YOUR ULTIMATE GUIDE TO THRIVING IN THE ED

# OXFORD HANDBOOK OF EMERGENCY MEDICINE

Jonathan P. Wyatt | Robert G. Taylor
Kerstin de Wit | Emily J. Hotton

The must-have handbook for emergency medicine

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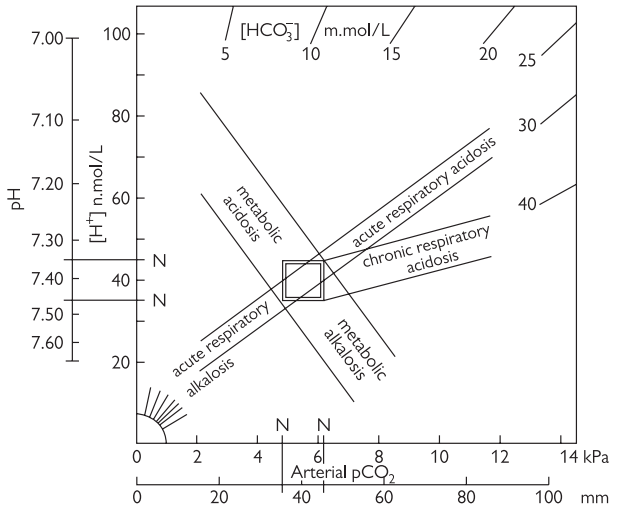


Acid-base nomogram in the interpretation of arterial blood gases

# Golden rules of Emergency Medicine

There are exceptions to every rule, but think very carefully before breaking the following:

## General Rules

- Turn up on time for every shift
- ED staff work as a team—thank members appropriately
- Always listen to nagging doubts
- Do not work beyond your expertise: when in doubt, seek senior advice
- If someone gives you advice, record what it was and who gave it
- Referral means referral and is usually a one-way process
- When making notes, write legibly, record times and print your name
- Always record what explanation and advice you give
- Avoid giving an opinion outside your expertise
- Always re-check drug doses (especially in children)

## Rules and your patient

- Allow patients to 'tell their story' or at least a summary of it
- Beware patients who are 'handed over' to you
- Treat patients as you would want to be treated
- Treat the patient (not just the investigation result)
- Do not bring patients back for a second opinion—get a first opinion
- Discuss with a senior if contemplating breaking patient confidentiality
- If a patient has ↓ GCS, check BMG
- Glass + skin wound = X-ray
- Beware using tourniquets on digits and limbs
- Check visual acuity for all eye problems
- X-ray high velocity eye injuries (eg hammering)
- Always check/document anatomical snuffbox tenderness in wrist injuries
- 'Worst headache ever' mandates exclusion of subarachnoid haemorrhage
- Call an anaesthetist early in possible airway burns
- Never assume ↓ GCS is due to alcohol alone (especially with head injury)
- Admit patients with even minor head injury and no one at home
- CT scan patients with head injury if they take anticoagulants
- Bleeding disorder + injury = discuss with a haematologist
- Do not place chest tubes through stab or bullet wounds
- Take it seriously if a parent says their baby (or child) is simply 'not right'
- Consider meningococcal disease with unexplained skin rashes
- Consider NAI in atypical paediatric presentations
- If NAI is a possibility, inform a senior and/or specialist at once
- Do not try to age bruises
- Ask about allergies before giving drugs

## Rules and you

- Ensure each shift contains regular refreshment breaks
- Do not try to 'work through' illness
- If you feel yourself becoming angry, take a deep breath and a short break
- If a fellow professional is rude, it may reflect stress on their part
- Each time you see a new condition, read up about it

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OXFORD HANDBOOK OF

# Emergency Medicine

FIFTH EDITION

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*Remembering especially Dr Rebecca Ovenden, née Steptoe (1984–2017), a much loved and respected colleague and friend.*

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# Abbreviations and symbols

| | |
|---|---|
| °C | degrees Centigrade |
| +ve | positive |
| –ve | negative |
| ± | plus or minus |
| > | greater than |
| < | less than |
| ≥ | equal to or greater than |
| ≤ | equal to or less than |
| ♂ | male |
| ♀ | female |
| ® | registered |
| ™ | trademark |
| ~ | approximately |
| ↑ | increase(d), increasing |
| ↓ | decrease(d), decreasing |
| ▶▶ | don't dawdle |
| ▶ | important |
| ➲ | cross-reference |
| 🖱 | online reference |
| α | alpha |
| β | beta |
| 5HT | 5-hydroxytryptamine |
| ABC | airway, breathing, circulation |
| ABG | arterial blood gas |
| AC | acromio-clavicular |
| ACE | angiotensin-converting enzyme |
| ACS | acute coronary syndrome |
| ACTH | adrenocorticotrophic hormone |
| ADH | antidiuretic hormone |
| AF | atrial fibrillation |
| AIDS | acquired immune deficiency syndrome |
| AIO | Ambulance Incident Officer |
| AIS | Abbreviated Injury Scale |
| AKI | acute kidney injury |
| ALS | Advanced Life Support |
| ALT | alanine aminotransferase |
| ALTE | apparent life-threatening event |
| ANDO | Allow Natural Death Order |
| AP | antero-posterior |
| APLS | Advanced Paediatric Life Support |
| APTT | activated partial thromboplastin time |
| ARDS | adult respiratory distress syndrome |
| ARF | acute renal failure |
| ASA | American Society of Anesthesiologists |
| AST | aspartate aminotransferase |
| ATLS | Advanced Trauma Life Support |
| ATP | adenosine triphosphate |
| AV | atrioventricular |
| BCG | bacille Calmette–Guérin |
| bd | twice daily |
| BE | base excess |
| BiPAP | bilevel positive airway pressure |
| BKPOP | below-knee plaster of Paris |
| BKWPOP | below-knee walking plaster of Paris |
| BLS | Basic Life Support |
| BMG | bedside strip measurement of venous/capillary blood glucose |
| BMI | body mass index |
| *BNF* | *British National Formulary* |
| *BNFC* | *British National Formulary for Children* |
| BNP | B-type natriuretic peptide |
| BP | blood pressure |
| BTS | British Thoracic Society |
| BURP | Backwards, Upwards, Rightwards Pressure |
| BZP | benzylpiperazine |
| $Ca^{2+}$ | calcium |
| CAMHS | child and adolescent mental health services |
| CBRN | chemical, biological, radiological, nuclear |
| CCU | coronary care unit |
| CDC | Centers for Disease Control |

| | |
|---|---|
| CIWA-Ar | revised Clinical Institute Withdrawal Assessment for Alcohol |
| CK | creatine kinase |
| CKD | chronic kidney disease |
| $Cl^-$ | chloride |
| cm | centimetre(s) |
| $cmH_2O$ | centimetre(s) of water (pressure) |
| CMV | cytomegalovirus |
| CN | chloroacetophenone |
| CNS | central nervous system |
| CO | carbon monoxide |
| $CO_2$ | carbon dioxide |
| COHb | carboxyhaemoglobin |
| COPD | chronic obstructive pulmonary disease |
| CPAP | continuous positive airway pressure |
| CPK | creatine phosphokinase |
| CPR | cardiopulmonary resuscitation |
| CRF | chronic renal failure |
| CRP | C-reactive protein |
| CRT | capillary refill time |
| CS | orthochlorobenzylidene malononitrile |
| CSF | cerebrospinal fluid |
| CT | computed tomography |
| cTnI | cardiac troponin I |
| cTnT | cardiac troponin T |
| CTPA | computed tomography pulmonary angiography |
| CVP | central venous pressure |
| CVS | cardiovascular system |
| CXR | chest X-ray |
| DC | direct current |
| Defra | Department for Environment, Food, and Rural Affairs |
| DIC | disseminated intravascular coagulation |
| DIPJ | distal interphalangeal joint |
| DKA | diabetic ketoacidosis |
| dL | decilitre(s) |
| DNA | deoxyribonucleic acid |
| DNACPR | Do Not Attempt CPR |
| DPG | diphosphoglycerate |
| DPL | diagnostic peritoneal lavage |
| DPT | diphtheria, pertussis, and tetanus |
| DVLA | Driver and Vehicle Licensing Agency |
| DVT | deep vein thrombosis |
| EBV | Epstein–Barr virus |
| ECG | electrocardiogram |
| ECT | electroconvulsive therapy |
| ED | emergency department |
| EDTA | ethylenediamine tetra-acetic acid |
| EEG | electroencephalogram |
| eGFR | estimated glomerular filtration rate |
| EMLA | eutectic mixture of local anaesthetics |
| ENT | ear, nose, and throat |
| EPAP | expiratory positive airway pressure |
| ERCP | endoscopic retrograde cholangiopancreatography |
| ESR | erythrocyte sedimentation rate |
| ET | endotracheal |
| $ETCO_2$ | end-tidal carbon dioxide |
| FAST | focussed assessment with sonography for trauma |
| FB | foreign body |
| FBC | full blood count |
| FFP | fresh frozen plasma |
| FG | French Gauge |
| FGM | female genital mutilation |
| $FiO_2$ | inspired oxygen concentration |
| FOB | faecal occult blood |
| ft | foot/feet |
| FTOCC | Fever, Travel, Occupation, Cluster and Contact |
| γ | gamma |
| G6-PD | glucose-6-phosphate dehydrogenase |
| g | gram(s) |
| G | gauge |
| GA | general anaesthesia/ anaesthetic |
| GCS | Glasgow Coma Score |
| GFR | glomerular filtration rate |
| GHB | gammahydroxybutyrate |
| GI | gastrointestinal |
| GMC | General Medical Council |

| | |
|---|---|
| GP | general practitioner |
| GTN | glyceryl trinitrate |
| GU | genitourinary |
| HAS | human albumin solution |
| Hb | haemoglobin |
| HCG | human chorionic gonadotrophin |
| HCM | hypertrophic cardiomyopathy |
| $HCO_3^-$ | bicarbonate |
| Hct | haematocrit |
| HDU | high dependency unit |
| HHS | hyperosmolar hyperglycaemic state |
| HIV | human immunodeficiency virus |
| $H_2O$ | water |
| HPV | human papillomavirus |
| hr | hour(s) |
| HTLV | human T-cell lymphotropic virus |
| Hz | hertz(s) |
| ICP | intracranial pressure |
| ICU | intensive care unit |
| IgA | immunoglobulin A |
| IgE | immunoglobulin E |
| IgG | immunoglobulin G |
| IHD | ischaemic heart disease |
| IM | intramuscular |
| INR | international normalized ratio (of prothrombin time) |
| IO | intra-osseous |
| IPAP | inspiratory positive airway pressure |
| IPJ | interphalangeal joint |
| IPPV | intermittent positive pressure ventilation |
| ISS | Injury Severity Score |
| IUD | intrauterine contraceptive device |
| IUS | intrauterine system |
| IV | intravenous |
| IVF | *in vitro* fertilization |
| IVI | intravenous infusion |
| IVRA | intravenous regional anaesthesia |
| IVU | intravenous urography |
| J | joule(s) |
| JVP | jugular venous pressure |
| $K^+$ | potassium |
| KCl | potassium chloride |
| KE | kinetic energy |
| kg | kilogram(s) |
| kPa | kilopascal(s) pressure |
| L | litre(s) |
| LA | local anaesthesia/ anaesthetic |
| LAD | left axis deviation |
| LBBB | left bundle branch block |
| LDH | lactate dehydrogenase |
| LET | lidocaine, epinephrine, tetracaine |
| LFTs | liver function tests |
| LMA | laryngeal mask airway |
| LMP | last menstrual period |
| LMWH | low-molecular weight heparin |
| LP | lumbar puncture |
| LSD | lysergic acid diethylamide |
| LV | left ventricle/ventricular |
| LVAD | left ventricular assist device |
| LVF | left ventricular failure |
| LVH | left ventricular hypertrophy |
| m | metre(s) |
| MAC | mid-arm circumference |
| MAOI | monoamine oxidase inhibitor |
| MAP | mean arterial pressure |
| max | maximum |
| MC | metacarpal |
| MCA | Mental Capacity Act |
| mcg | microgram(s) |
| MCPJ | metacarpophalangeal joint |
| MCV | mean corpuscular volume |
| MDDUS | Medical and Dental Defence Union of Scotland |
| MDMA | 3,4-methylene-dioxymetamphetamine |
| MDU | Medical Defence Union |
| MERS | Middle East respiratory syndrome |
| MERS-CoV | Middle East respiratory syndrome |
| mg | milligram(s) |
| $Mg^{2+}$ | magnesium |
| MI | myocardial infarction |
| min | minute(s) |

| | |
|---|---|
| MIO | Medical Incident Officer |
| mL | millilitre(s) |
| mm | millimetre(s) |
| mmHg | millimetre(s) of mercury pressure |
| mmol | millimole(s) |
| MMR | measles, mumps, and rubella |
| mOsm | milliosmole(s) |
| MRA | magnetic resonance angiography |
| MRI | magnetic resonance imaging |
| MRSA | meticillin-resistant *Staphylococcus aureus* |
| ms | millisecond(s) |
| MS | multiple sclerosis |
| MSU | mid-stream specimen of urine |
| MT | metatarsal |
| MTPJ | metatarsophalangeal joint |
| MUA | manipulation under anaesthesia |
| mV | millivolt(s) |
| $Na^+$ | sodium |
| NAI | non-accidental injury |
| NAIR | National Arrangements for Incidents involving Radioactivity |
| NAPQI | *N*-acetyl-*p*-benzoquinone imine |
| ND | notifiable disease |
| NEWS2 | National Early Warning Score (revised 2017) |
| NG | nasogastric |
| NHS | National Health Service |
| NICE | National Institute for Health and Care Excellence |
| NIHSS | National Institutes of Health Stroke Scale |
| NIV | non-invasive ventilation |
| $N_2O$ | nitrous oxide |
| NPIS | National Poisons Information Service |
| NPS | novel psychoactive substance |
| NSAID | non-steroidal anti-inflammatory drug |
| NSPCC | National Society for the Prevention of Cruelty to Children |
| NSTEMI | non-ST segment elevation myocardial infarction |
| $O_2$ | oxygen |
| OA | osteoarthritis; occiput anterior |
| OCP | oral contraceptive pill |
| od | once daily |
| OPG | orthopantomogram |
| ORIF | open reduction and internal fixation |
| PA | postero-anterior |
| PACS | picture archiving and communication system |
| PAN | polyarteritis nodosa |
| PCI | percutaneous coronary intervention |
| $pCO_2$ | arterial partial pressure of carbon dioxide |
| PCR | polymerase chain reaction |
| PE | pulmonary embolus/embolism |
| PEA | pulseless electrical activity |
| PEEP | positive end-expiratory pressure |
| PEFR | peak expiratory flow rate |
| PEG | percutaneous endoscopic gastrostomy (tube) |
| PERT | Paediatric Emergency Response Team |
| PGL | persistent generalized lymphadenopathy |
| PICU | paediatric intensive care unit |
| PID | pelvic inflammatory disease |
| PIPJ | proximal interphalangeal joint |
| PO | *per os* (orally/by mouth) |
| $pO_2$ | arterial partial pressure of oxygen |
| $PO_4^{2-}$ | phosphate |
| POP | plaster of Paris |
| PPE | personal protective equipment |
| PPI | proton pump inhibitor |
| PR | per rectum |
| PrEP | pre-exposure prophylaxis |
| PRF | patient report form |
| PRICE | Protection/Rest/Ice/Compression/Elevation |
| PRN | *pro re nata* (as required) |

| | |
|---|---|
| Ps | probability of survival |
| PSP | primary spontaneous pneumothorax |
| PV | per vaginam |
| qds | four times daily |
| qSOFA | quick SOFA |
| RA | rheumatoid arthritis |
| RAD | right axis deviation |
| RBBB | right bundle branch block |
| RBC | red blood cell |
| RCEM | Royal College of Emergency Medicine |
| Rh | rhesus |
| RIMA | reversible inhibitor of monoamine oxidase A |
| RNA | ribonucleic acid |
| RMO | responsible medical officer |
| ROSC | restoration of spontaneous circulation |
| ROTEM | rotational thromboelastometry |
| RR | respiratory rate |
| RSI | rapid sequence induction/ intubation |
| RSV | respiratory syncytial virus |
| rtPA | recombinant tissue plasminogen activator |
| RTS | Revised Trauma Score |
| RV | right ventricle/ventricular |
| s | second(s) |
| SA | sino-atrial |
| SARS | severe acute respiratory syndrome |
| SC | subcutaneous |
| SCIWORA | spinal cord injury without radiographic abnormality |
| SCRA | synthetic cannabinoid receptor agonist |
| SIDS | sudden infant death syndrome |
| SIGN | Scottish Intercollegiate Guidelines Network |
| SL | sublingual |
| SLE | systemic lupus erythematosus |
| SOFA | Sequential Organ Failure Assessment |
| SPECT | single-photon emission computed tomography |
| $SpO_2$ | arterial oxygen saturation |
| SSP | secondary spontaneous pneumothorax |
| SSRI | selective serotonin reuptake inhibitor |
| STEMI | ST segment elevation myocardial infarction |
| STI | sexually transmitted infection |
| SUDI | sudden unexplained death in infancy |
| SUDIC | sudden unexpected death in infancy and childhood |
| SVT | supraventricular tachycardia |
| T° | temperature |
| $T_3$ | tri-iodothyronine |
| $T_4$ | thyroxine |
| TAC | tetracaine, adrenaline, and cocaine |
| TACO | transfusion-associated circulatory overload |
| TB | tuberculosis |
| tds | three times daily |
| TEG | thromboelastography |
| TEP | Treatment Escalation Plans |
| TFCC | triangular fibrocartilage complex |
| TFTs | thyroid function tests |
| TIA | transient ischaemic attack |
| TIG | tetanus immune globulin |
| TIMI | thrombolysis in myocardial infarction |
| TORCH | *Toxoplasma*, rubella, CMV, herpes |
| tPA | tissue plasminogen activator |
| TSH | thyroid-stimulating hormone |
| TWOC | Trial WithOut Catheter |
| U | unit(s) |
| U&E | urea and electrolytes |
| UK | United Kingdom |
| URTI | upper respiratory tract infection |
| USS | ultrasound scan |
| UTI | urinary tract infection |
| V | volt(s) |
| VA | visual acuity |
| VBG | venous blood gas |
| VF | ventricular fibrillation |

| | |
|---|---|
| VHF | viral haemorrhagic fever |
| V/Q | ventilation–perfusion (scan) |
| VT | ventricular tachycardia |
| VTE | venous thromboembolism |
| vW | von Willebrand |
| WBC | white blood cell |
| WCC | white cell count |
| WPW | Wolff–Parkinson–White (syndrome) |
| y | year(s) |

# Normal values

Note that 'normal' values in adults may vary slightly between labs. Normal values in pregnancy are shown in ➔ Prescribing in pregnancy, p. 594.

## Arterial blood gas analysis

| | |
|---|---|
| $H^+$ | 35–45nanomol/L |
| pH | 7.35–7.45 |
| $pO_2$ (in air) | >10.6kPa, 75–100mmHg |
| $pCO_2$ | 4.5–6.0kPa, 35–45mmHg |
| Bicarbonate | 24–28mmol/L |
| Base excess | ± 2mmol/L |

## Biochemistry

| | |
|---|---|
| Alanine aminotransferase (ALT) | 5–35 IU/L |
| Albumin | 35–50g/L |
| Alkaline phosphatase | 30–300 IU/L |
| Amylase | 0–180 Somogyi U/dL |
| Aspartate transaminase (AST) | 5–35 IU/L |
| Bicarbonate | 24–30mmol/L |
| Bilirubin | 3–17 micromoles/L |
| Calcium (total) | 2.12–2.65mmol/L |
| Calcium (ionized) | 1–1.25mmol/L |
| Chloride | 95–105mmol/L |
| Creatine kinase (CK) | 25–195 IU/L |
| Creatinine | 70–150 micromoles/L |
| C-reactive protein (CRP) | <10mg/L |
| Glucose (fasting) | 3.5–5.5mmol/L |
| α-glutamyl transpeptidase (♀) | 7–33 IU/L |
| (♂) | 11–51 IU/L |
| Magnesium | 0.75–1.05mmol/L |
| Osmolality | 278–305mOsm/kg |
| Potassium | 3.5–5.0mmol/L |
| Sodium | 135–145mmol/L |
| Urea | 2.5–6.7mmol/L |
| Urate (♀) | 150–390 micromoles/L |
| (♂) | 210–480 micromoles/L |

## Haematology

| | |
|---|---|
| RBC (♀) | 3.9–5.6 × $10^{12}$/L |
| (♂) | 4.5–6.5 × $10^{12}$/L |
| Hb (♀) | 115–160g/L |
| (♂) | 135–180g/L |
| Hct (♀) | 0.37–0.47 |
| (♂) | 0.40–0.54 |
| MCV | 76–96fL |
| WCC | 4.0–11.0 × $10^{9}$/L |
| Neutrophils | 2.0–7.5 × $10^{9}$/L (40–75% of WCC) |
| Lymphocytes | 1.5–4.0 × $10^{9}$/L (20–40% of WCC) |
| Monocytes | 0.2–0.8 × $10^{9}$/L (2–10% of WCC) |
| Eosinophils | 0.04–0.40 × $10^{9}$/L (1–6% of WCC) |
| Basophils | <0.1 × $10^{9}$/L (<1% of WCC) |
| Platelets | 150–400 × $10^{9}$/L |
| Prothrombin time (factors I, II, VII, X) | 12–15s |
| APTT (factors VII, IX, XI, XII) | 23–42s |

## International normalized ratio (INR) therapeutic targets

| | |
|---|---|
| 2.0–3.0 | (for treating DVT and PE) |
| 2.5–3.5 | (embolism prophylaxis for AF) |
| 3.0–4.5 | (recurrent thromboembolic disease, arterial grafts, and prosthetic valves) |

| | | |
|---|---|---|
| ESR | (women) | < $^{(\text{age in years}+10)}/_{2}$ mm/hr |
| | (men) | < $^{(\text{age in years})}/_{2}$ mm/hr |

## Metric conversion

*Length*

| | |
|---|---|
| 1m = 3 feet 3.4 inches | 1 foot = 0.3048m |
| 1cm = 0.394 inch | 1 inch = 25.4mm |

*Weight*

| | |
|---|---|
| 1kg = 2.20 pounds | 1 stone = 6.35kg |
| 1g = 15.4 grains | 1 pound = 0.454kg |
| | 1 ounce = 28.4g |

*Volume*

1L = 1.76 UK pints = 2.11 US liquid pints
1 UK pint = 20 fluid ounces = 0.568L
1 US liquid pint = 16 fluid ounces = 0.473L
1 teaspoon = ~ 5mL
1 tablespoon = ~ 15mL

*Temperature*

T° in °C = (T° in Fahrenheit – 32) × 5/9

*Pressure*

1kPa = 7.5mmHg

# Chapter 1

# General approach

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# The emergency department

## Role of the emergency department

The emergency department (ED) occupies a key position in terms of the interface between primary and secondary care. It has a high public profile. Many patients attend without referral, but some are referred by minor injury units, general practitioners (GPs), other medical practitioners, and National Health Service (NHS) 111/NHS 24/NHS Direct Wales.

The ED manages patients with a huge variety of medical problems. Many of the patients who attend have painful and/or distressing disorders of recent origin.

*ED priorities*

- To make life-saving interventions.
- To provide analgesia.
- To identify relevant issues, investigations, and commence treatment.
- To decide upon need for admission or discharge.

*ED staff*

A key feature of the ED is the way that the staff work together as a team. Traditional roles are often blurred, with the important issue being what clinical skills a member of staff is capable of. Staff include:

- Nurses (including nurse practitioners, nurse consultants, health care assistants) and Advanced Care Practitioners.
- Doctors (permanent and fixed-term).
- Reception and administrative staff (receptionists, secretaries, managers).
- Radiographers, including reporting radiographers.
- Other specialist staff (eg psychiatric liaison nurses, plaster technicians, physiotherapists, paramedic practitioners, physician assistants, occupational therapists, clinic/ED ward staff).
- Supporting staff (security, porters, cleaners, police).

## The facilities of an ED

Most departments have the following areas:

- Resuscitation area—including a paediatric area (often separate).
- Majors area (sometimes called 'trolley area').
- Minors area—including theatre, plaster room, eye room.
- Paediatric area—with a separate waiting room.
- Adjacent or embedded imaging (X-ray department and CT scanner).
- Reception and waiting room.
- Relatives room (for seriously ill patients).
- Staff room.
- Teaching/resource room.
- Offices.

Depending upon the nature and configuration of the service which is being delivered, many EDs have other areas within or adjacent which are designated for a specific purpose (eg clinic area, short stay ward/clinical decision unit).

# Emergency medicine beyond the ED

## Additional roles of ED staff

In addition to their roles in providing direct clinical care in their departments, many ED staff provide related clinical care in other settings and ways, including in short stay wards, in outpatient follow-up clinics (eg for burns, soft tissue injuries), and through planned theatre lists (eg for wrist fracture manipulation) and telemedicine advice (for satellite and minor injury units).

## Short stay wards/clinical decision units

The intention is for admissions to these units to be short—most of the patients admitted are observed for relatively short periods (<24hr) and undergo assessments at an early stage to decide about the need for discharge or longer-term admission. The range of conditions which may be appropriately managed varies between units but may include the following:

- Head and neck injuries.
- Poisoning, self-harm, and some psychiatric presentations.
- Alcohol and/or drug intoxication.
- Falls (especially in the elderly), with no injuries requiring surgery.
- Anaphylaxis.
- Drowning and electrocution injuries.
- Low-risk chest pain.
- Soft tissue infection.
- Asthma.
- Low back pain (including suspected cauda equina syndrome).
- 'Social problems'.

The clinical staff required to provide support to a short stay ward depends upon the exact service being delivered, but typically includes occupational therapists, physiotherapists, psychiatric liaison staff, social workers, and alcohol and drug specialists.

## Emergency medicine in other settings

As the delivery of emergency care continues to develop and evolve, patients with emergency problems are now being assessed and treated in a variety of settings. In the overstretched, overcrowded world of acute/emergency medicine, different ways of working are being implemented, which enables ED clinicians, GPs, acute physicians, and other specialists to work in adjacent areas with a variety of different names (eg urgent care centre, primary care unit, walk-in centre, same-day emergency care, clinical decision unit, medical assessment areas).

Traditional distinctions between emergency medicine, acute medicine, and primary care have become blurred.

# Patient flow

## Discharge from the ED

To work efficiently, the overall hospital system needs to enable easy flow of patients out of the ED. Options available for continuing care of patients who leave the ED include:

- Discharge home with no follow-up.
- Discharge home with GP and/or other community support/follow-up.
- Discharge with hospital clinic follow-up arranged.
- Admission to hospital for further investigation and treatment.
- Transfer to another hospital with more specialist facilities.

Aim to make an early decision about the likely 'disposal' of each patient—this is very helpful for senior staff and managers who are focussed on patient flow.

## Patient flow

The flow of patients through the ED depends upon a number of factors, most particularly:

- The rate of patients arriving.
- The waiting time to be seen.
- The time to 'process' (examine/assess/investigate/decide a plan).
- The time to discharge/admit.

A problem with any of these factors can result in overcrowding in the ED, which, in turn, adds further delays to the process. Most EDs are designed and staffed to cope with an average rate of patients arriving, and so it is natural that, at some times, there will be fluctuations in the waits to be seen, which will occasionally rise. However, a bigger problem in the United Kingdom has become 'exit block' from the ED, whereby there are no beds available in the hospital for patients to be admitted to. Sometimes the block can be specific to certain areas or specialties (eg surgery or ICU).

At times of high demand and/or extreme pressure, some EDs have systems in place whereby additional support (eg medical and nursing staff) can be obtained from other parts of the hospital or where some elements of the usual procedures for processing patients (eg documentation in the ED) can be reduced.

## Government targets

Long waiting times to be seen in the UK EDs prompted the introduction of quality indicators, most notably the '4-hour target', which was adopted variably throughout the UK. Often misquoted in the press as '4hrs to be seen', it was actually set out as a percentage (eg 95%) to be seen, treated, and discharged/admitted within 4hrs. Lack of hospital beds, resulting in poor flow for patients admitted into the hospital, has impacted dramatically on the ability of hospitals to deliver on these targets. Irrespective of the politics, long stays in the ED impact very negatively on patient outcomes.

# Patient safety: overcrowding

## Overcrowding and queuing

Overcrowding disrupts the usually smooth processes which are in place, risking the safety of patients. Although the timely and efficient processing of patients can help to keep patients safe, experience suggests that when (especially junior) staff try to work too quickly or cut corners, mistakes are made. Do not compromise the quality of care by a less rigorous assessment of a patient simply because the department is busy.

### *Privacy and dignity*

Overcrowding inevitably adversely affects privacy and dignity, especially for patients arriving on stretchers. As capacity becomes saturated, patients end up waiting on trolleys in corridors. Many EDs have recognized that it is safer for selected patients to queue out waiting for a bed than for unselected patients to queue to get in. Whilst it may be necessary to undertake a limited assessment in a corridor, do not undress patients to examine them in a corridor.

## Patient safety checklist for an overcrowded department

The following is an example of the way that one ED has attempted to combat some of the risks posed by overcrowding:

- Provide reassurance to patients who arrive to a crowded ED.
- Explain to patients that although the environment is not ideal, they can expect to be looked after with the same quality of care from the ED team.
- Focus on minimizing any compromise of patient privacy and dignity.
- Provide a simple verbal explanation and an apology for the ED crowding situation (and document this in the notes).
- Hand out the written ED crowding information sheet to patients and their relatives.
- Ask patients for permission before taking a history, undertaking a non-intimate examination, or taking observations/bloods outside of an ED cubicle space. Explain the benefits of early diagnosis and treatment and apologize for the lack of privacy.
- Use a private area if requested by the patient.
- Use a private area for any examination or investigation where the patient needs to get undressed (eg chest/abdominal examination, recording an ECG).
- Document clearly in the notes any examinations which are suboptimal due to the inability to adequately assess patients in the corridor. Complete the examination fully at the earliest opportunity.
- Ensure routine nursing and clinical care (including analgesia, medication, regular observations, toileting, food and drink) continue wherever the patient is located. Keep patients informed about the progress of their care.

# Note keeping

## General aspects

The importance of making accurate notes cannot be over-emphasized. The principal role of ED patient notes is to record (and communicate with future carers) the history, investigations, possible diagnoses, and treatment plan. Rather than making comprehensive notes on every aspect of the patient's care, aim to focus notes in the ED on those important issues that need to be addressed within the first few hours—leave the 'comprehensive clerking' to doctors on the ward.

### *Medicolegal considerations*

Clinicians each treat hundreds of patients every month. With the passage of time, it is impossible to remember all aspects relating to these cases, yet it may be necessary to give evidence in court about them years after the event. The only reference will be the notes made much earlier. Medicolegally, the ED record is also the prime source of evidence in negligence cases. If the notes are deficient, it may not be feasible to defend a claim, even if negligence has not occurred.

The *Data Protection* and *Access to Medical Records Acts* give patients right of access to their medical notes. Remember, whenever writing notes, that the patient may in the future read exactly what has been written. Follow the following basic general rules.

## Layout

### *Presenting complaint*

Indicate from whom the history has been obtained (eg the patient, a relative, or the paramedic). Avoid attributing events to certain individuals (eg the patient was struck by 'Joe Bloggs').

### *Previous relevant history*

Note recent ED attendances, together with other information which may be available within the hospital electronic record system—in particular, check recent letters and investigation results. Previous ECGs can be very useful to compare against. Take a relevant social history (which needs to be detailed when there are potential concerns about the safety of later discharging the patient).

### *Current medications*

Many patients bring their medication (and/or list) to hospital. It is also often possible to cross-check against online/GP records. Remember to ask about non-prescribed drugs. Enquire about allergies and document the nature of any reaction.

### *Examination findings*

As well as +ve features, document relevant –ve findings (eg the absence of neck stiffness in a patient with headache and pyrexia). Always document the side of the patient which has been injured. For upper limb injuries, note whether the patient is left- or right-handed. Use 'left' and 'right', not 'L' and 'R'. Document if a patient is abusive or aggressive, but avoid non-medical judgemental terms (eg 'drunk').

*Investigation findings*

Record these clearly, plus what is still outstanding.

*Working diagnosis*

For patients being admitted, this may be a differential diagnostic list. Sometimes a problem list can help.

*Treatment given*

Document drugs, including the dose, time, and route of administration. Include medications given in the ED, as well as therapy to be continued (eg course of antibiotics). For patients who are being referred for admission, whilst it is not necessary to prescribe all regular medications in the ED, ensure that important medications are prescribed—these include drugs for epilepsy, Parkinson's disease, and diabetes, together with antibiotics.

Record other treatments in detail such as the number and type of sutures or staples used for wound closure (eg '5 × 6/0 nylon sutures').

*Advice and follow-up arrangements*

Document if the patient and/or relative is given a preprinted advice sheet (eg 'head injury advice'). Indicate when/if the patient needs to be reviewed (eg 'see GP in 5 days for suture removal').

## Basic rules

- Write legibly, ideally in black ink which photocopies well.
- Always date and time the notes.
- Sign the notes, and print your name and status below.
- Make your notes concise and to the point.
- Use simple line drawings or preprinted sheets for wounds/injuries.
- Avoid idiosyncratic abbreviations.
- Never make rude or judgemental comments.
- Always document the name, grade, and specialty of any doctor from whom you have received advice.
- When referring or handing a patient over, *always* document the time of referral/handover, together with the name, grade, and specialty of the receiving doctor.
- Inform the GP by letter/email (see ➲ Liaising with GPs, p. 12), even if the patient is admitted. Most EDs have computerized systems that generate letters. In complex cases, send also a copy of ED notes, with the results of investigations, and consider giving the patient a copy as well.

## Pro formas

Increasing emphasis on evidence-based guidelines and protocols has been associated with the introduction of pro formas for many patient presentations and conditions. Whilst they have some advantages, bear in mind the fact that, for some patients, satisfactory completion of a pro forma may not adequately capture all of the information required.

## Electronic records

Some departments (and hospitals) are now completely paperless, which has obvious advantages in terms of legibility, storage, and later access. When completing electronic records, follow the same principles as those outlined for written records.

# Radiological requests

## General aspects

The *Royal College of Radiologists'* booklet *iRefer: Making the Best Use of Clinical Radiology* (eighth edition, London, Royal College of Radiologists, 2017) contains useful information and is recommended.

- An X-ray is no substitute for a careful, thorough clinical examination. It is usually unnecessary to request X-rays to confirm the clinical diagnosis of uncomplicated fractures of the nose, the coccyx, a single rib, or toes (other than the big toe).
- If in doubt about the need for X-rays/CT or the specific test required, consider relevant guidelines (eg Ottawa rules for ankle injuries—see ➲ Approach to ankle injuries, p. 498; the National Institute for Health and Care Excellence (NICE) guidelines for CT scanning in head injuries—see ➲ Head injury: imaging, p. 370) and/or discuss with senior ED staff or the radiologist. The dose of radiation involved may need to be weighed up against the risk of missing or delaying making the diagnosis, in the context of the availability of the tests.
- When requesting imaging, describe the indication/mechanism of injury, clinical findings, including the side involved (right or left—spelt out in full, not abbreviated), and the suspected clinical diagnosis. This is important for the radiologist reporting the films without the advantage of being able to examine the patient.
- Do not worry about specifying exactly which X-ray views are required. The radiographer will know the standard views that are needed, based on the information provided (eg antero-posterior (AP) + simplified apical oblique views for a patient with suspected anterior shoulder dislocation). In unusual cases, discuss with senior ED staff, the radiographer, or the radiologist.
- Always consider the possibility of pregnancy in women of child-bearing age before requesting an X-ray (or CT scan) of the abdomen, pelvis, lumbar spine, hips, or thighs. If the clinical indication for X-ray/CT scanning is overriding, tell the radiographer, who will attempt to shield the fetus/gonads. If the risks/benefits of X-ray/CT scanning in pregnant or possibly pregnant women are not obvious, consult senior ED or radiology staff.
- Magnetic resonance imaging (MRI) can be dangerous in patients who have metalwork *in situ*. Its effects in pregnancy have not been established, but it is usually regarded as being safe in pregnancies after 3 months.
- Ultrasound scanning (USS) is particularly useful at identifying radiolucent foreign bodies (FBs) (see ➲ Approach to foreign bodies, p. 413).

## X-ray reporting system

Most hospitals have systems whereby all ED X-rays are reported by a specialist within 24hr. Reports of any missed abnormalities are returned to the ED for the attention of senior staff, so that appropriate action can be taken.

### *System for identifying abnormalities*

In addition to the formal reporting system described above, a system is commonly used whereby the radiographer taking the films applies a label to the image if they identify an abnormality. This alerts other clinical staff to the possibility of abnormal findings.

# Triage

The nature of ED work means that a sorting system is required to ensure that patients with the most immediately life-threatening conditions are seen first. A triage process aims to categorize patients based on their medical needs and the available departmental resources. One process used in the UK is the National Triage Scale (see Table 1.1).

**Table 1.1** National Triage Scale (UK)

| National Triage Scale | Colour | Time to be seen by doctor |
|---|---|---|
| 1 Immediate | Red | Immediately |
| 2 Very urgent | Orange | Within 5–10min |
| 3 Urgent | Yellow | Within 1hr |
| 4 Standard | Green | Within 2hr |
| 5 Non-urgent | Blue | Within 4hr |

On arrival in the ED, a patient is assessed by a dedicated triage nurse (a senior, experienced individual with considerable common sense). This nurse provides any immediate interventions that are needed (eg elevating injured limbs, applying ice packs or splints, and giving analgesia) and initiates investigations to speed the patient's journey through the department (eg ordering appropriate X-rays). The Royal College of Emergency Medicine (RCEM) guidelines are that patients should not have to wait >15min to be triaged. It is a brief assessment which should take no more than a few minutes.

Three points require emphasis:

- Triage is a dynamic process. The urgency (and hence triage category) with which a patient requires to be seen may change with time. For example, a middle-aged man who hobbles in with an inversion ankle injury is likely to be placed in triage category 4 (green). If in the waiting room, he becomes pale and sweaty and collapses with chest pain, he would require prompt re-triage into category 1 (red).
- Placement in a triage category does not imply a diagnosis, or even the lethality of a condition.
- Triage has its own problems. In particular, patients in non-urgent categories may wait inordinately long periods of time, whilst patients who have presented later, but with conditions perceived to be more urgent, are seen before them. Patients need to be aware of this and be informed of likely waiting times. Uncomplaining elderly patients can sometimes be poorly served by the process.

The triage process has evolved to include an initial assessment of the physiological status (NEWS2—see ➔ National Early Warning Score ('NEWS 2'), p. 61), which can assist in the identification of sick patients and also of deterioration of patients after arrival. Other systems aim to tackle the issues of patients waiting to be seen, including 'see and treat' which aims to enable patients to be assessed and treated immediately by a senior practitioner.

# Discharge, referral, and handover

Most patients seen in the ED are examined, investigated, treated, and discharged home, either with no follow-up or with advice to see their GP (for suture removal, wound checks, etc.). Give these patients (and/or attending relative/friend) clear instructions on when to attend the GP's surgery and an indication of the likely course of events, as well as any features that they should look out for to prompt them to seek medical help prior to this.

*Formal written instructions* are useful for patients with a range of conditions (eg minor head injury—see ➲ Discharging patients, p. 375; those with limbs in plaster of Paris (POP) or other forms of cast immobilization—see ➲ Casts and their problems, pp. 430–1; low back pain—see ➲ Atraumatic low back pain, pp. 508–9; spontaneous pneumothorax—see ➲ Spontaneous pneumothorax, pp. 118–20).

The referral of patients to an inpatient team can cause considerable anxiety, misunderstanding, and potential conflict between ED staff and other disciplines. Before making the referral, consider the following.

### Is it appropriate to refer this patient to the inpatient team?

Usually, this will be obvious. For example, a middle-aged man with a history of crushing chest pain and an ECG showing an acute myocardial infarction (MI) clearly requires urgent management in the ED and rapid admission for further investigation and treatment. Similarly, an elderly lady who has fallen, is unable to weight-bear, and has a fractured neck of femur will require analgesia, inpatient care, and surgery.

However, difficult situations occur where the clinical situation is less clear, eg a man who experienced 4–5min of atypical chest pain, has a normal ECG and chest X-ray (CXR), and is anxious to go home, or a patient who has no apparent fracture on X-ray but is struggling to weight-bear.

### Is there appropriate information to make this decision?

This requires a balance between availability, time, and appropriateness. In general, simple investigations which rapidly give the diagnosis, or clues to it, are all that are needed. These include ECGs, arterial blood gas (ABG)/venous blood gas (VBG), and plain X-rays. It is relatively unusual to have to wait for the results of investigations such as full blood count (FBC), urea and electrolytes (U&E), and liver function tests (LFTs) before referring a patient, since these rarely alter the immediate management. Simple trolleyside investigations are often of great value, eg Stix estimations of blood glucose (BMG) and urinalysis. If complicated investigations are needed, then referral for inpatient or outpatient specialist care is often required.

### Has the patient had appropriate treatment pending admission?

Do not forget, or delay, in providing analgesia. Treat every patient in pain appropriately as soon as possible. A patient does not have to 'earn' analgesia. Never delay analgesia to allow further examination or investigation. Concern regarding masking of signs or symptoms (eg in a patient with an acute abdomen) is inhumane and incorrect.

*Note*: clinical responsibility for a patient in the ED usually passes over to the admitting team once the patient has been seen by that team.

## How to refer patients

Referral is often by telephone, which can create problems. Adopt the following approach:

- Introduce yourself and ask for the name and grade of the specialist.
- Give a clear, concise summary of the history, investigations, and treatment that you have already undertaken.
- Early in the discussion, say clearly whether you are making a referral for admission or requesting a specialist opinion (it is usually better to obtain a senior ED opinion before a specialist one). With ever increasing pressure on hospital beds, inpatient teams can be reluctant to come and see patients, and may appear to be happier to give advice over the phone to avoid admission. If, however, the patient needs to be admitted, then clearly indicate this. If, for whatever reason, this is declined, do not get cross, rude, or aggressive, but contact senior ED medical staff to speak to the specialist team.
- When the specialist team comes to see the patient, or the patient is admitted directly to a ward, ensure the ED notes are complete and legible. Make sure that there is a list of the investigations already performed, together with the available results and, crucially, a list of investigations whose results remain outstanding. Similarly, summarize the treatment already given and the response.
- Encourage inpatient specialists who see patients in ED to write their findings and management plan in the notes.

## Referring to a specialist at another hospital

An increasing number of patients require discussion with a specialist at a remote hospital (eg regional neurosurgeon, plastic surgeon).
The two questions to ask the specialist are:

- Does the patient require transfer and if so, when?
- What treatment is required (whether transferred or not)?

Follow established protocols for referral/discussion. Increasingly, telephone discussions are being replaced by referral online, with systems in place to enable images (eg of skin burns) to be transferred securely.

## Handing over patients

*Dangers of handing over*

Handing over a patient to a colleague (usually because it is the end of the shift) is fraught with danger. It is easy for patients to be neglected or to receive suboptimal or delayed treatment. Hand over the patient carefully to the doctor who is taking over, update the electronic system/register as appropriate, and inform the nursing staff.

*How to hand over*

Include in the handover relevant aspects of the history and examination, investigation results, and treatment undertaken. Complete records on the patient as soon as possible. Note the time of handover and the name of the doctor or nurse handed over to. When accepting a 'handed-over patient' at the start of a shift, spend time establishing exactly what has happened so far. Finally, it is courteous (and will prevent problems) to tell the patient that their further care will be performed by another doctor or nurse.

## Liaising with GPs

Despite changes in the way that care (particularly out of hours) is delivered, GPs still have a pivotal role in co-ordinating medical care. Often the GP knows more than anyone about the past history, the social and family situation, and recent events of their patient's management. Therefore, consider contacting the GP when these aspects are relevant to the patient's ED attendance or where considerations of admission or discharge cannot be resolved during the consultation and by reviewing old online records.

Every attendance is followed routinely by a letter/email to the GP, detailing the reason(s) for presentation, clinical findings and relevant investigations, treatment given, and follow-up arrangements.

If a patient dies, contact the GP without delay—to provide a medical contact and assistance to the bereaved family, to prevent embarrassing experiences (eg letters requesting clinic attendances), and out of courtesy, because the GP is the patient's primary medical attendant. Finally, the GP may be asked to issue a death certificate by the Coroner (in Scotland, the Procurator Fiscal), following further enquiries.

Aim to contact the GP prior to the discharge of a patient where early follow-up (ie within the next 24–72hr) is required. This may occur with elderly patients where there is uncertainty about the home situation and their ability to manage. A typical example is an elderly woman with a Colles' fracture of her dominant wrist who lives alone. The ED management of this patient is relatively simple (see ➲ Colles' fracture, pp. 454–5). However, manipulating a Colles' fracture into a good position, supporting it in an adequate cast, and providing analgesia are only one facet of care. The GP may know that the patient has supportive relatives or neighbours who will help with shopping and cooking and who will help her to bathe and dress. The GP and the primary care team may be able to supplement existing support and check that the patient is coping. Equally, the GP may indicate that with additional home support (eg home helps, meals, district nurses), the patient could manage. Alternatively, the GP may indicate that the Colles' fracture merely represents the final event in an increasingly fragile home situation and that the patient will require hospital admission, at least in the short term.

For the same reasons, a GP who refers a patient to the ED and indicates that the patient requires admission does so in the full knowledge of that patient's circumstances. Always contact the GP if it is contemplated that such a patient is to be discharged—preferably after senior medical consultation.

Finally, remember that GPs are also under considerable pressure. Some situations may appear to reflect the fact that a patient has been referred inappropriately or the patient may report that they have tried to contact their GP unsuccessfully. Rather than irately ringing the practice and antagonizing them, inform the ED consultant who can consider this constructively and appropriately in a suitable environment.

# Telephone advice

## Advice for the public

Members of the public often try to call their local ED in an attempt to request medical advice. Most departments have a system in place whereby such requests are automatically redirected to telephone and/or Internet sources of help, including NHS 111 in England, NHS 24 in Scotland, or NHS Direct in Wales.

Not infrequently, patients who have been recently discharged (or their relatives) will call the department for advice on how to manage further symptoms, medication, or other treatment. Most EDs will consider these as their responsibility and will try to help the caller. Approach these calls in exactly the same way as a face-to-face consultation. Before giving any information over the phone, first establish who is calling and that the patient has given consent to discuss his/her medical details.

There is usually a book to record this information in. Formally document details of the call, including:

- The date and time of the call.
- The caller's telephone number.
- The caller's relationship to the patient.
- The patient's name, age, and sex.
- The nature of the problem.
- The advice given.

## Telephone advice calls from other health professionals

Other health professionals (eg paramedics and GPs) often telephone requesting advice regarding the management of patients in their care. Ensure that this advice is given by experienced ED staff and that the details are recorded in the appropriate book.

## Telemedicine

Increasingly, emergency health care is provided by integrated networks, which include EDs, minor injuries units, radiology departments, and GP surgeries connected by telemedicine links. This has advantages in remote or rural settings, enabling a wide range of injuries and other emergencies to be diagnosed and treated locally.

The combination of video and teleradiology may allow a decision to be made and explained directly to the patient. A typical example is whether a patient with an isolated Colles' fracture needs to have a manipulation of the fracture. Expertise is required to undertake telemedicine consultations safely. Ensure that this specialist advice is given by senior/experienced ED staff and that it is carefully documented.

## Liaising with the ambulance crew

Paramedics and ED staff have a close professional relationship. Paramedics and ambulance staff are professionals who work in conditions that are often difficult and sometimes dangerous. It is worth taking an off-duty day to accompany a crew during their shift to see the problems they face.

In the UK, a patient brought to the ED by ambulance will routinely have a patient report form (PRF) (see Fig. 1.1). This is usually completed by the crew at the scene and in transit, and printed on arrival at hospital. The information on these forms can be invaluable. In particular, the time intervals between the receipt of the 999 call and arrival at the scene and at hospital provide a time framework within which changes in the patient's clinical condition can be placed and interpreted.

The initial at-scene assessment will include details of the use of seat belts, airbags, crash helmets, etc., and is particularly valuable when amplified by specifically asking the crew about their interpretation of the event, likely speeds involved, types of vehicle, etc.

The clinical features of the Glasgow Coma Score (GCS), pulse rate, blood pressure (BP), and respiratory rate (RR) form baseline values from which trends and response to treatment can be judged. Useful aspects in the history/comments section include previous complaints, current medications, etc., which the crew may have obtained from the patient, relatives, or friends. The PRF will also contain important information about oxygen ($O_2$), drugs, and/or intravenous (IV) fluids administered, and the response to these interventions. Before the crew leave the department, confirm that they have provided all relevant information.

Do not be judgemental about the crew's performance. Remember the constraints under which they operate. Without the benefits of a warm environment, good lighting, and sophisticated equipment, it can be exceedingly difficult to make accurate assessments of illness or injury severity, or to perform otherwise simple tasks (eg airway management and IV cannulation).

Do not dismiss the overall assessment of a patient made by an experienced crew. Whilst the ultimate diagnosis may not be clear, their evaluation of the potential for life-threatening events is often extremely perceptive. Equally, take heed of their description (and photographs) of crash scenes. They will have seen far more than most ED staff, so accept their greater experience.

Most ambulance staff are keen to obtain feedback, both about specific cases and general aspects of medical care. Like everyone, they are interested in their patients. A few words as to what happened to 'Mrs Smith who was brought in last week' and her subsequent clinical course are a friendly and easy way of providing informal feedback and help to cement the professional relationship between the ambulance service and the ED.

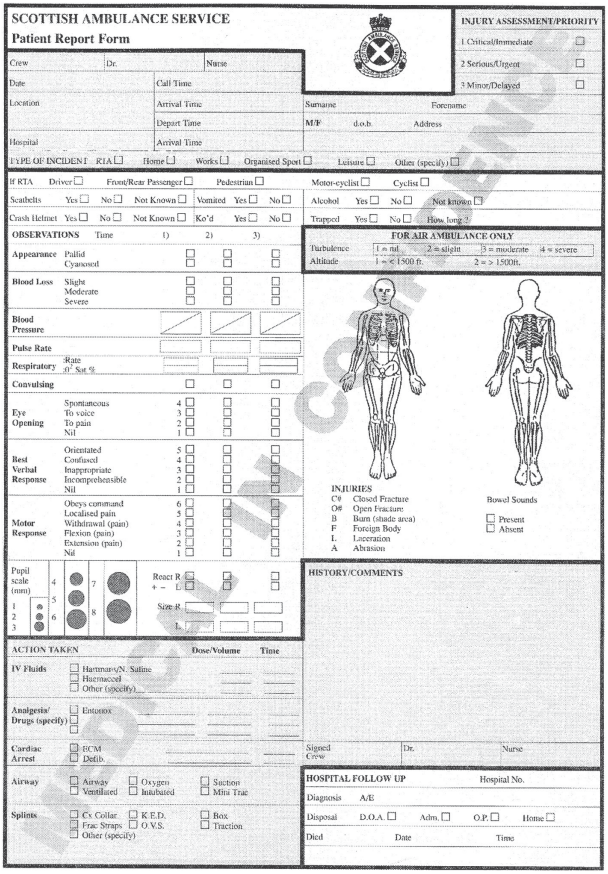

SCOTTISH AMBULANCE SERVICE
Patient Report Form
INJURY ASSESSMENT/PRIORITY
1 Critical/Immediate
2 Serious/Urgent
3 Minor/Delayed
Crew
Dr.
Nurse
Date
Call Time
Location
Arrival Time
Depart Time
Hospital
Arrival Time
Surname
Forename
M/F
d.o.b.
Address
TYPE OF INCIDENT RTA Home Works Organised Sport Leisure Other (specify)
If RTA Driver Front/Rear Passenger Pedestrian
Motor-cyclist Cyclist
Seatbelts Yes No Not Known
Vomited Yes No
Alcohol Yes No Not known
Crash Helmet Yes No Not Known
Ko'd Yes No
Trapped Yes No How long ?
OBSERVATIONS Time 1) 2) 3)
FOR AIR AMBULANCE ONLY
Turbulence 1 = nil 2 = slight 3 = moderate 4 = severe
Altitude 1 = < 1500 ft. 2 = > 1500ft.
Appearance Pallid Cyanosed
Blood Loss Slight Moderate Severe
Blood Pressure
Pulse Rate
Respiratory Rate $O^2$ Sat %
Convulsing
Eye Opening Spontaneous 4 To voice 3 To pain 2 Nil 1
Best Verbal Response Orientated 5 Confused 4 Inappropriate 3 Incomprehensible 2 Nil 1
Motor Response Obeys command 6 Localised pain 5 Withdrawal (pain) 4 Flexion (pain) 3 Extension (pain) 2 Nil 1
INJURIES
C# Closed Fracture
O# Open Fracture
B Burn (shade area)
F Foreign Body
L Laceration
A Abrasion
Bowel Sounds
Present
Absent
Pupil scale (mm) 1 2 3 4 5 6 7 8
React R + − L
Size R
L
HISTORY/COMMENTS
ACTION TAKEN
Dose/Volume
Time
IV Fluids Hartmann's/N. Saline Haemaccel Other (specify)
Analgesia/ Drugs (specify) Entonox
Cardiac Arrest ECM Defib.
Airway Airway Ventilated Oxygen Intubated Suction Mini Trac
Splints Cx. Collar K.E.D. Box Frac Straps O.V.S. Traction Other (specify)
Signed Crew
Dr.
Nurse
HOSPITAL FOLLOW UP
Hospital No.
Diagnosis A/E
Disposal D.O.A. Adm. O.P. Home
Died Date Time

**Fig. 1.1** An example of a patient reporting form.
Reproduced with kind permission from the Scottish Ambulance Service.

# Coping as a junior doctor in the ED

Although many junior doctors coming to the ED have completed >12 months of work since qualification, the prospect of working at the 'sharp end' can be accompanied by trepidation. As with many potentially worrying situations in life, the reality is not as terrifying as its anticipation. The number of hours worked may not appear long in comparison with other posts, but do not assume that this makes an ED job 'easy'. Being on duty inevitably involves much time standing, walking, working, thinking, and making decisions. It is unusual to come off-shift without feeling physically tired.

Active young doctors can usually cope with these physical demands, but a demanding professional life and a demanding social life are rarely compatible. Make the most of time off and try to relax from the pressures of the job. However, do not think it is possible to stay out all night and then work unimpaired the next day. Tired doctors make mistakes. They also tend to have less patience and, as a consequence, interpersonal conflicts are more likely.

A greater problem is the mental aspect of the job. Doctors often find that the ED is the first time in their careers when they have to make unequivocal decisions based on their own assessment and investigations. This is one of the great challenges and excitements of emergency medicine. It is also a worry. Decision-making is central to ED practice and, with experience, the process becomes easier. Developing a structured approach can pre-empt many problems and simplify your life. After taking an appropriate history and completing the relevant clinical examination of a patient, consider the following:

- What is likely to be wrong with this patient?
- What investigations are required to confirm the diagnosis?
- What treatment is needed and is the expertise available?
- Does this patient require referral to an inpatient team (see ➔ Discharge, referral, and handover, pp. 10–11)?
- If not, do they need to be reviewed in the ED or in another specialist clinic?

The wide spectrum of problems with which ED patients can present means that no individual can be expert in every possible condition. Recognize and accept when you are out of your depth. Seek help appropriately and do not just try to muddle through. Help may be readily available from senior ED staff, but in some departments, direct contact with a specialist team may be required.

One of the most difficult situations is where a specialist either refuses to come to see the patient or gives telephone advice that is clearly inappropriate. Always act as the patient's advocate. If, having referred a patient with a fractured neck of femur, the telephone message from the inpatient team is 'bring him back to the fracture clinic in 1 week', it is clearly wrong to carry this out. First, check that the doctor has understood the details of the patient's condition and the diagnosis. More conflict and aggravation are caused by communication errors (usually involving second-hand telephone messages) than by anything else. If the situation remains unresolved, consult senior ED staff. Whatever happens, remain cool in public and always put the patient's interests first.

### Learning in the ED

Try to learn something new every day. Keep a note of patients with interesting or unusual problems, and later check what happened to them. Ask senior staff for advice. Use ED reference books. Try to note all new conditions seen during a shift and read about them later.

### Staff interaction

The nature of the job, the patients, and the diversity of staff involved means that a considerable degree of camaraderie exists. For an outsider, this can initially be daunting. Junior medical staff are likely to work for 4–12 months in the department. Other staff may have spent a lifetime there, with long-established friendships (or sometimes animosities). Respect their position and experience, and learn from them.

The role of one individual and that of other individuals in the department are inextricably linked. Anyone who feels they are the most important individual in their working environment will have an extremely uncomfortable professional existence. In the ED, every member of staff has an important role to play.

Never consider any job 'beneath you' or someone else's responsibility. Patients come before pride. So if portering staff are rushed off their feet and you are unoccupied, wheel a patient to X-ray yourself—it will improve your standing with your colleagues and help the patient.

### Shifts

*Rule 1*

Never be late for your shift.

*Rule 2*

If, for whatever reason, you are unable to work a shift, let the senior staff in the ED know as soon as possible.

Ensure that you take a break. Two or three short breaks in an 8-hr shift are better than one long one. Remember to eat and maintain your fluid intake. Shift working may mean that you will work sometimes with familiar faces and perhaps occasionally with individuals with whom you find social contact uncomfortable. Put these considerations aside whilst you are at work, for the sake of the patients and your peace of mind.

### If you can't cope

Finally, if you feel that you are unable to manage or that the pressure of the job is too great—*tell someone*. Do not bottle it up, try to ignore it, or assume that it reflects inadequacy. It does not. Everyone, at some time, has feelings of inability to cope. Trying to disguise or deny the situation is unfair to yourself, your colleagues, and your patients. You need to tell someone and discuss things. Do it now. Talk to your consultant. If you cannot face him or her, talk to your GP or another senior member of staff—but talk to someone who can help you.

The *BMA Counselling Service for Doctors* (tel: 0330 123 1245) provides a confidential counselling service 24hr a day, 365 days of the year, to discuss personal, emotional, and work-related problems. The Doctors' Support Network (℔ http://www.dsn.org.uk) is a useful resource providing peer support for doctors and medical students with mental health concerns.

# Inappropriate attenders

This is an emotive and ill-defined term. Depending upon the department, such patients could comprise 4–20% of attendances.

The perception as to whether it is appropriate to go to an ED or attend a GP will vary between the patient, GP, and ED staff. Appropriateness is not simply related to the symptoms, diagnosis, or the time interval involved. It may not necessarily be related to the need for investigation. For example, not all patients who require an X-ray necessarily have to attend an ED.

Further blurring of 'appropriate' and 'inappropriate' groups relates to the geographical location of the ED. In some rural areas, GPs sometimes perform procedures such as suturing. In urban areas, these arrangements are less common. For ill-defined reasons, patients often perceive that they should only contact their GP during 'office' hours, and outside these times they may attend an ED with primary care complaints.

It is clearly inappropriate to come to an ED simply because of real or perceived difficulty in accessing primary care. Nevertheless, the term 'inappropriate attendance' is a pejorative one—it is better to use the phrase 'primary care patients'. It must be recognized that primary care problems are best dealt with by GPs. In the past, many departments have tried to prevent this primary care workload presenting to the ED. Most departments now tackle the problem by having GPs working alongside ED staff, either within the department or in an adjacent unit.

### Managing inappropriate attenders

Only through a continual process of patient education will these problems be resolved. Initiatives include nurse practitioner minor injuries units and hospital-based primary care services. Evaluations are under way, but to function effectively, such services require adequate funding and staffing.

It can sometimes be difficult to deal with primary care problems in the ED. After an appropriate history and examination, it may be necessary to explain to patients that they will have to attend their own GP. This may need direct contact between the ED and the practice to facilitate this.

### Inappropriate referrals

Sometimes, it may appear that another health professional (eg GP, emergency nurse practitioner, member of staff at NHS 111) has referred a patient to the ED inappropriately. Avoid making such judgements. Treat patients on their merits, but mention the issue to your consultant. Remember that the information available to the referring clinician at the time of the prehospital consultation is likely to have been different to that available at the time of ED attendance.

# The patient with a label

Some patients are referred by a medical practitioner with an accompanying letter which includes a presumptive diagnosis. The details in the letter are often extremely helpful, but do not assume the diagnosis is correct! Take particular care with patients who re-attend following an earlier attendance. The situation may have changed. Clinical signs may have developed or regressed. The patient may have not given the referring doctor and ED staff the same history. Do not pre-judge the problem—start with an open mind.

## Self-labelled patients

Take care with patients who label themselves. Those with chronic or unusual diseases often know significantly more about their conditions than ED staff! In such situations, take special notice of comments and advice from the patient and/or their relatives. Do not resent this or see it as a professional affront—rapport with the patient will increase markedly and management will usually be easier.

## Regular attenders

Every ED has a group of 'regular' patients who, with time, become physically, and sometimes emotionally, attached to the department. Some have underlying psychiatric illnesses, often with personality disorders. Some are homeless. Regular attenders frequently use the ED as a source of primary care. As outlined previously, make attempts to direct them to appropriate facilities, because the ED is unsuited to the management of chronic illness and is unable to provide the continuing medical and nursing support that these patients require.

Repeated presentations with apparently trivial complaints or with the same complaint often tax the patience of ED staff. This is heightened if the presentations are provoked or aggravated by alcohol intake. Remember, however, that these patients can and do suffer from the same acute events as everyone else. Keep an open mind, diagnostically and in attitude to the patient. Just because he/she has returned for the third time in as many days, complaining of chest pain, does not mean that, on this occasion, he does not have an acute MI! Maintain adequate documentation for each attendance. Sometimes, with intractable re-attenders, a multidisciplinary meeting can provide a plan of action for both the patient and the medical services, which can help to shape care in the ED (and, importantly, identify those investigations and treatments that are unlikely to help).

## Medically unexplained symptoms

A significant proportion of regular attenders present with ongoing problems relating to medically unexplained symptoms which have often been extensively investigated. These patients can be very difficult to manage, made more tricky by the fact that a small proportion will have an underlying treatable condition. Information from previous attendances can be extremely helpful in preventing unnecessary investigations. Acknowledge to the patient that the symptoms are real, and involve a senior ED clinician. Very often, the best approach is to focus on managing the symptoms, rather than making a new diagnosis.

# The difficult patient

## General approach

Accept the patient as he or she is, regardless of behaviour, class, religion, social lifestyle, or ethnicity. Given human nature, there will inevitably be some patients whom you immediately dislike or find difficult. The feeling is often mutual. Many factors that cause patients to present to the ED may aggravate the situation. These include their current medical condition, their past experiences in hospitals, their social situation, and any concurrent use of alcohol and/or other drugs. Your approach and state of mind during the consultation play a major role. This will be influenced by whether the department is busy, how much sleep you have had recently, and when you last had a break for coffee or food.

Given the nature of ED workload and turnover, conflict slows down the process and makes it more likely that you will make clinical errors. Many potential conflicts can be avoided by an open, pleasant approach. Introduce yourself politely to the patient. Use body language to reduce a potentially aggressive response.

Put yourself in the patient's position. Any patient marched up to by a doctor who has their hands on their hips, a glaring expression, and the demand 'Well, what's wrong with you now?' will retort aggressively.

## Defusing a volatile situation

Most complaints and acts of aggression occur when the department is busy and waiting times are long. Patients understand the pressures under which medical and nursing staff have to work, and a simple 'I am sorry you have had to wait so long, but we have had a number of emergencies elsewhere in the department' does much to diffuse potential conflict, and will often mean that the patient starts to sympathize with you as a young, overworked practitioner!

There is never any excuse for rude, abusive, or aggressive behaviour to a patient. If you are rude, complaints will invariably follow and, more importantly, the patient will not have received the appropriate treatment for their condition. It may be necessary to hand care of a patient to a colleague if an unresolvable conflict has arisen.

Management of the violent patient is considered in detail in ➲ Managing aggression, p. 626.

## Patients in police custody

Patients who are brought to the ED whilst in police custody can be very challenging to manage. Follow the 2016 RCEM guideline (*Emergency Department Patients in Police Custody*)—the key points of which are:

- Patients in custody are entitled to the same care as other patients.
- Treat patients in custody as a priority within their triage category.
- Liaise with the health care practitioner (nurse/doctor) at the police station if discharging the patient from the ED to a police station. Provide clear instructions and advice about continuing care to the health care practitioner and/or police personnel responsible for the patient.
- ED staff have a purely therapeutic role and should not act as surrogate forensic medical examiners (who also have a forensic role).

# Special patient groups

Attending the ED is difficult enough but can be even more so for certain patient groups. It is important that ED staff are sensitive to the needs of these groups and that there are systems in place to help them in what may be regarded as an intimidating atmosphere. The following list is far from exhaustive but includes some important groups who require particular consideration:

- *Children*: they are such an 'obvious' and large 'minority' group that they receive special attention to suit their particular needs (see ➔ Chapter 15).
- *Pregnant women* (see ➔ Chapter 13).
- *Those with mental health problems* (see ➔ Chapter 14).
- *The elderly*: who often have multiple medical problems and live in socially precarious circumstances.
- Patients with Alzheimer's disease and other states associated with chronic confusion.
- *Those with learning difficulties* (see ➔ The patient with learning difficulties, p. 24).
- Patients with hearing problems.
- The visually impaired.
- *Those who do not speak or understand English*: arrangements should be in place to enable the use of interpreters.
- *Patients with certain cultural or religious beliefs (particularly amongst 'minority groups')*: these can impact significantly upon a variety of situations (eg after unsuccessful resuscitation for cardiac arrest—see ➔ Breaking bad news, pp. 26–7).
- Those who are homeless or away from home, friends, and family (eg holiday makers).
- Those who have drug/alcohol dependency.

### Isn't everyone special?

Taken at face value, the concept that certain groups of patients are 'special', and so require special attention, does not meet with universal approval. There is a good argument that every patient deserves the best possible care. Whilst this is true, it is also obvious that certain patients do have additional needs that need to be considered. Many of these additional needs relate to effective communication. There are some tremendous resources available that can help practitioners to overcome communication difficulties (eg 🔗 http://www.communicationpeople.co.uk).

# Assessing the elderly patient

## Frailty

Although a large proportion of elderly patients attending the ED with serious illness are very frail, most elderly people in the general population are actually not frail. The degree of frailty of an elderly individual is a good predictor of their life expectancy. Early assessment of frailty can assist with the planning of patient management in ED and beyond. There are a number of different scoring systems in current use—an example is shown in Table 1.2.

**Table 1.2** Rockwood Clinical Frailty Scale

| Score | Label | Descriptor |
|---|---|---|
| 1 | Very fit | Robust, fit, and very active |
| 2 | Well | No active illness, exercises occasionally |
| 3 | Managing well | Medical problems well controlled |
| 4 | Vulnerable | Symptoms limit activities |
| 5 | Mildly frail | Requires help for activities of daily living |
| 6 | Moderately frail | Needs help in the home and with bathing |
| 7 | Severely frail | Completely dependent for personal care |
| 8 | Very severely frail | Could not recover from minor illness |
| 9 | Terminally ill | Life expectancy <6 months |



### *Frailty syndromes*

Elderly frail patients may present in a number of ways, some of which are relatively non-specific but warrant careful assessment: falls, immobility, delirium, incontinence, and medication issues.

## Risk indicators

Multiple pathologies and atypical symptoms render the elderly more vulnerable to the physical, functional, and social effects of acute illness. Past medical history and pre-admission status are especially important determinants for patients with dementia or psychiatric illness. Check for recently changed circumstances, recent bereavement, a change in medical or physical condition, ↑ confusion, or unusual behaviour. The patient may not be able to afford adequate food or heating. Community services may not be aware that support is needed, or help may have been offered but refused.

Other important indicators are: living alone, absence of close family support or community services, unsuitable home circumstances (eg external or internal stairs), and difficulty with mobility.

### *Cognitive impairment*

Perform an Abbreviated Mini-Mental Test or a Mini-Mental State Examination on every patient aged >75y.

# Discharging the elderly patient

There are no set predisposing factors that determine which patients are most at risk following discharge. Those that affect the chance of difficulties at home include the current medical problem and the underlying functional and social factors.

## Determining those unable to cope

Look for evidence of self-neglect that suggests that the elderly person is having difficulty coping at home (eg poor personal hygiene, unclean or unsuitable clothing). Evidence of recent weight loss may suggest difficulties with food preparation or eating or unavailability of food, or it may be due to serious pathology such as malignancy or tuberculosis. Signs of old bruising or other minor injuries may be consistent with frequent falls. Shortness of breath and any condition producing impaired mobility are important factors.

### *Falls*

These are a very common problem of old age—analyse what happened carefully. Avoid using the term 'mechanical fall', which does not really provide an explanation of what happened. Correctable factors include damaged walking aids, loose rugs, poor lighting, unsuitable footwear or glasses. Common medical causes include cerebrovascular disease, arthritis, and side effects of drugs.

Many elderly people claim that they can cope at home when they are unable to do so. If in doubt, ask relatives, the GP, and community support agencies. They may give helpful insight into the patient's mental state, which can be assessed further, whether it be a cognitive or a reactive condition.

### *Admission to a specialist unit*

If hospital admission is required, consider where the patient's overall needs would be best met. Specialty older persons or frailty units may be best placed to perform a full assessment.

## The decision to discharge

Hospital admission for an elderly person is a frightening experience and can lead to confusion and disorientation. If circumstances allow, aim to discharge the patient home. If there are concerns regarding the patient's functional ability and/or mobility, ask for an *occupational therapy* and/or *physiotherapy* assessment. On some occasions, it may be necessary to admit the patient for a short time (eg to a clinical decision unit) to complete this assessment. On other occasions, it may be possible to arrange a home assessment.

The elderly person is best seen in their home environment with familiar surroundings, especially if there is evidence of cognitive deficit. The provision of equipment and recommendations for adaptations can be made at this point, if required. A wide range of community services, including district nurse, health visitor, home help, crisis care, social work, hospital discharge, and rapid response therapy teams, can be contacted to provide immediate follow-up and support. These play a crucial role in preventing later breakdowns in home circumstances and unnecessary admissions for social reasons.

# The patient with learning difficulties

Patients with learning difficulties use the health care system more than the general population. Unfortunately, many health care professionals have little experience with these patients. However, understanding common illness patterns and using different techniques in communication can result in a successful consultation. Patients with learning difficulties often have complex health needs. There are many barriers to assessing health care, which may lead to later presentations of illness. Patients may have a high tolerance of pain—take this into consideration when examining them.

## Associated health problems

Patients with learning difficulties have a higher incidence of certain problems:

- Visual and hearing impairment.
- Poor dental health.
- Swallowing problems.
- Gastro-oesophageal reflux disease.
- Constipation.
- Urinary tract and other infections.
- Epilepsy.
- Mental health problems (↑ incidence of depression, anxiety disorders, schizophrenia, delirium, and dementia), with specific syndromes having their own particular associations (eg Down's syndrome is associated with depression and dementia; Prader–Willi with affective psychosis).
- Behavioural problems (eg Prader–Willi, Angelman syndrome).

*Leading causes of death*

These include pneumonia (relating to reflux, aspiration, swallowing, and feeding problems) and congenital heart disease.

## The patient's perspective

Past experiences of hospital are likely to have a significant impact on the patient's reaction to his/her current situation. Most patients have problems with expression, comprehension, and social communication. They may find it difficult to describe symptoms—behavioural change may be the best indication that something is wrong.

## Tips for communication

- Explain the consultation process before starting.
- Speak first to the patient, then to the carer.
- Use open questions, then re-phrase to check again.
- Aim to use language that the patient understands, modifying this according to comprehension.
- Patients may have difficulties with time, so try to relate symptoms to real-life temporal events (eg 'did the pain start before lunch?').
- They may not make a connection between something that they have done and feeling ill (eg several questions may be required in order to establish that they have ingested something).
- Take particular note of what the carer has to say—information from someone who knows the patient well is invaluable.

# End of life care

## Background

It was once unusual for patients to present to the ED requiring end of life care, but it is now relatively common, perhaps a reflection of the way that the work has evolved in recent years. Consider possible end of life care issues in any sick or frail patient who presents to the ED. Involve the patient and their family when making any decisions about end of life care.

Many patients have plans in place which cover their wishes regarding their end of life care—try to establish the nature of these plans through discussion with the patient, their family, carers, and paramedics and by checking electronic medical records. Frustratingly, sometimes patients who have clearly documented wishes of not to be brought to hospital in the event of a deterioration are still brought to the ED. On occasions, it can be appropriate to discharge a patient in the expectation that they will die in the community, rather than in hospital.

## Decisions about CPR

A key decision which needs to be made about sick patients who are brought to the ED is whether or not to start resuscitation in the event of a cardiac arrest. On some occasions, there is insufficient information available to make a considered decision—in an emergency, start CPR as the default position.

Try to make a considered decision about whether or not to start CPR, including discussion with a senior doctor, the patient, and their family, whenever possible, and document this on an appropriate form. In many hospitals, previously used 'Do Not Attempt CPR' (DNACPR) and 'Allow Natural Death Order' (ANDO) have been replaced with 'Treatment Escalation Plans' (TEPs) or the equivalent.

## Treatment Escalation Plans

DNACPR orders have been criticized as focussing simply on one aspect of end of life care and have sometimes been misinterpreted by patients and their families. TEPs are personalized to the patient and include considerations about whether or not it is appropriate to start CPR, but also include a number of wider issues (eg whether to treat in ICU/high dependency unit (HDU) and/or use non-invasive ventilation (NIV)). Such plans may reassure patients that, irrespective of a decision about CPR, they will still be treated actively (eg IV fluids, antibiotics, and analgesia) and may enable staff to set an agreed 'ceiling of care'. See the ReSPECT website (🔗 http://www.respectprocess.org.uk) which outlines the process for making a Recommended Summary Plan for Emergency Care and Treatment and has some useful resources.

## Other considerations

See RCEM recommendations (2015) for adult patients receiving end of life care in the ED. Focus upon symptom control and, in particular, provide analgesia, hydration, and personal care, as required. Consider opportunities for organ and tissue donation, as appropriate.

# Breaking bad news

A proportion of patients presenting to the ED have life-threatening conditions and some will die in the department. Often, the event will be sudden and unexpected by family and friends. It may already involve other family members (eg in the context of a road traffic collision). In contrast to hospital inpatients or those in general practice, an opportunity to forewarn relatives as to what has happened or the eventual outcome is unlikely. The relatives may already be distressed after witnessing the incident or collapse and may have been directly involved in providing first aid.

It is inappropriate for junior hospital staff without suitable experience to speak with distressed or bereaved relatives. The task must be undertaken by someone with sufficient seniority and authority, who also has the skills of communication and empathy. The most important component is time.

## Reception

Relatives usually arrive separately and after the patient. Anticipate this by designating a member of staff to meet them and show them to a relatives' room, which should afford privacy, comfortable seating, an outside telephone line, tea, coffee, and toilet facilities. Paper tissues, some magazines, and toys for small children are useful.

Whilst the relatives are waiting, a designated nurse should stay with them to act as a link with the department and the team caring for the patient. This nurse can pre-warn relatives of the life-threatening nature of the patient's condition and assist in building (an albeit short) relationship between staff and relatives. The link nurse should also check that important details have been recorded correctly, eg the patient's name, address, date of birth, religion (in case last rites are required), next of kin (name, relationship to patient, address, and phone number), and the patient's GP. This information should be collected as soon as possible, since later the relatives may be too upset to remember all these details or it may be difficult to ask for them.

## Breaking the news

Irrespective of who performs this task, remember a number of points. If you are the person who informs the relatives, ensure the link nurse is with you. After leaving the resuscitation room or clinical area, allow a minute or two of preparation to make yourself presentable, checking clothing for bloodstains, etc. Confirm that you know the patient's name. Enter the room, introduce yourself, and sit or kneel by the relatives so that you are at their physical level. Ensure that you speak with the correct relatives and identify who is who. Speak slowly and keep your sentences short and non-technical. Do not hedge around the subject. In their emotional turmoil, relatives very often misconstrue information. Therefore, you may need to re-emphasize the important aspects.

For many critically ill patients, their ultimate prognosis cannot be determined in the ED. In these situations, do not raise unrealistic expectations or false hopes, but be honest and direct with the relatives and the patients.

If the patient has died, then use the words 'death' or 'dead'. Do not use euphemisms such as 'passed away' or 'gone to a better place'.

After giving the news, allow the relatives a few minutes to collect their thoughts and ask questions. In some cases, these may be unanswerable. It is better to say 'We don't yet know', rather than confuse or give platitudinous answers.

Common responses to bad news or bereavement include emotional distress, denial, guilt, and aggression. The feelings of guilt and anger can be particularly difficult to come to terms with, and relatives may torture themselves with the idea that if only their actions had been different, the situation would never have arisen or the clinical outcome would have been different.

### Relatives seeing patients

Many relatives wish to see or touch their loved ones, however briefly. Television and cinema have prepared much of the population for the sights and sounds in the ED. In some departments, relatives are encouraged to be present in the resuscitation room. In selected situations, this stratagem has benefits. If the relatives are present during resuscitation, ensure that the link nurse is present with the relatives to provide support, explain what is happening, and accompany them if they wish to leave.

More frequently, the relatives can see the patient in the resuscitation room briefly or whilst they are leaving the ED (eg to go to the CT scan room or theatre). Even a few seconds, a few words, and a cuddle can be immensely rewarding for both the relative and the patient. The link nurse can give guidance beforehand as to the presence of injuries (especially those involving the face), monitors, drips, and equipment to diminish any threatening impact that these may have.

### When death occurs

Even before death has occurred, involvement of religious leaders can be valuable. As early as possible, inform the hospital chaplain, who can provide help to relatives and staff.

When a patient has died, offer the relatives the opportunity to see the body. This contact, which should be in a private quiet room, can greatly assist in the grieving process. With careful preparation, most patients who have died from multiple injuries can be seen by relatives in this fashion.

Remember that followers of some faiths, such as Muslims and Hindus, have important procedures and rituals to be followed after death, although these may not always be feasible after a sudden death, especially from trauma. In such situations, discuss the matter with the Coroner's or Procurator Fiscal's officer, and obtain help from an appropriate religious leader to look after the bereaved relatives.

# What to do after a death

## Who to contact

Any suspicious death must be immediately reported to the police who will liaise directly with the Coroner or Procurator Fiscal (in Scotland).

Following all deaths in the ED, a number of important contacts must be made as soon as possible:

- *Informing the next of kin*: if the relatives are not already present in the ED, it may be necessary to ask the police for assistance.
- Notifying the Coroner (Procurator Fiscal in Scotland).
- Informing the patient's GP.
- Cancelling hospital outpatient appointments.
- Informing social work and health visitor teams, as appropriate.

Ensure relatives of the deceased are given information about the process for death certification and registration, and how to organize funeral arrangements. Most EDs have useful leaflets that cover these matters and can answer many questions. Some departments have formal arrangements for counselling after bereavement. Often the GP is the best individual to coordinate bereavement care, but in any event, give the relatives a telephone number for the ED, so they can speak to a senior nurse or doctor if they need further information or help.

## Information for the Coroner or Procurator Fiscal

Report sudden deaths as soon as possible to the Coroner (in Scotland, the Procurator Fiscal). It is helpful to give the following information if it is available:

- Patient's name, address, and date of birth.
- Next of kin (name, relationship, address, phone number).
- Patient's GP.
- Date and time of patient's arrival in the ED.
- Date and time of patient's death.
- Name and job title of doctor who pronounced death.
- Details of the incident, injuries, or illness.
- Relevant past medical history.
- When the patient last saw a doctor (the Coroner may be happy for a GP or a hospital doctor to write the death certificate if they saw the patient recently for the condition that caused the death, eg a patient with known terminal cancer).
- *The patient's religion*: some faiths may wish to arrange burial before the next sunset, but this may not be feasible after a sudden death.
- Anything else that is important, eg difficulties in communication with the next of kin due to language or deafness.

## Looking after the staff

The death of a patient or the management of patients with critical illness inevitably affects ED staff. This is particularly so when some aspect of the event reminds staff of their own situation or relatives. These episodes often occur at the busiest times and when everyone in the ED is working under pressure.

One of the most difficult situations is to have to inform parents of the death of their child and help them in the initial grieving process, and then return to the busy department where many people are waiting with increasingly strident demands. It would be easy to respond that such individuals, with injuries or illnesses that are minor or have been present for days or weeks, are time-wasting. However, this approach will lead to conflict and is unfair to all concerned. Take 5–10min for a break in the staff room before returning to the fray. Remember that, in these circumstances, you too are a patient. Even senior and experienced staff may be distressed after difficult resuscitation situations and may require support—an informal or formal debrief can be very helpful.

## Organ donation

There is considerable potential to assist with the process of organ/tissue donation in the ED. However, the possibility of organ donation is sadly often overlooked in the ED. Many patients who die after an unexpected cardiac arrest are potential donors of corneal tissue and heart valves. Kidneys may also be retrieved from some patients who have died in the ED, if a protocol for this has been arranged with the transplant team and the local Coroner or Procurator Fiscal. Many other patients who are moribund, intubated, and ventilated (eg following massive subarachnoid haemorrhage) may be identified as potential donors of other tissues also. Consider the possibility of organ donation in patients who die in the ED or who are moribund with no hope of survival.

Most hospitals have specialist organ donation nurses (previously known as 'donor transplant co-ordinators') who will educate, advise, and assist with the process of organ donation. Useful information about organ/tissue transplantation is available on the website of the British Transplantation Society (http://www.bts.org.uk).

# Patient transfer

## The need to transfer

When patients have problems that exceed the capabilities of a hospital and/or its personnel, transfer to another hospital may be needed.

## Timing the transfer

Do not commence any transfer until life-threatening problems have been identified and managed and a secondary survey has been completed. Once the decision to transfer has been made, do not waste time performing non-essential diagnostic procedures that do not change the immediate plan of care. First, secure the airway (with tracheal intubation, if necessary). Ensure that patients with pneumothoraces have intercostal drains inserted prior to transfer where necessary. This is particularly important before sending a patient by helicopter or fixed wing transfer. Consider the need to insert a urinary catheter and a gastric tube.

## Arranging the transfer

Speak directly to the doctor at the receiving hospital. Provide the following details by telephone or electronic link:

- Details of the patient (full name, age, and date of birth).
- A brief history of the onset of symptoms/injury.
- The prehospital findings and treatment.
- The initial findings, diagnosis, and treatment in the ED and the response to treatment.

Write down the name of the doctor responsible for the initial reception of the patient after transfer. Establish precisely where, within the receiving hospital, the patient is to be taken. Where possible, prepare the receiving unit by sending details ahead by email. Preprinted forms can help in structuring the relevant details and avoiding omissions.

## Preparing for transfer

### *Transfer team*

If the patient to be transferred may require advanced airway care, ensure they are accompanied by a doctor who can provide this. Accompanying staff should be trained in resuscitation, with a good knowledge of the equipment used during transfer. Note that, in some instances, the transfer team is a retrieval team based in the regional centre.

### *Equipment*

'Transfer cases' containing a standardized list of equipment must be immediately available and regularly checked. Take all the emergency equipment and drugs that might prove necessary to maintain the 'Airway, Breathing, and Circulation' (ABC) during transfer. In particular, take at least twice the amount of $O_2$ estimated to be necessary (a standard 'F' cylinder contains 1360L of $O_2$ and will therefore last <3hr running at 10L/min). Before leaving, ensure that the patient and stretcher are well secured within the ambulance. Send all cross-matched blood (in a suitably insulated container) with the patient.

### Monitoring during transfer

Minimum monitoring during transfer includes ECG monitoring, pulse oximetry, and non-invasive BP measurement. If the patient is intubated and ventilated, end-tidal carbon dioxide ($CO_2$) monitoring is mandatory. An intra-arterial line may be recommended to monitor BP during the journey. Make allowances for limited battery life on long transfers—spare batteries may be needed. Plug monitors and other equipment into the mains supply, whenever possible.

### Accompanying documentation

Include the following:

- *Patient details*: name, date of birth, address, next of kin, telephone numbers, hospital number, and GP.
- History, examination findings, and results of investigations (including imaging).
- Type and volume of all fluids infused (including prehospital).
- Management, including drugs given (type, route, and time of administration) and practical procedures performed.
- Response to treatment, including serial measurements of vital signs.
- Names of referring and receiving doctors, and their hospitals and telephone numbers.

Some departments use standard forms to ensure that important information is complete.

### The relatives

Keep the patient's relatives informed throughout. Explain where and why the patient is going. Document what they have been told. Arrange transport for relatives to the receiving hospital.

### Before leaving

Prior to transfer, re-examine the patient. Check that the airway is protected, ventilation is satisfactory, chest drains are working, IV cannulae are patent and well secured, and the spine is appropriately immobilized, with pressure areas protected. Ensure that the patient is well covered to prevent heat loss. Inform the receiving hospital when the patient has left, and give an estimated time of arrival.

### After leaving

Communicate to the receiving hospital the results of any investigations that become available after the patient has left. Contact the receiving doctor afterwards to confirm that the transfer was completed satisfactorily and to obtain feedback.

### Intra-hospital transfers

In many respects, the only difference between intra- and inter-hospital transfers is the distance. The principles involved in organizing a transfer are the same, whether the patient is to be conveyed to the CT scanner down the corridor or to the regional neurosurgical unit miles away.

# Medicolegal aspects: avoiding trouble

Medicolegal problems are relatively common in the ED. Many of these problems may be avoided by adopting the correct approach.

## Attitude

Be polite and open with patients. Try to establish a good rapport. Be as honest as possible in explaining delays/errors.

## Consent

(See General Medical Council guidance, available at: https://www.gmc-uk.org)

Use the consent form liberally for anything that is complex or risky or involves sedation or general anaesthesia (GA). Ensure that the patient understands what is involved in the procedure, together with the potential benefits and risks. Whenever possible, attempt to obtain consent from the parent/guardian in minors, but do not delay life-saving treatment in order to obtain consent.

## Documentation

(See ➲ Note keeping, pp. 6–7.)

Good notes imply good practice. Keep careful notes, using simple, clear, unambiguous language. Write your name legibly, and document the time that you saw the patient. Remember that successful defence of a medical negligence claim may depend upon accurate, legible, comprehensive, contemporaneous notes. Try to avoid abbreviations, particularly where there is room for confusion. In particular, name the digits of the hand (thumb, index, middle, ring, and little fingers), and specify right or left by writing it in full.

Be meticulous in documenting the nature, size, and position of any wounds (see ➲ The approach to wounds, p. 410). Write down a diagnosis, together with a full interpretation of any investigations. Ensure that all attached documents (nursing observations, blood results, ECG) are labelled. Document all instructions and advice given to the patient, together with any follow-up arrangements made.

## Referral

(See ➲ Discharge, referral, and handover, pp. 10–1.)

Always seek senior help or refer those patients with problems beyond your knowledge or expertise. Record any referral made, together with the name and grade of the doctor referred to, the time it was made, and a summary of the facts communicated. After referral, be cautious about accepting telephone advice alone—an expert cannot usually provide an accurate opinion without seeing the patient.

## Return visits

Take special care with any patient who returns to the ED with the same presenting complaint, because it is no better or has deteriorated or the patient is simply dissatisfied. Do not automatically rely upon previous diagnosis and X-ray interpretations as being correct—treat the patient as if they were attending for the first time. Aim to involve a senior doctor in these cases.

### Discharge against advice

Always attempt to persuade the patient to accept the treatment offered, but if this is refused or the patient leaves before being seen, ask the patient to sign an appropriate form. Patients not deemed competent (see ➲ Mental Capacity Act, p. 645) to make this decision may need to be held against their wishes—seek senior help with this. Write full notes explaining what happened.

### Mental Capacity Act

(See ➲ Mental Capacity Act, p. 645.)

The Mental Capacity Act 2005 outlines how a person is unable to make a decision for himself/herself if he/she is unable to:

- Understand the information relevant to the decision.
- Retain the information.
- Use or weigh that information as part of the process of making the decision.
- Communicate his/her decision.

A patient lacks capacity if at the time he/she is unable to make a decision for himself/herself in relation to the matter because of an impairment or a disturbance in the functioning of the mind or brain.

### Access to records

All ED staff should bear in mind that patients may gain access to their medical records and read what has been written about them. Patients in the UK have a statutory right of access to information about themselves (set out in the Data Protection Act 1998) and this includes medical records. Competent patients may apply for access to, and copies of, their own records. Applications are usually made in writing via the hospital's legal department.

### Medical defence organization

Join a medical defence organization. The Medical Defence Union (MDU), Medical and Dental Defence Union of Scotland (MDDUS), and Medical Protection Society provide professional indemnity cover for emergencies outside hospital and advice and support for all sorts of medicolegal matters that are not necessarily covered by NHS trusts, eg statements to the Coroner or Procurator Fiscal, support at inquests or fatal accident inquiries, allegations of negligence, legal actions, and problems with the General Medical Council (GMC). They also provide members with useful information and booklets about consent, confidentiality, and other issues.

### Further information

http://www.themdu.com
http://www.mddus.com
http://www.medicalprotection.org

# Medicolegal aspects: the law

## Confidentiality

Medical information about every patient is confidential and should not be disclosed without the patient's consent. In the UK, the police do not have routine access to clinical information, but some information may be divulged in certain specific circumstances:

- The Road Traffic Act (1972) places a duty on any person to provide the police, if requested, with information that might lead to the identification of a vehicle driver who is suspected of an offence under the Act. The doctor is obliged to supply the person's name and address, but not clinical information.
- Suspicion of terrorist activity.
- Gunshot and knife wounds (see https://www.gmc-uk.org).
- Disclosure in the public interest. The GMC advises that this might include situations where someone may be exposed to death or serious injury (eg murder, rape, armed robbery, child abuse). Although this may provide ethical permission for the doctor to reveal details without consent, it does not place him/her under any legal duty to do so. Discuss these cases with your consultant and/or your medical defence organization. See GMC advice (https://www.gmc-uk.org).

## Ability to drive

A patient's ability to drive may be impaired by injury (especially limb or eye), by drugs (eg after GA, opiates, alcohol), or due to medical conditions (eg transient ischaemic attacks (TIAs), epilepsy, arrhythmias). In each case, warn the patient not to drive and ensure that this warning is documented in the notes. It may be prudent to provide this warning in the presence of a close relative.

For further information on medical aspects of fitness to drive, see https://www.gov.uk

## Police requests for blood alcohol

In the UK, the police may request a blood or urine sample under Section 5 of the Road Traffic Act (1988) from a patient they suspect to have been in charge of a motor vehicle with an illegal blood alcohol level (>80mg/100mL). In such circumstances, specimens should only be taken if they do not prejudice the proper care and treatment of the patient. The relevant specimens should only be taken by a clinical forensic physician (forensic medical examiner/police surgeon) and with the patient's consent.

A change in the law (Police Reform Act 2002) also allows a forensic physician to take a blood sample from an unconscious patient who is suspected of having been the driver of a motor vehicle whilst under the influence of alcohol and/or drugs. The blood sample is retained and tested later, depending upon the patient later giving consent. Again, only permit the forensic physician access to the patient if this will not delay or prejudice proper care and treatment of the patient.

### Reporting deaths to the Coroner (or Procurator Fiscal)

Many deaths that occur in (or in transit to) the ED are sudden and unexpected, and/or follow trauma. The exact cause of death is seldom immediately apparent. Accordingly, do not be tempted to sign death certificates. Instead, report all deaths to the Coroner (the Procurator Fiscal in Scotland). (See ➲ What to do after a death, p. 28 for details of the information required.)

### Police statements

Do not provide information to the police until patient consent has been obtained. Writing a police statement requires thought and care. Write the statement yourself. Keep statements brief and try to avoid hearsay, conjecture, or opinion on the likely outcome. List injuries using both medical and non-medical language, explaining terminology in detail as necessary. State the investigations and treatment provided as accurately as possible (eg what sutures and how many were used). Having written the statement, ask your consultant to read it and comment on it. Get the statement typed (a friendly ED secretary may help if you cannot type yourself and will also know how you can claim the relevant fee). Having checked it, sign and date the statement and give it to the officer concerned. Always keep a copy of the statement and the ED notes, so that they are easily available if you are called to court.

### Court appearances

*In advance* Discuss the case with your consultant, and review the notes, the questions that you might be asked, and the likely court procedures. Get a good copy of the notes and any investigations. Ask whether you should take the original records to court.

*On the day* Dress smartly, arrive early, and behave professionally. Be prepared for a long wait, so take a book to read. Turn off your mobile phone. Once in court, you have the option of taking an oath before God or affirming without religious connotation. You are equally bound to tell the truth, whichever you choose. Use the same form of address that others have already used (eg 'My Lord', 'Your Honour'). Answer directly and simply. Use comprehensible language, free of medical jargon. Remember that you are a professional witness, not an expert. Therefore, confine the expression of opinion to within the limits of your knowledge and experience—if asked something outside this, say so!

*Inquest/fatal accident inquiry* If you are called to give evidence at an inquest (in Scotland, a fatal accident inquiry), discuss the case with your consultant and also with your medical defence society.

*Further information and advice about reports and appearing in court* The medical defence organizations (see ➲ Medical defence organization, p. 33) have useful advice sheets for their members about writing reports and appearing in court.

# Infection control and prevention

Organisms such as *Staphylococcus aureus*, including meticillin-resistant *S. aureus* (MRSA) (see ➲ Staphylococcal infections, p. 245), can readily be transmitted by contaminated hands or equipment, causing infection of wounds, fractures, and indwelling devices (eg catheters or chest drains). Infected blood can transmit many infections, including hepatitis B and C (see ➲ Hepatitis, p. 249) and human immunodeficiency virus (HIV) (see ➲ Human immunodeficiency virus, pp. 250–1). Viral gastroenteritis usually spreads by the faecal–oral route, but vomiting may cause widespread viral contamination of the surroundings and equipment, with a risk of transmission to other patients and staff.

Coughing and sneezing produce small droplets of infected secretions, which could involve viruses such as influenza (see ➲ Influenza pandemics, avian flu, and swine flu, p. 262), COVID-19/severe acute respiratory syndrome (SARS) (see ➲ Severe acute respiratory syndrome, p. 259), and respiratory syncitial virus (RSV) (see ➲ Acute bronchiolitis, pp. 698–9). A nebulizer used on an infected patient may spread respiratory viruses widely, as occurred in the outbreak of SARS in Hong Kong in 2003.

## Standard precautions for preventing infection

Use standard precautions (also known as 'universal precautions') at all times and with all patients to ↓ risks of infection. Treat all blood and body fluids from patients as infected. Standard precautions include:

- Hand hygiene: essential, but often neglected. Decontaminate your hands before and after every patient contact, and after any activity that might contaminate hands, including removing gloves. Wash hands that are visibly dirty or possibly grossly contaminated with soap and water, then dry thoroughly. Use alcohol hand gel if hands look clean. Cover broken skin with a waterproof dressing.
- Personal protective equipment (PPE): wear suitable disposable *gloves* for any contact with blood, body fluids, mucous membranes, or non-intact skin. Latex gloves are widely used but cause allergic reactions in some patients and staff who need special nitrile gloves. Use a *disposable plastic apron* if there is a risk of blood or body fluids contaminating clothing. *Impervious gowns* are needed if there is a high risk of contamination. Use a *mask*, *face shield*, and *eye protection* if blood or body fluids might splash in your eyes or mouth. Protection against respiratory viruses, eg SARS or COVID-19, requires special *masks* or *respirators* (eg FFP3), which must be fitted and used properly. *Powered air-purifying respirators* should be used for high-risk procedures such as intubating patients with serious viral infections.
- Safe handling and disposal of sharps: avoid handling needles directly and never re-sheathe them. Place used needles immediately into a 'sharps bin'. If possible, use safety needles and cannulae, which ↓ the risk of needlestick injury. If, despite all precautions, a *needlestick injury* does occur, follow local approved procedures to ↓ the risk of infection and look after the people involved (see ➲ Needlestick injury, p. 425).
- Managing blood and bodily fluids: handle samples of blood or other body fluids safely, with care not to contaminate request forms or the outside of the container. Follow local approved procedures for dealing with spillages of blood or body fluids—wear suitable PPE (usually a disposable apron and gloves), and disinfect the spillage with an appropriate agent, such as diluted bleach.

## Planning for outbreaks of infectious diseases

Planning to cope with an outbreak of a serious infectious disease such as COVID-19, SARS, or pandemic flu (see ➔ Influenza pandemics, avian flu, and swine flu, p. 262) is a considerable challenge for ED staff and for the whole community. The ED must be organized, so that patients can be assessed properly, with a minimum risk of infecting staff or other patients. If possible, patients with serious airborne diseases should be treated in negative-pressure isolation rooms by staff in appropriate PPE who are fully trained to minimize the risks of spreading and acquiring the infection. In high-risk situations, a 'buddy' system for staff may be helpful, with each doctor or nurse being watched closely by another person to check that full safety precautions are maintained.

### *Assessment of febrile patients*

Hospitals in Hong Kong with experience of SARS use the *FTOCC* criteria when assessing febrile patients for potentially serious infectious diseases:

- F—fever (>38°C).
- T—travel history.
- O—occupational history.
- C—clustering of cases.
- C—contact history (eg someone with SARS or avian flu).

See also:

- *Pandemic influenza/avian flu* (see ➔ Influenza pandemics, avian flu, and swine flu, p. 262).
- *SARS* (see ➔ Severe acute respiratory syndrome, p. 259).
- COVID-19 (see ➔ COVID-19, p. 260).

# At the roadside

►► *If you arrive first at the scene of a collision, the initial priority is to ensure your own safety and that of other rescuers.*

- Park safely so your car will not obstruct other vehicles (including emergency vehicles), preferably where its presence will alert other road users to the collision. Put your hazard warning lights on. If you have a warning beacon, put it on the roof of the car and switch it on.
- If you have a mobile phone, dial '999' and request ambulance, fire service, and police to attend. Remember to give the exact location, a brief description of the incident, and the number of casualties. Tell the emergency service operator who you are, as well as the number of your mobile phone.
- Switch off the engine of your car and of any other vehicles.
- Ensure that no one is smoking or displays a naked flame.
- Events involving electricity or chemicals have specific hazards. Involvement of overhead or underground electric cables poses risks, compounded if water is involved or sparks are produced. The risk from high-tension cables extends for several metres. Phone the power company to ensure that the source is turned off before approaching. Electrified rail lines may be short-circuited by a trained individual using a special bar carried in the guard's compartment.

## Chemical incidents

Do not approach a chemical incident until declared safe by the fire service. Lorries carrying hazardous chemicals must display a 'Hazchem' board (see Fig. 1.2). This has:

- Information on whether the area should be evacuated, what protective equipment should be worn, aspects relating to firefighting, and if the chemical can be safely washed down storm drains (top left). A white plate means that the load is non-toxic.
- A 4-digit UN product identification number (middle left).
- A pictorial hazard diamond warning (top right).
- An emergency contact number (bottom).

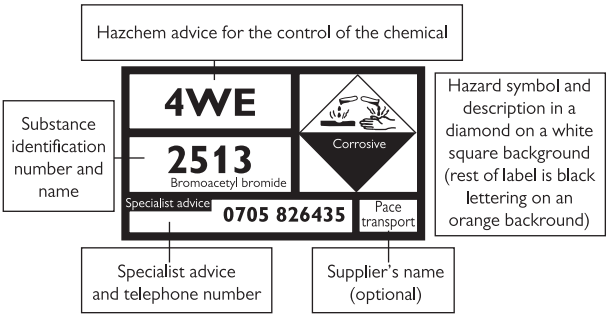
*The European 'Kemler' plate* Contains only the UN product number (bottom) and a numerical hazard code (top)—a repeated number means intensified hazard. Mixed loads of <500kg may only be identified by a plain orange square at the front and rear of the vehicle.

*The transport emergency card (TREM card)* Is carried in the driver's cab and gives information about the chemical for use at the scene of a crash. The fire tender may be equipped with CHEMDATA—a direct link with the National Chemical Information Centre at Harwell. Alternatively, contact a Poisons Information Centre or the transport company.

## Helicopters

If helicopters are used for transport/evacuation, remember:

- Ensure any loose objects are secured to prevent them from being blown away.
- Never enter the landing space area during landing or take-off.
- Only enter/leave the rotor disc area with the pilot's permission. Duck down in the rotor disc area, and only approach in full view of the pilot.
- If the helicopter cannot land and the winch is used, do not touch the cable before it has touched the ground to earth any static electrical charge.



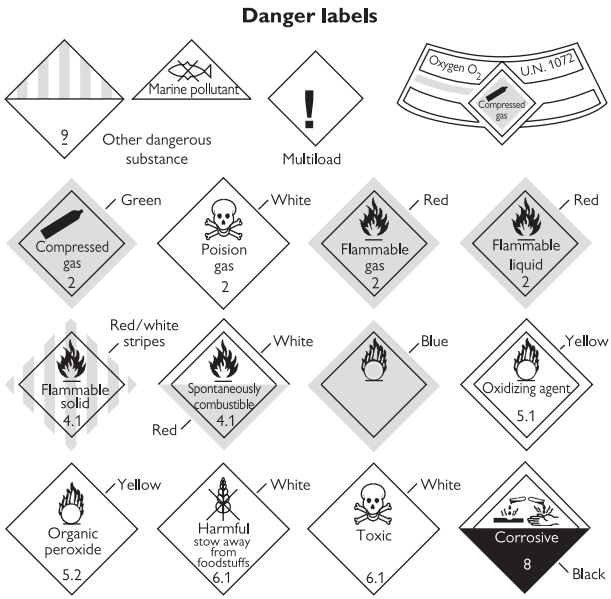




**Fig. 1.2** Hazchem advice and danger labels.

# Major incidents

A major incident involves a lot of people. Casualties may have multiple or minor injuries, burns, or other problems (eg food poisoning or chemical inhalation). Every hospital accepting emergencies has a Major Incident plan to use when normal resources are unable to cope and special arrangements are needed. Action cards for key staff detail their duties. All staff need to familiarize themselves with their roles in advance.

- *Call-in lists* must be up-to-date and available at all times.
- *Major incident practices* must be held regularly to check arrangements and contact details and to remind staff what they should do.

## Alert

The ambulance service or the police should warn the hospital of a possible or definite major incident. Initial messages are often inaccurate because they are based on confused and incomplete information from the scene. Occasionally, patients arrive without warning from a major incident near the hospital.

Ensure that the *ED consultant* on duty is informed immediately of any suspected major incident, enabling them to participate in the decision to start the major incident procedure. Senior medical, nursing, and administrative staff will set up the hospital's *Control Centre* and prepare for action. If the major incident is confirmed, the full hospital response is initiated, following the procedures in the plan.

Communications are vital, but switchboards rapidly become overloaded. Staff should therefore be called in using non-switchboard phones, if possible. All staff should wear their identification badges.

## Action in the ED

- Check that the ED consultant and hospital switchboard know about the incident and that the major incident procedure has been started.
- Inform all ED staff on duty (doctors, nurses, receptionists, porters).
- Call in other ED staff in accordance with the Major Incident plan.
- Clear the ED of any patients who are not seriously ill or injured. Prepare the department to receive patients from the incident.
- Doctors and nurses arriving to help should be given appropriate action cards. Staff should have labels or tabards, so that ED staff and other specialties (eg anaesthetists) can be identified easily.
- Prepare a triage point at the ambulance entrance. This should be staffed by a senior doctor and nurse who direct patients to the most appropriate area of the department. If possible, a nurse should stay with each patient until he/she is discharged or admitted to a ward.
- All patients should be labelled immediately with a unique Major Incident number, which is used on all notes, forms, blood samples, property bags, and lists of patients. Collect names, addresses, and other details as soon as possible, but this must not delay triage or emergency treatment. Keep lists of anyone leaving the ED.
- Ensure that the hospital *Control Centre* is regularly updated regarding the situation in the ED.

### Wards and theatres

Beds must be cleared to receive patients, preferably on one or two wards, rather than on many different wards. A senior surgeon should triage patients needing operations and co-ordinate theatre work.

### Relatives and friends

Relatives and friends of casualties should be looked after by social workers and chaplaincy staff in an area near to, but separate from, the ED, perhaps in the outpatient department. Keep relatives informed as soon and as much as possible. *Security staff* at each entrance to the ED should direct relatives and friends of casualties to the appropriate area and not allow them into the ED.

### Press

Journalists and television crews will arrive rapidly after a major incident. Keep them out of the ED—direct them to a pre-arranged room to be briefed by a press officer and senior staff.

### Arrangements at the site of a major incident

The police are in overall command. The fire service takes control of the immediate area if there is a fire or chemical risk. The police, fire, and ambulance services will each have a control vehicle, with an *Incident Officer* to co-ordinate their staff and the rescue work.

There may be a *Medical Incident Officer* (MIO) and also a *Mobile Medical Team* of doctors and nurses, who should, if possible, be sent from a supporting hospital, rather than the hospital receiving the first casualties. These staff must be properly clothed (yellow and green high-visibility jacket marked 'Doctor' or 'Nurse', overtrousers, green helmet with visor and chin strap, safety boots, gloves, knee pads, torch, ID badge) and must be trained and equipped with suitable medical supplies and action cards.

The mobile medical team must report to the MIO, who is in charge of all medical and nursing staff on site and works closely with the *Ambulance Incident Officer* (AIO). The MIO should record the names of the mobile medical team and brief them about their duties and the site hazards and safety arrangements. The MIO is responsible for supervising the team, arranging any necessary equipment and supplies, and making sure that the team are relieved when necessary. The MIO and AIO relay information to the hospitals and distribute casualties appropriately.

### Debriefing staff

Debriefing is important after a major incident, so that staff can discuss what happened and express their feelings. Mutual support of the team is essential. Counselling may be required. Senior staff should prepare a report on the incident and review the Major Incident plan.

### Further information

CBRN (chemical, biological, radiological, and nuclear) incidents (see ➲ Decontamination of the patient, p. 279)

NHS Emergency Planning guidance (see 🖱 https://www.gov.uk).

## Chapter 2

# Life-threatening emergencies

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Treatment algorithm for adults with anaphylaxis *46*
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Life-threatening emergencies in children are considered in Chapter 15, Paediatric emergencies p. 646

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➲ Choking from a foreign body p. 664
➲ Anaphylaxis in children p. 666
➲ Paediatric Advanced Life Support p. 668

# Anaphylaxis

(*Anaphylaxis in children* is covered in ➲ Anaphylaxis in children, p. 666.)

Anaphylaxis is a generalized immunological condition of sudden onset, which develops after exposure to a foreign substance. The mechanism may:

- Involve an immunoglobulin E (IgE)-mediated reaction to a foreign protein (stings, foods, streptokinase) or to a protein–hapten conjugate (antibiotic) to which the patient has previously been exposed.
- Be complement-mediated (human proteins, eg G-globulin, blood products).
- Be unknown (aspirin, 'idiopathic').

Irrespective of the mechanism, mast cells and basophils release mediators (eg histamine, prostaglandins, thromboxanes, platelet-activating factors, leukotrienes), producing clinical manifestations. Angio-oedema caused by angiotensin-converting enzyme (ACE) inhibitors and hereditary angio-oedema may present in a similar way to anaphylaxis. Hereditary angio-oedema is not usually accompanied by urticaria and is treated with C1 esterase inhibitor.

## Common causes

- Drugs and vaccines (eg antibiotics, streptokinase, suxamethonium, aspirin, non-steroidal anti-inflammatory drugs (NSAIDs), IV contrast agents).
- *Hymenoptera* (bee/wasp) stings.
- Foods (nuts, shellfish, strawberries, wheat).
- Latex.

## Clinical features

The speed of onset and severity vary with the nature and amount of the stimulus, but the onset is usually in minutes/hours. A prodromal aura or a feeling of impending death may be present. Patients on β-blockers or with a history of ischaemic heart disease (IHD) or asthma may have especially severe features. Usually two or more systems are involved:

- ***Respiratory*** Swelling of the lips, tongue, pharynx, and epiglottis may lead to complete upper airway occlusion. Lower airway involvement is similar to acute severe asthma—dyspnoea, wheeze, chest tightness, hypoxia, and hypercapnia.
- ***Skin*** Pruritus, erythema, urticaria, and angio-oedema.
- ***Cardiovascular*** Peripheral vasodilatation and ↑ vascular permeability cause plasma leakage from the circulation, with ↓ intravascular volume, hypotension, and shock. Arrhythmias, ischaemic chest pain, and ECG changes may be present.
- ***Gastrointestinal (GI) tract*** Nausea, vomiting, diarrhoea, abdominal cramps.

## Treatment

- Discontinue further administration of suspected factor (eg drug). Remove stings by scraping them carefully away from skin.
- Give 100% $O_2$ and IM adrenaline as indicated.
- Open and maintain airway. If upper airway oedema is present, get specialist senior help immediately. Emergency intubation or a surgical airway and ventilation may be required.
- In profound shock or *immediately life-threatening situations*, give CPR/Advanced Life Support (ALS) as necessary, and consider *slow* IV adrenaline 1:10,000 or 1:100,000 solution. This is recommended only for experienced clinicians who can also obtain immediate IV access. Note the different strength of adrenaline required for IV use. If there is no response to adrenaline, consider glucagon 1–2mg intramuscular (IM)/IV every 5min (especially in patients taking β-blockers).
- Give a $\beta_2$-agonist (eg salbutamol 5mg) nebulized with $O_2$ for bronchospasm, possibly with the addition of nebulized ipratropium bromide 500mcg.
- Give IV fluid if hypotension does not rapidly respond to adrenaline. Rapid infusion of 1–2L IV 0.9% saline may be required, with further infusion according to the clinical state.
- Antihistamine $H_1$ blockers (eg chlorphenamine 10–20mg slow IV) and $H_2$ blockers (eg ranitidine 50mg IV) are commonly given. They are second-line drugs that, with hydrocortisone 100–200mg slow IV, may reduce the severity/duration of symptoms.
- Admit/observe after initial treatment: prolonged reactions and biphasic responses may occur. Observe for at least 4–6hr after all symptoms have settled.

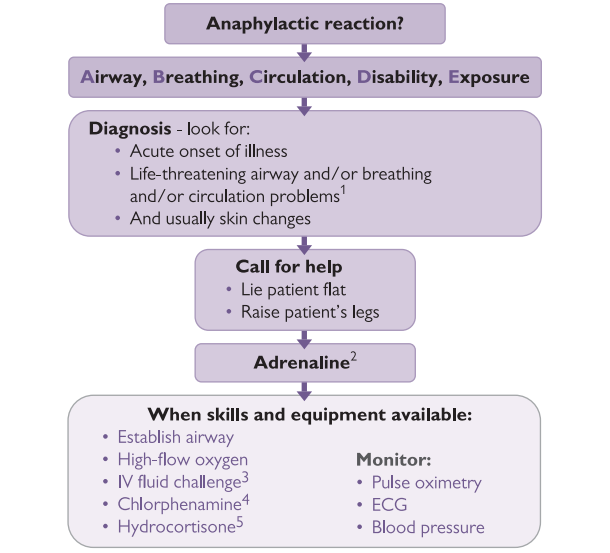
Report anaphylactic reactions related to drugs/vaccines to the Committee on Safety of Medicines. Further investigation of the cause (and possibly desensitization) may be indicated. Where identified, the patient and GP must be informed and the hospital records appropriately labelled. MedicAlert bracelets are useful.

## Notes on treatment algorithm

(See Fig. 2.1.)

1 An inhaled $\beta_2$-agonist, such as salbutamol, may be used as an adjunctive measure if bronchospasm is severe and does not respond rapidly to other treatment.
2 If profound shock judged *immediately* life-threatening, give CPR/ALS if necessary. Consider slow IV adrenaline (epinephrine) 1:10,000 solution. This is *hazardous* and is recommended only for an experienced practitioner who can also obtain IV access without delay. Note the different strength of adrenaline (epinephrine) that may be required for IV use.
3 If adults are treated with an EpiPen®, the 300mcg dose will usually be sufficient. A second dose may be required. Half doses of adrenaline (epinephrine) may be safer for patients on amitriptyline, imipramine, or a β-blocker.
4 A crystalloid may be safer than a colloid.

# Treatment algorithm for adults with anaphylaxis




**1 Life-threatening problems:**

| | |
|---|---|
| **Airway:** | Swelling, hoarseness, stridor |
| **Breathing:** | Rapid breathing, wheeze, fatigue, cyanosis, $SpO_2$ <92%, confusion |
| **Circulation:** | Pale, clammy, low blood pressure, faintness, drowsy/coma |

**2 Adrenaline** (*give IM unless experienced with IV adrenaline*)
IM doses of 1:1000 adrenaline (repeat after 5min if no better)

- Adult: 500 micrograms IM (0.5mL)
- Child more than 12 years: 500 micrograms IM (0.5mL)
- Child 6–12 years: 300 micrograms IM (0.3mL)
- Child less than 6 years: 150 micrograms IM (0.15mL)

Adrenaline IV to be given **only by experienced specialists**
Titrate: adults 50 micrograms; children 1 microgram/kg

**3 IV fluid challenge:**
Adult: 500–1000mL
Child: crystalloid 20mL/kg

Stop IV colloid if this might be the cause of anaphylaxis

| | **4 Chlorphenamine** (IM or slow IV) | **5 Hydrocortisone** (IM or slow IV) |
|---|---|---|
| Adult or child more than 12 years: | 10mg | 200mg |
| Child 6–12 years: | 5mg | 100mg |
| Child 6 months to 6 years: | 2.5mg | 50mg |
| Child less than 6 months: | 250 micrograms/kg | 25mg |

**Fig. 2.1** Anaphylaxis algorithm.
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# Choking

The management of choking is rightly taught as part of first aid. Recognition of the problem is the key to success. Clues include a person experiencing a sudden airway problem whilst eating, possibly combined with them clutching their neck.

## Severity of airway obstruction

Victims with severe airway obstruction may be unable to speak or breathe and may become unconscious (see Fig. 2.2).

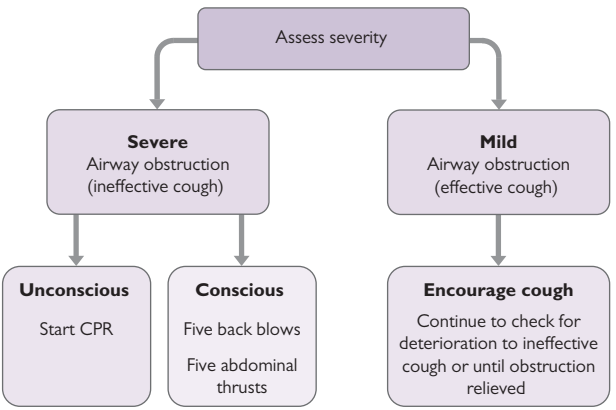


**Fig. 2.2** Adult choking algorithm.

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# Cardiac arrest

## Clinical features and recognition

Follow the resuscitation algorithm (https://www.resus.org.uk) in Fig. 2.3. Cardiac arrest is a *clinical diagnosis*:

- Suspect cardiac arrest in any patient who is unconscious and who does not have signs of life. If you check a pulse, examine only for a major (carotid or femoral) one and take no longer than 10s. Other 'confirmatory' clinical features (eg colour, pupil size/response) waste time and do not help. Note that some respiratory efforts, such as gasping, may persist for several minutes after the onset of cardiac arrest. Occasionally, an arrest may present as a grand mal fit of short duration.
- Most patients have had a sudden and unexpected out-of-hospital event.

Prior warning to the department is usually relayed by radio or direct telephone link from the ambulance service. Whilst resuscitation is continued, ensure that accompanying relatives/friends are met and taken to an appropriate room, which has a telephone and facilities for making tea and coffee and where privacy is possible. Arrange for a member of staff to stay with the relatives to act as a link with the resuscitation team.

## Information to obtain from ambulance crew/relatives

- *Patient details*, including age, past medical history, current medication, and chest pain before event.
- *Times of* collapse (often an approximation), 999 (or 112) call, arrival on scene, start of CPR, first defibrillating shock (if appropriate), other interventions (eg advanced airway management, drugs), restoration of spontaneous circulation (ROSC).
- *Was there any bystander CPR?*

Where a patient in cardiac arrest is brought to hospital by ambulance, the cardiac arrest team (ED staff, the hospital team, or a combination of both) should already be present in the resuscitation room, with all equipment ready, to receive the patient.

## The team leader

The team leader controls, co-ordinates, and organizes the team and makes decisions. Four to six team members are optimal. Each should know their role. Perform resuscitation in a calm, quiet, confident manner with minimal interruption to the performance of Basic Life Support (BLS) or defibrillation.

## Start the following procedures simultaneously

- Continue BLS.
- Remove/cut clothing from the upper body to allow defibrillation, ECG monitoring, chest compression, and IV access.
- Obtain the ECG trace (through defibrillator pads or monitor leads). If already attached to an ECG monitor, note (and print out, if possible) the rhythm. Beware movement artefact, disconnected leads, electrical interference, etc.
- Follow the ALS algorithm (see Advanced Life Support algorithm, p. 54).
- Do not interrupt CPR, except to perform defibrillation.

# In-hospital resuscitation algorithm

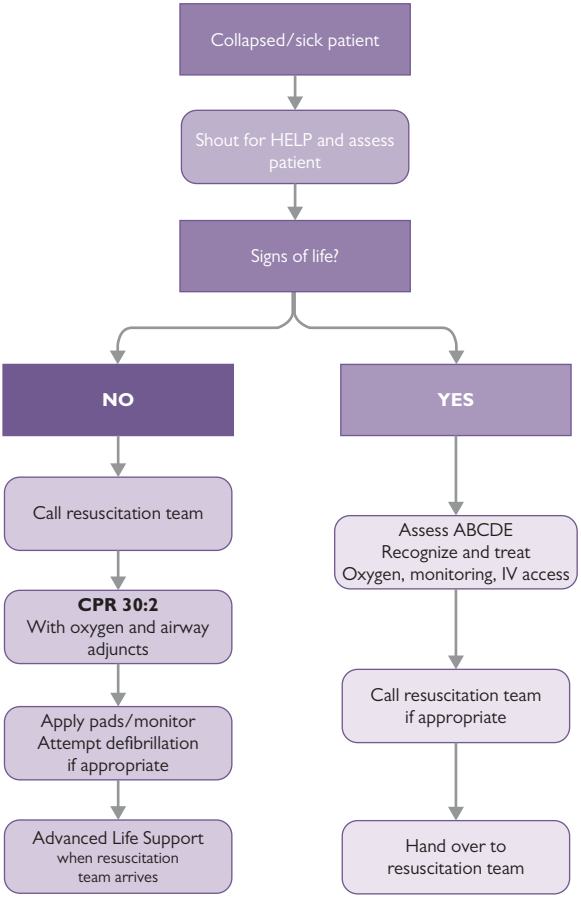


**Fig. 2.3** In-hospital resuscitation algorithm.
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# Adult Basic Life Support

## Airway and ventilation

Usually in the ED, advanced airway techniques will be used from the outset. If basic techniques are used (see Fig. 2.4):

- With the patient on his/her back, open the airway by tilting the head and lifting the chin (use jaw thrust instead if neck trauma suspected).
- Remove any visible obstructions from the mouth, but leave well-fitting dentures in place.
- Aim for each breath to last ~1s, and make the chest rise. After each breath, maintain the head tilt/chin lift; take your mouth away from the patient's, and watch for the chest to fall as the air comes out.

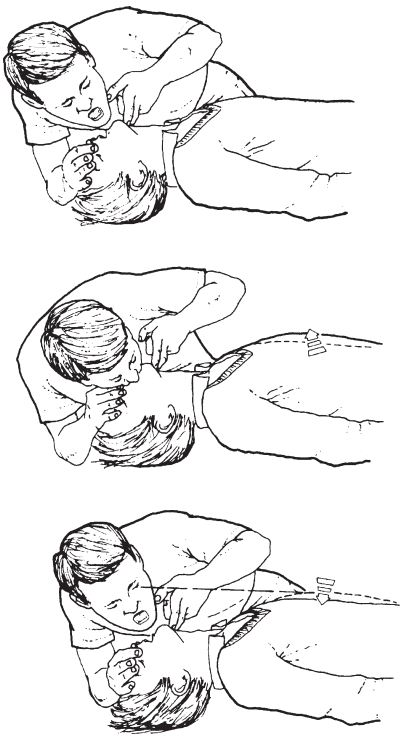

**Fig. 2.4** Mouth-to-mouth ventilation.

### Technique for chest compression

- Place the heel of one hand over the middle of the lower half of the patient's sternum, with the other hand on top. Extend or interlock the fingers of both hands, and lift them to avoid applying pressure to the patient's ribs (see Fig. 2.5.).
- Positioned above the patient's chest and with arms straight, press down to depress the sternum 5–6cm.
- Release all the pressure and repeat at a rate of 100–120/min.
- Compression and release phases should take the same time.
- Use a ratio of 30 chest compressions to two ventilations (30:2).
- Aim to change the person providing chest compressions every 2min, but ensure that this is achieved without causing significant pauses.

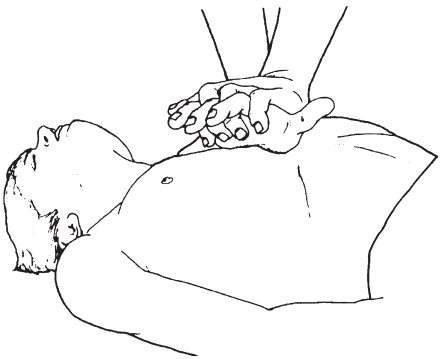

**Fig. 2.5** Chest compressions.

# Cardiac arrest management

## Defibrillation

- Most survivors have an initial rhythm of ventricular fibrillation (VF)/ventricular tachycardia (VT). The treatment for this is defibrillation. With time, the chances of successful defibrillation and survival ↓ dramatically. Adhesive defibrillator pads have replaced manual paddles in most hospitals. Place one pad to the right of the upper sternum below the clavicle, the other at mid-axillary line level with the $V_6$ ECG electrode position. Avoid placement over the ♀ breast. To avoid problems with pacemakers, keep pads >15cm away from them.
- With biphasic defibrillators, use shock energy of 150J; for (mostly older) monophasic defibrillators, select 360J energy—check with each machine.
- Plan for chest compressions to be as continuous as possible, with minimal delays. Having paused briefly to assess the rhythm, recommence compressions until the defibrillator is charged. Pause briefly to deliver a shock (removing $O_2$ sources and GTN patches), then immediately restart CPR with 30:2 compressions:ventilation, and continue for 2min before reassessing the rhythm or feeling for a pulse.
- In monitored patients with pulseless VT/VF where defibrillation is not immediately available, give a single *precordial thump*. With a tightly clenched fist, deliver one direct blow from a height of ~20cm to the lower half of the sternum.

## Airway management

Techniques for securing the airway, providing oxygenation, and ventilation are covered in ➲ Airway obstruction: basic measures, pp. 334–5. Although tracheal intubation has long been considered to be the gold standard definitive airway, only attempt this if suitably experienced. *Supraglottic airway* is a readily available, rapid alternative, which is easy to insert. Whatever method is used, aim to ventilate (preferably with 100% $O_2$) using an inspiratory time of 1s, a volume sufficient to produce a normal rise of the chest, at a rate of 10/min. For patients with tracheal tubes or laryngeal mask airways, ventilate without interrupting chest compressions, which should be continuous (except for defibrillation or pulse checks as appropriate).

*End-tidal $CO_2$* monitoring is very useful to confirm correct tracheal tube placement and indirectly measure cardiac output during CPR.

## Drugs

There is little evidence that *any* drug improves outcome. Central venous cannulation is difficult, has risks, and interrupts CPR. Peripheral access is easy and quick. Having given a peripheral IV drug, give a 20mL saline bolus and elevate the limb for 10–20s. If IV access is impossible, consider the intra-osseous route (➲ Intra-osseous infusion, pp. 656–7). It is no longer recommended for any drugs to be given by tracheal tube. Similarly, do not attempt intracardiac injections.

The first drug used in cardiac arrest (after $O_2$) is adrenaline. In the case of VF/VT, give adrenaline after three shocks, whereas in asystole/PEA, give it as soon as possible (see ➲ Advanced Life Support algorithm, p. 54).

## Non-shockable rhythms: PEA and asystole

*PEA* (pulseless electrical activity) is the clinical situation of cardiac arrest with an ECG trace compatible with a cardiac output. PEA may be caused by:

- Failure of the normal cardiac pumping mechanism (eg massive MI, drugs such as β-blockers and calcium ($Ca^{2+}$) antagonists, or electrolyte disturbances, eg hypokalaemia, hyperkalaemia).
- Obstruction to cardiac filling or output [eg tension pneumothorax, pericardial tamponade, myocardial rupture, pulmonary embolism (PE), prosthetic heart valve occlusion, and hypovolaemia].

Prompt and appropriate correction of these can result in survival. Remember potentially reversible causes as the 4Hs and 4Ts (see Table 2.1).

**Table 2.1** The 4Hs and 4Ts

| 4Hs | 4Ts |
|---|---|
| Hypoxia | Tension pneumothorax |
| Hypovolaemia | Tamponade (cardiac) |
| Hyper-/hypokalaemia/metabolic disorders | Toxic substances (eg overdose) |
| Hypothermia | Thrombosis (PE/MI) |

*Asystole* is the absence of cardiac (particularly ventricular) electrical activity. If unsure whether the rhythm is asystole or fine VF, continue chest compressions and ventilation in an attempt to increase the amplitude and frequency of VF and make it more susceptible to defibrillation.

## Length of resuscitation

The duration of the resuscitation attempt depends upon the nature of the event, the time since the onset, and the estimated prospects for a successful outcome. In general, continue resuscitation whilst VF/pulseless VT persists, always provided that it was initially appropriate to commence resuscitation. If VF persists despite repeated defibrillation, try changing pad position or defibrillator.

Asystole unresponsive to treatment and arrests which last >1hr are rarely associated with survival. However, exceptions occur—particularly in younger patients, hypothermia, near drowning, and drug overdose.

## Mechanical CPR

Several devices can provide mechanical CPR. These include the 'AutoPulse' circumferential load-distributing band chest compression device (comprising a pneumatically actuated constricting band and backboard) and the 'LUCAS' gas-driven sternal compression device (with an accompanying suction cup to provide active decompression). Mechanical CPR is potentially very useful in situations where the resuscitation attempt is prolonged (eg cardiac arrest associated with hypothermia or poisoning or following fibrinolytic treatment for PE), ensuring consistent CPR over a long period of time and freeing up an additional member of the team.

# Advanced Life Support algorithm

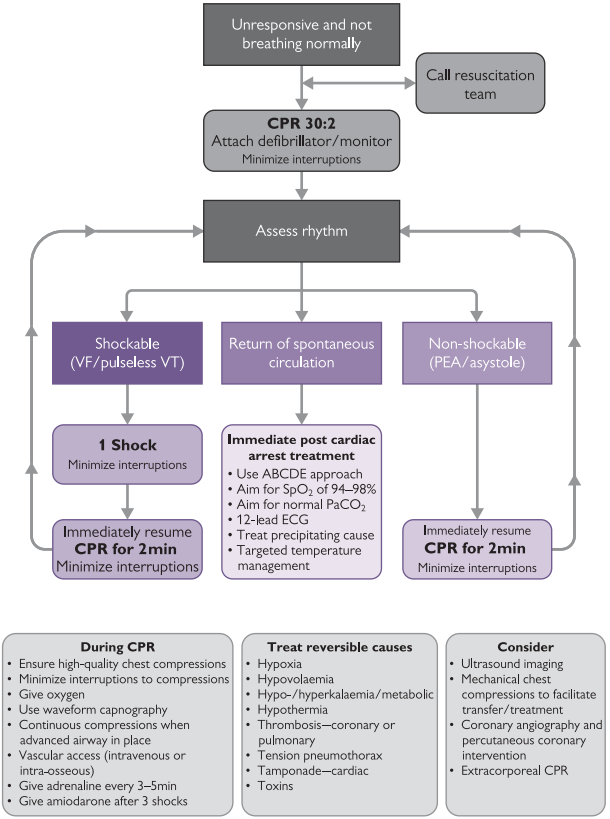


**Fig. 2.6** Advanced Life Support algorithm.

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# Notes on using the Advanced Life Support algorithm

- Establish the underlying cardiac rhythm as quickly as possible in order to determine which 'loop' to follow to provide appropriate treatment—for VF/pulseless VT, the initial focus is defibrillation and good CPR; for asystole/PEA, the initial focus is good CPR, IV adrenaline, and searching for potentially reversible causes (see Fig. 2.6).
- Do not interrupt CPR, except to perform defibrillation.
- Search for, and correct, potentially reversible causes of the arrest.
- Give IV adrenaline 1mg and amiodarone 300mg for VF/pulseless VT refractory to three shocks, followed by adrenaline 1mg every 3–5min. A further dose of IV amiodarone 150mg may be given for recurrent or refractory VF/VT. Lidocaine (1mg/kg) IV is an alternative to amiodarone, but do not give it if amiodarone has already been given.
- For torsades de pointes and refractory VF in patients with suspected digoxin toxicity or hypomagnesaemia (eg on potassium-losing diuretics), give IV magnesium sulfate 2g (= 8mmol = 4mL of 50% solution).
- In asystole and PEA, give IV adrenaline 1mg as soon as possible and thereafter every 3–5min.
- Exercise caution before using adrenaline in arrests associated with cocaine or other sympathomimetic drugs.
- Atropine is no longer routinely recommended in asystole or slow PEA.
- In PEA arrests associated with hyperkalaemia, hypocalcaemia, or $Ca^{2+}$ channel-blocking drug or magnesium overdose, give 10mL of 10% IV calcium chloride (6.8mmol).
- With good-quality CPR, acidosis develops slowly. Do not 'routinely' give an alkali. Give 50mL of sodium bicarbonate 8.4% solution (50mmol) if the arrest is associated with tricyclic overdose (see ➲ Tricyclic antidepressant poisoning, pp. 202–3) or hyperkalaemia, and consider it in patients with severe acidosis (arterial pH <7.1, base excess less than –10). Allow further administration to be guided by repeated ABG results.
- Follow loops of the algorithm for as long as it is considered appropriate for the resuscitation to continue. Provided that the attempt was commenced appropriately, it should not normally be stopped if the rhythm is still VF.

### Pacing and external cardiac percussion

Pacing may be of value in patients with extreme bradyarrhythmias, but its value in asystole is unproven (except for rare cases of asystole with P waves present). If there is a delay before pacing can be performed, external cardiac percussion can provide a cardiac output and 'buy time'. Perform external cardiac percussion using a clenched fist:

- Over the heart at a rate of 100/min.
- With a blow more gentle than a precordial thump.
- Each blow should generate a QRS complex. If this and a detectable output are not achieved, restart conventional CPR.

# Post-resuscitation care

Features such as coma or pupil reflexes are unreliable prognostic indicators in the early post-resuscitation phase. Accurate prognostication in an individual patient is rarely possible before 24–72hr. Involve the ICU/CCU team early.

### Pending this and following ROSC

- Ensure that the airway is protected (➲ Airway obstruction: basic measures, pp. 334–5).
- Maintain oxygenation and ventilation. Correct hypoxia and prevent hypercapnia under ABG guidance [may require intermittent positive pressure ventilation (IPPV)]. Use pulse oximetry to monitor arterial oxygen saturation ($SpO_2$) non-invasively, titrating the inspired $O_2$ concentration to achieve $SpO_2$ of 94–98%.
- In intubated patients, insert an oro- or nasogastric tube to decompress the stomach.
- Obtain a 12-lead ECG and a CXR (check the position of the tracheal tube, central lines, and presence of pneumothorax, etc.).
- Optimize cardiac output: inotropes, vasodilators, fluids, and/or diuretics may be needed under haemodynamic monitoring guidance. If the arrest is associated with an acute coronary syndrome (ACS), consider immediate thrombolysis and/or coronary revascularization (see Fig. 2.7).
- Cerebral blood flow autoregulation is deficient post-arrest. Maintaining arterial pressures 'normal' for the patient may prevent hypotensive hypoperfusion. ↑ BP above the normal for the patient may worsen cerebral oedema.
- Seizures aggravate brain injury by ↑ intracranial pressure (ICP) and cerebral metabolic requirements. Treat with appropriate anticonvulsants (as ➲ Treatment of status epilepticus, p. 157), and ensure adequate oxygenation and ventilation.
- Measure U&E, $Ca^{2+}$, magnesium ($Mg^{2+}$), and correct abnormalities appropriately.
- Obtain FBC to exclude anaemia contributing to myocardial ischaemia and to provide an admission baseline.
- Both hypo- and hyperglycaemia compromise neurological outcome. Monitor plasma glucose concentration regularly, and aim to avoid both hypo- and hyperglycaemia (keep the level ≤10mmol/L).
- No drug has been shown to improve cerebral outcome following a cardiac arrest. The routine use of steroids, mannitol, $Ca^{2+}$ channel blockers, etc. is unwarranted.
- When any drug is used, remember that pharmacokinetic profiles are often impaired post-resuscitation. Dose adjustment and careful monitoring are needed.
- Avoid/treat hyperthermia with an antipyretic or active cooling.

- There are compelling data to support early induction of targeted temperature management (32–36°C) in patients who are comatose following an out-of-hospital VF arrest. Mild hypothermia is believed to be neuroprotective in this situation (and, pending more data, may be of benefit in other situations as well, eg other arrest rhythms, in-hospital arrests, paediatric patients). Cooling may be initiated by external techniques (cooling blankets, water- or air-circulating blankets) or internally by an infusion of 30mL/kg of 4°C 0.9% saline—liaise with ICU. Mild hypothermia is typically maintained for 12–24hr.

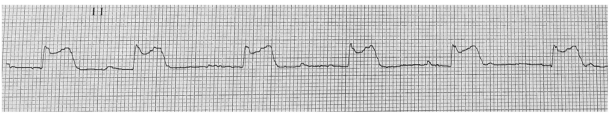

**Fig. 2.7** Post-resuscitation ECG showing ST segment elevation MI (STEMI) with complete heart block.

## Training

Theoretical knowledge is important, but many of the skills required during the management of a cardiac arrest need expert teaching and supervised practice. Attend an approved Resuscitation Council (UK) *Advanced Life Support* course (see https://www.resus.org.uk)—preferably before starting in the ED.

# Central venous access

## Indications

Central venous access may be required for:

- Administration of emergency drugs.
- Central venous pressure (CVP) measurement.
- Administration of IV fluids, especially when peripheral veins are collapsed or thrombosed. *Note*: other routes (eg femoral vein) are generally preferable for giving large volumes rapidly.
- Transvenous cardiac pacing.

## Choice of vein

*The external jugular vein* is often readily visible and can be cannulated easily with a standard IV cannula.

*The internal jugular and subclavian veins* are generally used for central venous access in the ED. Subclavian vein cannulation has a relatively high risk of pneumothorax, so the internal jugular vein is usually preferable via a 'high' approach. Use USS guidance and the right side of the neck (↓ risk of thoracic duct damage). If, however, a chest drain is already *in situ*, use the same side for central venous cannulation.

*The femoral vein* is useful for temporary access in severe trauma and burns and in drug users with many thrombosed veins.

## Seldinger technique for central venous access

This is the method of choice, because the relatively fine needle ↓ the risk of complications such as pneumothorax. The technique involves inserting a hollow metal needle into the vein. A flexible guidewire is threaded through the needle, which is then removed. A tapered dilator and plastic cannula are inserted over the guidewire and advanced into the vein. The guidewire and dilator are removed, and the cannula secured. Once the cannula is in place, check that venous blood can be freely aspirated and secure the cannula.

## Precautions and problems

Central venous access is a specialized technique with potentially life-threatening complications, including pneumothorax, haemothorax, arterial puncture, thoracic duct damage, air embolism, and infection.

- Expert supervision is essential. Cannulation is particularly difficult and hazardous in hypovolaemic, shocked, or agitated patients. In such situations, consider whether it is possible to defer the procedure.
- Use USS to guide central line insertion. USS ↓ complications and failure rates by clarifying the relative positions of the needle, vein, and surrounding structures. Variant anatomy and vein patency can also be assessed by USS.
- Bleeding dyscrasias and anticoagulant treatment are contraindications to internal jugular and subclavian vein access.
- Severe pulmonary disease is a relative contraindication to central venous access, especially by the subclavian route, because a pneumothorax would be particularly dangerous.

## Methods

Use an aseptic technique. If possible, tilt the trolley 10° head down to fill the internal jugular and subclavian veins and ↓ the risk of air embolus. After successful or attempted subclavian or internal jugular vein cannulation, take a CXR to check for pneumothorax and the position of the cannula.

*External jugular vein*

The vein can be seen and felt as it crosses superficially over the sternomastoid muscle and runs obliquely towards the clavicle. Gentle pressure on the lower end of the vein will distend it. A standard IV cannula can easily be inserted into the external jugular vein, but passing a catheter centrally may be difficult because of valves and the angle at which the vein joins the subclavian vein.

*Internal jugular vein*

The internal jugular vein runs antero-laterally in the carotid sheath, parallel to the carotid artery and deep to the sternocleidomastoid muscle. The high approach described has less risk of pneumothorax than lower approaches (see Fig. 2.8).

- Turn the patient's head away from the side to be cannulated.
- Identify the carotid artery and jugular vein on USS.
- Follow the needle tip with USS until the needle is seen to pierce the jugular vein and blood is freely aspirated.
- After inserting the guidewire, confirm positioning in the vein on transverse and longitudinal USS views.

*Subclavian vein (infraclavicular approach)*

(See Fig. 2.9.)

- Turn the patient's head away from the side of cannulation.
- Identify the subclavian artery and vein between the midpoint and the distal third of the clavicle with the USS probe.
- Align the needle with the midpoint of the USS probe which is centred over the subclavian vein. Follow the needle tip until it penetrates the vein and blood is aspirated freely. After inserting the guidewire, confirm the position on both transverse and longitudinal views.

*Femoral vein*

Insert the needle ~1cm medial to the femoral artery and just below the inguinal ligament, pointing slightly medially and with the needle at 20–30° to the skin. If time and expertise allow, use USS guidance.

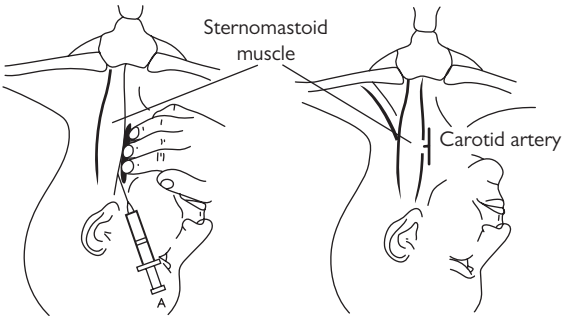


**Fig. 2.8** Internal jugular cannulation.

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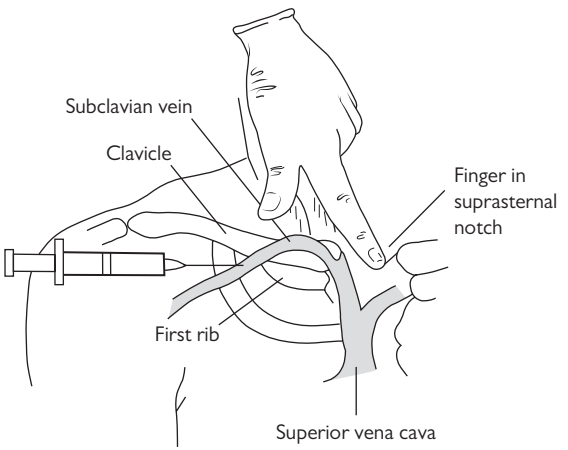


**Fig. 2.9** Subclavian vein cannulation.

# Recognition of the sick patient

## Time-critical illness

There are a relatively large number of conditions where urgent intervention is required in order to secure the best outcome for the patient. Examples include sepsis, MI, major trauma, ruptured abdominal aortic aneurysm, and ruptured ectopic pregnancy. Clinical diagnosis of some of these conditions is not always straightforward, but a common theme is that many present with deranged physiology and vital signs. Implementation of an early warning score on arrival at hospital has helped to identify seriously ill patients.

## National Early Warning Score ('NEWS2')

Following its introduction by the Royal College of Physicians in 2012, the National Early Warning Score was revised in 2017 to produce NEWS2. The original score has been adjusted to take into account the chronic hypoxia of patients with chronic obstructive pulmonary disease (COPD), which generated high baseline scores and led to some instances of excessive $O_2$ administration. Another hope associated with the updated system is that it would help with the early identification of sepsis. NEWS2 is based on six physiological simple, easily measured variables:

- RR.
- $SpO_2$.
- Systolic BP.
- Pulse rate.
- Level of consciousness or new confusion (delirium).
- Temperature.

In addition to NEWS2 being able to identify seriously unwell patients on presentation to hospital, it can also help in the early identification of patients who deteriorate after arrival at hospital and need urgent intervention.

## Clinical acumen

It is not always easy to pinpoint exactly why, but experienced clinicians are often able to quickly identify the fact that a patient is very sick, when compared with less experienced staff. Sometimes, however, the clues are in the history. On other occasions, scrutiny of the prehospital notes can provide vital information. Take particular note of abnormal vital signs (and/or abnormal ECG) as recorded by the paramedics or the GP, even if these have returned to within the normal range by the time of arrival at hospital.

# Sepsis

There is a high mortality rate associated with severe infection at all ages. Sepsis occurs when life-threatening organ dysfunction is associated with infection. It can be insidious in onset and challenging to identify in the ED.

Ask the question: 'does this patient have a suspected infection?'

The initial NEWS (see ➲ National Early Warning Score ('NEWS 2'), p. 61) can give an early indication of those patients with sepsis.

## Suspected sepsis: qSOFA

The Sequential Organ Failure Assessment score has been simplified to the quick SOFA (qSOFA) score to provide one way to assess for sepsis.

Consider sepsis and commence treatment as appropriate if any one (or more) of the following is present (score 1 for each):

- RR ≥22 breaths/min.
- Systolic BP ≤100mmHg.
- Altered mental state (lower GCS than usual).

Patients scoring 2 or 3 have been shown to have ↑ mortality.

Note that an ↑ lactate level can also be helpful in the initial identification of sepsis in the ED (see ➲ Venous blood gases, p. 103).

## Management: the sepsis bundle

Early treatment can improve outcome from sepsis—early IV antibiotics and IV fluids appear to be the most important. The following approach incorporates the 'sepsis 6' care bundle (to be delivered within 1hr of arrival in the ED): $O_2$, blood cultures, IV fluids, IV antibiotics, blood lactate measurement, and urine output monitoring.

- Obtain senior/ICU assistance immediately.
- Assess and manage airway, breathing, and circulation (ABC); in particular, provide high-flow $O_2$ as required—in most patients who require $O_2$, aim for $SpO_2$ of 90–96%. In patients with COPD, a target of 88–92% may be more appropriate (see ➲ Oxygen, p. 99).
- Secure good IV access and take blood samples, including FBC, C-reactive protein (CRP), U&E, LFTs, and a blood lactate level. If the blood lactate level is ≥2mmol/L, repeat the level in 2hr.
- Look for obvious sources of infection (consider a CXR).
- Take blood cultures before starting antibiotics if this does not delay the administration of antibiotics (the choice of antibiotics will depend upon the likely cause and is considered in ➲ Shock, p. 65), but broad-spectrum antibiotics are often appropriate.
- Begin administration of IV crystalloid according to response (eg 10–20mL/kg, although 30mL/kg may be required if hypotensive or if the blood lactate level is ≥4mmol/L). Be cautious with IV fluids in patients who have suspected COVID-19 as a large amount of IV fluid may worsen associated ARDS (see ➲ COVID-19, p. 260).
- Start vasopressors (such as a noradrenaline infusion) for persistent hypotension in order to maintain a mean arterial BP of ≥65mmHg.

See Surviving Sepsis Campaign at https://www.survivingsepsis.org

## Neutropenic sepsis

Systemic chemotherapy (or sometimes radiotherapy) can cause bone marrow suppression and limit the ability of the body to respond to infection. Do not underestimate the extent to which patients with neutropenic sepsis can suddenly deteriorate without much warning—they may not exhibit the typical progressive clinical signs of shock over a number of hours but instead drop their BP precipitously. For this reason, triage ahead and ensure these patients receive immediate attention and resuscitation. Most patients who have received chemotherapy are well aware of the potential risks of neutropenic sepsis and the need to seek urgent medical attention on becoming unwell.

See NICE guidance at https://www.nice.org.uk

Suspect neutropenic sepsis if a patient presents pyrexial or unwell (with symptoms/signs of clinically significant sepsis), with a neutrophil count of $<0.5 \times 10^9$/L, then start treatment accordingly. Do not delay the administration of IV antibiotics whilst waiting for the neutrophil count to be processed by the lab—if in doubt, assume that a patient who has recently received chemotherapy is likely to be neutropenic. Provide the 'sepsis bundle', as outlined in Management: the sepsis bundle, p. 62, including giving broad-spectrum antibiotics according to local protocols—an example of one regime is:

- Piperacillin–tazobactam IV 4.5g every 8hr or
- If penicillin-allergic: ceftazidime IV 2g every 8hr.

It may not be easy to immediately identify an obvious source of infection. Apart from chest, urine, or skin infections, other causes include endocarditis, meningitis, GI perforation, osteomyelitis, sinusitis, and line sepsis.

## Line sepsis

If the patient has a central line or another line *in situ*, consider infection of this and treat as appropriate (seek local advice from haematologist/microbiologist).

# Shock

Shock is a clinical condition characterized by failure to adequately perfuse and oxygenate vital organs. Clinically, shock is recognized by:

- *Hypotension*: generally considered to be systolic BP <90mmHg (in adults), but values may be higher in young, fit, or previously hypertensive patients. Associated *tachycardia* (>100/min) is common but may not be present in patients with cardiac or neurological causes or in those taking β-blockers. A few patients with haemorrhagic shock have paradoxical bradycardia.
- *Altered consciousness* and/or fainting (especially on standing or sitting up) may result from ↓ cerebral perfusion.
- *Poor peripheral perfusion*: cool peripheries, clammy/sweaty skin, pallor, and ↓ capillary return, but note that in the early phase of endotoxic septic shock, there may be vasodilatation with warm peripheries.
- *Tachypnoea*.
- *Purpuric rash*.
- *Oliguria*: ↓ renal perfusion with urine output <50mL/hr (in adults).

## Classification of shock

Traditional classification is artificial; mixed aetiologies are common.

*Hypovolaemic shock*

- Blood loss: trauma, GI bleed (haematemesis, melaena), ruptured abdominal aortic aneurysm, ruptured ectopic pregnancy.
- Fluid loss/redistribution ('third spacing'): burns, GI losses (vomiting, diarrhoea), pancreatitis, sepsis.

*Cardiogenic shock*

- Primary: MI, arrhythmias, valve dysfunction, myocarditis.
- Secondary: cardiac tamponade, massive PE, tension pneumothorax.

*Septic shock*

Includes relative hypovolaemia and cardiogenic shock (see ➲ Sepsis, pp. 62–3). More common at the extremes of age, with diabetes mellitus, renal/hepatic failure, and immunocompromise (eg HIV, underlying malignancy, post-splenectomy, steroid therapy), pregnancy/postnatal, IV drug users, recent surgery, *in situ* IV catheter. Note that fever, rigors and ↑ white cell count (WCC) may not be present.

- Organisms responsible include Gram +ve and –ve, especially *Staphylococcus aureus, Streptococcus pneumoniae, Neisseria meningitidis*, and coliforms including enterococci and *Bacteroides* (especially in patients with intra-abdominal emergencies such as ruptured diverticular abscess). In the immunocompromised, *Pseudomonas*, viruses, and fungi may cause septic shock.

*Anaphylactic shock* See ➲ Anaphylaxis, pp. 44–5.

*Neurogenic shock* See ➲ Spine and spinal cord injury, pp. 390–1.

*Other causes*

These include poisoning (see ➲ Poisons: supportive care, p. 191) and Addison's disease (see ➲ Addisonian crisis, p. 163).

### Management of shock

Investigation and treatment should occur simultaneously. Get senior help immediately.

- Address the priorities—ABC.
- Give $O_2$ as required, according to $SpO_2$ measurements.
- Secure adequate venous access and take blood for FBC, U&E, glucose, LFTs, lactate, coagulation screen, and, if appropriate, blood cultures.
- Monitor vital signs, including pulse, BP, $SpO_2$, and RR.
- Check ABG.
- Monitor the ECG and obtain a 12-lead ECG and a CXR.
- Insert a urinary catheter and monitor urine output hourly.
- For shock associated with ↓ effective circulating blood volume, give IV crystalloid (0.9% saline) titrated in small boluses up to 20–30mL/kg according to response. Give further IV fluids ± blood [aim for haematocrit (Hct) >30%] according to aetiology and clinical response (and, in particular, pulse, BP, CVP, and urine output). Consider using the passive leg raise manoeuvre to help dynamically determine fluid responsiveness. Use caution with IV fluid infusion in shock related to cardiogenic causes and in ruptured or dissecting aortic aneurysm.
- Look for, and treat specifically, the cause(s) of the shock. Echocardiography, USS, CT, and/or surgical intervention may be required. Specific treatments include:
  - *Laparotomy*: ruptured abdominal aortic aneurysm, splenic and/or liver trauma, ruptured ectopic pregnancy, intra-abdominal sepsis.
  - *Thrombolysis/angioplasty*: MI.
  - *Thrombolysis*: PE.
  - *Pericardiocentesis/cardiac surgery*: cardiac tamponade, aortic valve dysfunction.
  - *Antidotes*: for certain poisons.
  - *Antibiotics*: sepsis. The choice of antibiotic will depend upon the perceived cause and local policies (eg ceftriaxone for meningococcal disease). Where there is no obvious source, empirical combination therapy is advised (eg co-amoxiclav + gentamicin + metronidazole). Obtain specialist microbiological advice early, especially in neutropenic/immunocompromised patients.
- Inotropic and vasoactive therapy, assisted ventilation, and invasive monitoring (including arterial and CVP lines) are often needed. Get specialist ICU help early, especially for COVID-19 (see ➲ COVID-19, p. 260.

# Chapter 3

# Medicine

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# Electrocardiogram interpretation

The ECG is normally recorded so that a deflection of 10mm = 1mV. The recording rate is 25mm/s, 1mm = 0.04s, 1 large square = 0.2s. There is an ECG ruler on the inside back cover. Follow a systematic approach. The components of a normal ECG are shown in Fig. 3.1.

**Rate** Calculate the rate by dividing 300 by the number of large squares in one R–R interval.

**Frontal plane axis** Normally lies between –30° and +90° (see Fig. 3.2). With a normal axis, QRS complexes in I and II are both +ve. An axis more –ve than –30° (I +ve, aVF and II –ve) is *left axis deviation* (LAD) (causes: left anterior hemiblock, inferior MI, VT, Wolff–Parkinson–White (WPW) syndrome). An axis more +ve than +90° (I –ve, aVF +ve) is *right axis deviation* (RAD) (causes: PE, cor pulmonale, lateral MI, left posterior hemiblock).

**P wave** Normally <0.12s wide and <2.5mm tall. They are best seen in leads II and $V_1$ which are chosen for rhythm strips or monitoring. A tall peaked P wave in II may reflect right atrial hypertrophy, and a widened bifid P wave left atrial hypertrophy. P waves are absent in atrial fibrillation (AF).

**PR interval** Normally 0.12–0.2s (3–5 small squares). A short PR interval (abnormally fast conduction between atria and ventricles) implies an accessory pathway (eg WPW syndrome). A prolonged PR interval occurs in heart block (*first-, second-, or third-degree*) (see ➲ Bradyarrhythmias, p. 84).

**QRS width** Normally 0.05–0.11s (<3 small squares). Prolonged QRS complexes may be due to: right bundle branch block (RBBB) (RsR′ or M shape in $V_1$), left bundle branch block (LBBB) (QS or W shape in $V_1$ with RsR′ or M shape in $V_6$), tricyclic antidepressant poisoning (see ➲ Tricyclic antidepressant poisoning, p. 202), hypothermia, ventricular rhythms, and ectopics.

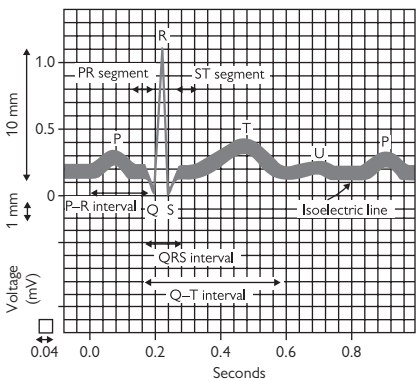


**Fig. 3.1** Components of a normal ECG trace.

**QRS amplitude** The QRS amplitude can indicate left ventricular hypertrophy (LVH). Signs of LVH are: (S in $V_2$ + R in $V_5$) >35mm; R in I >15mm; and R in $aV_L$ >11mm.

**Q waves** May be normal in III, $aV_R$, and $V_1$ but are abnormal in other leads if >0.04s or more than half the height of the subsequent R wave.

**ST segment elevation** Caused by: acute MI, pericarditis (concave up), ventricular aneurysm, Prinzmetal's angina, LVH, Brugada syndrome, hypertrophic cardiomyopathy (HCM), benign early repolarization.

**ST segment depression** Caused by: ischaemia, digoxin, LVH with strain.

**QT interval** = start of Q wave to end of T wave.

$QT_c$ = QT/√R–R (Bazett's formula). Normal $QT_c$ is <440ms. At rates of 60–100/min, QT should be less than half the R–R interval.

A *prolonged* $QT_c$ predisposes to 'torsades de pointes' (see ➔ Broad complex tachyarrhythmias, p. 91) and is caused by acute MI, hypothermia, hypocalcaemia, drugs (quinidine, tricyclic antidepressants), and certain congenital diseases (eg Romano–Ward syndrome).

**T waves** Abnormal if inverted in $V_4$ to $V_6$. Peaked T waves are seen in early acute MI and hyperkalaemia (see ➔ Hyperkalaemia, p. 170). Flattened T waves (sometimes with prominent U waves) occur in hypokalaemia.

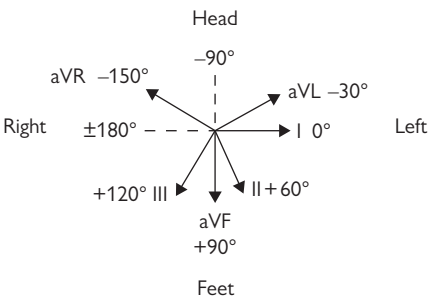


**Fig. 3.2** Diagram of the ECG frontal axis.

# Chest pain

Take chest pain seriously—it may reflect life-threatening illness. Triage as 'urgent', so patients are seen within a few minutes. IHD is understandably the first diagnosis to consider, but chest pain may be due to other disease processes, which may also be potentially life-threatening (see Table 3.1).

**Table 3.1** The differential diagnosis of chest pain

| Common causes | Less common causes |
|---|---|
| Musculoskeletal (eg costochondritis) | Aortic dissection* |
| Acute coronary syndrome* | Cholecystitis |
| Pneumothorax* | Herpes zoster |
| Oesophagitis | Oesophageal rupture* |
| Pneumonia | Pancreatitis* |
| Pulmonary embolism* | Vertebral collapse |
| Obscure origin (eg precordial catch) | Tabes dorsalis (very rare) |

* Potentially rapidly fatal.

Reaching the correct conclusion requires accurate interpretation of the history, examination, and investigations, bearing in mind recognized patterns of disease presentations.

## History

*Characterize the pain*

- Site (eg central, bilateral, or unilateral).
- Severity.
- Time of onset and duration.
- Character (eg 'stabbing', 'tight/gripping', or 'dull/aching').
- Radiation (eg to arms and neck in myocardial ischaemia).
- Precipitating and relieving factors (eg exercise/rest/GTN spray).
- Previous similar pains.

*Enquire about associated symptoms* Breathlessness, nausea, and vomiting, sweating, cough, haemoptysis, palpitations, dizziness, loss of consciousness.

*Document* Past history, drug history, and allergies. Old notes and old ECGs are invaluable—review them at an early stage.

*Quickly consider* Contacting cardiologists if ACS is likely (see ➲ STEMI: treatment, pp. 80–1).

## Examination and resuscitation

Evaluate ABC and resuscitate ($O_2$, venous access, IV analgesia), as appropriate. Listen to both lung fields to check for tension pneumothorax and severe left ventricular failure (LVF). Complete full examination.

## Investigations

These depend upon the presentation and likely diagnosis, but an ECG and a CXR are usually required. Remember that these may initially be normal in MI, PE, and aortic dissection. Ensure that all patients receive ECG monitoring in an area where a defibrillator is readily available.

# Cardiac ischaemia

Angina occurs when coronary artery blood flow fails to meet the myocardial $O_2$ demand (eg exercise, coronary artery spasm, anaemia). It may cause ST depression or T wave inversion, which resolves on recovery.

Patients may come to the ED with angina as a first presentation of IHD. Always consider the possibility of MI, particularly if any pain lasts >10min.

► *A normal examination, a normal ECG, and a normal baseline troponin do not exclude MI. If in doubt, undertake formal rule-out with serial troponins.*

If considering discharge (eg with daily aspirin and chest pain clinic follow-up), discuss with senior ED staff.

## Rule out strategies for acute coronary syndrome

Acute MI is still occasionally missed. Cardiac chest pain may be poorly localized and present with musculoskeletal features or GI upset. If the history is suspicious of being cardiac (especially with risk factors), formally exclude ACS. Interpret ECG and troponin in clinical context (old ECGs help). Become familiar with the hospital's troponin assay—there are many, with different 'cut' points to diagnose ACS. Unless significantly ↑, most high-sensitivity troponin assays use a 20% rise in absolute troponin level (over >3hr) to diagnose ACS, rather than a defined level. Clinical scores, such as the HEART score (see Table 3.2), may help to determine the probability of underlying myocardia ischaemia. If serial troponins are negative and HEART score low, ACS is an unlikely cause of the pain.

**Table 3.2** HEART score for myocardial ischaemia

| Risk factor | Feature | Points |
|---|---|---|
| History | Highly suspicious | 2 |
| | Moderately suspicious | 1 |
| | Slightly suspicious | 0 |
| ECG | Significant ST deviation | 2 |
| | Non-specific repolarization changes | 1 |
| | Normal | 0 |
| Age | >65y | 2 |
| | 45–65y | 1 |
| | <45y | 0 |
| Risk factors | ≥3 from: ↑ cholesterol, ↑ BP, smoker, diabetes, family history, obesity | 2 |
| | 1–2 risk factors | 1 |
| | No risk factors | 0 |
| Troponin | >3x normal limit | 2 |
| | Between 1–3x normal limit | 1 |
| | Under normal limit | 0 |

Total 0–3 = low score; 4–6 = moderate score; 7–10 = high score.

# Acute coronary syndromes

Coronary artery plaque rupture can result in a variety of ischaemic conditions which fall under the overall term of 'acute coronary syndrome' and include unstable angina, non-ST segment elevation MI (NSTEMI), and ST segment elevation MI (STEMI). Patients labelled as having ACS, but without initial ST elevation, comprise a relatively heterogenous group—some later proving (on the basis of elevated blood troponin levels) to have suffered an NSTEMI.

## The patient presenting with unstable angina or NSTEMI

Unstable angina can occur as worsening angina or as a single episode of 'crescendo' angina, with a high risk of infarction. Features include angina at rest, ↑ frequency, ↑ duration, and severity of pain (including response to GTN). It may be difficult to distinguish between unstable angina and NSTEMI in the ED. ECG changes may be subtle or non-existent (see Figs. 3.3 and 3.4).

- Provide $O_2$ if $SpO_2$ is <90%—aim to maintain $SpO_2$ at 90–94%.
- Attach a cardiac monitor.
- Administer IV opioid analgesia (± antiemetic) as required.
- Give aspirin 300mg orally (PO), if not already administered prehospital.
- Give clopidogrel 300mg PO (or prasugrel 60mg PO or ticagrelor 180mg PO), according to local guidelines.
- Start fondaparinux 2.5mg subcutaneous (SC) daily, unless there is a high risk of bleeding, renal impairment, or there is a plan to go to the Cath Lab, in which case give IV unfractionated heparin instead.
- If pain is unrelieved, commence GTN intravenous infusion (IVI). Start at 0.6mg/hr and ↑ as necessary, provided systolic BP is >90mmHg.
- Discuss all patients with NSTEMI and TIMI (thrombolysis in myocardial infarction) score >3 (see Table 3.3) or GRACE score >100 with the cardiology team—they may benefit from an early revascularization procedure (via angiography in the Cath Lab).
- If the patient has a high TIMI/GRACE score and a low risk of bleeding, consider glycoprotein IIb/IIIa inhibitors (eg eptifibatide and tirofiban) with IV heparin. *Note*: bivalirudin is an alternative—follow local protocols.
- If high risk of NSTEMI, haemodynamically stable, and no contraindications, consider atenolol (5mg IV slowly over 5min, repeated once after 15min), according to local policy. Contraindications include: hypotension, bradycardia, second- or third-degree heart block, heart failure, and severe reactive airways disease.
- Maintain blood glucose <11mmol/L.
- Refer for admission, repeat ECGs, and troponin.

**Table 3.3** TIMI risk score: increasing score predicts mortality or adverse event

| Risk factor | Points |
|---|---|
| Age >65 | 1 |
| 3+ risk factors for coronary artery disease: Family history of IHD, hypertension, hypercholesterolaemia, diabetes, or smoker | 1 |
| Known coronary artery disease with stenosis ≥50% | 1 |
| Aspirin use in last 7 days | 1 |
| Recent episode of angina prior to this event | 1 |
| Raised troponin levels (or other cardiac marker) | 1 |
| ST segment deviation ≥0.5mm on ECG | 1 |

### Normal lead II

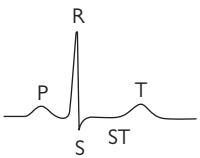


**Fig. 3.3** Normal lead II.

### Ischaemic changes in lead II

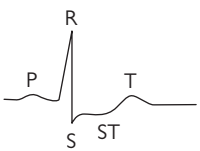


**Fig. 3.4** Ischaemic changes in lead II.

### Prinzmetal's or 'variant' angina

Angina associated with ST elevation may be due to coronary artery vasospasm. This may occur with or without a fixed coronary abnormality and may be indistinguishable from an acute MI until changes resolve rapidly with GTN as pain is relieved.

# ST segment elevation MI

IHD is the leading cause of death in the Western world. Contributory risk factors for MI include smoking, hypertension, age, ♂ sex, diabetes, hyperlipidaemia, and family history.

## MI pathology

MI mostly affects the left ventricle (LV). It usually results from sudden occlusion of a coronary artery or one of its branches by thrombosis over a pre-existing atheromatous plaque. Patients with IHD are at risk of sustaining an MI if additional stresses are placed upon their already critically impaired myocardial circulation (eg a high level of carboxyhaemoglobin (COHb) following smoke inhalation). MI may also occur in vasculitic processes, eg cranial arteritis (see ➲ Giant cell arteritis, p. 137) and Kawasaki disease.

## MI diagnosis

The diagnosis of acute MI requires two out of the following three features:

- A history of cardiac-type ischaemic chest pain.
- Evolutionary changes on serial ECGs.
- A rise in serum cardiac markers.

Note that 50–60% of patients will not have a diagnostic ECG on arrival and up to 17% will have an entirely normal initial ECG. Late presentation does not improve the diagnostic accuracy of the ECG.

## History

*The classic presentation* is of sudden onset, severe, constant central chest pain, which radiates to the arms, neck, or jaw. This may be similar to previous angina pectoris but is much more severe and unrelieved by GTN. The pain is usually accompanied by one or more associated symptoms: sweating, nausea, vomiting, and breathlessness.

*Atypical presentation* is common. Have a high level of suspicion. Many patients describe atypical pain, some attributing it to indigestion (be wary of new-onset 'dyspeptic' pain). Up to a third of patients with acute MI do not report any chest pain. These patients tend to be older, are more likely to be ♀, have a history of diabetes or heart failure, and have higher mortality. These patients may present with:

- LVF.
- Collapse or syncope (often with associated injuries, eg head injury).
- Confusion.
- Stroke.
- An incidental ECG finding at a later date.

In a patient who presents with possible MI, enquire about past medical history (IHD, hypertension, diabetes, hyperlipidaemia) and contraindications to thrombolysis. Ask about drug history, including drugs of abuse (particularly cocaine; see ➲ Cocaine, p. 223).

## Examination

Examination and initial resuscitation (maintain $SpO_2$ in the normal range, IV cannula, analgesia) go hand in hand. The patient may be pale, sweaty, and distressed. Examination is usually normal, unless complications have supervened (eg arrhythmias, LVF). Direct initial examination towards searching for these complications and excluding alternative diagnoses:

- Check pulse and BP, and monitor trace (? arrhythmia or cardiogenic shock).
- Listen to the heart (? murmurs or third heart sound).
- Listen to the lung fields (? LVF, pneumonia, pneumothorax).
- Check peripheral pulses (? aortic dissection).
- Check legs for evidence of deep vein thrombosis (DVT) (? PE).
- Palpate for abdominal tenderness or masses (? cholecystitis, pancreatitis, perforated peptic ulcer, ruptured aortic aneurysm).

## Investigations

The diagnosis of STEMI within the first few hours is based upon the history and ECG changes (serum cardiac markers may take several hours to rise—see below).

- Record an ECG as soon as possible, ideally within a few minutes of arrival at hospital. Sometimes patients arrive at hospital with ECGs of diagnostic quality already recorded by paramedics. If the initial ECG is normal, but symptoms are suspicious, repeat the ECG after 15min and re-evaluate (see ➲ Myocardial infarction: ECG changes 1, pp. 76–7 for a detailed explanation of the typical ECG features of acute MI).
- Review old notes (and, most importantly, previous ECGs for comparison).
- Ensure continuous cardiac monitoring and pulse oximetry.
- Monitor BP and RR.
- Obtain venous access and send blood for cardiac markers, U&E, glucose, FBC, and lipids.
- Obtain a CXR if there is suspicion of LVF or aortic dissection.

## Cardiac markers

Troponins are now universally used. Troponin T (cTnT) and troponin I (cTnI) are proteins virtually exclusive to cardiac myocytes. However, cardiac cells may release troponin into the blood when cardiac muscle is damaged by pericarditis, PE with a large clot burden, or sepsis. Renal impairment reduces the excretion of troponin, so can result in higher levels.

# Myocardial infarction: ECG changes 1

Infarction of cardiac muscle results in ECG changes that evolve over hours, days, and weeks in a relatively predictable fashion (see Fig. 3.5).

## Hyperacute changes

Frequently ignored, although often subtle, some or all of the following may be observed within minutes of infarction:

- ↑ *ventricular activation time*, since the infarcting myocardium is slower to conduct electrical impulses. The interval between the start of the QRS complex and the apex of the R wave may be prolonged >0.045s.
- ↑ *height of R wave* may be seen initially in inferior leads in inferior MI.
- *Upward-sloping ST segment*—having lost normal upward concavity, the ST segment straightens, then slopes upwards, before becoming elevated.
- *Tall, widened T waves.*

## Evolving acute changes

In isolation, none of these changes are specific to MI. In combination and with an appropriate history, they can diagnose MI:

- *ST elevation*: the most important ECG change. ST segments become concave down and are significant if elevated >1mm in two limb leads or >2mm in two adjacent chest leads (see Figs. 3.6 and 3.7).
- *Reciprocal ST depression* may occur on the 'opposite side' of the heart (see Fig. 3.6).
- *Pathological Q waves* (defined in ➲ Electrocardiogram interpretation, p. 69) reflect electrically inert necrotic myocardium. ECG leads over a large transmural infarct show deep QS waves. Leads directed towards the periphery of a large infarct or over a smaller infarct may show a QR complex or a *loss of R wave amplitude*.
- *T wave inversion*: typically deeply inverted, symmetrical, and pointed.
- *Conduction problems* may develop. LBBB in a patient with acute cardiac chest pain makes interpretation of the ECG very difficult. LBBB does not have to be new to be significant. Do not delay intervention in patients with a good clinical history of MI in order to obtain old ECGs.

*Sgarbossa criteria for diagnosing ACS in the presence of LBBB*

- ST segment elevation >1mm in leads with positive QRS complexes.
- ST segment depression in leads $V_1$, $V_2$, or $V_3$.
- ST segment elevation >5mm in leads with negative QRS complexes.

If all three are present, MI is likely.

## Chronic changes

In the months following an MI, ECG changes resolve to a variable extent. ST segments become isoelectric, unless a ventricular aneurysm develops. T waves gradually become +ve again. Q waves usually remain, indicating MI at some time in the past.

## Electrocardiogram changes following myocardial infarction


**Fig. 3.5** Electrocardiogram changes following myocardial infarction.

## Electrocardiograms after myocardial infarction

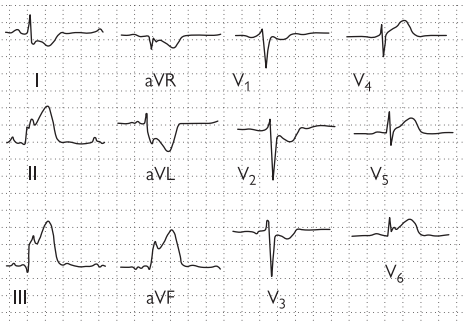


**Fig. 3.6** Acute inferolateral infarction with 'reciprocal' ST changes in I, $aV_L$, and $V_2$ to $V_3$.

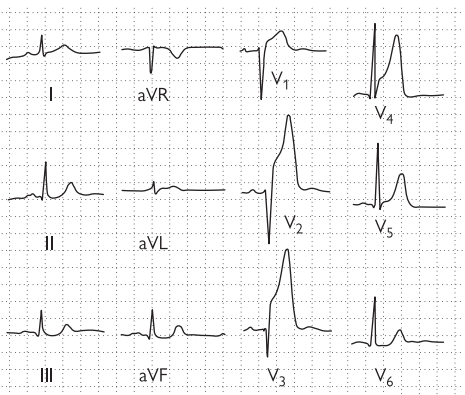


**Fig. 3.7** Acute anteroseptal infarction with minimal 'reciprocal' ST changes in III and $aV_F$.

# Myocardial infarction: ECG changes 2

## Localization of myocardial infarction

MI usually affects the LV and occasionally the right ventricle (RV), but virtually never the atria. The part of the myocardium affected is implied by which leads show changes (see Table 3.4).

**Table 3.4** ECG leads showing changes in various MIs

| ECG leads | Location of MI |
|---|---|
| $V_{1–3}$ | Anteroseptal |
| $V_{5–6}$, $aV_L$ | Antero-lateral |
| $V_{2–4}$ | Anterior |
| $V_{1–6}$ | Extensive anterior |
| I, II, $aV_L$, $V_6$ | Lateral |
| II, III, $aV_F$ | Inferior |
| $V_1$, $V_4R$ | RV |

## Posterior myocardial infarction

Posterior MI nearly always occurs as part of inferior (postero-inferior) or lateral (postero-lateral) MI. No conventional electrode views the posterior heart, since intervening tissues result in an attenuated signal. ECG diagnosis of true posterior MI may be made from the use of $V_{7–9}$ and from reciprocal changes seen in leads $V_{1–3}$: tall, slightly widened R (reciprocal of Q), concave-up ST depression (reciprocal of ST elevation), and upright tall, widened T (reciprocal of inverted T).

## Right ventricular infarct

This occurs most often as part of an inferior MI. In the presence of changes of acute MI in the inferior leads, ST elevation in $V_1$ suggests RV involvement. In this case, record an ECG trace from lead $V_4R$. The diagnosis of RV infarct helps determine treatment of ensuing cardiac failure. Treat RV failure with IV fluids to maintain adequate filling pressure, and exercise caution if considering use of nitrates.

## Blood supply to the heart and coronary artery dominance

The left anterior descending artery supplies the anterior and septal cardiac areas (see Fig. 3.8). The circumflex branch supplies the antero-lateral aspect of the heart. The right coronary artery supplies the RV. In most people, the right coronary artery also supplies the sino-atrial (SA) node, the inferior wall of the LV, and the ventricular septum. In 15% of individuals, the inferior wall is supplied by the circumflex branch of the left coronary artery (left dominance).

Subendocardial infarcts can produce dramatic ECG changes (see Fig. 3.9) without the ST elevation seen in transmural infarction.

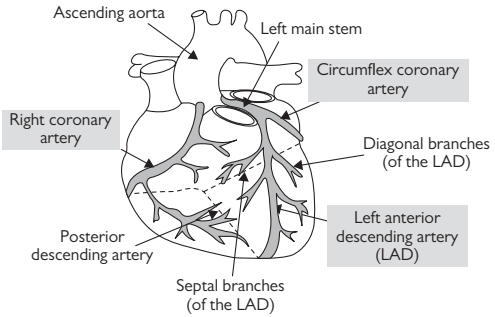


**Fig. 3.8** Blood supply to the heart.

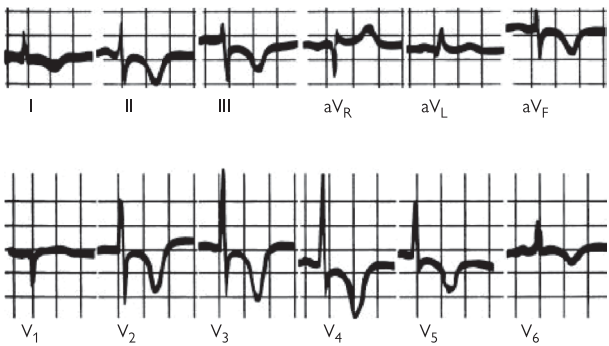


**Fig. 3.9** ECG of subendocardial infarct.

# STEMI: treatment

Speed is crucial—time is muscle. Work efficiently as a team to ensure treatment is not delayed.

- Give $O_2$ if needed (to maintain $SpO_2$ 90–94%; see ➔ Oxygen, p. 99), and attach a cardiac monitor.
- Contact cardiology for primary percutaneous coronary intervention (PCI).
- Provide IV morphine, titrated to effect (± antiemetic).
- Ensure the patient has had aspirin 300mg—this is likely to have been given prehospital, but check and give it if not.
- Obtain IV access and take samples for U&E, glucose, FBC, and troponin.
- Give a second antiplatelet agent as per local protocol (eg ticagrelor loading dose 180mg PO, or prasugrel or clopidogrel). Avoid ticagrelor if the patient has a history of intracranial haemorrhage, has active bleeding or moderate hepatic impairment, and is on an anticoagulant.
- Arrange immediate transfer to the Cath Lab for PCI if <12hr from symptom onset (or >12hr with cardiogenic shock).
- If PCI cannot be delivered within 90min, assess the risk of bleeding and offer thrombolysis using local protocol (tenecteplase, reteplase, or alteplase).
- If pain continues, give IVI GTN (start at 0.6mg/hr and ↑ as necessary), provided systolic BP is >90mmHg.
- Consider atenolol (5mg IV slowly over 5min, repeated once after 15min) or metoprolol, unless contraindicated (eg uncontrolled heart failure, hypotension, bradyarrhythmias, COPD).

### Indications for PCI or thrombolysis

- ST elevation of >1mm in two limb leads, or
- ST elevation of ≥2mm in two or more contiguous chest leads, or
- LBBB in the presence of a typical history of acute MI (*Note*: LBBB does not have to be new—see ➔ Sgarbossa criteria for diagnosing ACS in the presence of LBBB, p. 76).

### Primary angioplasty for ST segment elevation MI

Primary PCI (coronary angioplasty and stenting) is the treatment of choice for STEMI. Compared to thrombolysis, PCI administered within 12hr of symptom onset results in lower mortality and re-infarction rates. The sooner it is performed, the greater the benefits.

### Thrombolysis

If PCI cannot be performed within 90min of diagnosis, thrombolysis is an alternative. The benefits reduce markedly with time delay, so if PCI is not available, do not delay thrombolysis. Rural areas with long hospital transfers may have a protocol for ambulance-administered thrombolysis, aided by telemedicine advice from the ED or cardiology. Patients presenting >12hr after symptom onset will not benefit from thrombolysis.

Strokes, intracranial haemorrhage, and major bleeds are more common in patients given thrombolysis. Intracranial bleeding is more common in older patients, those with hypertension on admission, and those given tissue plasminogen activator (tPA). Prior to administering thrombolysis, always explain the benefits and risks. Obtain verbal consent to give it and record this in the notes.

*Contraindications to thrombolysis*

Absolute contraindications include:

- Stroke in past 3 months, neurosurgery within 6 months, intracranial bleed within a year.
- Any cerebral tumour or metastases.
- GI, genitourinary (GU), retroperitoneal, or intraocular bleeding within the last month.
- Coagulopathy (eg haemophilia), anticoagulation (warfarin, rivaroxaban, apixaban, dabigatran, edoxaban, or LMWH).
- Platelet count $<50 \times 10^9$/L.
- Severe hypertension: systolic BP >200mmHg, diastolic BP >120mmHg.
- Major surgery within the last 2 weeks.

Relative contraindications include:

- Pregnancy.
- Stroke within past year.
- Major bleeding within past 3 months.
- History of any intracranial bleed.
- Traumatic CPR.
- Puncture of non-compressible vessel (eg subclavian vein).
- Major surgery within past month.

*Choice of thrombolytic agents*

- Alteplase (recombinant tPA (rtPA)): give by an accelerated regimen, eg 15mg IV bolus, followed by 0.75mg/kg (max 50mg) IVI for 30min, then 0.5mg/kg (max 35mg) IVI over 60min. Give LMWH (eg enoxaparin 1mg/kg stat) or heparin concomitantly through a separate IV line (5000U IV bolus, then 1000U/hr IV), according to local protocols.
- Reteplase (modified tPA): give as two IV boluses of 10U each, exactly 30min apart. Give LMWH/heparin as for alteplase.
- Tenecteplase (modified tPA): give as a single IV bolus over 10s. Dose according to weight (<60kg = 30mg; 60–69kg = 35mg; 70–79kg = 40mg; 80–89kg = 45mg; >90kg = 50mg). Give LMWH/heparin as for alteplase.
- Streptokinase: give as 1.5 mega-units by IVI over 1hr. Streptokinase is allergenic (may need slow IV chlorphenamine 10mg and IV hydrocortisone 100mg) and causes hypotension (↓ IVI rate and tilt the bed head down—treatment rarely needs to be stopped). Patients can develop antibodies to streptokinase, so do not give if administered in the past year.

## Further management

*Arrhythmias* Occur commonly after MI. Occasional ventricular ectopics or transient AF (lasting <30s) require no treatment. Watch for sudden VT/VF and treat as described in ➲ Cardiac arrest, p. 48.

*Hypokalaemia* Treat if $K^+$ <4mmol/L.

*Pulmonary oedema* Treat as described in ➲ Cardiogenic pulmonary oedema, p. 105.

*Cardiogenic shock* Mortality is high. Contact ICU/cardiology. Emergency echocardiography may exclude conditions requiring urgent surgery (mitral regurgitation from papillary muscle rupture, aortic dissection, ventricular septum rupture, cardiac tamponade from ventricular wall rupture). If these are excluded, emergency coronary intervention may ↑ survival.

# Acute pericarditis

This characteristically produces chest pain, low-grade fever ± intermittent pericardial friction rub. Pericarditis and myocarditis often coexist.

## Causes

- Viruses (Coxsackie virus A9, B1–4, echovirus 8, mumps, EBV, CMV, varicella, HIV, rubella, parvovirus B19)) are believed to be responsible for most cases affecting previously well young or middle-aged adults.
- MI (including Dressler's syndrome—see ➔ Pericarditis, management, p. 83).
- Bacterial infection (pneumococcus, meningococcus, *Chlamydia*, gonorrhoea, *Haemophilus*).
- Tuberculosis (TB) (especially in patients with HIV) (see ➔ Tuberculosis, p. 242).
- Locally invasive carcinoma (eg bronchus or breast).
- Rheumatic fever (see ➔ Rheumatic fever, p. 513).
- Uraemia.
- Collagen vascular disease: systemic lupus erythematosus (SLE), polyarteritis nodosa (PAN), rheumatoid.
- After cardiac surgery or radiotherapy.
- Drugs (hydralazine, procainamide, methyldopa, minoxidil).

## Diagnosis

Classical features of acute pericarditis are pericardial pain, friction rub, and concordant ST elevation on ECG. The characteristic combination of clinical presentation and ECG changes often results in a definite diagnosis.

- *Chest pain* is typically sharp, central, retrosternal, and worse on deep inspiration, and change in position, on exercise, and on swallowing. A large pericardial effusion may cause dysphagia by compressing the oesophagus.
- A *pericardial friction rub* is often intermittent, positional, and elusive. It tends to be louder during inspiration and may be heard in both systole and diastole. Low-grade fever is common.
- *Appropriate investigations* include: ECG, CXR, FBC, CRP, U&E, and troponin. Note troponin may be ↑ in pericarditis—consider repeat/serial troponins ± other investigations if there is doubt about the underlying cause. Obtain blood cultures if there is evidence of sepsis or suspicion of a bacterial cause (eg spread of intrathoracic infection). A pericardial effusion is most quickly and easily demonstrated by bedside echocardiography; clinical evidence of cardiac tamponade is rare.

## ECG changes

In *acute pericarditis*, ECG changes result from associated epicardial inflammation (see Fig. 3.10). Sinus tachycardia is usual, but AF, atrial flutter, or atrial ectopics may occur. ST elevation is concave up (unlike MI—see ➔ Myocardial infarction: ECG changes 1, p. 76) and present in at least two limb leads and all chest leads (most marked in $V_{3-6}$). T waves are initially prominent, upright, and peaked, becoming flattened or inverted over several days. PR depression (reflecting atrial inflammation) may occur in the same leads as ST elevation (this PR–ST discordance is characteristic). Pathological Q waves are not present.

*Pericardial effusion* causes ↓ QRS amplitude in all leads. Electrical alternans is diagnostic, but rare.

## Management

The appropriate treatment depends on the underlying cause.

*Idiopathic pericarditis or viral pericarditis* in young patients is usually benign and self-limiting. Admit patients with high-risk features: pyrexia >38°C, ↑ WCC, large pericardial effusion/tamponade, acute trauma, immunosuppression, oral anticoagulation, failure of NSAID treatment (see https://www.rcemlearning.co.uk). Advise rest, with avoidance of exercise/sport until symptoms resolve. Occasionally, it follows a relapsing course before 'burning itself out'. If symptoms do not respond to NSAIDs, the GP may consider colchicine.

Admit patients with *other causes of pericarditis* for investigation and management. Dressler's syndrome (autoimmune pericarditis ± effusion 2–14 weeks after 3% of MIs) requires cardiology specialist care.

*Pericardial effusion* may occur with any type of pericarditis. It is relatively common in acute bacterial, tuberculous, and malignant pericarditis. Acute tamponade may occur following cardiac rupture with MI, aortic dissection, or after cardiac surgery. Summon senior help and arrange immediate bedside echocardiography for patients with signs of tamponade, with pericardiocentesis under USS guidance, and then a definitive drainage procedure.

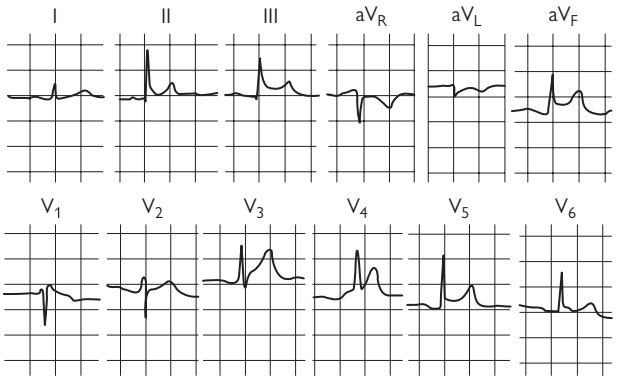


**Fig. 3.10** ECG of pericarditis.

# Bradyarrhythmias

Bradycardia is a ventricular rate of <60/min in the adult. It usually reflects influences on, or disease of, the SA node, or atrioventricular (AV) block. Intraventricular conduction disturbances may progress to AV block. Sinus bradycardia may be physiological (eg athletes), due to drugs (β-blockers), or pathological (hypothyroidism, hypothermia, hypoxia, ↑ ICP, sick sinus syndrome, MI (see Fig. 3.11), myocardial ischaemia). Bradycardia also occurs in up to one-third of patients with hypovolaemia (eg GI bleed, ectopic pregnancy).

## Sick sinus syndrome ('sinus node disease')

Is usually the result of ischaemia or degeneration of the SA node. It is characterized by sinus pauses (>2s) or sinus arrest. Junctional or other escape beats may occur, and occasionally a tachyarrhythmia may emerge ('tachy-brady' syndrome). Patients may present with dizziness, collapse, loss of consciousness, or palpitations. A continuous 24-hr ECG tape may demonstrate arrhythmias.

## AV block

*Causes* IHD, drugs (eg excess digoxin), or cardiac surgery.

*First-degree AV block* Conduction from atria to ventricles occurs every time but is delayed. The PR interval is >0.2s (five small squares on standard ECG) (see Fig. 3.12).

*Second-degree AV block* Only a proportion of P waves are conducted to the ventricles. There are two types:

- *Mobitz type I block (Wenckebach)*: the PR interval becomes increasingly lengthened until a P wave fails to conduct (see Fig. 3.13).
- *Mobitz type II block*: failure to conduct P waves may occur regularly (eg 3:1) or irregularly, but the PR interval remains constant (see Fig. 3.14).

*Third-degree (complete) heart block* Atrial activity is not conducted to the ventricles. With a proximal block (eg at the AV node), a proximal escape pacemaker in the AV node or bundle of His may take over, producing narrow QRS complexes at a rate of ~50/min. With distal AV block, a more distal escape pacemaker results in broad, bizarre complexes at a rate of ~30/min. Ventricular asystole may occur if the escape pacemaker stops discharging, unless a subsidiary pacemaker takes over (see Fig. 3.15).

## Intraventricular conduction disturbances

The intraventricular conducting system commences as the bundle of His and divides into right and left bundle branches—the latter subdivides further into antero-superior and postero-superior divisions. These two divisions and the right bundle branch are referred to as the 'fascicles'. Blockage of two out of three fascicles = *bifascicular block*.

- RBBB + left anterior hemiblock (blockage of the left antero-superior fascicle) causes LAD and an RBBB pattern on ECG.
- RBBB + left posterior hemiblock causes RAD and an RBBB pattern on ECG.

Bifascicular block accompanied by a prolonged PR interval is often referred to as *trifascicular block*. Note that true blockage of all three fascicles would cause complete heart block, so bifascicular block with prolonged PR interval represents impending progression to complete heart block.

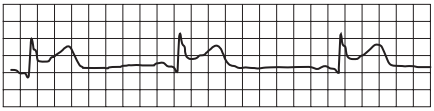

**Fig. 3.11** ECG of sinus bradycardia with STEMI.

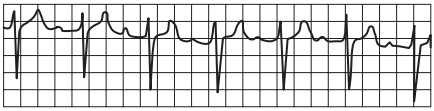

**Fig. 3.12** ECG of first-degree heart block.

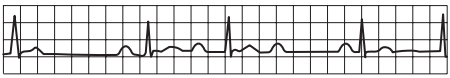

**Fig. 3.13** ECG of Mobitz type I AV block.

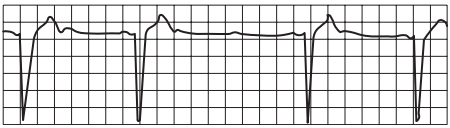

**Fig. 3.14** ECG of Mobitz type II AV block.

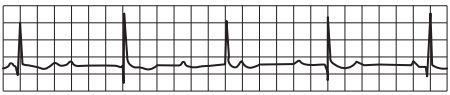

**Fig. 3.15** ECG of complete AV block.

# Treatment of bradyarrhythmias

The emergency treatment of bradycardia depends upon two important factors: the clinical condition of the patient and the risk of asystole. Give $O_2$ if hypoxic; insert an IV cannula, and follow the Resuscitation Council guidelines shown in Fig. 3.17 (https://www.resus.org.uk).

*Atropine* is the first-line drug. The standard dose is 500mcg IV, which may be repeated to a total of 3mg. Further doses are not effective and may result in toxic effects (eg psychosis, urinary retention).

*Adrenaline (epinephrine)* can be used as a temporizing measure prior to transvenous pacing if an external pacemaker is not available. Give by controlled infusion at 2–10mcg/min, titrating up according to response (adrenaline 6mg in 500mL of 0.9% saline infused at 10–50mL/hr). A (traditional) alternative to using an IVI of adrenaline is to use an isoprenaline IVI.

*External transcutaneous pacing* allows a pacing current to be passed between adhesive electrodes placed over the front of the chest and the back. Select external demand pacing mode at a rate of 70/min, then gradually ↑ the pacing current from zero until capture is shown on the monitor. Clinically, capture results in a palpable peripheral pulse at the paced rate and clinical improvement. Provide small doses of IV opioid ± sedation as needed.

*Transvenous cardiac pacing* is the treatment of choice for bradycardic patients who are at risk of asystole. The technique should only be performed by an experienced doctor. The preferred route of access is the internal jugular or subclavian vein. However, if thrombolysis has recently been given or is contemplated, or if the patient is taking anticoagulants, use the right femoral vein instead. Obtain a CXR to exclude complications. A correctly functioning ventricular pacemaker results in a pacing spike followed by a widened and bizarre QRS (see Fig. 3.16).

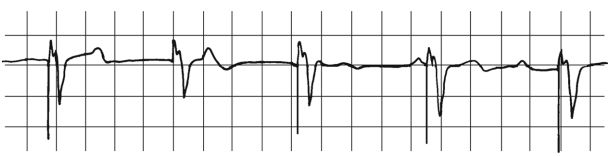

**Fig. 3.16** Paced rhythm.

## Algorithm for the management of bradycardia

(See Fig. 3.17; see also https://www.resus.org.uk)

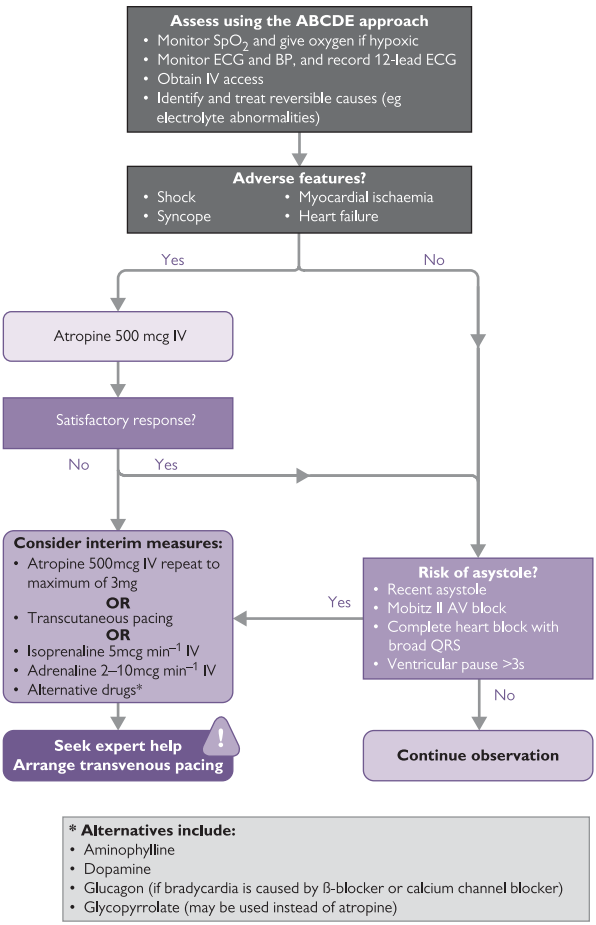


**Fig. 3.17** Algorithm for the management of bradycardia, 2015.

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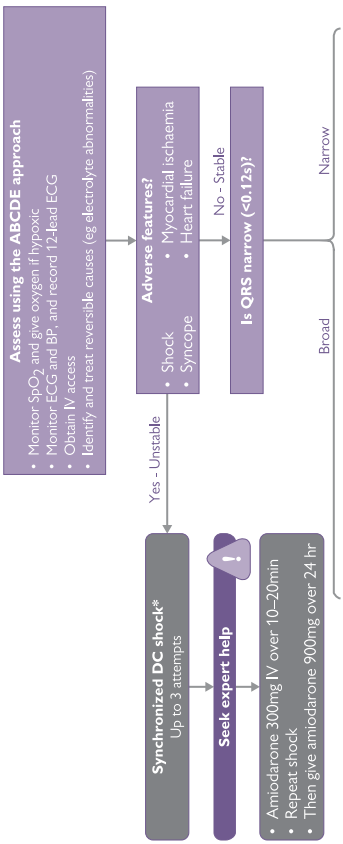
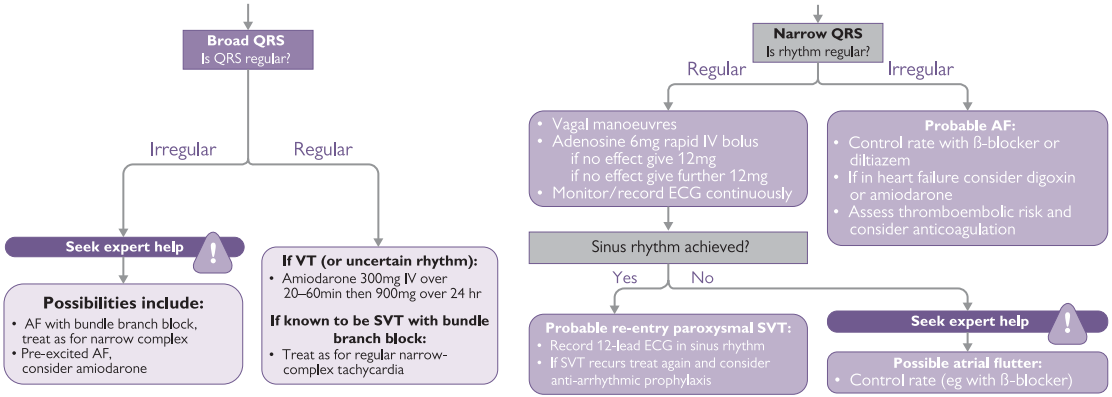
# Tachycardia algorithm—with pulse

(See Fig. 3.18.)






*Conscious patients require sedation or general anaesthesia for cardioversion

**Fig. 3.18** Tachycardia algorithm with pulse, 2015.
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# Tachyarrhythmias

The single Resuscitation Council 2015 tachycardia algorithm (see Fig. 3.18) (see https://www.resus.org.uk) is based on the fact that, irrespective of the exact underlying cardiac rhythm, many of the initial management principles in the peri-arrest setting are the same:

- Rapidly assess Airway, Breathing, and Circulation.
- Monitor cardiac rhythm and record a 12-lead ECG.
- Provide $O_2$ if hypoxic.
- Identify and treat reversible causes.
- Assess for adverse features (signs of shock, syncope, signs of heart failure, or myocardial ischaemia)—these indicate the need for urgent intervention, initially in the form of synchronized cardioversion.

## The unstable patient with tachyarrhythmia

### *Synchronized cardioversion*

This requires two doctors—one to perform cardioversion, the other (experienced in anaesthesia) to provide sedation/anaesthesia and manage the airway. The patient will not be fasted and is therefore at particular risk of aspiration. The arrhythmia will almost certainly ↓ cardiac output and ↑ circulation times, so IV drugs take much longer to work than usual. If the 'sedation doctor' does not appreciate this and gives additional doses of anaesthetic drugs, hypotension and prolonged anaesthesia may result.

Synchronize electrical cardioversion so that it occurs with the R wave to minimize the risk of inducing VF. Synchronized cardioversion is effective in treating patients who exhibit evidence of instability with underlying rhythms of supraventricular tachycardia (SVT), atrial flutter, AF, and VT—choose an initial level of energy according to the rhythm and defibrillator:

- For broad complex tachycardia or AF, start with 120–150J (biphasic). If unsuccessful, ↑ in increments.
- Start with a lower energy level for atrial flutter and paroxysmal SVT—use 70–120J (biphasic). If this is unsuccessful, ↑ in increments to 150J.

### *Amiodarone*

If cardioversion is unsuccessful after three synchronized shocks, give amiodarone IV 300mg over 10–20min and repeat the shock. Give amiodarone by central vein, when possible, as it causes thrombophlebitis when given peripherally. However, in an emergency, it is acceptable to use a large peripheral vein.

## Clinically stable patient with tachyarrhythmia

Tailor treatment according to the likely underlying rhythm. Establish if the QRS is broad or narrow (<0.12s) and if the rhythm is regular or not, then treat as outlined in Fig. 3.18.

# Broad complex tachyarrhythmias

May be caused by VT (see Fig. 3.19) or, rarely, by SVT with aberrant conduction. The default position should be that broad complex tachycardia is VT. Provide $O_2$ as appropriate; insert an IV cannula, and follow the Resuscitation Council guidelines (see Fig. 3.18 and https://www.resus.org.uk).

The priorities in broad complex arrhythmias associated with tricyclic antidepressant overdose are airway management, oxygenation, ventilation, and correction of metabolic disorders—give IV bicarbonate, but avoid antiarrhythmic drugs (see Tricyclic antidepressant poisoning, pp. 202–3).

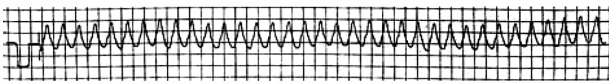

**Fig. 3.19** ECG of VT.

## Evaluating ECGs: VT or SVT with aberrant conduction?

VT is much more likely as a cause of broad complex tachycardia if:

- The patient is >60y.
- The patient has a history of IHD or cardiomyopathy.
- There is clinical evidence of AV dissociation (intermittent cannon 'a' waves seen on JVP, first heart sound of variable intensity).
- Inverted P waves in lead II.
- The frontal plane axis is bizarre (–90° to –180°).
- The QRS is >0.13s.
- There are 'capture' or 'fusion' beats.
- The QRS is bizarre, not resembling a bundle branch block pattern.
- All chest leads ($V_{1-6}$) concordant (QRS complexes point the same way).
- R > R′ (or r′) in $V_1$.
- There is a deep S wave (QS, rS, or RS) in $V_6$.

## Torsades de pointes

Rare form of polymorphic VT, associated with hypomagnesaemia, hypokalaemia, long QT interval (congenital or drug-related, eg sotalol, antipsychotics, antihistamines, antidepressants). A constantly changing electrical axis results in QRS complexes of undulating amplitude (see Fig. 3.20). Usually paroxysmal, it may degenerate to VF. Stop drugs that might prolong QT and avoid amiodarone. Correct electrolyte abnormalities. Get expert help and treat with IV magnesium sulfate (2g over 10min = 8mmol or 4mL of 50% magnesium). Refractory cases may require overdrive pacing. Arrange synchronized cardioversion if adverse features.

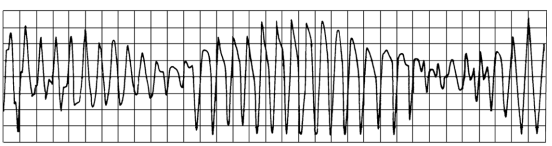

**Fig. 3.20** ECG of torsades de pointes.

# Regular narrow complex tachyarrhythmias

Most patients presenting with regular narrow complex tachycardia (see Fig. 3.21) that is not sinus tachycardia have paroxysmal SVT which responds to vagal manoeuvres or adenosine. If the ventricular rate is exactly 150/min, atrial flutter with 2:1 block is likely. *Note*: vagal manoeuvres/adenosine may temporarily slow the heart rate and reveal the rhythm.

Give $O_2$ if hypoxic; insert an IV cannula, and follow the algorithm in Fig. 3.18 (see also https://www.resus.org.uk).

## Unstable patients

*Treat the compromised patient* (shock, syncope, acute cardiac failure, or cardiac ischaemia) with emergency electrical cardioversion. Consider vagal stimulation and/or giving IV adenosine whilst arranging the cardioversion, as long as this does not delay the procedure.

## Stable patients

- *Try vagal stimulation*. The most effective way is a modified Valsalva manoeuvre. Whilst semi-recumbent, instruct the patient to attempt to blow the plunger out of a 20mL syringe for 15s, then lie the patient supine and manually raise the legs for 15s.
- *Carotid sinus massage* of the carotid sinus for 15s (one side only), by gently rubbing in a circular action lateral to the upper border of the thyroid cartilage, is used less frequently now. It may be dangerous (especially if there is a carotid bruit or risk of stroke/TIA).
- *Adenosine* temporarily blocks conduction through the AV node. It has a very short half-life (10–15s) and can successfully terminate re-entrant tachycardias and may 'unmask' other conditions (eg atrial flutter) by temporarily producing a conduction block. It is contraindicated in second- or third-degree AV block, severe hypotension, and patients with asthma. The effects are blocked by theophylline and potentiated markedly (and dangerously) in the presence of dipyridamole or carbamazepine or in a denervated heart—seek advice. Warn the patient about transient flushing and chest discomfort. Give adenosine by fast IV bolus 6mg into an IV cannula in the antecubital fossa and flush with 0.9% saline (see Fig. 3.18) whilst recording a rhythm strip. If unsuccessful, repeat with 12mg (then use 12mg again, if needed).
- If adenosine is contraindicated, consider IV *verapamil* 2.5–5mg over 2min. Avoid verapamil in patients with cardiac failure, hypotension, concomitant β-blocker therapy, or WPW.

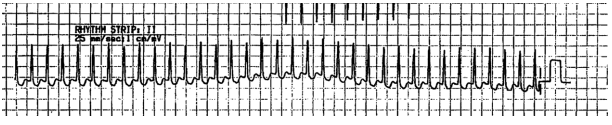


**Fig. 3.21** Narrow complex tachycardia.

# Atrial fibrillation

Most patients with a fast, irregular pulse are in AF. This is characterized by rapid, irregular, unco-ordinated atrial activity, associated with an irregular ventricular response. It is a common condition with numerous causes.

## Causes

Acute AF may be associated with: IHD (33%), heart failure (24%), hypertension (26%), and valvular heart disease (7%). *Other cardiac causes* are sick sinus syndrome, pericarditis, infiltrative heart disease, cardiomyopathy, myocarditis, congenital heart disease, and post-cardiac surgery.

*Non-cardiac causes* include: sepsis, PE, thyrotoxicosis, electrocution, lung or pleural disease, chest trauma, hypokalaemia, hypovolaemia, hypothermia, drug abuse (eg cocaine), and 'holiday heart syndrome'. Paroxysmal AF sometimes occurs in fit athletes.

## Clinical features

Some patients may present to the ED with palpitations and other symptoms as a result of suddenly developing AF; some may develop AF as part of a serious acute illness (eg sepsis), whilst in others it may be an apparently incidental finding. AF ↓ cardiac output by 10–20%, irrespective of the underlying ventricular rate.

Clinical presentation varies according to the cause and effect of AF. Some patients are asymptomatic; others suffer life-threatening complications (heart failure, angina). Patients with underlying IHD may develop ischaemia during periods of rapid ventricular rate.

## Treatment

Patients in AF can be treated with rate or rhythm control. Rhythm control can be achieved by either chemical or electrical cardioversion.

If signs of shock, syncope, acute cardiac failure, or ischaemia, consider electrical cardioversion under sedation (as shown in Fig. 3.18). Patients may be chemically cardioverted with flecainide 50–150mg IV or 300mg PO (contraindicated in patients with cardiac disease) or amiodarone 300mg IV (safer in patients with cardiac disease). Both drugs may cause hypotension.

If the patient has had symptoms for longer than 48hr, he/she is at risk of cardiac thromboembolism and stroke when cardioverted, so instead, give rate control medications and commence oral anticoagulant or LMWH. Rate control drugs include metoprolol 5mg IV and diltiazem (IV form not available in the UK). Digoxin 500mcg IV is the drug of choice in patients with congestive cardiac failure. (See NICE guidelines at https://www.nice.org.uk)

AF ↑ the risk of stroke. This risk can be quantified by applying the $CHADS_2VA_2SC$ score (see https://www.mdcalc.com)—any patient scoring >1 should be considered for anticoagulation (eg apixaban, rivaroxaban, edoxaban, or dabigatran). The decision on whether and what treatment to commence will be informed by current renal function, previous bleeding events, and the need to minimize co-prescription of antiplatelet drugs. Local policy will determine if this will occur in the ED or in early GP follow-up.

# Hypertensive problems

- Most patients with hypertension are asymptomatic.
- Hypertension is an important risk factor for cardiovascular disease and stroke.
- Most patients found to be hypertensive in the ED do not require any immediate intervention or treatment, but do require careful follow-up—usually by their GP.
- Never intervene on the basis of a single raised BP measurement in the absence of any associated symptoms and signs.

## Hypertensive emergency

↑ BP with rapid-onset neurological signs, retinopathy, myocardial ischaemia, or renal failure. BP is often >230/130mmHg. Search for evidence of hypertensive encephalopathy: headache, nausea, vomiting, confusion, retinal changes (haemorrhages, exudates, papilloedema), fits, focal neurological signs, ↓ conscious level. Check for symptoms of aortic dissection. Consider recent drug ingestion (eg ecstacy or cocaine).

*Investigations*

Insert an IV cannula and send blood for U&E, creatinine, and glucose. Obtain a CXR and an ECG, and perform urinalysis. If there is ↓ conscious level, focal signs, or other clinical suspicion that the hypertension may be secondary to stroke or intracranial haemorrhage, arrange an emergency CT scan. If there is concern for aortic dissection, request a CT angiogram.

*Management*

In a true hypertensive emergency (eg encephalopathy, aortic dissection, or intracranial haemorrhage), aim to reduce BP by no more than 25% in the first hour. If treatment is appropriate, commence an IVI of sodium nitroprusside, labetalol, or GTN, with continuous BP monitoring via an arterial line and admit to HDU or ICU. Sodium nitroprusside has a very short half-life (~1–2min) and acts as a vasodilator of both arterioles and veins. IV labetalol may be preferred if aortic dissection (see ➲ Aortic dissection, p. 97) or phaeochromocytoma are suspected.

β-blockers are contraindicated in hypertension caused by cocaine, amphetamine, or related sympathomimetic drugs (see ➲ Recreational drugs, pp. 222–3), since β-blockade may cause unopposed α-adrenergic activity with paradoxical hypertension and ↓ coronary blood flow.

## Hypertension in pregnancy

Hypertension may be part of pre-eclampsia or eclampsia (see ➲ Medical complications of pregnancy, p. 606). Pre-eclampsia is diagnosed with two or more of: hypertension (>140/90mmHg), proteinuria, and oedema. This can be associated with haemolysis, elevated LFTs, low platelets (HELLP syndrome). Check urine for protein, and check blood for FBC, LFTs, platelets, uric acid level, and coagulation screen. Call for senior obstetric help. Eclampsia is diagnosed with the onset of grand mal seizures after 20 weeks' gestation and carries a significant mortality rate.

# Implantable cardiac devices

## Pacemaker letter codes

Enable pacemaker identification:

- First letter: chamber paced (A = atria; V = ventricles; D = dual chamber).
- Second letter: chamber sensed (A = atria; V = ventricles; D = dual; 0 = none).
- Third: pacemaker response (T = triggered; I = inhibited; D = dual; R = reverse).
- Fourth (P = programmable; M = multi-programmable).
- Fifth (P = the pacemaker will pace in tachycardia; S = the pacemaker shocks in tachycardia; D = dual ability to pace and shock; 0 = none of these).

## Types of pacemaker malfunction

*Failure to capture* Pacing spikes, but no QRS (battery or lead problem, exit block caused by infarcted muscle or electrolyte abnormality).

*Undersensing* Inappropriate pacing spikes (lead displacement, low-voltage native QRS complexes).

*Oversensing* Too few pacing spikes (picking up T waves or extracardiac potentials, mobile phones).

*Abnormal rates* Low battery, conduction from ventricles to atria.

## Types of defibrillator problems

*Increased shocks* Oversensing of T waves causing ↑ shocks. Sensing of extracardiac potentials causing ↑ shocks. External transcutaneous pacing will provide temporary support whilst the problem is resolved. A special magnet may be needed to inactivate an implantable defibrillator which fires repeatedly. Admit patients whose implantable defibrillator has fired.

*VT/VF* Sustained or recurrent VT/VF cardiac arrest from lead displacement, low battery, failure to respond to shock.

*Infection and pocket haematoma* Common early after insertion of both pacemaker and defibrillator.

## Left ventricular assist devices

Placed either as a 'bridge' to cardiac transplant or as 'destination therapy' to prolong life in end-stage cardiac failure. They pump blood from LV into aorta; battery-dependent. Patient carries backup battery and charger (as a backpack or belt). Future LVADs may have internal implantable batteries. Battery may last 4–6hr and will alarm when low. LVAD patients are anticoagulated with warfarin. They may not have a palpable pulse. Doppler or arterial line will measure BP. Do not give chest compressions in cardiac arrest because of risk of displacement. The device may have a hand pump for use in cardiac arrest.

Problems with these devices are most common soon after insertion:

- Battery depletion: check that the light is on. Ensure the device is plugged in and backup battery is to hand.
- Bleeding: intracranial, pulmonary, GI, and other. Do not reverse anticoagulation without seeking expert advice, as it carries a high risk of LVAD thrombosis (and patient demise).
- Clotting: embolic stroke, ischaemic limb or gut, LVAD thrombosis causing device failure. Machine may feel hot to touch in LVAD thrombosis.
- Infection: treat for severe sepsis.

# Aortic dissection

►► *Remember*: patients (especially those with hypertension) with sudden severe chest and/or back pain may have acute aortic dissection.

## Pathology

Aortic dissection is longitudinal splitting of the muscular aortic media by a column of blood. The dissection may spread proximally (possibly resulting in aortic incompetence, coronary artery blockage, cardiac tamponade) or distally (possibly involving the origin of various arteries), or rupture internally back into the aortic lumen or externally (eg into the mediastinum, resulting in rapid exsanguination).

More than 70% of patients have a history of hypertension. It occurs more frequently in those with a bicuspid aortic valve, Marfan's syndrome, or Ehlers–Danlos syndrome. Up to 20% follow recent cardiac surgery or recent angiography/angioplasty.

Dissection may be classified as Stanford type 'A' or 'B', according to whether the ascending aorta is involved or not, respectively (see Fig. 3.22).

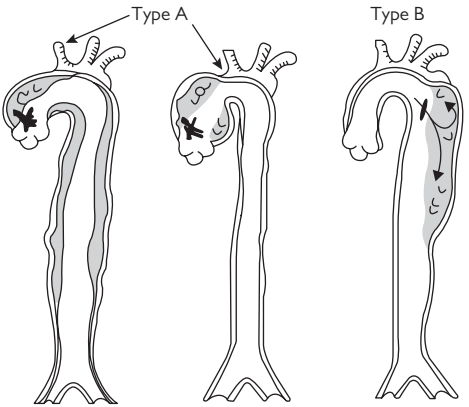


**Fig. 3.22** Stanford classification of aortic dissection.

## History

Aortic dissection may mimic an MI, so adopt a high index of suspicion. It typically presents with abrupt-onset sharp, tearing, or ripping pain (maximal at onset) in the anterior or posterior chest. The pain can resolve, then recur in the epigastrium or elsewhere. Pain migration may reflect dissection extension. Sometimes the patient is pain-free after the initial insult. Syncope occurs in ~10%, sometimes without any pain. Occasionally, patients present with an acute stroke, with neurological deficit plus chest pain. Involvement of the coeliac artery can cause bowel ischaemia. Likewise, involvement of the renal arteries can cause acute kidney injury (AKI).

## Examination

The patient is usually apprehensive and distressed, with pain which is difficult to alleviate, even with using IV opioid. Clues to the diagnosis include:

- An aortic regurgitation murmur (30%).
- Asymmetry or absence of peripheral pulses or a pulse deficit.
- Hypertension.
- Hypotension with features of tamponade or neurological signs in association with pain (eg secondary to spinal/carotid artery involvement).

## Investigations

Send blood for U&E, glucose, FBC, coagulation, and cross-matching. Obtain an ECG and a CXR. Thoracic aortic dissection usually results in an abnormal CXR. One or more of the following changes may be seen:

- A widened or abnormal mediastinum (present in ~75%).
- Left pleural effusion (~20%).
- Deviation of the trachea or nasogastric (NG) tube to the right.
- A 'double-knuckle' aorta and/or separation of the two parts of the wall of a calcified aorta by >5mm (the 'calcium sign').

The ECG may show MI, LVH, or ischaemia. *Note*: ~12% of patients with aortic dissection have a normal CXR and ~30% have a normal ECG. CT angiography will provide the definitive diagnosis (see Fig. 3.23).

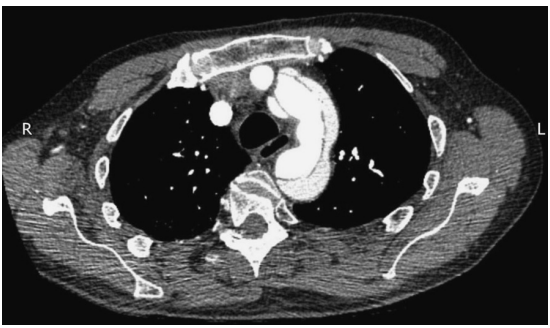


**Fig. 3.23** CT scan showing dissection of the aortic arch.

## Management

On suspicion of an aortic dissection:

- Provide $O_2$ by face mask as appropriate (see ➲ Oxygen, p. 99).
- Insert two large-bore (14G) IV cannulae and cross-match for 6U.
- Give IV morphine and titrate according to response (± antiemetic).
- Call the cardiothoracic team and the cardiologist at an early stage.
- Insert an arterial line (preferably right radial artery), and discuss with specialist teams how to control the BP (eg labetalol infusion).
- Arrange further investigation based upon specialist advice and available resources (eg aortography, echocardiography, CT scan, MRI).

Type A dissections are usually treated surgically; type B lesions are usually treated medically.

# Haemoptysis

Haemoptysis may be the chief or sole complaint of patients presenting to the ED. It always warrants investigation. For causes of haemoptysis, see Table 3.5.

**Table 3.5** Causes of haemoptysis

| | |
|---|---|
| *Respiratory* | • Carcinoma (bronchial or laryngeal)<br>• Infection (upper respiratory tract infection, pneumonia, TB, lung abscess)<br>• Bronchiectasis |
| *Cardiovascular* | • Pulmonary oedema<br>• PE<br>• Ruptured aortic aneurysm (aorto-bronchial fistula) |
| *Coagulation disorder* | • Drugs (eg warfarin, rivaroxaban, apixaban, dabigatran)<br>• Inherited (eg haemophilia) |
| *Trauma* | • Penetrating or blunt |
| *Other* | • Goodpasture's, granulomatosis with polyangiitis |

## Presentation

Ascertain the exact nature and volume (eg 'bright red streaks' or 'dark brown granules'). Patients sometimes have surprising difficulty distinguishing vomited blood from that coughed up. Enquire about weight loss, and take a drug history and a smoking history.

## Investigations

- Send blood for FBC, coagulation screen, U&E, and LFTs.
- Request group and save if evidence of significant haemorrhage.
- If $SpO_2$ <90% on air or the patient has COPD, check ABG.
- Obtain CXR and ECG. Request CT if lung cancer suspected.
- Perform urinalysis—if shocked, insert a catheter and monitor output.
- Collect sputum samples. Send for microscopy, culture, and sensitivity.
- Initiate further investigations according to the likely diagnosis.

## Treatment

- Airway: clear and secure (coughing/suction). Put on a face mask and shield if maintaining the airway or intubating. Ensure nearby high-flow suction. Massive haemorrhage may require tracheal intubation. Whilst preparing for this, tilt the trolley so that the patient is head down.
- Breathing: provide $O_2$ to maintain $SpO_2$ at 90–94%. If ventilation is inadequate, assist with bag and mask or tracheal tube.
- Circulation: insert a large-bore (14G) IV cannula (use two if hypovolaemic). Give IV fluids/blood/clotting factors, as clinically indicated (see ➔ Blood transfusion overview, pp. 180–1).

*Further treatment* Commence specific treatment measures aimed at the life-threatening underlying cause (eg LVF, PE, infection, coagulopathy). In cases of large haemoptysis, admit for further investigation and treatment. If the patient is stable and has only had a small amount of bloodstained sputum, urgent outpatient investigation may be appropriate.

# Oxygen

$O_2$ is the most commonly administered hospital therapy. $O_2$ therapy is controlled and targeted, as it has been recognized that giving too much $O_2$ can ↑ mortality for medical patients in hospital.

## Oxygen requirements

*Patients with high oxygen requirements*

A small number of patients with certain specific conditions may benefit from the provision of high-flow $O_2$, with the target of $SpO_2$ approaching 100%, including:

- Carbon monoxide (CO) poisoning (see ➔ Carbon monoxide poisoning, p. 216).
- Cluster headaches (see ➔ Cluster headache, p. 138).
- Sickle-cell crisis (see ➔ Sickle-cell disease, pp. 184–5).
- Pneumothorax (see ➔ Spontaneous pneumothorax, pp. 118–20).

*Most patients with acute medical illness*

The aim of $O_2$ therapy is to optimize tissue $O_2$ delivery. Use pulse oximetry to guide whether the patient requires supplemental $O_2$. In most previously healthy patients with acute medical illness who require $O_2$, aim for $SpO_2$ of ≤96%, with a target range of $SpO_2$ of 90–94%.

*Patients with COPD*

In patients with known COPD or type II respiratory failure, aim for $SpO_2$ of 88–92%. Take an ABG in patients with chronic lung disease, to assess their optimal $O_2$ treatment (see ➔ Arterial blood gases, p. 102 on ABG interpretation). Repeat the ABG within 30min after changing the inspired $O_2$ concentration ($FiO_2$).

*Prescribing oxygen*

Include the target $SpO_2$, the $O_2$ mask type, and the $O_2$ flow rate in the $O_2$ prescription. In an emergency, it is appropriate to administer $O_2$ prior to prescribing, but do not forget to prescribe the $O_2$ after resuscitation. (See https://www.brit-thoracic.org.uk)

*Oxygen cylinders*

When administering $O_2$ in the ED, always use piped $O_2$ from the wall outlet. Only use an $O_2$ cylinder when transporting the patient to the radiology department or ward. $O_2$ is highly flammable. Do not take a cylinder out of its support cage. In the UK, $O_2$ cylinders are colour-coded white. The most common small cylinder is B (or M-6), which holds 170L of $O_2$. The most common large cylinder is E (or M-24), holding 680L of $O_2$. Before a patient leaves the ED, check that the cylinder is full. If the patient is being transferred to another hospital, ensure there is enough $O_2$ for the journey. The formula is:

Volume of cylinder in L/flow rate = how long cylinder will last in minutes

# The dyspnoeic patient

The normal adult RR is 11–18/min, with a tidal volume of 400–800mL. Acute dyspnoea is a common presenting symptom.

## Common causes of acute dyspnoea

*Cardiac*

- Cardiogenic pulmonary oedema (see ➲ Cardiogenic pulmonary oedema, pp. 104–5).
- MI (see ➲ Acute coronary syndromes, pp. 72–3).
- PE (see ➲ Pulmonary embolism, pp. 124–5).
- Arrhythmias (see ➲ Bradyarrhythmias, p. 84).

*Respiratory*

- Asthma (see ➲ Acute asthma: assessment, pp. 108–9) or exacerbation of COPD (see ➲ Chronic obstructive pulmonary disease, pp. 112–13).
- Pneumonia (see ➲ Pneumonia, pp. 114–15).
- Pleural effusion (see ➲ Pleural effusion, p. 107).
- Pneumothorax (see ➲ Spontaneous pneumothorax, pp. 118–20).

*Trauma*

- Aspiration of FB or vomit (see ➲ Pulmonary aspiration, pp. 116–17).
- Pneumothorax/haemothorax (see ➲ Traumatic pneumothorax, pp. 344).
- Flail chest (see ➲ Flail segment, p. 342).
- Drowning incident (see ➲ Drowning or near drowning, pp. 268–9).

*Other*

- Hypovolaemia or fever from any cause.
- Hyperventilation syndrome (see ➲ Hyperventilation, p. 101).
- Respiratory compensation for metabolic acidosis (diabetic ketoacidosis (DKA), salicylate overdose).

## Approach

Follow the ABC approach and resuscitate as necessary. The main aim of treatment is to correct life-threatening hypoxia. Enquire about the speed of onset of dyspnoea, past medical history, and associated symptoms (cough, haemoptysis, fever, wheezing, chest pain). Examine carefully, paying attention to the RR, depth, and pattern. Apply a pulse oximeter.

## Pulse oximetry

Simple, rapid, safe, and non-invasive, but it does *not* provide information about ventilation or arterial partial pressure of $CO_2$ ($pCO_2$). A normal $SpO_2$ does not exclude significant lung pathology (eg PE). Pulse oximetry may be inaccurate or misleading in:

- Poor peripheral perfusion/shock and hypothermia.
- Methaemoglobinaemia.
- CO poisoning (see ➲ Carbon monoxide poisoning, p. 216). $SpO_2$ values may be falsely high as COHb reads as oxyhaemoglobin. COHb can be measured on VBG testing or with a COHb pulse oximeter.
- Nail varnish/synthetic fingernails (if a finger probe is used).
- Excessive movement.

Correlate readings with clinical findings—a non-pulsatile trace (or a heart rate different from that on the cardiac monitor) suggests the saturation reading is probably inaccurate.

# Hyperventilation

Hyperventilation is breathing which occurs more deeply and/or more rapidly than normal. $CO_2$ is 'blown off', so that $pCO_2$ ↓. Hyperventilation may be primary ('psychogenic') or secondary. A classical secondary cause is DKA—Kussmaul's respiration represents respiratory compensation for metabolic acidosis.

## Secondary causes of hyperventilation

- Metabolic acidosis (eg DKA, uraemia, sepsis, hepatic failure).
- Poisoning (eg aspirin, methanol, CO, cyanide, ethylene glycol).
- Pain/hypoxia.
- Hypovolaemia.
- Respiratory disorders (eg PE, asthma, pneumothorax).

## Primary (psychogenic or inappropriate) hyperventilation

Typically, the patient is agitated and distressed, with a past history of panic attacks or episodes of hyperventilation. They may complain of dizziness, circumoral paraesthesiae, carpopedal spasm, and occasionally sharp or stabbing chest pain. Initial examination reveals tachypnoea, with equal air entry over both lung fields, and no wheeze or evidence of airway obstruction. It is important to consider secondary causes (such as PE or DKA). Therefore, perform the following investigations:

- $SpO_2$.
- ECG.
- ABG if $SpO_2$ ↓ or if symptoms do not completely settle in a few minutes.
- BMG.

If symptoms do not completely settle in a few minutes, obtain:

- CXR.
- U&E, blood glucose, FBC.

*Treatment*

Do not sedate a patient who is hyperventilating. Once serious diagnoses have been excluded, use this information to help reassure the patient with primary hyperventilation. Often this is all that is required, but it may be helpful to try simple breathing exercises (breathe in through the nose—count of 8, out through the mouth—count of 8, hold for count of 4, and repeat). Discharge the patient with arrangements for GP follow-up. If these simple measures fail, reconsider the diagnosis and refer the patient to the medical team for subsequent observation and treatment.

# Arterial blood gases

## Assessing respiratory function

Arterial sampling helps in the assessment of a patient with low $SpO_2$ or patients with known lung disease (especially if they are receiving supplemental $O_2$). Document the $FiO_2$. Look specifically for:

- Hypoxia ($pO_2$ <10.6kPa on air).
- Hypercarbia ($pCO_2$ >6.0kPa).
- Bicarbonate retention ($HCO_3^-$ >28mmol/L).
- Acidosis (pH <7.35).

### *Differentiating between type I and type II respiratory failure*

In type I failure, there is hypoxia with normal or ↓ $pCO_2$. In type II failure, there is hypoxia with ↑ $pCO_2$ and frequently ↑ $HCO_3^-$. In type II failure, the patient may develop life-threatening respiratory failure if administered high concentrations of $O_2$. Aim to maintain $SpO_2$ at 88–92% in COPD, and recheck ABGs in 30min.

### *Differentiating between acute and chronic type II respiratory failure*

Patients who normally have a slightly ↑ $pCO_2$ will also show ↑ $HCO_3^-$ on ABG. The kidneys adapt over a period of days to retain $HCO_3^-$, in an attempt to buffer the respiratory acidosis (see ➲ nomogram inside front cover). Respiratory acidosis in a patient with chronic type II respiratory failure (↑ $pCO_2$, ↑ $HCO_3^-$, and pH <7.35) indicates life-threatening impairment of lung function.

In acute respiratory failure, the lungs are unable to eliminate $CO_2$ (caused by ↓ GCS or hypoventilation from any cause), which results in ↑ $pCO_2$ and respiratory acidosis. Patients may require ventilatory support.

## Metabolic acidosis

The usual pattern of results in metabolic acidosis is pH <7.35, $HCO_3^-$ <24mmol/L, and base excess (BE) <–2mmol/L. There may be compensatory hypocarbia ($pCO_2$ <4.5kPa). Metabolic acidosis has many possible causes:

- ↑ acid load (lactic acidosis, ketoacidosis, or ingestion of salicylates, methanol, ethylene glycol, or metformin).
- ↓ removal of acid (renal failure or renal tubular acidosis types 1 and 4).
- Loss of $HCO_3^-$ from the body (diarrhoea, pancreatic or intestinal fistulae, acetazolamide, or renal tubular acidosis type 2).

## The anion gap

The anion gap is the quantity of anions not balanced out by cations (a measurement of negatively charged plasma proteins). The normal value is 12–16mmol/L. It is measured by (all measured in mmol/L):

$$\left(Na^+ + K^+\right) - \left(Cl^- + HCO_3^-\right)$$

Measuring the anion gap helps distinguish the cause of metabolic acidosis. A high anion gap indicates that there is excess $H^+$ in the body. The most common cause of a high anion gap metabolic acidosis is lactic acidosis. Most blood gas analysers measure lactate (normal <2.0mmol/L).

*Causes of lactic acidosis*

- Tissue hypoperfusion (trauma with major haemorrhage, sepsis).
- Tissue hypoxia (hypoxaemia, CO or cyanide poisoning).
- Hepatic failure.
- Renal failure.
- Ethylene glycol or methanol poisoning (see ➲ Ethylene glycol poisoning, p. 212 and ➲ Methanol poisoning, p. 211).
- Cocaine or amphetamines (see ➲ Recreational drugs, pp. 222–3).
- Salicylate poisoning (see ➲ Salicylate poisoning, p. 197) or iron poisoning (see ➲ Iron poisoning, p. 209).
- Biguanides (metformin).
- Isoniazid.
- Strenuous exercise.

The other causes of a high anion gap metabolic acidosis are *ketoacidosis* (diabetic or alcohol-induced) and renal failure.

Causes of a normal anion gap metabolic acidosis are chronic diarrhoea, pancreatic or intestinal fistulae, acetazolamide, and renal tubular acidosis.

### The osmolal gap

This is the difference between the calculated serum osmolarity and the laboratory-measured serum osmolality. Serum osmolarity can be calculated by (all measured in mmol/L):

$$\left(2 \times Na^{+}\right) + \text{urea} + \text{glucose}$$

Subtract the calculated result from the laboratory-measured osmolality to give the osmolal gap. Normally this is <10mOsm/kg.

An elevated osmolal gap can be caused by alcohol, methanol, ethylene glycol or acetone ingestion, mannitol, or sorbitol.

## Venous blood gases

Whilst not providing quite as much information as arterial samples, VBGs are incredibly useful in the initial work-up of many 'trolley' cases presenting to the ED. The availability and ease with which samples can be processed by modern analysers has meant that VBG analysis has become routine. The biggest advantage for the patient is that VBG analysis does not require a painful arterial puncture—it can be performed on a sample that was going to be taken for lab analysis, rather than done as an additional test.

A venous blood sample will give accurate readings for $K^{+}$, lactate, glucose, $HCO_3^{-}$, haemoglobin (Hb), and COHb. In addition, a normal venous $pCO_2$ will exclude hypercarbia.

Venous lactate levels are useful in helping with the early identification of patients who are sicker than they initially appear to be, particularly those with sepsis.

Use serial VBG analyses to establish the response to treatment, especially in terms of lactate and $K^{+}$.

# Cardiogenic pulmonary oedema

Left heart failure results in ↑ LV end-diastolic pressure, causing ↑ pulmonary capillary hydrostatic pressure. Fluid collects in extravascular pulmonary tissues faster than the lymphatics clear it.

## Causes of cardiogenic pulmonary oedema

Often an acute complication of MI and IHD, or an exacerbation of pre-existing cardiac disease (eg hypertension, aortic/mitral valve disease). Other causes are:

- Arrhythmias.
- Failure of prosthetic heart valve.
- Ventricular septal defect.
- Cardiomyopathy.
- Negatively inotropic drugs (eg β-blockers).
- Acute myocarditis.
- Left atrial myxoma (may cause syncope, fever, ↑ ESR))—very rare.
- Pericardial disease.

**History** Frequently dramatic. Dyspnoea and distress may prevent a full history from being taken. Find out the length of the history and whether there is any chest pain. Check current drug therapy/allergies, and establish what emergency prehospital treatment has been administered.

**Examination** Usually reveals a tachypnoeic, tachycardic, and anxious patient. If pulmonary oedema is severe, the patient may be cyanosed, coughing up frothy pink sputum and unable to talk. Check pulse and BP; auscultate the heart for murmurs and third/fourth heart sounds of gallop rhythm. Look for ↑ JVP (also a feature of PE and cardiac tamponade). Listen to the lung fields—fine inspiratory crepitations (crackles) may be limited to the bases or be widespread. Wheeze may be more prominent than crepitations. Cardiogenic pulmonary oedema is associated with evidence of ↓ cardiac output (sweaty, peripherally cool, and pale). Consider other diagnoses (eg sepsis) in patients with warm, flushed extremities.

## Investigations

Commence treatment before completing investigations:

- Attach a cardiac monitor and check $SpO_2$ with a pulse oximeter.
- Obtain an ECG. Check for arrhythmias, LAD, LVH, LBBB, and recent or evolving MI.
- Send blood for U&E, glucose, FBC, troponin, and B-type natriuretic peptide (BNP).
- If severely ill or $SpO_2$ <90% on air, obtain an ABG.
- Obtain a CXR and look for features of cardiogenic pulmonary oedema:
  - Upper lobe diversion (distension of upper pulmonary veins).
  - Cardiomegaly (LV and/or left atrial dilatation).
  - Kerley A, B, or C septal lines (see Fig. 3.24).
  - Fluid in interlobar fissures.
  - Peribronchial/perivascular cuffing and micronodules.
  - Pleural effusions.
  - Bat's wing hilar shadows.
- Request old hospital notes/ECGs. In newly diagnosed heart failure, an urgent transthoracic echo will identify the presence or absence of cardiac abnormalities.

## Treatment

- Check that the airway is clear.
- Raise the trolley to sit the patient up (support with pillows, if needed).
- Provide high-flow $O_2$, as required, by a tight-fitting face mask.
- Give furosemide IV 40mg. Note that larger doses may be needed in patients already taking oral furosemide.
- Nitrates and opioids are no longer recommended routinely (https://www.nice.org.uk). Reserve the use of IV nitrates for specific circumstances (eg concomitant myocardial ischaemia, severe hypertension, or regurgitant aortic or mitral valve disease), starting IVI slowly (eg GTN IVI, starting at 10mcg/min), ↑ every few minutes according to clinical response; monitor BP closely—take special care to avoid hypotension. If the patient has chest pain, consider giving very small titrated increments of IV opioid (with antiemetic). Do not give opioids to patients who are drowsy, confused, or exhausted, as this may precipitate respiratory arrest.
- Consider NIV (continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP)) if the patient is very breathless with acidaemia and there is no immediate improvement.
- Monitor urine output (inserting a urinary catheter if necessary).
- Treat the underlying cause and associated problems (arrhythmias, MI, cardiogenic shock, acute prosthetic valve failure).

Monitor the $SpO_2$ and clinical response to initial treatment. Rapid improvement may occur, due to venodilatation and reduction of preload. If the patient does not improve, recheck the ABG and consider the following:

- If hypotensive, refer to ICU for treatment of cardiogenic shock (see ➲ STEMI treatment, pp. 80–1). An intra-arterial line, a Swan–Ganz catheter, and inotropic support (dobutamine) are likely to be required.
- Echocardiography may help to exclude valve or septal rupture and guide treatment.
- Rapid sequence intubation (RSI) in the presence of cardiogenic pulmonary oedema may be associated with cardiovascular collapse. Stop nitrates prior to administering anaesthesia and be ready to give pressors ± fluids immediately post-induction.

## Prosthetic valve failure

Always consider valve failure in patients with prosthetic valves—a large variety are in common use. All are associated with some risks (eg embolism, failure, obstruction, infection, haemorrhage from associated anticoagulation), which vary according to the design. Acute failure of a prosthetic aortic or mitral valve results in dramatic acute-onset pulmonary oedema with loud murmurs. The patient may deteriorate rapidly and not respond to standard drug treatment. Resuscitate as described earlier. A CXR will show a prosthetic heart valve ± pulmonary oedema. Call urgently for expert help (ICU team, cardiologist, and cardiothoracic surgeon). Emergency transthoracic or transoesophageal echocardiography will confirm the diagnosis. Immediate valve replacement is required.

# Non-cardiogenic pulmonary oedema

Pulmonary oedema may occur in the absence of ↑ pulmonary venous pressure. The following mechanisms may be responsible:

- ↑ capillary permeability.
- ↓ plasma oncotic pressure.
- ↑ lymphatic pressure.

Changes in capillary permeability, secondary to a variety of triggers, is the mechanism most frequently implicated in non-cardiogenic pulmonary oedema, when it occurs as adult respiratory distress syndrome (ARDS). Since the mechanisms producing cardiogenic and non-cardiogenic pulmonary oedema differ, so does the approach to treatment.

## Causes of non-cardiogenic pulmonary oedema

- ARDS (sequel to sepsis, trauma, pancreatitis, COVID-19).
- Intracranial (especially subarachnoid) haemorrhage.
- IV fluid overload.
- Hypoalbuminaemia (liver failure, nephrotic syndrome).
- Drugs/poisons/chemical inhalation.
- Lymphangitis carcinomatosis.
- Smoke inhalation.
- Near drowning incidents.
- High altitude mountain sickness.

## Approach

Distinguishing non-cardiogenic from cardiogenic pulmonary oedema is usually apparent from the history. Evaluate the patient and resuscitate according to ABCs. Direct treatment towards the underlying cause and according to the physiological disturbance. Use NIV early and consider urinary, intra-arterial, and central venous lines. Involve ICU early and provide appropriate IV fluids and inotropes—deterioration may require intubation, whilst being mindful of the risk of hypotension afterwards.

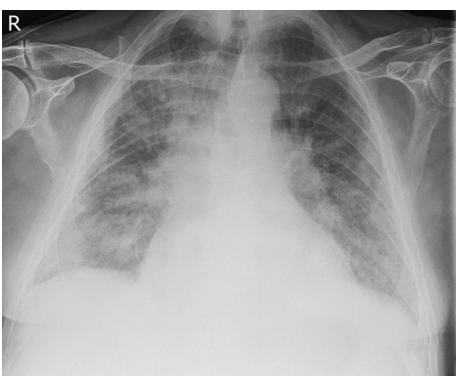


**Fig. 3.24** CXR showing pulmonary oedema.

# Pleural effusion

Under normal circumstances, each pleural cavity contains <20mL of fluid.

An exudate is diagnosed if the pleural fluid:serum protein is >0.5, fluid:serum lactate dehydrogenase (LDH) >0.6, or fluid LDH more than two-thirds the upper limits of the laboratory normal value for serum LDH.

**Table 3.6** Causes of pleural effusion

| Exudates | Transudates |
|---|---|
| Pneumonia | Cardiac failure |
| Malignancy | Nephrotic syndrome |
| TB | Hepatic failure |
| PE with pulmonary infarction | Ovarian hyperstimulation |
| Collagen vascular disease | Peritoneal dialysis |
| Abscess (subphrenic and amoebic liver) | Ovarian fibroma (Meig's syndrome) |
| Pancreatitis | |
| Chylothorax (thoracic duct injury) | |

## Clinical presentation

*Symptoms* are usually due to the underlying disease process. A mild dull ache and dyspnoea (initially on exercise, later at rest) may occur if the effusion is large. A history of vomiting, followed by chest pain, points to a ruptured oesophagus—a surgical emergency.

*Signs* are not apparent until >500mL are present. Dyspnoea and stony dullness to percussion, with absent breath sounds over the effusion, are characteristic. Bronchial breathing may be heard just above the effusion. Very large unilateral effusions may produce evidence of mediastinal shift.

## Investigation

*CXR* can demonstrate pleural effusions as small as 250mL as blunting of the costophrenic angle (see Fig. 3.25). Other investigations depend on the likely cause. For causes of pleural effusion, see Table 3.6.

## Treatment

Provide $O_2$ and resuscitate as necessary, according to the underlying pathology. Emergency therapeutic pleural aspiration is rarely required in the ED, except where haemothorax is suspected. Refer to the medical team for further investigation (including USS-guided diagnostic pleural aspiration).

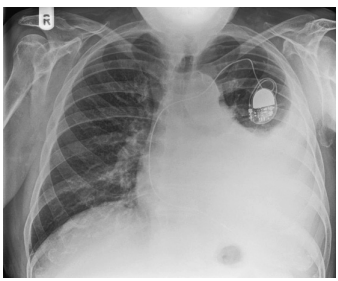


**Fig. 3.25** CXR showing left pleural effusion and pacemaker.

# Acute asthma: assessment

Follow the British Thoracic Society (BTS)/Scottish Intercollegiate Guidelines Network (SIGN) guidelines (https://www.brit-thoracic.org.uk) to assess and manage adults presenting with asthma (see Acute asthma: management, pp. 110–11). The guidelines reflect continuing concern over asthma deaths. Patients with severe asthma and one or more adverse psychosocial factors (psychiatric illness, alcohol or drug abuse, unemployment) have ↑ mortality. Measure the peak expiratory flow rate (PEFR) and compare it against that expected (see Fig. 3.26). The peak flow acts as an immediate triage tool—remember that patients with life-threatening asthma may be too dyspnoeic to do this.

Make an initial assessment of the severity of acute asthma based upon a combination of clinical features, peak flow measurement, and pulse oximetry, as outlined below.

### Moderate exacerbation of asthma

- ↑ symptoms.
- Peak flow 50–75% best or predicted.
- No features of acute severe asthma (see below).

### Acute severe asthma

Any one of:

- Peak flow 33–50% best or predicted.
- RR ≥25/min.
- Heart rate ≥110/min.
- Inability to complete sentences in one breath.

### Life-threatening asthma

A patient with severe asthma with any one of:

- Peak flow <33% best or predicted.
- $SpO_2$ <92%.
- $pO_2$ <8kPa.
- Normal $pCO_2$ (4.6–6.0kPa).
- Silent chest.
- Cyanosis.
- Poor respiratory effort.
- Arrhythmia.
- Exhaustion.
- Altered conscious level.
- Hypotension.

### Near-fatal asthma

- ↑ $pCO_2$ and/or requiring mechanical ventilation with ↑ inflation pressures.

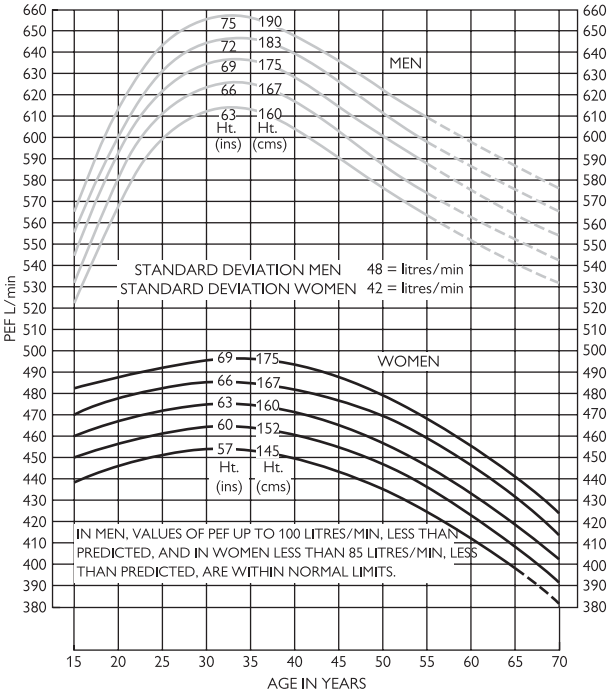


**Fig. 3.26** Peak expiratory flow rates in normal adults.

Source: data from Nunn AJ, Gregg I. (1989). New regression equations for predicting peak expiratory flow in adults. *Br Med J* 298: 1068–70.

## Investigations in asthma

*Peak flow* is most useful when expressed as a percentage of that patient's previous best, but the percentage of predicted is a rough guide.

*Pulse oximetry ($SpO_2$)* determines the adequacy of $O_2$ therapy and the need for ABG measurement. Use $O_2$ to aim for $SpO_2$ of 94–98%.

Obtain *ABG* if $SpO_2$ <92% or if there are other features of life-threatening asthma.

Obtain a *CXR* (without delaying treatment) if there is:

- Suspected pneumomediastinum or pneumothorax.
- Suspected consolidation.
- Life-threatening asthma.
- Failure to respond to treatment satisfactorily.
- Requirement for ventilation.

# Acute asthma: management

## Initial treatment

Follow BTS/SIGN guidelines (https://www.brit-thoracic.org.uk), summarized as follows:

- Provide high-flow $O_2$.
- Put the trolley back and side rails up, so the patient is sitting up and holding on to the side rails (to use the pectoral muscles as accessory muscles of respiration).
- If the patient cannot talk, start treatment, but get senior ED and ICU help in case intubation and ventilation are required.
- Check the trachea and chest signs for pneumothorax.
- Ask about previous admissions to ICU.
- Administer high-dose ($O_2$-driven) nebulized $\beta_2$-agonist (eg salbutamol 5mg or terbutaline 10mg), or ten puffs of salbutamol into a spacer device and face mask. For severe asthma or asthma that responds poorly to the initial nebulizer, consider continuous nebulization. Reserve the use of IV salbutamol for those patients in whom inhaled therapy cannot be used reliably (in which case, draw up salbutamol 5mg into 500mL of 5% glucose and run at a rate of 30–60mL/hr).
- Give a corticosteroid to all patients with acute asthma—either prednisolone 40–50mg PO or hydrocortisone (preferably as sodium succinate) 100mg IV.
- Add nebulized ipratropium bromide (500mcg 4- to 6-hourly) to $\beta_2$-agonist treatment for patients with acute severe or life-threatening asthma or those with a poor initial response to $\beta_2$-agonist therapy.
- Consider a single dose of IV magnesium sulfate (1.2–2g IVI over 20min), after consultation with senior medical staff, for patients with acute severe asthma without a good initial response to inhaled bronchodilator therapy or for those with life-threatening or near-fatal asthma.
- The use of IV aminophylline remains controversial and is not likely to result in any additional bronchodilation, compared to standard care. Use IV aminophylline only after consultation with senior medical staff. Some individual patients with near-fatal or life-threatening asthma with a poor response to initial therapy may gain additional benefit. The loading dose of IVI aminophylline is 5mg/kg over 20min, unless on maintenance therapy, in which case check blood theophylline level and start IVI of aminophylline at 0.5–0.7mg/kg/hr.
- A patient who cannot talk will be unable to drink fluids and may be dehydrated.
- Avoid 'routine' antibiotics.
- Repeat ABG within an hour if: initial $pO_2$ is <8kPa (unless subsequent $SpO_2$ is >92%), or $pCO_2$ is normal or ↑, or if the patient deteriorates.
- Hypokalaemia may be caused or exacerbated by $\beta_2$-agonist and/or steroid therapy. Correct electrolyte abnormalities.

## Criteria for admission

Admit patients with any features of:

- A life-threatening or near-fatal attack.
- Severe attack persisting after initial treatment.

## Management of discharge

Consider for discharge those patients whose peak flow is >75% best or predicted 1hr after initial treatment. Prescribe a short course of oral prednisolone (eg 40–50mg for 5 days) if initial PEFR is <50%, and ensure an adequate supply of inhalers. If possible, arrange for review by an asthma liaison nurse before discharge. At a minimum, the inhaler technique and peak expiratory flow monitoring should be reviewed. Arrange/advise GP/asthma liaison nurse follow-up within 2 days. Email/send the discharge summary to the GP. Advise to return to hospital if symptoms worsen/recur.

## Referral to intensive care unit

Refer any patient requiring ventilatory support or with acute severe or life-threatening asthma failing to respond to therapy, as evidenced by:

- Deteriorating peak flow.
- Persisting or worsening hypoxia.
- Hypercapnia.
- ABG showing ↓ pH.
- Exhaustion, feeble respiration.
- Drowsiness, confusion, altered conscious state, or respiratory arrest.

*Post-intubation care*

Mechanical ventilation is associated with a high risk of barotrauma and dynamic hyperinflation. Breath stacking can result in critically reduced venous return and hypotension. High inspiratory presssures and volumes can cause pneumothorax. Typically, set the ventilator to provide low tidal volumes (6–8mL/kg) at a low RR (7–8/min) and inspiratory:expiratory ratio of 1:4.

## Cardiac arrest in acute asthma

The underlying rhythm is usually PEA. This may reflect one or more of the following: prolonged severe hypoxia (secondary to severe bronchospasm and mucus plugging), hypoxia-related arrhythmias, or tension pneumothorax (may be bilateral). Give ALS according to the guidelines in ➲ Cardiac arrest, p. 48, and treat tension pneumothorax if present (see ➲ Tension pneumothorax, pp. 338–9). Aim to achieve tracheal intubation early in view of the higher than normal required lung inflation pressures and the attendant risk of gastric inflation in the absence of a tracheal tube.

# Chronic obstructive pulmonary disease

COPD is characterized by chronic airflow limitation due to impedance to expiratory airflow, mucosal oedema, infection, bronchospasm, and bronchoconstriction due to ↓ lung elasticity. Smoking is the main cause, but other causes are chronic asthma, α-1 antitrypsin deficiency, and chronic infection (eg bronchiectasis).

## History

Exertional dyspnoea, cough, and sputum are usual complaints. Ask about:

- *Present treatment*: including inhalers, steroids, antibiotics, theophyllines, nebulizers, opiate analgesia, and home $O_2$ treatment.
- *Past history*: enquire about previous admissions and comorbidity.
- *Exercise tolerance*: how far can they walk on the flat without stopping? How many stairs can they climb? Do they get out of the house?
- *Recent history*: ask about wheeze and dyspnoea, and sputum volume and colour. Chest injuries, abdominal problems, and other infections may cause respiratory decompensation.
- *Read the hospital notes*: have there been prior ICU assessments? Has the respiratory consultant advised whether ICU would be appropriate?

## Examination

Examine for dyspnoea, tachypnoea, accessory muscle use, and lip-pursing. Look for hyperinflation ('barrel chest'), and listen for wheeze or coarse crackles (large airway secretions). Cyanosis, plethora (due to secondary polycythaemia), and right heart failure (cor pulmonale) suggest advanced disease. Look for evidence of hypercarbia: tremor, bounding pulses, peripheral vasodilatation, drowsiness, or confusion.

Check for evidence of other causes of acute dyspnoea, particularly: asthma (see ➲ Acute asthma: assessment, pp. 108–9), pulmonary oedema (see ➲ Cardiogenic pulmonary oedema, pp. 104–5), pneumothorax (see ➲ Spontaneous pneumothorax, pp. 118–20), and PE (see ➲ Pulmonary embolism, pp. 124–5). Remember that these conditions may coexist with COPD.

## Investigation

- $SpO_2$, RR, pulse rate, BP, T°, and peak flow (if possible).
- CXR (look for pneumothorax, hyperinflation, bullae, and pneumonia).
- ECG.
- ABG (or VBG), documenting the $FiO_2$. Use $pCO_2$ to guide $O_2$ therapy.
- FBC, U&E, glucose, theophylline levels, and, if pneumonia is suspected and/or pyrexial, blood cultures, CRP, and pneumococcal antigen.
- Send sputum for microscopy and culture if purulent.

## Treatment

*Give oxygen* Remember that hypercapnia with $O_2$ is multifactorial. The aim is to maintain $SpO_2$ of 88–92% without precipitating respiratory acidosis or worsening hypercapnia (see ➲ Oxygen, p. 99 and ➲ Arterial blood gases, pp. 102–3). If the patient is known to have COPD and is drowsy or has a documented history of previous hypercapnic respiratory failure, give an $FiO_2$ of 28% via a Venturi mask and obtain an ABG. Titrate up the $FiO_2$ with serial ABG sampling until the minimum $FiO_2$ that achieves $SpO_2$ of 88–92%. Reduce inhaled $O_2$ concentration if $SpO_2$ is >92%.

*Give bronchodilators and steroids*

- Give nebulized salbutamol 5mg or terbutaline 5–10mg.
- Consider adding nebulized ipratropium 0.5mg.
- Use $O_2$-driven nebulizers unless the patient has hypercapnic, acidotic COPD, in which case use nebulizers driven by compressed air, supplemented by $O_2$ via nasal prongs at 1–4L/min.
- Give steroids (eg prednisolone 30mg PO stat, then continued once daily for 7 days). Use hydrocortisone 100mg IV if the patient cannot take prednisolone PO.

*Other drug treatments*

- Give antibiotics (eg amoxicillin, doxycycline, or clarithromycin) if the patient reports ↑ purulent sputum or there is clinical evidence of pneumonia and/or consolidation on CXR.
- Only consider IV aminophylline if there is an inadequate response to nebulized bronchodilators. Beware interactions with other drugs and the potential for toxicity if the patient is already taking oral theophylline.
- Consider naloxone if the patient is taking an opioid analgesic that may cause respiratory depression.

(See NICE guideline on COPD, 2018 at https://www.nice.org.uk)

## Non-invasive ventilation

NIV is standard early therapy for hypercapnic ventilatory failure during exacerbations of COPD. NIV improves blood gas measurements in the ED and ↓ intubation rates, mortality, and length of hospital stay. Ensure patients started on NIV have a plan in the event of deterioration (agreed ceiling of care).

NIV takes two forms—CPAP and BiPAP (which may be more suitable for treating type II respiratory failure in COPD). Both CPAP and BiPAP have been used to treat acute cardiogenic pulmonary oedema. Patients with sleep apnoea use CPAP at night. The positive airway pressure is delivered by a tightly adhered face mask, which is sized to fit the patient. The patient is awake and must be compliant with wearing the mask.

Unlike tracheal intubation, NIV does not protect the airway, so coma and vomiting are contraindications. Absolute contraindications include apnoea and cardiac arrest. Check CXR before starting—a pneumothorax will be converted into a tension pneumothorax with NIV. Severe agitation may make effective NIV impossible.

The patient should always be cared for by staff who are familiar with the ventilator and mask, in the resuscitation room.

Start BiPAP at 10cm$H_2O$ inspiratory positive airway pressure (IPAP)/ 5cm$H_2O$ expiratory positive airway pressure (EPAP), and titrate upwards:

- To treat persistent hypercapnia, ↑ IPAP by 2cm$H_2O$ at a time.
- To treat persistent hypoxia, ↑ IPAP and EPAP by 2cm$H_2O$ at a time.
- The maximum IPAP/EPAP is 25/15cm$H_2O$.
- For CPAP, commence treatment at 5–8cm$H_2O$.

# Pneumonia

Pneumonia involves symptoms and signs of lower respiratory tract infection (breathlessness, productive cough, and fever) usually associated with CXR abnormalities. *Pneumocystis* pneumonia may occur with minimal or no CXR changes. Consider pneumonia in patients with septicaemia or acute confusional states.

## Causes

*Bacterial (80–90%)* *Streptococcus pneumoniae* is the most common cause of community-acquired pneumonia. Others include *Mycoplasma pneumoniae, Haemophilus influenzae, Legionella, Chlamydia psittaci*, and *Staphylococcus aureus* (can cause fulminant pneumonia in patients with influenza). Gram –ve and anaerobic infections are rare. Always consider TB, particularly in chronic alcoholism, poor social circumstances, immigrants and those travelling to developing countries, or individuals not BCG-vaccinated. Immunosuppressed patients (eg HIV, steroid therapy) are at ↑ risk of TB and *Pneumocystis jirovecii* pneumonia.

*Viral (10–20%)* Predominantly COVID-19, influenza A and B, RSV, rarely varicella and SARS.

*Rickettsial (1%)* Rarely *Coxiella burnetii*.

## Signs and symptoms

Fever, cough, and production of sputum are common complaints. Breathlessness, pleuritic chest pain, myalgia, rigors, or haemoptysis may occur. Pneumonia can present without obvious chest signs. *Mycoplasma* pneumonia may present in children and young adults with sore throat, headache, nausea, abdominal pain, and diarrhoea. *Legionella* can present with constitutional upset, diarrhoea, or confusion, particularly in the elderly. *Pneumocystis* pneumonia in immunosuppressed patients may present with cough, dyspnoea, and marked hypoxia, with relatively few other findings.

## Examination and investigation

- If there is suspicion of COVID-19 infection, place the patient in isolation, restrict staff interaction, and ensure staff don PPE prior to entering the room.
- Assess for signs of severe sepsis.
- Check RR, pulse, and BP.
- Auscultation may reveal a patch of inspiratory crackles; signs of consolidation are present in <25%.
- Check BMG and $SpO_2$ (obtain ABG if <90% or known to have COPD).
- Take blood for U&E, FBC, and CRP, and blood cultures before giving IV antibiotics.
- Obtain a CXR. Look for patchy or lobar opacification (see Fig 3.27), mass lesions, or an air bronchogram. In early pneumonia, the CXR may be normal.
- Obtain blood cultures and sputum cultures, and consider urinary pneumococcal and *Legionella* antigen testing. If suspicion of COVID-19 infection, take a nasopharyngeal swab for viral testing.

*Assessment: admit or discharge*

Some patients with 'mild' illness, good social circumstances, and no significant comorbidity may be safely discharged with appropriate antibiotics (eg amoxicillin 0.5–1g PO tds), simple analgesia for pleuritic pain to aid deep breathing/coughing, and GP follow-up.

Patients with a CURB-65 score of ≥3 (see Table 3.7) have severe pneumonia with a high risk of death; those who score 2 are at ↑ risk of death and should be considered for inpatient treatment or hospital-supervised outpatient care; patients with a CURB-65 score of 0 or 1 are at low risk of death and may be suitable for home treatment (℞ https://www.brit-thoracic.org.uk).

**Table 3.7** CURB-65 score for pneumonia

| | Score |
|---|---|
| Confusion | 1 |
| Urea >7mmol/L | 1 |
| RR ≥30/min | 1 |
| Low BP (systolic <90mmHg or diastolic ≤60mmHg) | 1 |
| Age ≥65y | 1 |

Reproduced from Lim WS *et al*., *Thorax* 2003;58:377–82, Copyright © 2003 *Thorax*, with permission from BMJ Publishing Group Ltd.

## Treatment

(See NICE guideline 2014 at ℞ https://www.nice.org.uk)

*Patients deemed suitable for discharge*

Give analgesia, oral antibiotics for 5 days, and GP follow-up (including a decision about the need to repeat CXR in 6 weeks to confirm changes have cleared). Explain to the patient what rate of recovery to expect.

*Patients admitted, but not severely unwell*

Start either PO or IV antibiotics, as follows:

- *Either* amoxicillin 0.5–1g PO tds + erythromycin 500mg PO qds (or clarithromycin 500mg bd).
- *Or* if IV therapy is needed: ampicillin 500mg IV qds + erythromycin 500mg IV qds (or clarithromycin 500mg bd). Local guidelines will apply.
- *Monitor* $SpO_2$ and provide $O_2$ accordingly.
- *Provide* simple analgesia.

*Patients with sepsis*

(See ➔ Sepsis, pp. 62–3.)

Commence IV crystalloid fluids; take blood cultures and administer IV antibiotics (eg co-amoxiclav 1.2g IV tds + clarithromycin 500mg IV bd) immediately. Contact ICU and insert a urinary catheter. Aim for mean arterial pressure (MAP) of >65mmHg and urine output of >0.5mg/kg/hr. (See sepsis guidelines at ℞ http://www.survivingsepsis.org)

## Differential diagnosis

Pneumonia-like presentations can occur with pulmonary oedema, pulmonary infarction, pulmonary vasculitis (eg SLE, PAN, Churg–Strauss syndrome, and granulomatosis with polyangiitis), aspergillosis, allergic alveolitis, bronchial or alveolar cell carcinoma, acute pancreatitis, and subphrenic abscess.

# Pulmonary aspiration

Aspiration of solid or liquid material into the upper and lower airways is likely when one or more of the following features are present:

- ↓ GCS: head injury, stroke, overdose, seizures, sedation, anaesthesia.
- ↓ cough and/or gag reflexes: related to above factors and/or bulbar dysfunction, intubation/extubation, Guillain–Barré syndrome, multiple sclerosis (MS), myasthenia gravis.
- Tendency to regurgitate/vomit: alcohol, full stomach, upper GI tract pathology (including hiatus hernia, oesophageal obstruction, pregnancy).
- May occur in infirm or elderly fed via NG tube.

## Clinical features

*Large food particles* sufficient to cause complete airway obstruction cause choking, inability to speak, ↑ respiratory effort, cyanosis, loss of consciousness, and death. Smaller particles may pass through the vocal cords, causing coughing, stridor, tachypnoea, and wheeze. 80% of patients are aged <4y, with peanuts being the classic inhaled objects. Delayed presentation with cough, wheeze, haemoptysis, unresolved pneumonia, abscess formation, or empyema occurs in ~30%, often days or weeks later.

*Vomiting/regurgitation* is often witnessed, and pulmonary aspiration confirmed by seeing gastric contents in the oropharynx or trachea during intubation or following suction. Gastric content is a mixture of semi-solid and liquid material—aspiration leads to a sudden onset of severe dypsnoea, wheeze, and cyanosis. Its acid nature causes severe damage to the alveolar–capillary membrane, with denaturation of pulmonary surfactant and ↑ pulmonary permeability, with oedema and atelectasis.

*Hydrocarbons* (eg petrol, paraffin) cause severe pulmonary toxicity if aspiration occurs during ingestion or following regurgitation/vomiting.

## Investigation

### *ABG*

These show hypoxaemia within minutes of acid aspiration. Initially, patients may hyperventilate, with ↓ $pCO_2$, until pulmonary compliance ↑ work of breathing, sufficient to result in hypoventilation.

### *CXR*

Abnormalities develop in >90% of patients but may take hours/days. Appearances depend on the nature of the aspirated material and the patient's position at the time of the episode (the right lower lobe is most frequently and severely affected, followed by the left lower lobe and the right middle lobe). In severe aspiration, diffuse bilateral infiltrates and pulmonary oedema similar to ARDS appearances are present. Less severe episodes produce atelectasis, followed by alveolar infiltration.

### *Intrapulmonary foreign body (including peanuts)*

Rarely radio-opaque. Resulting collapse, hyperinflation, or consolidation are usually obvious and depend on whether obstruction is complete or partial and if supervening infection is present. If the history strongly suggests an inhaled FB, but CXR is normal, consider bronchoscopy or CT.

### Prevention

Prevention is everything. Pay meticulous attention to airway protection. This may involve positioning (tilt head down on the right-hand side), suction to the oropharynx (Yankauer catheter avoiding stimulation of the gag reflex), and, if necessary, tracheal intubation. Tracheal intubation does not completely protect against aspiration of fluid into the lungs, but it is the best preventative measure. In at-risk patients, pass an NG tube to empty the stomach. However, NG tubes can also predispose to aspiration by preventing closure of the oesophageal sphincters and interfering with coughing and clearing the pharynx.

### Treatment

Correct hypoxia and give nebulized salbutamol for associated bronchospasm. If particulate aspiration is present, refer for urgent bronchoscopy. Although secondary infection is common, the use of antibiotics or steroids is not routinely indicated.

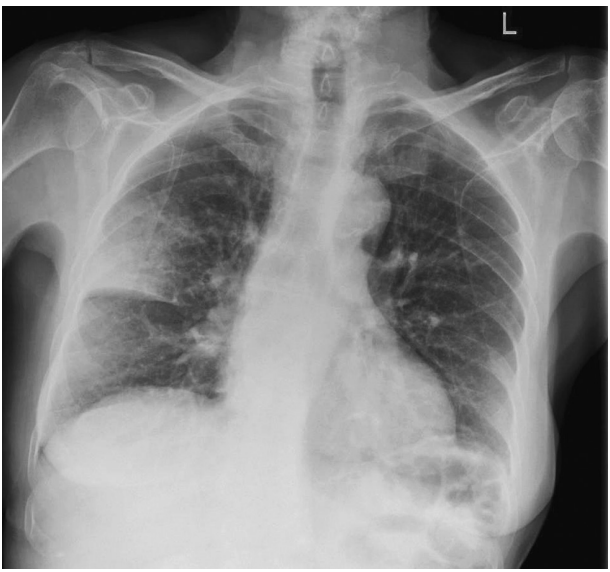


**Fig. 3.27** CXR showing right upper lobe pneumonia.

# Spontaneous pneumothorax

Primary spontaneous pneumothorax (PSP) may occur in previously healthy individuals. Secondary spontaneous pneumothorax (SSP) occurs in older patients with pre-existing chronic lung disease (like COPD or TB) and may also occur with asthma, bronchial carcinoma, Marfan's syndrome, infection, cystic fibrosis, and oesophageal rupture.

## Presentation

Most patients present with unilateral pleuritic chest pain and dyspnoea. Classical physical signs may not be present (depending upon the size of the pneumothorax): tachypnoea, tachycardia, normal/hyper-resonant percussion note with ↓ air entry on the affected side. Rarely, there may be a clicking sound at the cardiac apex.

*Severe symptoms* (inability to speak, gasping, low $SpO_2$) should prompt rapid assessment for tension pneumothorax: tracheal deviation, tachypnoea, tachycardia, and hypotension. Treat tension pneumothorax with immediate decompression using a needle in the second intercostal space (just above the third rib) in the mid-clavicular line (see ➲ Tension pneumothorax, pp. 338–9). Severe symptoms are also found in patients with SSP (disproportionate to the pneumothorax size). In the absence of signs of tension pneumothorax, obtain an emergency portable CXR and involve an experienced doctor.

## Initial assessment and management

- Monitor pulse, $SpO_2$, and BP. Ensure IV access.
- Administer high-flow $O_2$ (in patients with COPD, aim for an $SpO_2$ of 90–92%).
- When there are no signs of tension, an ABG will help assess patients with chronic lung disease and guide $O_2$ therapy.
- *Erect CXR* is the principal way of making the diagnosis (see Fig. 3.28), but beware pitfalls (see ➲ Pitfalls in CXR analysis for possible pneumothorax, p. 120).
- *CT scan* is not the primary diagnostic modality but can identify small pneumothoraces not apparent on the CXR. CT is also of use in the subacute setting for assessing bullous lung disease in a stable patient.

## Intervention

- Follow the algorithm shown in Fig. 3.29.
- Be guided primarily by the patient's symptoms. If the patient is breathless, they should undergo an intervention.
- The size of the pneumothorax can be estimated on CXR by measuring from the chest wall to the lung edge at the level of the hilum. This is only an estimate and assumes symmetrical lung collapse. The cut-off of 2cm is used to determine treatment.
- Intervention for PSP is needle aspiration. If unsuccessful, do not repeat aspiration. Instead insert a Seldinger chest drain.
- Treatment for symptomatic SSP is chest drain insertion and admission.
- Treatment for SSP without breathlessness is admission. Aspiration should be performed by an experienced doctor and may require CT.
- Adopt a very low threshold for inserting chest drains for bilateral pneumothoraces.

- Always insert a chest drain immediately following emergency needle decompression.
- Pleural aspiration and drain insertion should be performed by a doctor who has prior training and experience.
- Ensure the patient has IV access. Perform in a monitored environment with an assistant and appropriate supervision. Use aseptic technique.
- Always discuss the procedure with the patient, and document that they have given their consent.
- If the patient is on anticoagulation or has a known coagulopathy disorder, discuss with a haemotologist first.

### Aspiration technique

Confirm the side of the pneumothorax. Sit the patient upright. Infiltrate 1% lidocaine, then insert a 16G IV cannula just above the third rib (in the second intercostal space) in the mid-clavicular line. Alternatively, lay the patient on their side, with the pneumothorax side upwards. Insert a cannula in the fifth intercostal space in the anterior axillary line. Remove the needle; attach a three-way tap, then aspirate air with a 50mL syringe. Continue aspiration until the patient coughs excessively or until 2.5L of air is removed.

### Seldinger chest drain insertion

Confirm the side of the pneumothorax. Keep the patient comfortable; ensure adequate analgesia (this may require 1mg increments of morphine IV), but avoid sedation. Sit the patient upright, and rest their hand behind their head. Infiltrate 10mL of 1% lidocaine at the anterior axillary line in the fifth intercostal space. Aspirate a small amount of pleural air during infiltration and note the depth of the pleural space. Locate the pleural space with the introducer needle (aspirate whilst advancing through the chest wall), then advance the guidewire through the needle. Remove the introducer needle; make a small skin incision, and gently pass the dilator over the guidewire using a twisting action. Do not push the dilator >1cm past the depth of the pleural space. Pass the chest drain over the guidewire to a depth of 10–12cm. Remove the guidewire; connect to an underwater seal drain and suture in place. Check the drain is bubbling and swinging, and organize a CXR.

### Discharge

Patients without breathlessness with a small PSP may be considered for discharge. Give the patient verbal and written instructions to return if their symptoms worsen and warn them not to fly or go diving. Ensure they have a plan for follow-up (and repeat CXR) with a respiratory physician or GP in 2–4 weeks (see https://www.brit-thoracic.org.uk).

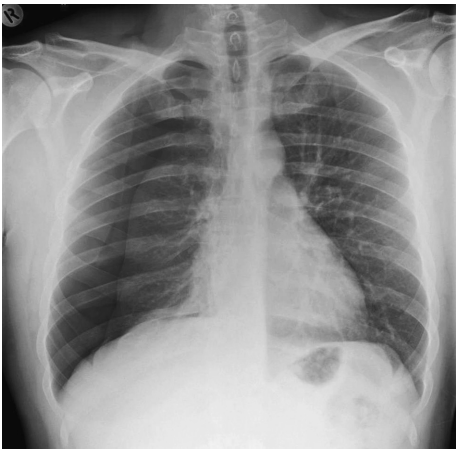

**Fig. 3.28** CXR showing spontaneous right pneumothorax.

### Pitfalls in CXR analysis for possible pneumothorax

- When using digital images, always use a picture archiving and communication system (PACS) workstation. The signs of pneumothorax may be subtle and difficult to spot. Compare with previous CXRs, if available.
- Look for a displacement of the pleural line.
- Do not mistake the scapular edge for the lung edge. Similarly, clothing, $O_2$ tubing, and overlying sheets can cause artefacts which mimic the edge of the lung.
- Some patients with COPD have emphysematous bullae, which can mimic pneumothorax. If in doubt, ask for senior review prior to treating for pneumothorax.
- An air–fluid level at the costophrenic angle may be present.

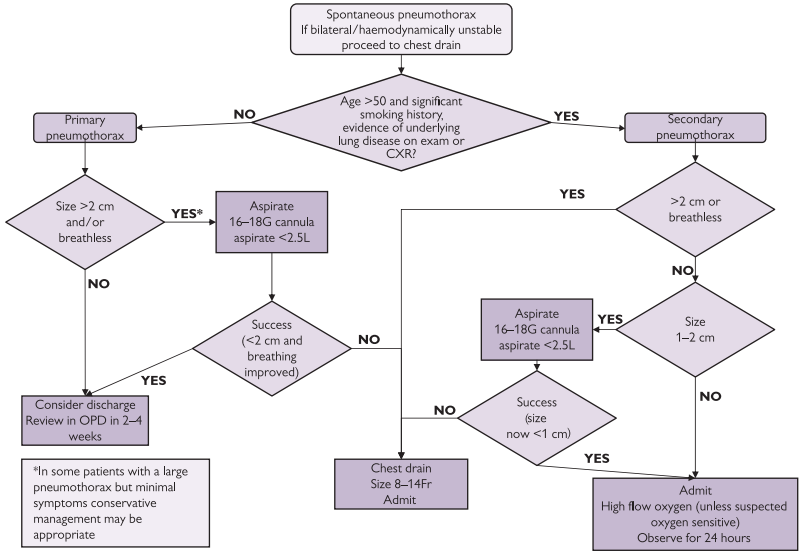


**Fig. 3.29** Management of pneumothorax.

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# Deep vein thrombosis

DVT and PE are manifestations of the same disease process whereby abnormal clotting occurs in the veins of the legs or pelvis. The clots may break from the vein wall and embolize to the lungs. Untreated DVTs are associated with 1% mortality from PE. Around half of those with DVT will go on to develop post-thrombotic syndrome, with lifelong pain and swelling of the leg.

## Risk factors

- Recent surgery (where a general anaesthetic was administered, especially orthopaedic, abdominal, spinal, and obstetric).
- Recent admission to hospital.
- Current malignancy.
- Being bedbound.
- Sepsis.
- IV drug use (where the patient injects in the femoral vein).
- Pregnancy/pelvic masses.
- Limb immobility such as recent fracture with crutches and plaster cast.
- Previous DVT/PE.
- Thrombophilia or family history of venous thromboembolism.

## Clinical features

DVT classically produces leg pain with swelling, warmth, tenderness, and dilated superficial veins in the affected leg. These signs are non-specific and often not present. A small or partially occluding thrombus may be asymptomatic. History and clinical examination alone cannot safely exclude DVT—if a DVT is suspected, investigate further. Investigate for PE instead if the patient has tachycardia, hypoxia, ↑ RR, or breathlessness (see ➲ Pulmonary embolism, pp. 124–5).

## Differential diagnosis

- *Muscular tear*: typically acute onset.
- *Rupture of a Baker's cyst*: again, typically acute onset.
- Cellulitis or other infection.

## Investigation and management

- Record pulse rate, RR, BP, $SpO_2$, and T° in all patients.
- Take a full history, including concurrent illness, past history, recent operations, travel, and family history.
- Examine the affected leg for signs of plethora, deep vein tenderness, swelling (measure both legs, 10cm distal to the tibial tuberosity), oedema, and dilatation of the skin veins.
- Perform a full examination, checking for signs of PE or occult carcinoma.
- Calculate the clinical probability assessment score. The Wells score (see Table 3.8) is the most widely used clinical prediction score.
- Take FBC, U&E, CRP, and glucose.
- Take D-dimer if the Wells score indicates DVT is 'unlikely' (<2 points).
- If D-dimer normal *and* DVT 'unlikely', DVT has been ruled out.

**Table 3.8** Wells clinical probability assessment score for DVT

| Clinical feature | Score |
|---|---|
| Active cancer (treatment ongoing or within 6 months or palliative) | 1 |
| Paralysis, paresis, or recent POP immobilization of a leg | 1 |
| Recently bedridden for >3 days or major surgery <12 weeks | 1 |
| Localized tenderness along the distribution of the deep venous system | 1 |
| Entire leg swelling | 1 |
| Calf swelling >3cm, compared with asymptomatic leg | 1 |
| Pitting oedema (greater in the symptomatic leg) | 1 |
| Dilated superficial veins (non-varicose) | 1 |
| Previously documented DVT | 1 |
| Another diagnosis more likely than DVT | –2 |

Total score ≤1 means DVT is 'unlikely'. Score of ≥2 signifies DVT is 'likely'.

All patients investigated for DVT with a 'likely' Wells score (≥2) or an elevated D-dimer level require USS. A normal whole leg USS (femoral, popliteal, and calf vein scan) will exclude DVT. If a thigh scan is performed (femoral and popliteal veins), DVT can only be excluded by two normal thigh USS, 1 week apart.

Anticoagulate with LMWH all patients with a 'likely' score whilst awaiting an outpatient USS (see Table 3.9).

Treat patients diagnosed with calf or thigh DVT with LMWH, rivaroxaban, or apixaban, and discharge with a sufficient supply and medical outpatient follow-up. Advise them to return immediately if they become breathless or have chest pain. *Note*: if prescribing apixaban, be sure to include the dose reduction at 7 days (see Table 3.10).

### Upper limb DVT

Often seen with chemotherapy central or long lines. May be associated with plethora and swelling of the arm or face. If suspected, request an USS of the axillary, subclavian, and jugular veins, or a CT scan of the central veins. Treat as for lower limb DVT.

### Superficial thrombophlebitis

Patients present with a painful, tender area of the skin. The diagnosis is made clinically with a firm, tender superficial vein and overlying erythema. This may coexist with DVT. If there is any doubt as to the presence of a DVT, investigate using the DVT protocol. Otherwise, treat with an NSAID or a 6 week course of an oral anticoagulant. Arrange follow-up either in the ED, in a medical clinic, or with the GP to check for resolution.

# Pulmonary embolism

The mortality of diagnosed and treated PE is 7%. Pulmonary embolic disease can result in a variety of symptoms often misdiagnosed as asthma, anxiety, pneumonia, and ACS.

## History

Most patients with PE experience dyspnoea, commonly without other symptoms. Syncope with cyanosis, cardiac arrest, or angina are signs of massive PE. A minority present with pleuritic chest pain, some with additional haemoptysis. Always consider PE in patients with unexplained hypoxia or breathlessness. Take a full history of concurrent illness, surgical procedures, recent hospital admission, past history, including DVT and PE, and travel and family history.

## Examination

Examination may be normal.

- Tachycardia and tachypnoea are common.
- Pyrexia following lung infarction is common.
- 30% of all patients with PE have normal $SpO_2$.
- Always record BP. Hypotension indicates massive PE.
- Perform a full respiratory and cardiovascular examination.
- Always examine the legs for signs of DVT.

**Table 3.9** Modified Wells clinical probability assessment score for PE

| Clinical feature | Score |
|---|---|
| Signs of DVT (minimum of objective leg swelling and tenderness) | 3.0 |
| IV drug use | 3.0 |
| PE is the most likely diagnosis | 3.0 |
| Heart rate >100 | 1.5 |
| Prior PE or DVT diagnosis | 1.5 |
| Bedridden for >3 days or surgery within the past 4 weeks | 1.5 |
| Cancer (treated actively or with palliation within last 6 months) | 1.0 |
| Haemoptysis | 1.0 |

Total score ≤4.0 = PE unlikely; score ≥4.5 = PE likely.

Any patient scoring ≥4.5 on the Wells score OR who has an elevated D-dimer requires pulmonary imaging. Only a normal D-dimer AND a 'PE unlikely' Wells score will safely exclude PE.

## Investigations for suspected PE

- If hypoxic, tachycardic, or hypotensive, insert an IV cannula.
- All patients should have blood taken for FBC and U&E.
- Take a D-dimer test on any patient who scores ≤4.0 on the Wells score (see Table 3.9). A normal D-dimer in a patient scoring <4.0 excludes PE.
- Arrange an ECG (to look for MI or pericarditis) and a CXR (to look for pneumothorax or pneumonia). ECG and CXR are often normal in PE.

## Diagnostic imaging for pulmonary embolus

There are two forms of imaging for PE: CT pulmonary angiography (CTPA) and ventilation–perfusion (V/Q) scanning. CTPA uses a higher dose of radiation (not good for young patients) but will give a definitive answer, as well as diagnose other conditions (like aortic dissection).

Planar V/Q and V/Q single-photon emission CT (SPECT) use a lower dose of radiation but may not give a definitive answer. The V/Q scan result must concord with the clinical probability to diagnose or exclude PE (both PE unlikely or both PE likely). Other combinations are non-diagnostic and necessitate CTPA.

## Treatment of DVT/PE

Aim to treat patients with PE as outpatients after senior review if they are ambulant and have normal $SpO_2$, RR, and heart rate. Outpatient treatment is dependent on reliable follow-up. Admit those who are hypoxic, hypotensive, tachycardic, tachypnoeic, or unable to cope at home (see https://www.brit-thoracic.org.uk).

As soon as venous thrombosis is confirmed or if there is a delay of >4hr to diagnose in a high-risk patient, give a dose of anticoagulation (see Table 3.10).

**Table 3.10** Choice of anticoagulant drug for PE in the ED

| Anticoagulant | Dose |
|---|---|
| Rivaroxaban | 15mg PO bd for 21 days, then 20mg PO daily |
| Apixaban | 10mg PO bd for 7 days, then 5mg PO bd |
| Enoxaparin | 1mg/kg SC bd, max 100mg bd |
| Dalteparin | 200U/kg SC daily, max 18,000U |
| Tinzaparin | 175U/kg SC daily, max 18,000U |
| Unfractionated heparin | IVI—arrange admission for warfarinization<br>Give if estimated glomerular filtration rate (eGFR) <20mL/min |

*Suspected massive PE*

- In patients with cardiovascular compromise, call for urgent ICU help.
- Bedside echo will demonstrate a dilated RV.
- Bedside USS may demonstrate DVT.
- Do not take unstable patients for CT or V/Q scanning.
- If suspicion of PE is high and the patient is haemodynamically unstable, administer thrombolytic therapy. Do not delay. Administer alteplase (rtPA) 10mg slow IV over 1–2min, followed by 90mg IVI over 2hr (max dose 1.5mg/kg if patient is <65kg).
- If thrombolysis is contraindicated or does not work, liaise with experts to consider other alternatives, if available (eg surgical embolectomy, catheter-directed thrombolysis).
- After thrombolysis, start unfractionated heparin IVI, with the dose based on the patient's weight.

# Upper gastrointestinal bleeding

## Causes of upper gastrointestinal bleeding

*Common*

- Peptic ulceration.
- Mucosal inflammation (oesophagitis, gastritis, or duodenitis).
- Oesophageal varices.
- Mallory–Weiss tear.
- Gastric carcinoma.
- Coagulation disorders (thrombocytopenia, warfarin).

*Rare*

- Aorto-enteric fistula (especially after aortic surgery).
- Benign tumours (eg leiomyomas, carcinoid tumours, angiomas).
- Congenital (eg Ehlers–Danlos, Osler–Weber–Rendu, pseudoxanthoma elasticum).

## History

Take a detailed history, whilst resuscitating as necessary. Upper GI bleeding usually presents with haematemesis and/or melaena, and bleeding involving the lower GI tract with fresh per rectum (PR) bleeding. However, major upper GI bleeding may present with fresh PR bleeding.

Ask about the amount and duration of bleeding, any past history of GI bleeding or liver problems, and associated symptoms (abdominal pain, weight loss, anorexia). Syncope usually infers a significant bleed. Take a full drug history (ask about aspirin, NSAIDs, anticoagulants, iron), and enquire about alcohol consumption.

## Examination

Check ABCs. Rapidly assess for hypovolaemic shock (pulse and RR, BP, GCS, skin colour/T°, capillary refill time). Look at any available vomit or faeces. Check for abdominal masses, tenderness, or surgical scars (including aortic grafting). Look for stigmata of liver disease. Perform a PR examination and check for faecal occult blood (FOB).

## Investigations and diagnosis

Review the patient's old hospital notes, and send blood for FBC, clotting screen, U&E, blood glucose, and group and save or cross-matching (according to clinical features). Urea may be ↑, but creatinine will be normal unless renal function is impaired. Check $SpO_2$ (obtain ABG if <94%), and consider CXR and ECG. Endoscopy is the investigation of choice to identify the source of the bleeding.

## Risk of further bleeding and death

The risk of mortality and further complications ↑ with ↑ age, comorbidities (especially cancer and heart failure), liver disease, continued bleeding, ↑ urea, and passage of PR blood. Scoring systems can provide an indication of the chance of a rebleed and/or death. The Glasgow–Blatchford score (see Table 3.11) is more useful in the ED than the Rockall score to identify which patients do not need admission.

**Table 3.11** Glasgow–Blatchford score for upper GI bleeding

| Blood urea (mmol/dL) | | Systolic BP (mmHg) |
|---|---|---|
| 6.5–8 = 2pt | | 100–109 = 1pt |
| 8–10 = 3pt | | 90–99 = 2pt |
| 10–25 = 4pt | | <90 = 3pt |
| >25 = 6pt | | |
| Hb (g/L) for men | Hb for women | Other markers: |
| 120–129 = 1pt | 100–119 = 3pt | Pulse ≥100/min = 1pt |
| 100–119 = 3pt | <100 = 6pt | Presentation with melaena = 1pt |
| <100 = 6pt | | Presentation with syncope = 2pt |
| | | Hepatic disease = 2pt |
| | | Cardiac failure = 2pt |

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Only consider patients scoring 0 on the initial Blatchford score (see Table 3.11), with no further evidence of bleeding, for discharge home from the ED with follow-up. Any patient scoring >0 requires urgent endoscopy.

### Treatment of moderate/severe haemorrhage

- Check airway/breathing. Give $O_2$ as needed (see Oxygen, p. 99). Insert two large-bore (14G) IV cannulae, and send blood for FBC, U&E, clotting, and cross-matching.
- Start IV fluids, followed by blood, as necessary.
- Avoid omeprazole acutely, unless the patient has known peptic ulcer disease (give 40mg diluted in 100mL of saline as IVI over 30min).
- If the patient is anticoagulated or has altered clotting (eg liver disease), talk to haematology. Reverse anticoagulation giving vitamin K/prothrombin complex concentrate/idarucizumab, as needed.
- Insert a urinary catheter and monitor the urine output.
- Ensure that patients with severe uncontrolled variceal bleeding, severe encephalopathy, hypoxia, acute agitation, or evidence of aspiration have their airways secured, if necessary by tracheal intubation.
- Transfuse using the massive transfusion protocol to maintain Hb >70g/L.

### Managing severe haemorrhage possibly due to varices

For unstable patients with a past history of varices or clinical features of hepatic failure, arrange emergency endoscopic treatment.

- Commence fluid rescucitation.
- Give terlipressin (2mg IV, repeated every 4–6hr).
- Check the international normalized ratio (INR), and give IV vitamin K if prolonged.
- Give prophylactic antibiotics, eg ciprofloxacin or second-/third-generation cephalosporin, which may ↓ mortality in severe haemorrhage.
- Consider balloon tamponade as a salvage procedure in a patient with massive haemorrhage at risk of death. If experienced in the technique, insert a 4-lumen Sengstaken/Minnesota tube. Inflate the gastric balloon, then the oesophageal balloon to a pressure of 30–40mmHg in order to tamponade the bleeding varices. Regularly aspirate both ports.

(See https://www.nice.org.uk)

# Lower gastrointestinal bleeding

The most common cause of apparent lower GI bleeding is upper GI haemorrhage. ~20% of acute GI haemorrhage is from the colon or rectum. Angiodysplasia and bleeding from diverticula are the most frequent causes, but inflammatory bowel disease or, very rarely, aorto-enteric fistulae may be responsible. Lower GI haemorrhage often settles spontaneously—localization of the bleeding source may be difficult.

## History

*Nature of bleeding* Melaena may occur following small bowel or proximal colon bleeding, as well as upper GI haemorrhage. Conversely, large volumes of fresh or 'plum-coloured' rectal bleeding may follow upper GI haemorrhage. Bloody diarrhoea suggests inflammatory bowel disease or infective colitis.

*Associated symptoms* Weight loss, anorexia, or a change in bowel habit raise suspicion of colonic carcinoma. Abdominal pain may be a feature of ischaemic colitis, inflammatory bowel disease, or carcinoma. Anal pain commonly occurs with anal fissure or as a complication of haemorrhoids.

*Syncope or postural dizziness* May indicate significant haemorrhage.

*Past medical history* Ask about inflammatory bowel disease, peptic ulceration, or other illnesses. Previous aortic surgery with graft insertion can rarely result in the formation of an aorto-enteric fistula (symptoms include sporadic or fulminant bleeding, often with syncope).

*Drug history* Ask about salicylates, NSAIDs, corticosteroids, and anticoagulants.

*Family and social history* Note any family history of peptic ulcers or inflammatory bowel disease. Enquire about alcohol consumption.

## Examination

First assess for signs of hypovolaemia, and commence resuscitation if necessary. Document pulse, BP (comparing erect and supine, noting any postural drop), T°, and $SpO_2$. Examine the abdomen, and PR in all cases.

## Investigation

Obtain blood for cross-matching (ask for 4–6U of type-specific if urgent), FBC, U&E, glucose, and coagulation studies. Perform an ECG on any patient >50y. Review old patient notes, especially in relation to previous colonoscopy or GI pathology.

## Risk of further bleeding and death

The risk of mortality and further complications ↑ with ↑ age, comorbidities, haemodynamic disturbance, and the use of NSAIDs or aspirin. Only consider discharge if the patient is young and otherwise healthy, has passed only a small amount of blood PR, and does not take NSAIDs or anticoagulants. Always arrange follow-up for these patients.

### Treatment

Patients with signs of hypovolaemia require immediate resuscitation:

- Give $O_2$ as required (see ➔ Oxygen, p. 99).
- Attach monitoring (cardiac monitor, $SpO_2$, BP monitoring).
- Insert two large-bore IV cannulae.
- Give 1L of 0.9% saline or Hartmann's solution IV stat, and give further fluids according to response.
- Insert a urinary catheter.
- If the patient is anticoagulated or has a clotting disorder (eg due to liver disease), reverse anticoagulation and discuss with a haematologist.
- Consider the need for a central venous line.
- Contact the surgical team and ICU.

(See https://www.sign.ac.uk)

## Gastrostomy tube problems

An ↑ number of individuals are being managed in the community in a variety of care settings with indwelling percutaneous endoscopic gastrostomy (PEG) tubes, which are being used for enteral nutrition and administration of medications.

### Tube misplacement

Gastrostomy tubes are not infrequently pulled out or 'fall out' (become inadvertently misplaced). The track can close off within hours, so triage ahead and contact gastroenterology—if not immediately available, gently attempt to pass a replacement tube. A lubricated Foley catheter (of size ≤ original PEG tube) can act as a temporary measure to keep the track open (do not feed through this).

### Other problems

Tube blockage may respond to gentle flushing with warm water using a 20mL syringe. Contact the gastroenterology team for other problems (infection, bleeding, or gastric leakage).

## Refeeding syndrome

This relatively rare, but potentially life-threatening, condition is not always recognized early. Any patients who have not eaten for ≥5 days are at risk. Patients with low body mass index (BMI)/anorexia and alcoholics are at particular risk. Hypophosphataemia, hypomagnesaemia, and hypokalaemia can occur and worsen as food is reintroduced, so check baseline blood glucose and U&E (including phosphate and magnesium) in patients at risk, before considering starting to feed. Give oral thiamine and oral or IV vitamin B, and get expert advice before feeding. Rehydrate and start to correct electrolyte abnormalities. Ensure that, when feeding is commenced, it starts very slowly and with regular monitoring of electrolytes.

# Jaundice

Serum bilirubin levels are usually >51 micromoles/L before clinical jaundice occurs. Causation may be categorized as pre-hepatic, intrahepatic, and post-hepatic, although a mixed aetiology may be present (see Table 3.12).

**Table 3.12** Causes of jaundice

| | |
|---|---|
| Pre-hepatic | • Haemolytic anaemia<br>• Malaria |
| Intrahepatic | • Viral infection (eg hepatitis A–E, EBV, leptospirosis)<br>• Alcohol<br>• Gilbert's syndrome<br>• Paracetamol poisoning/drugs<br>• Autoimmune liver disease<br>• Non-alcoholic fatty liver disease<br>• Biliary malignancy |
| Post-hepatic | • Gallstones<br>• Malignancy (eg pancreatic, biliary, hepatic)<br>• Pancreatitis |

Excessive ingestion of β-carotene can lead to pseudo-jaundice (although sclerae remain normal in colour).

## History and examination

Ask about the duration of symptoms and any other associated features such as itching and weight loss. Pale stools and dark urine may be seen in obstructive jaundice. Ascertain recent prescribed, over-the-counter, and illicit drug use (including herbal medicines), alcohol intake, foreign travel, tattoos, and piercings. Red flags include evidence of hepatic dysfunction/encephalopathy (see ➲ Acute confusional state, pp. 140–1), haematemesis or melaena, or signs of sepsis (see ➲ Sepsis, pp. 62–3). Check for hepatosplenomegaly, abdominal tenderness, and masses.

## Investigations

- Check FBC, coagulation screen, U&E, LFTs, and amylase. Alkaline phosphatase is ↑ more than alanine aminotransferase (ALT) in cholestatic aetiologies—↑ gamma glutamyl transpeptidase can confirm this. ↑ ALT suggests a hepatic cause. Isolated ↑ bilirubin most commonly reflects Gilbert's syndrome. Aspartate aminotransferase (AST):ALT ratio of >1 is seen in alcohol-induced liver disease.
- Consider a viral hepatitis screen.
- Urinalysis: bilirubin in the urine suggests conjugated hyperbilirubinaemia.
- USS can identify gallstones, biliary duct dilatation, and some tumours.
- CT can identify smaller liver and pancreatic lesions.

## Management

Admit the patient if acutely unwell, appears septic, is encephalopathic, or has red flags. Generally, admit obstructive causes under surgeons, and others under a medical team—follow local policy. Refer patients who do not require admission to an appropriate outpatient clinic.

# Ascites and liver failure

## Background

Ascites is an abnormal accumulation of fluid within the peritoneal cavity (deriving from the Greek 'askos', meaning pot or bag). Up to 20mL of fluid may be present physiologically in women of child-bearing age. The most common cause of ascites is hepatic cirrhosis, but it can occur with heart failure, peritonitis, pancreatitis, TB, acute hepatitis, and intra-abdominal malignancy. Patients having peritoneal dialysis also have excess fluid within the peritoneal cavity and are at particular risk of bacterial peritonitis. Cirrhotic ascites is associated with a poor prognosis. If refractory, the survival at 1y is <50%.

## Clinical features

Patients may attend the ED when the volume of ascitic fluid causes discomfort due to local pressure effects on the GI tract. Loss of appetite, nausea, and altered bowel habit are often features. Respiratory difficulty may occur due to diaphragmatic compression. Look for stigmata of liver disease. Gross ascites is often clearly visualized with a taut, distended abdomen out of proportion with the patient's body habitus. Examine for shifting dullness and fluid thrill. Check for other evidence of hepatic decompensation, i.e. jaundice, encephalopathy, and variceal haemorrhage. If clinical evidence of infection, consider spontaneous bacterial peritonitis.

## Investigations

- FBC, U&E, LFTs, amylase, coagulation screen, group and save (cross-match if variceal bleed suspected).
- Ammonia level if hepatic encephalopathy suspected.
- Hepatitis screen.
- Bedside USS may confirm ascites.
- USS or CT may help identify the cause if it is not apparent.
- Perform an ascitic tap under USS guidance and send fluid for microscopy, culture and sensitivity, neutrophil count (EDTA tube), protein, amylase, and cytology if infection is suspected.

## Management

- Resuscitate as appropriate.
- If there is evidence of an upper GI bleed, consider treatment as for oesophageal varices, even if no previous endoscopy (see ➲ Upper gastrointestinal bleeding, pp. 126–7).
- If evidence of spontaneous bacterial peritonitis or peritoneal dialysis infection, commence empirical IV antibiotics (e.g. ciprofloxacin PO 500mg bd, or in severe infection/unable to swallow piperacillin–tazobactam IV 4.5g tds).
- Treat hyponatraemia (see ➲ Sodium derangements, p. 162).
- If encephalopathic, prescribe lactulose.
- Stop NSAIDs and ACE inhibitors.
- Refer to gastroenterology for ascitic drainage and albumin replacement. If the patient is not overtly unwell, they may be able to have drainage the next day in an ambulatory setting.

# Headache

Headaches of non-traumatic origin account for ~0.5% of ED attendances—10–15% of these have serious underlying pathology. Patients typically present in one of three ways:

- Severe headache, unlike any previous one ('first severe' or 'worst ever').
- Headache with associated worrying features (altered mental status, fever, focal neurology).
- Chronic severe headache unresponsive to treatment.

## Causes

*Primary headaches*

- Migraine.
- Tension headaches.
- Cluster headaches.
- Miscellaneous (benign cough headache, benign exertional headache, headache associated with sexual activity).

*Secondary headaches*

- Head injury.
- Vascular (stroke, intracranial haematoma, subarachnoid haemorrhage, unruptured arteriovenous malformation, venous thrombosis, hypertension).
- Non-vascular intracranial disorder (↑ CSF pressure, post-LP, intracranial tumour).
- Substance misuse or withdrawal (including analgesia withdrawal or rebound).
- Infection (encephalitis or meningitis).
- Metabolic (hypoxia, hypercapnia, hypoglycaemia, CO poisoning, dialysis).
- Craniofacial disorder (pathology of skull, neck, eyes, nose, ears, sinuses, teeth, mouth, temporomandibular joint dysfunction).
- Neuralgias (trigeminal, occipital, and other cranial nerves).

## Approach

Use a detailed history and examination (including vital signs and neurological examination) to search for potentially serious causes. Look particularly for the following (some typical features in brackets):

- Subarachnoid haemorrhage (sudden, severe onset, syncope)—see ➲ Subarachnoid haemorrhage, pp. 134–5.
- Meningitis or encephalitis (fever, neck rigidity, ± indwelling ventriculoperitoneal shunt)—see ➲ Meningitis, pp. 232–3.
- Head injury (history or signs of trauma)—see ➲ Head injury: introduction, pp. 362–3.
- ↑ ICP (papilloedema, loss of retinal vein pulsation).
- Stroke (focal neurological signs)—see ➲ Stroke, pp. 150–1.
- Acute glaucoma (painful red eye, ↓ VA, irregular semi-dilated pupil)—see ➲ The red eye, p. 558.
- Cranial arteritis (jaw pain, temporal artery tenderness)—see ➲ Giant cell arteritis, p. 137.

### History

Features suggesting possible serious pathology are:
- Sudden-onset headache.
- Worst headache ever.
- Dramatic change in pattern of headache.
- Known HIV or malignancy.
- Presence of a ventriculoperitoneal shunt.
- Headache coming on during exertion.
- New-onset headache in those aged >50y.

Ask about drugs and the possibility of toxins (eg CO).

### Examination

- Check GCS, pulse rate, RR, BP, T°, and $SpO_2$.
- Feel the head for muscular tenderness, arterial tenderness, and trigger points for neuralgia, and look for evidence of head injury.
- Examine the eyes for VA, pupil reactions, and eye movements. Look at the fundi for papilloedema.
- Palpate the sinuses for tenderness.
- Look in the ears for haemotympanum or infection.
- Check the oral cavity for infection.
- Look for evidence of purpura/rash of meningococcal infection.
- Complete a full neurological examination (include cranial nerves, limb tone, power, sensation, co-ordination, and reflexes).
- Check for *Kernig's sign*: straightening the knee, whilst the hip is flexed, produces discomfort in the presence of meningeal irritation.

### Management

Tailor investigation and emergency treatment according to the presentation, based upon the likely diagnosis.
- Check FBC, ESR, CRP, U&E, and blood glucose.
- If pyrexial and no other obvious source of infection found, take blood cultures and consider cefotaxime 2g IV ± aciclovir. Start IV fluids and refer—a CT head scan may be required (and LP if no sign of ↑ ICP).
- Give paracetamol PO (or IV if vomiting) and an NSAID.
- Consider metoclopramide 10mg IV with IV fluid (eg 1L of 0.9% saline), which can be an effective treatment for some headaches.
- Arrange an emergency CT brain scan for any patient with an acute severe headache or with a history of seizure or an abnormal neurological exam. Adopt a low threshold for CT scan for any patient with HIV.
- Use the Ottawa rule to rule out subarachnoid haemorrhage in patients aged 15–39y (see ➔ Ottawa rule to exclude subarachnoid haemorrhage, p. 134).

It may be safe to discharge home a patient with slow-onset headache that has resolved following treatment, and with a normal examination and blood tests. Advise GP follow-up and to re-attend if symptoms worsen.

# Subarachnoid haemorrhage

►► *Consider subarachnoid haemorrhage in any 'worst ever' or sudden-onset headache.*

Atraumatic subarachnoid haemorrhage is an important cause of sudden collapse and death at any age. Most bleeds follow the rupture of saccular ('berry') aneurysms in the circle of Willis (see Fig. 3.30). Other bleeds may be due to arteriovenous malformations, tumours, or connective tissue disorders.

## History

Up to 70% of patients with subarachnoid haemorrhage report rapid-onset or 'worst ever' headache. This is classically described as 'like a blow to the back of the head', accompanied by neck pain, photophobia, and vomiting. In 25%, exertional activities precede the event. The patient may present after syncope or fits. Drowsiness and confusion are common. 'Warning headaches' may precede subarachnoid haemorrhage. Unilateral eye pain may occur.

## Examination

Document pulse rate, BP, T°, and GCS. An unconscious patient with signs of Cushing's response signifies ↑ ICP. Perform a full cranial and peripheral nerve examination. There may be focal motor and sensory signs due to intracerebral extension of the haemorrhage or vasospasm, subhyaloid haemorrhages (blotchy haemorrhages seen in the fundi), or cranial nerve palsies. Oculomotor nerve palsy is characteristic of a berry aneurysm involving the posterior communicating artery. Neck stiffness is often absent in ED presentations, either because meningeal irritation has not yet occurred or because the patient is deeply unconscious.

## Ottawa rule to exclude subarachnoid haemorrhage

This excludes subarachnoid haemorrhage in alert patients aged 15–39y, with new severe atraumatic headache, with maximum intensity within 1hr, who have no neck pain or stiffness, no witnessed loss of consiousness, no onset during exertion, no thunderclap headache (defined as peak within 1s), and no limited neck flexion on examination. The rule does not apply to patients with new neurological deficits, previous aneurysm/bleed, known brain tumour, or chronic recurrent headaches.

## Investigations

This may need to proceed alongside resuscitation in seriously ill patients:

- Assess ABC. If the patient is unconscious, open the airway and contact ICU. Consider urgent RSI, tracheal intubation, and IPPV.
- Obtain venous access, and check BMG, FBC, clotting screen, and U&E.
- CXR may show changes of neurogenic pulmonary oedema.
- ECG may demonstrate ischaemic changes.
- Modern high-resolution CT scanning within 6hr will identify >98% of subarachnoid haemorrhages. If the scan does not show a bleed, but the patient is deemed to be at high risk, admit for LP and CSF analysis (to be done >12hr after headache onset).

Once diagnosed on plain CT, involve the neurosurgical team and consider the need and timing for a CT angiogram. It may be useful to use the Hunt and Hess score (see Table 3.13) when communicating the severity by phone.

**Table 3.13** Hunt and Hess scale for subarachnoid haemorrhage

| Grade | |
|---|---|
| 1 | Asymptomatic, mild headache, slight nuchal rigidity |
| 2 | Moderate to severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy |
| 3 | Drowsiness/confusion, mild focal neurological deficit |
| 4 | Stupor, moderate to severe hemiparesis |
| 5 | Coma, decerebrate posturing |

## Treatment

Tailor this according to the presentation and the need for resuscitation:

- Give $O_2$ as required.
- Provide adequate analgesia and antiemetic. Codeine (30–60mg PO), paracetamol (1g PO), and/or NSAID may suffice. Some patients require more potent analgesics (eg morphine titrated in 1mg increments IV, according to response)—proceed slowly to avoid drowsiness.
- If unconscious (GCS <8), severely agitated, or combative, tracheal intubation (with GA) will allow IPPV and control of $pCO_2$ to within normal levels. Insert a urinary catheter and an arterial line.

Contact the neurosurgical team—further treatment options include:

- Nimodipine (60mg PO every 4hr or 1mg/hr IVI) to prevent and treat ischaemic neurological deficits secondary to vasospasm.
- Mannitol IV (eg 200mL of 10%) if there is evidence of ↑ ICP.

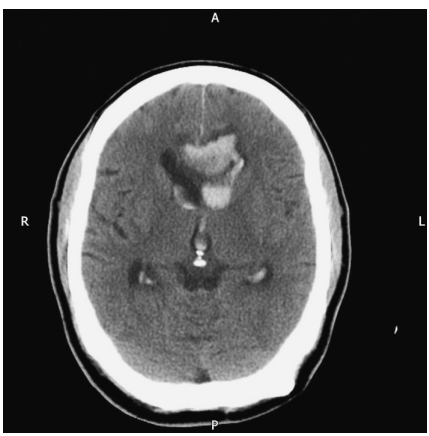


**Fig. 3.30** CT showing acute subarachnoid haemorrhage following rupture of berry aneurysm.

# Migraine

Patients with recurrent migraine rarely attend the ED unless symptoms are different from usual—take care to avoid missing more serious conditions. The pathogenesis of migraine is not entirely clear, but there is initial vasoconstriction and subsequent vasodilatation of both intracranial and extracranial blood vessels.

## Presentation

Precipitants include fatigue, alcohol, menstruation, oral contraceptive pill (OCP), hunger, chocolate, cheese, shellfish, and red wine.

A *prodrome* lasting 5–30min occurs in a third of patients, with blurred vision, photophobia, or scintillating scotomata (an area of blurred or absent vision surrounded by moving zigzag lines), malaise, anorexia, and vomiting. A few experience hemiparaesthesiae, mild unilateral weakness, ataxia, or dysphasia. The following headache may last 4–72hr and is usually 'throbbing' and unilateral, but may be generalized. Photophobia, nausea, or phonophobia are common.

## Rare forms of migraine

*Hemiplegic migraine* Profound hemiplegia precedes the development of the headache by 30–60min. Weakness and other focal deficits usually resolve quickly. Occasionally, they may be slow or fail to resolve.

*Basilar migraine* Brainstem disturbances, with impaired consciousness, vertigo, dysarthria, diplopia, and limb weakness.

*Ophthalmoplegic migraine* Transient unilateral ophthalmoplegia and ptosis, which may last several days.

*Acephalgic migraine* Very occasionally, neurological defects may be present without headache.

## Examination

Look for evidence of other serious diagnoses.

## Treatment of acute attacks

- Give simple analgesia (eg paracetamol 1g PO PRN qds or soluble aspirin 600–900mg PO or an NSAID), in combination with a $5HT_1$ agonist (eg sumatriptan 50–100mg PO or 6mg SC). Oral triptans are not licensed in patients under 18y—consider nasal sumatriptan instead for patients aged 12–17y. Triptans cause vasoconstriction and are contraindicated in IHD, uncontrolled hypertension, and basilar and hemiplegic migraine. Rebound headache may occur in up to 45%—offer a second dose of triptan if there is initial improvement but then relapse within 2–4hr. Advise GP follow-up for all patients treated with a triptan.
- Consider an antiemetic (eg metoclopramide 10mg PO or IV).
- Refer for admission patients who have neurological signs or altered mental status or where there is diagnostic uncertainty (including a change in severe headache pattern).
- Avoid ergotamine and opioids (see https://www.nice.org.uk).

## Giant cell arteritis

Also known as 'temporal arteritis' or 'cranial arteritis' (see ➔ Giant cell (temporal) arteritis, p. 557).

Consider this in all patients >50y with a recent onset of headache or a change in headache pattern. There may be weight loss, night sweats, low-grade fever, jaw claudication, and ↓ vision (up to 10% present with acute visual loss), shoulder girdle stiffness, and muscular aches (polymyalgia). Involvement of carotid or vertebral arteries may lead to TIAs or stroke.

**Examination** The temporal arteries may be tender, reddened, pulseless, or thickened. Fundoscopy is usually normal, but papilloedema can occur later in the disease.

**Investigation** ↑ CRP and/or ↑ ESR >> 40mm/hr, often with low-grade anaemia and leucocytosis. A normal CRP/ESR does not exclude temporal arteritis—a minority (10%) with the condition will have normal markers at presentation.

**Treatment** In view of the serious risk of rapidly progressive visual loss, if suspected, give hydrocortisone 200mg IV (or prednisolone 40mg PO) immediately. Refer to the neurologist or ophthalmologist as an emergency—the diagnosis may be confirmed by temporal artery biopsy.

## Space-occupying lesions

If the headache is always located on the same side, consider space-occupying lesions and arteriovenous malformations. Headaches that are dull, aching, and made worse by lying down or straining are typical of space-occupying lesions. Space-occupying lesions include primary tumours, metastases (see Fig. 3.31), aneurysms, haematomas, and abscesses. They may present with personality change, seizures, focal neurological signs ± ↑ ICP.

Diagnosis is apparent on CT scan. Provide analgesia (eg paracetamol and codeine), as required. Liaise with the neurosurgeon—if there is associated cerebral oedema, give dexamethasone 4mg PO and discuss the need for mannitol (eg 0.5g/kg IVI over 20min).

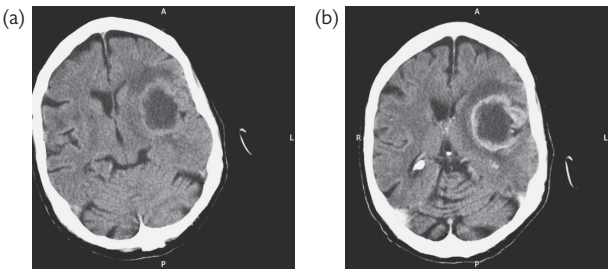


**Fig. 3.31** CT showing cerebral metastasis from a lung primary (a), 'ring-enhancing' with contrast (b).

# Other causes of headache

## Cluster headache

These are more common in men. Often there is a family history. Headache usually occurs at night, waking the patient. Sometimes alcohol may act as a precipitant. Headaches are typically 'clustered' into up to eight attacks per day, each lasting between 15 and 180min. Pain is usually severe, centred upon the eye. Associated symptoms, often unilateral, include conjunctival injection, lacrimation, nasal congestion, rhinorrhoea, forehead and facial sweating, miosis, and ptosis.

*Treatment* High-flow $O_2$ (12L/min via reservoir mask) for 15min sometimes provides relief. Otherwise, use paracetamol/NSAID. Consult before contemplating starting ergotamine or sumatriptan.

## Trigeminal neuralgia

Characterized by stabbing unilateral pain within the distribution of the trigeminal nerves. Stimulation of the 'trigger area' (eg by touching, hairbrushing, or even chewing) induces very severe pain. Treat with carbamazepine and oral analgesia. Admit if the pain is severe and unrelieved.

## Malignant hypertension

Hypertension is an unusual cause of headaches but is seen in patients with malignant hypertension and diastolic BP >130mmHg (see ➲ Hypertensive problems, p. 94).

## Ventricular shunts

Assume that any patient who presents with headaches associated with a ventricular shunt has infection/blockage and refer as an emergency. Associated drowsiness is a particular pointer to blockage.
Send bloods (including WCC and CRP) and request a CT scan.

## CSF spinal leak headache

Headache may occur in the first few days after a spinal or epidural anaesthetic as a result of a CSF leak. Provide simple analgesia for mild symptoms. Patients with more severe headache may need treatment in the form of an injected 'blood patch' (contact the anaesthetic team).

## Analgesic headache

Chronic use of simple analgesics, sympathomimetics, ergotamine, or cocaine is associated with headaches. Stopping or starting certain medications (eg OCP) can also cause headache, as can withdrawal from caffeine. Exclude serious causes and advise GP follow-up with advice on medication use.

### Cerebral venous thrombosis

More common than previously realized. It presents in a similar fashion to subarachnoid or subdural haemorrhage—sudden-onset headache with nausea and vomiting. It may be associated with sinus infections, pregnancy, and the post-partum period. Presenting features partly reflect the sinus involved (most commonly the sagittal sinus or transverse sinus). Cerebral venous thrombosis may not be apparent on initial plain CT but can be diagnosed on contrast CT or MRI. Treat with heparinization and refer for admission.

### Idiopathic intracranial hypertension

Previously known as 'benign intracranial hypertension'. More common in women with a high BMI. ↑ ICP can cause permanent damage to the optic nerve and result in blindness if not treated. Presenting symptoms and signs reflect ↑ ICP: headache (worse on coughing/sneezing), vomiting, cranial nerve palsies, and papilloedema. The diagnosis is made by a combination of a normal CT scan with ↑ opening pressure on LP.

**Meningitis** See ➲ Meningitis, pp. 192–3.

**Encephalitis** See ➲ Acute encephalitis, p. 234.

### Miscellaneous causes

Headaches may also result from:

- *Hypoxia and hypercapnia*.
- *Poisons*, eg CO and solvents (see ➲ Carbon monoxide poisoning, p. 216).
- *Drugs*, eg nitrates, sildenafil, and after alcohol ('hangover').
- *Post-traumatic* (see ➲ Post-concussion symptoms, p. 376).
- *Glaucoma* (see ➲ The red eye, p. 558).
- *Sinusitis* (see ➲ Paranasal sinusitis, p. 571).

### Tension headache

The diagnosis is only made after exclusion of more serious pathology.

*The history* may be described in a dramatic manner. The headache is usually continuous, pressing, or tight ('band-like') in nature. It is usually bitemporal or occipital. Usual features of migraine are absent and the headache does not worsen with exertion.

*Examination* often reveals pericranial muscle tenderness but is otherwise normal.

*Treat* with simple analgesia (eg paracetamol 1g PO qds PRN) and advise GP follow-up. Reassure the patient that a thorough history and examination have not revealed any worrying features.

# Acute confusional state (delirium)

## Definition of delirium

Delirium is a form of organic brain syndrome characterized by:

- Disturbed conscious level and mood (overactivity, excitement, drowsiness, or stupor).
- Global disturbance of cognition (memory, orientation, attention, speech, motor function).
- Rapid onset with fluctuating course (often worse at night, with reversal of usual sleep–wake cycle) and brief duration.
- Perceptual distortions and hallucinations (especially visual).

## Causes of acute confusion

One or more of the following may be the underlying cause of an acute confusional state (several causes frequently coexist):

- *Prescribed medication*: digoxin, cimetidine, steroids, analgesics, diuretics, anticholinergics, antiparkinsonian drugs.
- *Drugs of abuse*: opioids, benzodiazepines, ecstasy, amphetamines, hallucinogens.
- *Withdrawal*: from alcohol, opioids, hypnotics, or anxiolytics.
- *Infection*: pneumonia, urinary tract infection (UTI), septicaemia, meningitis, encephalitis.
- *Metabolic*: hypoxia, hypercapnia, hypoglycaemia, acidosis, hyponatraemia, hypercalcaemia.
- *Cardiac*: acute MI, cardiac failure, endocarditis.
- *Neurological*: head injury, chronic subdural haematoma, meningitis, post-ictal state.
- *Organ failure*: respiratory, renal, and hepatic failure.
- *Endocrine*: myxoedema, thyrotoxicosis, diabetes, Addison's disease.

## Differential diagnosis

Delirium can occur at any age but is much more common in the elderly. It is often misdiagnosed as schizophrenia, depression, or dementia (see Dementia, p. 141). Differentiation can be difficult, but the following are more suggestive of physical illness:

- Non-auditory hallucinations.
- Dysarthria.
- Ataxia.
- Gait disturbance.
- Incontinence.
- Focal neurological signs.

## Approach

Search systematically for (and exclude) the physical causes of acute confusion outlined above.

### Investigation of acute confusion

Perform a thorough, careful physical and mental state examination (see ➲ Mental state examination, pp. 622–3) on acutely confused patients. It may be impossible to obtain an accurate history from the patient, so actively seek other sources of information: relatives, carers, GP, and previous medical records.

*Look* for evidence of alcohol/drug intoxication or withdrawal states. Examine for focal neurological signs and acute cardiac, respiratory, or abdominal abnormalities (including acute urinary retention). Document basic vital signs (GCS, pulse, BP, RR, and T°) in all cases.

*Mandatory basic investigations*

- BMG.
- U&E, FBC, and blood glucose.
- Urinalysis.
- $SpO_2$ and ABG.
- ECG.
- CXR.

Adopt a low threshold for additional tests based on clinical suspicion—blood cultures, thyroid function tests (TFTs), serum digoxin, paracetamol and salicylate, CT brain scan, and even LP may be indicated.

*Be careful not to miss*: hypoglycaemia, head injury, Wernicke's encephalopathy, opioid intoxication, acute alcohol withdrawal, and CO poisoning.

### Dementia

Dementia is an acquired, progressive decline in intellect, behaviour, and personality. It is irreversible and typically occurs with a normal level of consciousness. Note that patients with dementia are at risk of delirium resulting from an acute infective or metabolic origin—a clue to this may be an acute deterioration in mental state.

The *most common causes* of dementia are Alzheimer's disease, vascular dementia, and Lewy body dementia.

## Transient global amnesia

This curious and poorly understood condition is probably more common than generally appreciated. It is characterized by sudden unexplained memory loss in a middle-aged/elderly patient, not accompanied by any other neurological or other abnormalities (such as weakness). The patient is able to follow simple instructions but may appear bewildered and have poor short-term memory. Family members may also be understandably distressed. Investigations do not reveal any abnormality. Often mistaken for delirium or stroke/TIA, transient global amnesia is characterized by the way that memory is so dramatically affected, but without other signs being present. Spontaneous recovery within 24hr is the norm. Transient global amnesia is not a particular predictor of future stroke or other vascular event—advise GP follow-up.

# The unconscious patient: 1

### Common causes

- Hypoglycaemia.
- Drug overdose.
- Head injury.
- Stroke.
- Subarachnoid haemorrhage.
- Convulsions.
- Alcohol intoxication.

### Uncommon causes

- Type II respiratory failure.
- Cardiac failure.
- Arrhythmias.
- Hypovolaemic shock.
- Anaphylaxis.
- Hepatic/renal failure.
- Hypo-/hyperthermia.
- Meningitis/encephalitis.
- Malaria.
- DKA/hyperosmolar hyperglycaemic state (HHS).
- Non-convulsive status epilepticus.
- Wernicke's encephalopathy.

Treatment may be needed before any diagnosis is made. Remember:

- *Airway*.
- *Breathing*.
- *Circulation*.

### Initial resuscitation

*Airway and cervical spine* Whatever the cause of coma, a patient may die due to airway obstruction, respiratory depression, or circulatory failure. Clear and protect the airway immediately, and immobilize the cervical spine if trauma is suspected. Arrange intubation if no improvement.

*Breathing* If breathing is inadequate, ventilate with $O_2$ using a self-inflating bag with an $O_2$ reservoir. An uninjured patient who is breathing adequately can be examined supine, but nurse in the recovery position to ↓ the risk of airway obstruction. Record the RR.

*Circulation* Measure pulse and BP. Observe and feel the skin for colour, sweating, and T°. Obtain reliable venous access. Monitor ECG. Replace IV fluid if indicated.

*Conscious level* Assess the level of consciousness using GCS (see ➔ Head injury: examination, pp. 368–9). Check blood glucose (initially by BMG) and treat hypoglycaemia immediately (see ➔ Hypoglycaemia, pp. 158–9). Record pupil size. Give slow IV thiamine (i.e. two pairs of Pabrinex® ampoules in 100mL of 5% glucose over 30min—see the (*BNF*) to patients with a history of alcoholism or who appear malnourished.

## History

Obtain a history from the ambulance crew and the patient's relatives and friends. Ask:

- How was the patient found?
- When was he/she last seen?
- Is there any suggestion of trauma?
- Is there any history of fits?
- Has there been recent foreign travel?
- Previous symptoms and medical history (including depression).
- Note any drugs available.

Check previous ED records and hospital notes.

## Examination

Examine thoroughly for illness and injury. Check clothes and possessions for tablets and cards/bracelets warning of pre-existing disease.

- ↑ *RR* may reflect obstructed airway, aspiration, pneumonia, DKA, liver/renal failure, salicylate poisoning, methanol, or ethylene glycol.
- *Respiratory depression* may be due to poisoning (eg barbiturates, opioids, tricyclics) or ↑ ICP. Brainstem compression or damage by stroke may cause rapid, irregular, or intermittent (Cheyne–Stokes) breathing.
- *If bradycardic*, consider: hypoxia, complete heart block, ↑ ICP, digoxin or β-blocker poisoning (see ➲ Beta-blocker poisoning, p. 206).
- *If tachycardic*, consider: airway obstruction, hypoxia, hypovolaemia, SVT, VT, or anticholinergic overdose.
- *AF* may be associated with cerebral emboli.
- *Hypotension* suggests hypoxia, shock (hypovolaemic, anaphylactic, septic), or poisoning.
- *Hypertension* may be due to ↑ ICP.
- *Skin*: look for pallor, cyanosis, jaundice, spider naevi, skin crease/scar pigmentation (Addison's disease), rashes (eg purpura in meningococcal infection or (DIC), injection marks (drug addiction or medical treatment), and signs of trauma. Erythema or blistering over pressure points indicate the patient has been unconscious for some hours.
- Measure rectal *T*° with a low-reading thermometer if the skin feels cold. Coma is common at <30°C (see ➲ Hypothermia: presentation, pp. 264–5).

# The unconscious patient: 2

*Neurological examination* includes GCS, limb strength, muscle tone and reflexes, optic fundi, eardrums, neck stiffness (except in neck injury), and palpation of the fontanelle in babies. Lateralizing signs, such as facial or limb weakness, may be caused by a stroke, intracranial bleeding, or pre-existing problems (eg previous stroke or Bell's palsy). Ocular nerve palsy or divergent squint with coma can indicate Wernicke's encephalopathy, requiring IV thiamine, or tricyclic poisoning. Look for subtle signs of seizure activity (eg twitching of ocular muscles or eyelids, unusual limb movements), which may indicate non-convulsive status epilepticus. Look at the fundi—spontaneous central retinal venous pulsations are rare with ↑ ICP. Subhyaloid haemorrhages (blotchy fundal haemorrhages) suggest subarachnoid haemorrhage.

*Hypoglycaemia* can cause localized weakness/coma and mimic stroke.

*Coma without lateralizing signs* is usually due to poisoning, a post-ictal state, brainstem stroke, or hepatic failure—extensor plantar reflexes are common in these conditions.

*Tricyclic antidepressants* often cause coma with dilated pupils, a divergent squint, ↑ muscle tone, jerky limb movements, and extensor plantars. In severe poisoning, there may be muscle flaccidity with respiratory depression and ↓ reflexes (see ➲ Tricyclic antidepressant poisoning, pp. 202–3).

*Coma with small pupils* and respiratory depression suggests opioid poisoning (see ➲ Opioid poisoning, p. 196). In unexplained coma, give a therapeutic trial of naloxone (0.4–0.8mg IV), observing for changes in conscious level, RR, and pupil size.

## Investigations

- BMG and blood glucose. If BMG is low, do not wait for the laboratory result to confirm this before starting treatment.
- VBG/ABG (record $FiO_2$ and whether breathing spontaneously or with IPPV).
- FBC, prothrombin time, U&E.
- Check paracetamol and salicylate levels if poisoning is suspected—paracetamol alone does not cause coma (except in late cases with liver failure), but a mixture of drugs may have been taken. Do not routinely send blood for drug screening for sedatives/hypnotics, but in unexplained coma, keep blood for later analysis if necessary.
- ECG may show arrhythmias (see ➲ Tricyclic antidepressant poisoning, pp. 202–3).
- CXR may show pneumonia, aspiration, trauma, or tumour.
- CT scan will identify subarachnoid haemorrhage, stroke, or head injury.

## Psychogenic coma

Patients sometimes pretend to be unconscious. It can be difficult to be certain of this—exclude other causes first. Suspect psychogenic coma if serious pathology has been excluded, and when the eyes are opened, only the sclerae show as the eyes deviate upwards (Bell's phenomenon).

# Falls in the elderly

With an increasingly elderly population living in more isolated, and often precarious, situations, it is not surprising that many elderly present to the ED following a fall. In addition to the standard approach, pay particular attention to the following questions.

### What caused the fall?

Trying to distinguish between a trip/stumble, a dizzy spell, or medical collapse can be very difficult. Basic observations, lying/standing BPs, ECG, and BMG act as basic screening, but be aware that a medical problem responsible for the fall may not be immediately apparent.

### What injuries resulted?

The classic injury resulting from a fall in an elderly individual who is then unable to get up after is a hip fracture—adopt a low threshold for requesting an X-ray of the pelvis. Fractures of the pubic rami (see ➲ Pelvic fractures, pp. 480–1) are also common as a result of a fall (see Fig. 3.32) and can tip the balance in terms of whether a patient is safe to discharge home. Falls, especially down steps or stairs, can result in significant head and neck injuries which are not always obvious at first.

### Did the patient lie for a long period?

Be aware that as a result of being unable to get up after a fall, the patient may have experienced a 'long lie', with attendant risks of hypothermia, dehydration, pressure sores, and muscle damage (with rhabdomyolysis, hyperkalaemia, and AKI).

### Is it safe to consider discharge home?

Having established that the patient does not have medical problems requiring treatment and hospital admission, determining whether a patient is safe to discharge is very often a complex issue. It requires input from a number of sources, including the patient, relatives, GP, and other specialists (see ➲ Discharging the elderly patient, p. 23).

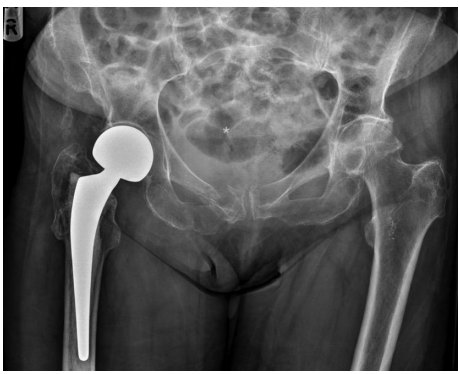


**Fig. 3.32** Right-sided pubic ramus fractures in an elderly patient with previous hemiarthroplasty.

# Collapse and syncope

Syncope is a sudden, transient loss of consciousness, with spontaneous recovery. If a patient suddenly loses consciousness in the ED, assess responsiveness and check for a pulse. Keep the airway clear; give $O_2$, and monitor pulse and ECG. Note any neurological signs during the episode, and obtain BP, $SpO_2$, and BMG.

## Priorities

- Identify serious or life-threatening problems and institute treatment.
- Decide which patients require admission.
- Decide which patients require follow-up.

## History of syncopal episode

*Was it a simple faint?* Vasovagal or neurally mediated syncope is a common response to an overwarm environment or prolonged standing, and can be precipitated by sudden fright or visual stimuli (eg the sight of blood). Other contributors are large meals, prolonged starvation, or alcohol. There are usually premonitory symptoms of feeling unwell, nauseated, dizzy, or tired, with yawning, blurred or 'tunnel' vision, or altered hearing. If the fainter cannot get supine (eg bystanders keeping them upright), seizure-like twitching may occur (*convulsive syncope*). Vomiting and incontinence may occur and do not reliably discriminate seizures from faints.

*Was it a seizure?* Look at the ambulance records. An eyewitness account is crucial. Ask what the witnesses *actually saw* (do not assume they know what a 'fit' looks like). There should typically be no prodrome, and there is often a cry followed by tonic/clonic movements. Cyanosis, saliva frothing from the mouth, heavy breathing, tongue biting, or incontinence suggest a generalized seizure. Post-ictal drowsiness or confusion is normal—very rapid recovery questions the diagnosis.

*Was it a cardiac event?* Cardiac syncopal events are also abrupt in onset (eg collapse due to HCM) and may be accompanied by pallor and sweating. Recovery may be rapid, with flushing and deep/sighing respiration in some cases (eg Stokes–Adams attacks). Nausea and vomiting are not usually associated with syncope from arrhythmias. Ask about previous episodes and chest pain, palpitations, history of cardiac disease, and family history of sudden death. Syncope associated with exertion is a worrying feature—possible causes include aortic or mitral stenosis, pulmonary hypertension, cardiomyopathy, or coronary artery disease.

*Other causes* Carotid sinus syncope is neurally mediated and often occurs with shaving or turning the head. Syncope may be secondary to the effects of medication (eg GTN, β-blockers, antihypertensive drugs). Syncope may also be the presenting feature of subarachnoid haemorrhage, ruptured ectopic pregnancy, aortic or carotid dissection, PE, or GI bleed. Syncope is rarely caused by a TIA.

## Assessment and treatment

Obtain a detailed account from the patient and witnesses. Look for signs of tongue biting, incontinence, or other injuries, and examine the cardiovascular system (CVS) for murmurs, arrhythmias, or abnormalities. Perform a neurological examination and look for focal signs. Do postural tests (supine and standing or sitting pulse and BP). A degree of postural hypotension is common, but postural symptoms (eg dizziness, weakness) are always significant (look for causes of hypovolaemia, eg GI bleed, ectopic pregnancy). Check BMG to exclude hypoglycaemia, and an ECG looking for arrhythmias, LVH, ischaemia, previous or acute MI, and QT prolongation. An abnormal ECG may be the only clue to an underlying HCM or Brugada syndrome (various ECG patterns, including ST elevation in $V_{1-3}$ and RBBB).

### *Disposal*

Admit patients for cardiology review within 24hr if they present with:

- An ECG abnormality.
- Heart failure.
- Loss of consciousness on exertion.
- Family history of sudden death <40y or an inherited cardiac condition.
- New or unexplained breathlessness.
- A heart murmur.

Treat patients as if they have had a 'first fit' (see ➔ Seizures and status epilepticus, pp. 156–7) if they present with one or more of:

- A bitten tongue.
- Amnesia, unresponsiveness, unusual posturing or prolonged limb jerking, head turning to one side.
- History of an aura.
- Post-ictal confusion.

Aim to discharge patients who have made a full recovery and have an appropriate history for vasovagal syncope and a normal examination. Consider suggesting to the GP to arrange outpatient ECG monitoring/investigation if discharging a patient >65y with unexplained syncope.

(See NICE guideline CG109 at https://www.nice.org.uk)

## Diagnoses not to be missed

- *GI bleed*: syncope (± postural symptoms) indicate significant blood loss and hypovolaemia. Perform PR examination to check for blood/melaena.
- *Ectopic pregnancy*: suspect this in women with syncope and abdominal pain or gynaecological symptoms. Do a pregnancy test.
- Ruptured abdominal aortic aneurysm.
- PE (see ➔ Pulmonary embolism, pp. 124–5). A witness may give a history of cyanosis. Indicative of massive thrombus.

# Acute generalized weakness

Weakness may be a feature of common neurological problems (eg TIA/stroke) or accompany many of the causes of collapse (see ➲ Collapse and syncope, pp. 146–7). Less commonly, generalized muscle weakness may be the presentation of a number of other diseases.

Clinical features which may help to distinguish between upper and lower motor neurone lesions are shown in Table 3.14.

**Table 3.14** Distinguishing between upper and lower motor neurone lesions

| Feature | Upper motor neurone | Lower motor neurone |
|---|---|---|
| Wasting | No | Yes |
| Fasciculation | No | Yes |
| Tone | ↑ | ↓ |
| Power | ↓ | ↓ |
| Reflexes | ↑ | ↓ |
| Plantars | Upgoing | Downgoing |

## Guillain–Barré syndrome

Guillain–Barré syndrome follows a respiratory or GI infection and is characterized by progressive symmetrical weakness, spreading from distal muscles to involve proximal muscles. Symptoms and signs include muscle tenderness, back pain, loss of muscle reflexes, sensory symptoms (paraesthesiae of fingers and toes), and disturbance of the autonomic nervous system (hyper- or hypotension, tachy- or bradycardia, bladder atony). Beware respiratory failure, which can rapidly progress to respiratory arrest. Serial vital capacity measurements are advised. Refer to the medical team/ICU.

## Multiple sclerosis

This demyelinating disease of the central nervous system (CNS) is more common in ♀ and usually presents at 20–50y. It follows a relapsing and remitting course with sensory loss, stiffness, weakness of legs, ataxia, autonomic impairment (bladder dysfunction), and diplopia. Patients may present with these symptoms during their first exacerbation or with optic neuritis (pain in one eye, with visual blurring and ↓ VA). Admit under neurology. If there are eye symptoms, arrange urgent ophthalmology review.

## Polymyositis

Polymyositis is an inflammatory myopathy that presents with symmetrical proximal muscle weakness, arthritis, and sometimes muscular tenderness. Patients report difficulty climbing stairs, standing from a low chair, or lifting arms to brush hair. Creatine kinase (CK) levels are raised. Refer to a rheumatologist for treatment.

### Myasthenia gravis

This is a rare autoimmune disease with antibodies to the nicotinic acetylcholine receptors. Crises can be precipitated by infection, with painless weakness in which the muscles are fatiguable, but tendon reflexes and pupil responses are normal. Usually, cranial nerves are involved to a greater extent than limb muscles and the distribution is asymmetrical. Ptosis, diplopia, and blurred vision are the most common presentations, which can be treated with pyridostigmine. Crises may present with severe muscle weakness when the major concern relates to respiratory compromise. The patient may require emergency RSI using rocuronium for paralysis. ICU treatment includes plasmapheresis.

As a diagnostic adjunct in the ED, placing an ice pack over the eyelids improves ptosis.

Patients with known myasthenia gravis may present with weakness due to under-treatment or over-treatment (cholinergic crisis) or as an adverse reaction to an unrelated drug. Refer to the medical team for investigation.

### Periodic paralysis

This encompasses a family of hereditary diseases associated with defects in muscle ion channels. Episodes of weakness can be associated with fluctuations in serum $K^+$ levels, lasting a few hours to a week. On occasions, this is associated with eating a large meal. Patients may develop myotonia between attacks and fixed proximal muscle weakness. Treatment tends only to be required for hypokalaemic periodic paralysis with oral $K^+$ supplementation.

### Wound botulism

Botulism has made a comeback in the IV drug-injecting community. Botulinum toxin inhibits the release of acetylcholine at neuromuscular junctions and sympathetic and parasympathetic synapses. Wound infection with *Clostridium botulinum* presents with diplopia, blurred vision, ptosis, and neck weakness, which can progress to respiratory failure. Treatment is with antitoxin, benzylpenicillin, and metronidazole, along with respiratory support.

Note that generalized weakness may also be caused by:

- Spinal cord compression.
- Tetanus.
- Alcoholic myopathy.
- Diphtheria.
- Lead poisoning.

# Stroke

A *stroke* is an acute onset of a focal neurological deficit of vascular origin which lasts >24hr. The blood supply to the brain has two sources—the internal carotid and the basilar arteries. The internal carotids supply the anterior and middle cerebral arteries, known as the anterior circulation. The basilar artery supplies the posterior cerebral artery in 70% of people (the posterior circulation). Anterior and posterior communicating arteries in the circle of Willis provide collateral circulation in cases of carotid artery stenosis.

## Pathogenesis

70% of strokes occur in those aged >70y, but they can occur at *any* age.
Cerebral infarction (80%) results from:

- Thrombosis secondary to atherosclerosis, hypertension, and arteritis.
- Cerebral embolism from AF, valve disease/replacement, post-MI, ventricular aneurysm, myxoma, endocarditis, or cardiomyopathy.
- An episode of hypoperfusion.

Cerebral haemorrhage (20%) is associated with:

- Hypertension (rupture of small arteries in the brain).
- Subarachnoid haemorrhage (see ➲ Subarachnoid haemorrhage, pp. 134–5).
- Arteriovenous malformations.
- Intracranial tumours
- Bleeding disorders (including anticoagulants) and intracranial tumours.

## Presentation

Stroke preceded by neck pain may indicate carotid/vertebral artery dissection or subarachnoid haemorrhage. Headache is an unusual presentation of ischaemic stroke and may indicate cerebral haemorrhage. Be alert to the possibility of different pathology requiring urgent treatment (eg hypoglycaemia, Todd's paresis, hemiplegic migraine, meningitis, encephalitis, brain abscess, head injury, Bell's palsy, 'Saturday night palsy', tumours).
Undertake a thorough examination, including:

- Assessment of mental status/GCS and signs of meningeal irritation.
- Examination of pupils, fundi, and cranial nerves.
- Assessment of motor function (tone, power, and reflexes).
- Assessment of sensory function (including speech and comprehension).
- Examination for cerebellar signs (co-ordination, speech).
- Record initial NIHSS (National Institutes of Health Stroke Scale).
- Check for sources of embolism (AF, murmurs, carotid bruits).

Localization on clinical grounds alone can be difficult, and differentiation between infarction and haemorrhage requires CT/MRI. NICE recommends use of the ROSIER score to identify patients presenting with acute stroke (see Table 3.15). The ROSIER score will pick up the majority of patients who are having a stroke but may not identify patients with posterior circulation infarcts.

**Table 3.15** ROSIER score for stroke recognition

| Criteria | Points |
|---|---|
| Facial weakness (asymmetrical) | 1 |
| Arm weakness (asymmetrical) | 1 |
| Leg weakness (asymmetrical) | 1 |
| Speech disturbance | 1 |
| Visual field defect | 1 |
| Loss of consciousness or syncope | –1 |
| Seizure | –1 |

Stroke is unlikely if score is 0 or lower.

## Investigations

Examine and investigate first to exclude other conditions, and second to confirm the diagnosis of stroke. As a minimum requirement: BMG, FBC, ESR, U&E, blood glucose, ECG, and CXR. Apply a pulse oximeter (if $SpO_2$ <94%, consider ABG) and a cardiac monitor.

Arrange emergency non-enhanced CT scan where:

- Stroke thrombolysis or thrombectomy may be indicated. If considering thrombectomy, also perform CT contrast angiography.
- The patient is on oral anticoagulant and/or has a bleeding tendency.
- The GCS is <13.
- There are unexplained progressive or fluctuating symptoms.
- There is papilloedema, neck stiffness, or fever.
- There was a severe headache at the onset of symptoms.

## Initial management

Quickly decide if emergency thrombolysis or thrombectomy is indicated—if it is, involve the stroke service and follow protocols (see ➲ Stroke thrombolysis, p. 152).

If the CT scan reveals a bleed, treat accordingly (see ➲ Intracerebral haemorrhage, p. 154).

If emergency thrombolysis is not indicated and the CT shows no bleed:

- Immediately correct hypoglycaemia if present (see ➲ Hypoglycaemia, pp. 158–9).
- If hypoxic, give $O_2$—aim for $SpO_2$ of 90–94% (see ➲ Oxygen, p. 99).
- Screen the patient's ability to swallow (try a teaspoon of water). If unable to safely swallow, prescribe maintenance IV fluids.
- Give aspirin 300mg as soon as possible—PO, or if unable to swallow, PR. Give a proton pump inhibitor (PPI) (eg omeprazole) if previous dyspepsia. If allergic to aspirin, give an alternative antiplatelet drug (eg clopidogrel).
- Do not routinely give anticoagulants or start statins in the ED.
- Hypertension and labile BP are common in the early post-stroke period. Do not attempt to reduce the BP at presentation, unless there is: aortic dissection, pre-eclampsia/eclampsia, hypertensive encephalopathy/nephropathy/cardiac failure/MI, or in some patients with intracerebral bleed.
- Get specialist advice if vertebral artery dissection is suspected.
- Admit directly to a stroke unit.

# Stroke thrombolysis

A treatment which has generated a lot of debate but is in widespread use, stroke thrombolysis benefits some patients but carries a risk of life-threatening haemorrhage. Many departments have protocols which offer thrombolysis to patients aged >18y within 3hr of onset (and up to 4.5hr after onset for those aged 18–80y) for significant stroke symptoms which are not improving, provided CT shows no bleed (see Fig. 3.33) and there are no contraindications.

Deciding whether to thrombolyse can be difficult—defer to senior staff, with involvement of the patient. It is particularly difficult to plan treatment for patients who have relatively minor symptoms or present slightly later or whose symptoms are improving but not resolved.

## Contraindications/clinical exclusions to stroke thrombolysis

- Awoke with symptoms/time of onset unknown.
- Seizure at onset.
- Clinical presentation suggestive of subarachnoid haemorrhage.
- Known bleeding diathesis or low platelets ($<100 \times 10^9$/L).
- Arterial puncture at non-compressible site or LP within past 7 days.
- GI or urinary tract haemorrhage in the past 3 weeks.
- Head injury, intracranial surgery, or stroke in the past 3 months.
- Any previous intracranial haemorrhage, brain tumour, arteriovenous malformation, or aneurysm.
- Diastolic BP >140mmHg (*Note*: if systolic BP is >180mmHg and/or diastolic BP is 105–140mmHg, consider thrombolysis if BP reduces to <180/105mmHg after intervention, eg IV labetalol 10mg IV over 2min, repeated once after 15min if not responding enough).

## Procedure for stroke thrombolysis

- Having decided to thrombolyse, time is of the essence.
- Ensure patient comfort; insert two venous cannulae and monitoring.
- Confirm there is no bleeding on the CT brain scan.
- Do not give aspirin.
- Give alteplase (0.9mg/kg, up to a maximum of 90mg) as an IVI over 1hr, with the first 10% as a slow bolus over 1–2min.
- Monitor closely with regular observations. Stop the IVI of alteplase if there is any suspicion of intracranial haemorrhage (new headache, ↓ GCS, acute hypertension, seizure, vomiting) and get a new CT scan. If this shows haemorrhage, liaise with the stroke team/neurosurgeon.
- After administration of thrombolysis, admit under the care of the stroke team to an acute stroke unit. Do not give any anticoagulant or antiplatelet therapy—this will be considered at 24hr if a repeat CT scan at that time shows no bleeding.

# Stroke thrombectomy

Evidence is growing to support the use of intra-arterial clot extraction. It is becoming increasingly available in specialist centres—follow local protocols. Consider mechanical thrombectomy if there is proximal intracranial large vessel occlusion causing a disabling neurological deficit (NIHSS ≥6) in individuals who have a pre-stroke modified Rankin score of ≤3 (in other words, they are not moderately to severely disabled or severely disabled). NICE (2019) outlines situations where thrombectomy may be offered as soon as possible for certain situations:

- Patients within 6hr of symptom onset (together with IV thrombolysis, if not contraindicated and delivered within the licensed time window) with an acute ischaemic stroke, with confirmed occlusion of the proximal anterior circulation demonstrated by CT angiography or magnetic resonance angiography.
- Patients who were last known to be well between 6 and 24hr previously (including those who woke up with symptoms) who have an acute ischaemic stroke and confirmed occlusion of the proximal anterior circulation demonstrated by CT/magnetic resonance angiography, and imaging confirms there is potential to salvage brain tissue.
- Patients who were last known to be well up to 24hr previously (together with IV thrombolysis, if not contraindicated and delivered within the licensed time window), including those who woke up with symptoms, who have an acute ischaemic stroke and confirmed occlusion of the proximal posterior circulation on CT/magnetic resonance angiography, and imaging confirms there is potential to salvage brain tissue.

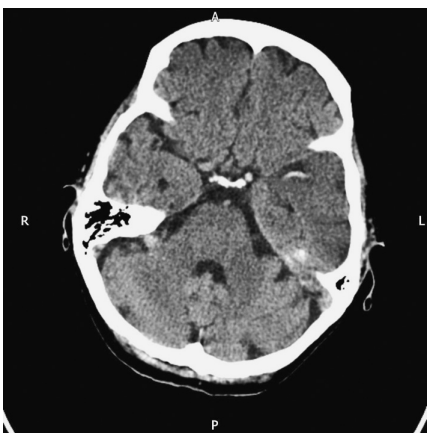


**Fig. 3.33** Hyperdense left middle cerebral artery seen on CT scan as an early sign of acute stroke.

# Intracerebral haemorrhage

Primary acute intracerebral haemorrhage (haemorrhagic stroke) is responsible for a significant proportion (~10%) of acute strokes and ranges from minor dot haemorrhages to an overwhelming bleed (see Fig. 3.34). Treat supportively and medically in the first instance, and seek advice from the relevant specialist team regarding definitive management. For some patients with a large bleed ± significant comorbidities, palliative measures may be appropriate. For others:

- Aim to urgently reverse any anticoagulation (eg give prothrombin complex concentrate and vitamin K for patients taking warfarin, idarucizumab for those on dabigatran, andexanet for rivaroxaban or apixaban).
- Treat significant hypertension—aim to lower systolic BP to <140mmHg within 1hr. Options to achieve this include IVI of isosorbide dinitrate (eg 2mg/hr, titrating up to 12mg/hr) or IV labetalol (initially 10mg slow IV over 2min, repeated every 2min up to 200mg). Do not rapidly lower BP in patients who have an underlying tumour, aneurysm, arteriovenous malformation, or GCS <6/15, or who are going to have surgical evacuation, or who have a massive haematoma with a poor prognosis.
- Liaise with the neurosurgical team. Surgical intervention is not often indicated.

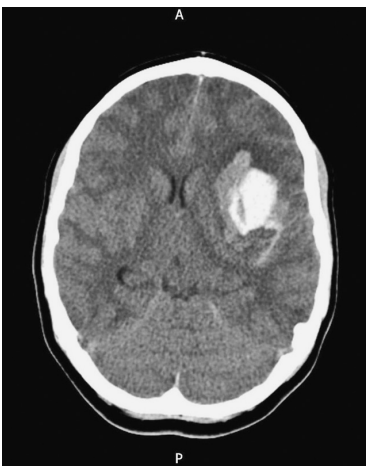


**Fig. 3.34** Large acute left intracerebral haemorrhage, with associated swelling.

# Transient ischaemic attacks

A *TIA* is an episode of transient focal neurological deficit of vascular origin lasting <24hr. A TIA gives major warning for the development of stroke (5% within 48hr, up to 50% in 5y). Even in patients with resolution of symptoms/signs, most have evidence of infarction on CT/MRI.

## Presentation

- *Carotid territory involvement* produces unilateral weakness or sensory changes, dysphasia, homonymous hemianopia, or amaurosis fugax.
- *Vertebrobasilar territory involvement* produces blackouts, bilateral motor or sensory changes, vertigo, and ataxia.

## Causes

Most result from thromboembolic disease involving the heart (AF, mitral stenosis, artificial valves, post-MI) or extracranial vessels (carotid artery stenosis). Other causes are: hypertension, polycythaemia/anaemia, vasculitis (temporal arteritis, PAN, SLE), sickle-cell disease, hypoglycaemia, hypoperfusion (eg arrhythmia, hypovolaemia), and syphilis.

## Assessment

To diagnose a TIA, the symptoms must have resolved within 24hr. Document vital signs, and perform a thorough neurological examination. Look for possible sources of emboli, eg arrhythmias (especially AF), heart murmurs, carotid bruits, and MI (mural thrombus).

## Investigation

- Check BMG and send blood for FBC, ESR, U&E, glucose, lipids, and INR.
- Record an ECG to search for MI and arrhythmia.
- Many patients who end up being diagnosed as having a TIA are symptomatic when they present to the ED, so they will have a CT brain. For patients whose symptoms have resolved by the time of arrival at the ED, do not request a CT scan unless there is a clinical suspicion of a different diagnosis which CT could identify.

## Management

- Give aspirin 300mg PO immediately and continue aspirin until TIA clinic specialist review (which should be in <24hr). The $ABCD^2$ score is no longer recommended to risk-stratify patients (https://www.nice.org.uk).
- Unless there is a contraindication, start treatment with a statin (eg atorvastatin 20–80mg od at night).
- Liaise with the medical team (and follow local guidelines) for patients with newly diagnosed AF to start anticoagulation, providing CT has excluded haemorrhage and there is no uncontrolled hypertension.

### *Advice for discharged patients*

Provide patients who are discharged after a TIA with verbal and written advice, including not to drive until seen in a TIA clinic and to call for an ambulance if any symptoms of TIA or stroke occur.

# Seizures and status epilepticus

## First fit

► *A first fit has enormous consequences—do not diagnose without good evidence.*

A detailed history from both the patient and any witnesses is crucial to the diagnosis. The presence of jerking movements or incontinence does not necessarily reflect epilepsy. Carefully document what was seen, in order to avoid confusion with vasovagal syncope or other types of collapse. Full rapid recovery suggests a syncopal event. Always consider alcohol/drug use, withdrawal states, hypoglycaemia, arrhythmia, head injury, subarachnoid haemorrhage, stroke/TIA, infection (including meningitis), or metabolic disturbance.

As part of the general examination, carefully examine the CNS, documenting: GCS, confusion, focal abnormalities, and findings on fundoscopy. Examine the CVS and check for signs of aspiration.

*Todd's paresis* May follow seizures—focal deficit or hemiparesis may persist for up to 24hr and indicates a high chance of structural lesion.

*Investigations* BMG, glucose, FBC, U&E, blood cultures if pyrexial, ECG, and, if there are chest signs, a CXR. Check urine pregnancy test if of childbearing age. All patients with new-onset seizures need brain imaging at some stage—a significant number have structural CNS abnormalities.

Arrange an emergency CT scan for patients with focal signs, head injury, known HIV, suspected intracranial infection, bleeding disorder (including anticoagulants), or where conscious level fails to improve as expected.

*Disposal* A patient presenting with a first seizure may be discharged home, accompanied by an adult, if they have normal neurological and cardiovascular examinations, the ECG and electrolytes are normal, and there is an appointment with an epilepsy specialist in the coming week. Admit any patient with more than one seizure that day or who does not fit the above criteria. Ensure clear documentation of follow-up arrangements, including booked clinic appointment. Meanwhile, advise the patient not to drive or use machinery and to take sensible precautions, with supervision when performing activities such as swimming/bathing until reviewed. *Document this advice* in the notes.

## Seizures in known epileptics

Ask about any change from the patient's normal seizure pattern. Possible causes of poor seizure control include: poor compliance with medication, intercurrent illness/infection, alcohol, or drug ingestion. Examine to exclude any injury occurring from the fit, especially to the head. Occult dislocations (eg shoulder) may occur. Check vital signs, BMG, and anticonvulsant levels if toxicity or poor compliance is suspected. Refer patients with a significant change in seizure pattern to the medical team. Discharge to the care of a responsible adult those patients who are fully recovered with no injuries, symptoms, or other concerns.

## Status epilepticus

This is continuous generalized seizures lasting >30min or without intervening recovery. Cerebral damage ↑ with duration. Precipitants include cerebral infection, trauma, cerebrovascular disease, toxic/metabolic disturbances, and childhood febrile seizures. Mortality is ~10% (due to underlying pathology). Although seizures typically start as generalized tonic/clonic, these features may gradually diminish, making diagnosis difficult (coma with virtually no motor evidence of seizure, eg minimal twitching of ocular muscles only). Complications include hypoglycaemia, pulmonary hypertension, and pulmonary oedema, and precipitous ↑ ICP can also occur.

*Treatment of status epilepticus*

- Establish a clear airway (a nasopharyngeal airway may help).
- Give $O_2$ as needed.
- Monitor ECG, $SpO_2$, T°, pulse rate, and BP.
- Obtain IV access; check BMG and correct hypoglycaemia if present (50mL of 20% glucose IV).
- Give lorazepam 4mg IV slowly into a large vein (diazepam 10mg is an alternative). Repeat IV lorazepam 4mg slowly after 10min if seizures continue.
- Buccal midazolam 10mg (can be repeated once) or rectal diazepam solution 10–20mg (can be repeated up to a total of 30mg) are alternatives if there is no venous access.
- If alcohol abuse or malnutrition is suspected, give slow IVI thiamine in the form of Pabrinex® two pairs of ampoules in 100mL of 0.9% saline (this occasionally causes anaphylaxis; be prepared to treat—see *BNF*).
- Consider the possibility of pregnancy-related fits (eclampsia) in women of child-bearing age and treat accordingly (with IV magnesium sulfate—as outlined in ➲ Eclampsia, p. 611).
- Check ABG and save blood for cultures, FBC, U&E, glucose, $Ca^{2+}$, $Mg^{2+}$, LFTs, clotting, and drug levels (and toxicology screen if poisoning/overdose is suspected).
- Search for features of injury (especially head injury) and infection (look for a rash).
- If seizures continue despite above therapy, call ICU and consider the use of phenytoin (20mg/kg IV, up to a max of 2g, at a rate of 50mg/min), with ECG monitoring, or fosphenytoin (20mg/kg phenytoin equivalent IV, <150mg/min). A 70kg patient would require 1400mg phenytoin equivalent of fosphenytoin (28mL Pro-Epanutin®) diluted in 100mL of 0.9% saline or 5% glucose, given over 10–15min.
- After 30min, contact ICU and proceed without delay to rapid sequence induction (RSI) (ideally with thiopental) and tracheal intubation, and continue anticonvulsant medication.

# Hypoglycaemia

Hypoglycaemia can mimic any neurological presentation, including coma, seizures, acute confusion, or isolated hemiparesis.

►► *Always exclude hypoglycaemia in any patient with coma, altered behaviour, and neurological symptoms or signs.*

Plasma glucose is normally maintained at 3.6–5.8mmol/L. Cognitive function deteriorates at levels of <3.0mmol/L, but symptoms are uncommon at levels of >2.5mmol/L. In diabetics, however, the threshold for symptoms can be very variable. Hypoglycaemia is potentially fatal and accounts for 2.4% of deaths in patients with type 1 diabetes. Even mild episodes aggravate pre-existing microvascular complications and lead to cumulative brain damage.

## Causes

In patients with diabetes, the most common cause is a relative imbalance of administered vs required insulin or oral hypoglycaemic drug. This may result from undue or unforeseen exertion, insufficient or delayed food intake, and excessive insulin administration (due to time, dose, or type of insulin). Other causes are:

- Alcohol (in addition to alcohol directly causing hypoglycaemia, the features of hypoglycaemia may be mistaken for alcohol intoxication or withdrawal).
- Addison's disease.
- Pituitary insufficiency.
- Post-gastric surgery.
- Liver failure.
- Malaria.
- Insulinomas.
- Extra-pancreatic tumours.
- Attempted suicide or homicide with large doses of insulin or oral hypoglycaemic drug.

## Symptoms and signs

*Common features* Sweating, pallor, tachycardia, hunger, trembling, altered mental state or loss of consciousness, irritability, irrational or violent behaviour, fitting, focal neurological deficit (eg hemiplegia). Look for MedicAlert bracelet/chain.

## Diagnosis

Check venous or capillary blood with glucose oxidase strip (BMG). If <3.0mmol/L, take a venous sample for a formal blood glucose level, but *give treatment* without waiting for the result. Take appropriate samples if overdose of insulin, oral hypoglycaemic agent, or other drugs is suspected.

## Treatment

This depends upon the conscious state and degree of co-operation of the patient. Choose the appropriate option from the following:

- A fast-acting oral carbohydrate 5–15g (eg Lucozade®, sugar lumps, Dextrosol®, followed by biscuits and milk).
- *Glucagon 1mg*: SC, IM, or IV. Can be administered by relatives, by ambulance crew, and when venous access is difficult. Glucagon is not suitable for treatment of hypoglycaemia due to sulfonylurea drugs, liver failure, or in chronic alcoholism (as there may be little liver glycogen available for mobilization).
- Glucose 10% solution 50mL IV, repeated at 1–2min intervals until the patient is fully conscious or 250mL (25g) has been given.
- Glucose 50% solution (25–50mL IV) is hypertonic, liable to damage veins, and no more effective than glucose 10%. If glucose 50% is used, give it into a large vein and follow with a saline flush.
- The time taken for return of consciousness and the incidence of nausea, vomiting, and other adverse effects are similar for IV glucagon and glucose.

*Persistence of an altered conscious level* suggests another underlying pathology (eg stroke) or may reflect the development of cerebral oedema due to hypoglycaemia, which has high mortality. Maintain plasma glucose at 7–11mmol/L; contact ICU, and consider mannitol and/or dexamethasone. Arrange urgent investigation (eg CT scan) and search for other causes of altered consciousness.

*Overdose* Glucose infusions may be needed for 24hr or longer after poisoning with insulin or an oral hypoglycaemic drug, depending upon exactly what and how much has been taken. Hypokalaemia may be a problem. Block excision of the injection site has been used as successful treatment for insulin overdose. Octreotide may be helpful in recurrent hypoglycaemia due to overdose of a sulfonylurea drug (see ➲ Sulfonylurea poisoning, p. 205).

## Discharge

90% of patients fully recover in 20min. Provided that the cause for the episode has been identified and fully corrected, it is reasonable to discharge the patient after observation in the ED, with appropriate follow-up.
Arrange follow-up, having considered the following:

- Why did this episode occur?
- Has there been a recent change of regimen, other drugs, alcohol, etc.?
- Is the patient developing hypoglycaemic unawareness or autonomic dysfunction?

If the patient is a driver, advise them to inform the Driver and Vehicle Licensing Agency (DVLA) of the hypoglycaemic episode.

# Hyperglycaemic crises

*Diabetic ketoacidosis (DKA)* is caused by absolute or relative ↓ insulin levels. Plasma glucose ↑ causes an osmotic diuresis, with $Na^+$ and water loss (up to 8–10L), hypotension, hypoperfusion, and shock. Normal compensatory hormonal mechanisms are overwhelmed and lead to ↑ lipolysis. In the absence of insulin, this results in the production of non-esterified fatty acids, which are oxidized in the liver to ketones.

Younger undiagnosed patients with diabetes often present with *DKA* developing over 1–3 days. Plasma glucose levels may not be grossly ↑; euglycaemic ketoacidosis can occur. Urinalysis demonstrates ketonuria.

*Hyperosmolar hyperglycaemic state (HHS)* is caused by intercurrent illness, inadequate diabetic therapy, and dehydration. It develops over days/weeks and is more common in the elderly. HHS is characterized by ↑ glucose levels (>30mmol/L), ↑ blood osmolality, and a lack of urinary ketones. Mortality is ~5–10% but may be even higher in the elderly.

## Causes

Think of the four '*I*'s separately or (often) in combination:

- *Infection:* common primary foci are the urinary tract, respiratory tract, and skin.
- *Infarction:* myocardial, stroke, GI tract, peripheral vasculature.
- *Insufficient insulin*.
- *Intercurrent illness*: many underlying conditions precipitate or aggravate DKA and HHS.

## Clinical features

Hyperglycaemic crisis may present in various ways. Some of the following are usually present:

- *Signs of dehydration:* thirst, polydipsia, polyuria, ↓ skin turgor, dry mouth, hypotension, tachycardia.
- *GI symptoms:* are common in DKA, with nausea, vomiting, and abdominal pain. This can be severe and mimic an 'acute surgical abdomen'.
- *Hyperventilation* (respiratory compensation for metabolic acidosis), with deep rapid breathing (Kussmaul respiration) and the smell of acetone on the breath, is pathognomonic of DKA.
- *True coma* is uncommon, but altered conscious states and/or focal neurological deficits (which may correct with treatment) are seen particularly in older patients with HHS.

## Diagnosis and investigations

Aim to confirm the diagnosis and search for possible underlying cause(s):

- Check BMG and test the urine for glucose and ketones.
- Send blood for U&E, blood glucose, creatinine, and osmolality (or calculate it): mOsm/L= (2 × $Na^+$) + glucose (mmol/L) + urea (mmol/L).
- Check ABG (look for metabolic acidosis ± respiratory compensation).
- FBC and CXR (to search for pneumonia).
- ECG and cardiac monitoring (look for evidence of hyper-/hypokalaemia).
- Blood cultures and, if appropriate, throat or wound swabs.
- Urine/sputum microscopy and culture.

## Treatment of DKA

- If altered consciousness/coma, open/maintain a patent airway.
- Give $O_2$ by mask, as required. Consider the possible need for GA and IPPV for coma ± severe shock.
- Commence *IVI* with 0.9% saline. Give 1000mL of 0.9% saline over 0.5–1hr, then 500mL/hr for the next 2–3hr. Persistent hypotension may require ↑ in infusion rate and/or colloid administration. Avoid over-rapid infusion with the risks of pulmonary oedema and ARDS, especially in the elderly and patients with IHD.
- *Insulin*: start an infusion of soluble insulin after IV fluids have started using an IV pump or a paediatric burette at 0.1U/kg/hr (typically 6U/hr). Check blood glucose and ketone levels every hour initially—aim for blood glucose to drop by at least 3mmol/L/hr, and blood ketone by at least 0.5mmol/L/hr. Continue insulin infusion until blood ketone is <0.3mmol/L and blood pH is >7.3.
- When plasma glucose is <14mmol/L, add 10% glucose IVI at a rate of 125mL/hr (through a large vein) to help ketone clearance and acid–base state.
- *Electrolyte balance*: although total body $K^+$ is low, plasma $K^+$ may be normal, ↑, or ↓. With treatment, $K^+$ enters cells and plasma levels ↓—therefore, unless initial $K^+$ levels are >5.5mmol/L, give 20mmol/hr of potassium chloride (KCl), monitor ECG, and check $K^+$ levels hourly. Despite the presence of metabolic acidosis, do not give sodium bicarbonate. Other electrolytes such as $Ca^{2+}$, $Mg^{2+}$, and phosphate ($PO_4^{2-}$) are commonly disturbed but rarely need emergency correction.
- Consider an NG tube to ↓ the risk of gastric dilation and aspiration.
- Monitor urine output (most accurate with a urinary catheter).
- Consider a central venous catheter to monitor CVP to guide treatment in the elderly or severe illness.
- Arrange admission to ICU, HDU, or acute medical admissions unit.

## Other aspects of treatment

*Signs of infection* Are often masked. T° is rarely ↑, and ↑ WCC may only reflect ketonaemia. If in doubt, treat with a broad-spectrum antibiotic.

*Over-rapid fluid replacement* Can cause cardiac failure, cerebral oedema, and ARDS, especially in patients with underlying cardiac disease or the elderly. CVP monitoring may be needed.

*Clotting* Hyperglycaemia causes a hypercoagulable state—DVT or PE may occur. Administer prophylactic anticoagulation with LMWH in DKA or hyperosmolar states.

*Treat HHS* with IV 0.9% saline. Do not start insulin unless significant ketonaemia, or glucose does not fall with fluid therapy. Seek advice before starting insulin as it risks cardiovascular collapse.

# Sodium derangements

Abnormal $Na^+$ states can occur with hypervolaemia, euvolaemia, or hypovolaemia, depending on the underlying pathophysiological process.

## Hyponatraemia

*Causes* Include excessive fluid loss replaced by hypotonic fluids (diarrhoea, burns, prolonged exercise such as marathon running), polydipsia, ecstasy ingestion, syndrome of inappropriate antidiuretic (ADH) secretion, nephrotic syndrome, renal impairment, hepatic cirrhosis, cardiac failure, and many prescription drugs (including diuretics, heparin, and ACE inhibitors).

*Treatment of acute hyponatraemia (<24hr duration)* Those with mild symptoms can be effectively treated by fluid restriction. Patients who present with seizures or signs of ↑ ICP are at risk of death and require more aggressive treatment. Serum $Na^+$ <120mmol/L is associated with risk of brain herniation. Give up to 200mL of 2.7% saline IV over 30min and recheck serum $Na^+$ levels.

*Treatment of chronic hyponatraemia (>24hr duration)* Is associated with central pontine myelinolysis, particularly in patients with low $K^+$ levels or alcoholic patients. Chronic hyponatraemia should be corrected no faster than 10mmol/L in 24hr. Treat the underlying cause. This may be as simple as discontinuing a diuretic. Patients with cardiac failure, cirrhosis, or nephrotic syndrome (hypervolaemic patients) should be fluid-restricted. Severe hyponatraemia in association with seizures or ↓ GCS may be cautiously treated with hypertonic saline (200mL of 2.7% saline over 30min and recheck serum $Na^+$). Aim to ↑ serum $Na^+$ by no more than 5mmol/L using this method.

## Hypernatraemia

Usually reflects a loss of water in excess of loss of $Na^+$.

*Causes* Include diabetes insipidus (lack of ADH or lack of renal response to ADH), diarrhoea, vomiting, diuretics, hypertonic saline, sodium bicarbonate administration, or Cushing's syndrome.

*Treatment* Do not correct $Na^+$ concentration faster than 1mmol/L/hr. Use 0.9% saline to correct hypovolaemia (patients who have tachycardia, hypotension, or postural hypotension). Once the patient is euvolaemic, use an infusion of 0.45% saline or 5% glucose. The *free water deficit* can be calculated using the formula:

$$\text{Free water deficit (L)} = 0.6 \times \text{weight (kg)} \times [(\text{serum } Na^+ / 140) - 1]$$

Replace the deficit over 48hr (in addition to normal maintenance fluids). Check serum $Na^+$ after 2–3hr to monitor correction rate.

*Complications* Include seizures, subdural and intracerebral haemorrhages, ischaemic stroke, and dural sinus thrombosis. Rapid correction of $Na^+$ levels (particularly in chronic hypernatraemia) can cause cerebral oedema and further neurological complications.

# Addisonian crisis

Acute adrenocortical insufficiency is rare and easily missed. By far, the most common cause is sudden withdrawal of chronic steroid therapy (deliberately or inadvertently). An Addisonian crisis may also be precipitated in these patients by intercurrent injury, infection, or stress—↑ steroid requirement. 80% of Addison's disease in the UK is idiopathic (autoimmune) and may be associated with Graves' disease, Hashimoto's thyroiditis, type 1 diabetes mellitus, pernicious anaemia, hypoparathyroidism, and ovarian failure. Other causes include TB, fungal infections, metastatic disease, congenital adrenal hyperplasia, drugs (eg metyrapone or cytotoxic agents), haemorrhage into the adrenal glands occurring as a complication of anticoagulation, or meningococcal septicaemia (Waterhouse–Friderichsen syndrome). Look for a MedicAlert bracelet indicating that the patient is taking steroids.

## Precipitating factors

Infection, trauma, MI, cerebral infarction, asthma, hypothermia, alcohol, pregnancy, exogenous steroid withdrawal or reduction.

## Clinical features

Addison's disease frequently has an insidious onset with weakness, apathy, anorexia, weight loss, abdominal pain (which may be severe enough to mimic an acute abdomen), and oligomenorrhoea. In crisis, the main features may be shock (tachycardia, peripheral vasoconstriction, severe postural hypotension occasionally with syncope, oliguria, profound muscle weakness, confusion, altered consciousness leading to coma) and hypoglycaemia. Chronic features of Addison's disease are: areas of vitiligo and hyperpigmentation in the palmar creases, buccal mucosa, areolae, scars, and axillae.

## Investigation

Obtain IV access, and send blood to check for hyperkalaemia, hyponatraemia, hypoglycaemia, uraemia, mild acidosis, hypercalcaemia, and eosinophilia which may be present. Also, take blood for cortisol (10mL in a heparinized tube) and adrenocorticotrophic hormone (ACTH) if possible—contact the biochemistry lab to warn them that these tests will be required. Take blood cultures, urine cultures, and sputum for culture and sensitivity.

## Management

- If an Addisonian crisis is suspected, take appropriate blood samples, but start treatment without waiting for results.
- If features of haemodynamic compromise are present, commence volume replacement with IV 0.9% saline if shocked.
- Give hydrocortisone sodium succinate 100mg IV stat.
- Treat hypoglycaemia with 50mL of 10% glucose IV (repeated if necessary).
- If infection is suspected as a precipitating cause, consider giving broad-spectrum antibiotics.
- Refer for admission.

# Thyrotoxic crisis

A rare condition, occurring in 1–2% of patients with established hyperthyroidism (usually toxic diffuse goitre—'Graves' disease'). Mortality is significant (~10%).

## Causes

It is often precipitated by a physiological stressor:

- Premature or inappropriate cessation of anti-thyroid therapy.
- Recent surgery or radio-iodine treatment.
- Intercurrent infection (especially chest infection).
- Trauma.
- Emotional stress.
- DKA, hyperosmolar diabetic crisis, insulin-induced hypoglycaemia.
- Thyroid hormone overdose.
- Pre-eclampsia.

## Clinical features

Onset may be sudden with features of hyperthyroidism and adrenergic overactivity. Fever and cardiovascular and neurological symptoms are common. Weight loss, ↑ appetite, tremor, irritability, emotional lability, heat intolerance, sweating, itch, oligomenorrhoea, agitation, anxiety, confusion, coma, palpitations, tachycardia, AF (rarely, complete heart block). It may mimic an 'acute abdomen', with abdominal pain, diarrhoea, and vomiting.

## Differential diagnosis

Includes acute pulmonary oedema, neuroleptic malignant syndrome, septic shock, anticholinergic or sympathomimetic overdose, drug withdrawal, or acute anxiety states.

## Investigation

- U&E, BMG and blood glucose, $Ca^{2+}$ (hypercalcaemia occurs in ~10%).
- FBC, differential WCC, coagulation screen.
- *Screen for infection*: mid-stream urine (MSU), blood cultures, sputum.
- Thyroxine ($T_4$) and tri-iodothyronine ($T_3$) (for later analysis), thyroid-stimulating hormone (TSH).
- CXR (searching for pulmonary infection or congestive heart failure).
- ECG (looking for arrhythmias).

## Treatment

- Manage the airway and give $O_2$ if indicated.
- Obtain IV access and commence IVI 0.9% saline (initially 500mL 4-hourly).
- Give propranolol (1mg slow IV over 1 min or 60mg PO) to ↓ heart rate.
- Give hydrocortisone 100mg IV.
- If sedation is required, give small titrated amounts of benzodiazepine (eg diazepam 5–20mg PO/IV) or haloperidol.
- Give broad-spectrum antibiotic if infection is suspected.
- Consider cooling measures in hyperthermia.
- Refer for admission (consider admission to ICU).
- Once admitted, carbimazole will normally be given with iodine.
- Do not give aspirin (this can exacerbate the clinical problem by displacing thyroxine from thyroid-binding globulin).

# Acute kidney injury

## Background

AKI is diagnosed by:

- A serum creatinine rise ≥26 micromoles/L over 48hr.
- A serum creatinine rise ≥50% over 7 days.
- ↓ urine output of <0.5mL/kg/hr for >6hr.

AKI may be present if creatinine is high, even if the above criteria are not met. If it is unclear whether a patient has worsening of their chronic kidney disease (CKD) or AKI, treat as the latter.

## Causes

- Pre-renal: hypovolaemia (haemorrhage, burns, pancreatitis), cardiogenic shock, sepsis, renal vasoconstriction (drugs).
- Renal: glomerulonephritis, vasculitis, acute tubular necrosis, interstitial nephritis.
- Post-renal: obstruction within renal system (calculi, stricture, tumour) or from outside (prostatic hypertrophy, pelvic malignancy, retroperitoneal fibrosis).

## History and examination

Consider AKI in anyone with acute illness aged >65y, a history of kidney disease, those on nephrotoxic drugs, and patients who are systemically unwell (eg with sepsis). Look for signs of dehydration, diarrhoea and vomiting, and ↓ urine output.

## Investigation

- U&E, VBG, and other bloods, depending on underlying cause (eg CK in suspected rhabdomyolysis).
- Urinalysis (blood and protein may indicate glomerular disease)—if ↓ urine output, consider a bladder scan and catheterization.
- If a catheter is already *in situ*, consider flushing or replacing it.
- Perform an ECG (looking especially for changes suggesting hyperkalaemia—see ➲ Hyperkalaemia, pp. 170–1).

## Management

- Treat the underlying cause.
- Assess hydration status and commence a fluid balance chart. If dehydrated, commence IV fluid therapy with a crystalloid. Stop all nephrotoxic drugs (eg NSAIDs and ACE inhibitors).
- Treat hyperkalaemia (see ➲ Hyperkalaemia, pp. 170–1).
- Refer to the renal team.
- Indications for haemodialysis include intractable hyperkalaemia, severe acidosis, fluid overload, toxin removal, and severe uraemic symptoms. If severely unwell, haemofiltration is more appropriate—refer to ICU.

# Chronic kidney disease

Patients with established CKD are likely to be very well known to the hospital. Review old notes and recent blood results, and liaise early with inpatient specialist teams.

## Established CKD (not on dialysis)

Patients with mild CKD (GFR >40–100mL/min) are unlikely to have specific problems related to their underlying renal failure. With GFR of <40mL/min, and especially if GFR is <10mL/min, complications may influence presentation and treatment. These patients are prone to pathological fractures.

- *Secondary hyperparathyroidism and osteomalacia* (lack of active vitamin D) occur in moderate CKD. In severe CKD, aluminium bone disease and β2-microglobulin-related amyloidosis may be associated with pathological fractures.
- '*Pseudogout*' due to high $Ca^{2+}/PO_4^-$ production and twitching/tetany due to hypocalcaemia may occur.

*Other problems include*

- *Defective regulation of extracellular fluid volume*: there is an ↑ risk of fluid depletion in moderate CKD and fluid retention in severe CKD. High-dose diuretics may be required in severe disease—the combination of furosemide and metolazone may be effective, even with very low GFRs.
- *Hyperkalaemia*: most patients preserve $K^+$ balance but cannot deal with sudden $K^+$ loads (eg dietary, tissue damage/catabolism, GI bleed). Associated ↓ $Ca^{2+}$ compounds the cardiac effects. Plasma $K^+$ may ↑ very quickly, so monitor ECG and check $K^+$ frequently.
- *Hypertension*: often severe and resistant, with an ↑ incidence of accelerated phase. Ciclosporin and erythropoietin ↑ BP and can precipitate hypertensive encephalopathy.
- *Drug effects:* drugs may accumulate (eg opioids, aciclovir, some antibiotics), worsen renal failure (eg NSAIDs, ACE inhibitors, which ↓ renal perfusion), and cause hyperkalaemia (eg $K^+$-sparing diuretics, ACE inhibitors, NSAIDs).
- *Infections:* impaired white blood cell (WBC) function, with ↑ risk of severe infection, and features of infection (eg pain, fever) may be masked by the relative immunocompromised state.
- *Bleeding:* platelet function is impaired.
- *Pericarditis:* a sign of severe chronic renal failure (CRF), indicating the need for dialysis.
- *Neurological dysfunction:* usually a sign of severe uraemia—convulsions and/or altered conscious state indicate a global metabolic disturbance.

## Haemodialysis patients' problems

*Pulmonary oedema* Usually occurs shortly before the next dialysis session and may reflect fluid overload due to non-compliance with diet and fluid restriction. Most are virtually anuric, so diuretics are ineffective. Get the patient on dialysis without delay. Whilst this is being arranged, give $O_2$ as needed and sublingual (SL), buccal, or IV nitrates.

*Pre-dialysis hyperkalaemia* May present with neuromuscular symptoms (eg muscle spasms, weakness, paralysis, paraesthesiae) or arrhythmias, including cardiac arrest. Standard treatment (see ➔ Hyperkalaemia, pp. 170–1) can buy time whilst emergency dialysis is arranged. When giving glucose/insulin, give 6U of insulin at most (there is a risk of late hypoglycaemia, since insulin half-life will be ↑).

*Complications of vascular access* Arteriovenous fistulae are a dialysis patient's lifeline—never occlude the limb with BP cuffs or tourniquets. Do not use for vascular access unless a life-threatening emergency. Acute shunt thrombosis (loss of palpable thrill, often local pain/redness) is a vascular emergency. Arteriovenous fistulae and central lines are common infection sources (usually staphylococcal), often with no overt external abnormality, but presenting with acute 'viral illness' symptoms.

## Continuous ambulatory peritoneal dialysis

*Bacterial peritonitis* Occurs every 12–18 patient-months. Features are cloudy drained dialysate bags, abdominal pain, and peritonism. Systemic sepsis is usually absent or minimal. Staphylococci are the most common organisms. Suspect an underlying surgical cause (most often diverticular abscess) if Gram –ve organisms or anaerobes present in drainage fluid, and particularly if >1 type of organism is found on microscopy or culture.

*Hyperglycaemia* Diabetic patients on continuous ambulatory peritoneal dialysis can develop acute severe (usually non-ketotic) hyperglycaemia, related to high dialysate glucose concentrations (80–140mmol/L).

*Hernias* of all types, leakage of dialysate into the abdominal wall or the pleural cavity, and scrotal swelling (open processus vaginalis) may occur.

## Transplant patients

Contact the transplant team whenever any transplant patient presents to the ED. They will know the patient well and will advise about drug therapy and intercurrent problems, and help with follow-up.

*Acute rejection* Signs include pain, tenderness, and swelling over graft, ↓ urine output, fever, systemic upset, and biochemical deterioration. Often indistinguishable from acute bacterial infection—if in doubt, treat for both, pending results of further testing by specialists (renal biopsy, blood and urine cultures).

*Infections* May be opportunistic, whilst 'conventional' infections are unduly severe, with response modulated by steroids.

*Poor wound healing, avascular necrosis, and pathological fractures* May be caused by steroids.

# Urinary tract infection

The urinary tract is normally bacteriologically sterile. Urine infection is present if $>10^5$ colony-forming units are present per mL of urine. Except at the extremes of age, UTIs are much more common in ♀ due to the shorter urethral length. Most UTIs occur because of organisms invading the bladder via the urethra. Proximal invasion via the ureter may result in acute or chronic pyelonephritis, particularly if anatomical derangement exists with impaired ureteric or bladder emptying. In both sexes, an underlying structural abnormality ↑ UTI risk. Blood-borne spread of infection to the urinary tract can occur (eg in bacterial endocarditis or systemic Gram –ve infection). UTI is usually caused by a single organism. The most common organism (90%) at all ages is *Escherichia coli*. *Proteus, Klebsiella*, and saprophytic staphylococci account for most of the remainder in adults. Other organisms (eg *Pseudomonas*) more commonly cause UTI in hospitalized patients or following instrumentation.

## Presentation

*Lower UTI (cystitis)*

Dysuria, frequency, haematuria, suprapubic discomfort, urgency, burning, cloudy urine with an offensive smell. Patients with acute urethral syndrome have identical symptoms, but –ve urine culture.

*Upper UTI (acute pyelonephritis)*

Often systemically unwell with malaise, fever, loin and/or back pain, vomiting, rigors, and occasionally Gram –ve septicaemia. Diagnose pyelonephritis if there is evidence of UTI with loin pain and T° >38°C.

## Investigations

Traditional investigations are:

- *Reagent strip (dipstix) urinalysis* may show haematuria, proteinuria, and +ve nitrite and leucocyte esterase tests. A patient with clear urine, –ve on dipstix testing, is extremely unlikely to have a UTI. False +ve results may be secondary to urinary tract tumours or excessive exercise.
  A false –ve nitrite test may reflect pathogens that do not convert dietary nitrates to nitrites.
- *Urine microscopy* may show leucocytes (>100/mL correlates well with infection but may be due to contamination or other urinary tract pathology). Red blood cells (RBCs) are commonly seen on microscopy but, in isolation, have a low degree of sensitivity or specificity for UTI. Underlying renal pathology is suggested by finding urinary crystals, RBCs, or granular casts.
- *MSU* for culture and sensitivity. Transport the sample to the laboratory without delay to ensure that bacterial overgrowth does not artificially ↑ the count. Dipslides dipped into freshly passed urine and transported in a plastic container to the laboratory are an alternative.

Do not routinely perform urine microscopy and culture for women with uncomplicated UTIs, but do perform them if there is haematuria, impaired renal function, immunosuppression, or abnormality of the renal tract.

## Treatment

*Lower UTI*

- Aim to discharge women with uncomplicated lower UTIs with *antibiotics*. Commence a 3-day course of trimethoprim (200mg bd) or nitrofurantoin (50mg qds). Provide *advice* regarding fluid intake, no 'holding on', and voiding after intercourse. (*Note:* urinary alkalinization renders nitrofurantoin ineffective.)
- Consider a 5 to 10 day course for women with impaired renal function, immunosuppression, or abnormality of the renal tract. Advise the patient to see her GP for review and MSU result.
- In pregnancy, treat symptomatic bacteriuria (eg amoxicillin 250mg tds for 7 days or cefalexin 500mg tds for 7 days), and arrange GP follow-up for a repeat MSU. Also treat with antibiotics a pregnant woman who is asymptomatic and has a +ve urine dipstick for leucocytes and nitrites, again advising GP follow-up. Remember that trimethoprim is contraindicated in the first trimester and nitrofurantoin is contraindicated in the third trimester.
- Do not give antibiotics to elderly men and women with asymptomatic bacteriuria, unless they show signs of being unwell.
- Treat men with symptoms of a lower UTI with a 7-day course of nitrofurantoin (50mg qds) or trimethoprim (200mg bd). Consider the possibility of alternative diagnoses: chlamydial infection, prostatitis, and epididymitis.

*Upper UTI*

- Assess and treat for severe sepsis (see ➔ Shock, pp. 64–5). Admit if there is evidence of sepsis and/or systemic symptoms, dehydration, or no response to oral antibiotics. Provide parenteral antibiotics (eg gentamicin + amoxicillin IV), fluid replacement, and analgesia.

*Older patients with suspected UTI*

UTI is likely if an elderly person without obvious infection elsewhere has two or more of: dysuria, urgency, frequency, urinary incontinence, rigors, flank or suprapubic pain, frank haematuria, and new confusion (or worsening of pre-existing confusion). Do not use urinalysis to diagnose UTI, but if clinically suspected, send an MSU. Treat with antibiotics (eg trimethoprim or nitrofurantoin). Consider analgesia and the need to admit to hospital (eg signs of sepsis and/or fever with rigors). If there is an indwelling catheter, remove and replace it. (*Note*: do not treat catheterized patients with asymptomatic bacteriuria with antibiotics.)

(See https://www.sign.ac.uk)

# Hyperkalaemia

Hyperkalaemia is classified as follows: mild ($K^+$ 5.5–6.0mmol/L), moderate ($K^+$ 6.1–6.9mmol/L), or severe ($K^+$ >7.0mmol/L).

## Causes

- Spurious: sample haemolysed or taken from limb with IVI containing $K^+$.
- ↓ renal excretion: AKI, patients with CKD or on dialysis with $K^+$ load, $K^+$ sparing diuretics (eg spironolactone, amiloride).
- Cell injury: crush injury and other causes of rhabdomyolysis, burns, tumour cell necrosis, massive or incompatible blood transfusion.
- $K^+$ cellular shifts: acidosis from any cause (eg DKA), drugs (suxamethonium, β-blockers).
- Hyperaldosteronism: Addison's disease, drug-induced (NSAIDs, ACE inhibitors).

## Clinical features

There may be muscle weakness/cramps, paraesthesiae, hypotonia, focal neurological deficits. Dangerous hyperkalaemia may be asymptomatic.

## ECG changes

ECG changes typically progress as hyperkalaemia worsens, as follows (see Fig. 3.35):

- Peaked T waves.
- Small, broad, or absent P waves.
- Widening QRS complex.
- Sinusoidal ('sine wave' pattern) QRST.
- AV dissociation or VT/VF.

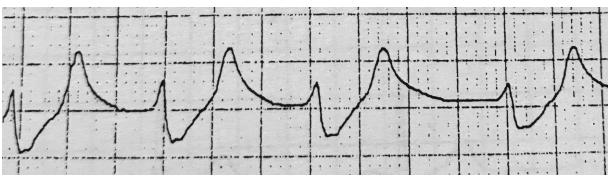

**Fig. 3.35** ECG strip of severe hyperkalaemia: peaked T waves, absent P waves, and widened QRS.

## Management of hyperkalaemic cardiac arrest

If a patient in cardiac arrest is known to have hyperkalaemia, follow the standard ALS guidelines (see ➲ Cardiac arrest, p. 48), plus one or more of the following:

- Give 10mL of 10% calcium chloride IV by rapid bolus injection.
- Consider giving 10U of short-acting insulin + 100mL of 50% glucose rapidly IV.
- If there is severe acidosis, give 50mL of 8.4% sodium bicarbonate rapidly IV.
- Consider haemodialysis for cardiac arrest induced by hyperkalaemia which is resistant to medical treatment.

### Management of severe hyperkalaemia

Urgent treatment is needed if $K^+$ is >6.5mmol/L, unless this is a spurious and incorrect result. If $K^+$ is reported as >6.5mmol/L, obtain venous access; monitor and review the ECG. If there are no ECG signs of hyperkalaemia, take another blood sample for U&E, with care to avoid haemolysis, and a heparinized sample to measure $K^+$ on a blood gas machine.

Start treatment immediately if there are ECG changes of hyperkalaemia:

- Give 10mL of 10% calcium chloride slowly IV (over 5min). This does not lower $K^+$ but antagonizes cardiac membrane excitability. Hypercalcaemia may possibly potentiate toxicity in patients on digoxin, so give as an IVI over 30min in these patients.
- Give 10U of short-acting human soluble insulin (eg Actrapid®) with 50mL of 50% glucose IV. This helps ↑ cellular uptake of $K^+$, lowering serum levels by up to 1mmol/L within 1hr and lasting up to 4hr.
- Give nebulized salbutamol 5mg, repeated once as necessary. This will lower $K^+$ in most patients, acting in ~30min.
- Correct volume deficits/acidosis with IV fluids and isotonic (1.26%) sodium bicarbonate or aliquots (25–50mL) of 8.4%. Beware fluid overload/osmolar effects, especially in dialysis patients.
- Correct the underlying cause, if possible (eg steroid therapy for Addison's disease).
- Contact the nephrology team urgently for patients with acute or chronic renal failure, as emergency dialysis may be needed.

*Hyperkalaemia in children* See ➲ Hyperkalaemia, p. 714.

### Management of moderate hyperkalaemia

Provided that the result is not spurious, a $K^+$ level of 6–6.5mmol/L may be regarded as 'moderately' severe hyperkalaemia.

- Obtain venous access and monitor ECG.
- If there are ECG changes, treat as for severe elevation (as outlined previously).
- If there are no ECG changes, give 10U of short-acting human soluble insulin with 50mL of 50% glucose IV over 15–30min.
- Look for and treat the underlying cause and consider diuretics (eg furosemide 1mg/kg IV slowly) and dialysis.

### Management of mild hyperkalaemia

$K^+$ level of 5.5–6mmol/L. Treat the underlying cause and any associated hypovolaemia. Discuss the need for specific intervention (diuretic, dialysis) with the medical team.

# Hypokalaemia

Defined as $K^+$ <3.5mmol/L; it is relatively common. Moderate hypokalaemia may result in lethargy, weakness, and leg cramps. In severe cases ($K^+$ <2.5mmol/L), rhabdomyolysis and respiratory difficulties may occur.

*ECG changes* include prominent U waves and flattened T waves (see Fig. 3.36). The U waves can be mistaken for T waves and so give the erroneous impression of a long QT interval.

## Treatment

In most instances, aim to replace $K^+$ gradually. The maximum recommended IVI rate of $K^+$ is 20mmol/hr. Restrict more rapid rates of IVI (eg 20mmol in 20–30min) to those patients who have unstable arrhythmias when cardiac arrest is imminent (obtain senior/expert advice). Ensure cardiac monitoring occurs during any $K^+$ IVI.

### *Associated magnesium deficiency*

Many patients with $K^+$ deficiency are also $Mg^{2+}$ deficient. Consider replacing $Mg^{2+}$ in those patients who have severe hypokalaemia.

(See also https://www.resus.org.uk)

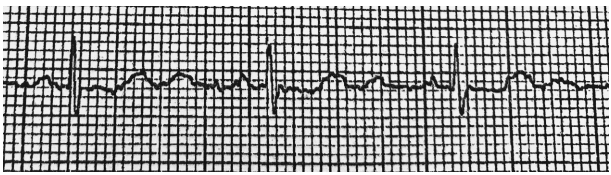

**Fig. 3.36** ECG strip of hypokalaemia with prominent U waves.

# Porphyria

Porphyrias are haem biosynthesis disorders in which enzyme deficiencies cause accumulation of porphyrin and porphyrin precursors. Most cases are hereditary, but abnormal porphyrin metabolism may develop in iron deficiency, alcohol excess, and lead poisoning. Acute porphyrias (acute intermittent porphyria, variegate porphyria, and hereditary coproporphyria) affect ~1 in 10,000 people in the UK. Non-acute porphyrias (eg porphyria cutanea tarda) do not produce acute attacks but cause skin photosensitivity sometimes associated with liver disease.

Attacks of acute porphyria are often caused by drugs: barbiturates, oestrogens, progesterones, sulfonamides, methyldopa, carbamazepine, phenytoin, sulfonylureas, chloramphenicol, tetracyclines, danazol, and some antihistamines. Other precipitants include: alcohol, smoking, dieting, emotional and physical stress, infection, substance misuse, and pregnancy.

## Clinical features of acute porphyria

- Abdominal pain is common and can be severe, with nausea, vomiting, and constipation. Abdominal examination may be normal or there may be mild generalized tenderness.
- Peripheral neuropathy is usually motor, rather than sensory, and may progress to paralysis and respiratory failure.
- Tachycardia, hypertension, and postural hypotension.
- Psychiatric manifestations: agitation, depression, mania, and hallucinations.
- Hyponatraemia due to inappropriate ADH secretion can cause fits or coma.

## Investigation and management of acute porphyria

Look for a MedicAlert bracelet. Review old medical notes.

If an acute attack is suspected, send a fresh urine sample (protected from light) to test for aminolevulinic acid and porphobilinogen. In an attack, urine goes dark red or brown, especially if left exposed to light (due to polymerization of porphobilinogen).

Treat acute attacks supportively (if necessary in ICU). Maintain carbohydrate intake (PO or IV). Control mild pain with paracetamol or aspirin; moderate/severe pain with morphine (± antiemetic). Consider chlorpromazine for agitation, and propranolol to control severe hypertension. Management of status epilepticus is difficult as many anticonvulsants are contraindicated—choose IV diazepam in the first instance. Haem arginate helps some patients with acute crises (take specialist advice).

## Prescribing for patients with porphyria

Many drugs can precipitate attacks, so check with the patient and the *BNF*.

However, the safety of many drugs in porphyria is uncertain and effects vary between patients. If in doubt, obtain specialist advice. In addition to those mentioned earlier, safe drugs appear to be: ibuprofen, penicillin, ciprofloxacin, and bupivacaine.

Data are also available on the Internet at http://www.porphyria.org.uk

# Bleeding disorders: assessment

► *Contact a haematologist whenever treating a patient with a known or suspected bleeding disorder.*

Haemostasis requires co-ordination between the *vascular system, platelets, and coagulation pathways* to limit blood loss from the circulation. Platelets interact with the vascular subendothelium, forming a primary platelet plug, which is strengthened by cross-linked fibrin strands formed via the coagulation cascade to allow restoration of vascular integrity (see Fig. 3.37). The fibrinolytic systems prevent excess clot formation and inappropriate local or generalized thrombosis by promoting lysis of fibrin.

## Recognition of bleeding

Bleeding is expected after trauma, but suspect a bleeding disorder if spontaneous or excess haemorrhage occurs from multiple or uninjured sites into deep tissues and joints, or delayed bleeding occurs (hours/days). Bleeding disorders may be congenital or acquired. Ask about previous bleeding after trauma, dentistry, or surgery and about the family history.

*Congenital disorders* Haemophilia A (factor VIII deficiency), haemophilia B (factor IX deficiency), and von Willebrand's disease. Most adults with a congenital disorder know the nature of it and carry a National Haemophilia card or a MedicAlert bracelet giving details. Many haemophiliacs know more about their required treatment than the ED clinician! They will be registered and known at a haemophilia centre.

*Acquired disorders* May be due to liver disease, uraemia, drug use (ask specifically about aspirin, NSAIDs, warfarin/anticoagulants, alcohol), or unrecognized conditions such as haematological malignancy.

*Hypothermia* From whatever cause—aggravates any bleeding tendency. For example, an INR assay performed at 32°C will be prolonged to the same extent as would occur with a factor IX level of 2.5% of normal. The severity of this may not be recognized merely from standard tests as these are performed at 37°C. (See ➲ Hypothermia: presentation, pp. 264–5.)

*Site of bleeding* Can give a clue as to the abnormality. Platelet problems (usually thrombocytopenia) often present with mucocutaneous bleeding (eg epistaxis, GI, GU, or heavy menstrual bleeding, bruising, purpura, and petechial haemorrhages). Bleeding into joints or potential spaces (eg retroperitoneal) and delayed bleeding are more often due to coagulation factor deficiencies. Patients with mucocutaneous bleeding and haemorrhage into deep spaces may have a combined platelet and coagulation factor abnormality (eg DIC).

## Investigations

*FBC* Remember that in acute bleeds, Hb and Hct values fail to demonstrate the severity of red cell loss as haemodilution takes time. Platelet counts $<100 \times 10^9$/L indicate thrombocytopenia, and those $<20 \times 10^9$/L are associated with a risk of spontaneous bleeding. If platelet function is abnormal (eg with aspirin or clopidogrel), serious bleeding can occur with normal platelet levels.

*INR* Used to monitor anticoagulant control in patients on coumarin drugs. May be prolonged in liver disease. A normal INR makes clinically relevant effects of apixaban or rivaroxaban unlikely.

*Activated partial thromboplastin time (APTT)* Tests components of the intrinsic and common coagulation pathways (essentially all factors, except VII and XIII) but may be normal in the presence of mild deficiency. Used to dose IV unfractionated heparin. A normal APTT makes clinically relevant effects of dabigatran unlikely.

*Anti-Xa assays* Calibrated for apixaban, edoxaban, rivaroxaban, enoxaparin, dalteparin, and tinzaparin—can be used to measure each drug effect. However, these blood tests may not be available in an emergency.

*Dilute thrombin time* Very sensitive to dabigatran and remains elevated (for days), even after the clinical effect of dabigatran has gone.

*Ecarin clotting time* May help in determining the effect of dabigatran.

*Individual factor levels* Can be determined by specific assays, together with inhibitor screening tests for antibodies that can prolong normal clotting.

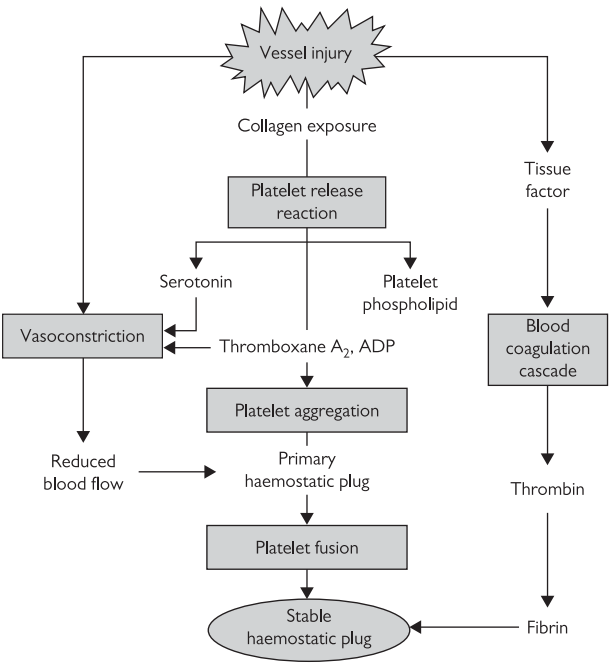


**Fig. 3.37** Reactions involved in haemostasis.

# Bleeding disorders: treatment

## General aspects of treatment

- Liaise with the haematologist for patients with bleeding disorders.
- Perform routine wound/fracture management of patients with bleeding disorders, but consider the need for prior or simultaneous administration of factor concentrates/platelets under haematological guidance.
- Spontaneous or traumatic bleeding into the neck or pharynx may cause rapid airway compromise.
- Always consider intracranial haemorrhage in a patient with headache, neurological symptoms, or minor head trauma.
- Never give IM injections.
- Do not attempt central line placement, except *in extremis*.
- Before giving any drug, check whether it may aggravate the condition or interfere with intercurrent therapy.

## Specific conditions

*Vascular lesions* May be inherited (Ehlers–Danlos syndrome, pseudoxanthoma elasticum, osteogenesis imperfecta, haemorrhagic telangiectasia) or acquired (eg due to steroids, infection such as meningococcaemia, thrombotic thrombocytopenic purpura, vasculitis, scurvy).

*Platelet disorders* Capillary-related mucocutaneous bleeding is common and may occur immediately after injury/surgery (eg dental extractions). The platelet count may be normal or ↓. Acquired thrombocytopenia may be due to drugs, toxins, infections, autoimmune conditions (eg immune thrombocytopenia), DIC, or massive blood transfusion. Abnormal platelet function occurs with uraemia, myeloproliferative disorders, and drugs (eg aspirin).

*Coagulation pathway disorders* Congenital coagulation pathway disorders predominate in ♂. They cause intramuscular or deep soft tissue haematomas. Bleeding onset after injury/surgery may be delayed 2–3 days.

*von Willebrand's disease* The most common congenital bleeding disorder, with von Willebrand (vW) factor and factor VIII deficiency and abnormal platelet function. Clinically, the condition is similar to a platelet disorder, but milder. Bleeding is commonly mucosal (eg epistaxis) and usually treated with desmopressin or factor VIII concentrate (which includes vW factor).

*Haemophilia A* Caused by a lack of functional factor VIII which is needed for clot formation. Often presents with bleeding into deep muscles, large joints, or the urinary tract. Intracranial bleeding is a major cause of death at all ages. Anticipate bleeding up to 3 days after trauma.

Haemophilia A associated with bleeding or potential bleeding is normally treated with factor VIII concentrate (some patients have 'home supplies' and may bring them to hospital). The volume (dose) depends upon the severity of haemophilia of the individual patient and the purpose of treatment (ie prophylaxis or therapy for current bleeding). Mild haemophilia A may also be treated with desmopressin.

*Haemophilia B (Christmas disease)* Involves a deficiency of factor IX activity and is genetically and clinically indistinguishable from haemophilia A, but much less common. It is normally treated with factor IX concentrate.

## Disseminated intravascular coagulation

Patients may present with DIC due to infection (especially Gram –ve sepsis), trauma, malignancy, pregnancy (amniotic fluid embolism, placental abruption, toxaemia, retained products), any cause of shock, incompatible blood transfusion, or massive volume replacement. Following triggering of the coagulation process, consumption of platelets and coagulation factors (particularly fibrinogen, V, VIII, and XIII) occurs, with thrombin formation overwhelming the normal inhibition system, resulting in systemic fibrin deposition (see Fig. 3.38). Activation of the fibrinolytic system results in dissolution of fibrin and release of fibrin degradation products.

*Investigations* Platelet count is usually ↓, INR ↑ and APTT ↑, fibrinogen level ↓, fibrin degradation products ↑.

*Treatment* Is complex and requires control of the primary cause of the DIC to avoid total depletion of clotting factors. Obtain expert advice about replacement therapy with platelets, fresh frozen plasma (FFP), cryoprecipitate, prothrombin complex concentrate, heparin, and red cells (particularly required if the patient is actively bleeding).

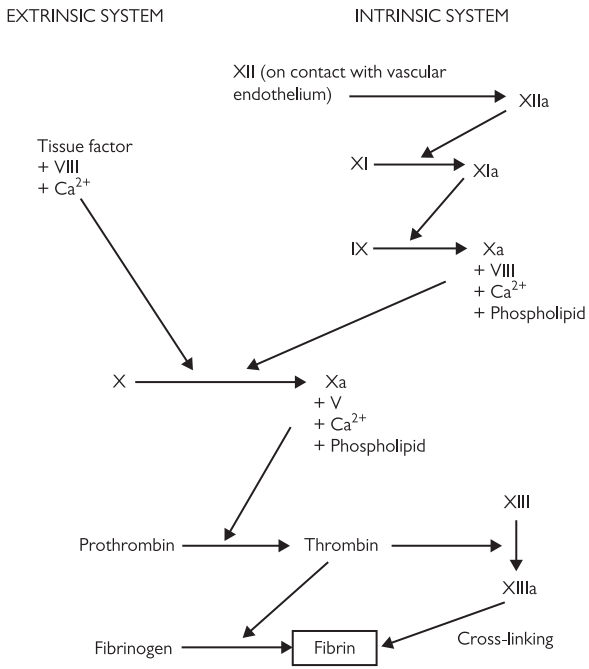


**Fig. 3.38** Coagulation cascade.

# Patients on anticoagulants

## Warfarin

A vitamin K antagonist inhibiting the production of factors II (prothrombin), VII, IX, and X. Expect patients with mechanical prosthetic heart valves to be prescribed warfarin, as this is a contraindication to the newer anticoagulants. Warfarin is also indicated for stroke prevention in AF and in vascular disease. Warfarin treatment cannot be started on its own for the treatment of DVT or PE without heparin (either IV or LMWH), as it initially induces a prothrombotic state. The effect of warfarin (measured by the INR) is influenced by intercurrent illness, liver disease, and changes in diet and/or alcohol consumption and other medications.

## Newer oral anticoagulants

Apixaban, rivaroxaban, and edoxaban are all anti-Xa inhibitors. Dabigatran is a direct thrombin inhibitor. Apixaban and rivaroxaban are indicated for the emergency treatment of DVT and PE (see ➲ Treatment of DVT/PE, p. 125), at a higher dose for the initial treatment period. Dabigatran and edoxaban cannot be started for the treatment of acute DVT or PE until 5–7 days of LMWH has been given. Any of the four drugs may be used for stroke prevention in patients with AF who do not have mechanical valves, as well as extended prophylaxis post-arthroplasty. These drugs are preferred by patients over warfarin as they do not require monitoring with blood tests and are not affected by diet.

Renal and hepatic impairment reduces drug clearance and so may promote bleeding. Newer oral anticoagulants have numerous drug interactions, including with antiepileptic, anti-TB, anti-HIV, antibiotic, and anti-arrhythmic drugs.

## Low-molecular weight heparin

Enoxaparin, tinzaparin, and dalteparin are short-chain heparins which are given SC. They are often used for cancer-associated DVT and PE. They are also used in acute DVT or PE, if warfarin, dabigatran, or edoxaban oral therapy is planned. LMWH is the drug of choice for prophylaxis in medical inpatients. Syringes are designed for patients to self-administer as an outpatient. Contraindicated in severe renal impairment.

## Unfractionated heparin

The anticoagulant of choice in acute DVT/PE in patients with severe renal impairment. Also used when there is a high risk of major bleeding because of its short half-life. The IV rate is adjusted using the APTT as a guide and discontinued when warfarin has achieved a therapeutic INR level.

## Fondaparinux

A synthetic pentasaccharide administered as an od SC injection. Indicated in ACS.

### Risks of bleeding on anticoagulation

When prescribing an anticoagulant or assessing a patient on anticoagulation, always check their bleeding risk. Patients with a recent bleed have a high risk of recurring bleeding. Patients who are very bruised are also at risk, as are those on antiplatelet (aspirin) or dual antiplatelet drugs. Patients with renal impairment have a high risk of bleeding, especially those on the new anticoagulants, LMWH, or fondaparinux. A platelet count of $<50 \times 10^9$/L ↑ bleeding risk. If in doubt, discuss with a haematologist.

### Managing major bleeding on anticoagulation

- Commence resuscitation measures (IV fluids, massive transfusion protocol as necessary).
- Identify the bleeding source and target haemorrhage control.
- Establish the time of the last anticoagulant dose and the anticoagulant name (new oral drugs will wear off after 24hr in patients with normal renal function).
- Send blood for APTT, INR, and cross-matching.
- Reverse warfarin with prothrombin complex concentrate (eg Beriplex®) and vitamin K 10mg IV.
- Reverse dabigatran with idarucizumab—if it is not available, consider dialysis.
- Andexanet alpha can be used to reverse rivaroxaban and apixaban.
- Give protamine sulfate if the patient has had a dose of LMWH in the past 24hr.
- Consider giving tranexamic acid 1g IV.

### Patients with less severe bleeding on anticoagulation

Patients with muscle haematomas, haematuria, or epistaxis may require hospital admission for observation and specific local treatment. Stop anticoagulant therapy for one or more days. Take expert advice when the patient is also at high risk of thrombosis (prosthetic heart valve or recent DVT/PE). Hold antiplatelet drugs.

(See https://www.b-s-h.org.uk/guidelines)

### Anticoagulation control check in inpatients on warfarin

For patients who have INR 4.0–7.0 without haemorrhage, withhold warfarin therapy for 1 or 2 days, and arrange review by an appropriate specialist team or GP. For patients with INR >7.0 without haemorrhage, withhold warfarin and obtain specialist consultation before considering phytomenadione (vitamin $K_1$) 5mg by slow IV injection or PO.

# Blood transfusion overview

► *It is better to stop bleeding than to have to replace blood loss.*

## General aspects

Correctly documenting and labelling blood tubes and forms, combined with checking blood products prior to administration, are crucial for safe patient care. If a patient's name(s), date of birth, clinical details, and address are unknown or uncertain, provide the gender and approximate age and identify them for transfusion purposes by a unique number (usually their unique ED number) and inform the blood transfusion laboratory.

To avoid confusion, the practitioner taking the blood sample must label and sign the tube at the patient's bedside, complete the form, and contact the transfusion service. Only take blood from one patient at a time. Label tubes before leaving the bedside to minimize the risk of mislabelling. Blood banks will refuse to handle incorrectly labelled forms/tubes. If samples are handwritten, the lab usually requires a second sample from fresh venepuncture to be supplied before issuing cross-matched blood.

If you knowingly give a blood product (or an animal product, eg gelatin) to a patient whom you know would not accept this (eg a Jehovah's Witness), you are likely to face an indefensible medicolegal claim. Document verbal consent and the discussion regarding benefits, risks, and alternative treatments. Always consider giving alternatives to blood products (eg iron for iron deficiency anaemia).

## What to request

When faced with major haemorrhage (eg major trauma or massive GI bleed), activate the Major Transfusion Protocol (see ➲ Massive blood transfusion, p. 182). ED staff can obtain emergency O rhesus D-negative red cells (this may be O +ve red cells for ♂ patients), platelets, and FFP. Cross-matched blood will follow soon after, at which point, switch from transfusing O –ve to cross-matched blood. Take group and screen samples before giving emergency red cells.

Assessment of a patient with hypovolaemic shock is complex and includes recognition of the clinical situation and the potential blood loss, together with ongoing assessment of the patient and investigations. Hb and Hct values may be misleading—it may take hours for their values to equilibrate to indicate the degree of blood loss.

*Group and screen* The patient's ABO and rhesus D groups are determined and the serum tested for unexpected red cell antibodies. Subsequently, if required, electronically matched red cells can be provided within 5min, assuming the antibody screen is clear. Request group and screen where a patient does not need transfusion in the ED but may require it later.

*Cross-match* Full blood compatibility testing may take up to 1hr. If blood is required more urgently, ABO- and Rh-compatible units can usually be provided within 15min, including an 'immediate spin cross-match' as a final check on ABO compatibility. In exsanguinating haemorrhage, uncross-matched group O rhesus –ve blood can be issued immediately.

## Blood products

*Red cells (additive solution)* Each pack (volume 300mL) derives from a single donor and has a Hct of 0.65–0.75 (0.55–0.65 for RBCs in additive solution). A transfusion of 4mL/kg will ↑ circulating Hb by ~10g/L.

*Platelet concentrate* Either pooled or from a single donor by platelet pheresis.

*Fresh frozen plasma* Contains clotting factors and fibrinogen.

*Cryoprecipitate* Derived from FFP when it is thawed. It is rich in factor VIII, fibrinogen, and vW factor.

*Prothrombin complex concentrate* A combination of vitamin K-dependent factors II, VII, IX, and X. Use prothrombin complex concentrate to reverse warfarin.

## Transfusion precautions

(See UK Blood Safety and Quality Regulations, 2005.)

- A practitioner must confirm all the following steps before commencing transfusion. If there is ANY discrepancy, DO NOT transfuse.
  *Note*: some sites use two practitioners, but these should perform single independent checks (ie not simply reading numbers out to each other).
- Confirm the details on the traceability label on the blood component match the patient's full name, date of birth, and hospital number (all patients must be wearing a wristband before transfusion is given).
- Check that the traceability label is attached to the blood bag.
- Ensure the donation number and the patient's blood group/rhesus D type all match and that any special requirements are covered.
- Check every component before starting transfusion for signs of discoloration, leaks, clots, etc., and the expiry date.
- If all checks are satisfactory, ensure that the component has been prescribed (prescription form and/or fluid balance chart) and sign the front of the traceability label before commencing the transfusion.
- Infuse all components through a giving set with an integral filter to trap large aggregates. Microaggregate filters are not routinely required.
- Never add any drug to a blood component infusion.
- Do not use giving sets which previously contained glucose or gelatin.
- Use a blood warmer for large and/or rapid transfusions.
- Once the transfusion has started, peel off the portion of the signed label and attach to the appropriate place in the prescription chart (or fluid chart).
- Sign the prescription form to confirm the patient identity checks.
- Complete and sign the traceability label and return it to the laboratory.

(See https://www.transfusionguidelines.org)

*Routine prescribing rates*

- For 'routine' transfusion, prescribe red cells to be given over 90min, unless there is a risk of transfusion-associated circulatory overload (TACO), in which case prolong it to 3.5hr.
- Platelets, FFP, and cryoprecipitate are usually given over 30min.

# Massive blood transfusion

Loss of 50% of circulating blood volume within 3hr is perhaps the most relevant ED definition of massive blood loss. Resuscitation requires an interdisciplinary team and clear organization.

## In the event of massive blood loss

- Protect the airway and give $O_2$ as required.
- Get help—two nurses and a senior doctor.
- Insert two large-bore cannulae.
- Activate the Massive Haemorrhage Protocol ahead of sending samples.
- Take blood for FBC, U&E, LFTs, coagulation, and cross-matching. Label the blood tubes and ensure they are sent directly to the laboratory. Do not leave them unlabelled or lying around in the resuscitation room.
- Accurate patient ID is essential, even if the patient is unknown. Ensure that the patient is wearing an identifying wristband.
- Call an appropriate senior surgeon—to stop the bleeding as soon as possible.
- Give tranexamic acid 1g IV if within 3hr of injury/bleed.
- Start the Massive Haemorrhage Protocol as per local guidelines. Usually give 1U of FFP per 1U of RBCs (starting with O –ve blood in ♀; O –ve or O +ve blood in ♂).
- Reverse anticoagulation (see ➲ Patients on anticoagulants, pp. 178–9).
- Repeat all bloods, including FBC, clotting, U&E, $Ca^{2+}$, $Mg^{2+}$, and fibrinogen levels, every 30min.
- Start platelet transfusion if platelet count falls below $75 \times 10^9$/L or if large volumes of blood and FFP have been given.
- Aim to maintain fibrinogen >1.0g/L and INR and APTT <1.5 times normal. Once fibrinogen begins dropping below 2.0g/L, give cryoprecipitate to maintain levels >1.5g/L.
- Recombinant factor VIIa might have been used as a 'last ditch attempt' to control bleeding in the past in some patients but is contraindicated due to thrombotic risk.

## Massive transfusion complications

Rapid infusion of blood products may lead to:

*Hypothermia* Blood products are normally stored at 2–6°C. Rapid infusion can cause significant hypothermia. Use blood warmers routinely for rapid transfusions (eg >50mL/kg/hr or 15mL/kg/hr in children). Never warm a blood product by putting a pack into hot water, on a radiator, or any other heat source.

*Electrolyte disturbances* With massive transfusion, the citrate anticoagulant may cause significant toxicity, ↓ plasma $Ca^{2+}$ (impairing cardiac function), and acid–base balance disturbance. This is aggravated in patients with underlying liver disease, hypotension, or hypothermia. Citrate may also bind $Mg^{2+}$, causing arrhythmias. Prophylactic or routine administration of IV $Ca^{2+}$ salts is not recommended. Monitor ECG and measure ionized plasma $Ca^{2+}$ levels during massive transfusion. $K^+$ levels ↑ in stored blood, and hyperkalaemia may follow massive infusion—check plasma $K^+$ levels regularly. Transient hypokalaemia may follow 24hr after a large transfusion.

# Transfusion reactions

Perform a full set of observations at baseline, after 15min, and at the end of each unit transfused, monitoring to detect early clinical evidence of acute reactions. If the patient develops an ↑ T°, shortness of breath, chest or abdominal pain, or hypotension, suspect a transfusion reaction. Treat allergic reactions, including itching, urticaria, bronchospasm, and fever, conventionally (see ➲ Anaphylaxis, pp. 44–5).

## Mismatched transfusion

By far, the most common cause is a clerical error when labelling, ordering, or administering blood. Transfusion of ABO-incompatible blood causes acute severe haemolysis and circulatory collapse. In a hypovolaemic, shocked, or anaesthetized patient, these features may be obscured and missed.

## If a transfusion reaction is suspected

ABO incompatibility, haemolytic reaction, bacterial infection, severe allergic reaction, or transfusion-related acute lung injury:

- Stop the transfusion.
- Keep the IV line open with 0.9% saline.
- Record all observations, and give supplemental $O_2$ as required.
- Double-check the blood unit label with the patient's wrist identity band and other identifiers.
- Send the unit of blood product and the giving set to the blood bank.
- Take blood and send it as follows:
  - An anticoagulated sample for blood bank.
  - U&E, LFTs, immunoglobulin A (IgA) level.
  - Serial mast cell tryptase (at 3hr and 24hr) if severe anaphylaxis.
  - Coagulation screening.
  - Blood cultures if sepsis suspected.
- Contact the blood bank directly by phone.
- Contact the haematologist directly.
- Give broad-spectrum antibiotic if infection suspected.
- Monitor fluid balance and urinary output, and check for Hb in urine.

## Transfusion-associated circulatory overload

This is defined as acute or worsening pulmonary oedema within 6hr of transfusion. It is currently the largest cause of mortality and morbidity from transfusion. Assess all patients for their risk of TACO—if at high risk, give units more slowly (over 3.5hr), together with a diuretic and more frequent observations.

## The role of blood products

Blood transfusion is not a panacea. An improvement in $O_2$ delivery cannot be assumed. RBC function deteriorates during storage, and changes in $O_2$ affinity occur with ↓ 2,3-diphosphoglycerate (DPG) levels, whilst ↓ adenosine triphosphate (ATP) levels alter RBC membrane deformability, causing ↑ cell stiffness and micro-circulatory problems. UK donations are routinely screened for hepatitis B, HIV, human T-cell lymphotropic virus (HTLV), syphilis, and, where necessary, CMV. However, blood cannot be sterilized—small, but definite, risks of infection transmission exist.

# Sickle-cell disease

Sickle-cell disease occurs in African, Indian, Middle Eastern, Caribbean, USA, and Mediterranean populations. It is caused by a genetic mutation in one of the chains of the Hb molecule. The normal adult Hb genotype AA produces HbA. In heterozygotes (sickle-cell trait), one gene is abnormal (HbAS) and about 40% of the patient's Hb will be HbS. In homozygotes (sickle-cell anaemia), both genes are abnormal (SS) and >80% of the Hb will be HbS. HbS molecules polymerize in deoxygenated or acidotic conditions, causing RBC sickling. Sickle cells are rigid and fragile. They may haemolyse or block small vessels, leading to tissue ischaemia, infarction, and further sickling (see Fig. 3.39). Sickling also occurs with genes coding for other analogous amino acid substitutions (eg HbSC and SD diseases).

## Clinical features

*Sickle-cell trait* causes no disability, except during conditions of severe hypoxia (eg sudden depressurization in aircraft or during cardiac arrest).

Patients with *sickle-cell anaemia* have chronic anaemia (Hb 80–100g/L), with alternating good health and acute crises. Later, chronic ill health supervenes with renal failure, bone necrosis (evident in 50% of patients by age 35y), osteomyelitis, leg ulcers, and iron overload as a consequence of transfusions. There is predisposition to infection, especially *Staphylococcus, Pneumococcus*, and *Haemophilus*.

*Sickle-cell crises* can occur *de novo* or follow infection, cold, dehydration, or any situation where tissue hypoxia/ischaemia occurs. The crisis may involve thrombosis, haemolysis, marrow aplasia, or acute splenic/liver sequestration (especially in children aged <5y). Any acute medical or surgical emergency may be mimicked (eg acute abdomen, PE, stroke). Severe aching bony pain and low-grade fever (even in the absence of infection) are common. Cerebral sickling may present with bizarre behaviour, psychosis, fits, TIAs, stroke, or other focal neurological signs. Priapism, jaundice, and painful swelling of the hands and feet may occur.

### *Acute chest syndrome*

The leading cause of death in sickle-cell anaemia. It presents as chest pain, hypoxia, and pulmonary infiltrates. There may be cough, tachypnoea, and wheezing. Poorly understood, but infection may be a precipitant.

### *Acute splenic sequestration*

Sudden trapping of large numbers of RBCs in the spleen results in severe anaemia, an enlarging spleen, hypovolaemia, and thrombocytopenia. It occurs most commonly in young children—those with sickle-cell disease have a 30% chance of having acute splenic sequestration by the age of 5y. It may present with shock and splenomegaly, with a mortality of >15%.

### *Osteomyelitis and septic arthritis*

Osteomyelitis and septic arthritis occur more commonly in sickle-cell disease. Be suspicious if a patient presents with high fever, soft tissue swelling, or pain in a different pattern to normal. *Salmonella* is frequently implicated.

### Investigations

No specific tests can detect a sickle-cell crisis:

- All patients in the at-risk groups require a sickle test before any anaesthetic procedure (including regional anaesthesia and Bier's block).
- Sickle testing (using an oxidizing agent) will detect sickling in homo- and heterozygous forms. Hb electrophoresis can then distinguish between HbSS, HbAS, and other Hb variants.
- FBC typically reveals significant anaemia (Hb 60–80g/L, but Hb may be much lower if acute haemolysis, sequestration, or aplasia are present). Post-splenectomy features may be seen on blood film. WCC may be ↑ (20–60 × $10^9$/L) in the absence of infection and platelet count is also usually ↑.
- Infection screen, including blood cultures, MSU, and CXR.
- Joint aspiration for culture if septic arthritis is suspected.
- U&E, ABG, ECG.
- Arrange CT brain scan if there are neurological symptoms or signs.

### Management of crises

Provide supportive therapy, directed to the patient's symptoms:

- Get expert help!
- Keep the patient warm and rested, and give $O_2$ if any obvious symptoms or $SpO_2$ <94%.
- Opioids (given IV and titrated to response) are often required for pain. Consider morphine IVI or a patient analgesia pump.
- Commence rehydration with PO or IV fluids, but take care not to precipitate heart failure.
- Transfusion may be required if severe anaemia from acute haemolysis, sequestration, or aplasia occurs, or if there are CNS or lung complications.

Empirical antibiotic therapy may be required if infection is thought to be the trigger for the sickling crisis.

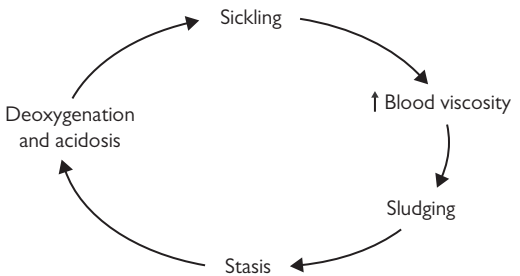


**Fig. 3.39** The sickling cycle.

## Chapter 4

# Toxicology

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# Poisons: background

## Types of poisoning

*Unintentional poisoning* Most common in inquisitive small children (1–4y) who eat tablets, household chemicals, and plants. Older children and adults may be poisoned by chemicals at school or work, or by drinking toxic fluids decanted into drink bottles. Poisoning by drugs may result from miscalculation or confusion of doses or by taking the same drug under different names. Drug packers and stuffers who swallow drugs wrapped in condoms or polythene or who stuff them in the rectum or vagina may suffer poisoning if the packages leak (see ➔ Body packers, p. 225).

*Self-poisoning* The most common form of poisoning in adults and may occur in children as young as 6y (usually with a family history of self-poisoning). Drugs or poisons are often taken impulsively, sometimes to manipulate friends or family. Suicidal intent may be relatively uncommon, but assess all patients for this (see ➔ Self-harm, pp. 628–9). Sometimes patients leave suicide notes and conceal drugs/poison to evade detection.

*Non-accidental poisoning of children* A form of fabricated or induced illness (see ➔ Fabricated or induced illness, p. 760), in which a parent deliberately poisons a child. Homicidal poisoning is rare and may involve acute or chronic poisoning with chemicals such as arsenic or thallium.

*Chemical plant incidents and terrorism* Potential threats to large numbers of people.

## National Poisons Information Service

*TOXBASE*, the UK National Poisons Information Service (NPIS) database on clinical toxicology, is available at 🖱 https://www.toxbase.org and can be downloaded as an app for phones and tablets. It includes information about poisoning with drugs, household products, plants, and fungi, as well as industrial and agricultural chemicals and agents which might be deliberately released by terrorists. Access to TOXBASE is password-protected and is restricted to NHS staff in the UK and hospitals in Ireland.

When accessing TOXBASE for clinical guidance, print off the information and place it in the notes to guide ongoing care. TOXBASE was most frequently consulted in 2015/16 in relation to poisoning by the following: paracetamol, ibuprofen, codeine, diazepam, sertraline, citalopram, mirtazapine, zopiclone, quetiapine, and tramadol.

TOXBASE provides sufficient information for most cases of poisoning—refer to it 'routinely', as toxicology is a dynamic specialty and advice frequently changes and is updated. More detailed information/advice is available from Poisons Information Centres and is especially useful for complex cases or severe poisoning.

The UK NPIS has four centres, with a single telephone number 0344 892 0111 which directs the call to the nearest centre, or to the on-call centre out of hours. In Ireland, advice is available from the National Poisons Information Centre, Dublin, telephone (01)809 2566.

Enquiries to Poisons Information Centres are usually answered initially by an information officer using TOXBASE and other reference sources. Medical staff with specialist toxicology experience are available for advice about seriously poisoned patients. Poisons Information Centres can also advise about sources of supply of antidotes that are needed only occasionally and about laboratory analyses that may be helpful in managing some patients.

An additional useful source of information is the *BNF*, which contains a chapter on the emergency treatment of poisoning.

*Advice for the public*

NHS 111 in England, NHS 24 in Scotland, and NHS Direct in Wales will provide advice to members of the public who have concerns about poisoning.

## Psychiatric assessment and admission after poisoning

*Adults*

Admit patients who are seriously poisoned to a medical ward or, if appropriate, to ICU. However, most patients who take overdoses suffer no serious ill effects and can be treated on an ED observation ward or in a Clinical Decisions Unit. Even if there is no risk of toxicity, admission overnight provides an opportunity for a 'cooling off' period for the patient to get away from the situation that precipitated the overdose and/or time to sober up. This should allow a more rational appraisal of the problems and may reduce the risk of further self-poisoning.

*Look for the causes* of every episode of self-harm. Observe carefully in the ED and on the ward any patient who appears suicidal (see ➔ Self-harm, pp. 628–9), because of the risk of further self-harm.

*Children with poisoning*

Serious poisoning is uncommon in children. Many children appear well but have been exposed to an unknown amount of a compound which could be toxic. Admit such children to a paediatric ward for observation—they can be discharged after a few hours if no toxic effects occur. A child may be discharged home directly from the ED if the substance taken is known to be non-toxic. The health visitor may usefully visit the home to advise about poisoning prevention. In children >6y, consider the possibility of intentional self-harm and the need for assessment by the child and adolescent mental health services (CAMHS).

# Diagnosis of poisoning

The patient or relatives/friends may state what drugs or poison have been taken, but this information is not always accurate. Self-poisoning is often an impulsive act whilst under the influence of alcohol—the patient may not know which tablets (and how many) he/she took. There is often confusion around compound analgesics and the use of trade names. Check any bottles or packets for the names and quantities of drugs or poisons that were available. If a patient is unconscious or severely poisoned, check hospital records for details of previous overdoses and find out from the GP what drugs had been prescribed. Record the time of ingestion of the drug or poison. Examine for signs of poisoning, injection marks, or self-injury. Exclude other processes mimicking poisoning (eg head injury, meningitis). Traditional Chinese medicines or herbal preparations can cause significant toxicity. Drugs of abuse and 'legal highs' (new psychoactive substances) pose an ↑ burden on EDs.

## Toxidromes: features suggesting a particular poison

- Coma with dilated pupils, divergent squint, tachycardia, ↑ muscle tone, and ↑ reflexes and extensor plantars suggest tricyclic antidepressant or orphenadrine poisoning (see ➲ Tricyclic antidepressant poisoning, pp. 202–3).
- Coma with hypotension, respiratory depression, and ↓ muscle tone suggest barbiturates, clomethiazole (see ➲ Clomethiazole poisoning, p. 204), benzodiazepines with alcohol, or severe tricyclic antidepressant poisoning (see ➲ Tricyclic antidepressant poisoning, pp. 202–3).
- Coma with slow respiration and pinpoint pupils is typical of opioid poisoning (give naloxone) (see ➲ Opioid poisoning, p. 196).
- Tinnitus, deafness, hyperventilation, sweating, nausea, and tachycardia are typical of salicylate poisoning (see ➲ Salicylate poisoning, p. 197).
- Agitation, tremor, dilated pupils, and tachycardia suggest amphetamines, ecstasy, cocaine, sympathomimetics (see ➲ Recreational drugs, pp. 222–3), tricyclic antidepressants (see ➲ Tricyclic antidepressant poisoning, pp. 202–3), or selective serotonin reuptake inhibitors (SSRIs) (see ➲ Serotonin syndrome, p. 224).

## Assessment and monitoring

- Assess and record conscious level (see ➲ Glasgow Coma Score (adults), p. 369). Observe frequently.
- Check blood glucose in patients with confusion, coma, or fits.
- Monitor breathing and RR. Use a pulse oximeter—note that the $SpO_2$ may be misleading in CO poisoning (see ➲ Carbon monoxide poisoning, p. 216).
- Check ABG (or VBG) if the patient is deeply unconscious or breathing abnormally.
- Record and monitor the ECG if the patient is unconscious, has tachy- or bradycardia, or has taken drugs or poisons with a risk of arrhythmias.
- Record BP and T°.

## Investigations in poisoned patients

Most useful are: paracetamol and salicylate levels, blood glucose, ABG/VBG, and U&E. Measure paracetamol if there is any possibility of paracetamol poisoning (this includes all unconscious patients). Record the time of the sample accurately. Many labs can measure salicylate, iron, and lithium and even check for paraquat, if necessary. Comprehensive drug screening is rarely needed and is only available in specialist centres (discuss with NPIS) (see ➲ National Poisons Information Service, pp. 188–9).

# Poisons: supportive care

## Protect airway, monitor breathing, and ventilate if necessary

Hypoxia and $CO_2$ retention are common in deep coma. In an unconscious patient, use a cuffed endotracheal (ET) tube if there is no gag reflex. If an oral or nasal airway is needed, nurse in the recovery position to minimize the risk of aspiration in case vomiting or regurgitation occurs.

## Hypotension

This may result from relative hypovolaemia, arrhythmias, and cardiodepressive effects of drugs. Treat according to the cause. Elevate the foot of the trolley. If BP is <90mmHg, consider giving saline 500mL IV. Vasopressors, inotropes, glucagon, or high-dose insulin may be required, under expert guidance.

## Cardiac arrhythmias

Generally rare in poisoned patients. Drugs most implicated are tricyclics, β-blockers, chloral hydrate, digoxin, $K^+$, bronchodilators, verapamil, and amphetamines. Correct hypoxia, respiratory depression, metabolic acidosis, and electrolyte abnormalities. Anti-arrhythmic drugs are rarely needed—get expert help.

## Convulsions

Dangerous because they cause hypoxia and acidosis and may precipitate cardiac arrest. Drugs responsible include tricyclic antidepressants, mefenamic acid, and theophylline. Check for, and correct, hypoxia and hypoglycaemia. Do not give anticonvulsants if fits are single and brief, but if fits are repeated or prolonged, give lorazepam 4mg IV (or PR diazepam or buccal midazolam if venous access is not available).

## Hypothermia

May occur with any drug causing coma, especially barbiturates, clomethiazole, and phenothiazines. Check rectal T° with a low-reading thermometer. Insulation and passive rewarming are usually adequate.

## Hyperthermia

(See ➲ Heat illness, pp. 274–5.)

May occur with amphetamines, cocaine, ecstasy, monoamine oxidase inhibitors (MAOIs), sympathomimetics, and theophylline. Consider serotonin syndrome (see ➲ Serotonin syndrome, p. 224). Convulsions and rhabdomyolysis are common. Active cooling, cyproheptadine, chlorpromazine, and possibly dantrolene are needed. Get expert help.

## Complications of immobility

Prolonged immobility (eg due to tricyclics and barbiturates) risks pressure areas. Treat blisters like minor burns. Immobility may cause rhabdomyolysis (leading to renal failure), nerve palsies, and compartment syndrome—if this is suspected, check CK; test urine for myoglobinuria, and get urgent orthopaedic advice about measuring compartment pressures.

## Urinary retention

Common in coma, especially after tricyclic poisoning. Suprapubic pressure often stimulates reflex bladder emptying. Catheterization may be needed to empty the bladder or to measure urine output.

# Reducing absorption of poison

Several methods aiming to reduce absorption of a poison have been described, but none can be recommended routinely.

## Gastric lavage

Now almost only of historical interest, gastric lavage (for an adult) involves the insertion of a large orogastric tube (36 or 40FG), then after clinically confirming the position, pouring 300mL aliquots of tepid water down the tube, then siphoning it out until the effluent is clear.

This does not empty the stomach of solids and may force gastric contents through the pylorus into the small bowel. It may cause hypoxia, aspiration pneumonia, and occasionally oesophageal perforation. Gastric lavage >1hr after an overdose is ineffective in ↓ the absorption of poisons.

## Induced emesis

Never use emetics. *Ipecacuanha* (ipecac) was once used frequently, but there is no indication for its use. *Salt solutions* may cause fatal hypernatraemia and must never be used as an emetic.

## Activated charcoal

Given within 1hr, this ↓ the absorption of therapeutic doses of many drugs, but there is little evidence of clinical benefit when it is used after an overdose. Charcoal ↓ the half-life of some drugs (eg digoxin), which undergo entero-hepatic recycling. However, charcoal is messy and unpleasant to take, and often causes vomiting. Aspiration into the lungs can result in fatal pneumonitis. Various formulations of activated charcoal are available (eg *Charcodote*® and *Carbomix*®). *Carbomix*® may cause severe constipation, especially if given in repeated doses.

Do not give activated charcoal for substances which do not bind to it. These include: iron, lithium, boric acid, cyanide, ethanol, ethylene glycol, methanol, organophosphates, petroleum distillates, and strong acids and alkalis. Charcoal is most likely to be useful for poisons which are toxic in small quantities (eg tricyclic antidepressants and theophylline derivatives). If a dangerous overdose has been taken in the previous 1hr, give charcoal (PO or via an orogastric tube: adult 50g; child 1g/kg, max 50g). Charcoal may be effective for >1hr for sustained-release formulations or drugs that delay gastric emptying (eg tricyclic antidepressants and opioids). Obtain expert advice before giving charcoal in repeated doses, which are only helpful in life-threatening poisoning with a few drugs (eg carbamazepine, dapsone, digoxin, phenobarbital, quinine, theophylline, and salicylate, and a few other drugs rarely taken in overdose).

### Whole-bowel irrigation

Whole-bowel irrigation is rarely needed and should only be used on expert advice. The aim of whole-bowel irrigation is to empty the bowel rapidly of solid contents by giving fluid PO or down an NG tube until the rectal effluent becomes clear. The value of this is uncertain. It may be useful for poisoning with sustained-release drug formulations or for poisons such as iron or lithium, which are not absorbed by activated charcoal. It has also been used to remove packets of cocaine from body packers and button batteries from children.

Bowel-cleansing solutions of polyethylene glycol and electrolytes (eg Klean-Prep®) are used in whole-bowel irrigation—2L/hr in adults (500mL/hr in small children) for up to 6hr, or occasionally longer (up to a maximum of 12hr). Do not use normal saline, since it may cause fluid overload and hypokalaemia. Nausea, vomiting, abdominal pain, and electrolyte disturbances may occur. Monitor ECG, U&E, and urine output.

# Antidotes for poisons

Antidotes are available for only a few drugs and poisons (see Table 4.1) and are often not necessary. More information is available from TOXBASE and Poisons Information Centres (see ➲ National Poisons Information Centre, pp. 188–9).

**Table 4.1** Antidotes for poisons

| Poison | Antidote | Notes |
|---|---|---|
| Benzodiazepines | Flumazenil‡ | See ➲ Benzodiazepine poisoning, p. 204 |
| β-blockers | Glucagon, atropine | See ➲ Beta-blocker poisoning, p. 206 |
| Calcium channel blockers | Calcium, glucagon, high-dose insulin euglycaemic therapy | See ➲ Beta-blocker poisoning, p. 206 |
| CO | $O_2$ | See ➲ Carbon monoxide poisoning, p. 216 |
| Cyanide | Sodium nitrite, sodium thiosulfate, dicobalt edetate, hydroxocobalamin | See ➲ Cyanide poisoning, p. 215 |
| Digoxin | Digoxin antibodies (DigiFab®)† | See ➲ Digoxin poisoning, p. 207 |
| Ethylene glycol | Ethanol, fomepizole† | See ➲ Ethylene glycol poisoning, p. 212 |
| Hydrofluoric acid | Calcium gluconate | See ➲ Chemical burns, p. 405 |
| Iron salts | Desferrioxamine | See ➲ Iron poisoning, p. 209 |
| Local anaesthetics | Lipid emulsion (Intralipid®) | See ➲ Lipid emulsion (Intralipid®) therapy for drug toxicity, p. 195; ➲ Local anaesthetic toxicity, p. 294 |
| MDMA | Dantrolene† | See ➲ Recreational drugs, pp. 222–3 |
| Methanol | Ethanol, fomepizole† | See ➲ Methanol poisoning, p. 211 |
| Opioids | Naloxone | See ➲ Opioid poisoning, p. 196 |
| Organophosphates | Atropine, pralidoxime† | See ➲ Organophosphate poisoning, p. 214 |
| Paracetamol | Acetylcysteine | See ➲ Paracetamol poisoning, pp. 198–201 |
| Serotonin syndrome (eg SSRI overdose) | Cyproheptadine | See ➲ Serotonin syndrome, p. 224 |
| Sulfonylureas | Glucose, octreotide | See ➲ Sulfonylurea poisoning, p. 205 |
| Tricyclic antidepressants | Sodium bicarbonate, Intralipid® | See ➲ Lipid emulsion (Intralipid®) therapy for drug toxicity, p. 195; ➲ Tricyclic antidepressant poisoning, pp. 202–3 |
| Warfarin | Vitamin K, prothrombin complex concentrate, FFP | See ➲ Patients on anticoagulants, pp. 178–9 |
| Adder bites | Snake venom antiserum | See ➲ Snake bites, p. 423 |
| Foreign snakes | Antivenoms† | Expert advice (see ➲ National Poisons Information Service, pp. 188–9) |

† Very rarely needed—get expert advice. See main text.
‡ Flumazenil is not licensed for use as a standard antidote, except in specific circumstances. See main text.

Antidotes are also available for arsenic, lead, mercury, thallium, and certain other metals. Some antidotes (marked†) are very rarely needed—get expert advice (see ➲ National Poisons Information Service, pp. 188–9) about when and how to use these antidotes (and where to obtain them). Flumazenil (marked‡) is not licensed for use as a standard antidote, except in specific circumstances (see ➲ Benzodiazepine poisoning, p. 204).

### Increasing elimination of poisons

The vast majority of poisoned patients recover with supportive care plus appropriate antidotes, if necessary. Active removal of absorbed poison is only needed in special circumstances. Alkalinization of the urine may help in salicylate poisoning (see ➲ Salicylate poisoning, p. 197). Haemodialysis is occasionally used for severe poisoning with salicylates, ethylene glycol, methanol, lithium, phenobarbital, and chlorates. Haemoperfusion is rarely needed but might be helpful (on specialist advice) in severe poisoning with barbiturates, chloral hydrate, or theophylline.

### Lipid emulsion (Intralipid®) therapy for drug toxicity

IV lipid emulsion is rarely needed but can be life-saving in overdoses of local anaesthetics such as lidocaine or bupivacaine (see ➲ Local anaesthetic toxicity, p. 294). Haemodialysis is occasionally used for severe poisoning with salicylates. It may be useful in cardiac arrest caused by some other drugs—the indications are unclear, but case reports record dramatic recovery from cardiac arrests due to a variety of drugs. Consider lipid emulsion in drug-induced cardiac arrest unresponsive to standard treatment (see ➲ Cardiac arrest management, pp. 52–3). EDs, theatres, and ICUs should stock it.

Lipid emulsion acts as a 'lipid sink', binding lipophilic drugs and reducing the amount of active free drug. It may also affect myocardial metabolism. Lipid emulsion is not licensed for use in drug overdose, and the safety of rapid infusion is unknown. Lipid interferes with analysis of blood samples, so if possible, take these before starting lipid emulsion, including blood for later measurement of drug concentrations.

Give 20% Intralipid® 1.5mL/kg IV as a bolus (for a 70kg patient, give 100mL), followed by 0.25mL/kg/min for 20–30min to an initial maximum of 500mL. Consider repeating the bolus once or twice for persistent cardiovascular collapse or asystole. Titrate the infusion rate against the clinical response.

Report cases in which lipid emulsion is used to the Poisons Information Service (see ➲ National Poisons Information Service, pp. 188–9).

### Insulin therapy in poisoning

Poisoning with cardiac drugs, such as calcium channel blockers (see ➲ Calcium antagonist poisoning, p. 206) and β-blockers (see ➲ Beta-blocker poisoning, p. 206), may cause severe hypotension. If standard treatments are ineffective, get expert advice (see ➲ National Poisons Information Service, pp. 188–9) and consider using insulin therapy, which may improve myocardial carbohydrate metabolism and ↑ BP and cardiac output.

### Low toxicity substances

Many ingestions by children, particularly within the household, cause extreme anxiety for parents but are, in reality, of low toxicity. Sometimes mild abdominal discomfort may occur, but severe features are unlikely. Mild symptoms can be treated with small amounts of oral fluids. These substances are numerous and include soil, fresh dog faeces, crayons, felt tip pen ink, and aftersun lotion. A more comprehensive list and poster are available from NPIS (🖱 http://www.npis.org/lowtoxposter2017.pdf).

# Opioid poisoning

The opioids include morphine, diamorphine (heroin), pethidine, codeine, buprenorphine, and methadone. These are used as analgesics (sometimes combined with paracetamol, as in co-codamol and co-dydramol), cough suppressants, and anti-diarrhoeal agents. Acute opioid poisoning often occurs in recreational users. Consider opioid poisoning in patients presenting with coma of unknown aetiology (remembering to check for transdermal patches).

## Clinical features

Opioid poisoning causes the triad of coma, ↓ RR, and pinpoint pupils. Cyanosis, apnoea, convulsions, and hypotension may occur. Effects of opioids are potentiated by alcohol. Non-cardiogenic pulmonary oedema may result from injecting heroin or other opioids.

*Respiratory depression* may cause death within 1hr of an opioid overdose. However, delayed respiratory depression can occur in poisoning with co-phenotrope (diphenoxylate and atropine), in which the opioid effects usually predominate over atropine toxicity. Delayed toxicity may occur with slow-release formulations of drugs, and also with methadone which has a very long duration of action (half-life 15–60hr).

## Treatment

Clear and maintain the airway. If breathing is inadequate, ventilate with $O_2$ using a bag and mask or an ET tube. *Naloxone* is a specific antagonist for opioids and reverses coma and respiratory depression if given in sufficient dosage. Give naloxone as a therapeutic trial in suspected opioid poisoning—record coma level, pupil size, and RR, and check for any response. The usual initial dose of naloxone for adults is 0.4mg IV, followed by a further dose of 0.8mg after 60s if no response. The aim is to reverse respiratory depression, not to restore full consciousness. In suspected toxicity due to therapeutic excess or reduced elimination in chronic opioid users or palliative care patients, consider lower doses.

For children, give 100mcg/kg (IV, IM or IN) up to 2mg, repeated as necessary. Intranasal naloxone, given by dripping or spraying the IV solution into the nose over 60s, enables rapid absorption. For children at risk of opioid withdrawal, give 1–10mcg/kg every 60s, titrated according to the response.

Naloxone has a much shorter duration of action than most opioids and so coma and respiratory depression often recur when naloxone wears off. More naloxone is often needed, given IV, by IVI, or IM, the dose adjusted depending on the response. Observe for at least 6hr after the last dose of naloxone and up to 24hr with methadone overdose. If repeat doses are required, consider starting a naloxone infusion.

Persuade patients at risk of respiratory depression to stay in hospital—consider using the Mental Capacity Act (see ➲ Mental Capacity Act, p. 645) if a patient is determined to leave.

# Salicylate poisoning

If <125mg/kg body weight of aspirin is ingested and the patient is asymptomatic, harm is unlikely. If the patient denies taking salicylate and has no clinical evidence of poisoning, blood tests to check salicylate levels are not necessary.

## Clinical features

- *Commonly*: vomiting, tinnitus, deafness, sweating, vasodilatation, hyperventilation, and dehydration. Hypokalaemia may occur.
- *Severe poisoning* may produce confusion, coma, and convulsions.
- *Children* are prone to developing hyperpyrexia and hypoglycaemia.
- *Rare features* include non-cardiogenic pulmonary oedema, cerebral oedema, and renal failure.

## Metabolic and acid–base disturbances

May be complex—adults usually have mixed metabolic acidosis and respiratory alkalosis, but respiratory effects predominate. In small children (± a few adults), acidosis predominates, often with confusion or coma.

## Management

Consider giving activated charcoal if a patient has ingested >125mg/kg of salicylate in the previous 1hr (or any amount of methyl salicylate).

Gastric lavage may be of benefit if a patient has ingested >500mg/kg body weight in the previous 1hr. Measure plasma salicylate concentration after at least 2hr in symptomatic, and 4hr in asymptomatic, patients. Take a repeat sample after a further 2hr in those who are symptomatic or have an initial level of ≥200mg/L. Check U&E, glucose, clotting, and ABG/VBG. Consider a second dose of charcoal if the plasma salicylate ↑, suggesting delayed gastric emptying or if enteric-coated tablets have been taken.

*Mild poisoning* Asymptomatic patients with plasma salicylate <300mg/L (2.2mmol/L) and a normal VBG are medically fit for discharge at 6hr.

*Moderate poisoning* Patients with salicylate levels of 300–700mg/L (2.2–5.1mmol/L) require treatment. Replace $K^+$ if low (max 20mmol/hr IV). Give sodium bicarbonate 50–100mmol over 30min. If plasma level is >500mg/L (or 350mg/L in children), consider urinary alkalinization to enhance elimination. Aim for a urinary pH of 7.5–8.5. In adults, this can be achieved by giving 3–5mmol/kg of sodium bicarbonate (eg ~1.5L of 1.26% over 1hr). Beware using 4.2% or 8.4% sodium bicarbonate solution as it is a venous irritant and can cause tissue necrosis if extravasation occurs.

*Severe poisoning* CNS features, acidosis, or salicylate >700mg/L (5.1mmol/L) are associated with significant mortality. Consider urgent haemodialysis/haemodiafiltration. In life-threatening poisoning (coma and extreme hyperventilation), consider paralysis and IPPV, whilst haemodialysis removes salicylate and corrects the electrolyte disturbances.

# Paracetamol poisoning

Paracetamol may cause severe liver damage if >150mg paracetamol/kg body weight are taken. Severe toxicity is unlikely if <75mg/kg has been ingested. In obese patients (>110kg), calculate the toxic dose in mg/kg, and the dose of acetylcysteine using a weight of 110kg, rather than the patient's actual weight.

A metabolite of paracetamol (*N*-acetyl-*p*-benzoquinone imine, NAPQI) binds glutathione in the liver and causes hepatic necrosis when stores of glutathione are exhausted. Renal failure from acute tubular necrosis occurs occasionally, but renal failure without liver failure is rare.

## Risk factors for paracetamol toxicity

Previously, some patients were deemed to be at ↑ risk of liver damage (eg alcoholics and patients on enzyme-inducing drugs) and were treated differently. However, this is of historical interest only, as all patients are now treated as if 'high risk', with a low threshold for acetylcysteine use.

## Clinical features

Nausea, vomiting, and abdominal discomfort are common within a few hours. In untreated patients developing liver damage, vomiting continues beyond 12hr and there is pain and tenderness over the liver (from 24hr), jaundice (at 2–4 days), and sometimes coma from hypoglycaemia (at 1–3 days) and hepatic encephalopathy (onset at 3–5 days). Loin pain, haematuria, and proteinuria suggest incipient renal failure. Hepatic failure causes bleeding from coagulation abnormalities and hyperventilation from metabolic acidosis. In fatal cases, cerebral oedema, septicaemia, and DIC are common. However, many patients survive severe liver damage and recover completely.

*LFTs* are normal until >18hr after the overdose. The most sensitive lab evidence of liver damage is often a prolonged INR (from 24hr after overdose). Liver enzymes (ALT and AST) may reach >10,000U/L at 3–4 days. Bilirubin rises more slowly (max at about 5 days).

## Paracetamol antidotes

*Acetylcysteine* is given by IVI in 5% glucose. Initial dose is 150mg/kg body weight in 200mL of glucose over 1hr, 50mg/kg in 500mL over 4hr, then 100mg/kg in 1L over 16hr. Acetylcysteine can cause side effects (which are more likely if the plasma paracetamol level is low): erythema and urticaria around the infusion site or more generalized rashes, itching, nausea, angio-oedema, bronchospasm, and rarely hypotension or hypertension. Side effects are dose-related and usually start in the first hour of treatment. If they occur, stop the infusion and give an antihistamine (eg chlorphenamine 10mg IV over 1min). When symptoms have settled, resume acetylcysteine at a slower rate—consider giving the first bag over 2hr and the rest at the normal rate.

Evidence suggests a modified 12hr regimen may be as efficacious with fewer side effects, but it is not yet in widespread use. *Note:* in rare circumstances (eg lack of venous access), oral acetylcysteine has been given, but it remains an unlicensed indication.

### Children

Serious paracetamol poisoning is rare in children. Young children rarely take large amounts of paracetamol, and they metabolize it differently from adults and may have lower risk of hepatotoxicity. However, there are no data for assessing the risk in children, so use the same treatment guidelines as for adults.

If it is certain that <75mg/kg has been taken, then no investigation or treatment is needed—discharge with advice to return if symptoms develop.

Treatment with acetylcysteine is rarely needed in children. Doses are as for adults (see ➲ Paracetamol antidotes, p. 198), but with smaller volumes of fluid for IVI.

### Pregnancy

Assess the risk of toxicity and treat as for non-pregnant patients. Acetylcysteine does not seem to carry any risk to the fetus and may protect the fetal liver from damage. Calculate the dose ingested based on the patient's pre-pregnancy weight, and the treatment dose of acetylcysteine based on the current weight.

Paracetamol overdose does not appear to cause teratogenic effects.

### Staggered overdoses

If paracetamol has been taken in excess (≥75mg/kg) over >1hr, consider this to be a 'staggered overdose'. Do not use the graph to guide treatment for patients with staggered overdoses. If the patient has symptoms of toxicity or the amount taken was >75mg/kg, take blood for INR, LFTs, U&E, and paracetamol level (which may confirm that some was taken), and treat with acetylcysteine. If in doubt, start treatment and get expert advice.

### Outcome of treatment

Treatment with acetylcysteine within 8hr of an overdose is very effective in preventing liver and renal damage. Later treatment is less effective, but still worthwhile.

### Late presentation after paracetamol poisoning

Patients who present late are more likely to be severely poisoned than those who present soon after ingestion. Late presenters often have continuing vomiting and abdominal pain, which are symptoms of liver damage. The treatment graph (see Fig. 4.1) may be unreliable at >15hr, because of insufficient data on untreated patients.

### Liver transplantation

Liver transplantation is occasionally needed for hepatic failure due to paracetamol overdose in patients who presented or were treated late. Aim to identify and refer patients to a liver transplant unit as soon as possible. Transplant criteria include arterial pH <7.30 ($H^+$ >50nmol/L) after resuscitation, or PT >100s (INR >6.7), and creatinine >300 micromoles/L in patients with grade 3 or 4 hepatic encephalopathy.

# Management of paracetamol poisoning

The time since ingestion is crucial in interpreting paracetamol concentrations and assessing the need for specific treatment. Record the time of ingestion as accurately as possible. When taking blood for paracetamol levels, record the precise time in the notes and on blood forms. Start treatment immediately if the time of ingestion is unknown.

### Management within 4hr of ingestion

Consider activated charcoal (see ➲ Activated charcoal, p. 192) if 150mg/kg paracetamol has been taken in the previous 1hr. Take blood at 4hr from ingestion and use the treatment graph (see Fig. 4.1) to assess the risk of liver damage; if the result is above the treatment line, give IV acetylcysteine (see ➲ Paracetamol antidotes, p. 198).

### Management at 4–8hr from ingestion

Measure paracetamol level, and use the graph to assess the risk of liver damage. If above the treatment line, or only just below it, give IV acetylcysteine (for doses, see ➲ Paracetamol antidotes, p. 198). Treatment is most effective if started before 8hr—start it at once if the paracetamol level is not available by this time and >150mg/kg has been taken. Patients treated with acetylcysteine within 8hr of an overdose should be medically fit for discharge at the end of the treatment course.

### Management at 8–24hr from ingestion

Urgent action is needed—start treatment with IV acetylcysteine immediately if >150mg/kg paracetamol has been taken. Measure plasma paracetamol level (plus creatinine, LFTs, and INR), and use the treatment graph to assess the risk of liver damage. If the paracetamol level is well below the line and the patient is asymptomatic, stop acetylcysteine treatment. Continue acetylcysteine if the level is above the treatment line, if there is doubt about the time of ingestion, or if the patient has nausea or vomiting. After 12–15hr, the graph is less reliable and some laboratories may have a higher limit of detection than the treatment line. If there is any doubt, treat with acetylcysteine, especially if ALT is ↑, even if the level is below the treatment line.

### Management at >24hr from ingestion

Measure paracetamol level, LFTs, U&E, creatinine, INR, and ABG. Start treatment with IV acetylcysteine if the patient is clinically jaundiced or has hepatic tenderness. Otherwise, wait for investigation results—treat with acetylcysteine if abnormal and seek advice from NPIS or a liver unit. If the patient is asymptomatic, with non-detectable paracetamol level and normal bloods, treatment is not required.

### Management of staggered overdose

Commence acetylcysteine and take bloods at least 4hr after the last ingestion. If the patient is asymptomatic, has normal ALT and U&E, INR <1.3, and a paracetamol level <10mg/L, consider discontinuing acetylcysteine.

Treat similarly for patients with therapeutic excess—see TOXBASE.

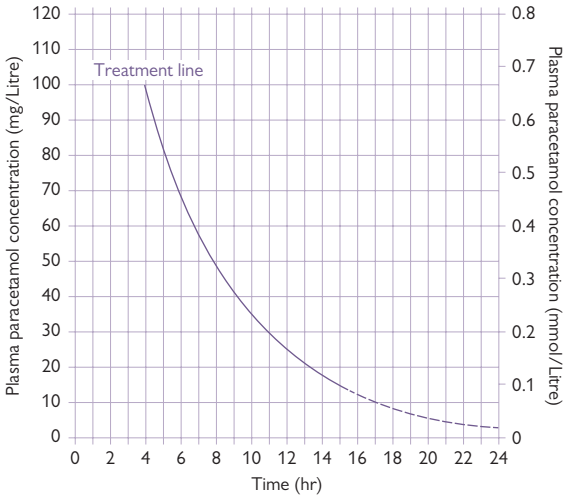


**Fig. 4.1** Paracetamol treatment graph.

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*Note*: check whether the laboratory reports paracetamol in mg/L or mmol/L. Start treatment at once if in doubt about the time of the overdose or if the plasma paracetamol level is only just below the treatment line.

# Tricyclic antidepressant poisoning

Tricyclic antidepressants are highly toxic in overdosage. They have anticholinergic effects. They also cause α-receptor and $Na^+$ channel blockade.

## Clinical features

Commonly: tachycardia, dry skin, dry mouth, dilated pupils, urinary retention, ataxia, jerky limb movements, and drowsiness leading to coma. Unconscious patients often have a divergent squint, ↑ muscle tone and reflexes, myoclonus, and extensor plantar responses. The pupils may be dilated and unreactive. In deep coma, there may be muscle flaccidity with undetectable reflexes and respiratory depression requiring IPPV. Fits occur in ~10% of unconscious patients and may precipitate cardiac arrest. Patients recovering from coma often suffer delirium with hallucinations and have jerky limb movements and severe dysarthria.

## ECG changes

Sinus tachycardia is usual, but as poisoning worsens, the PR interval and QRS duration ↑. These help to confirm clinical diagnoses of tricyclic poisoning in unconscious patients. The P wave may be superimposed on the preceding T wave, so the rhythm can look like VT when it is sinus tachycardia with prolonged conduction. In severe poisoning, ventricular arrhythmias and bradycardia may occur, especially in hypoxic patients (see Figs. 4.2, 4.3, and 4.4).

## Management

- Clear airway, intubate, and ventilate if necessary, and give nursing care.
- Observe continuously, in view of the potential for rapid deterioration.
- Monitor ECG and check ABG in unconscious or post-ictal patients.
- Consider activated charcoal by mouth or gastric tube if a toxic dose has been taken within 1hr—see TOXBASE.
- Do not give anticonvulsants for single brief fits, but give lorazepam or diazepam IV if fits are frequent or prolonged.
- Most arrhythmias occur in unconscious patients within a few hours of overdose. Treat arrhythmias by correcting hypoxia and acidosis. *Sodium bicarbonate* (8.4%, adult: 50–100mL IV; child: 1mL/kg) may dramatically improve the cardiac rhythm and output (by altering protein binding and ↓ active free tricyclic drug). Consider further bicarbonate, depending on the clinical response (especially hypotension), ECG (especially QRS >120ms), and arterial pH. Aim for pH 7.5–7.55, avoiding excessive alkalosis (pH >7.65), which may be fatal.
- Avoid anti-arrhythmic drugs. If arrhythmias do not respond to bicarbonate, discuss with a poisons specialist (see ➲ National Poisons Information Service, pp. 188–9).
- Correct hypotension by giving IV fluids. Glucagon may help in severe hypotension (see ➲ Beta-blocker poisoning, p. 206). Vasopressors or inotropes may be required, on specialist advice.
- Consider Intralipid® (see ➲ Lipid emulsion (Intralipid®) therapy for drug toxicity, p. 195) for severe arrhythmias or cardiac arrest.
- Do not use physostigmine or flumazenil (risk of precipitating fits).
- Unconscious patients usually improve in ~12hr and regain consciousness in 36hr. In extreme cases, veno-arterial extracorporeal membrane oxygenation or cardiac bypass may be required.

## ECG changes in tricyclic antidepressant poisoning

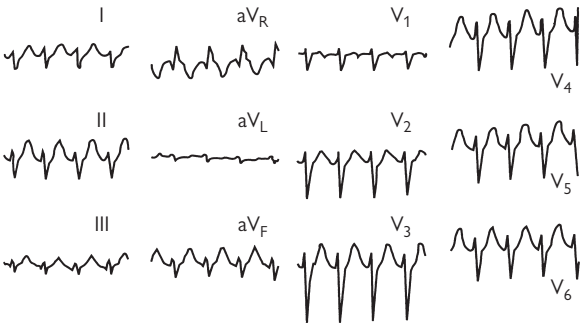


**Fig. 4.2** ECG in tricyclic antidepressant poisoning, showing sinus tachycardia with prolonged conduction, which may be mistaken for VT.

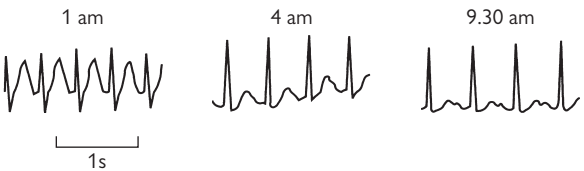


**Fig. 4.3** Serial ECG rhythm strips in amitriptyline poisoning, showing spontaneous recovery with supportive care.

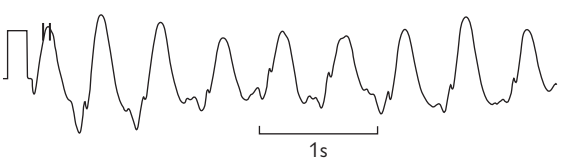


**Fig. 4.4** ECG trace in very severe tricyclic antidepressant poisoning. The patient was unconscious, with GCS of 3, and was intubated and ventilated, with BP of 70/50mmHg.

# Benzodiazepine poisoning

Benzodiazepines (eg diazepam, nitrazepam, temazepam) rarely cause serious poisoning when taken alone in overdose. However, they potentiate other CNS depressants (eg alcohol, tricyclics, and barbiturates).

### Clinical features

Drowsiness, dizziness, ataxia, and dysarthria. Rarely, coma, respiratory depression, and mild hypotension. Fatal poisoning is unusual but may occur from respiratory depression in elderly patients and those with chronic COPD.

### Management

Clear the airway and maintain ventilation if necessary. Provide supportive care. Consider giving activated charcoal if the patient has taken a potentially toxic dose within the past 1hr.

Many benzodiazepines have long-acting metabolites, which may affect driving and other motor skills for several days or even weeks after an overdose. Give appropriate warnings about this.

*Flumazenil* is a specific benzodiazepine antagonist. It reverses the effects of benzodiazepines within 1min but has a short duration of action (<1hr)—as a result, toxic effects may recur. Flumazenil can cause convulsions and cardiac arrhythmias and may precipitate a withdrawal syndrome in patients who are dependent on benzodiazepines. It is particularly dangerous in patients with combined benzodiazepine and tricyclic antidepressant poisoning, in whom it may cause convulsions and cardiac arrest. Flumazenil can be considered to improve ventilation in patients who would otherwise require mechanical ventilation.

# Clomethiazole poisoning

Clomethiazole overdosage may cause coma, respiratory depression, ↓ muscle tone, hypotension, and hypothermia. Excessive salivation and a characteristic smell of clomethiazole on the breath are often noticeable. Treat supportively. In severe cases, consider flumazenil and IPPV.

# Phenothiazine poisoning

Phenothiazines (eg chlorpromazine), butyrophenones (eg haloperidol), and related drugs are used as antipsychotics and antiemetics. In overdosage, they may cause drowsiness, coma, hypotension, and hypothermia. Deep coma and respiratory depression are uncommon. Some conscious patients suffer *dystonic reactions* with oculogyric crises and muscle spasms causing torticollis or opisthotonus. Convulsions may occur. ECG changes of prolonged PR, QRS, and ST intervals and arrhythmias are seen particularly with thioridazine poisoning.

Treat supportively. Activated charcoal may help. If cardiac arrhythmias occur, correct hypoxia, acidosis, and electrolyte abnormalities before giving any anti-arrhythmic drug. Consider sodium bicarbonate if QRS is >120ms. Treat dystonic reactions with procyclidine (5–10mg IV or IM), or alternatively diazepam.

# Barbiturate poisoning

Barbiturate poisoning may cause coma, respiratory depression, hypotension, and hypothermia, with effects potentiated by alcohol. There are no specific neurological signs. Skin blisters and rhabdomyolysis may result from prolonged immobility. Treat supportively, with IPPV if necessary. Repeated doses of activated charcoal may help remove barbiturates. Very rarely, charcoal haemoperfusion or haemodialysis helps in some patients with deep/prolonged coma and respiratory complications.

# Lithium poisoning

**Clinical features** Often due to chronic accumulation from therapeutic overdosage or drug interactions (eg with diuretics or NSAIDs), rather than self-harm. Acute-on-chronic overdose is particularly likely to cause serious toxicity. Symptoms may start up to 24hr after an overdose, especially with slow-release tablets. Nausea, vomiting, and diarrhoea are followed by tremor, ataxia, confusion, ↑ muscle tone, and clonus. In severe cases, there may be convulsions, coma, and renal failure. Lithium-induced nephrogenic diabetes insipidus may complicate treatment.

**Investigations** Measure lithium levels immediately in those on chronic therapy, and at 6hr if lithium-naive (plain tube, not lithium heparin!). Check U&E, creatinine, LFTs, $Ca^{2+}$, and TFTs. Therapeutic lithium levels are 0.4–1.0mmol/L. In poisoning, interpret levels with caution, with treatment dictated by the type of overdose. Toxic effects are often seen at >1.5mmol/L. Soon after a large overdose, higher levels may occur with few clinical effects, before lithium is distributed to tissues.

**Management** Activated charcoal does not absorb lithium. Whole-bowel irrigation (see ➲ Whole-bowel irrigation, p. 193) may be considered for slow-release tablets—discuss this with a poisons specialist (see ➲ National Poisons Information Service, pp. 188–9). Use standard supportive measures and control convulsions with diazepam. Observe all patients for >24hr. Give oral fluids in conscious patients. Forced diuresis is contraindicated. Haemodialysis is the best treatment in severe poisoning but often has to be repeated because of rebound release of lithium from tissue stores.

# Sulfonylurea poisoning

Overdosage causes hypoglycaemia, which may recur over several days after long-acting drugs such as glibenclamide. Check U&E, glucose, and LFTs. Correct hypoglycaemia with PO or IV glucose (see ➲ Hypoglycaemia, pp. 158–9). Observe and check BMG hourly. To prevent recurrent hypoglycaemia, give 10% glucose IVI; in severe cases, 20% glucose may be needed, via a central line because of venous irritation. Hypokalaemia may occur. In severe poisoning, get expert advice (see ➲ National Poisons Information Service, pp. 188–9) and consider octreotide (unlicensed indication) which blocks pancreatic insulin release—initial dose for adults 50mcg SC or IV. Consider discharge after 6hr (12hr in sustained-release preparations) if asymptomatic and normal BMG not requiring glucose.

# Beta-blocker poisoning

## Clinical features

Overdose usually causes sinus bradycardia, but the heart rate can be normal. Hypotension, coma, convulsions, and cardiac arrest may occur. ECG changes include marked QRS prolongation and ST and T wave abnormalities. Propranolol may cause bronchospasm in asthmatics and hypoglycaemia in children. Sotalol can cause prolonged QTc and VT, with torsades de pointes.

## Management

Monitor ECG, heart rate, and BP. Obtain venous access. Check U&E, $Ca^{2+}$, FBC, and blood glucose. Consider activated charcoal (see ➲ Activated charcoal, p. 192). Bradycardia and hypotension may respond to atropine (0.5–1.2mg for adult; 0.02mg/kg for child), but this is often ineffective.

*Glucagon* is the best treatment for severe cardiotoxicity and seems to work by activating myocardial adenylyl cyclase in a way not blocked by β-blockade. Glucagon 5–10mg IV (50–150mcg/kg for a child) usually produces a dramatic improvement in pulse and BP, with return of cardiac output and consciousness. Glucagon often causes vomiting—anticipate this and position the patient appropriately. In severe poisoning, glucagon has a transient effect on cardiac output—commence an infusion after the initial bolus (adults: 50–150mcg/kg/hr; children: 50mcg/kg/hr).

If glucagon is unavailable or ineffective, consider vasopressors (eg metaraminol) or inotropes (eg adrenaline) under expert guidance—see TOXBASE.

Cardiac pacing is an option for bradycardia but is often ineffective. Occasionally, circulatory support is needed by prolonged chest compressions or extracorporeal cardiac bypass whilst more glucagon is obtained or the β-blocker metabolized. Consider insulin therapy (see ➲ Insulin therapy in poisoning, p. 195) for severe hypotension, and Intralipid® (see ➲ Lipid emulsion (Intralipid®) therapy for drug toxicity, p. 195) in cardiac arrest.

# Calcium antagonist poisoning

Poisoning with verapamil, nifedipine, diltiazem, or other $Ca^{2+}$ channel blockers is rare but may be fatal. Nausea, vomiting, dizziness, and confusion may occur. Bradycardia and AV block may lead to AV dissociation, with profound peripheral vasodilatation, hypotension, and cardiac arrest (especially in patients taking β-blockers). Metabolic acidosis, hyperkalaemia, and hyperglycaemia may occur.

Treat supportively—monitor ECG and BP. Consider charcoal. Check U&E, glucose, and $Ca^{2+}$. Give atropine (0.5–1.2mg; child 0.02mg/kg) for symptomatic bradycardia. Pacing may be needed. Calcium chloride (0.2mL/kg of 10% IV up to 10mL over 5min, observing ECG) may ↓ prolonged intracardiac conduction—consider repeating every 10–20min up to four doses. Glucagon may help, as in β-blocker poisoning. Inotropic support with dobutamine, isoprenaline, adrenaline, or high-dose insulin therapy (see ➲ Insulin therapy in poisoning, p. 195) may be needed to maintain cardiac output. In severe poisoning or cardiac arrest, consider Intralipid® (see ➲ Lipid emulsion (Intralipid®) therapy for drug toxicity, p. 195).

# Digoxin poisoning

Toxicity from the therapeutic use of digoxin is relatively common. Acute poisoning is rare but may be fatal. Similar effects occur with digitoxin and, very rarely, with plants containing cardiac glycosides (foxglove, oleander, and yew).

### Clinical features

Nausea, vomiting, malaise, delirium, and xanthopsia (yellow flashes or discoloration of vision). Acute poisoning usually causes bradycardia with PR and QRS prolongation. There may be AV block, AV dissociation, and escape rhythms, sometimes with ventricular ectopics or VT. Hyperkalaemia occurs and, in severe cases, metabolic acidosis due to hypotension and ↓ tissue perfusion.

### Management

Provide supportive treatment. Monitor ECG and BP. Obtain venous access. Give activated charcoal to ↓ absorption and prevent entero-hepatic recycling of digoxin (see Activated charcoal, p. 192). Measure U&E, $Mg^{2+}$, plasma digoxin (after 6hr), and ABG/VBG in severe poisoning. Get expert help for severely poisoned patients. Digoxin levels do not correlate well with toxic features, but treat hyperkalaemia actively. Use digoxin-specific antibodies for severe bradycardia, VT, and severe hyperkalaemia ($K^+$ >6.5mmol/L)—consider repeat doses. Correct severe metabolic acidosis with sodium bicarbonate. Bradycardia and AV block often respond to atropine IV (total 1.2mg; child 0.02mg/kg). Cardiac pacing may be helpful. Treat severe poisoning with digoxin antibodies (DigiFab®), which, together with insulin/glucose, may rapidly reduce serum $K^+$ level. Anaphylactoid reactions occur in up to a quarter of patients.

# ACE inhibitor poisoning

Overdosage with ACE inhibitors (eg captopril, enalapril, lisinopril) may cause drowsiness, hypotension, hyperkalaemia, and, rarely, renal failure. Monitor BP and ECG. Give IV 0.9% saline if BP is low. Check U&E, FBC, and LFTs. Consider activated charcoal (see Activated charcoal, p. 192).

# Theophylline poisoning

Theophylline and aminophylline can cause fatal poisoning. Many preparations are slow-release and may not produce serious toxicity until 12–24hr after ingestion, so ensure careful observation.

### Features

Nausea, vomiting (often severe and not helped by antiemetics), abdominal pain, haematemesis, restlessness, ↑ muscle tone, ↑ reflexes, headache, and convulsions. Coma, hyperventilation, hyperpyrexia, and rhabdomyolysis may occur. Sinus tachycardia may be followed by supraventricular and ventricular arrhythmias and VF. BP may initially ↑ but later ↓ in severe poisoning. Complex metabolic disturbances include respiratory alkalosis, followed by metabolic acidosis, hyperglycaemia, severe hypokalaemia, and hypomagnesaemia.

### Management

- Treat supportively and monitor ECG, heart rate, and BP.
- Measure U&E, $Ca^{2+}$, $Mg^{2+}$, $PO_4^-$, glucose, ABG, and plasma theophylline (repeated after a few hours). If symptomatic, repeat $K^+$ hourly, as correcting hypokalaemia may prevent dangerous arrhythmias. Correct hypokalaemia with $K^+$ (no faster than 20mmol/hr).
- Consider gastric lavage if <1hr since ingestion. Give repeated activated charcoal (see ➲ Activated charcoal, p. 192), by NG tube if necessary.
- Consider ondansetron (8mg slowly IV in adult) for intractable vomiting.
- GI bleeding may require transfusion and ranitidine.
- Observe closely if there is tachycardia with an adequate cardiac output. Non-selective β-blockers (eg propranolol) may help severe tachyarrhythmias and hypokalaemia, but cause bronchospasm in asthmatics. Lidocaine and mexiletine may precipitate fits, so disopyramide is preferable for ventricular arrhythmias.
- Control convulsions with diazepam or lorazepam.
- Treat ventricular arrhythmias with direct current (DC) cardioversion.
- Consider charcoal haemoperfusion or haemodialysis in severe poisoning, especially if PO or NG activated charcoal is impracticable due to vomiting. Serious hyperkalaemia may occur during recovery from theophylline poisoning if large amounts of $K^+$ were given earlier.

# Salbutamol poisoning

Poisoning with $\beta_2$-agonists may cause vomiting, agitation, tremor, tachycardia, palpitations, hypokalaemia, and hypertension. Rarely, hallucinations, hyperglycaemia, delayed hypoglycaemia, ventricular tachyarrhythmias, myocardial ischaemia, and convulsions occur.

Treat supportively:

- Correct hypokalaemia by infusion of $K^+$ (max 20mmol/hr).
- Monitor ECG and BP.
- Activated charcoal may ↓ drug absorption.
- Do not treat tachycardia if there is an adequate cardiac output. Metoprolol or esmolol may help severe tachyarrhythmias and hypokalaemia, but can precipitate bronchospasm in asthmatics.

# Iron poisoning

Iron tablets may resemble sweets and so can be ingested by inquisitive children. Serious poisoning is uncommon, but fatalities can occur. Note that iron is present in some weed/seed preparations.

Different preparations contain the equivalent of 35–105mg of elemental iron per tablet, sometimes in slow-release form.

## Features

In the first few hours after ingestion, nausea, vomiting, diarrhoea, and abdominal pain are common. Vomit and stools are often grey or black and may contain blood. Hyperglycaemia and ↑ WCC may occur. Most patients do not develop further features.

*In severe poisoning*, early effects include haematemesis, drowsiness, convulsions, coma, metabolic acidosis, and shock.

Early symptoms settle after 6–12hr, but a few patients then deteriorate 24–48hr after ingestion, with shock, hypoglycaemia, jaundice, metabolic acidosis, hepatic encephalopathy, renal failure, and occasionally bowel infarction. Survivors may develop gastric strictures or pyloric obstruction 2–5 weeks after the overdose.

## Management

- Employ supportive measures, as required.
- Check serum iron, FBC, glucose, LFTs, INR, and also ABG/VBG in severe poisoning. Severe poisoning causes metabolic acidosis and may cause RBCs to haemolyse.
- Do not give charcoal, as it does not absorb iron. Iron tablets are radio-opaque and can be counted on a plain abdominal X-ray film. Whole-bowel irrigation (see ➲ Whole-bowel irrigation, p. 193) may be useful if many tablets remain in the gut, especially with slow-release formulations.
- Obtain expert advice in serious poisoning. Coma and shock indicate severe poisoning needing immediate treatment with *desferrioxamine* by IVI (15mg/kg/hr, until a max of 80mg/kg has been given). If there are features of severe poisoning, commence desferrioxamine prior to obtaining the serum iron concentration. Measurement of total iron-binding capacity may give misleading results after iron poisoning.
- Desferrioxamine causes hypotension if infused too rapidly and can produce rashes and, rarely, anaphylaxis, pulmonary oedema, or ARDS. The iron–desferrioxamine complex makes the urine orange or red, which confirms that free iron has been bound and that desferrioxamine was required.
- Patients who still have no symptoms 6hr after an iron overdose have probably not ingested toxic amounts and may be discharged, with advice to return if symptoms develop.
- Pregnancy does not alter the treatment needed for iron poisoning—use desferrioxamine if indicated.

# Ethanol poisoning

## Features

Alcohol initially causes disinhibition and later ataxia, dizziness, dysarthria, and drowsiness. It potentiates the CNS-depressant effects of many drugs. In addition to the characteristic odour of alcoholic beverages, there may be nystagmus to horizontal gaze.

*Severe poisoning*

Patients who present with severe alcohol intoxication may be comatose, with respiratory depression, hypotension, hypothermia, and metabolic acidosis.

Hypoglycaemia is a particular problem in children who have consumed alcohol and may occur after some hours.

Death may result from respiratory failure or aspiration of vomit. For an adult, the *fatal dose of ethanol* alone is ~300–500mL of absolute alcohol—whisky and gin usually contain 40–50% ethanol. *Note*: the UK legal limit for driving is 80mg/dL; patients who have a blood alcohol level of >350mg/dL are at significant risk of death from acute alcohol intoxication. The rate of clearance of alcohol from the blood varies enormously between individuals, with typical quoted values of 10–20mg/dL/hr in most adults, although this can be much higher in some chronic alcoholics.

Never assume that ↓ GCS is due to alcohol until other causes have been excluded (especially hypoglycaemia, head injury, post-ictal state, meningitis/encephalitis, hepatic encephalopathy, and intoxication with other drugs).

## Management

- Maintain a clear airway and adequate ventilation. Nurse in the recovery position to protect the airway.
- Measure blood alcohol—if low, it may challenge the diagnosis and indicate an alternative aetiology.
- Check blood glucose every 1–2hr in severe poisoning. Correct hypoglycaemia with glucose, not glucagon (unless IV access is difficult).
- Look for signs of injury, especially head injury, and adopt a low threshold for a CT brain scan to search for intracranial haemorrhage.
- Treat co-ingested poisons appropriately.
- If obtunded, check CK for evidence of rhabdomyolysis (especially if there is a history of the patient having been lying for several hours).
- Gastric lavage and activated charcoal are ineffective in ethanol intoxication.
- Involve ICU and consider dialysis in extreme cases.

# Methanol poisoning

Ingestion of 10mL of pure methanol may cause blindness, and 30mL can be fatal, the toxic effects being due to the metabolites formaldehyde and formic acid. *Methylated spirits* contain toxicologically insignificant amounts of methanol—toxicity is almost entirely due to ethanol.

## Clinical features

Methanol initially causes only mild transient drowsiness. Serious toxicity develops after a latent period of 12–24hr with vomiting, abdominal pain, headache, dizziness, blurring of vision, and drowsiness leading to coma. There is severe metabolic acidosis, hyperglycaemia, and ↑ serum amylase. Survivors may be blind from optic nerve damage and develop Parkinsonian problems.

## Management

- Consider gastric lavage if <1hr since ingestion. Do not give charcoal.
- Measure ABG, U&E, $Cl^-$, $HCO_3^-$, glucose, FBC, LFTs, ethanol, osmolality, and plasma methanol if possible—if not available locally, discuss with Clinical Chemistry. Calculate the osmolar gap and anion gap.
- Read TOXBASE advice (see ➲ National Poisons Information Service, pp. 188–9). Discuss with NPIS.
- Observe for at least 6hr after ingestion, even if asymptomatic.
- Early use of fomepizole or ethanol (as for ethylene glycol—see ➲ Ethylene glycol poisoning, p. 212) minimizes methanol toxicity and should be started if poisoning is likely, especially if there is a high anion gap metabolic acidosis.
- Consider sodium bicarbonate to correct metabolic acidosis (aim for pH 7.44). Hypernatraemia is a risk if a large amount is needed.
- Give folinic acid (30mg IV every 6hr for 48hr).
- In severe poisoning, refer to ICU for haemodialysis and possibly IPPV.

# Ethylene glycol poisoning

Ethylene glycol is used mainly as antifreeze. The fatal dose for an adult is about 100g (90mL of pure ethylene glycol). Toxic effects are due to the metabolites glycolaldehyde, glycolic acid, and oxalic acid. Fomepizole or ethanol block ethylene glycol metabolism, preventing toxicity.

## Clinical features

In the first 12hr after ingestion, the patient appears drunk but does not smell of alcohol. Ataxia, dysarthria, nausea, vomiting, and sometimes haematemesis occur, followed by convulsions, coma, and severe metabolic acidosis.

From 12–24hr after ingestion, hyperventilation, pulmonary oedema, tachycardia, cardiac arrhythmias, and cardiac failure may develop. Hypocalcaemia may be severe. Acute tubular necrosis and renal failure occur at 24–72hr. Cranial nerve palsies may develop.

Urine microscopy shows calcium oxalate monohydrate crystals which are diagnostic of ethylene glycol poisoning. Some makes of antifreeze contain fluorescein, which makes urine fluoresce in ultraviolet light (eg a Wood's lamp from a dermatology department). This helps to confirm ethylene glycol poisoning, but the absence of fluorescence does not exclude poisoning.

## Management

- Consider gastric lavage if <1hr since ingestion. Do not give charcoal.
- Measure ABG, U&E, $Cl^-$, $HCO_3^-$, glucose, FBC, LFTs, osmolality, and plasma ethylene glycol if possible (check with the local Clinical Chemistry department). Calculate the osmolar gap and anion gap.
- Read TOXBASE advice and discuss with NPIS (see ➲ National Poisons Information Service, pp. 188–9).
- Observe for at least 6hr after ingestion, even if asymptomatic.
- Monitor ECG, pulse, BP, RR, and urine output.
- High anion gap metabolic acidosis (see ➲ The anion gap, pp. 102–3) occurs in ethylene glycol poisoning (and also methanol poisoning, DKA, alcoholic ketoacidosis, and renal failure), but acidosis only develops after some ethylene glycol has been metabolized.
- Early use of fomepizole or ethanol minimizes toxicity, so commence this if poisoning is likely. Consider fomepizole—discuss with NPIS (see ➲ National Poisons Information Service, pp. 188–9) about indications, dosage, and where to obtain it.
- If fomepizole is not available, give a loading dose of ethanol PO as whisky, gin, or vodka (40% ethanol, 2.5mL/kg PO or 10mL/kg of 10% ethanol IV over 30min). Follow by IVI of ethanol—the dose depends on the usual alcohol consumption and whether dialysis is being used.
- Use sodium bicarbonate to correct metabolic acidosis which has not improved with adequate ventilation and fluid resuscitation (aim for pH 7.44). Large amounts may be needed and hypernatraemia may occur.
- Correct hypocalcaemia with calcium gluconate (10–20mL of 10% slowly IV) only if there are seizures or QTc >500ms. Note that correction risks calcium oxalate stone formation.
- Correct hypomagnesaemia.
- Consider haemodialysis in severe poisoning, with frequent measurements of blood ethylene glycol concentrations (and ethanol if this is used) ± ICU and IPPV.

# Paraquat poisoning

Paraquat is a weedkiller which is very toxic if ingested—death is likely after 10mL of liquid paraquat ingestion. Paraquat poisoning is now rare in the UK where paraquat is no longer approved for sale or use.

### Clinical features of paraquat ingestion

Paraquat is corrosive and causes immediate burning pain in the mouth and throat, nausea, and vomiting, followed by abdominal pain and diarrhoea. Large amounts result in rapid deterioration, with shock, pulmonary oedema, metabolic acidosis, coma, convulsions, and death within 24hr.

Paraquat lung usually develops by 5–7 days, with pulmonary oedema and fibrosis causing breathlessness and cyanosis. Lung shadowing is seen on CXR. Death from hypoxia occurs 7–14 days after poisoning.

### Management

- Avoid supplemental $O_2$, where possible, as it may ↑ pulmonary toxicity.
- Avoid gastric lavage as oesophageal perforation may occur.
- Consider activated charcoal PO.
- Measure lactate—this may be of prognostic value.
- Send urine to test for paraquat (TOXBASE can advise which hospital labs provide this). A negative test after 4hr of suspected ingestion excludes significant poisoning.

# Petrol and paraffin poisoning

Petrol, paraffin (kerosene), and other petroleum distillates contain mixtures of hydrocarbons, often with small quantities of other chemicals. Unintentional poisoning occurs after liquids have been stored in inappropriate containers or siphoning fuel from a vehicle. The major problem is pneumonitis caused by aspiration of hydrocarbons into the lungs.

### Clinical features

In many cases, no symptoms occur. There may be nausea, vomiting, and occasionally diarrhoea. Aspiration into the lungs causes choking, coughing, wheeze, breathlessness, cyanosis, and fever. X-ray changes of pneumonitis (shadowing in the mid or lower zones) may occur without respiratory symptoms or signs. Occasionally, pleural effusions or pneumatoceles develop. In severe cases, there may be pulmonary oedema, drowsiness, convulsions, or coma.

### Management

Many patients remain well and need no treatment. Avoid gastric lavage. Aim to obtain a CXR at 6–8hr, but request this earlier if required.

Discharge (with advice to return if symptoms develop) those patients who are free of symptoms or signs 8hr after ingestion.

If symptoms occur, treat supportively with $O_2$ and bronchodilators, together with steroids. Consider CPAP/IPPV if the patient deteriorates.

# Organophosphate poisoning

Organophosphates are widely used as insecticides. Poisoning is rare in the UK, but common in many developing countries. Organophosphates are absorbed through the skin, bronchial mucosa, and gut and inhibit cholinesterases, causing accumulation of acetylcholine at nerve endings and neuromuscular junctions. The speed of onset, severity, and duration of toxicity vary between different compounds. Irreversible binding of cholinesterase ('ageing') develops after some minutes or hours. Pralidoxime reactivates cholinesterase if given promptly, before ageing occurs.

*Organophosphate nerve gas agents*, such as sarin, may be released deliberately by terrorists. Information is available from TOXBASE (see ➲ National Poisons Information Service, pp. 188–9).

*Carbamate insecticides* act similarly to organophosphates, but poisoning with carbamates is generally less severe and pralidoxime is not needed.

## Clinical features

Minor exposure to organophosphates may cause subclinical poisoning with ↓ cholinesterase levels, but no symptoms or signs. Symptoms may be delayed by 12–24hr after skin exposure.

*Early features of toxicity* include anxiety, restlessness, insomnia, tiredness, headache, nausea, vomiting, abdominal colic, diarrhoea, sweating, hypersalivation, and miosis. Muscle weakness and fasciculation may develop.

*In severe poisoning*, there is widespread paralysis with respiratory failure, pulmonary oedema, profuse bronchial secretions, bronchospasm, convulsions, and coma. Hyperglycaemia and cardiac arrhythmias may occur. Occasionally, delayed effects of poisoning develop 1–4 days after acute poisoning, with cranial nerve palsies, muscle weakness, and respiratory failure which resolve after 2–3 weeks. Peripheral neuropathy may develop after 2 weeks, usually involving the legs.

## Management

- Ensure all staff in contact with the patient wear protective clothing to avoid getting contaminated.
- Provide supportive treatment as needed.
- Clear the airway and remove secretions. Give $O_2$ and IPPV if needed.
- Insert IV cannulae. Take blood for cholinesterase (EDTA tube on ice).
- Give diazepam to treat agitation and control convulsions.
- If there are profuse bronchial secretions or bronchospasm, give atropine IV (adult 2mg; child 0.02mg/kg), repeated every 5min, with the dose doubled each time until the chest sounds clear, systolic BP >80mmHg, and pulse >80. Some patients need >100mg of atropine. If atropine is required, give pralidoxime in addition.
- In moderate or severe poisoning, give pralidoxime. The dose of pralidoxime is 30mg/kg IV over 30min, followed by an IVI at 8mg/kg/hr. Improvement is usually apparent within 30min.
- NPIS can advise on pralidoxime supply and use. In a terrorist incident, the UK has stocks of antidotes in reserve for mass casualty poisonings.

# Cyanide poisoning

Cyanide compounds are widely used in industry and may be ingested or inhaled inadvertently or deliberately. Cyanides produced by burning polyurethane foam ↑ mortality from smoke inhalation—if there is severe acidosis, consider cyanide toxicity. Cyanide poisoning may be caused by the drug sodium nitroprusside or ingestion of amygdalin (laetrile) from the kernels of apricots, cherries, and other fruits. Solutions for removing artificial fingernails may contain acetonitrile (methyl cyanide).

*Cyanides inhibit cytochrome oxidase*, blocking the tricarboxylic acid cycle and stopping cellular respiration. This process is reversible. Inhalation of hydrogen cyanide often causes death within minutes. Ingestion of cyanides can produce rapid poisoning, but food in the stomach may delay absorption and the onset of symptoms. Delayed poisoning may follow absorption of cyanides through the skin. Ingested cyanide compounds react with gastric acid to form hydrogen cyanide, which could poison first-aiders giving mouth-to-mouth resuscitation.

## Clinical features

Acute poisoning causes dizziness, anxiety, headache, palpitations, breathlessness, and drowsiness. In severe cases, there may be coma, convulsions, paralysis, pulmonary oedema, cardiac arrhythmias, and cardiorespiratory failure, with metabolic acidosis. Most of the clinical features result from severe hypoxia, but cyanosis is uncommon. Classically, there is a smell of bitter almonds on the breath, but many people cannot detect this.

## Management

- Avoid staff getting contaminated.
- Provide supportive measures—give 100% $O_2$ and monitor ECG.
- Remove contaminated clothing and wash exposed skin.
- Consider activated charcoal or gastric lavage within 1hr of ingestion.
- In mild poisoning, reassurance, $O_2$, and observation may be all that is required. Exposure to cyanide causes great anxiety—it may be hard to distinguish fear of poisoning and early symptoms of toxicity.
- *Specific antidotes* should be available but are not always needed.

*Some specific antidotes* to cyanide are dangerous in the absence of cyanide—only give if poisoning is moderate or severe (eg coma). In severe cyanide poisoning, give *dicobalt edetate* (Kelocyanor®) 300mg IV over 1min, repeated if there is no improvement after 1min. In the absence of cyanide, dicobalt edetate may cause cobalt poisoning with facial, laryngeal, and pulmonary oedema, vomiting, tachycardia, and hypotension. In mild poisoning, treat with *sodium thiosulfate* (adult dose 25mL of 50% solution IV over 10min; child 400mg/kg) or with *sodium nitrite* (adult dose 10mL of 3% solution IV over 5–20min; child dose 0.13–0.33mL of 3% solution/kg, ie 4–10mg/kg). Sodium thiosulfate often causes vomiting. Sodium nitrite may cause hypotension. High doses of *hydroxocobalamin* (5g IV over 15min, Cyanokit®) are useful and relatively safe in cyanide poisoning, especially in victims of smoke inhalation.

# Carbon monoxide poisoning

*Carbon monoxide (CO)* is a tasteless and odourless gas produced by incomplete combustion. Poisoning may occur from fires, (old) car exhausts, and faulty gas heaters. CO is also produced by metabolism of methylene chloride (used in paint strippers and as an industrial solvent). CO ↓ the $O_2$-carrying capacity of blood by binding haemoglobin (Hb) to form carboxyhaemoglobin (COHb). This impairs $O_2$ delivery from blood to the tissues and also inhibits cytochrome oxidase, blocking $O_2$ utilization. These effects combine to cause severe tissue hypoxia.

*The elimination half-life of CO* is 320min on breathing air, 80min on 100% $O_2$, and 23min on $O_2$ at 3 atmospheres pressure.

## Clinical features

Early features are headache, malaise, nausea, and vomiting (sometimes misdiagnosed as a viral illness or gastroenteritis, especially if several members of a family are affected).

*In severe poisoning*, there is coma with hyperventilation, hypotension, ↑ muscle tone, ↑ reflexes, extensor plantars, and convulsions. Cherry-red colouring of the skin may be seen in fatal CO poisoning but is rare in live patients. Skin blisters and rhabdomyolysis may occur after prolonged immobility. Pulmonary oedema, MI, and cerebral oedema can occur. Neurological and psychiatric problems sometimes develop later.

## Management

- Clear the airway and maintain ventilation with as high a concentration of $O_2$ as possible. For a conscious patient, use a tight-fitting mask with an $O_2$ reservoir, but if unconscious, intubate and provide IPPV on 100% $O_2$.
- Record ECG and monitor cardiac rhythm—look for arrhythmias and signs of acute MI.
- Check VBG or ABG—$SpO_2$ measurements are misleading in CO poisoning, as are $p_aO_2$ values, but acidosis indicates tissue hypoxia.
- Check COHb levels (in blood or with a special pulse oximeter)—although these correlate poorly with clinical features, COHb >20% after arrival at hospital suggests serious poisoning. COHb may be up to 8% in smokers without CO poisoning. A nomogram (see ➲ Nomogram of decay of COHb with time, p. 403) can help to estimate COHb at the time of exposure.
- Correct metabolic acidosis by ventilation and $O_2$—try to avoid bicarbonate, which may worsen tissue hypoxia.
- Consider mannitol if cerebral oedema is suspected.
- *Hyperbaric $O_2$* therapy is logical, but of no proven benefit for CO poisoning. Transfer to a hyperbaric chamber and pressurization may take hours, and so hyperbaric treatment may be no more effective than ventilation on 100% normobaric $O_2$. Caring for a critically ill patient in a small-pressure chamber may be impracticable. NPIS no longer recommends hyperbaric $O_2$ treatment. Previously used criteria were: if a patient has been unconscious at any time, has COHb >20%, is pregnant, or has cardiac complications or neurological or psychiatric features. Details of some hyperbaric chambers are shown in Table 6.2.

## Chlorine poisoning

Chlorine gas causes lacrimation, conjunctivitis, coughing, wheezing, breathlessness, and chest pain. Laryngeal and pulmonary oedema may develop within a few hours.

- Give $O_2$, with bronchodilators if necessary. If there is laryngeal or pulmonary oedema, get senior help and give prednisolone in high dosage (adult 60–80mg/day initially). In severe cases, consider the need for tracheal intubation, IPPV, and admission to ICU.
- If the eyes are painful, irrigate with water or saline, and examine with fluorescein for corneal damage.
- Allow home casualties with minor exposure to chlorine but no symptoms, with advice to rest and return if symptoms develop.
- Patients with symptoms when seen in hospital usually need admission for at least 12hr for observation.

## CS gas (tear gas)

*CS (orthochlorobenzylidene malononitrile)* is used for riot control and police self-protection. It is an aerosol or smoke, rather than a gas. Exposure to CS causes immediate blepharospasm and lacrimation, uncontrollable sneezing and coughing, a burning sensation in the skin and throat, and tightness of the chest. Vomiting may occur. These symptoms usually improve within 10min in fresh air, but conjunctivitis may persist for 30min. Exposure in a confined space may cause symptoms for some hours and is particularly dangerous in people with pre-existing lung disease. Redness or blistering of the skin may develop, due to the solvent in the spray.

*Treat* patients exposed to CS gas in a well-ventilated area. Ensure that staff wear gloves and close-fitting goggles. Remove contaminated clothes and wash affected skin thoroughly. Remove contact lenses, and give $O_2$ and bronchodilators if necessary. Reassure the patient that the symptoms will resolve.

If the eyes remain painful, instil local anaesthetic drops and irrigate them with water or saline. When symptoms have settled, record visual acuity and examine the corneas using fluorescein. Refer to an ophthalmologist if symptoms persist.

*CN gas (chloroacetophenone)* is used in some countries for riot control and in personal defence devices. CN has similar effects to CS but is more toxic.

# Chemical incidents

Chemical incidents involving single or multiple casualties may result from accidents (eg release of chlorine gas) or deliberate release of chemicals (by terrorists or others). CBRN (chemical, biological, radiological, and nuclear) incidents have many features in common.

If you know or suspect that a patient has been involved in a chemical incident:

- Inform senior ED staff.
- Avoid contaminating other staff or patients.
- Ensure that you are wearing suitable PPE, unless the patient has already been decontaminated.
- Decontaminate the patient according to departmental guidelines if this has not been done already (see ➲ Decontamination of patients, p. 218).
- Resuscitate as necessary—airway, breathing, and circulation.
- Assess the clinical features and toxic agent.
- Give antidotes if appropriate, and reassess the patient.
- Enquire whether other patients are expected.
- Inform the local health protection team.
- Get expert advice from TOXBASE (see ➲ National Poisons Information Service, pp. 188–9) or the Department for Environment, Food, and Rural Affairs (Defra) CBRN emergency contact: tel 0300 1000 316.
- If deliberate release is suspected, inform the police and involve other agencies and the press officer.

Chemicals which might cause a chemical incident include: chlorine—see ➲ Chlorine poisoning, p. 217; CS gas (tear gas)—see ➲ CS gas (tear gas), p. 217; cyanide—see ➲ Cyanide poisoning, p. 215; and organophosphates—see ➲ Organophosphate poisoning, p. 214.

## Information about chemical incidents

- Liaise with NPIS.
- TOXBASE (see ➲ National Poisons Information Service, pp. 188–9) gives details of toxicity and antidotes, with medical, public health, and public briefing documents about 60 chemicals that might be deliberately released.

*Infection control and prevention* See ➲ Infection control and prevention, pp. 36–7.

*Major incidents* See ➲ Major incidents, pp. 40–1.

*Radiation incidents* See ➲ Radiation incidents, pp. 278–9.

## Decontamination of patients

Decontamination after exposure to a chemical, biological, or radiation hazard is intended to reduce the risks to the patient and to other people.

Casualties should be decontaminated at the scene after a CBRN incident, but some contaminated patients may arrive at the ED without warning. Many people will be worried about contamination but not actually be at risk. Even with advance planning, it will be challenging to organize the ED, keep 'clean' areas clean, maintain order, and communicate between the 'decon' team (in PPE) and other ED staff.

# Plants, berries, and mushrooms

## Plants and berries

Many children eat plant leaves or brightly coloured berries, but serious poisoning from plants is very rare. Identify the plant if possible, using reference books. Advice on toxicity and any necessary treatment is available from Poisons Information Services. Many garden and house plants are non-toxic and no treatment is needed after ingestion.

Serious poisoning from *laburnum* is very rare, with only one death recorded in the UK in 50y. No treatment needs to be provided for children who eat laburnum seeds, except for the very few with symptoms (nausea, salivation, vomiting, headache, and rarely convulsions).

## Mushroom poisoning

Serious poisoning from mushrooms or fungi is rare. Most deaths are due to *Amanita phalloides* (death cap mushroom). Reference books are useful, but identification of mushrooms from the description or fragments available is often uncertain. Advice on toxicity and treatment is available from Poisons Information Services (see ➲ National Poisons Information Service, pp. 188–9).

Mushrooms found in gardens are unlikely to produce severe poisoning but may cause vomiting and occasionally hallucinations, usually within 2hr of ingestion. Mushrooms which cause symptoms within 6hr are unlikely to be seriously toxic. Delayed toxicity occurs with *Amanita phalloides* and some other species, which grow throughout the UK.

*Amanita phalloides* poisoning causes vomiting and profuse watery diarrhoea after a latent period of 6–12hr, followed by hepatic and renal failure. The interval between ingestion and the onset of symptoms is crucial in distinguishing between non-serious and potentially fatal poisoning.

Try to ascertain if:

- More than one variety of mushroom was eaten (since poisonous and edible mushrooms often grow together).
- Whether the mushrooms were cooked (since some toxins are inactivated by heat).
- Whether alcohol was taken (since disulfiram-like effects may occur with *Coprinus* species, ink cap mushrooms).

For most toxic mushrooms, only symptomatic treatment is required. Activated charcoal may ↓ absorption if given within 1hr. Get expert advice immediately if *Amanita* poisoning is suspected (see ➲ National Poisons Information Service, pp. 188–9).

# Button batteries

Small children often swallow button or disc batteries intended for toys, watches, hearing aids, and other electrical equipment. Older patients sometimes mistake them for tablets.

Larger batteries may become stuck in the oesophagus, causing perforation or, later, stenosis. They can be mistaken for coins on X-ray.

Most batteries that reach the stomach pass through the gut without any problem. Corrosive damage could occur from electrical discharge, but toxicity from battery contents is rare. Mercury poisoning is very unlikely since mercuric oxide batteries are no longer sold. The NPIS may identify the type of battery involved from the reference number, if this is available on the packet or on a similar battery to that ingested.

## Management

See advice on TOXBASE.

X-ray the chest and abdomen or use a metal detector to find the battery. A battery stuck in the oesophagus should be removed immediately by endoscopy, which allows inspection for oesophageal damage.

An asymptomatic child with a battery in the stomach can be sent home, with advice to return if any symptoms develop. If the battery has not been passed after 2 days, use a metal detector or repeat X-ray to look for the battery. If it is still in the stomach (which is rare), refer to consider removal by endoscopy to avoid any risk of perforation or absorption of battery contents. Note that ingestion of honey prior to removal may limit GI injury.

Batteries in the small or large bowel almost always pass spontaneously. Encourage stool inspection, but bear in mind that it may take up to 2 weeks to pass. If abdominal pain, vomiting, diarrhoea, or rectal bleeding occur, an abdominal X-ray is needed to localize the battery, which may require removal by endoscopy or surgery.

## Batteries in the nose or ear

Button batteries lodged in the nose may cause corrosive burns and bleeding, sometimes with septal perforation after a few weeks. A battery in the ear may perforate the tympanic membrane and cause facial nerve injury. Liaise with a ear, nose, and throat (ENT) specialist to remove batteries as soon as possible.

### *Ingestion of magnets*

Occasionally, patients (usually children) ingest magnets. When a single magnet is ingested on its own, it usually passes without incident. However, when more than one magnet has been ingested (or one magnet together with another ferrometallic object), there is a risk of bowel necrosis and perforation—refer for removal.

# Novel psychoactive substances

Previously known as 'legal highs', new or novel pyschoactive substances (NPS) contain one or more chemical substances which produce similar effects to illegal drugs such as cocaine, cannabis, and ecstasy. In the UK, it is currently illegal to sell or distribute NPS, but possession is not a criminal offence.

NPS are often taken with other recreational drugs ± alcohol, which may potentiate their effects. Sometimes, NPS may be adulterated with other chemicals such as caffeine or lidocaine.

NPS comprise an unknown combination of chemicals, and therefore, treatment relies upon identification of the toxidrome and managing the symptoms.

The four groups of NPS are summarized below (most commonly encountered are stimulants and synthetic cannabinoids).

## Stimulant NPS (eg mephedrone)

Synthetic cathinone compounds are sometimes known as 'plant food' or 'bath salts' which are snorted or swallowed. Toxic effects are similar to those of amphetamines: agitation, sweating, tachycardia, palpitations, and hypertension. Some have nausea, hallucinations, fits, muscle spasms, nausea, peripheral vasoconstriction, and myocardial ischaemia. Nasal irritation and epistaxis may occur after snorting these. Treat as for MDMA/amphetamines.

## Cannabinoid NPS

Synthetic cannabinoid receptor agonists (SCRAs), such as 'spice' and 'noids', are often smoked or inhaled (eg using vaporizers). Suspect if there is a history of smoking a herbal product and the patient has features similar to those of cannabis intoxication: relaxation, altered consciousness, and disinhibition.

Some patients may become agitated and confused, with evidence of adrenergic stimulation (see TOXBASE section on SCRAs).

## Depressant (sedative) NPS

Features include reduced conscious level, hypoventilation, and bradycardia. Treat as for benzodiazepine or opioid poisoning (see ➲ Benzodiazepine poisoning, p. 204; ➲ Opioid poisoning, p. 196).

## Hallucinogenic NPS

These may have psychedelic effects (similar to LSD—see ➲ Recreational drugs, pp. 222–3), in which case treat as for tryptamine toxicity (see TOXBASE).

Some patients present with dissociative symptoms (similar to ketamine—see ➲ Ketamine, p. 287), in which case treat as for dissociative drugs, as per TOXBASE.

A general rule for any patient who has taken an unknown recreational drug is that if there are no symptoms present at 4hr and no treatment has been required, then they are safe to be discharged with advice regarding returning for review if symptoms develop.

# Recreational drugs

Toxicity is often seen from heroin (see ➲ Opioid poisoning, p. 196), cocaine, ecstasy, and related drugs. Street names for drugs vary and may be confusing. TOXBASE (see ➲ National Poisons Information Service, pp. 188–9) has lists of slang names about drugs.

Illicit drugs vary in strength and are often mixed with other drugs or chemicals, which may cause unexpected effects. Drugs may be smoked, sniffed ('snorted'), swallowed, or injected. Injecting drug users are at ↑ risk of hepatitis (see ➲ Hepatitis, p. 249), HIV (see ➲ Human immunodeficiency virus, pp. 250–1), necrotizing fasciitis (see ➲ Necrotizing fasciitis, p. 244), botulism (see ➲ Botulism, p. 247), anthrax (see ➲ Anthrax, p. 243), and endocarditis (see ➲ Infective endocarditis, p. 244).

## Ecstasy (MDMA)

'Ecstasy' (3,4-methylenedioxymetamphetamine, MDMA) is an amphetamine derivative used as an illegal stimulant drug. The name 'ecstasy' is also used for benzylpiperazine (BZP), another illegal drug. 'Liquid ecstasy' is GHB (see ➲ Gammahydroxybutyrate (GHB, GBH, 'liquid ecstasy'), p. 223). MDMA is taken PO as tablets or powder, often at raves or parties. Some people who have previously tolerated the drug have idiosyncratic reactions, with severe toxicity from a single MDMA tablet.

MDMA causes release of serotonin, catecholamines, and other hormones. Inappropriate ADH secretion, abnormal thirst, and excessive water intake may result in hyponatraemia and cerebral oedema, especially in women.

### *Clinical features*

Euphoria, agitation, sweating, dilated pupils, ataxia, teeth grinding, headache, tachycardia, and hypertension. Severe poisoning can cause hyperpyrexia, muscle rigidity, rhabdomyolysis, convulsions, coma, cardiac arrhythmias, renal failure, hepatic failure, cerebral haemorrhage, and DIC. Metabolic acidosis is common. Features of serotonin syndrome may occur, as may hypoglycaemia, severe hyponatraemia, and hyperkalaemia.

### *Treatment*

Consider activated charcoal (see ➲ Activated charcoal, p. 192) if <1hr since ingestion. Observe asymptomatic patients for at least 4hr. Monitor ECG, pulse, BP, and T°. Record ECG, and check U&E, creatinine, glucose, LFTs, and CK. Test urine for blood. In severe cases, check ABG and coagulation.

Support ABC. Get expert advice (see ➲ National Poisons Information Service, pp. 188–9) and ICU help in severe poisoning. RSI may be needed because of trismus and fits—avoid suxamethonium which may cause hyperkalaemia. Control agitation with PO or IV diazepam or lorazepam—large doses may be needed. For severe hypertension, give IV diazepam and GTN. Do not treat single short fits, but give diazepam or lorazepam for repeated or prolonged fits.

Correct metabolic acidosis (possibly with sodium bicarbonate), checking ABG and U&E. Treat hyperkalaemia (see ➲ Hyperkalaemia, pp. 170–1). Treat mild hyponatraemia by fluid restriction. IV saline may be needed for severe hyponatraemia—rapid correction of chronic hyponatraemia can cause brain injury (central pontine myelinolysis), but this is less likely with acute hyponatraemia caused by MDMA. Cool as for heat stroke (see ➲ Heat illness, pp. 274–5) if hyperpyrexial. If rectal T° is >40°C, consider dantrolene 1–2.5mg/kg IV (up to 10mg/kg in 24hr). (See also ➲ Serotonin syndrome, p. 224.)

### Amphetamine (amfetamine)

Can be swallowed, snorted, smoked, or injected. Body packers may suffer severe poisoning. Toxic features are euphoria, agitation (excited or agitated delirium), psychosis, sweating, dilated pupils, tachycardia, hypertension, vomiting, abdominal pain, fits, hyperpyrexia, and metabolic acidosis. Severe poisoning may cause stroke, MI, rhabdomyolysis, renal failure, and DIC. Cardiac arrest can occur in violent agitated patients who need physical restraint. Treat amphetamine poisoning as for MDMA (see ➲ Ecstasy (MDMA), p. 222).

### Cocaine

Cocaine base ('crack') is usually smoked. Cocaine salt ('coke') is snorted, eaten, or injected. Toxic effects (due to catecholamines, serotonin, and amino acid stimulation and $Na^+$ channel blockade) are euphoria, agitation, delirium, ataxia, dilated pupils, sweating, vomiting, fits, tachycardia, arrhythmias, and hypertension. Chest pain may be due to myocardial ischaemia or MI (from ↑ catecholamines, ↑ $O_2$ demand, coronary vasospasm, and thrombosis), aortic dissection, or pneumothorax. Cerebral haemorrhage, hyperpyrexia, rhabdomyolysis, renal failure, gut ischaemia, and serotonin syndrome may occur. Cocaine is an LA, so hot air from smoking crack can cause airway burns.

*Treatment*

Treat as for MDMA (see ➲ Ecstasy (MDMA), p. 222). Give diazepam for agitation (5–10mg IV, repeated every 5min if needed, up to 100mg). Treat chest pain with diazepam, GTN, $O_2$, and aspirin. GTN and phentolamine may ↓ BP and ↑ coronary blood flow. Avoid β-blockers (may cause paradoxical hypertension and ↑ coronary vasoconstriction), except labetalol which may be an option (antagonizes both α- and β-adrenoceptors). If ECG suggests acute MI, consider angioplasty or thrombolysis.

### Gammahydroxybutyrate (GHB, GBH, 'liquid ecstasy')

GHB is used illegally as a body-building agent and psychedelic drug. It is ingested or injected. Intoxication may cause vomiting, diarrhoea, drowsiness, confusion, ataxia, and agitation. Severe poisoning results in coma, respiratory depression, fits, bradycardia, and hypotension. Consider activated charcoal (see ➲ Activated charcoal, p. 192) if <1hr since ingestion. Observe for at least 4hr and monitor pulse rate, BP, and breathing. Provide supportive treatment as needed. Control agitation and convulsions with diazepam.

### LSD (lysergic acid diethylamide)

Causes visual hallucinations, agitation, excitement, tachycardia, and dilated pupils. Hypertension and pyrexia may occur. Paranoid delusions may require sedation. Massive overdose of LSD is rare but may cause coma, respiratory arrest, and coagulation disturbances. Treat supportively.

### Amyl nitrite

This volatile liquid is often inhaled recreationally. As a vasodilator, it can cause headache and hypotension. Oxidation of iron in Hb to the ferric form causes methaemoglobinaemia, causing ↓ $O_2$-carrying capacity. $SpO_2$ may be normal. Give high-flow $O_2$ and measure methaemoglobin level. Treat as per TOXBASE—consider methylene blue (methylthioninium chloride) 1–2mg/kg over 5min, which can be repeated after 30–60min.

# Serotonin syndrome

## Background

The clinical picture of serotonin syndrome is increasingly recognized amongst those taking SSRIs. The syndrome can occur in patients who have taken therapeutic doses of SSRIs, and this is especially likely if they have recently started on the medication or if it is taken in combination with other drugs which ↑ production, availability, or release of serotonin (eg cocaine, MDMA, amphetamines) or reduce metabolism (eg MAOIs). Serotonin syndrome can also occur after an acute overdose. Numerous drugs have been implicated—in addition to those mentioned previously, they include: tricyclic antidepressants, venlafaxine, tramadol, pethidine, buprenorphine, St John's wort, olanzapine, and lithium.

## Clinical features

*Altered mental status*

Confusion, hallucinations, and agitation may occur, with drowsiness and reduced conscious level in severe cases.

*Neuromuscular features*

Rigidity, shivering/tremor, teeth grinding, ataxia, and hyper-reflexia (especially affecting the lower limbs) may occur.

*Autonomic effects*

These include tachycardia, hypertension (or hypotension), flushing, diarrhoea, vomiting, and hyperthermia.

Severe cases can result in fits, rhabdomyolysis, renal failure, and coagulopathy.

## Differential diagnosis

This includes neuroleptic malignant syndrome, malignant hyperthermia, severe infection (eg encephalitis), and other direct effects of drug overdose or withdrawal.

## Investigations

Check U&E, glucose, LFTs, CK, FBC, urinalysis, ABG/VBG, and ECG.

## Treatment

Provide supportive measures and obtain expert advice (TOXBASE or call NPIS—see ➲ National Poisons Information Service, pp. 188–9). Agitation, hyperthermia, myoclonic jerking, and fits may benefit from diazepam therapy. Treat rhabdomyolysis with IV fluids and urine alkalinization.

*Cyproheptadine* (12mg PO, then 4–8mg 6-hourly) is a serotonin receptor antagonist. Although data are lacking, there is a good theoretical basis for its use in serotonin syndrome. Consider chlorpromazine (12.5–25mg IV) in severe cases.

# Body packers

Body packers try to smuggle drugs such as cocaine or heroin by ingesting multiple packages of drugs wrapped in condoms, latex, foil, or even fibreglass. Packages may also be hidden in the rectum or vagina (individuals who do this are sometimes referred to as '*body pushers*'). Serious or even fatal poisoning may occur if any packages leak and the drugs are absorbed.

Patients are often in police custody—take care to ensure that correct procedures are undertaken (see https://www.rcem.ac.uk).

In England and Wales, imaging and intimate examination to search for class A drugs requires the patient's written consent and an authorization from a police inspector. In Scotland, the police will seek a Sheriff's warrant.

Rectal and/or vaginal examination should ideally be performed by a forensic physician (police surgeon). Limit the number of individuals handling any retrieved packages to the police or forensic physician. If the patient lacks capacity, consider whether radiological investigation can be undertaken in their best interests (get senior advice).

## Management

Advice is available from NPIS (see ➲ National Poisons Information Service, p. 190). Ensure that suspected body packers receive careful assessment and observation. Features of toxicity depend upon the concealed drug and any accompanying adulterants (eg strychnine). Consider the need for intimate examination (as outlined previously).

Try to establish the drug involved, the number of packages, and the type of packaging used. Check basic signs. Urine toxicology screening is not widely available but may detect heroin or cocaine and provide an indication of those occasions when leakage has occurred.

Abdominal X-ray is sometimes used as a screening tool (97% specificity) but is less sensitive than CT. Gastric packages can sometimes be seen on an erect CXR. USS or MRI may play a role in pregnant patients.

Isotonic laxatives and whole-bowel irrigation may aid expulsion of packages. Consider a naloxone infusion (see ➲ Opioid poisoning, p. 196) for symptomatic heroin body packers. In symptomatic patients, refer early as surgery may be required—this is particularly urgent if cocaine or amphetamines are implicated. Endoscopy may help to remove small gastric packages, but there is a risk of damaging packaging and ↑ drug leakage.

## Body stuffers

This term is sometimes applied to individuals who swallow drugs (or hide them rectally or vaginally as body pushers) immediately prior to being apprehended by the police. The quantity of drugs ingested in this way may be less than that by body packers. However, any packaging is likely to be much less robust than that used by body packers, thereby ↑ the risk of the packages leaking. Consider activated charcoal and admission for 6–8hr (up to 24hr if suspicion is high).

## Parachuting

Recreational drugs are ingested in delicate packaging, with the aim of delaying absorption. Treat as body stuffers (see ➲ Body stuffers, p. 225).

# Chapter 5

# Infectious diseases

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# Incubation periods

## Incubation period usually <1 week

| | |
|---|---|
| Staphylococcal enteritis | 1–6hr |
| *Salmonella* enteritis | 6–48hr (usually 12–24hr) |
| Bacillary dysentery (*Shigella*) | 1–7 days (usually 1–3 days) |
| Botulism | 12–96hr (usually 18–36hr) |
| Cholera | 12hr to 6 days (usually 1–3 days) |
| COVID-19 | 7–14 days (median 5 days) |
| Dengue | 4–7 days |
| Diphtheria | 2–5 days |
| Gas gangrene | 6hr to 4 days |
| Legionnaires' disease | 2–10 days (usually 7 days) |
| Meningococcaemia | 1–7 days (usually 3 days) |
| Scarlet fever | 1–4 days |
| Yellow fever | 3–6 days |

## Incubation period usually 1–3 weeks

| | |
|---|---|
| Brucellosis | 7–21 days (occasionally some months) |
| Chickenpox | 10–20 days (usually about 14 days) |
| Lassa fever | 6–21 days |
| Leptospirosis | 2–26 days (usually 7–12 days) |
| Malaria (falciparum) | 7–14 days (occasionally longer) |
| Malaria (vivax, malariae, ovale) | 12–40 days (occasionally >1y) |
| Measles | 10–18 days (rash usually 14–18 days) |
| Mumps | 14–18 days |
| Pertussis (whooping cough) | 5–14 days (usually 7–10 days) |
| Rubella | 14–21 days |
| Tetanus | 1 day to 3 months (usually 4–14 days) |
| Typhoid | 3–60 days (usually 7–14 days) |
| Typhus | 7–14 days |

## Incubation period usually >3 weeks

| | |
|---|---|
| Amoebiasis | 2 weeks to many months |
| Hepatitis A | 2–6 weeks (usually 4 weeks) |
| Hepatitis B and C | 6 weeks to 6 months |
| HIV | 2 weeks to 3 months (anti-HIV antibody appears) |
| Infectious mononucleosis | 4–7 weeks |
| Rabies | 4 days to 2y (usually 3–12 weeks) |
| Syphilis | 10 days to 10 weeks (usually 3 weeks) |

### Duration of infectivity of infectious diseases

| | |
|---|---|
| Chickenpox | 3 days before rash until last vesicle crusts |
| Hepatitis A | 2 weeks before until 1 week after jaundice starts |
| Measles | 4 days before rash until 5 days after rash appears |
| Mumps | 3 days before to 1 week after salivary swelling |
| Pertussis | 3 days before to 3 weeks after start of symptoms (5 days if on appropriate antibiotic) |
| Rubella | 1 week before to 1 week after onset of rash |
| Scarlet fever | 10–21 days from onset of rash (1 day if on penicillin) |

## Notifiable infectious diseases

In the UK, certain infectious diseases are 'notifiable'. A doctor who knows or suspects that a patient has one of these diseases is obliged to notify the local health protection department. Use the special notification form, if available. Telephone the consultant in communicable disease control if investigation or control of an outbreak may be needed (see https://www.gov.uk).

### Notifiable infectious diseases list

The list of notifiable diseases from Public Health England is as follows:

- Acute encephalitis, acute infectious hepatitis, acute meningitis, acute poliomyelitis, anthrax, botulism, brucellosis, cholera, COVID-19, diphtheria, enteric fever (typhoid or paratyphoid fever), food poisoning, haemolytic uraemic syndrome, infectious bloody diarrhoea, invasive group A streptococcal disease, legionnaires' disease, leprosy, malaria, measles, meningococcal septicaemia, mumps, plague, rabies, rubella, SARS, scarlet fever, smallpox, tetanus, TB, typhus, viral haemorrhagic fever (VHF), whooping cough, yellow fever.

Note that there is a separate list of notifiable organisms (causative agents) which laboratories report—there is some inevitable overlap between the two lists.

# Childhood infectious diseases

## Children at risk

Unimmunized children are at risk of infections which would be prevented by the standard immunization schedule (see ➲ Standard immunization schedule, pp. 652–3). Always ask about vaccination status in any febrile, unwell child. The common infectious diseases of childhood can be very serious in children with *immune deficiency* or those on *immunosuppressant drugs*. Refer such children for specialist advice if they develop an infectious disease or have been in contact with one. Children with cystic fibrosis can become very ill with measles, whooping cough, or chickenpox—refer these also. Neonates rarely develop the common exanthems of childhood, but refer if these present to the ED. Chickenpox can be particularly serious in this age group.

## Measles[ND]

A viral infection spread by airborne droplets. It is very contagious.

*Incubation period* Is 10–18 days. Infectious from just before the onset of symptoms until 5 days after the rash appears.

*Initial features (lasting ~3 days)* Fever, malaise, coryza, conjunctivitis, and cough. Koplik's spots (small white spots like grains of salt) appear inside the cheeks. One to 2 days later, a red maculopapular rash starts behind the ears and spreads to the face and down the body and onto the limbs.

*Treatment* Is symptomatic unless there are complications (eg otitis media or bacterial pneumonia). Febrile convulsions may occur. Encephalitis is relatively rare (~0.1%) but can be fatal. Hospital admission is rarely needed, unless the child is very ill or has pre-existing disease. In the tropics, many malnourished children die from measles, but in the UK, mortality is very low.

## Mumps[ND]

Mumps is a virus infection spread by saliva and respiratory droplets. Infectivity is greatest at the onset of symptoms, but many subclinical cases also spread infection. There has been a large increase in cases in the UK amongst young adults (especially those at college/university), including many who did receive all childhood immunizations.

*Incubation period* 14–18 days.

*Typical features* Fever with pain and swelling in one or both parotid glands. The submandibular glands may sometimes be affected. Aseptic meningitis may occur. Orchitis affects 10–15% of post-pubertal ♂ but rarely causes sterility. The pain of orchitis may be relieved by analgesia and a short course of steroids. Orchitis is uncommon before puberty, so consider torsion of the testis if a child presents with testicular pain and swelling (see ➲ Inguinal and scrotal swellings, pp. 722–3).

### Rubella (German measles)[ND]

Rubella is usually a mild disease, but infection during pregnancy may cause severe congenital disorders, particularly eye defects, heart defects, and deafness. Guidance on the management of, and exposure to, rubella during pregnancy is available from the Health Protection England/Wales/Scotland websites (https://www.gov.uk). The virus is spread mainly by the airborne route, with an incubation period of 2–3 weeks and infectivity from 1 week before symptoms until 1 week after the rash appears. A macular rash occurs on the face and trunk, with mild fever, occipital lymphadenopathy, and sometimes transient arthralgia. Rare complications are encephalitis and thrombocytopenia.

*Treatment* Generally symptomatic. The clinical diagnosis of rubella is unreliable—similar rashes may occur with enterovirus and parvovirus infections. If there is concern about rubella infection in pregnancy, take blood for viral antibody levels and arrange urgent follow-up by the GP or obstetrician.

### Whooping cough[ND]

See Whooping cough[ND], p. 700.

### Infectious mononucleosis (glandular fever)

Infection with *Epstein–Barr virus* is common in children and young adults and is spread by saliva or droplets. Infection often occurs without clinical disease. In glandular fever, there is malaise, fever, a sore throat, and cervical lymphadenopathy. The throat may be very red, and in 25% of cases, there is also infection with β-haemolytic *Streptococcus*. In severe cases, there is marked oedema of the throat, with tonsillar swelling and a membranous exudate ('anginose' infectious mononucleosis), with difficulty in swallowing and breathing. A rash is uncommon, unless ampicillin or amoxicillin are given, causing a widespread erythematous maculopapular rash (which does not signify allergy to penicillins in general).

*Complications* Include respiratory obstruction, ruptured spleen (spontaneously or after minor trauma, so advise avoid contact sports for 1 month), thrombocytopenia, jaundice, meningitis, encephalitis, facial palsy, and acute polyneuritis (occasionally causing respiratory failure).

*Investigations* FBC and blood film (for atypical lymphocytes), Monospot test or Paul–Bunnell test (which may be –ve initially).

*Differential diagnosis* Includes CMV and toxoplasmosis.

*Treatment* Unnecessary in most patients. Severe or complicated cases need specialist assessment and follow-up. In anginose infectious mononucleosis, a short course of high-dose oral steroids gives rapid relief of symptoms (prednisolone 80mg on day 1; 15mg tds on days 2–3; 10mg tds on days 4–5; 5mg tds on days 6–7). Steroids are also helpful in patients with neurological complications. Concurrent β-haemolytic streptococcal infection requires erythromycin (500mg qds), which would also treat the rare unrecognized case of diphtheria.

# Meningitis

## Causative organisms

Meningitis may be *bacterial, viral*, or occasionally *fungal*. Bacterial causes of meningitis include meningococci, pneumococci, *Haemophilus influenzae, Listeria*, and TB. Other bacteria may also cause meningitis in neonates, the elderly, and immunosuppressed patients.

## Clinical features of bacterial meningitis

Some patients have classic features of headache, neck stiffness, photophobia, fever, and drowsiness. However, the clinical diagnosis of meningitis may be very difficult in early cases. Neonates may present with anorexia, apnoea, or fits. Meningitis may start as a 'flu-like' illness, especially in the immunosuppressed or elderly. Consider meningitis in any febrile patient with headache, neurological signs, neck stiffness, or ↓ conscious level.

*Meningococcal meningitis*[ND] Caused by *Neisseria meningitidis*. It can result in septicaemia, coma, and death within a few hours of the first symptoms. Skin rashes occur in 50% of patients, often starting as a maculopapular rash before the characteristic petechial rash develops. There may be DIC and adrenal haemorrhage (Waterhouse–Friderichsen syndrome). Meningococcal septicaemia (see ➲ Meningococcal disease, pp. 682–3) may occur without meningitis.

## Management

Resuscitate, giving $O_2$ as required, and obtain venous access.

Start antibiotics *immediately* (without waiting for investigations) if the patient is shocked or deteriorating or there is any suspicion of meningococcal infection (especially a petechial or purpuric rash)—give IV ceftriaxone (adult 2g; child 80mg/kg) or cefotaxime. Chloramphenicol is an alternative if there is a history of anaphylaxis to cephalosporins (see *BNF*). In adults >55y, add ampicillin 2g qds to cover *Listeria.* Give vancomycin ± rifampicin if penicillin-resistant pneumococcal infection is suspected. Give IV dexamethasone (0.15mg/kg, max 10mg, qds for 4 days), starting with or just before the first dose of antibiotics, especially if pneumococcal meningitis is suspected.

*Initial investigations* FBC, U&E, glucose, clotting screen, VBG, CRP, blood cultures, EDTA sample for polymerase chain reaction (PCR), and clotted blood for serology. LP is needed if meningitis is suspected, unless there is a coagulopathy or ↑ ICP—do a CT scan if there is suspicion of ↑ ICP (confusion/coma, hypertension, bradycardia, or papilloedema) or focal neurological signs.

*Provide supportive treatment, including*

- IV fluids.
- Pressure area care.
- Monitor conscious level, T°, BP, ECG, $SpO_2$, and fluid balance.

Get expert help promptly and organize ICU care.

For latest advice, see https://www.meningitis.org and https://www.nice.org.uk.

For meningitis and LP in children, see ➲ Meningococcal disease, pp. 682–3.

### Prophylaxis of meningococcal infection

Whilst intubating a patient with suspected meningococcal infection, wear a suitable mask (eg FFP3) and a face shield to reduce the risk of infection.

Meningococcal infection is spread by droplets from the nose of an infected carrier, who may be well. Notify the consultant in communicable disease control (see ➲ Notifiable infectious diseases, p. 229) immediately about any suspected meningococcal infection and obtain advice about antibiotic prophylaxis. Prophylactic antibiotics (rifampicin, ciprofloxacin, or ceftriaxone) are needed for the patient's family and close contacts. Hospital and ambulance staff do not need prophylaxis unless they have given mouth-to-mouth ventilation or intubated the patient without using protective equipment.

*Rifampicin* is given 12-hourly for 2 days (5mg/kg for a child aged <1y; 10mg/kg at 1–12y; 600mg at age >12y). It makes the urine orange or brown, discolours soft contact lenses, and ↓ effectiveness of OCP for ~4 weeks (see *BNF*)—give appropriate warnings and record this in the notes.

*Ciprofloxacin* is given as a single PO dose of 500mg (adults), 250mg (child 5–12y), or 125mg (child 2–5y), although it is not licensed for chemoprophylaxis of meningitis.

*Ceftriaxone* is given as a single IM dose of 250mg (adults and children >12y) or 125mg (children <12y).

Warn contacts of meningococcal patients to report to a doctor at once if they develop symptoms.

### TB meningitis

Often gradual onset, with malaise, anorexia, vomiting, headache, and eventually signs of meningitis. Cranial nerve palsies, spastic paraplegia, and coma can occur. Meningitis may be part of miliary TB (see ➲ Tuberculosis[ND], p. 242), which may be apparent on CXR. Ophthalmoscopy may show choroidal tubercles and papilloedema, which is found more commonly than in other forms of meningitis. Refer for specialist investigation and treatment.

### Viral meningitis

Viral causes of meningitis include Coxsackie virus, mumps, and echoviruses. Viral meningitis produces similar clinical features to those of bacterial infection, but the illness is often less severe. Initial management is the same as for suspected bacterial meningitis. Refer for admission and investigation.

See also ➲ Acute encephalitis, p. 234.

### Fungal meningitis

Fungal meningitis is usually part of disseminated infection in immunosuppressed patients (eg those with AIDS—see ➲ Human immunodeficiency virus, pp. 250–1), or lymphoma, or on steroid therapy). *Cryptococcus neoformans* is the most common organism. Symptoms usually develop slowly, as with TB meningitis. There may be papilloedema and focal neurological signs. Admit for specialist investigation and treatment.

# Acute encephalitis[ND]

### Causative organisms

Acute encephalitis is most commonly attributed to herpes simplex virus in the UK (hence, early treatment with aciclovir is a high priority). Other viral causes include CMV, EBV, herpes varicella-zoster virus, measles, mumps, HIV, and rabies. Non-viral causes include TB and malaria, in addition to the wide range of causes of bacterial meningitis.

### Presentation

The diagnosis of acute encephalitis is not always an easy one to make. There may not always be a history of headache and fever. Patients may present with odd or bizarre behaviour ± confusion. Sometimes the presentation is acute with collapse, seizures, or ↓ conscious level. Keep the diagnosis in mind when assessing patients with unexplained neurological symptoms and/or focal neurological signs.

### Investigation

- Take blood for FBC, ESR, CRP, U&E, clotting, blood glucose, viral PCR, and cultures.
- If there is a relevant history of foreign travel, send thin and thick films for malaria (see Malaria[ND], p. 255).
- Contrast-enhanced CT may reveal changes suggestive of herpes simplex encephalitis. MRI is an alternative, if available.
- LP may yield CSF which has ↑ lymphocytes. Send a sample for viral PCR.

### Management

- Involve ICU and resuscitate with $O_2$ and IV fluids as required.
- Start IVI aciclovir (10mg/kg IVI over 1hr).
- Consider treating with IV antibiotics (eg IV ceftriaxone 2g) for bacterial meningitis (see Meningitis, pp. 232–3).

# Herpes simplex virus

Primary herpes simplex infection causes painful vesicles and ulceration of the mouth or genitalia (see Sexually transmitted diseases, p. 234). The virus may be inoculated into skin by trauma (herpes gladiatorum, scrumpox) or by contamination of fingers causing herpetic paronychia (whitlow). Infection of the cornea may cause dendritic ulcers (see Ulcerative keratitis, p. 559).

*Herpes simplex meningitis* and *encephalitis*[ND] are uncommon but may be fatal, especially in immunodeficient patients.

Herpes simplex virus persists in sensory ganglia and may be reactivated by stimuli such as sun, cold, trauma, or viral infections. Recurrence of cold sores of the lips is often preceded by tingling—aciclovir cream or tablets may prevent the development of vesicles. Secondary bacterial infection may require antibiotics. Do not incise a suspected whitlow. Cover it with a dressing and advise care to avoid spreading infection to the lips or eyes.

## Herpes varicella-zoster virus

*Chickenpox* results from primary infection with varicella-zoster virus, which then remains dormant in the dorsal root ganglia. Reactivation of the virus causes *shingles*. Chickenpox is usually a mild disease of childhood. An itchy vesicular rash appears, most densely on the trunk and face, but ↓ peripherally. The lesions appear in crops and crust over in 3–4 days. Fever, malaise, and muscle aches may occur in adults. Infectivity starts 3 days before the rash appears and continues until the last lesion has crusted.

Treat symptomatically, eg calamine lotion for itching and paracetamol for fever. Avoid NSAID use in chickenpox—this is associated with ↑ risk of skin and soft tissue infections. Occasionally, antibiotics are needed for secondary bacterial skin infection (usually *Staphylococcus* or *Streptococcus*). Pneumonia is rare and in children is usually staphylococcal, but in adults it may be caused by chickenpox virus. Chickenpox may be severe in neonates and in those with cystic fibrosis or immune deficiency—refer for specialist assessment and treatment with aciclovir and/or varicella-zoster immune globulin. Consider aciclovir also for adults and older adolescents (see *BNF*).

*Shingles* often occurs in the elderly and may affect any dermatome, most often thoracic. The pain of shingles may cause diagnostic difficulty until the rash appears, usually after 1–4 days. Erythema is followed by vesicles and then crusting of lesions in a unilateral distribution over one dermatome or two adjacent dermatomes. Ophthalmic shingles may affect the eye via the long ciliary nerves—skin lesions on the side of the tip of the nose imply a high risk of eye involvement (Hutchinson's sign). Oral lesions occur in maxillary and mandibular shingles. Infection of the geniculate ganglion causes a facial palsy, with lesions in the pinna of the ear and on the side of the tongue and hard palate (*Ramsay–Hunt syndrome*). In severe shingles, there may be weakness of muscles supplied by nerves of the same spinal root.

*Antiviral treatment* (aciclovir, famciclovir, or valaciclovir) ↓ the risk of postherpetic pain if given early (within 72hr of start of rash). *Dose*: aciclovir 800mg five times daily for 7 days. In renal failure, antiviral drugs may cause severe toxicity, so use much smaller or less frequent doses. Patients with immune deficiency or ophthalmic zoster need immediate specialist referral and antiviral treatment. Give analgesia. Antibiotics may be required for secondary infection.

## Zika virus

Zika virus infection is mainly transmitted to humans by mosquitos in the Caribbean and South America, although human-to-human transmission can occur. It causes a usually mild illness, which may be subclinical or cause fever, rash, headache, myalgia, and conjunctivitis lasting up to a week. The incubation period is 3–14 days. The principal concern regarding zika virus infection is that infection in pregnancy produces fetal abnormalities (especially microcephaly) in a significant number of pregnant women.

# Gastroenteritis/food poisoning[ND]

## Diarrhoea

This is the usual presenting symptom of gastroenteritis, but it is also a feature of many other conditions as diverse as otitis media, appendicitis, and ulcerative colitis. Antibiotics often cause diarrhoea. Constipation may present as diarrhoea if there is overflow around an obstructing stool. A rectal tumour may present similarly.

*Diarrhoea and vomiting* May be caused by many types of bacteria and viruses (eg norovirus), and also by some toxins and poisons. Many episodes of gastroenteritis result from contaminated food, usually meat, milk, or egg products, which have been cooked inadequately or left in warm conditions. The specific cause is often not identified. Some infections are spread by faecal contamination of water (eg cryptosporidiosis from sheep faeces). Rotavirus infection (common in children) may be transmitted by the respiratory route. Severe illness with bloody diarrhoea, haemolysis, and renal failure may result from infection with verocytotoxin producing *Escherichia coli* (VTEC O157).

*Food poisoning* Is a notifiable disease (see ➲ Notifiable infectious diseases, p. 229). Immediate notification by telephone is mandatory if an outbreak is suspected. The food eaten, symptoms, and incubation period may suggest the organism or toxin involved (see Table 5.1). CO poisoning (see ➲ Carbon monoxide poisoning, p. 216) may cause malaise and vomiting in several members of a family and be misdiagnosed as food poisoning.

Ensure that patients who present with diarrhoea are flagged up as posing a potential risk of infection, so that they can be isolated appropriately to protect other patients from cross-infection.

## History

Record the duration of symptoms and the frequency and description of stools and vomit, including the presence of blood. Document other symptoms (eg abdominal pain, fever), food and fluid ingested, and any drugs taken. Enquire about affected contacts, foreign travel, and occupation (especially relevant if a food handler).

## Examination

Check vital signs, including pulse rate, RR, BP, $SpO_2$, and T°. Examine for abdominal tenderness and other signs of infection. Record the patient's weight and compare this with any previous records. Assess the *degree of dehydration*—this is especially important in children and is traditionally classified as mild (<5%), moderate (5–10%), or severe (>10%) (see also ➲ Gastroenteritis in children, pp. 718–19).

Evidence of severe dehydration includes: weakness, confusion, shock, and ↓ urine output.

**Table 5.1** Food poisoning characteristics

| Cause | Incubation | Food | Symptoms* |
|---|---|---|---|
| *Staphylococcus aureus* | 1–6hr | Meat, milk | D, V, P, shock |
| *Bacillus cereus* | 1–16hr | Rice | D, V, P |
| *Salmonella* | 6–48hr | Meat, eggs | D, V, P |
| *Escherichia coli* | 1–2 days | Any food | D, V, P |
| *E.coli* VTEC O157 | 1–2 days | Meat, milk | D, V, P |
| *Campylobacter* | 1–3 days | Meat, milk | Fever, P, D |
| *Shigella* | 1–3 days | Any food | Bloody D, V, fever |
| *Vibrio* parahaem | 2–3 days | Seafood | Watery D |
| Cholera | 12hr to 6 days | Water, seafood | D (watery), shock |
| Rotavirus | 1–7 days | | D, V, fever, cough |
| Botulism | 12–96hr | Preserved food | V, paralysis |
| Histamine fish poisoning (scombrotoxin) | <1hr | Fish | Flushing, headache, D, V, P (see ➲ Fish poisoning, p. 239) |
| Ciguatera fish poisoning | 1–6hr (rarely 30hr) | Fish from tropical coral reef | D, V, P, paraesthesiae, muscle weakness (see ➲ Fish poisoning, p. 239) |
| Paralytic shellfish poisoning | 30min to 10hr | Shellfish | Dizziness, paraesthesiae, weakness, respiratory failure (see ➲ Fish poisoning, p. 239) |
| Chemicals | <2hr | Food, water | Various |
| Mushrooms | <24hr | Mushrooms | D, V, P, hallucinations (see ➲ Plants, berries, and mushrooms, p. 219) |

* D, diarrhoea; V, vomiting; P, abdominal pain.

## Investigations

Consider sending blood tests (including U&E) if the patient is shocked or unwell.

*Stool culture* is unnecessary in most cases of gastroenteritis, but obtain if the patient is systemically unwell, has blood or pus in the stool, is immunocompromised, has recently been hospitalized and/or has had antibiotics or PPI, has been abroad or has prolonged symptoms, is a resident in a care home, or works as a food handler, or the diagnosis is uncertain.

# Managing gastroenteritis/food poisoning[ND]

## Treatment

(See also ➲ Gastroenteritis in children, pp. 718–19.)

- Isolate and take precautions to prevent spread of infection to staff or other patients (see ➲ Infection control and prevention, pp. 36–7).
- Rehydrate aggressively with IV fluids those patients who are shocked and/or severely dehydrated, then reassess and refer for admission. Admit patients who have persistent vomiting and cannot keep down oral fluids. Most illnesses are self-limiting and do not require hospital admission, but remember that some patients may need special attention and/or cannot manage to cope at home (eg significant comorbidity, frail elderly living alone). Other features which may influence the decision to admit include: fever, bloody diarrhoea, abdominal pain, recent foreign travel, diarrhoea for >10 days, drugs that may exacerbate renal impairment, and dehydration.
- Aim to discharge less severe cases with oral fluids (± soup and fruit juice) and advice. Consider supplementing diet with oral rehydration therapy (eg Dioralyte®) in patients >60y or with comorbidities. Warn that home-made salt and sugar mixtures may be dangerously inaccurate versions of the professional oral rehydration products on offer. Advise patients to recommence normal diet after symptoms settle. Most patients can return to work 48hr after the first normal stool (advise food handlers to contact their employer and/or public health authorities).

*Antiemetic drugs* are sometimes helpful in gastroenteritis. In adults, an antiemetic (eg metoclopramide 10mg IM or prochlorperazine 12.5mg IM or 3mg buccal) may help, but do not use in children as it often causes troublesome side effects.

*Anti-diarrhoeal drugs* (eg loperamide) are contraindicated in children and rarely needed in adults—they may aggravate nausea and vomiting and occasionally cause ileus. However, they may provide symptomatic control in mild to moderate diarrhoea, but avoid if there is bloody diarrhoea and/or possible *Shigella* infection.

*Antibiotics* are only needed in special circumstances. Most episodes of gastroenteritis are brief, and many are caused by viruses and not helped by antibiotics. Patients with amoebiasis, giardiasis, and *Campylobacter* or *Shigella* infections may need antibiotics—refer to a specialist and/or an infectious diseases unit for treatment and follow-up.

Antibiotics are occasionally useful in traveller's diarrhoea before a long journey or an important meeting (ciprofloxacin 500mg bd PO for 3 days—see the *BNF* or data sheet about side effects and warnings).

# Fish poisoning

## Histamine fish poisoning

Also known as scombroid fish poisoning or scombrotoxin poisoning, this is caused by ingesting toxins in fish such as tuna, mackerel, and other dark-meat fish, which have been stored improperly. If the fish is not cooled rapidly after it is caught, an enzyme in bacteria converts histidine into histamine and other toxins, which are heat-stable and so are unaffected by cooking. The patient may notice that the fish tastes metallic, bitter, or peppery and the flesh looks honeycombed. Symptoms start within a few minutes to 2hr, with flushing of the face and upper body, headache, nausea, vomiting, abdominal pain, diarrhoea, dizziness, and palpitations. Urticaria and bronchospasm are less common. Symptoms usually settle within 6hr without treatment but resolve more quickly with antihistamines (eg chlorphenamine 10mg IV in adults, 250mcg/kg in children). In severe cases, cimetidine and, rarely, adrenaline might be needed, with $O_2$, IV fluids, and bronchodilators.

Tell the patient that histamine fish poisoning is caused by improper fish handling and storage. It is not an allergic reaction, and so the patient would not have to avoid eating fish in future.

## Ciguatera fish poisoning

This is caused by a neurotoxin called ciguatoxin which is produced by a dinoflagellate (a unicellular plankton) associated with coral reefs. Fish imported from the tropics may cause ciguatera poisoning in the UK and elsewhere. Symptoms usually start 1–6hr after ingestion with nausea, vomiting, watery diarrhoea, and abdominal pain, followed by neurological symptoms, including paraesthesiae of the lips, tongue, and feet, ataxia, and muscle weakness. A classic feature is paradoxical temperature reversal (cold objects feel hot and hot objects feel cold). Alcohol makes these symptoms worse. Bradycardia and hypotension may occur. Treatment is symptomatic and supportive. GI symptoms usually settle within a day, but paraesthesiae may persist for weeks or months.

## Paralytic shellfish poisoning

This can be caused by eating molluscs such as mussels, clams, cockles, and scallops which concentrate a neurotoxin called saxitoxin produced by dinoflagellate plankton. This plankton proliferates when sea temperatures rise in summer and may make the sea look red ('red tide'). Symptoms start 30min to 10hr after ingestion with dizziness, ataxia, paraesthesiae, and muscle weakness, which may progress to respiratory failure. Treatment is supportive, with assisted ventilation if necessary. Complete recovery is usual within 24hr.

# Infestations

## Worms

The most common helminthic infection seen in the UK is the threadworm *Enterobius vermicularis*. This causes anal itching, especially at night. Sometimes intact worms (length 5–13mm, diameter 0.1–0.5mm) are seen in the faeces. Unwashed fingers transmit ova from the perianal skin to the mouth. Personal hygiene is important in treatment and preventing reinfection (handwashing and nail-scrubbing before each meal and after every visit to the toilet). A bath immediately after getting up removes ova laid overnight. Treat all members of the family with mebendazole, unless contraindicated (see *BNF*), and arrange GP follow-up.

*Other helminthic infections* include roundworms, hookworms, and tapeworms. Obtain advice from departments of infectious diseases or tropical medicine (see ➲ Imported infectious diseases, p. 254).

## Lice

Humans may be infected by the body louse (*Pediculosis humanis corporis*), head louse (*Pediculosis humanis capitis*), or 'crab'/pubic louse (*Phthirus pubis*).

*Head lice* Common in schoolchildren. Infection is not related to lack of hygiene or the length of hair. Adult lice are 3–4mm long, vary in colour from white to grey-black, and attach themselves firmly to the scalp at the base of hairs. The egg cases ('nits') are white and 1–2mm in diameter, glued firmly to the base of hairs and moving outwards as the hair grows. Head lice cause intense itching, which may suggest the diagnosis. Secondary infection may result in impetigo. Treatment options include physical insecticide, chemical insecticide (malathion), or wet combing (see https://www.nice.org.uk), and advise GP follow-up.

*Infection by body lice* Related to poor hygiene and infrequent washing of clothes. Body lice are found in the seams of clothing and sometimes in body hair. Treat with malathion. Clothes can be disinfected by boiling or machine laundering and ironing. Body lice may transmit rickettsial diseases (louse-borne typhus) and other infections.

*Crab lice* Usually transmitted sexually. They cause itching in pubic hair areas. Occasionally, children become infested on eyelashes or eyebrows. Treat with permethrin cream or malathion 0.5% aqueous solution (see *BNF*). Sexual partners or other family members may also need treatment. There may be other coexisting sexually transmitted infections.

## Fleas

There are many different types of flea. They cause itchy bites with linear erythematous papules. Treat with calamine lotion and an oral antihistamine (eg chlorphenamine) if itching is severe. Consider a long-acting insecticide in the house, especially in cracks in the floor and under furniture. Advise all household cats and dogs be treated for fleas. Fleas can transmit many infections, including plague, typhus, and Q fever.

## Scabies

Scabies is caused by infestation with the mite *Sarcoptes scabiei*, which is about 0.2–0.4mm long and burrows into the skin. It is most often found in the finger webs and on the flexor aspect of the wrists. After 4–6 weeks, intense itching occurs, especially at night or after a hot shower. Burrows (3–15mm long) may be apparent, especially on palpation of affected skin. Genital lesions are reddish and nodular. Secondary bacterial infection may occur. Scabies can be confirmed by microscopy of scrapings from suspected lesions. Treat with topical permethrin 5% cream or malathion aqueous 0.5% (see *BNF*). Treat all members of the household at once. Calamine lotion and an oral antihistamine may help to relieve itching.

## Ticks

Ticks may be acquired from domestic animals or whilst walking through undergrowth or exploring caves. Aim to remove the tick completely. Remove ticks by grasping them with tweezers or curved forceps close to the skin and pulling them out straight and perpendicular to the skin. Ticks can carry several diseases, including Lyme disease (see below), tick-borne encephalitis, typhus, and Rocky Mountain spotted fever. Tick paralysis occurs in North America and Australia, with progressive paralysis which is often misdiagnosed as poliomyelitis.

The risk of infection from tick bites is low in most areas, and so routine prophylaxis with antibiotics is not recommended. However, do warn patients to seek medical attention if a rash develops at the site of the bite or they develop a fever.

*Lyme disease (Lyme borreliosis)* Is caused by the tick-borne spirochaete *Borrelia burgdorferi* and occurs in the UK, most of Europe, the USA, and parts of Asia and Australia. Most cases occur in summer and early autumn and are transmitted by ticks from deer or sheep. The initial tick bite may go unnoticed. Clinical illness develops after about 7–14 days (range 2–30 days), with an expanding red area around the site of the bite (erythema migrans). The second clinical stage of the disease occurs some weeks or months later, with fever, muscle and joint pains, and sometimes facial palsy or other cranial nerve or peripheral nerve palsies. Meningitis, encephalitis, and arthritis may develop. Myocarditis and heart block occur occasionally. Refer to an infectious diseases specialist for confirmation (usually initially including an enzyme-linked immunosorbent assay for Lyme disease) and treatment if Lyme disease is suspected. Treatment for adults and older children is usually with oral doxycycline, unless there is CNS involvement or carditis with haemodynamic instability, when IV ceftriaxone is preferred (see https://www.nice.org.uk).

# Tuberculosis[ND]

The *Mycobacterium* genus is characterized by acid-fast staining (ie it is not decolourized by acid after staining with hot carbol fuchsin).

Infection with *Mycobacterium tuberculosis* is common throughout the world. There is growing concern about the re-emergence of TB in the UK and other countries. Many cases of TB occur in the lower socio-economic groups, ethnic minorities, and the immunocompromised. The incidence of TB ↑ with age. Transmission is by the inhalation route.

## Presentation

TB can involve almost any organ of the body.

### *Primary infection*

This is usually pulmonary and often asymptomatic.

### *Post-primary infection*

This may present with malaise, weight loss, and night sweats, with localized symptoms, depending on the organs involved.

### *Pulmonary TB*

A relatively common way for TB to present, pulmonary TB may cause cough (initially dry, then productive), haemoptysis, pneumonia, and pleural effusion (see ➲ Pleural effusion, p. 107). CXR typically shows fibronodular/linear opacities in the upper lobes, but they can be seen in the middle and lower lobes ± calcification, cavitation, lymphadenopathy, and pleural effusion.

### *Miliary TB*

This involves blood-borne infection of many organs and develops over 1–2 weeks with fever, weight loss, malaise, and breathlessness. CXR may show multiple small opacities throughout the lung fields, and choroidal tubercles may be visible in the optic fundi.

### *TB meningitis*

This causes headaches and vomiting, sometimes with neck stiffness, cranial nerve palsies, and papilloedema (see ➲ Meningitis, pp. 232–3).

### *Tuberculous osteomyelitis*

This usually affects the spine and progresses slowly over weeks/months, with collapse of adjacent vertebrae and the development of paravertebral abscesses.

### *Tuberculous lymphadenitis*

Patients may present with swollen lymph nodes from tuberculous lymphadenitis or with sinuses or cold abscesses from bone or soft tissue infection. Microscopy of the discharge will show acid-fast bacilli.

## Treatment

Refer patients with suspected TB to an appropriate specialist for assessment and treatment. Initial treatment of confirmed TB involves the combination of rifampicin, isoniazid, ethambutol, and pyrazinamide. Isolate patients with untreated pulmonary TB. Notify the local health protection department (see ➲ Notifiable infectious diseases, p. 229).

# Anthrax[ND]

Anthrax is caused by the bacterium *Bacillus anthracis* which affects cows and other herbivorous animals, especially in warm climates. The bacterium forms spores, which may remain infective for years. Most human cases of anthrax are *cutaneous anthrax* caused by direct skin contact with infected tissues and occur in people working with animal products such as imported hides. Less common, but more serious, are *inhalation anthrax* caused by inhalation of anthrax spores, and *intestinal anthrax* which is a rare form of food poisoning caused by undercooked infected meat. Anthrax spores released deliberately in terrorist attacks could cause cutaneous anthrax or inhalation anthrax, which is often fatal.

Cutaneous anthrax starts 2–7 days after infection, with a red papule which develops into an ulcer with a black leathery eschar, surrounded by non-pitting oedema. The lesion is painless but may itch. Small satellite lesions may surround the original lesion. Malaise and fever may occur, with septicaemia in 10–20% of cases. Penicillin ↓ the risk of complications from cutaneous anthrax. Clinical diagnosis is confirmed by microscopy and culture of the pustule.

Inhalation anthrax starts within 48hr of exposure (rarely up to 6 weeks) with a flu-like illness, followed by breathlessness, cyanosis, stridor, and sweating, often with subcutaneous oedema of the chest and neck. CXR and CT show mediastinal widening from lymphadenopathy and pleural effusions. Shock, septicaemia, and meningitis are common and usually fatal, despite antibiotics and intensive treatment.

Airborne transmission of anthrax from one person to another does not occur, but cutaneous anthrax could result from direct contact with anthrax lesions. Obtain expert advice immediately if anthrax is suspected. It is a notifiable disease (see ➲ Notifiable infectious diseases, p. 229). Post-exposure antibiotics (eg ciprofloxacin) can prevent anthrax if started early enough. Anticipate press enquiries after any case of anthrax, especially if anthrax has been released deliberately.

## Anthrax in drug users

After a serious anthrax outbreak in heroin users in Scotland in 2010, Health Protection Scotland advised doctors to suspect anthrax in a drug user presenting with any of the following:

- Severe soft tissue infection and/or signs of severe sepsis/meningitis.
- Clinical features of inhalational anthrax.
- Respiratory symptoms + features of meningitis or intracranial bleeding.
- GI symptoms (eg pain, bleeding, nausea, vomiting, diarrhoea, ascites).

*Approach* Get expert help early to advise on management (microbiology, hospital infection control team, Public Health, ICU, surgeons). Start IV antibiotics according to advice (eg combination of ciprofloxacin, clindamycin + penicillin) or if there is soft tissue infection (ciprofloxacin, clindamycin, penicillin, flucloxacillin + metronidazole). Experts will advise on the use of anthrax immune globulin.

# Streptococcal infections

*Streptococcus pyogenes* and other streptococci may reside in the pharynx without symptoms but can cause sore throats (see ➲ Sore throat, pp. 570–1), soft tissue infections (see ➲ Infected wounds and cellulitis, p. 419; ➲ Cellulitis and erysipelas, p. 545), scarlet fever, endocarditis, and septicaemia. Later, non-suppurative sequelae of streptococcal infections include erythema nodosum, rheumatic fever (see ➲ Acute arthritis: 2, p. 513), and glomerulonephritis. Streptococci and staphylococci may cause necrotizing fasciitis, impetigo, and toxic shock.

## Scarlet fever[ND]

Some streptococcal infections are associated with scarlet fever. A diffuse blanching scarlet rash often involves the neck, chest, axillae, and groin. Occlusion of sweat glands makes the skin feel rough, like sandpaper. During the first 1–2 days of illness, there is a 'white strawberry tongue', with red papillae protruding through white furry material. After a few days, the white fur separates, leaving a shiny 'raspberry tongue'. Ten to 14 days after onset of the rash, the skin may peel from the palms and soles. Treat with penicillin V (or azithromycin if penicillin-allergic) for 14 days. Complete recovery is usual.

## Infective endocarditis

Endocarditis may develop on previously normal heart valves, as well as on diseased or prosthetic valves. The most common organism is *Streptococcus viridans*. Many acute cases present with heart failure and involve *Staphylococcus aureus*. Injecting drug users are liable to staphylococcal infection of the tricuspid valve, with fever and pneumonia from septic PE.

*Clinical features* Fever and changing murmurs suggest endocarditis. Emboli may cause strokes. Ask about weight loss, malaise, and night sweats. Look for clubbing, splinter haemorrhages, splenomegaly, anaemia, and microscopic haematuria.

*Treatment* On suspicion of endocarditis, admit immediately for investigation (blood cultures, echocardiography) and treatment.

## Necrotizing fasciitis

Rare and severe bacterial infection of soft tissues. It can occur with or without obvious trauma and may follow illicit IM heroin injection ('muscle popping'). *Streptococcus* pyogenes is often involved, sometimes with *S. aureus* or other bacteria. Often there are both aerobic and anaerobic organisms. Infection involves the fascia and subcutaneous tissues, with gas formation and the development of gangrene. Infection may spread to adjacent muscles, causing myonecrosis or pyogenic myositis. Similar infections may involve the abdomen and groin (Fournier's gangrene).

*Initial symptoms and signs* May be vague, with severe pain, but little on examination—the affected area may be tender, sometimes with slight erythema and swelling. Pyrexia is usual. Infection can spread rapidly and cause marked soft tissue swelling with discoloration, bruising, haemorrhagic blisters, or overlying skin necrosis. Toxic shock may develop—mortality is high. X-rays may show gas in the soft tissues.

*Treatment* Resuscitate with IV fluids and antibiotics (eg meropenem 2g and clindamycin 900mg), urgent surgery to debride the affected area and excise necrotic tissues, and ICU.

# Staphylococcal infections

*S. aureus* is involved in many infections of wounds, soft tissues (see ➔ Infected wounds and cellulitis, p. 419), joints, and bones (see ➔ Acute arthritis: 2, pp. 512–13; ➔ Osteomyelitis, p. 727; ➔ Septic arthritis, p. 511). Staphylococci also cause impetigo, scalded skin syndrome, food poisoning, toxic shock syndrome, endocarditis, pneumonia, septicaemia, and meningitis.

## Impetigo

This highly infectious superficial skin infection is caused by staphylococci or streptococci. It may involve normal skin or complicate a pre-existing condition such as eczema or scabies. Lesions often start around the mouth and nose, spreading rapidly on the face and to other parts of the body. Irregular golden-yellow crusted lesions occur, particularly in streptococcal infections. Staphylococci may cause bullous impetigo, with bullae containing pus which rupture and dry to form crusts. Treat with topical fusidic acid (usually qds for 7 days) and give PO flucloxacillin for 7 days (or azithromycin if allergic) if lesions are widespread or there is cellulitis or pyrexia.

## Scalded skin syndrome

*S. aureus* may produce an exotoxin causing separation of the outer layers of the epidermis, large sections of which slide off with minimal pressure, leaving large raw areas resembling a severe scald. Drug allergies can cause similar lesions. Most cases of scalded skin syndrome (toxic epidermal necrolysis, Lyell's syndrome) occur in children. Admit for nursing and medical care.

## Toxic shock syndrome

Caused by exotoxins from *S. aureus* or (less commonly) *S. pyogenes*. Some cases during menstruation are related to tampons, whilst other cases occur after surgical operations, burns, other trauma, or local infections. There is high fever, a generalized erythematous rash, confusion, diarrhoea, muscle pains, hypotension, and renal failure. Subsequently, scales of skin separate from the hands and feet. Death may occur from multi-organ failure. Treat for septic shock with IV fluids and anti-staphylococcal antibiotics. Remove tampons and send for culture. Refer to ICU. Involve a surgeon if an associated abscess requires drainage.

## Staphylococcal septicaemia

Occurs particularly in debilitated or immunocompromised patients and in injecting drug users. There may be endocarditis, with metastatic infection of lungs, bone, or soft tissues and gangrene due to emboli or arterial thrombosis. Signs of meningitis and DIC may suggest meningococcal septicaemia (see ➔ Meningitis, pp. 232–3) and the rash may be similar.

## Meticillin-resistant *Staphylococcus aureus* (MRSA)

MRSA causes particular concern because of antibiotic resistance and is carried by many asymptomatic people (patients and staff). Transmission is minimized by handwashing (see ➔ Infection control and prevention, pp. 36–7) and other infection control measures. Information about MRSA for patients is available online at https://www.nhs.uk

# Tetanus[ND]

An acute and often fatal disease, common in much of Asia, Africa, and South America, especially in neonates. Now rare in developed countries—30–40 cases/y in the UK, many involving the elderly. Injecting drug users (eg those 'skin popping') are also at particular risk. Spores of the Gram +ve organism *Clostridium tetani* (common in soil and animal faeces) contaminate a wound, which may be trivial. The spores germinate in anaerobic conditions, producing tetanospasmin, an exotoxin which blocks inhibitory neurones in the CNS and causes muscle spasm and rigidity.

Incubation period is usually 4–14 days but may be 1 day to 3 months. In 20% of cases, there is no known wound. Tetanus occasionally occurs after surgery or IM injections.

## Clinical features

Stiffness of the masseter muscles causes difficulty in opening the mouth (trismus, lockjaw). Muscle stiffness may spread to all facial and skeletal muscles and muscles of swallowing. Characteristically, the eyes are partly closed and the lips pursed and stretched (risus sardonicus). Spasm of chest muscles may restrict breathing. There may be abdominal rigidity, stiffness of limbs, and forced extension of the back (opisthotonus). In severe cases, prolonged muscle spasms affect breathing and swallowing. Pyrexia is common. Autonomic disturbances cause profuse sweating and tachycardia and hypertension, alternating with bradycardia and hypotension. Cardiac arrhythmias and arrest may occur.

## Differential diagnoses

Dystonic reaction to metoclopramide or phenothiazines, strychnine poisoning, quinsy, dental abscess, meningitis, and rabies. Procyclidine relieves muscle spasms from drug-induced dystonia but will not affect tetanus; diazepam may relieve dystonia or tetanic spasms.

## Management

Obtain senior medical and anaesthetic help. Monitor breathing, ECG, and BP. Refer to ICU. Control spasms with diazepam. Paralyse and ventilate if breathing becomes inadequate. Clean and debride wounds. Give penicillin, metronidazole, and human tetanus immunoglobulin.

## Prognosis

Depends on severity of disease and quality of care. Short incubation (<4 days) and rapid progression suggest severe disease, with a high mortality. With expert intensive care, mortality in adults is <10%, but neonatal tetanus is often fatal.

## Immunization

Tetanus is eminently preventable by immunization and proper care of wounds (see ➲ The approach to wounds, p. 410; ➲ Tetanus prophylaxis, p. 424).

## Gas gangrene

This is a rapidly spreading infection of muscle caused by toxin-producing clostridial bacteria (anaerobic Gram +ve bacilli), usually *Clostridium perfringens*. It is fatal if untreated. It may involve wounds of the buttocks, amputations for vascular disease, or severe muscle injuries (eg gunshot wounds). Occasionally gas gangrene of the perineum occurs without trauma.

### Incubation period

Is usually <4 days (sometimes a few hours). Sudden severe pain occurs at the wound site. Generalized toxicity develops, with tachycardia, sweating, and fever. Swelling and skin discoloration occur around the wound, with a serous ooze, marked tenderness, and sometimes haemorrhagic vesicles and crepitus. Shock and AKI develop, with death often within 2 days of the first symptoms.

### Diagnosis

Depends on clinical features. Severe pain necessitates wound inspection (remove or window any POP). Obtain immediate senior surgical advice if gas gangrene is suspected. Wound discharge may contain Gram +ve bacilli. X-rays may show soft tissue gas, but its absence does not exclude gas gangrene.

### Treatment

IV antibiotics (eg penicillin and clindamycin), immediate surgical removal of all infected tissue, and ICU. Hyperbaric $O_2$ and gas gangrene antitoxin are rarely available and of no proven benefit.

## Botulism[ND]

*Clostridium botulinum* exotoxin paralyses autonomic and motor nerves by blocking acetylcholine release at neuromuscular junctions and nerve synapses. Infection follows eating tinned or preserved food contaminated with *C. botulinum* spores. Rarely, *C. botulinum* infects wounds or colonizes the gut. Injecting drug users may develop botulism after IM or SC injections of contaminated drugs.

**Incubation period** 12–72hr. Initial symptoms may be GI (nausea, vomiting, abdominal discomfort, dryness of the mouth) or neurological (dizziness, blurred vision, diplopia). Later problems include dysarthria, dysphagia, muscle weakness or paralysis, constipation and urinary retention, respiratory failure, and sudden death. Susceptibility varies—some people who eat contaminated food develop no symptoms or suffer only mild fatigue.

**Clinical signs** Result from involvement of autonomic and motor nerves: dry mouth, cranial nerve palsies (ptosis, squint, fixed pupils, weakness of tongue), and limb weakness with flaccid muscles. Consciousness and sensation are preserved. Hypotension and ileus may occur. Fever is unusual.

**Differential diagnoses** Guillain–Barré syndrome, myasthenia, brainstem stroke, diphtheria, and rabies. It may be misdiagnosed as staphylococcal food poisoning, paralytic shellfish poisoning, and CO or mushroom poisoning.

**Management** Get senior help. Assess breathing; ventilate if necessary, and admit to ICU. Botulinum antitoxin ↓ mortality and morbidity—see *BNF* and TOXBASE (see ➲ National Poisons Information Service, pp. 188–9). Inform Public Health—others who have eaten contaminated food may need urgent treatment. Anticipate media enquiries.

# Sexually transmitted infections

The most common sexually transmitted infection (STI) is *Chlamydia*. Other common diseases include gonorrhoea, genital herpes, trichomoniasis, genital warts, *Mycoplasma genitalium*, *Pediculosis pubis*, HIV, and syphilis. Many patients have more than one disease. Suspicion of STI necessitates prompt referral to a GU medicine clinic for proper diagnosis, treatment, and follow-up of the patient and contacts. Some GU departments allow self-referral. Others provide an on-call service. Only prescribe antibiotics for suspected STIs on the advice of a GU specialist.

## Genital ulcers and sores

Most genital ulcers/erosions are either multiple and painful or single and painless. In the UK, multiple genital ulcers are most often due to herpes simplex; other causes are Behçet's disease, chancroid, Stevens–Johnson syndrome, and scabies. Multiple painful sores may occur with gonorrhoea, *Candida*, or other conditions. Syphilis can cause painless or painful ulceration. Primary chancre is a single ulcer, and secondary syphilis often multiple—both are highly infectious—and the incidence has ↑ recently. Other causes of painless ulcers include carcinoma and trauma.

## Urethritis

In men, dysuria and urethral discharge are the most common presenting symptoms of an STI. However, 5–10% of men with gonococcal or non-gonococcal urethritis have no symptoms. Urethritis may result from physical trauma, FBs, or attempts at self-treatment with intra-urethral chemicals.

*Gonorrhoea* Usually has a shorter incubation period (3–5 days) than non-gonococcal urethritis (eg *Chlamydia* 7–14 days) but do not rely on a clinical diagnosis—refer to a GU clinic for diagnosis, management, and follow-up. If no GU advice is available and treatment cannot wait for attendance at a GU clinic, give ceftriaxone 500mg IM + azithromycin 1g PO. If possible, make a glass slide of the discharge, dried in air, for the patient to take to the clinic. Advise the patient not to pass urine for 2hr before the appointment, in order to allow serial urine samples to be taken.

*Reactive arthritis (previously called Reiter's syndrome)* Is a rare complication of non-gonococcal urethritis. There is arthritis (mainly of the knees, ankles, and feet) and sometimes conjunctivitis, rashes, and cardiac and neurological problems.

## Gonorrhoea

Gonorrhoea may infect the urethra, cervix, rectum, pharynx, or conjunctiva. Men usually have dysuria and urethral discharge, with rectal discharge and tenesmus in rectal infection. Women are often asymptomatic but may have dysuria and vaginal discharge.

*Complications* Include prostatitis, epididymitis, salpingitis, and Bartholin's abscess; rarely, septicaemia with arthritis, fever, rash (maculopapular initially, then pustular), and endocarditis.

# Hepatitis[ND]

## Hepatitis A (infectious hepatitis)[ND]

Hepatitis A occurs throughout the world but is particularly common in the tropics and subtropics. It is transmitted by contamination of food or water with infected faeces or urine. Many infections are asymptomatic. The incubation period is 2–6 weeks (usually ~4 weeks). Fever, malaise, anorexia, and nausea may last for 2–7 days before jaundice develops. Jaundice is more common in adults than in children and is associated with dark urine, pale stools, and tender hepatomegaly.

*Treatment* Is symptomatic, but advise avoidance of alcohol. Infectivity is greatest before jaundice develops, so isolation is of little value. Arrange follow-up by a specialist or GP. Complete recovery is usual. Consider hepatitis A vaccine for close contacts (see *BNF*).

## Hepatitis B[ND]

Hepatitis B is transmitted by infected blood (eg shared needles in drug users, tattooing, needlestick injury) and sexual intercourse. Incubation period is 6 weeks to 6 months. Symptoms are similar to hepatitis A, often with arthralgia and skin rashes. Most patients with hepatitis B recover completely. A few develop liver failure or chronic hepatitis, with a risk of liver cancer. Asymptomatic carriers of hepatitis B virus are common (~0.1% of UK population, but ~20% in parts of Africa and Asia). All health care workers should be immunized against hepatitis B and use 'standard precautions' (see ➲ Infection control and prevention, pp. 36–7) when handling all blood samples and 'sharps'. The management of needlestick injury is described in ➲ Needlestick injury, p. 425.

## Hepatitis C, D, and E[ND]

*Hepatitis C and D* Are spread in the same way as hepatitis B and may cause hepatic failure or chronic liver disease. New treatments for hepatitis C (eg ledipasvir + sofosbuvir) have transformed the prognosis.

*Hepatitis E* Is similar to hepatitis A but has high mortality in pregnancy. Refer to a specialist for follow-up.

# Leptospirosis (Weil's disease)[ND]

Leptospirosis, caused by the spirochaete *Leptospira interrogans* and other *Leptospira* species, is spread by contact with infected rat's urine, often in rivers, canals, or sewers. Leptospires enter the body through small breaks in the skin or via mucous membranes of the eyes or nose. About 10 days after exposure (range 2–26 days), the illness starts with fever, severe muscle pains, headache, sore throat, nausea, and vomiting. Conjunctival reddening is common. A haemorrhagic rash, jaundice, renal failure, and pulmonary haemorrhage may occur (Weil's disease).

Refer to an infectious diseases unit. Treatment is with penicillin or doxycycline, with supportive care and haemodialysis if necessary. Prophylactic doxycycline is reasonable for people who fall into waterways likely to be contaminated with leptospires.

# Human immunodeficiency virus

First reports of AIDS involved the homosexual community in the USA in 1981. HIV was identified as the causative agent in Paris in 1983.

## Structure and pathogenesis

HIV is an RNA retrovirus. Retroviruses are characterized by having the enzyme reverse transcriptase. This allows viral RNA to be transcribed (copied) into DNA and incorporated into host cells, which then make a new virus. This mechanism has proved difficult to overcome—despite much effort, no 'vaccine' is yet available and a cure is elusive.

Glycoproteins on the surface of HIV bind to specific receptors on target cells. The cellular receptor for HIV is the CD4 molecule. CD4 receptors are found on a variety of cells, particularly helper/inducer T lymphocytes ('CD4 cells'), but also monocytes and macrophages. CD4 cells normally play a crucial role in co-ordinating the immune response—as HIV infection progresses and CD4 cell counts ↓, the patient develops profound cellular immunodeficiency. Although other complex mechanisms are also involved, CD4 cell counts provide a useful index of disease stage and progress.

## Transmission

HIV has been found in many body fluids but is mostly transmitted via blood, semen, cervical secretions, and perhaps breast milk. It may be acquired by:

- Sexual intercourse (vaginal or anal), with an ↑ risk of transmission where individuals already have a genital mucosal breach (eg coexisting STI).
- Risk of transmission from HIV +ve pregnant mother to baby is ~15%.
- Transfusion of unscreened blood/blood products (screening started in 1985 in the UK).
- Contaminated needles shared amongst IV drug users. Needlestick injuries from an HIV +ve source carry a risk of ~0.3%.

## Prevention

Post-exposure prophylaxis can significantly ↓ the risk of acquiring HIV after a needlestick injury or unprotected sexual contact (see ➲ Needlestick injury, p. 425).

An ↑ number of those at high risk of acquiring HIV are taking antiretroviral therapy as pre-exposure prophylaxis ('PrEP').

## Diagnosis and HIV testing

Antibodies to HIV provide evidence of infection and form the basis of many blood tests, but these antibodies may not appear until 3 months after exposure. Over-the-counter test kits are now available.

Many HIV +ve patients attending the ED are aware of their HIV status. Some patients, however, present with HIV-related illness, without knowing (or admitting) that they are HIV +ve. Test for HIV (with consent) in ED patients where it may influence immediate management.

Routine screening for HIV in ED patients in the UK has been proposed where there is a relatively high prevalence (>2/1000 population).

## Natural history of HIV infection

Acute infection is often subclinical, but 2–6 weeks after exposure, there may be non-specific febrile illness with lethargy, myalgia, sore throat, lymphadenopathy, and often a maculopapular rash on the face and trunk. This illness usually resolves after 1–2 weeks but sometimes persists for longer. A long asymptomatic period (~10y) follows the initial illness.

Some patients develop persistent generalized lymphadenopathy (PGL), with lymphadenopathy (>1cm) at two non-inguinal sites for 3 months.

Following a latent phase of chronic infection, patients (especially without treatment) are at risk of developing symptoms as immunity ↓, developing unusual infections and tumours.

Antiretroviral therapy delays the progression of HIV-related illness and ↑ length of survival.

## Initial presentation of HIV to the ED

Presentation of any of the diseases listed below should arouse particular suspicion.

*Centers for Disease Control HIV infection classification*

A patient's CD4 cell count helps to provide information on immune function and disease progression—the combination of this with clinical status underpins the Centers for Disease Control (CDC) classification:

- Stage 1—no AIDS-defining condition and either a CD4 cell count of ≥500 cells/mm$^3$ or CD4 percentage of ≥29%.
- Stage 2—no AIDS-defining condition and either a CD4 cell count of 200–400 cells/mm$^3$ or CD4 percentage of 14–28%.
- Stage 3 (AIDS)—a documented AIDS-defining condition or a CD4 cell count of <200 cells/mm$^3$ or a CD4 percentage <14%.

*Some AIDS-defining diseases in HIV +ve patients*

- *Pneumocystis jiroveci* pneumonia (previously called *P. carinii*).
- Kaposi's sarcoma.
- Tracheobronchial or oesophageal candidiasis.
- Cerebral toxoplasmosis.
- Pulmonary TB.
- Recurrent pneumonia.
- CMV retinitis.
- Cerebral lymphoma.
- Recurrent *Salmonella* septicaemia.
- Disseminated histoplasmosis.
- Invasive cervical carcinoma.
- Disseminated or extrapulmonary coccidioidomycosis.
- Extrapulmonary cryptococcosis.
- Chronic intestinal cryptosporidiosis for >1 month.
- Progressive multifocal leukoencephalopathy.
- Oesophageal or bronchial herpes simplex for >1 month.
- Histoplasmosis.
- Wasting syndrome attributed to HIV.

# Presentation of HIV +ve patients

Some patients with symptomatic HIV infection bypass the ED and liaise directly with the specialist unit caring for them. Assessment of HIV +ve patients is difficult in the ED where advanced infections may present with relatively few signs and little past history is available. Similarly, interpretation of investigations is difficult without knowledge of previous results. On this basis, adopt a low threshold for specialist referral. HIV +ve patients may present with a variety of complications.

## Respiratory problems

As CD4 counts ↓, pneumonia due to *Pneumocystis jiroveci* (previously *Pneumocystis carinii*) becomes more likely and it is a common indicator diagnosis of AIDS. A non-productive cough occurs with dyspnoea and fever. CXR may show bilateral interstitial mid-zone shadowing but may be normal. Obtain blood and sputum cultures; rehydrate with IV fluids as necessary, and refer urgently for IV co-trimoxazole or pentamidine ± steroids. Occasionally, *Pneumocystis* infection may present with fulminant respiratory failure, needing emergency tracheal intubation and IPPV. Other common infections include *Aspergillus, Cryptococcus*, and TB. Injecting drug users are at an ↑ risk of bacterial infection, especially *Haemophilus influenzae* and *Streptococcus pneumoniae*.

## Neurological problems

*Cryptococcus neoformans* meningitis may present with headache, fever, and sometimes ↓ conscious level. Neck stiffness and photophobia are rare. Obtain a CT scan to exclude space-occupying lesions before LP and CSF examination. *Cerebral toxoplasmosis* may present similarly, often with focal signs or fits. Neurological problems may also be caused by cerebral lymphoma, progressive leucoencephalopathy (focal deficits secondary to papovaviruses), CMV encephalitis (retinopathy is usually present—see below), and HIV-associated delirium or dementia.

## Eye problems

The most significant eye problem is *CMV retinitis*, occurring in 15% of patients. This presents with blurred vision, blind spots, 'floaters' or flashing lights, and ↓ VA. Characteristic retinal changes are irregular yellow-white lesions and perivascular haemorrhages that have been called 'pizza pie'. Retinal detachment may occur. Refer urgently for ophthalmological assessment and treatment with ganciclovir.

## Mucocutaneous problems

Oral candidiasis, seborrhoeic dermatitis, and oral hairy leukoplakia (white ridges on the lateral border of the tongue) are often seen before AIDS develops. As immunity ↓, patients may develop herpes simplex, herpes zoster, and molluscum contagiosum. Gum bleeding and dental problems are common—the former may be due to thrombocytopenia. Kaposi's sarcoma is seen in the skin and mucous membranes. It is rarely life-threatening but requires specialist evaluation and treatment.

### Gastrointestinal problems

Nausea, vomiting, diarrhoea, and weight loss are common complaints and can be due to drug therapy. Dysphagia may result from oesophageal candidiasis, herpes simplex, CMV, or Kaposi's sarcoma, all of which require specialist investigation and treatment.

*CMV colitis* can cause a serious illness, characterized by abdominal pain, diarrhoea, and fever. Obtain plain X-rays if the recognized complication of toxic dilatation is suspected. Other frequently implicated infective causes of diarrhoea include *Cryptosporidium, Giardia, Microsporidium*, and *Salmonella*. Send stool specimens (including for *Clostridium difficile*) and treat severe diarrhoea by IV rehydration and correction of electrolyte imbalance before referral.

*Hepatitis viruses* are likely to complicate the picture in injecting drug users, many of whom are infected with hepatitis B and C.

### Drug reactions and side effects

Some patients present with symptoms due to drug therapy. This may not be initially apparent—the safest approach is to exclude tumours and opportunistic infection first.

### HIV and ED staff

ED staff are often concerned about the possibility of acquiring HIV from patients. The need to perform invasive emergency procedures on 'high-risk' patients makes these concerns understandable. Additionally, apparently 'low-risk' patients with untreated HIV may also pose a greater threat than patients known to have HIV who are taking antiretroviral therapy. Therefore, treat every patient as 'high risk'. The risk to ED staff is largely in the form of needlestick injury [although the risk of acquiring HIV following a needlestick injury from a HIV +ve source can be ↓ by post-exposure prophylaxis (see ➲ Needlestick injury, p. 425]. Safe practice is reflected in the recommended standard precautions (see ➲ Infection control and prevention, pp. 36–7)—follow these in all patients. Pregnant staff should not treat patients with AIDS (because of concern about CMV and herpes simplex virus).

### Handling HIV +ve patients

Despite vigorous attempts to educate the general public, HIV remains a taboo subject amongst many in society. It is imperative to treat all patients, including those who are HIV +ve, with sensitivity and compassion. In view of prevailing attitudes, patient confidentiality is of the utmost importance. Remember that family and friends accompanying the patient may be unaware of the patient's HIV status.

### HIV +ve staff

The risk to patients from ED staff infected with HIV is minimal but remains a theoretical possibility. Staff who believe that they may be HIV +ve must obtain and follow occupational health advice.

### Needlestick injury

See ➲ Needlestick injury, p. 425.

# Imported infectious diseases

Patients may present to the ED with infectious diseases acquired abroad. It is essential to ask where a patient has been, especially in the 6 weeks before the onset of symptoms. The most common imported diseases are bowel infections causing diarrhoea (see ➔ Gastroenteritis/food poisoning[ND], pp. 236–7). Less common, but very important, diseases include malaria (see ➔ Malaria[ND], p. 255), typhoid (see ➔ Typhoid[ND] and parathyroid[ND] (enteric fever), p. 256), legionnaires' disease (see ➔ Pneumonia, pp. 114–15), and hepatitis (see ➔ Hepatitis[ND], p. 249). Rabies (see ➔ Rabies[ND], p. 257) and viral haemorrhagic fevers, such as Lassa fever (see ➔ Viral haemorrhagic fevers[ND], p. 258), are very rare in the UK.

Occasionally, tropical diseases are acquired in Britain from bites by infected insects carried by plane (eg 'airport malaria').

Advice about tropical diseases is available from departments of infectious diseases or tropical medicine, including:

- The Hospital for Tropical Diseases (based in London) (🔗 http://www.thehtd.org; telephone 0203 456 7890.
- A public access website provided by the NHS which gives information for people travelling abroad from the UK (🔗 https://www.fitfortravel.scot.nhs.uk).
- An alternative is 🔗 https://travelhealthpro.org.uk

## Pyrexia of unknown origin in travellers

Think of, and check for, malaria (see ➔ Malaria[ND], p. 255) in any febrile patient who has been in a malarious area. Consider Lassa fever (see ➔ Viral haemorrhagic fevers[ND], p. 258) in someone who has been in West Africa in the previous 3 weeks. Typhoid (see ➔ Typhoid[ND] and parathyroid[ND] (enteric fever), p. 256) often presents as a septicaemic illness with constipation, rather than diarrhoea. TB (see ➔ Tuberculosis[ND], p. 242) and brucellosis may cause fever and sweating at night.

### *Investigations*

Warn the lab of possible risk of infection.

- FBC, thick and thin blood films for malaria.
- U&E, blood glucose, blood culture.
- Urine stick testing, microscopy, and culture.
- CXR.

Further investigations may include LFTs and viral titres.

### *Management*

Barrier nurse in a cubicle (ideally use a negative-pressure room if available). Wear gown, gloves, goggles, and mask. Record vaccination and prophylaxis history, with countries and areas visited and dates of travel and onset of symptoms. Look particularly for confusion, dehydration, jaundice, rashes, chest signs, liver and spleen enlargement and tenderness, lymphadenopathy, neck stiffness, and photophobia. Seek expert advice at once if the patient is very ill or there is concern about typhoid or Lassa fever or other VHFs. Refer to an infectious diseases specialist.

# Malaria[ND]

Malaria is very common in the tropics and subtropical regions, and is a parasitic infection transmitted by mosquitos. The five species which cause malaria in humans are *Plasmodium falciparum, P. vivax, P. malariae, P. ovale*, and *P. knowlesi*. Falciparum ('malignant tertian') malaria is the most important, since it may be rapidly fatal and drug-resistant strains are common. Serious complications are unusual in the other types of malaria, but they may cause febrile convulsions in children.

In the UK, malaria occurs in travellers from malarious areas, especially *P. vivax* from the Indian subcontinent and *P. falciparum* from Africa, South East Asia, and Central and South America. Malaria often develops despite antimalarial tablets, because of drug resistance or incorrect dosage. Check for malaria in any febrile illness within 2 months of visiting a malarious area. Common misdiagnoses are influenza and viral hepatitis.

Specific country information about the risk of malaria is available on https://travelhealthpro.org.uk

## Clinical features

The incubation period is usually 7–14 days for *P. falciparum* and 12–40 days for other types of malaria, but occasionally it is much longer (>1y), especially in *P. malariae* and *P. vivax* infections. Malaise, fatigue, fever, and headache are followed by paroxysms (lasting 8–12hr) of rigors, vomiting, and then severe sweating. Fever may be periodic (classically 48hr in *P. ovale* or *P. vivax*, and 72hr in *P. malariae*). Haemolytic anaemia, jaundice, and splenomegaly may occur, but lymphadenopathy is not a feature. *P. falciparum* may cause cerebral malaria with coma, fits, and focal neurological signs. Diarrhoea, cardiac failure, pulmonary oedema, and shock may occur. Deterioration can be rapid.

## Investigations

Consider Lassa fever (see Viral haemorrhagic fevers[ND], p. 258) in recent visitors to West Africa. Send blood for thin and thick film examination for malaria in any ill person who has visited a malarious area. Repeated blood films may be needed. Also arrange FBC (malaria may cause anaemia, thrombocytopenia, neutropenia), blood glucose (hypoglycaemia may occur), and U&E (AKI possible), and test the urine for blood ('black water fever').

## Treatment of malaria

Involve ICU and get urgent expert advice from a tropical disease specialist (see Imported infectious diseases, p. 254). Artemisinin derivatives (such as artemether or artesunate) are more effective than quinine for the management of falciparum malaria—treat with an artemisinin combination, such as artemether–lumefantrine (Riamet®) according to the UK malaria treatment guidelines (2016) (see https://www.journalofinfection.com/article/S0163-4453(16)00047-5/pdf). These guidelines also outline the management of less severe non-falciparum malaria.

# Typhoid[ND] and paratyphoid[ND] (enteric fever)

These fevers, caused by *Salmonella typhi* and *S. paratyphi A, B,* or *C*, occur throughout the world, especially where hygiene is inadequate. They are spread by contamination of food or water by urine or faeces from a patient or an asymptomatic carrier. Typhoid may occur despite immunization. Typhoid and malaria are the first diseases to consider if fever develops soon after a visit to the tropics. The *incubation period* is usually 7–14 days but may range from 3 to 60 days.

### Initial symptoms

Headache, fever, and a dry cough, with abdominal discomfort and anorexia. Constipation is common, but diarrhoea may occur, especially in children. Confusion and hallucinations may develop.

### Physical examination

This may be normal, except for fever. There may be relative bradycardia (ie less than the usual 10 beats/min ↑ in pulse rate per °C of fever). Splenomegaly and abdominal tenderness occur, but there is no lymphadenopathy. 'Rose spots' are pink macular spots on the lower chest or upper abdomen which blanch on pressure. There may be signs of pneumonia or dehydration. Intestinal perforation or haemorrhage occur occasionally.

### Investigations

FBC (mild anaemia is common, WCC usually normal), blood films for malaria, U&E, LFTs, blood cultures, and CXR (for signs of TB or pneumonia).

### Treatment

Isolate and barrier nurse. Admit suspected cases to an infectious diseases unit and notify the local consultant in communicable disease control. The usual drug treatment is with ciprofloxacin or cefotaxime, but other antibiotics may be needed for drug-resistant infections.

# Dengue

Dengue is a mosquito-borne viral infection which is common in Southern Asia, the Western Pacific, Central Africa, and Central and South America. Most infections are asymptomatic. Symptoms start after an incubation period of 4–7 days with fever, malaise, nausea and vomiting, headache, and severe muscle and bone pains ('break bone fever'). Some patients have a transient macular rash, petechiae, lymphadenopathy, hepatomegaly, ↓ WCC, ↓ platelets, and ↑ liver enzymes.

Most patients recover after 3–7 days with symptomatic treatment. A few develop dengue shock syndrome (DSS) with hypotension, pleural effusions, ascites, ↓ plasma protein, and bleeding problems. Abdominal pain may be severe. Treatment is supportive, with careful fluid balance management and IV fluids in DSS. With expert care, most patients with severe dengue eventually make a full recovery.

# Poliomyelitis[ND]

Paralytic poliomyelitis is rare in developed countries where vaccination is routine. Fever is followed by signs of meningitis, pain, and spasm in limb muscles. Respiratory failure may be fatal.

*Resuscitate and ventilate* if necessary and refer to ICU. The differential diagnosis includes Guillain–Barré syndrome (see Acute generalized weakness, pp. 148–9) and organophosphate poisoning (see Organophosphate poisoning, p. 214).

# Rabies[ND]

Rabies is a viral infection of mammals that occurs in most parts of the world, including much of the Arctic, as well as tropical and temperate regions. At present, it is not endemic in the UK, Norway, Sweden, Iceland, Australasia, or Japan. Human and animal rabies is most common in the Indian subcontinent, China, Thailand, the Philippines, and parts of South America. Most human infections result from dog bites, but rabies can be transmitted by many other domesticated or wild animals such as cats and foxes. Rabies virus in an animal's saliva may cause infection by contamination of a bite or scratch, or by absorption through mucous membranes of the eye, mouth, or nose. Rarely, infection occurs from inhalation of the virus in bat-infested caves.

Prevention of rabies after a bite is described in Bite wounds, pp. 420–1. Detailed information about rabies is available at http://www.gov.uk/government/publications/rabies-the-green-book-chapter-27. This document includes detailed advice on both pre-exposure prophylaxis and post-exposure treatment.

## Clinical features

The *incubation period* of rabies is usually 3–12 weeks but can vary from a few days to >2y.

The first symptoms are itching, tingling, or pain at the site of the bite wound. Headache, fever, and malaise occur, with spreading paralysis and episodes of confusion, hallucination, and agitation. Hydrophobia is characteristic—attempts at drinking cause spasm of muscles involved in breathing and swallowing and also profound terror. In ~20% of cases, there is 'dumb rabies', with ↑ paralysis, but no episodes of spasm or hyperactivity. Rabies is almost always fatal, even with ICU treatment.

## Management

If rabies is suspected, barrier nurse the patient in a quiet room with a minimum of staff who must wear gowns, gloves, eye protection, and masks. Obtain advice immediately from a specialist in infectious diseases. Anticipate press enquiries. Record the names of all staff involved, so that they can be offered rabies immunization.

# Viral haemorrhagic fevers[ND]

## Lassa fever

Lassa fever occurs in many rural parts of West Africa. It is a viral infection acquired from infected blood or secretions, transmitted by inadvertent innoculation (eg needlestick injuries) or contamination of mucous membranes or broken skin. In Africa, it is transmitted by multimammate rats. The incubation period is up to 3 weeks. There is high mortality.

*Early symptoms* Are non-specific with fever, malaise, headache, sore throat, retrosternal chest pain, and backache. Periorbital oedema, swelling of the neck, and conjunctival injection are common. Suspect Lassa fever in any pyrexial patient who has been in rural West Africa (south of the Sahara) in the previous 3 weeks. However, malaria and typhoid are much more common and need urgent diagnosis and treatment.

*Management* If Lassa fever is possible, barrier nurse the patient in a cubicle by staff wearing gloves, gowns, goggles, and masks. Take special care to avoid needlestick injuries, which may cause fatal infection. Before taking any blood samples, discuss the case with a tropical diseases specialist and the local consultant in communicable disease control. Start treatment immediately for falciparum malaria (see ➲ Malaria[ND], p. 255). Warn the laboratory about Lassa fever and send blood for examination for malaria. The patient will be admitted to an isolation bed, possibly in a high-security infectious diseases unit.

## Ebola fever and Marburg fever

These are VHFs which occur in West and Central Africa (Democratic Republic of the Congo, Uganda, Kenya, and Sudan) and have similar clinical features and high mortality. Transmission is usually by infected blood, but the viruses may be acquired from monkeys or apes. The incubation period is usually 4–10 days. Illness starts suddenly with severe headache, high fever, and generalized pains, especially in the back, followed by severe diarrhoea, abdominal pain, dry throat, a maculopapular rash, conjunctivitis, and GI bleeding. Isolate and treat as for suspected Lassa fever.

## Other viral haemorrhagic fevers

Diseases with similar features (plus, in some cases, jaundice) include dengue (see ➲ Dengue, p. 256), Crimean–Congo fever (Central Africa, parts of Eastern Europe, and Asia), and yellow fever (Africa and South America). The initial management is the same as for Lassa fever.

## Middle East respiratory syndrome

Due to a coronavirus, this is also known as 'MERS' or 'MERS-CoV' which was first identified in 2012. It is a severe respiratory illness (mortality is >30%) which presents with fever, cough, and breathlessness. Consider it in patients with respiratory symptoms who have returned from Saudi Arabia and neighbouring areas. Obtain specialist and ICU help. Treatment is supportive, and at the present time, the risk of human-to-human transmission appears to be relatively low.

# Severe acute respiratory syndrome

## Background

Severe acute respiratory syndrome (SARS) is a viral respiratory illness caused by a coronavirus. SARS was first recognized in March 2003 but probably originated in November 2002 in the Guangdong province of China where the virus has been found in wild animals. SARS spread to several countries, causing deaths in South East Asia and Canada in March to May 2003. Few cases have occurred since then. No cases are known at the time of writing, but there is concern that SARS may re-emerge from China.

## Spread

SARS is spread by respiratory droplets produced when an infected person coughs, sneezes, or uses a nebulizer. The virus can also spread when someone touches an object contaminated by infectious droplets and then touches his/her mouth, nose, or eyes.

## Features

The incubation period of SARS is usually 2–7 days but may be up to 10 days. The illness starts with fever (>38°C), usually associated with rigors, headache, muscle pains, and malaise. Diarrhoea may occur. Some patients have mild respiratory symptoms initially. A dry cough develops after 2–7 days, with ↑ breathlessness from hypoxia caused by pneumonia. CXR may be normal or may show patchy infiltrates, and later areas of consolidation. WCC is usually normal or ↓ initially (lymphopenia).

## Management

If SARS is suspected, get expert help (ED consultant, infectious diseases specialist, and infection control staff) and isolate the patient (if possible, in a negative-pressure room). Ensure that a minimum number of staff have contact with the patient. Staff who do have contact must wear masks or respirators (of FFP3 standard), goggles, gowns, and gloves, with strict handwashing and careful disposal of all items. Provide the patient with an N95 mask or a surgical mask. Record $SpO_2$ and give $O_2$ if necessary, but avoid flow rates of >6L/min to minimize virus aerosolization. If bronchodilators are needed, use a spacer inhaler, rather than a nebulizer. Maintain a list of all contacts. Expect press enquiries.

An expert will help to assess to decide about admission. Those admitted should ideally be placed in a negative-pressure isolation room with full infection control measures. Treat as for community-acquired pneumonia (see ➲ Pneumonia, pp. 114–15).

Further information about SARS is available at 🖱 https://www.hse.gov.uk

# COVID-19

This section has been written whilst current knowledge of this illness is at an early stage. Refer to latest guidance (https://www.gov.uk).

## Background, origin, and spread

In 2019, illness from a coronavirus (SARS-CoV-2) emerged from Wuhan in China and spread throughout the world. It was quickly apparent that it was a relatively mild illness for most, but a small proportion developed ARDS requiring IPPV, with an estimated 2% mortality rate. The initial response to this coronavirus disease 2019 (COVID-19) varied considerably between countries, in terms of testing, contact tracing, availability of ventilators, and PPE for health care workers. Within 6 weeks of a senior politician telling the public that the 'US has contained the virus', that country had the highest number of deaths in the world and the illness had been declared a pandemic.

## Transmission and incubation period

COVID-19 is spread principally by close contact between individuals, especially by respiratory droplets (coughing, sneezing, breathing), some of which may contaminate surfaces. The incubation period is 2–14 days, typically 5 days, with individuals believed to be infectious before displaying symptoms.

## Clinical features

In March 2020, an ED consultant in Cornwall, UK reported: 'An initial feeling of headache, intense tiredness and cold, progressive over several hours, gave way to fever, shivering, and myalgia.' He also described 'some tight feelings in my chest and loss of sense of smell'. He recovered completely within 10 days.

The main symptoms are fever, persistent cough, and breathlessness. Some patients also experience headache, myalgia, chest tightness, GI disturbance, and loss of sense of smell. There are no distinctive features on examination, even in those with CXR abnormalities. Patients who become seriously ill typically deteriorate 7–10 days after onset of symptoms. They may have hypoxia on minimal exertion, a high NEWS2, and features of ARDS.

## High risk factors

The mortality rate is much higher for older individuals, especially men and those with pre-existing health problems (including immunosuppression, diabetes, hypertension, cardiovascular and respiratory disease). Healthy children and young adults may still require hospital treatment, including IPPV, but are at relatively low risk of death. Of relevance to health care workers, there is evidence that being exposed initially to a large amount of virus may predispose to more severe illness.

### Management

- On initial suspicion, isolate the patient and put a mask on him/her; protect staff and other patients: don appropriate PPE ('full PPE' including FFP3 respirator, visor, long-sleeved gown and gloves for high risk procedures such as airway management/intubation, suctioning, NIV).
- Consider early discharge with advice for patients not seriously ill who have NEWS2 of <3 and no hypoxia on minimal exertion. High risk patients seen early (in the first 7 days) may benefit from community follow up at 7–10 days to check for deterioration.
- Take blood for FBC, U&E, CRP, D-dimer, LFTs, coagulation, ferritin, procalcitonin, troponin, cultures.
- Take upper respiratory swabs for COVID-19.
- Consider imaging, depending upon local resources. CXR may show peripheral and basal ground glass opacities in patients predisposed to ARDS. USS correlates well with CXR (and CT) findings.
- Provide $O_2$ as required: target $SpO_2$ 94–98% (88–92% in COPD).
- Give paracetamol and oral fluids. Avoid IV fluids in ARDS. If needed, start with 250mL IV 0.9% saline bolus.
- Consider alternative diagnoses (eg bacterial pneumonia) and treat accordingly.
- Refer seriously unwell patients early to ICU to consider intubation and IPPV.
- Decide early about the ceiling of care/treatment escalation plan.
- Do not start CPR (or other aerosol-generating procedures) unless all staff are wearing PPE.

### Health care workers

Tragically, many health care workers have already died as a result of treating patients with COVID-19 (see dedication). Strict adherence to correct use (including donning and doffing) of PPE helps reduce risks.

### Community strategy

Measures to prevent spread of COVID-19 in the community have included social distancing, isolation, testing, contact tracing, shutdown of schools, restaurants, and businesses, with dramatic social and economic consequences.

### Future treatments

There is hope that in the short term, antiviral and antibody treatments will reduce the mortality rate. Longer term hopes are focussed on a vaccine.

# Influenza pandemics, avian flu, and swine flu

## Background

*Influenza* is common in the UK and many other countries, particularly during winter. Most people are ill for only a few days with fever, muscle aches, coughing, and nausea, but there are some deaths, especially in elderly people.

*Pandemic influenza* occurs when a new subtype of influenza A emerges, which can spread easily from person to person and which is different from previous strains (so there is no pre-existing immunity). Influenza pandemics occurred in 1918–1919 (with 40–50 million deaths worldwide, including many children and young adults) and also in 1957 and 1968. Another pandemic could develop at any time. There was concern about influenza A subtype H5N1, which infected poultry in Hong Kong in 1997 and 2003 and spread to birds across South East Asia, with carriage by migrating birds across Asia and to Europe and Africa. This avian flu infected many millions of birds and some people in South East Asia and Turkey who had been in close contact with infected chickens. Mortality in these cases was high. In 2009, influenza A subtype H1N1 caused a pandemic of swine flu which started in Mexico and spread to many other countries. Most patients with swine flu had only mild illness, but a minority developed severe infection, and some died.

Human-to-human spread of H1N1 or H5N1 flu is rare at the time of writing, but another pandemic could develop if the virus mutates again.

## Spread

Like SARS (see ➲ Severe acute respiratory syndrome, p. 259), flu is spread by droplets coughed or sneezed into the air, or by direct contact with hands contaminated with the virus.

## Features

Consider the possibility of avian flu or swine flu in a patient with fever of ≥38°C and cough or breathlessness, who, in the last 7 days, has been in an area affected by H1N1 or H5N1 influenza. Laboratory staff and health care workers in contact with cases of severe unexplained respiratory illness could also be at risk.

## Management

Isolate the patient and treat with precautions against transmission of the virus, as for SARS (see ➲ Severe acute respiratory syndrome, p. 259). Antiviral treatment with oseltamivir or zanamivir may be considered, depending on current guidelines.

Clinical guidelines about the assessment of suspected cases and the management of influenza patients are updated as the situation changes and if another pandemic develops.

## Chapter 6

# Environmental emergencies

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# Hypothermia: presentation

## Definitions

Hypothermia is defined as a core T° of <35°C.

*Hypothermia may be classified as in Table 6.1.*

**Table 6.1** Classification of hypothermia

| | |
|---|---|
| Mild hypothermia | 32–35°C |
| Moderate hypothermia | 30–32°C |
| Severe hypothermia | <30°C |

## Background

Hypothermia in young adults often reflects environmental exposure (eg hill-walking or cold water immersion) or immobility and incapacity from alcohol and/or drugs. The elderly more typically become hypothermic indoors; common factors include unsatisfactory housing, poverty, immobility, lack of cold awareness (autonomic neuropathy, dementia), drugs (sedatives, antidepressants), alcohol, acute confusion, hypothyroidism, and infection.

## Clinical features

Severe hypothermia can mimic death. As core T° ↓, cerebral and cardiovascular function deteriorate. At 32–35°C, apathy, amnesia, ataxia, and dysarthria are common. At <32°C, consciousness falls progressively, leading to coma, ↓ BP, arrhythmias (check pulse for at least 1min before diagnosing cardiac arrest), respiratory depression, and muscular rigidity. Shivering is an unreliable sign. VF may occur spontaneously when T° falls <28°C and may be provoked by limb movement or invasive procedures (especially in the presence of hypoxia).

## Diagnosis

Check tympanic T° (or rectal T° with an electronic probe or low-reading thermometer). Tympanic and rectal T° may lag behind core (cardiac) T° during rewarming. Oesophageal T° reflects core levels more accurately but requires special equipment.

## Investigations

- U&E, FBC, toxicology (including alcohol level), and clotting screens. *Note*: hypothermia can cause or aggravate coagulation disturbances.
- Blood glucose (BMG reading may be falsely ↓).
- Amylase or lipase (↑ levels are common but do not necessarily imply pancreatitis).
- Blood cultures and ABG.
- ECG: look for prolongation of elements in the PQRST complex, J waves (also called Osborn waves), and arrhythmias (AF and bradycardias are the most common) (see Fig. 6.1).
- CXR: look for pneumonia, aspiration, and LVF. Consider other X-rays after rewarming (eg suspected fractured hip).
- Consider CT scan if underlying head injury or stroke is suspected.

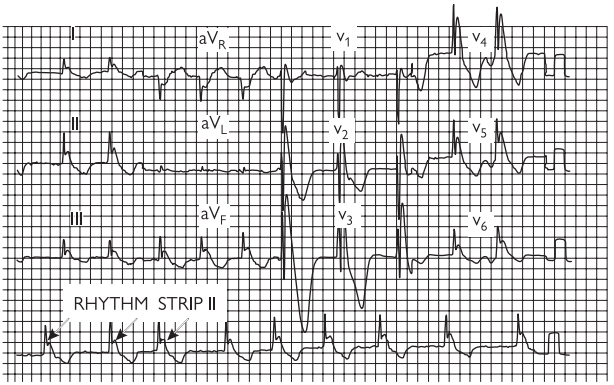


**Fig. 6.1** ECG in hypothermia.

*Notes on the ECG shown in Fig. 6.1*

- Rhythm disturbance: AF with slow ventricular response.
- Prolongation of QRS.
- Delayed repolarization 'J waves' (arrowed).
- ST–T wave abnormalities.

# Hypothermia: management

## Principles

- Treat in a warm room (>21°C), remove wet clothes, and dry the skin.
- Handle the patient gently and place on a cardiac monitor.
- Give warmed, humidified $O_2$ by mask.
- Intubation, if needed, should be preceded by oxygenation.
- Secure IV access. IV fluid is rarely required unless there is loss of volume from another cause. If BP ↓ during rewarming, give 300–500mL of warmed 0.9% saline or colloid. In unstable patients, consider CVP and urinary catheter. Warm IV fluid administration is an inefficient rewarming method and runs the risk of fluid overload and precipitating arrhythmias.
- Correct hypoglycaemia with IV glucose.
- In cardiac arrest, give CPR at standard rates. Note that the heart may be unresponsive to defibrillation, pacing, and drug therapy. Drug metabolism is ↓ and unpredictable—avoid drugs until core T° >30°C.
- Defibrillation is appropriate at normal energy levels if VF/VT occurs. If three shocks are unsuccessful, defer further shocks until core T° >30°C.

## Rewarming methods

Choice depends on the severity and duration of the condition, available facilities, and the individual patient.

*Passive rewarming* Easy, non-invasive, and suitable for mild cases (T° >32°C). ↓ evaporative and conductive losses by wrapping in warm blankets (remember to cover the back and sides of the head) ± polythene sheets. Avoid space blankets which are noisy and have no advantages over polythene sheets. Endogenous metabolism and shivering usually generate enough heat to allow spontaneous rewarming. Aim for a rate of 0.5–2°C/hr, but do not rewarm the elderly with prolonged hypothermia too rapidly (>0.6°C/hr), as hypotension or cerebral/pulmonary oedema may develop.

*Active rewarming* A water bath at ~41°C is rapid and useful for immersion hypothermia but cannot be used in injured patients or if CPR is required. Airway care, ventilation, and monitoring are difficult, and hazards include core T° afterdrop and BP ↓ due to peripheral vasodilatation. Hot water bottles and heat pads are less efficient and can cause burns. A hot air blanket is more convenient than a water bath, provides some heat, and reduces heat loss.

*Core rewarming*

- Airway warming with heated (40–45°C) humidified $O_2$ provides some additional heat and reduces heat loss. It can be combined with other rewarming methods. It may reduce the risk of cardiac arrhythmias.
- Consider peritoneal lavage—run in saline at 45°C via a catheter; leave for 10–20min, then replace with a fresh warm supply. The fluid directly heats the liver and blood in the IVC. Other options are warm irrigation of the pleural cavity, stomach, or bladder.

Extracorporeal rewarming with cardiopulmonary bypass maintains brain and organ perfusion and, if available, is the method of choice in patients with severe hypothermia or cardiac arrest. Cardiopulmonary bypass can result in rapid rewarming, with core T° ↑ at 1−2°C/5min.

# Frostbite and non-freezing cold injury

## Frostbite

Frostbite[1] occurs when tissues freeze at sub-zero temperatures. Predisposing factors include inadequate clothing/footwear, hypothermia, exhaustion, alcohol (which impairs judgement), drugs (eg β-blockers), peripheral vascular disease, smoking, and previous cold injury. Frostbite usually involves extremities, especially the fingers, toes, nose, and ears.

*Frostnip* May precede frostbite. The skin of the nose, face, or fingers goes white and numb but recovers rapidly on protection from the cold, with transient paraesthesiae, but no tissue loss and no permanent damage.

*Superficial frostbite* Involves the skin and subcutaneous tissues. The frozen area is numb and looks white and waxy. Tissues feel firm or hard but are still pliable. Rewarming is painful. Oedematous hyperaemic skin becomes mottled or purple, with serum-filled blisters. A hard black eschar forms, and after ~3 weeks, this separates, revealing sensitive red, shiny skin.

*Deep frostbite* Involves muscles, nerves, and sometimes bone, as well as the skin and superficial tissues. The damaged area is hard and remains grey or white after rewarming. Blood-filled blisters develop. The dead tissue mummifies and then separates after several weeks or months.

*Treatment of frostbite* Varies with the situation and facilities. Only frostnip should be treated in the field. Frostbitten tissues need rewarming as soon as possible, but further damage from refreezing needs to be avoided. Treat hypothermia before frostbite. Rewarm frostbitten limbs in water at 37–39°C until skin circulation returns (usually ~30min). Give analgesia and ibuprofen (which inhibits prostaglandins). After rewarming, let the area dry in warm air (do not towel dry). Elevate the limb. Expose the area, with a bed cradle to avoid pressure of bedclothes. Clean the area daily in a whirlpool bath, and encourage movement. If necessary, split eschar to relieve stiffness, but avoid surgical debridement and amputations and allow the eschar to separate spontaneously; premature surgery causes avoidable tissue loss. Expert advice is helpful in severe frostbite—the British Mountaineering Council (https://www.thebmc.co.uk) has a frostbite advice service. Bone scans or MRI/magnetic angiography (MRA) may help to define deep tissue injury. In severe frostbite, early thrombolysis with tPA may reduce the risk of eventual amputations.

## Non-freezing cold injury[1]

*Trench foot (immersion foot)* Caused by prolonged immersion in cold water or wet boots at temperatures just above freezing. Vasoconstriction causes tissue ischaemia and nerve damage. The feet are initially cold, numb, and pale or mottled. On rewarming, they become red, swollen, and very painful. Blisters may develop.

*Treatment* Keep the feet clean, warm, dry, and elevated to reduce oedema.

*Outcome* Most patients recover fully, but some have continued pain, paraesthesiae, and sensitivity to cold.

1 *State of Alaska Cold Injury Guidelines, 2014*. Available at: http://dhss.alaska.gov/dph/Emergency/Documents/ems

# Drowning and near drowning

## Definitions

*Drowning* Death by suffocation from submersion in any liquid. Drowning is a common cause of death in young people; 40% of drownings occur in children aged <4y.

*Near drowning* Survival (at least temporarily). In adults, the most common predisposing factor is alcohol, sometimes with other drugs. A significant proportion reflect attempted suicide. In the UK, marine near drowning is usually associated with hypothermia (see ➲ Hypothermia: presentation, pp. 264–5).

## Pathophysiology

*Wet drowning* Involves significant aspiration of fluid into the lungs. This causes pulmonary vasoconstriction and hypertension with V/Q mismatch, aggravated by surfactant destruction and washout, ↓ lung compliance, and atelectasis. Acute respiratory failure is common. ABG shows hypoxia, hypercarbia, and mixed respiratory/metabolic acidosis. The onset of symptoms can occur rapidly, but in lesser insults, symptoms may be delayed.

*Contamination* Water contaminated with chemical waste, detergents, etc. may induce further lung injury.

*Electrolytes* Irrespective of whether aspirated water is salt water, fresh water, or swimming pool water, changes in serum electrolytes and blood volume are similar and rarely immediately life-threatening.

*Gastric fluid* Swallowing of fluid into the stomach, with gastric dilatation, vomiting, and aspiration, is common.

*Dry drowning* In ~10–20% of deaths from drowning, a small amount of water entering the larynx causes persistent laryngospasm, which results in asphyxia and an immediate outpouring of thick mucus, froth, and foam, but without significant aspiration—this is 'dry drowning'.

*Secondary drowning* Deterioration in a previously apparently well patient, following successful resuscitation after submersion. It may occur in 5–10% of initial survivors.

## The mammalian diving reflex

This is probably seen only in young children but may explain why successful resuscitation without neurological deficit can occur after prolonged immersion. Cold water stimulates facial nerve afferents, whilst hypoxia stimulates the carotid body chemoreceptors. These effects reflexively ↓ the heart rate and vasoconstrict skin, GI tract, and skeletal muscle vessels, redistributing blood to the brain and heart. Associated hypothermia results in ↓ metabolic demands, delaying cerebral hypoxia.

## Management

- Consider associated injury (eg to the cervical spine from diving into a shallow pool or surfing), and treat appropriately.
- Maintain the airway. Remove regurgitated fluid/debris by suction of the upper airway. Ensure adequate ventilation and correction of hypoxia. If the patient does not have a gag reflex or is apnoeic, ventilate with a bag and mask and proceed to early tracheal intubation and IPPV. In spontaneously breathing patients, give the highest $FiO_2$ possible. IPPV will be required if hypoxia and/or hypercapnia are present despite $O_2$ therapy or if there are signs of pulmonary oedema. Ventilation with positive end-expiratory pressure (PEEP) may significantly improve oxygenation by ↑ functional residual capacity, improving V/Q mismatch and enhancing fluid resorption from the pulmonary bed. However, PEEP may ↓ venous return to the heart.
- If the patient is in cardiac arrest, commence CPR (see ➲ Cardiac arrest, p. 48). Defibrillation may not be successful until core T° is >30°C (see ➲ Hypothermia: management, pp. 264–5). Appropriate rapid core rewarming techniques are required.
- Remove all wet/cold clothing.
- Monitor core T° and start rewarming (see ➲ Hypothermia: management, pp. 264–5).
- NG tube to relieve gastric dilatation.
- Check U&E, blood glucose, ABG/VBG, FBC, and ECG.
- Obtain CXR if symptomatic.
- Consider the possibility of alcohol, illegal drugs, or drug overdose. Keep urine and blood samples and test if appropriate, eg paracetamol.
- Do not use 'prophylactic' steroids or barbiturates.
- Antibiotics may be warranted if contaminated water (eg sewage) is involved (see ➲ Leptospirosis (Weil's disease), p. 249).
- Inhalation of mud/sand, etc. may require bronchoscopy for clearance.

## Outcome

Resuscitation without cerebral deficit is possible after prolonged submersion (even after >60min), particularly if associated with hypothermia. 50% of children recovered apparently lifeless will survive, and even adults with a GCS of 3–4 out of 15 and fixed dilated pupils can survive unimpaired.

Respiratory effort is a sensitive prognostic sign, but in hypothermic patients, its absence does not necessarily imply poor outcome. Note the time to the first spontaneous inspiratory gasp.

*Poor prognostic factors* Include extremes of age, severe acidosis, immersion for >5min, and coma on admission.

*Good prognostic factors* Include patients who are alert on admission, hypothermia, older children/adults, brief submersion time, and those who receive rapid on-scene BLS and respond to initial resuscitation measures.

*Asymptomatic patients* Those who have no abnormality on repeated clinical examination, ABG, and CXR require observation for at least 4–6hr prior to considering discharge. Admit all others to ICU or a general ward as appropriate.

# Diving emergencies: 1

Consider any symptom developing within 48hr of a dive as related to the dive until proven otherwise. On suspicion of a diving-related episode, seek specialist advice urgently (see Table 6.2).

Diving-related emergencies fall into four main categories: drowning (see ➲ Drowning and near drowning, pp. 268–9), barotrauma, decompression illness, and marine bites or stings (see ➲ Specific bites and stings, pp. 422–3).

## Barotrauma

May occur in any gas-containing body cavity during descent or ascent.

Descent barotrauma ('squeeze') results from compression of gas in enclosed spaces as the ambient pressure ↑. Commonly, the ears, sinuses, and skin are affected. Middle ear squeeze may be precipitated by Eustachian tube congestion and leads to erythema, haemorrhage, or tympanic membrane perforation with conductive hearing loss. Round or oval window rupture (inner ear squeeze) occurs with sudden pressure changes between the middle and inner ear and results in acute tinnitus, vertigo and deafness, and a perilymphatic fistula. ENT opinion is urgently required if a perilymphatic fistula is suspected and for cases of severe or continuing symptoms. If tympanic membrane rupture has not occurred, middle ear squeeze can usually be managed with decongestants/simple analgesics. If it has ruptured, give antibiotics (see ➲ Traumatic tympanic membrane rupture, p. 567). Instruct the patient not to dive until the symptoms have resolved and the drum has completely healed.

Sinus barotrauma has a similar aetiology to middle ear injury and is often associated with upper respiratory tract infection (URTI), mucosal polyps, and sinusitis. Treat similarly to ear barotrauma.

Divers who fail to exhale periodically via the nose into their face mask during descent may develop 'face mask squeeze' (skin barotrauma). Erythema, bruising, and petechial and conjunctival haemorrhages develop in the enclosed area. Skin tightly enclosed by parts of the diving suit can have similar appearances. Usually no treatment is required.

*Ascent barotrauma* is the reverse of squeeze and particularly affects the lungs. It may be caused by breath-holding during rapid uncontrolled ascent or by air trapping in patients with asthma or congenital lung bullae. Mediastinal emphysema is the most common event and presents with ↑ hoarseness, neck swelling, and retrosternal chest discomfort. Symptoms usually resolve spontaneously with high concentrations of $O_2$. Pneumothorax is a potentially life-threatening complication if it develops during the dive, as intrapleural gas cannot be vented and ↑ ascent will precipitate tension. Conventional treatment by needle decompression, aspiration, or chest drain insertion (see ➲ Chest drain insertion, p. 346) is required.

Dental pain may occur on ascent or descent barotrauma in carious teeth or those which have had recent fillings. The affected tooth is tender on tapping. Treat symptomatically with analgesics and arrange dental referral.

**Table 6.2** Sources of advice on diving emergencies and hyperbaric chambers

| | |
|---|---|
| *England, Wales, Northern Ireland* | |
| Diving Incident Telephone Advice Line, Institute of Naval Medicine, Gosport, Hampshire | Telephone 07831 151523 (24hr)<br>Ask for the Duty Diving Medical Officer |
| Diving Diseases Research Centre, Plymouth<br>http://www.ddrc.org | Telephone 01752 209999 (24hr)<br>Ask for the Duty Diving Doctor |
| *Scotland* | |
| Hyperbaric Medicine Unit, Aberdeen Royal Infirmary | Telephone 0345 408 6008<br>State 'diving emergency'. Give your name and telephone number<br>Ask for the Duty Hyperbaric Doctor |
| In the event of any difficulties in contacting these agencies in the UK, telephone 999 and ask for COASTGUARD | |
| *Other countries* | |
| Divers Alert Network (DAN)<br>https://www.diversalertnetwork.org | DAN Diving Emergency Hotline (USA) +1 919 684 9111 |
| This has links to diving emergency contact numbers throughout the world | |

## Further information

British Hyperbaric Association. Available at: http://www.hyperbaric.org.uk
Divers Alert Network. Available at: https://www.diversalertnetwork.org
Diving Diseases Research Centre (Hyperbaric Medical Centre, Plymouth, England). Available at: https://www.ddrc.org
Scottish Diving Medicine. Available at: https://www.sdm.scot.nhs.uk

# Diving emergencies: 2

## Decompression illness

There are two forms of decompression illness. The first occurs when dissolved nitrogen in blood and tissues is not expelled at a sufficient rate to prevent bubble formation. The second occurs when air bubbles are released into the circulation because of pulmonary barotrauma. This follows if air bubbles enter the pulmonary capillaries from ruptured alveoli. The bubbles travel via the left side of the heart to the systemic circulation. Cerebral air embolism usually causes symptoms as the diver surfaces, with loss of consciousness, fits, cardiovascular collapse, and chest pain. Clinically, differentiation between the two forms is difficult and initial management is the same. In general, the sooner the onset of symptoms, the greater the likely severity. Symptoms may be attributed by the patient (and the unwary doctor) to musculoskeletal sprains/strains or other minor injury.

Decompression illness is more likely in divers who have not followed safe ascent recommendations, the obese, in cold water, and when excessive exercise has occurred during the dive. It may be precipitated by air travel if insufficient time is left between diving and flying for residual nitrogen to leave the body in a controlled fashion. Bubbles have direct mechanical and local inflammatory effects, commonly involving the joints, skin, CNS, lungs, and ears.

Joint pain, 'the bends', most often affects the shoulders and elbows. A dull aching sensation, ↑ by movement, but without localized tenderness, is common. Pruritic rashes, local swelling, and a peau d'orange effect may occur. Back pain, limb weakness, sensory abnormalities, or urinary retention imply spinal cord involvement. Central effects include focal deficits, cerebellar disturbance, and mood changes.

*Treatment for decompression illness* Is recompression. If delayed, the risks of permanent damage to the brain and spinal cord greatly ↑. The diagnosis of decompression sickness may only follow the response to recompression. Pending this, give the highest possible concentration of $O_2$. Analgesics and sedatives can mask recompression responses and should only be used on specialist advice. Entonox® is absolutely contraindicated.

If intubation is required, inflate the ET tube cuff with sterile water, since during recompression, an air-filled cuff will deflate. IV fluids (0.9% saline or a plasma expander) assist oxygenation of ischaemic tissues and facilitate discharge of excess tissue nitrogen load into the venous system by ensuring adequate circulating volume. Some centres may recommend aspirin and/or dextran solutions to ↓ capillary sludging which accompanies severe decompression sickness.

Despite dry or wet suits, hypothermia is common. Treat with appropriate passive or active rewarming (see ➲ Hypothermia: management, p. 266).

*Air evacuation* If, after consultation with the diving medical centre, air evacuation is necessary, unpressurized aircraft should not fly above 300m. The diver should breathe 100% $O_2$. On reaching the diving centre, recompression to a simulated depth of 18m with 100% $O_2$ occurs, interspersed with periods of air breathing to ↓ $O_2$ toxicity risk. Slow decompression then follows standard treatment protocols.

Divers usually dive in pairs. If a diver has symptoms of decompression sickness or pulmonary barotrauma, his 'buddy' will be at risk also. Although recompression may not be required in the buddy, transfer him/her, along with the affected diver and their diving equipment, to the recompression facility.

*Obtain the following information before referral, if possible*

- The patient's current condition, progression since onset, and response to treatment.
- Time of onset of symptoms related to the dive.
- Dive profile and history (depth, duration, activity during the dive, speed of ascent including details of any stoppages, environmental conditions (water temperature, currents, etc.), pre-dive exercise, alcohol, drugs and food, type and condition of diving equipment used, clothing worn, other recent dives). Many divers store much of this information in a dive computer.
- Previous medical history, previous diving-related episodes, and drug history.

# Heat illness

Body T° is normally kept at 36–38°C by the hypothalamus. Hyperthermia occurs when homeostatic mechanisms are overwhelmed by factors acting individually or (commonly) together. This can occur even in temperate climates. At-risk groups include the young and the elderly in conditions of ↑ T° and humidity, patients with unaccustomed or prolonged muscular activity (eg at 'raves', associated with ecstasy or other drugs), grand mal fitting, athletes, marathon runners, and armed forces recruits.

## Predisposing medical factors

- Alcohol use or withdrawal (including delirium tremens).
- Cardiac disease.
- Any condition which may cause or aggravate $Na^+$/water ($H_2O$) loss (eg gastroenteritis, cystic fibrosis).
- Drugs, including: alcohol, diuretics, salicylates, anticholinergics (antihistamines, tricyclic antidepressants), sympathomimetics (amphetamines, ecstasy, LSD, cocaine, phencyclidine, appetite suppressants), phenothiazines, antipsychotics, MAOIs, and SSRIs (see Serotonin syndrome, p. 224).

## Heat illness has a spectrum of severity

Heat cramps ⇔ Heat exhaustion ⇔ Heat stroke.

In *heat cramps/exhaustion*, homeostatic mechanisms still function but are overwhelmed.

In *heat stroke*, all thermoregulatory control is lost and body T° ↑ rapidly to very high levels (>41°C), causing widespread severe tissue and organ damage. Mortality ~10%.

## Heat cramps

Core T° of 37–39°C. Mental function is normal. Sweating during exercise and replacement with hypotonic fluid lead to $Na^+$ deficiency. Brief cramps occur in muscles used in heavy work, usually after exertion.

## Heat exhaustion

Core T° <40°C. Mental function is normal. Characterized by mixed $Na^+$/$H_2O$ depletion. Sweating and tachycardia are usually present. Symptoms of weakness, fatigue, headache, vertigo, nausea and vomiting, postural dizziness, and syncope. Patients will recover with rest and fluids.

*In mild cases*, remove from heat and use simple cooling techniques. Rehydrate with oral electrolyte solutions.

*More severe cases* require IV 0.9% saline or 0.45% saline/5% glucose. Use clinical assessment, U&E, and Hct to guide infusion rate. Up to 4L of fluid may be required over 6–12hr. Avoid over-rapid infusion which may cause pulmonary and/or cerebral oedema.

## Measurement of core temperature

Tympanic or rectal T° measurement is appropriate in the ED but may underestimate core T° and respond slowly as this changes. Oesophageal and intravascular probes give the most accurate readings of core T° but require special equipment.

## Heat stroke

Suspect in collapse during or after exercise and in high-risk groups. Core T° is >41°C (but significant cooling can occur before arrival in the ED). Outcome depends upon the height and duration of ↑ T°.

- *CNS*: oedema + petechial haemorrhages cause focal/generalized damage.
- *Muscle injury*: releases enzymes, myoglobin, urate, $K^+$, $PO_4^-$.
- *Liver*: jaundice commonly develops after 24hr.
- *Kidneys*: acute renal failure (ARF) from hypovolaemia, muscle breakdown products, acidosis, and DIC.
- *Blood*: DIC, thrombocytopenia, leucocytosis.
- *Metabolic*: ↑ or ↓ $K^+$, metabolic acidosis, respiratory alkalosis, hypoglycaemia.

*Features*

Sweating may be present, but the skin surface may feel deceptively cool due to peripheral vasoconstriction.

- *CNS*: confusion, delirium, fitting, coma, oculogyric crisis, dilated pupils, tremor, muscle rigidity, decerebrate posturing, cerebellar dysfunction.
- *CVS*: tachycardia, hypotension, arrhythmias.
- *Coagulopathy*: purpura, conjunctival haemorrhages, melaena, haematuria.

*Investigations*

- ABG, U&E, BMG, CK, clotting screen, LFTs, urate, $Ca^{2+}$, $PO_4^-$, ECG, CXR.

*Treatment*

- Treat immediately and involve ICU staff. Remove all clothing.
- Secure the airway (intubation/IPPV if needed). Give $O_2$ as needed.
- Cooling techniques depend upon facilities available and the clinical state of the patient. Do not give 'antipyretics' such as aspirin/paracetamol. Evaporative cooling is the most efficient and applicable treatment. Spray the naked patient with tepid tap water and blow air with fans. Ice-packs can be applied to the axillae, groins, neck, and scalp (but avoid prolonged contact). Consider cold gastric or peritoneal lavage, or cardiopulmonary bypass if these techniques fail. Aim to cool ≥0.1°C/min. Stop active cooling when core T° is <39°C.
- *IV fluids*: give 50mL of 10% glucose IV if BMG is <3mmol/L. Severe hypovolaemia is uncommon, but if hypotension persists despite ↓ T°, give IV 0.9% saline (1–1.5L over 1–2hr). Avoid overloading the circulation with a risk of pulmonary/cerebral oedema. CVP monitoring may be needed. CVP may be initially ↑ due to peripheral vasoconstriction.
- Insert a urinary catheter. If myoglobinuria is present, aim for ↑ urine output and consider giving IV bicarbonate and/or mannitol.
- If fits occur, give IV lorazepam—but beware of respiratory depression.

*Neuroleptic malignant syndrome* An idiosyncratic reaction in patients on anti-psychotics (especially haloperidol, thioridazine, chlorpromazine). Features are muscle rigidity, extrapyramidal signs, autonomic dysfunction, and severe dyskinesia. Stop the antipsychotic, cool the patient, and give dantrolene.

*Malignant hyperpyrexia* A rare autosomal dominant condition related to use of suxamethonium and volatile anaesthetics. Dantrolene prevents $Ca^{2+}$ release from skeletal muscle and is very effective—the initial dose is 2–3mg/kg IV, then give 1mg/kg as needed (max total dose 10mg/kg) (see https://www.aagbi.org).

# Electrical injuries

Electric shocks can cause cardiac and respiratory arrest. The heart often restarts spontaneously, but respiratory arrest may be prolonged, causing fatal hypoxia. The electric current may produce burns and muscle damage. Spasms from a shock may result in dislocations or fractures or precipitate a fall causing major trauma. Fatal electrocution can occur from domestic electricity (in the UK, 230V, alternating current at 50 cycles/s), but severe injury is more common with high-voltage shocks (>1000V).

*Lightning* causes a DC shock at a very high voltage (up to 100,000,000V), but with a short duration (0.1–1ms).

## Electrical flash and arc burns

An electrical short-circuit near to a person may cause sudden vaporization of metal and deposit a thin layer of hot metal on the skin, without any electricity passing through the casualty. Electrical flash burns may look dramatic because of skin discoloration but are often superficial and heal well. In contrast, electrical arcing produces high temperatures and may cause deep dermal or full-thickness burns, especially if clothing is set alight.

## Contact burns

If electricity has passed through the patient, there are usually two or more entry or exit wounds, comprising full-thickness burns with white or charred edges. Tissue damage is more extensive than the visible burns, especially with high-voltage injuries. Deeper layers of skeletal muscle may be involved and muscle damage can cause myoglobinuria and renal failure. Myonecrosis and oedema of muscles may produce compartment syndrome (see ➲ Crush syndrome, pp. 406–7).

If current passes through the torso (especially from arm to arm), cardiac arrhythmias are more likely than if only a single limb is involved. Myocardial damage may occur, often in association with vascular injuries.

*Neurological effects* of electric shocks include coma, fits, headaches, transient paralysis, peripheral neuropathy, and mood disturbances.

*Ophthalmic injuries* are common after electrical burns of the head. Cataracts and glaucoma may develop later.

*Electrocution in pregnancy* carries risks to the fetus (spontaneous abortion may occur). Obtain obstetric advice.

## Lightning

Sudden vaporization of sweat and rainwater caused by lightning may explode clothes and shoes off the victim and rupture ear drums. Lightning burns are superficial, often with a characteristic feathered or fern-like appearance (Lichtenberg figures). The limbs are often cold and mottled due to arterial spasm, which usually resolves over a few hours. Deep muscle damage and myoglobinuria are rare. Coma may result from direct brain injury, head injury due to a fall, or cardiac arrest. CPR, if indicated, may be successful, even if required for prolonged periods. Survivors may be confused and amnesic for several days and may have fits and temporary paralysis. Cataracts are common.

### Management

- At the scene, make sure that the current is turned off before anyone approaches or touches the casualty. Remember that high-voltage electricity can arc through the air or pass through the ground.
- Check the airway, breathing, and circulation. Electrical burns of the mouth and throat may cause oedema and airway obstruction.
- Perform CPR as necessary, remembering that a good outcome may follow prolonged resuscitation. Minimize movement of the spine in case there has been spinal trauma.
- Examine thoroughly for head, chest, abdominal, and skeletal injuries.
- Examine all over for skin entry/exit burns, and check pulses and sensation.
- Check the ECG—there may be arrhythmias (eg AF), conduction defects, ST elevation, and T wave changes—if present, place on a cardiac monitor.
- Test the urine for blood (except in an asymptomatic young healthy person who has suffered a domestic shock, when it is unnecessary). If the stick test is +ve for blood, but there are no RBCs on microscopy, treat for myoglobinuria to prevent AKI—obtain specialist advice; maintain a high urine output, and consider using mannitol ± isotonic sodium bicarbonate.
- Except in minor low-voltage (domestic) electrical injury with no associated worrying features (such as burns, ECG abnormalities, hypotension, reduced conscious level), check FBC, U&E, and CK.
- If there is a reduced conscious level or there are focal neurological abnormalities, request a CT brain scan.
- Significant electrical injuries may cause fluid loss into muscle, resulting in hypovolaemia—if there are significant burns or soft tissue damage, treat with IV fluid (start with 1000mL of 0.9% saline).
- High-voltage injuries with associated burns and tissue damage may require widespread fasciotomies—involve surgical experts to help with excision or amputation of non-viable tissues and inspection and further debridement after 48hr.

### Admission or discharge

Allow home asymptomatic patients with domestic and minor low-voltage burns who have a normal ECG, no history suggestive of arrhythmia (eg palpitations), no pre-existing history of cardiac problems, no significant skin burns, and no myoglobinuria, but advise review if any problem develops. Note that late-onset arrhythmias are very unlikely.

Admit children who bite electric flexes for observation, because of the risk of delayed bleeding from labial blood vessels.

Admit for observation (± treatment) all patients with high-voltage conduction injuries (including lightning) and those with cardiac arrhythmias, chest pain or ECG abnormalities, vascular injury, significant skin burns, or myoglobinuria.

# Radiation incidents

In the UK, 24hr advice and assistance is available via NAIR (National Arrangements for Incidents involving Radioactivity) by telephone (0800 834153) or via the police. Try to distinguish between external irradiation of a person and contamination with radioactive material. Someone exposed to X-rays or to gamma rays in a radiation sterilizing unit receives no further radiation after removal from the source, and there is no risk of contaminating anyone else. However, a person contaminated with radioactive material is still exposed to radiation and needs urgent, careful decontamination to minimize the risks to himself and other people. Some hospitals are officially designated for the care of casualties contaminated with radioactive substances, but in an emergency, a patient may be taken to any ED where a plan for such events should exist.

## Anticipation of a radiation incident

- Inform the ED consultant immediately if a patient from a radiation incident arrives or is expected.
- Get advice and help from a radiation physicist (from the medical physics or radiotherapy department).
- Implement the appropriate Radiation Incident Plan to deal with the patient.
- Expect media enquiries.

## Treatment of contaminated casualties

Where possible, treatment should take place in a designated decontamination room. This room should have a separate entrance, ventilation arrangements, decontamination facilities with a shower, and contaminated water collection facilities. Cover the floor of this room and entrance/exit corridors with disposable sheeting. All staff must themselves be decontaminated and checked before leaving this area.

- Turn off air conditioning.
- Pregnant and potentially pregnant staff should not be involved.
- Provide any necessary life-saving treatment, but avoid spreading contamination.
- 'Barrier nurse', as for an infectious disease.
- Assume patients are contaminated until they have been checked by the radiation physicist.
- Instruct patients and staff not to eat, drink, or smoke.
- Involve a minimum number of staff who should wear face masks, theatre clothing with impermeable gowns or plastic aprons, two pairs of gloves, and overshoes or rubber boots.
- Restrict and record the movements of people in and out of the room.
- Ensure that the ambulance crew waits for monitoring of themselves and their vehicle.
- Keep everything that may be contaminated for radiation testing.
- Collect the patient's clothes, dressings, swabs, and any equipment used in plastic bags, and keep them in the decontamination room.
- All blood/urine samples must be specially labelled and the laboratories informed of the radiation risk.
- Life-threatening injuries may take precedence over all of the above, such that patients may need to be managed in the resuscitation room.

## Decontamination of the patient

The radiation physicist should determine the sites of contamination and monitor the effectiveness of treatment. The object is to remove any contaminating substance and minimize absorption into the body, especially via the mouth, nose, and wounds.

- Cover any wounds prior to decontamination.
- Avoid splashing.
- Radioactive material can usually be removed from intact skin by washing with soap and water. Gentle scrubbing may be needed, but it is important to avoid damaging the skin. Carefully clean wounds and irrigate with water or saline.
- Clean the mouth using a mouthwash and a soft toothbrush, with care to avoid swallowing any fluid.
- Instruct the patient to blow their nose into paper handkerchiefs. If the nose is still contaminated, irrigate it with small amounts of water.
- Irrigate each eye from the medial side outwards to avoid draining contaminated water into the nasolacrimal duct.
- Clean the hair by washing with shampoo and by clipping if contamination persists, but do not shave the scalp.
- If monitoring shows that all contamination has been removed, treat the patient as for an irradiated, but uncontaminated, patient. However, if contamination persists or if radioactive material has been ingested or inhaled, further treatment will be needed after discussion with a radiation specialist.
- Check all staff involved in treating the patient for radioactive contamination before they leave the treatment area.

## The irradiated patient

A patient who has been irradiated or contaminated with radiation may be at risk of radiation sickness or other ill effects. Admit to a designated unit for assessment and follow-up by a radiotherapist or other specialist.

*Initial symptoms of radiation sickness* are malaise, nausea, vomiting, and diarrhoea, starting a few hours after exposure. There is then a latent period before the main effects of radiation sickness appear. Record any symptoms and the time of onset. The effects of anxiety and stress may be similar to the early features of radiation sickness.

*Take blood* for FBC, U&E, and blood group, recording the time on the blood tubes and in the notes. Measurement of the lymphocyte count and analysis of chromosomes at known times after exposure are helpful in assessing the amount of radiation received and determining the prognosis. A low ($<1.0 \times 10^9$/L) or falling lymphocyte count indicates serious radiation exposure.

## Further information

National Operational Guidance. https://www.ukfrs.com

## Chapter 7

# Analgesia and anaesthesia

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# Approach to pain

## Pain hurts!

Many patients who present to the ED are in pain. Ascertaining the site and characteristics of this pain is often very important in diagnosing the underlying problem. Relief of pain is an essential and urgent part of treatment and is usually the initial top priority for patients. Pain and distress may prevent patients from giving important details of the history and may prevent them from co-operating fully with investigations or treatment.

## Standard assessment of pain

The traditional way to assess the severity of pain is to ask patients to grade the severity of their pain on a scale of 1 to 10. This simple linear system is useful in that it is easily understood by patients, who can convey the extent to which analgesia has worked by reporting changing scores over time. Establishing a pain score may work for most adults and some older children, but is not appropriate for younger children, adults with dementia, and patients with learning difficulties.

### *Assessment of pain in children*

The numerical pain scale may be replaced by a 'faces scale' or 'pain ladder' (eg 'No pain at all'—'Stinging'—'Quite bad'—'Very bad'—'Worst ever'). Look for verbal and non-verbal clues that a child is in pain—formal scales are available such as the Alder Hey Triage Pain Score.

### *Assessment of pain in patients with dementia*

The combination of impaired cognition and communication can make it difficult to establish if an older patient is in pain. Always consider this and remember to ask patients directly if they are experiencing any pain or discomfort. Look for signs of distress and/or agitation which are out of character with usual behaviour.

## More pain than expected

If an injury or illness appears to be more painful than would be expected, consider if there are complications and/or reconsider the diagnosis. Examples in the context of trauma are:

- Severe pain despite immobilization of a fracture may be due to a vascular injury, compartment syndrome (see ➲ Crush syndrome, pp. 406–7), or a tight plaster (see ➲ Casts and their problems, pp. 430–1).
- Severe pain in patients with limb trauma where no injury is identified on X-ray may be due to missed fracture/dislocation (eg lunate dislocation, Lisfranc fracture/dislocation).
- Consider the possibility of infection (eg necrotizing fasciitis—see ➲ Necrotizing fasciitis, p. 244) or vascular compromise.
- Reflex sympathetic dystrophy (Sudeck's atrophy) may also cause severe pain starting a few days after relatively minor trauma.
- In the context of chest pain, consider aortic dissection (see ➲ Aortic dissection, pp. 96–7), and in abdominal pain, consider mesenteric infarction/ischaemia if the degree of pain is out of proportion to the physical signs.

# Options for relieving pain

## Analgesics

Before prescribing any drug, check what treatment has been taken at home and/or given prehospital. Consider interactions and allergies.

## Splintage

Immobilizing fractures helps to relieve pain and ↓ analgesic requirements. Entonox® (see ➲ Analgesics: Entonox® and ketamine, p. 287) may help whilst a splint or cast is being applied.

## Cold

Cool burns as soon as possible, usually by running under cold water, to ↓ pain and stop continuing thermal injury. Chemical burns from hydrofluoric acid (see ➲ Chemical burns, p. 405) are often extremely painful and need prolonged cooling in iced water. Pain from recent sprains and muscle injuries may be ↓ by cooling with ice-packs (or a pack of frozen peas) applied for 10–15min at a time, with towelling between the ice-pack and the skin.

## Heat

Pain following sprains and strains of the neck, back, and limbs is often caused by muscle spasm. Symptomatic relief may be provided by heat from a hot bath, hot water bottle, or heat lamp.

## Elevation

Many limb injuries produce considerable swelling, which causes pain and stiffness. Elevate the limb to ↓ swelling, which will help to relieve the pain and allow mobilization as soon as possible.

## Dressings

Pain from minor burns and fingertip injuries often resolves after a suitable dressing is applied.

## Local anaesthesia

Local anaesthesia (LA) provides excellent pain relief for fractured shaft of femur in the form of a femoral nerve or fascia iliaca compartment block (see ➲ Femoral nerve block p. 313; ➲ Shaft of femur fractures, pp. 486–7). Similarly, digital nerve and other blocks are useful for some finger and hand injuries (see ➲ Local anaesthetic nerve blocks, p. 302). Sometimes it can help to give analgesia in the form of LA before obtaining X-rays. Always check for a nerve injury (and document this) before injecting LA. Consider using a small bleb of LA SC before taking ABG.

## Definitive treatment

Reducing a pulled elbow or trephining a subungual haematoma usually gives immediate relief of pain, so no analgesia is needed.

## Psychological aspects of pain relief

Anxiety and distress accompany pain and worsen suffering. Psychological support is needed, as well as physical relief from pain. Patients are helped by caring staff who explain what is happening and provide support and re-assurance. The presence of family and/or a friend can help.

# Analgesics: aspirin and paracetamol

## Aspirin

Good for headaches, musculoskeletal pain, and dysmenorrhoea. It has antipyretic and mild anti-inflammatory actions. It interacts with some anticonvulsants and may exacerbate asthma and cause gastric irritation. Aspirin ↑ the risk of bleeding in patients on anticoagulants.

- Do not use aspirin in children <16y or during breastfeeding.
- Adult dose for analgesia is PO 300–900mg 4- to 6-hourly (max 4g daily).

## Paracetamol ('acetaminophen' in the USA)

Paracetamol has similar analgesic and antipyretic actions to aspirin and causes less gastric irritation, but has no anti-inflammatory effects.

The therapeutic dose range of paracetamol in children and adults is 10–15mg/kg. Weigh and calculate the dose for adults and children who are small for age. In most patients, the dose can be based safely on age.

- Adult dose is 0.5–1g PO 4- to 6-hourly (max 4g in 24hr), but note some patients can get hepatotoxicity at normal doses—↓ dose if at risk (eg weight <50kg, chronic alcohol consumption, chronic malnutrition). IVI paracetamol is useful in certain circumstances—give 1g IVI over 15min in adults >50kg. ↓ dose to 15mg/kg if the adult weighs <50kg.
- For children aged <6y, use PO paracetamol infant suspension (120mg/5mL):
  - 1–2 months: 30–60mg every 8hr as required, max 60mg/kg/day.
  - 3–5 months: 60mg every 4–6hr, max four doses per day.
  - 6 months to 1y: 120mg every 4–6hr, max four doses per day.
  - 2–3y: 180mg every 4–6hr, max four doses per day.
  - 4–5y: 240mg every 4–6hr, max four doses per day.
- For children aged ≥6y, use paracetamol six plus suspension (250mg/5mL) every 4–6h, max four doses per day:
  - 6–7y: 240–250mg.
  - 8–9y: 360–375mg.
  - 10–11y: 480–500mg.
  - 12–15y: 480–750mg.
  - 16–17y: 0.5–1g.
- Paracetamol may be repeated 4- to 6-hourly. Adults and children aged ≥3 months may have a maximum of four doses in 24hr.
- Overdosage can cause liver and renal damage (see ➲ Paracetamol poisoning, pp. 198–201).

## Compound analgesics (paracetamol + opioid)

Tablets containing paracetamol and low doses of opioids are widely used but have little benefit over paracetamol alone and cause more side effects, such as constipation and dizziness, particularly in the elderly. These compound preparations include:

- *Co-codamol 8/500* (codeine phosphate 8mg, paracetamol 500mg).
- *Co-dydramol* (dihydrocodeine tartrate 10mg, paracetamol 500mg).

Compound preparations of paracetamol and full doses of opioids, such as co-codamol 30/500 (codeine phosphate 30mg, paracetamol 500mg), are more potent but cause opioid side effects, including nausea, vomiting, constipation, dizziness, drowsiness, and respiratory depression.

# Analgesics: NSAIDs

## Non-steroidal anti-inflammatory drugs

NSAIDs are often used to treat musculoskeletal pain, with or without inflammation, although paracetamol is usually tried first. NSAIDs can cause gastric irritation, diarrhoea, GI bleeding, and perforation, with an ↑ risk at higher drug dosage and in patients aged >60y and those with a history of peptic ulcer. NSAIDs may exacerbate asthma and can precipitate AKI in patients with heart failure, cirrhosis, or renal insufficiency. Interactions occur with diuretics, anticoagulants, lithium, and other drugs (see *BNF*). Advise NSAIDs be taken after food to ↓ the risk of GI side effects. Avoid giving NSAIDs to patients who are already taking aspirin. Note there is evidence that NSAIDs impair healing after injury, so tend to be avoided by those involved in professional sport.

Many NSAIDs are available and all can cause serious adverse effects. Ibuprofen has the lowest incidence of side effects and may be bought without prescription. It is useful in children as an analgesic and an antipyretic.

- *Ibuprofen dosage in adults*: 200–400mg tds.
- *Doses for children* according to age are:
  - 1–2 months: 5mg/kg tds to qds.
  - 3–5 months: 50mg tds, max 30mg/kg/day.
  - 6–11 months: 50mg tds, max 30mg/kg/day.
  - 1–3y: 100mg tds, max 30mg/kg/day.
  - 4–6y: 150mg tds, max 30mg/kg/day.
  - 7–9y: 200mg tds, max 30mg/kg/day.
  - 10–11y: 300mg tds, max 30mg/kg/day.
  - 12–17y: 200–400mg tds.

Naproxen is more effective than ibuprofen but has fewer side effects than diclofenac:

- *Naproxen*: 500mg initially, then 250mg 6- to 8-hourly (max 1.25g daily).
- *Acute gout*: 750mg initially, then 250mg 8-hourly until pain resolves.

Diclofenac is similar in efficacy to naproxen but has more side effects:

- *Diclofenac* (PO or PR): 75–150mg daily in 2–3 divided doses.

## Injectable NSAIDs

Some NSAIDs may be given by injection for musculoskeletal pain (eg for ureteric colic). The contraindications and side effects are the same as for oral treatment. IM injections are painful and can cause sterile abscesses, so PO or PR treatment is preferable.

- *Ketorolac* may be given IM or slowly IV (initial dose 10mg over at least 15s—see *BNF*). It is useful as an adjunct for manipulations under anaesthesia (MUAs).
- *Diclofenac* must be given by deep IM injection (not IV, which causes venous thrombosis). Dose: 75mg, repeated if necessary after 30min (max 150mg in 24hr).

## Topical NSAIDs

NSAID gels or creams applied to painful areas provide some analgesia but are less effective than oral treatment. Systemic absorption can occur and cause adverse effects as for oral NSAIDs.

# Analgesics: morphine

The standard analgesic for severe pain is morphine. It often causes nausea and vomiting in adults, so consider giving an antiemetic (cyclizine 50mg IV/IM or prochlorperazine 12.5mg IM) with it. Antiemetics are not usually necessary in children aged <10y.

Other side effects of opioids include drowsiness and constipation. Respiratory depression and hypotension may occur, especially with large doses. Pinpoint pupils can complicate neurological assessment. Naloxone (see ➲ Opioid poisoning, p. 196) reverses the effects of opioids.

## IV morphine

In acute conditions, give morphine by slow IV injection, which provides rapid, but controlled, analgesia. The dose varies with the patient and the degree of pain. Titrate the dose depending on the response—2mg may be enough for a frail elderly person, but sometimes >20mg is needed in a young fit person with severe injuries. Dilute morphine with 0.9% saline to 1mg/mL (label the syringe clearly) and give it slowly IV (1–2mg/min in adults) in 1mg increments until pain is relieved. Give further analgesia if pain recurs. The dose of IV morphine in children is 100–200mcg/kg, given in increments, repeated as necessary. Patient-controlled analgesia using a computerized syringe pump is very good for post-operative analgesia, but rarely appropriate initially in the ED.

## IM morphine

Provides slower and less controlled effects than IV analgesia—avoid its use, especially in shocked patients.

## Oral morphine

Morphine may be given PO (eg as *Oramorph*®  oral solution) but is not usually a first-line choice when a patient presents in pain to the ED.

### *Smooth muscle spasm due to opioids*

In a (very) few patients, opioids such as morphine can cause severe pain due to smooth muscle spasm, especially spasm of the sphincter of Oddi. About 5–20min after morphine has been given, severe colicky abdominal pain develops. This may be typical of biliary colic but can mimic renal colic, intestinal perforation, or MI.

Pain from spasm of the sphincter of Oddi may be relieved by glucagon (1mg IV, repeated if necessary), although this is liable to cause vomiting. Naloxone (0.2mg IV, repeated if necessary) is also effective but may reverse the desired analgesia. GTN is another option.

# Analgesics: Entonox® and ketamine

## Entonox®

Entonox® is a mixture of 50% nitrous oxide ($N_2O$) and 50% $O_2$. It is stored as a compressed gas in blue cylinders with a blue and white shoulder. It is unsuitable for use at <–6°C, since the gases separate and a hypoxic mixture could be given. Entonox® diffuses more rapidly than nitrogen and so is *contraindicated* with the following: undrained pneumothorax (since it may produce a tension pneumothorax), after diving (↑ risk of decompression sickness), facial injury, base of skull fracture, intestinal obstruction, and ↓ conscious level.

Entonox® is controlled by a demand valve and inhaled through a mask or mouthpiece, often held by the patient. It gives rapid and effective analgesia and is widely used in prehospital care. In the ED, Entonox® is useful for initial analgesia, eg whilst splinting limb injuries, and for many minor procedures such as reduction of a dislocated patella or finger. Tell the patient to breathe deeply through the mask or mouthpiece, and warn that they may feel drowsy or drunk but that this will wear off within a few minutes.

## Ketamine

This dissociative anaesthetic drug may be given IM or IV by experts and provides strong analgesia in sub-anaesthetic dosage. There is reluctance to use it in adult hospital practice, because it can cause hallucinations, but these are less of a problem in children. It is useful for sedating children for procedures such as minor wound suturing. Ketamine is useful in prehospital care, especially to help extrication or emergency amputation.

*Airway-protective reflexes* are maintained better with ketamine than with other induction agents, but airway obstruction and aspiration of gastric contents are still potential hazards. Respiratory depression is uncommon at normal dosage. Ketamine is a bronchodilator and may be used in asthmatics. It stimulates the cardiovascular system and often causes tachycardia and hypertension, so avoid it in severe hypertension. Hallucinations are less likely if a small dose of midazolam is given and the patient is not disturbed during recovery from anaesthesia.

Ketamine is available in *three strengths*: 10, 50, and 100mg/mL, which are easily confused. The IV dose for GA is 1–2mg/kg over 1min, which is effective after 2–7min and provides surgical anaesthesia for 5–10min. The IM dose for GA is 10mg/kg, which is effective after 4–15min and gives surgical anaesthesia for 12–25min. Further doses (10–20mg IV or 20–50mg IM) can be given if major limb movements or ↑ muscle tone prevent extrication of the patient.

For sedation of children undergoing suturing or other minor procedures, ketamine may be given IM (2.5mg/kg) or IV (1mg/kg over at least 1min). With this dose of ketamine, LA is needed for cleaning and suturing of wounds, but little physical restraint should be needed to allow the procedure to take place. Occasionally, a second dose of ketamine (1mg/kg IM or 0.5mg/kg IV) is required to achieve adequate sedation. Larger initial doses provide deeper sedation but are more likely to cause side effects (eg vomiting or agitation) during recovery. With low doses of ketamine, agitation is unlikely and there is no need to add midazolam.

# Analgesics: other opioids

### Codeine

This is a weak opioid which is used PO for moderate pain (30–60mg 4-hourly, max 240mg daily) and has side effects similar to those of morphine. Codeine may also be given IM (but not IV, because it can cause hypotension). Note that codeine is a pro-drug, so its efficacy is variable.

### Dihydrocodeine

This is very similar to codeine. As with codeine, opioid dependency can occur with prolonged usage.

### Tramadol

Stronger than codeine, tramadol inhibits reuptake of serotonin and noradrenaline, in addition to its opioid action. Its metabolism varies between individuals, and so its analgesic effects can be unpredictable. However, tramadol may be useful for the management of chronic pain in some patients, especially where NSAIDs are not an option. Start with 50mg PO bd, ↑ as necessary to 100mg PO qds.

### Diamorphine

Otherwise known by the street name 'heroin', diamorphine has similar effects to morphine but is more soluble and so can be dissolved in a very small volume of diluent. Nasal diamorphine provides effective analgesia in children (see ➲ Nasal diamorphine for analgesia in children, p. 291).

### Fentanyl

This short-acting opioid is particularly useful for patients undergoing brief procedures in the ED such as manipulation of fractures or dislocations. The dose of fentanyl is 0.5mcg/kg slow IV, repeated as required. The rapid onset (and offset) is an advantage, but depending upon the dose used, there is an ↑ risk of inducing apnoea when compared with morphine.

### Pethidine

Providing rapid, but brief, analgesia, pethidine is less potent than morphine. It is associated with a number of problems and has fallen out of favour and routine use.

# Penthrox®

Methoxyflurane (Penthrox®) is very useful for managing pain after trauma in adults, especially during manipulations of dislocations and fractures. It is easy to use and works quickly after inhalation. It does not usually require any specific additional monitoring. One or two 3mL doses may be given in 24h (eg for shoulder dislocation, one whilst getting X-rays, another whilst being relocated). Do not use it in patients with known renal impairment or liver disease or if there is a history of serious reaction to inhaled anaesthetics.

# Analgesia for trauma

## Multiple injuries

Entonox® may be useful for analgesia during transport and initial resuscitation but only allows administration of 50% $O_2$ and is contraindicated if there is an undrained pneumothorax. As soon as practicable, use other forms of analgesia, such as IV morphine (see ➲ Analgesics: morphine, p. 286) and/or nerve blocks (see ➲ Local anaesthetic nerve blocks, p. 302), and splintage of fractures to ↓ pain and blood loss.

## Head injury

Relief of pain is particularly important in head-injured patients, since pain and restlessness ↑ ICP, which can exacerbate secondary brain injury. Aim to treat headache following a head injury with paracetamol, an NSAID, or codeine (which causes less central depression than stronger opioids such as morphine). If headache is severe or ↑, arrange a CT scan to look for an intracranial haematoma. Try to avoid strong opioids, because of concern about sedation and respiratory depression, but if pain is severe, give morphine in small IV increments—the effects can be reversed, if necessary, with naloxone. Femoral nerve block (or fascia iliaca compartment block) is particularly useful in a patient with a head injury and a fractured femur, since it ↓ the need for opioids.

*Small children* with minor head injuries often deny having headaches but look and feel much better if given paracetamol (see ➲ Analgesics: aspirin and paracetamol, p. 284). Give further doses, if necessary, over the following 12–24hr.

## Chest injury

Chest injuries are often extremely painful. Good analgesia is essential to relieve distress, enable deep breaths to be taken, and ↓ risk of complications such as pneumonia and respiratory failure. Avoid Entonox® if a pneumothorax is a possibility, until this has been excluded or drained. Give high-concentration $O_2$, as necessary, and check $SpO_2$ and ABG. Give morphine in slow IV increments (see ➲ Analgesics: morphine, p. 286) and monitor for respiratory problems. Intercostal nerve blocks (see ➲ Intercostal nerve block, p. 303) provide good analgesia for fractured ribs but may cause a pneumothorax and so are only used in patients being admitted. In severe chest injuries, get anaesthetic or ICU help—thoracic epidural anaesthesia can sometimes avoid the need for IPPV. Before a thoracic epidural is performed, check X-rays/CT of the thoracic spine for fractures. Many patients who are admitted with chest injuries benefit from patient-controlled analgesia.

# Analgesia in specific situations

## Children

Injured children are distressed by pain and fear. Enlist parental support. Explanation and reassurance are important, but give analgesia as needed.

*Start oral analgesia* with paracetamol (see ➲ Analgesics: aspirin and paracetamol, p. 284), but if this is inadequate, add ibuprofen (see ➲ Analgesics: NSAIDs, p. 285), dihydrocodeine elixir, or morphine sulfate oral solution (eg Oramorph®):

- *Ibuprofen*—see ➲ Analgesics: NSAIDs, p. 285 for dose.
- *Dihydrocodeine elixir dose*: 0.5–1mg/kg PO 4- to 6-hourly.
- Children in severe pain may benefit from PO morphine
  - 1–2 months: 50–100mcg/kg.
  - 3–5 months: 100–150mcg/kg.
  - 6–11 months: 200mcg/kg.
  - 1–11y: 200–300mcg/kg.
  - 12–17y: 5–10mg.

*Entonox®* (see ➲ Analgesics: Entonox® and ketamine, p. 287) gives rapid analgesia without the need for an injection.

*IV morphine* is appropriate in severe injuries, but beware sedation.

*Femoral nerve block* (see ➲ Femoral nerve block, p. 313) provides good analgesia for femoral fractures and is usually well tolerated.

*Digital nerve block* with bupivacaine (see ➲ Digital nerve block, pp. 304–5) is useful for painful finger injuries, especially crush injuries. Consider before X-ray—when the child returns from X-ray, the finger may then be treated painlessly.

*IM morphine* could be used to provide analgesia for small burns or fractured arms, but PO morphine or nasal diamorphine are preferable, since IM injections are painful and unpleasant.

*Nasal diamorphine* is playing an ↑ role in the provision of pain relief in children (see Table 7.1).

## Acute abdominal pain

It is cruel and unnecessary to withhold analgesia from patients with acute abdominal pain. Adequate analgesia allows the patient to give a clearer history and often facilitates examination and diagnosis—tenderness and rigidity become more localized, and masses more readily palpable. Good X-rays cannot be obtained if the patient is distressed and restless because of renal colic or a perforated ulcer.

*Morphine* by slow IV injection (see ➲ Analgesics: morphine, p. 286) is appropriate in severe pain, unless this is due to renal or biliary colic, in which an NSAID (see ➲ Analgesics: NSAIDs, p. 285) may be preferred. Morphine occasionally causes severe abdominal pain due to smooth muscle spasm of the sphincter of Oddi (see ➲ Smooth muscle spasm due to opioid analgesics, p. 286).

## Toothache

Toothache or pain after dental extractions can often be eased by aspirin, an NSAID, or paracetamol. Do not give opioids such as codeine or dihydrocodeine, which may make the pain worse. Drainage of a dental abscess may be required to relieve toothache.

## Nasal diamorphine for analgesia in children

In the UK, diamorphine is licensed for use IV, IM, SC, and PO. Nasal diamorphine is an effective and acceptable method of analgesia for children with limb fractures or small burns who do not need immediate venous access. It should be given as soon as possible, prior to X-rays.

*Contraindications* Age <1y (or weight <10kg), nasal obstruction or injury, basal skull fracture, opioid sensitivity.

Verbal consent for nasal diamorphine should be obtained from the child's parents (and the child, if appropriate). Follow local protocols and see https://bnfc.nice.org.uk/drug/diamorphine-hydrochloride.html

One method delivering nasal diamorphine is described as follows:

The dose of nasal diamorphine is 0.1mg/kg, given in a syringe in a volume of 0.2mL. The child is weighed. The appropriate concentration of solution for the weight of child is achieved by adding a suitable volume of 0.9% saline to a 10mg ampoule of diamorphine.

**Table 7.1** Dosage of nasal diamorphine in children

| Weight (kg) | Volume of saline (mL) | Dose of diamorphine (mg) in 0.2mL |
|---|---|---|
| 10 | 2.0 | 1.0 |
| 15 | 1.3 | 1.5 |
| 20 | 1.0 | 2.0 |
| 25 | 0.8 | 2.5 |
| 30 | 0.7 | 2.9 |
| 35 | 0.6 | 3.3 |
| 40 | 0.5 | 4.0 |
| 50 | 0.4 | 5.0 |
| 60 | 0.3 | 6.7 |

A volume of 0.2mL of this solution is drawn up into a syringe and given in one or both nostrils, whilst the child's head is tilted backwards. Turn the head to each side, maintaining each position for several seconds. A small syringe can be used to drip the solution into the nose, but, if possible, use an aerosol device (eg MAD®), allowing for the dead space of the device (0.1mL for MAD®, so draw up 0.3mL). Record the time of administration. Monitor the conscious level for 20min. Respiratory depression is unlikely, but resuscitation facilities and naloxone must be available. Nasal diamorphine provides rapid analgesia which lasts up to 4hr.

Fentanyl may also be used nasally (initial dose 2mcg/kg) as an alternative to nasal diamorphine.

# Local anaesthesia

## Indications for local anaesthesia in the ED

LA is indicated in any situation in which it will provide satisfactory analgesia or safe and adequate conditions for operations or procedures. These include the following:

- *Insertion of venous cannulae* (0.1mL of 1% lidocaine SC 30s prior to cannulation ↓ the pain of cannulation without affecting the success rate).
- *Obtaining ABG*.
- *Cleaning, exploration, and suturing* of many wounds.
- *Analgesia for some fractures*, eg shaft of femur.
- *Minor operations/procedures*, eg manipulation of some fractures and dislocations, insertion of chest drain, drainage of paronychia, removal of corneal FB.

## Contraindications to local anaesthetic

- *Refusal or poor patient co-operation*.
- *Allergy to LA*: severe allergic reactions to LA are rare, but anaphylaxis can occur. If allergy to LA is alleged, obtain full details of the circumstances and the drug involved and check with a senior before giving any LA. It may be possible to use a different drug. Some reactions are caused by the preservative in multi-dose vials, rather than the drug itself, so single-dose ampoules may not cause a problem. Some alleged 'allergies' are actually toxic effects due to overdosage, or faints due to fear and pain.
- *Infection at the proposed injection site*: injection into an inflamed area is painful and could spread infection. High tissue acidity from inflammation ↓ the effectiveness of LA drugs. Hyperaemia causes rapid removal of the drug, and so a short duration of action and an ↑ risk of toxicity. LA nerve block at a site away from the infected area can provide good anaesthesia (eg digital nerve block for paronychia or nerve blocks at the ankle for an abscess on the sole of the foot).
- *Bleeding disorder*: anticoagulant therapy and thrombocytopenia are contraindications for nerve blocks in which there is a risk of inadvertent arterial puncture (eg femoral nerve block and fascia iliaca block).

## Special cautions (increased risk of toxicity)

- Small children.
- Elderly or debilitated.
- Heart block.
- Low cardiac output.
- Epilepsy.
- Myasthenia gravis.
- Hepatic impairment.
- Porphyria.
- Anti-arrhythmic or β-blocker therapy (risk of myocardial depression).
- Cimetidine therapy (inhibits metabolism of lidocaine).

### Lidocaine (previously known as 'lignocaine')

Lidocaine is the LA used most often for local infiltration and nerve blocks. It is available in 0.5%, 1%, and 2% solutions, either 'plain' (without adrenaline) or with adrenaline 1:200,000. For routine use, the most suitable choice is 1% plain lidocaine.

- *Duration of action*: lidocaine starts to work within a few minutes—the effects last from 30 to 60min (for plain lidocaine) to 90min (lidocaine with adrenaline). The duration of action varies with the dosage and local circulation.
- *For plain lidocaine*: the maximum dose is 200mg (20mL of 1% solution) in a healthy adult or 3mg/kg in a child.
- *For lidocaine with adrenaline*: the maximum dose is 500mg (50mL of 1% solution) in a healthy adult or 7mg/kg in a child.

These are the maximum total doses for one or more injections of LA given together for local infiltration or nerve block (with care to avoid intravascular injection). Reduce the dose in debilitated or elderly patients, or if there is a particular risk of toxicity (see ➲ Local anaesthetic toxicity, p. 294).

Lidocaine can also be used for anaesthesia of the skin (with prilocaine in EMLA® cream—see ➲ Topical anaesthesia, p. 298), urethra, and cornea, and also as a spray for anaesthetizing mucous membranes in the mouth and throat.

### Bupivacaine

Bupivacaine is particularly useful for nerve blocks since it has a long duration of action (3–8hr), although its onset of anaesthesia is slower than that of lidocaine. It may also be used for local infiltration, but not for Bier's block (see ➲ Bier's block, pp. 300–1). Bupivacaine is available in concentrations of 0.25% and 0.5%, with or without adrenaline—the usual choice is 0.5% bupivacaine without adrenaline. The maximum dose of bupivacaine (with or without adrenaline) for a fit adult is 150mg (30mL of 0.5% or 60mL of 0.25%) and for a child 2mg/kg.

*Note*: levobupivacaine is similar but has a longer duration of action.

### Prilocaine

Prilocaine has a similar duration of action to lidocaine. It can be used for local infiltration or nerve blocks but is particularly useful for Bier's block (see ➲ Bier's block, pp. 300–1). High doses (usually >600mg) may cause methaemoglobinaemia. The maximum dose of prilocaine for a healthy adult is 400mg (40mL of 1% solution) and for a child 6mg/kg.

### Tetracaine (amethocaine)

Tetracaine is used for topical LA of the cornea (see ➲ LA drops to aid examination, p. 551) and skin (see ➲ Topical anaesthesia, p. 298).

### Proxymetacaine

Proxymetacaine is also used for topical LA of the cornea. It causes less initial stinging than tetracaine and so is particularly useful in children.

# Local anaesthetic toxicity

## Toxic effects

These result from overdosage of LA or inadvertent intravascular injection. The first symptoms and signs are usually neurological, with numbness of the mouth and tongue, slurring of speech, light-headedness, tinnitus, confusion, and drowsiness. Muscle twitching, convulsions, and coma can occur.

Cardiovascular toxicity may initially result in tachycardia and hypertension, but later there is hypotension with bradycardia and heart block. Ventricular arrhythmias and cardiac arrest occur occasionally, especially with bupivacaine.

## Early signs of toxicity

These may be detected if the doctor maintains a conversation with the patient whilst injecting the LA. Toxic effects may start immediately if an intravascular injection is given. However, peak blood levels usually occur ~10–25min after injection—so if a relatively large dose has been given, do not leave the patient alone whilst anaesthesia develops.

Occasionally, patients initially agree to LA but become 'hysterical' or faint when an injection is given. In such circumstances, it may be difficult to distinguish immediately between the effects of anxiety and those of drug toxicity.

## Management of LA toxicity

- Stop the procedure.
- Call for help.
- Clear and maintain the airway.
- Give 100% $O_2$ and ensure adequate lung ventilation.
- Obtain reliable IV access. If possible, take blood for U&E, FBC, and LFTs.
- Monitor ECG. Record pulse, BP, RR, and conscious level.
- If convulsions occur, ensure adequate oxygenation and give lorazepam (adult dose 2–4mg slowly IV; child 100mcg/kg, max 4mg) or diazepam (adult 5–10mg slowly IV; child 100mcg/kg).
- Treat hypotension by raising the foot of the trolley. If systolic BP remains <90mmHg in an adult, give IV fluids (eg 500mL of 0.9% saline). In a child, give 20mL/kg if systolic BP is <70mmHg.
- Bradycardia usually resolves without treatment. If bradycardia and hypotension persist, give atropine and consider IV lipid emulsion. Dobutamine, isoprenaline, or temporary pacing are potential options if bradycardia is associated with hypotension.
- In cardiac arrest due to LA toxicity, give lipid emulsion using Intralipid® 20% 1.5mL/kg IV over 1min (bolus of 100mL for a 70kg patient), then 15mL/kg/hr (500mL over 30min for a 70kg patient). Continue CPR. If circulation is still inadequate, repeat IV bolus of Intralipid® twice at 5min intervals, then IVI 30mL/kg/hr (500mL over 15min). The maximum total dose of 20% lipid emulsion is 12mL/kg. Note that propofol is not an alternative to lipid emulsion.

(See ➲ Lipid emulsion (Intralipid®) therapy for drug toxicity, p. 195 and https://anaesthetists.org/Home/Resources-publications/Guidelines/Management-of-severe-local-anaesthetic-toxicity)

# Adrenaline (epinephrine) in LA

Most LAs cause vasodilatation, so adrenaline is sometimes added as a vasoconstrictor. This ↓ blood loss, ↑ duration of anaesthesia, and ↓ toxicity by delaying absorption of the LA. Lidocaine with adrenaline is often useful in scalp wounds, in which bleeding can be profuse, but the bleeding point is not visible.

*Bupivacaine with adrenaline* is used for intercostal nerve block to ↓ the risk of toxicity from rapid absorption in a relatively vascular area.

*Lidocaine with adrenaline* can be used in some situations (see below for contraindications) if a relatively large volume of LA is needed, since the maximum dose for a healthy adult is 500mg (50mL of 1% solution), compared to 200mg (20mL of 1%) for plain lidocaine. Other possibilities in such circumstances include 0.5% lidocaine, prilocaine (max dose 40mL of 1% solution), or GA.

*The maximum concentration of adrenaline* in LA is 1 in 200,000, except for dental anaesthesia in which 1 in 80,000 may be used. The maximum total dose of adrenaline in a healthy adult is 500mcg.

## Contraindications and cautions

Never use adrenaline for injections in the nose, ears, or penis, nor in Bier's block (see Bier's block, pp. 300–1). Avoid adrenaline for injections in or near flap lacerations, since vasoconstriction could cause ischaemic necrosis. Adrenaline is traditionally regarded as dangerous in digital nerve blocks of fingers and toes, because of the risk of ischaemia, but some hand surgeons have used LA with adrenaline uneventfully to ↓ bleeding and avoid the need for a finger tourniquet. However, use plain LA (without adrenaline) routinely in fingers and toes to avoid concerns about ischaemia.

*Avoid adrenaline in*

- IHD.
- Hypertension.
- Peripheral vascular disease.
- Thyrotoxicosis.
- Phaeochromocytoma.
- Patients on β-blockers.

The *BNF* states that LA with adrenaline appears to be safe in patients on tricyclic antidepressants.

## Storage

Keep ampoules and vials of LA with adrenaline in a locked cupboard, separate from those without adrenaline, so that they are only available by special request and are not used inadvertently or inappropriately.

# General principles of local anaesthesia

Obtain a brief medical history and record drug treatment and allergies. Think about possible contraindications and cautions for LA (see ➲ Local anaesthesia, pp. 292–3). Obtain expert advice if there is any query or concern.

## Consent for local anaesthesia

Explain to the patient what is planned. Verbal consent is adequate for most LA procedures in the ED.

*Written consent is advised*

- If there is a significant risk of a toxic reaction or complication, including procedures needing large doses of LA.
- For Bier's block (see ➲ Bier's block, pp. 300–1).
- For intercostal nerve block (risk of pneumothorax).

## Safety

Ensure that resuscitation equipment and drugs for toxic reactions are readily available. Monitoring and IV access are not needed for routine simple LA but are essential if there is a risk of complications or toxicity. Calculate the maximum dose of LA that could be used (see ➲ Local anaesthesia, pp. 292–3) and think how much might be needed. Before drawing up any LA, check the drug label carefully, especially if adrenaline is contraindicated.

## Giving local anaesthetic

- Lie the patient down in a comfortable position, with the site of injection accessible and supported. Some patients faint if LA is injected whilst they are sitting up.
- Warm the LA to body T° prior to use.
- Wash hands, use gloves, and clean the skin.
- Use a fine needle, if possible. Before inserting the needle, warn the patient and hold the relevant part firmly to prevent movement.
- Aspirate and check for blood in the syringe before injecting any LA. If the needle moves, aspirate again.
- Inject LA slowly to ↓ pain. Do not use force if there is resistance to injection.
- Maintain a conversation with the patient, to allay anxieties and also to detect any early signs of toxicity (see ➲ Local anaesthetic toxicity, p. 294).

Further details of techniques and precautions are listed in:

- ➲ Topical anaesthesia, p. 298.
- ➲ Local infiltration anaesthesia and ➲ Field block, p. 299.
- ➲ Haematoma block, p. 299.
- ➲ Bier's block, pp. 300–1.
- ➲ Local anaesthetic nerve blocks, p. 302; ➲ Digital nerve block, pp. 304–5; ➲ Median and ulnar nerve blocks, pp. 306–7; ➲ Radial nerve block at the wrist, p. 308 ➲ Nerve blocks of forehead and ear, pp. 310–11; ➲ Dental anaesthesia, p. 309; ➲ Intercostal nerve block, pp. 303; ➲ Femoral nerve block, p. 313; ➲ Nerve blocks at the ankle, pp. 314–15.

## Recording the local anaesthetic

Write clearly in the notes to record the time and site of injection and the type and quantity of LA given.

### Local anaesthesia in children

The general principles are the same as for adults. LA is very useful in children but requires experienced staff. Many children tolerate LA without any problem, but in some, sedation with midazolam (see ➔ Sedation in children, p. 319) or ketamine (see ➔ Ketamine, p. 287) can be helpful.

Weigh the child, if possible, and calculate the maximum dose of LA (see ➔ Lidocaine (previously known as 'lignocaine'), p. 293). In an average-size child, a simple initial estimate of the maximum dose of 1% plain lidocaine is 1mL/y of age (ie 3mL for a 3y old child). If a larger volume may be needed, consider using 0.5% solution or lidocaine with adrenaline (see ➔ Adrenaline (or epinephrine) in local anaesthesia, p. 295), or possibly GA instead.

Prepare everything before bringing the child into the room—rattling equipment and drawing up LA within sight of a child causes unnecessary anxiety. Most parents prefer to stay with their child during the procedure and this is usually helpful. Position the child and parent comfortably. Explain simply and honestly what is going to happen. Have adequate help to keep the child still. Use a small needle, if possible, and inject slowly to minimize pain from the injection.

# Topical anaesthesia

LA applied directly to mucous membranes of the mouth, throat, or urethra will diffuse through and block sensory nerve endings. Development of anaesthesia may take several minutes, and the duration is relatively short because of the good blood supply. Overdosage is relatively easy because most topical preparations contain high concentrations of lidocaine (2% in lidocaine gel, 5% in ointment, and 4% or 10% in lidocaine spray).

*Lidocaine gel* has been used to allow cleaning of gravel burns, but this is not advisable—absorption of lidocaine can easily cause toxicity and the degree of anaesthesia is rarely satisfactory. Scrubbing is often necessary to remove embedded gravel, so proper anaesthesia is essential. Field block may be adequate for a small area, but GA is often necessary for cleaning large or multiple gravel burns, in order to avoid tattooing.

## Topical anaesthesia

### *Lidocaine with prilocaine (eg EMLA®) cream*

Lidocaine 2.5% with prilocaine 2.5% cream (eg EMLA®—'eutectic mixture of local anaesthetics') can usefully ↓ pain of an injection or cannulation. It must only be applied to intact skin (not wounds) and the onset of anaesthesia is slow, usually ~1hr. Apply a thick layer of cream to the skin and cover it with an occlusive dressing, which must be left undisturbed for 1hr.

### *Tetracaine (amethocaine) gel (Ametop®)*

This is similar to EMLA® but acts more quickly (~30–45min) and causes vasodilatation, which aids venous cannulation. Do not use it in wounds because of the risk of rapid absorption and toxicity.

### *Other topical LA agents*

Topical agents such as TAC (tetracaine, adrenaline, and cocaine) or LET (lidocaine, epinephrine, and tetracaine) are sometimes used to provide anaesthesia for wound repair. These preparations can provide effective anaesthesia, but toxic effects may occur from excessive absorption (especially of cocaine).

## Ethyl chloride

Ethyl chloride is a clear fluid which boils at 12.5°C. Spraying the liquid on the skin causes rapid cooling and freezing of the surface. In the past, ethyl chloride was used for incision of paronychias and small abscesses, but it rarely provides adequate anaesthesia and is not recommended. Ethyl chloride is highly inflammable and is a GA, so it must always be handled with care.

# Local anaesthetic administration

## Local infiltration anaesthesia

Local infiltration is the technique used most often in the ED. The LA injected SC in the immediate area of the wound acts within 1–2min. Anaesthesia lasts 30–60min with plain lidocaine or ~90min with lidocaine and adrenaline.

In clean wounds, consider ↓ the pain of injection by inserting the needle through the cut surface of the wound. Do not do this in dirty or old wounds, because of the risk of spreading infection. Less pain is produced by injecting slowly through a thin needle, injecting in a fan-shaped area from a single injection site, and inserting the needle in an area already numbed by an earlier injection. Rapid injection of LA, especially in scalp wounds, can cause spraying of the solution from the tip of the needle or from separation of the needle from the syringe. Slow injection and the use of goggles will ↓ the risk of transmission of infection.

## Field block

This involves infiltration of LA SC around the operative field. Sometimes it is only necessary to block one side of the area, depending on the direction of the nerve supply. Field block can be useful for ragged and dirty wounds and for cleaning gravel abrasions. Check the maximum safe dose before starting a field block. If relatively large volumes of anaesthetic might be needed, consider using 0.5% lidocaine or lidocaine with adrenaline (see ➲ Adrenaline (or epinephrine) in local anaesthesia, p. 295).

## Haematoma block

A Colles' fracture (see ➲ Colles' fracture, pp. 454–5) can be manipulated after infiltration of LA into the fracture haematoma and around the ulnar styloid. This may provide less effective anaesthesia than Bier's block (see ➲ Bier's block, pp. 300–1) and risk a poorer reduction. It also technically converts a closed fracture into an open one, and so there is a theoretical risk of infection, but in practice, this is rarely a consideration if an aseptic technique is employed.

*Contraindications and warnings*

- Fractures >24hr old (since organization of the haematoma prevents spread of the LA).
- Infection of the skin over the fracture.
- Methaemoglobinaemia (avoid prilocaine).

*Drug and dosage* 15mL of 1% plain prilocaine. Lidocaine can be used, but it has a lower margin of safety. Never use solutions containing adrenaline.

*Technique* Use a 20mL syringe and 0.6 × 25mm needle, with full asepsis. Insert the needle into the fracture haematoma and aspirate blood to confirm this—it is usually easiest to do this by angling the needle slightly and approaching the fracture from the dorsum of the wrist, proximal to the fracture. Inject slowly to ↓ pain and the risk of high blood levels and toxicity. Anaesthesia develops in ~5min and lasts 30–60min. Sometimes anaesthesia is inadequate, so an alternative anaesthetic is needed.

# Bier's block

Bier's block (IVRA) is often used to provide anaesthesia for reduction of Colles' fractures or for minor surgery below the elbow. Bier's block uses a large dose of LA, and so there is a risk of a toxic reaction, although this is minimized by correct technique. Undertake a preoperative assessment and checklist, including recording of BP and weight. Obtain written consent for the operation. Ensure that there are at least two trained staff present throughout the procedure, including a practitioner who is competent to deal with severe toxic reactions.

## Contraindications

- Allergy to LA.
- Severe hypertension (>200mmHg systolic) or obesity (cuff unreliable).
- Severe peripheral vascular disease.
- Infection or lymphoedema in the affected arm.
- Raynaud's syndrome or scleroderma.
- Sickle-cell disease or trait.
- Methaemoglobinaemia.
- Children aged <7y (older children may also not be suitable for Bier's block, depending upon the child).
- Unco-operative or confused patient.
- Procedures needed in both arms.
- Surgery that may last >30min.
- Surgery that may need the tourniquet to be released.

Proceed with caution in patients who have epilepsy because of the risk of a fit from LA toxicity.

## Drug and dose

The most suitable drug for Bier's block is prilocaine, from a single-dose vial without preservative. Never use solutions with adrenaline. Do not use lidocaine or bupivacaine, which are more likely than prilocaine to cause toxic effects. The ideal concentration of prilocaine is 0.5%. If only 1% prilocaine is available, dilute it with an equal volume of 0.9% saline to make 0.5% prilocaine.

The dose of prilocaine is 3mg/kg, which is 42mL of 0.5% prilocaine for a 70kg adult, or 30mL of 0.5% prilocaine for a 50kg patient.

## Equipment

- A special tourniquet apparatus is required, including a double cuff with a fail-safe mechanism option to ensure the proximal cuff cannot be deflated until the distal cuff is inflated.
- Check the tourniquet apparatus and cuff regularly.
- Ordinary BP cuffs and sphygmomanometers are not reliable enough and should not be used for Bier's blocks.
- Check that resuscitation equipment and drugs are available (including Intralipid®).
- Ensure that the patient is on a tipping trolley.
- Monitor the ECG, BP, and $SpO_2$ throughout.

### Technique for Bier's block

- Insert a small IV cannula in the dorsum of the hand on the side of the operation (ready for injection of prilocaine) and another IV cannula in the opposite arm (for emergency use, if needed).
- Check the radial pulse. Place the double tourniquet high on the upper arm over padding, but do not inflate it yet.
- Elevate the arm for 3min, whilst pressing over the brachial artery, to try to exsanguinate the limb. (Do not use an Esmarch bandage for this purpose, because of pain.)
- Whilst the arm is elevated, inflate the proximal cuff tourniquet to 300mmHg, or at least 100mmHg above the systolic BP. Lower the arm onto a pillow, and check that the tourniquet is not leaking.
- Record the tourniquet time. Observe the tourniquet pressure constantly during the procedure.
- Slowly inject the correct volume of 0.5% plain prilocaine into the isolated limb, which will become mottled. If the operation is on the hand, squeeze the forearm during injection to direct the LA peripherally. Test for anaesthesia after 5min. If anaesthesia is inadequate, inject 10–15mL of 0.9% saline to flush prilocaine into the arm. Occasionally, no adequate anaesthesia is achieved and GA or IV sedation is needed instead.
- Complete the manipulation or operation. Before applying a POP back slab, remove the cannula from the injured arm.
- Keep the proximal tourniquet cuff inflated for at least 20min and a maximum of 45min. *Note*: if after 20min, there is severe pain due to the cuff and/or a second manipulation is required, use the fail-safe option and inflate the distal cuff over the now anaesthetized skin, then deflate the proximal cuff.
- Obtain a check X-ray whilst the proximal tourniquet cuff is still inflated (in case remanipulation is required).
- If the check X-ray is satisfactory, deflate the tourniquet slowly and record the time. Maintain a conversation with the patient and watch carefully for any sign of toxicity. If any toxic effects occur, re-inflate the tourniquet and give any necessary treatment (see ➲ Local anaesthetic toxicity, p. 294).
- After release of the tourniquet, the arm becomes warm and flushed. Sensation returns after a few minutes.
- Observe the patient carefully for at least 30min after a Bier's block, in case of delayed toxicity. Check the circulation of the limb before the patient is discharged home. Reactive swelling can occur—elevate the limb in a sling and give POP instructions.

(See the RCEM best practice guideline, available at https://www.rcem.ac.uk)

# Local anaesthetic nerve blocks

LA nerve blocks are very useful to enable minor procedures and to provide analgesia.

## Equipment for nerve blocks

Ordinary injection needles can be used for most local blocks in the ED. Anaesthetists sometimes use special pencil-point or short bevel needles when blocking large nerve trunks and plexuses.

## General procedure for nerve blocks

- Follow the general principles of LA (see ➲ Local anaesthesia, pp. 292–3).
- Review the relevant anatomy for the block. Determine the site of injection by feeling for local structures such as arteries or tendons.
- When performing a nerve block, hold the needle with the bevel in the line of the nerve (rather than across it), to reduce the risk of cutting nerve fibres.
- Ask the patient about tingling in the area supplied by the nerve. Do not try to elicit paraesthesiae. If paraesthesiae occurs, withdraw the needle 2–3mm before injecting.
- Wait for the nerve block to work, but do not leave the patient alone during this time. Estimate when a nerve block should be effective and do not test sensation before then. Small nerves may be blocked in 5min, but large nerves may take up to 40min.

## Failed nerve block

If a nerve block does not work, consider waiting longer or giving another injection. Before giving any more LA, review the relevant anatomy; consider using USS guidance, and check that the maximum safe dose of the drug will not be exceeded. Entonox® can be helpful as a supplement to LA for some procedures such as reduction of dislocations. Alternatively, sedation (see ➲ Approach to sedation, pp. 316–17) may be useful, or occasionally GA instead.

## Ultrasound guidance for nerve blocks

USS guidance can help in identifying nerves and other structures and allows visualization of the needle position and the spread of LA. Precise injection of LA adjacent to a nerve gives a faster onset and a longer duration of anaesthesia, with a smaller volume of LA, less pain, and a ↓ risk of complications. USS is unnecessary for some nerve blocks, eg digital nerves and supraorbital nerve block. USS allows nerve blockade away from identifying structures (eg medial nerve block in the forearm).

Successful use of USS for LA requires appropriate equipment, knowledge of the relevant anatomy, and training in USS techniques.

# Intercostal nerve block

Intercostal nerve blocks (see Fig. 7.1) can give useful analgesia for patients with rib fractures who are admitted to hospital. Inserting a block is not routine but requires training and experience. These blocks must not be used in outpatients and should not be performed bilaterally because of the risk of pneumothorax. Patients with obesity or severe obstructive airways disease have ↑ risk of complications. Alternative procedures used in the ICU are interpleural analgesia and thoracic epidurals, but these are not appropriate initially in the ED.

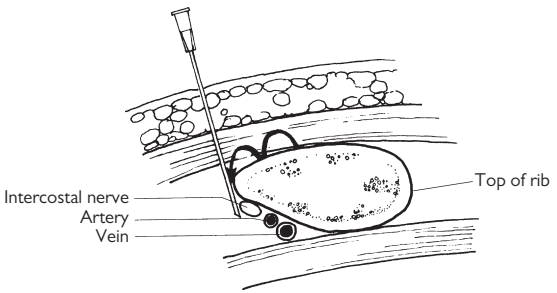



**Fig. 7.1** Intercostal nerve block (note that the neurovascular bundle runs below the rib).
Reproduced from Allman KG *et al*., *Oxford Handbook of Anaesthesia* (2016), with permission from Oxford University Press.

# Digital nerve block

Digital nerve block (see Fig. 7.2) is used frequently for simple procedures on the fingers and toes. (The term 'ring block' is often used but is incorrect since it implies that LA is injected in a ring around the finger, which is unnecessary and might cause ischaemia due to vascular compression.)

A dorsal nerve and a palmar digital nerve run along each side of the finger and thumb. Similarly, there are dorsal and plantar nerves in the toes.

Plain lidocaine (1%) is often used, but bupivacaine (0.5% plain) is preferable because it is less painful on injection and gives prolonged anaesthesia and analgesia. Traditional advice is never to use adrenaline or any other vasoconstrictor. In an adult, use 1–2mL of LA on each side of the finger, thumb, or big toe. Use smaller volumes in the other toes or in children.

## Technique

- Use a 0.6 × 25 mm (23G) needle [0.5 × 16 mm (25G) in small children].
- Insert the needle from the dorsum on the lateral side of the base of the digit, angled slightly inwards towards the midline of the digit, until the needle can be felt under the skin on the flexor aspect.
- Aspirate to check the needle is not in a blood vessel.
- Slowly inject 0.5–1mL. Continue injecting as the needle is withdrawn.
- Repeat on the medial side of the digit.
- If anaesthesia is needed for the nail bed of the great toe, give an additional injection of LA SC across the dorsum of the base of the proximal phalanx, to block the dorsal digital nerves and their branches. This is also required for anaesthesia of the dorsum of the digit proximal to the middle phalanx.

Anaesthesia develops after ~5min. Autonomic nerve fibres are blocked, as well as sensory nerve fibres, so when the block works, the skin feels dry and warm. Occasionally, anaesthesia remains inadequate and another injection is needed. The maximum volume for a finger, thumb, or big toe is 5mL. Use less in the other toes or in children.

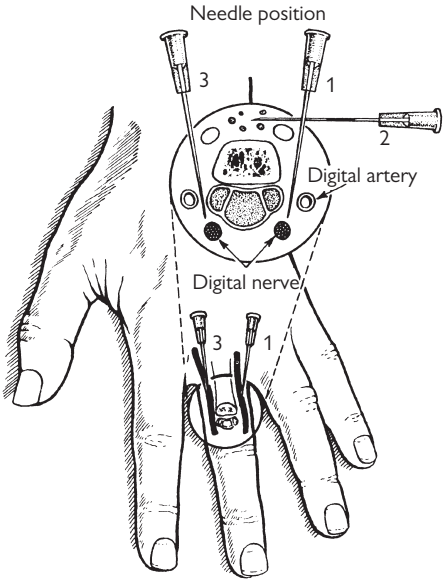
## Single injection digital nerve block

Anaesthesia of the distal phalanx and distal interphalangeal joint can be achieved by a single SC injection on the volar aspect of the base of the finger. Pinch the soft tissues just distal to the proximal skin crease. Insert a 25G needle just beneath the skin at the midpoint of the skin crease, and inject 2–3mL of 0.5% bupivacaine. Massage the LA into the soft tissues.

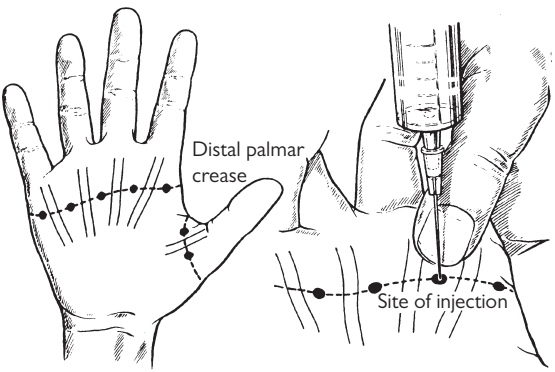
## Digital nerve block at the metacarpal level

Digital nerves can be blocked where they run in the interspaces between the metacarpals. Insert a thin needle in the palm through the distal palmar crease, between the flexor tendons of adjacent fingers. Injection of 3–4mL of 1% plain lidocaine will block the adjacent sides of these two fingers. Anaesthesia develops after 5–10min. Alternatively, a dorsal approach can be used—this is often preferred because it is less painful, but there is an ↑ risk of inadvertent venepuncture and the digital nerves are further from the dorsal surface, so a deep injection is needed.

## Digital nerve block




Digital nerve block at the base of the finger




Digital nerve block at the metacarpal level

**Fig. 7.2** Digital nerve block.

# Median and ulnar nerve blocks

The median nerve supplies sensation to the radial half of the palm, the thumb, the index and middle finger, and the radial side of the ring finger. The ulnar nerve supplies the ulnar side of the hand, the little finger, and the ulnar side of the ring finger. The radial nerve supplies the dorsum of the radial side of the hand. The different nerve distributions overlap. In some people, the radial side of the ring finger and the ulnar side of the middle finger are supplied by the ulnar, rather than the median, nerve. LA block of one or more nerves at the wrist provides good anaesthesia for minor surgery on the hand and fingers (see Fig. 7.3).

## Median nerve block

At the wrist, the median nerve lies under the flexor retinaculum on the anterior aspect of the wrist, under or immediately radial to the tendon of the palmaris longus and 5–10mm medial to the tendon of the flexor carpi radialis. Just proximal to the flexor retinaculum, the median nerve gives off the palmar cutaneous branch, which travels superficially to supply the skin of the thenar eminence and the central palm. Carpal tunnel syndrome is a contraindication to median nerve block.

*Technique*

- Use a 0.6mm (23G) needle and ~5–10mL of 1% lidocaine.
- Ask the patient to flex the wrist slightly and bend the thumb to touch the little finger, in order to identify the palmaris longus.
- Insert the needle vertically at the proximal wrist skin crease, between the palmaris longus and the flexor carpi radialis, angled slightly towards the palmaris longus, to a depth of 1cm. If paraesthesiae occurs, withdraw the needle by 2–3mm.
- Inject ~5mL of LA slowly.
- Block the palmar cutaneous branch by injecting another 1–2mL SC, whilst withdrawing the needle.
- Some people do not have a palmaris longus tendon—in this case, identify the flexor carpi radialis and insert the needle on its ulnar side.
- USS enables blockade of the median nerve in the forearm.

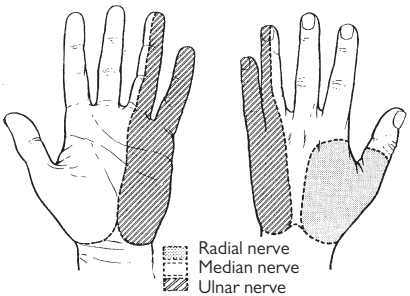
## Ulnar nerve block

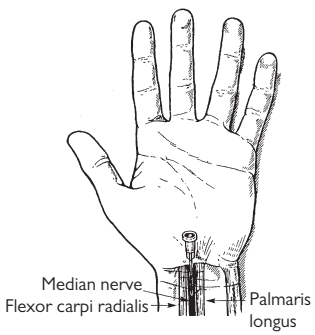
In the distal forearm, the ulnar nerve divides into a palmar branch (which travels with the ulnar artery to supply the hypothenar eminence and the palm) and a dorsal branch (which passes under the flexor carpi ulnaris to supply the ulnar side of the dorsum of the hand).

*Technique*

- Use a 0.6mm (23G) needle and 5–10mL of 1% lidocaine. Avoid adrenaline in peripheral vascular disease.
- Check the radial pulse before blocking the ulnar nerve.
- Feel the ulnar artery and flexor carpi ulnaris tendon, and insert the needle between them at the level of the ulnar styloid process.
- Aspirate and look for blood in the syringe. Withdraw the needle 2–3mm if paraesthesiae occurs.
- Inject 5mL of LA.
- Block the dorsal branch of the ulnar nerve by infiltrating SC 3–5mL of LA from the flexor carpi ulnaris around the ulnar border of the wrist.

## Nerve blocks at the wrist



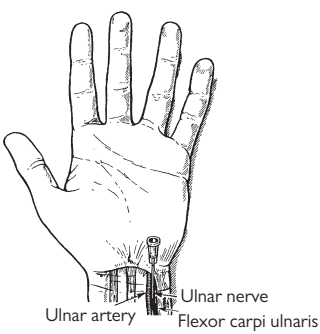




**Fig. 7.3** Nerve blocks at the wrist.

# Radial nerve block at the wrist

## Radial nerve block

In the distal part of the forearm, the radial nerve passes under the tendon of the brachioradialis and lies subcutaneously on the dorsum of the radial side of the wrist where it separates into several branches and supplies the radial side of the dorsum of the hand.

*Technique*

- Use a 0.6mm (23G) needle and 5mL of 1% lidocaine, with or without adrenaline.
- Infiltrate LA SC around the radial side and the dorsum of the wrist from the tendon of the flexor carpi radialis to the radio-ulnar joint. Beware of inadvertent IV injection.

Radial nerve block (see Fig. 7.4) involves an infiltration technique and often has a more rapid onset and a shorter duration of action than median and ulnar nerve blocks. In combined blocks, experts may use lidocaine with adrenaline, in order to prolong the anaesthesia and ↓ the risk of lidocaine toxicity.

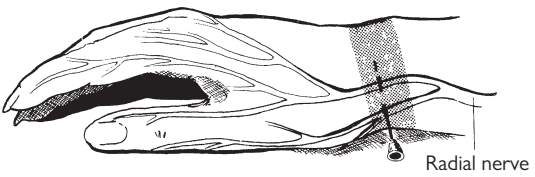



**Fig. 7.4** Radial nerve block at the wrist.

## Other nerve blocks in the arm

*Nerve blocks at the elbow* The median, ulnar, and radial nerves can be blocked at the level of the elbow, but this is rarely necessary. The onset of anaesthesia is slower than with blocks at the wrist.

*Brachial plexus blocks* These should only be used by doctors with anaesthetic training. Brachial plexus blocks can provide good anaesthesia for operations on the arm, but the onset of anaesthesia is often slow (30–45min) and there is a risk of LA toxicity because of the large dose required. The axillary approach can be used in patients who are being discharged later, but if the supraclavicular approach is used, admission to hospital is necessary, because of the risk of a pneumothorax. USS guidance helps to allow accurate positioning of the injection, which improves the effectiveness of the block and ↓ the risk of complications.

# Dental anaesthesia

Intraoral injections of LA are used frequently for dental procedures but can also be extremely useful for cleaning and repair of wounds of the lips, cheeks, and chin. Some basic training/experience in dental techniques is helpful. Give dental LA with dental syringes and cartridges of LA. An appropriate drug for most purposes is lidocaine 2% with adrenaline 1 in 80,000. Some dental syringes do not allow aspiration prior to injection. Disposable dental syringes are preferable to reusable syringes, to ↓ the risk of needlestick injury from re-sheathing of needles.

## Infra-orbital nerve block

The infra-orbital nerve supplies the skin and mucous membrane of the cheek and upper lip, and also the lower eyelid and the side of the nose. The nerve emerges from the infra-orbital foramen, which is 0.5cm below the infra-orbital margin and vertically below the pupil when the eyes are looking forward. The nerve can be blocked at the infra-orbital foramen by injection through the skin, but the intraoral approach is preferable, because it is less unpleasant for the patient. Insert the needle into the buccogingival fold between the first and second premolars, and direct it up towards the infra-orbital foramen.

## Mental nerve block

The mental nerve supplies sensation to the lower lip and chin. It emerges from the mental foramen, which is palpable on the mandible on a line between the first and second premolar teeth. The nerve can be blocked at the mental foramen with 1–2mL of LA, using either an intra- or extra-oral approach. Avoid injecting into the mental canal, since this may damage the nerve. If the wound to be repaired extends across the midline, bilateral mental nerve blocks will be needed.

The nerves to a single lower incisor may be blocked by submucous infiltration of LA in the buccal sulcus adjacent to the tooth.

# Nerve blocks of forehead and ear

## Nerve blocks of the forehead

Many wounds of the forehead and frontal region of the scalp can be explored and repaired conveniently under LA block of the supraorbital and supratrochlear nerves (see Fig. 7.5).

*The supraorbital nerve* divides into medial and lateral branches, and leaves the orbit through two holes or notches in the superior orbital margin, ~2.5cm from the midline. The branches of the supraorbital nerve supply sensation to most of the forehead and the frontal region of the scalp.

*The supratrochlear nerve* emerges from the upper medial corner of the orbit and supplies sensation to the medial part of the forehead.

*Technique*

- Use 5–10mL of 1% lidocaine, with or without adrenaline.
- Insert the needle in the midline between the eyebrows and direct it laterally.
- Inject LA SC from the point of insertion along the upper margin of the eyebrow.
- If the wound extends into the lateral part of the forehead, SC infiltration of LA may be needed lateral to the eyebrow, to block the zygomaticotemporal and auriculotemporal nerves.

*Possible complications*

- Injury to the eye can occur if the patient moves during the injection.
- It is possible to block the supraorbital nerve at the supraorbital foramen, but this is not advisable since inadvertent injection into the orbit may cause temporary blindness if the LA reaches the optic nerve.

## Nerve blocks of the ear

The auricle (pinna) of the ear is supplied by branches of the greater auricular nerve (inferiorly), the lesser occipital nerve (posteriorly), and the auriculotemporal nerve (anteriorly/superiorly). These nerves can be blocked by SC infiltration of up to 10mL of 1% plain lidocaine in the appropriate area or in a ring around the ear (see Fig. 7.5).

To block the *greater auricular nerve*, infiltrate 1cm below the earlobe from the posterior border of the sternomastoid muscle to the angle of the mandible.

Block the *lesser occipital nerve* by infiltration just behind the ear.

When blocking the *auriculotemporal nerve* by infiltration just anterior to the external auditory meatus, aspirate carefully to avoid inadvertent injection into the superficial temporal artery.

## Nerve blocks: the forehead and ear

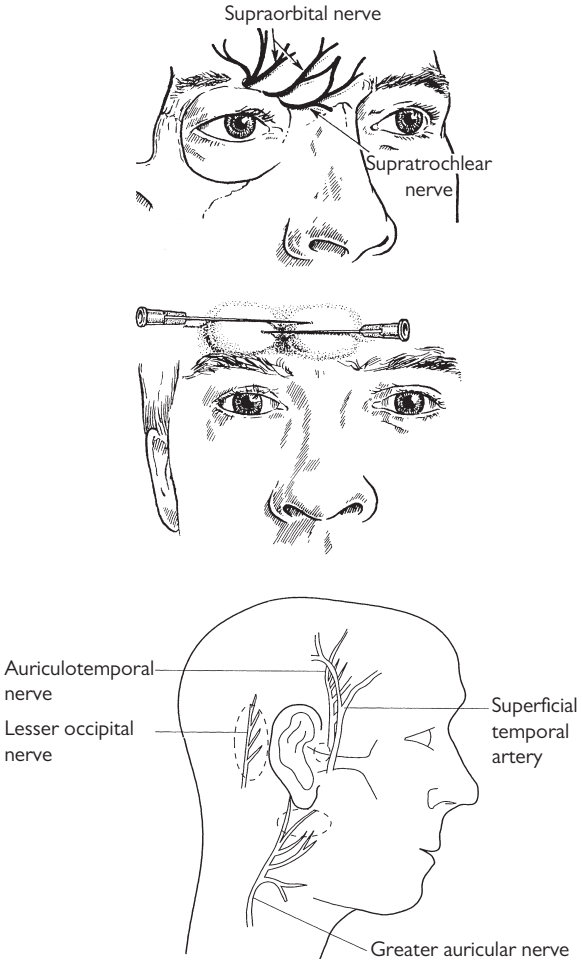



**Fig. 7.5** Nerve blocks of the forehead and ear.

# Fascia iliaca compartment block

## Anatomy

The femoral nerve passes under the inguinal ligament, lateral to the femoral artery. The femoral nerve supplies the hip and knee joints, the skin of the medial and anterior thigh, and the quadriceps, sartorius, and pectineus muscles in the anterior compartment of the thigh. The lateral femoral cutaneous nerve passes under the inguinal ligament near the anterior superior iliac spine, to supply sensation to the lateral aspect of the thigh.

## Background

The principal advantage of a fascia iliaca compartment block over a femoral nerve block is that it can be performed 'blind' (ie without USS guidance), with minimal risk of inadvertent puncture of neurovascular structures. It has been advocated for use with hip and/or femoral shaft fractures. The block works by LA spreading throughout the compartment, rather than targeting a specific individual nerve, so a relatively large volume of (diluted) LA is required. It can be reasonably expected to block the femoral and lateral femoral cutaneous nerves.

## Contraindications

- Allergy to LA.
- Infection and/or inflammation over the injection site.
- Previous femoral bypass surgery or near a graft site.
- Anticoagulation.

## Technique

- Explain the procedure and gain consent from the patient.
- Prepare the equipment, including relevant specialized long needles and LA:
  - Use 30mL of 0.25% levobupivacaine in adults weighing ≤50kg.
  - Use 40mL of 0.25% levobupivacaine in adults weighing >50kg.
- Clean the skin and identify the anterior superior iliac spine and pubic tubercle on the side of the fracture, and place a finger on each of these, then draw an imaginary line between them and divide this line up into thirds, thereby identifying the junction of the lateral third and medial two-thirds—the injection site is 1cm below (caudal to) this point. The femoral pulse is well medial to this.
- Direct the needle in the sagittal plane, and having pierced the skin, advance through two distinct palpable 'pops' (penetration through first the fascia lata and then the fascia iliaca). Advance a further 1–2mm, then attempt to aspirate, and if no blood is aspirated, inject LA slowly—if there is resistance, stop and withdraw slightly, then try again. Stop if there is pain or paraesthesiae on injection. Attempt to aspirate after every 5mL of injection.
- At the end of the procedure, withdraw the needle and apply a small amount of pressure to the injection site.
- Following the procedure, continue regular observations (pulse, BP, RR, $SpO_2$) every 5min for at least 30min—as pain improves, the possible effect on the RR of any IV opioids which have been given may be accentuated.

# Femoral nerve block

This provides good analgesia within a few minutes for pain from a fractured neck or shaft of femur. It may be used in children, as well as in adults.

## Technique (without using ultrasound)

(See Fig. 7.6.)

- Use a mixture of lidocaine and bupivacaine to give both rapid-onset and prolonged anaesthesia. In an adult, give 5mL of 1% lidocaine and 5mL of 0.5% bupivacaine. In a child, use 0.1mL/kg of 1% lidocaine and 0.1mL/kg of 0.5% bupivacaine. Check the maximum dose carefully, especially in children or if bilateral femoral nerve blocks are needed.
- Use a 0.8 × 40mm (21G) needle in adults, and a 0.6 × 25mm (23G needle in children.
- Blocking the right femoral nerve is easiest to perform by standing on the patient's left side (and vice versa).
- Using your non-dominant hand, palpate the femoral artery just below the inguinal ligament.
- Clean the skin.
- Insert the needle perpendicular to the skin and 1cm lateral to the artery to a depth of ~3cm. If paraesthesiae occurs, withdraw the needle 2–3mm.
- Aspirate and check for blood.
- Inject LA whilst moving the needle up and down and fanning out laterally to ~3cm from the artery. (The distances quoted refer to adults.)
- If the femoral artery is punctured, compress it for 5–10min. If no bleeding is apparent, continue with the femoral nerve block.

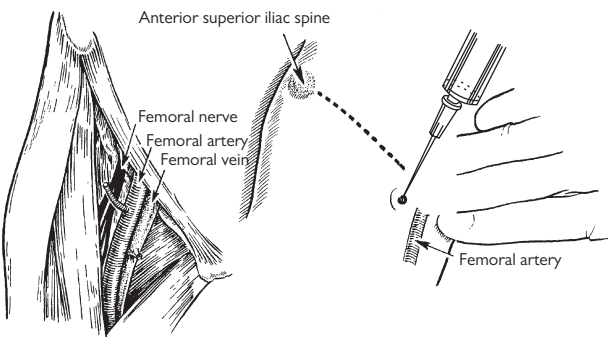



**Fig. 7.6** Femoral nerve block.

## Ultrasound-guided technique

The 'blind' technique for femoral nerve block has a high success rate and usually provides rapid and effective analgesia. However, USS (see ➲ Local anaesthetic nerve blocks, p. 302) is very helpful to delineate the anatomy of the femoral nerve (which can vary between patients) and to allow precise positioning of the injection adjacent to the nerve.

# Nerve blocks at the ankle

## Indications

- Cleaning, exploration, and suturing of wounds of the foot.
- Removal of FBs, drainage of small abscesses on the sole of the foot.
- Analgesia for crush injuries of the forefoot.
- LA blocks at the ankle are particularly useful for anaesthetizing the sole of the foot where local infiltration is very painful and unsatisfactory.

## Anatomy

Sensation in the ankle and foot is supplied by five main nerves:

- The saphenous nerve (medial side of the ankle).
- The superficial peroneal nerve (front of the ankle and dorsum of the foot).
- The deep peroneal nerve (lateral side of the big toe and medial side of the second toe).
- The sural nerve (heel and lateral side of the hind foot).
- The tibial nerve (which forms the medial and lateral plantar nerves, supplying the anterior half of the sole).

There are individual variations and significant overlap between the areas supplied by different nerves, especially on the sole of the foot. It is often necessary to block more than one nerve (see Fig. 7.7).

For each of these blocks, use a 0.6mm (23G) needle and 5mL of 1% lidocaine (with or without adrenaline) or 0.5% bupivacaine. Check the maximum dose (see ➲ Local anaesthesia, pp. 292–3), especially for multiple blocks. USS can help to allow accurate injection of LA, so smaller amounts are needed.

Do not use adrenaline in patients with peripheral vascular disease.

## Saphenous nerve

Infiltrate LA SC around the great saphenous vein, anterior to and just above the medial malleolus. Aspirate carefully because of the risk of IV injection.

## Superficial peroneal nerve

Infiltrate LA SC above the ankle joint from the anterior border of the tibia to the lateral malleolus.

## Deep peroneal nerve

Insert the needle above the ankle joint between the tendons of the tibialis anterior and the extensor hallucis longus. Inject 5mL of LA.

## Sural nerve

Lie the patient prone. Insert the needle lateral to the Achilles tendon, and infiltrate subcutaneously to the lateral malleolus.

## Tibial nerve

Lie the patient prone. Palpate the posterior tibial artery. Insert the needle medial to the Achilles tendon and level with the upper border of the medial malleolus, so the needle tip is just lateral to the artery. Withdraw slightly if paraesthesiae occurs. Aspirate. Inject 5–10mL of LA.

## Nerve blocks at the ankle

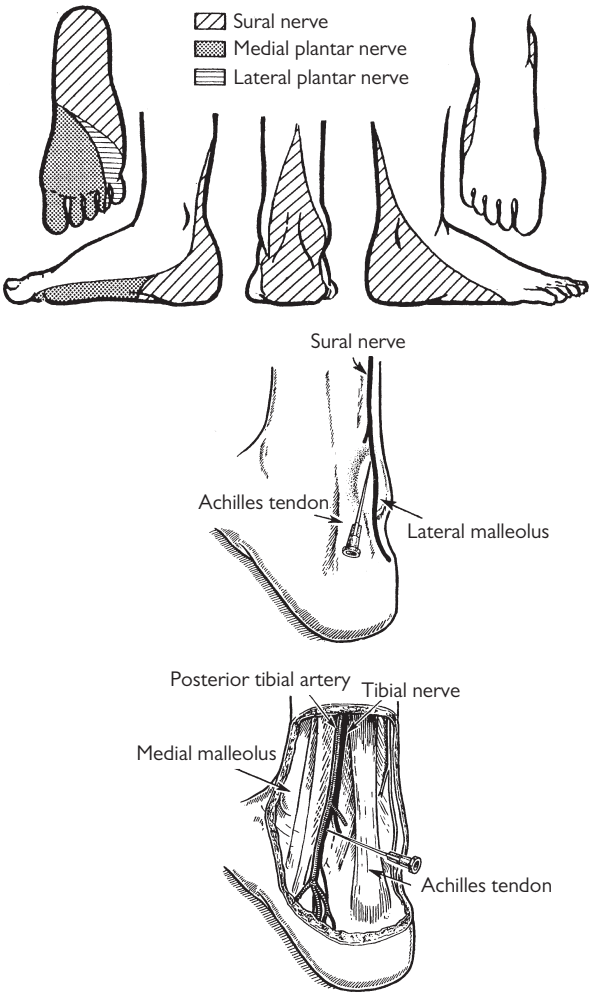



**Fig. 7.7** Nerve blocks at the ankle.

# Approach to sedation

Sedation is extremely important in everyday ED work to help patients tolerate painful and distressing procedures such as reduction of dislocations.

## Definitions

Sedation is a continuum ranging from being fully conscious through to unresponsiveness. The American Society of Anesthesiologists (ASA) has developed a classification, outlined in Table 7.2. For most purposes, the aim of sedation in the ED is to achieve 'minimal' or 'moderate' sedation using this scheme. Separate from this classification is 'dissociative sedation' using ketamine, in which the patient has a trance-like cataleptic state with significant analgesia and amnesia, but with relatively preserved airway reflexes, breathing, and circulation.

**Table 7.2** ASA classification of depth of sedation

| | |
|---|---|
| Minimal sedation (anxiolysis) | Normal response to voice. Airway, breathing, and circulation unaffected |
| Moderate sedation/ analgesia ('conscious sedation') | Purposeful response (not reflex withdrawal) to voice or touch. Airway maintained, breathing adequate, and circulation usually maintained |
| Deep sedation/ analgesia | Purposeful response to repeated or painful stimulation. Airway may need protection and ventilation may need support. Circulation usually maintained |
| General anaesthesia (GA) | Unrousable, even by painful stimulus. Airway often requires intervention; ventilation is frequently inadequate, and cardiovascular function may be impaired |

## Indications

Sedation is usually indicated in the ED to enable brief painful procedures or interventions to occur. Sedation is most commonly used in the ED for emergency reduction of dislocations and/or fractures and cardioversion for potentially life-threatening arrhythmias, but it is also sometimes used for less urgent conditions (eg the exploration/cleaning/closure of wounds).

When appropriate, sedation may be used with an analgesic or LA. Similarly, do not use sedation when it would be more appropriate to use GA.

## Consent and documentation

Explain and discuss the procedure and associated risks with the patient, and record this within the notes. Complete a standard pro forma for each patient who has sedation. In addition to recording observations and drugs given as part of the procedure, ensure that pre- and post-sedation checks are all recorded.

## Risk assessment

Sedation carries many of the same risks and complications as those of GA. The principal concerns are obstructed airway, aspiration of gastric contents, respiratory depression, and ↓ cardiac output. Assess the risks before committing to provide sedation. Ideally, patients should be fasted before IV sedation. Ask about and record pre-existing medical conditions, drug therapy, allergies, and time of last food and drink. Record vital signs: pulse, BP, RR, and $SpO_2$. Establish the ASA physical status classification (see ➲ General anaesthesia in the ED, pp. 320–1).

### *Airway problems*

Clues to possible difficulties managing the airway during sedation include: older, obese patients with a beard, a short neck, or a small mouth opening, history of sleep apnoea, previous airway problems during anaesthesia, and rheumatoid arthritis.

### *Aspiration of gastric contents*

Vomiting or regurgitation and aspiration of gastric contents is a particular risk in patients who are fasted for <4hr, especially for deeper sedation. Delaying procedures may not help as gastric emptying can be delayed in patients who have just been injured and/or given opioid drugs. Fasting is not needed for sedation using $N_2O/O_2$ mixtures alone or for minimal or moderate sedation where verbal contact is maintained. For procedures requiring deeper sedation where there is a concern about the lack of fasting time, consider whether more experienced staff and/or GA may be needed instead. Discuss the risk of aspiration vs the benefits of performing the procedure under sedation when obtaining consent.

### *Respiratory and cardiac complications*

Patients at ↑ risk of respiratory or cardiac complications include the elderly, those with obesity, and those with pre-existing heart/lung disease and/or ASA ≥3.

## Staff

Ensure that sedation is only given by appropriately trained staff. The number and training of staff required will vary according to the depth of sedation. RCEM recommendations for minimum staffing for moderate sedation/analgesia ('conscious sedation') are one physician to provide sedation and one physician or nurse as operator and one nurse.

Some sedatives cause amnesia and transient confusion—a chaperone may avoid difficulties if there is any allegation of impropriety.

## Equipment

Use a trolley which can be tilted head-down. Ensure suction, resuscitation equipment, and reversal drugs are immediately available.

## Monitoring during IV sedation

Ensure sedated patients have a venous cannula and receive $O_2$, pulse oximetry monitoring, and capnography (most accurately using nasal prongs with an end-tidal $CO_2$ outlet). Monitor the ECG. Continue monitoring observations every 5min for at least 30min after the last drug was given.

# Drugs for IV sedation

Sedative drugs may be given PO, IM, IV, or by inhalation. All IV sedative drugs will produce anaesthesia if given in excessive dosage. Aim to use the minimum dose that will give adequate sedation and allow the procedure to be completed satisfactorily. Note that patients with renal or hepatic disease may require ↓ drug dosage.

- Midazolam is the most suitable benzodiazepine. It has a plasma half-life of ~2hr in young adults (longer in the elderly or obese) and metabolites are relatively inactive. In fit adults, the initial dose of midazolam is 2mg IV over 1min. If sedation is inadequate after 2min, give incremental doses of 0.5–1mg. When fully sedated, the patient will be drowsy, with slurred speech, but will obey commands. A typical dose is 2.5–5mg. Elderly patients are more susceptible to benzodiazepines—give smaller doses. Give 0.5–1mg as an initial dose—the total dose needed is usually 1–3mg.
- Diazepam is not suitable for IV sedation of patients for planned later discharge, since it has a prolonged action and an active metabolite with a plasma half-life of ~3–5 days.
- Opioids such as fentanyl (dose 0.5mcg/kg—see ➲ Analgesics: other opioids, p. 288) may be used IV combined with midazolam, but there may be a synergistic effect with an ↑ risk of respiratory depression. Give the opioid first, followed by careful titration of midazolam.
- Other drugs: propofol (see ➲ General anaesthetic drugs, p. 326–7) can give excellent sedation for short procedures, with rapid recovery, but its use requires anaesthetic training. Ketamine (see ➲ Ketamine, p. 287) may be given IV or IM, but it requires special training.

### Antagonists

Ensure that the specific antagonists flumazenil (for benzodiazepines) and naloxone (for opioids) are available immediately, although they should be needed very rarely. If respiratory depression occurs, standard techniques to maintain the airway and breathing are more important than giving antagonists. Flumazenil and naloxone have shorter durations of action than the drugs they antagonize, so continue careful observation if either drug is used.

### Recovery and discharge after sedation

If IV sedation is used, monitor carefully until recovery is complete. Minimum criteria for discharging a patient are:

- Stable vital signs.
- Ability to walk without support.
- Toleration of oral fluids and minimal nausea.
- Adequate analgesia.
- Adequate supervision at home by a responsible adult.

Instruct the patient (both verbally and in writing) not to drive, operate machinery, make any important decisions, or drink alcohol for 24hr. Arrange appropriate follow-up. Ensure the adult accompanying the patient knows who to contact if there is any problem.

# Sedation in children

Many children (and their parents and staff) are distressed by procedures such as suturing of minor wounds under LA. Sedation is helpful to prevent distress and allows procedures to take place with minimal physical restraint. A suitable environment within the ED is required in order to enable sedation of children—this includes a quiet child-friendly area with $O_2$, suction, and monitoring and resuscitation equipment available, together with a quiet recovery area.

Sedation may be given by PO or nasal routes, IM, or IV. Paediatric IV sedation requires anaesthetic experience because of the narrow therapeutic margin between sedation and anaesthesia.

Inhalational sedation and analgesia with $N_2O$ (Entonox®—see ➲ Analgesics: Entonox® and ketamine, p. 287) are rapidly reversible and relatively risk-free, and can be used, when appropriate, in adults and some children.

## Ketamine in children

*Ketamine* given IM in a dose of 2–2.5mg/kg is often used for paediatric sedation in the ED by staff with appropriate training (see ➲ Analgesics: Entonox® and ketamine, p. 287). An alternative is 1mg/kg IV over 1–2min. These doses of ketamine do not provide full anaesthesia, and so local anaesthesia is still required for cleaning and suturing of wounds. Three members of staff are needed: one doctor to manage sedation and the airway, one clinician to perform the procedure, and a nurse to support the patient/family and assist with regular observations (every 5min) for at least 30min after giving sedation.

### *Adverse effects*

- Vomiting occurs in up to 10% of children, usually during recovery.
- Agitation is unusual but responds to small doses of midazolam.
- Laryngospasm can occur occasionally following administration of ketamine, but this small risk is reduced by using low doses of IM ketamine.

### *Recovery and discharge*

Children have usually recovered within 1–2hr. They can be safely discharged once able to walk, talk, and interact normally. Provide the parents with advice (including a written advice sheet) on what to do following discharge—in particular, advise them that their child should not eat or drink for 2hr after discharge because of the risk of vomiting.

## Other drugs

*Oral midazolam* is used by some specialists. Oral sedation with *promethazine* is not advisable, since it is often ineffective.

# General anaesthesia in the ED

GA may be needed in the ED for many different conditions:

- Minor surgery (eg drainage of abscesses, manipulation of fractures).
- Cardioversion.
- Airway problems (eg facial trauma, burns, epiglottitis).
- Respiratory failure (eg asthma, COPD, pulmonary oedema, chest injuries).
- To protect the airway and control ventilation after head injuries and to keep the patient immobile for a CT scan.
- To protect the airway and maintain ventilation in status epilepticus unresponsive to standard drug therapy.
- Immediate major surgery (eg thoracotomy or laparotomy for trauma, ruptured ectopic pregnancy, or aortic aneurysm). If at all possible, take the patient to the operating theatre before anaesthesia, as the loss of sympathetic tone after the onset of anaesthesia can cause catastrophic hypotension in a hypovolaemic patient. In extreme emergencies, it may be necessary to operate in the ED.

GA in the ED tends to be stressful for the anaesthetist and potentially hazardous for the patient, who is usually unprepared for anaesthesia, with a full stomach and an ↑ risk of aspiration. GA should only be given by doctors with anaesthetic training, but other staff should know what is required, so they can help when necessary.

## Preoperative assessment

This is essential for safe anaesthesia. If time allows, assess the patient before contacting the anaesthetist to arrange anaesthesia. However, if emergency anaesthesia is needed, call the anaesthetist immediately, so that he/she can come and assess the patient, and get senior help if necessary. For a checklist of questions to ask before GA, see ➲ Checklist for preoperative assessment in the ED, p. 321.

*Fitness for GA* The ASA classification of preoperative fitness is widely used by anaesthetists:

1 Healthy patient with no systemic disease.
2 Patient with a mild to moderate disease which does not limit their activity in any way (eg treated hypertension, mild diabetes, smoker).
3 Patient with a severe systemic disturbance from any cause which limits activity (eg IHD with ↓ exercise tolerance, severe COPD).
4 Patient with a severe systemic disease which is a constant threat to life (eg severe chronic bronchitis, advanced liver disease).
5 Moribund patient who is unlikely to survive 24hr with or without treatment.

The risk of complications from GA correlates well with the ASA group. Only patients in ASA groups 1 and 2 should be given an elective anaesthetic by a junior anaesthetist in the ED. Children aged <7y should not usually have GA in the ED, except in an emergency.

### Preoperative investigations

No investigation is needed, unless preoperative assessment reveals a problem. Measure Hb in any patient who appears anaemic. Ask about sickle-cell disease in any patient of Afro-Caribbean, Cypriot, or Indian origin. Measure U&E in patients on diuretics, and blood glucose in patients with diabetes. ECG and CXR are not needed, unless clinically indicated. Perform a pregnancy test if pregnancy is possible.

### Checklist for preoperative assessment in the ED

- Age.
- Weight.
- Time of last drink.
- Time of last food.
- Drugs.
- Drugs given in the ED.
- Time of last analgesia.
- Allergies.
- Sickle-cell risk?
- Infection risk?
- Family history of GA problems?
- Airway problem?
- Dentures/crowns/loose teeth?
- Chest disease?
- Smoker?
- Cardiac disease?
- BP.
- GI problem?
- Other illness?
- Possibility of pregnancy?
- Previous GA (problems?)?
- Consent form signed?
- Is the patient expected to go home after recovery from anaesthesia?
- Is there a responsible adult who can look after the patient at home?

### Preparation for GA

Ideally, the patient should have nothing to drink for 4hr and no food for 6hr before anaesthesia. Explain why this is necessary. Fasting does not guarantee an empty stomach. Trauma, pregnancy, and opioids delay gastric emptying.

If the patient is in pain, give analgesia and an antiemetic. Discuss with the anaesthetist any other drug treatment that is required. Patients with a hiatus hernia or gastro-oesophageal reflux need antacid prophylaxis (eg ranitidine 50mg IV and an antacid).

Explain the proposed operation and anaesthesia to the patient (and relatives, if appropriate) and ensure valid consent is obtained. The patient must be clearly labelled with a wristband. Remove contact lenses, false teeth, and dental plates.

### Recovery and discharge after anaesthesia

When the operation has finished, place the patient in the recovery position and ensure continuous observation by trained staff until recovery is complete. The anaesthetist should stay with the patient until consciousness is regained and the airway is controlled. Monitoring and resuscitation equipment and drugs must be available. The minimum criteria for discharging a patient are the same as those following sedation (see ➲ Recovery and discharge after sedation, p. 318).

Importantly, tell the patient (both verbally and in writing) not to drive, operate machinery, make any important decisions, or drink alcohol for 24hr. Arrange appropriate follow-up and make sure that the adult accompanying the patient knows who to contact if there is a problem.

# Emergency anaesthesia and rapid sequence induction

Emergency anaesthesia and intubation are often needed to protect the airway and provide adequate ventilation in a patient with a head injury or multiple trauma. There is a high risk of aspiration of gastric contents into the lungs, so use a cuffed ET tube (uncuffed in infants). In a patient with a gag reflex, any attempt to intubate without anaesthesia may cause vomiting and aspiration. Anaesthesia before intubation is essential in head-injured patients to minimize the ↑ in ICP.

## Rapid sequence induction

RSI involves administration of a sedative or an induction agent virtually simultaneously with a neuromuscular-blocking agent to allow rapid tracheal intubation.

►► *RSI should only be performed by staff who have had specific training and experience in the techniques and the drugs used, and the recognition and management of possible problems.* However, it is useful if ED staff who have not had such training understand the principles of RSI, so that they can assist as needed.

- Call for senior ED/anaesthetic/ICU help.
- Check all drugs and equipment using a standardized checklist including: suction, bag–valve–mask (or water circuit and T piece), laryngoscope (and spare with large blade), bougie, appropriate-sized tracheal tubes (× 2) with cuffs checked, then deflated, syringe, connector to attach tracheal tube to bag/ventilator, in-line end-tidal $CO_2$ monitoring, and tube holder/tie. Ensure that an appropriately sized supraglottic device [i-Gel or laryngeal mask airway (LMA)] and equipment for a surgical airway are immediately available.
- Check that the trolley can be tilted head-down easily.
- Check monitoring equipment (ECG, BP, pulse oximeter, end-tidal $CO_2$ monitor).
- Explain the procedure to the patient, if possible.
- Assess the risks and any conditions which might cause problems with intubation (eg trauma to the face or neck, ↓ mouth opening, receding chin). Identify and always verbalize a backup plan for failed intubation prior to commencing the procedure (see Difficult intubation, pp. 324–5) and communicate this to the team. The Difficult Airway Society has produced some useful guidelines on this (https://www.das.uk.com).
- Establish monitoring (ECG and pulse oximetry) and secure IV access.
- Protect the cervical spine in all trauma patients—an assistant should provide in-line immobilization during intubation. In other patients, use a pillow and position the head and neck to aid intubation.
- If possible, pre-oxygenate for 3min with 100% $O_2$ via a tight-fitting mask, with the patient breathing spontaneously. If breathing is inadequate, ventilate for 2min on 100% $O_2$ with a bag and mask.
- Ask an assistant to apply cricoid pressure whilst the patient is pre-oxygenated by pressing firmly downwards with the thumb and index finger on the cricoid cartilage. Note that although evidence for cricoid pressure is lacking, it is still widely used.
- Administer the appropriate drugs.

## Standard drugs for RSI

A balanced RSI requires an induction drug in an appropriate dose and a neuromuscular-paralysing drug to facilitate intubation. A widely used combination is fentanyl (3mcg/kg), followed by ketamine (2mg/kg), with immediate administration of a muscle relaxant, typically rocuronium (1mg/kg).

*Pre-induction opioid*

The use of a pre-induction dose of opioid to ↓ sympathetic response to laryngoscopy benefits haemodynamically stable patients and those with ↑ ICP.

*Ketamine*

Note that ketamine is useful in trauma RSI as it helps maintain haemodynamic stability. ↑ ICP is not a contraindication to ketamine at induction as cerebral perfusion is augmented and pre-treatment with opioid will offset the sympathetic response. However, the sympathomimetic effects of ketamine can result in tachycardia and hypertension, which may be harmful in some patients with cardiac disease—these effects can be mitigated with pre-induction fentanyl.

*Rocuronium*

The standard drug for neuromuscular blockade is rocuronium, which is reliable, lasts longer (hence less unwanted 'wake-up'), and has fewer contraindications than suxamethonium.

## Intubation technique

- Maintain the cricoid continuously until the airway is secure.
- Keep the face mask tightly applied until the anaesthetic and relaxant are effective.
- Then intubate and inflate the cuff quickly.
- Try to confirm tracheal placement of the tube—ideally it will have been seen passing through the cords, but this may not always be possible in an emergency intubation.
- Check air entry in both sides of the chest.
- Check end-tidal $CO_2$ (but this may be misleading if oesophageal intubation occurs in a patient who has recently consumed antacids or fizzy drinks). If $CO_2$ is not detected, oesophageal intubation has occurred.
- Release cricoid pressure when the ET tube is correctly positioned, the cuff has been inflated, and ventilation is satisfactory.
- Secure the tracheal tube.
- Continue observation and monitoring.

# Difficult intubation

Difficulties with intubation may result from problems with the equipment, the patient, and the circumstances of intubation, and from a lack of experience or skill.

## Equipment

Ensure proper working equipment is available where intubation may be needed: pillow, suction, laryngoscope (and spare) with interchangeable blades, ET tubes of different diameters (cut to suitable lengths, but with uncut tubes available), syringe and clamp for cuff, connectors, flexible stylet, gum-elastic bougie, lubricating jelly, Magill's forceps, and tape for securing ET tube. A face mask and a ventilating bag and oral/nasal airways must be immediately available. LMAs and cricothyroidotomy equipment must be accessible. Fibre-optic/video laryngoscopes are proving to be increasingly useful.

## The patient

Patients may be difficult to intubate because of facial deformity or swelling, protruding teeth, ↓ mouth opening from trismus or trauma, ↓ neck movement or instability of the cervical spine, epiglottitis or laryngeal problems, tracheal narrowing or deviation, and blood, vomit, or FB in the airway.

## Circumstances and skills

Intubation is much easier in the controlled environment of an operating theatre than in an emergency in the ED or in prehospital care. Skilled help is vital—in-line immobilization of the neck, cricoid pressure, and assistance with equipment and cuff inflation are needed. Practice intubating manikins regularly.

## Practical points

Before attempting intubation, oxygenate by bag-and-mask ventilation. Take a deep breath as you start intubation—if the patient is not intubated successfully when you have to breathe again, remove the ET tube and laryngoscope, and ventilate with $O_2$ for 1–2min using a bag and mask before another attempt. Consider adjusting the patient's position, using a different size of laryngoscope blade or ET tube or a stylet or bougie. Cricoid pressure can be changed to laryngeal manipulation by pushing the larynx backwards into view. The BURP (Backwards, Upwards, Rightwards Pressure) manoeuvre on the thyroid cartilage may be useful in a difficult intubation.

## Oesophageal intubation

Fatal if unrecognized. The best way of confirming tracheal intubation is to see the ET tube pass between the vocal cords. Inadvertent oesophageal intubation can produce misleadingly normal chest movements and breath sounds. End-tidal $CO_2$ measurement helps to confirm tracheal intubation, but it can be misleadingly ↑ in patients who have taken antacids or fizzy drinks. If in doubt, remove the ET tube and ventilate with a bag and mask.

## Difficult intubation guidelines

Persistent unsuccessful attempts at intubation cause hypoxia and an ↑ risk of aspiration and damage to the teeth and other structures. Get senior help early. (See Fig. 7.8 and the Difficult Airway Society guidelines available at https://www.das.uk.com)

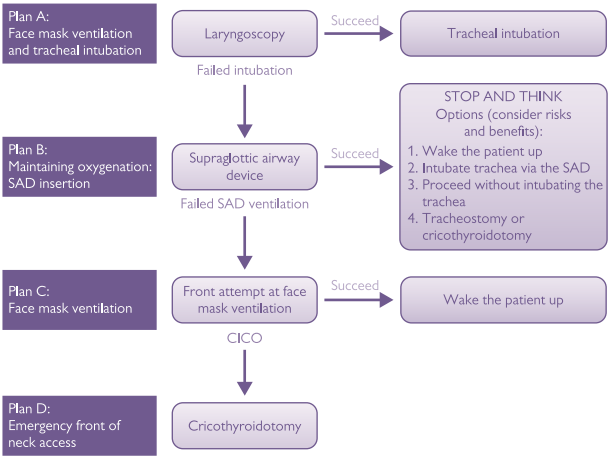



**Fig. 7.8** Overview of the Difficult Airway Society's difficult airway guidelines. CICO, Cannot Intubate Cannot Oxygenate; SAD, supraglottic airway device.

Reproduced from Frerk, C. *et al*. (2015). Difficult Airway Society 2015 guidelines for management of unanticipated difficult intubation in adults, BJA: *British Journal of Anaesthesia*, Volume 115, Issue 6, December 2015, Pages 827–848, https://doi.org/10.1093/bja/aev371

## Laryngospasm

Laryngospasm occurs when the laryngeal muscles contract and occlude the airway, preventing ventilation and causing hypoxia.

*Causes*

- Stimulation of the patient during light anaesthesia.
- Airway irritation by secretions, vomit, blood, or oropharyngeal airway.
- Extubation of a lightly anaesthetized patient.

*Treatment*

- Give 100% $O_2$ and get expert help.
- Clear the airway of secretions, using gentle suction.
- Gently ventilate the patient using a bag and mask, with gentle application of PEEP. Over-inflation is liable to fill the stomach and cause regurgitation.
- Monitor the ECG for bradycardia or arrhythmias.

In severe laryngospasm, an experienced anaesthetist may consider deepening anaesthesia or giving a muscle relaxant to allow intubation or ventilation with a bag and mask.

# General anaesthetic drugs

► *GA should only be given after anaesthetic training.*

IV induction agents are used for induction of anaesthesia, as the sole drug for short procedures (eg cardioversion), for treatment of status epilepticus unresponsive to other anticonvulsants (see ➲ Seizures and status epilepticus, pp. 156–7), for total IV anaesthesia, and for sedation of a ventilated patient. They are particularly hazardous in patients with upper airway obstruction or severe hypovolaemia. Thiopental, etomidate, and many other drugs are unsafe in acute porphyria (see ➲ Porphyria, p. 173 and *BNF*).

## Propofol

This is particularly useful in day-case surgery and for manipulation of fractures and dislocations, because recovery is rapid. Injection causes a burning sensation, which may be painful. Hypotension is common and severe bradycardia may occur. Induction dose is 1.5–2.5mg/kg. Propofol has profound vasodilatory effects which can result in haemodynamic instability, but consider it for those patients with a purely neurological indication for RSI (particularly if there is coexisting hypertension or seizures).

## Ketamine

This is used in prehospital care and increasingly in hospital care as well (see ➲ Ketamine, p. 287). It may be useful for RSI in hypotensive patients and in acute asthma. Induction dose is 1–2mg/kg IV.

## Etomidate

This causes less hypotension than propofol or thiopental (and so may be useful in patients who are already hypotensive), and recovery is rapid. However, the injection is painful and uncontrolled muscle movements and adrenocortical suppression may occur—avoid it in patients who have, or are at risk of developing, sepsis. Induction dose is up to 0.3mg/kg.

## Thiopental (thiopentone)

This is a barbiturate drug. Thiopental sodium solution is unstable and has to be prepared from powder to form a 2.5% solution (25mg/mL). Care is needed with injections because extravasation causes irritation and arterial injection is particularly dangerous. Hypotension may occur, especially with overdosage. The induction dose in a fit adult is up to 4mg/kg (child: 2–7mg/kg).

## Muscle relaxants

*Rocuronium* is a muscle relaxant that is now considered the standard drug to allow intubation, especially in RSI of anaesthesia (see ➔ Emergency anaesthesia and rapid sequence induction, pp. 322–3). The muscle relaxant effects of rocuronium last for much longer than those of suxamethonium (typically ~45min).

*Atracurium and vecuronium* are non-depolarizing muscle relaxants, which act for ~20–30min. They cause fewer adverse effects than older relaxants (eg pancuronium). Paralysis from these drugs can be reversed with neostigmine, which is given with atropine or glycopyrronium to prevent bradycardia.

*Suxamethonium* is a short-acting depolarizing muscle relaxant, which is now used much less frequently. In a dose of 1mg/kg, it causes muscle fasciculation, followed rapidly by flaccid paralysis. It is contraindicated in hyperkalaemia and also in burns, paraplegia, or crush injuries where dangerous hyperkalaemia may develop if suxamethonium is used 5–120 days after injury. Suxamethonium causes ↑ ICP and ↑ intra-ocular pressure. Usual duration of action is ~5min, but prolonged paralysis occurs in patients with abnormal pseudo-cholinesterase enzymes.

## Inhalational anaesthetics

These can be used for analgesia (especially Entonox®), induction of anaesthesia (particularly in upper airway obstruction, when IV induction of anaesthesia is hazardous), and maintenance of anaesthesia.

*$N_2O$* is widely used for analgesia as Entonox®, a 50:50 mixture with $O_2$ (see ➔ Analgesics: Entonox® and ketamine, p. 287). It is also used frequently in GA in a concentration of 50–70% in $O_2$, in combination with other inhaled or IV anaesthetics. $N_2O$ is contraindicated in certain circumstances (eg undrained pneumothorax—see ➔ Analgesics: Entonox® and ketamine, p. 287).

*Methoxyflurane (Penthrox®)* is an inhalational agent used as an analgesic in trauma by senior/suitably trained clinicians.

*Halothane, enflurane, isoflurane, and sevoflurane* are inhalational anaesthetic agents that are given using specially calibrated vaporizers in $O_2$ or a mixture of $N_2O$ and $O_2$. Sevoflurane is particularly useful for gas induction of anaesthesia in upper airway obstruction and can play a role in ongoing bronchospasm in life-threatening asthma. Halothane is also effective for gas induction but is now rarely used because of the risk of hepatotoxicity, especially after repeated use. Halothane sensitizes the heart to catecholamines, so adrenaline must not be used with halothane. Inhalational anaesthetic drugs can cause malignant hyperpyrexia (see ➔ Heat illness, pp. 274–5) in susceptible patients.

Chapter 8

# Major trauma

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# Major trauma: treatment principles

Suspect major trauma in:

- High-speed road collisions, vehicle ejection, rollover, and prolonged extrication.
- Death of another individual in the same collision.
- Pedestrians thrown up or run over by a vehicle.
- Falls of >2m.

### Airway control

Use basic manoeuvres (suction, chin lift, oropharyngeal airway) to open the airway and apply $O_2$ by face mask. Avoid head tilting or moving the neck if there is possible neck injury. If the airway remains obstructed despite these measures, consider advanced manoeuvres (see ➲ Airway obstruction: surgical airway, p. 336).

### Oxygen

Provide $O_2$ as required. Treat patients who are apnoeic or hypoventilating with bag-and-mask ventilation prior to tracheal intubation and IPPV.

### Cervical spine control

This is an early priority in a patient who presents with possible spine injury (eg neck pain, loss of consciousness). There are concerns about routine use of collars (eg ↑ ICP), so consider the need for adhesive tape and sandbags, but bear in mind that these may cause problems (eg patients who are vomiting or unco-operative patients who have drunk alcohol).

### IV fluids

Insert two large cannulae into forearm or antecubital fossa veins. If initial attempts fail, consider intra-osseous (IO) access. Avoid inserting lines into the lower limbs in suspected pelvic trauma. Treat hypovolaemia from blood loss with IV blood and blood products, rather than large amounts of crystalloid. Consider starting the Massive Haemorrhage Protocol (see ➲ Massive blood transfusion, p. 182).

### Tranexamic acid

In suspected major haemorrhage (and within 3hr of injury), give tranexamic acid 1g IV over 10min (if not already given by paramedics prehospital). Follow this with an infusion of tranexamic acid 1g IVI over 8hr.

### Analgesia

Give morphine IV (diluted in saline to 1mg/mL), titrated in small increments according to response. Provide an antiemetic (eg cyclizine 50mg IV) at the same time. Consider other forms of analgesia (eg regional nerve blocks, immobilization, splintage of fractures). *Note*: ketamine in low dose is increasingly being used to manage pain (especially prehospital) and where there is a concern about opioids and hypotension.

### Tetanus

Ensure tetanus prophylaxis in all patients (see ➲ Tetanus prophylaxis, p. 424).

### Antibiotics

Give prophylactic IV antibiotics for compound fractures and penetrating wounds of the head, chest, or abdomen. Antibiotic choice follows local policy—a broad-spectrum antibiotic (eg cefuroxime) is useful.

# Approach to trauma resuscitation

## Advanced Trauma Life Support (ATLS®)

The ATLS® concept was introduced by the American College of Surgeons in an attempt to improve the immediate treatment of patients with serious injury. The ATLS® (and now the European Trauma Course) approach enables standardization of trauma resuscitation. Treatment of all patients with major trauma passes through the same phases:

- Primary survey.
- Resuscitation phase.
- Secondary survey.
- Definitive care phase.

A key feature of ATLS® is frequent re-evaluation of the patient's problems and the response to treatment. Any deterioration necessitates a return to evaluate the 'ABC' (airway, breathing, and circulation).

Treatment is particularly designed to prevent trauma-induced coagulopathy, acidosis, and hypothermia—the so-called 'triad of death'.

## Primary survey

On initial reception of a seriously injured patient, identify and address life-threatening problems as rapidly as possible. Adopt a 'cABCDE' approach, quickly evaluating and treating:

- c—catastrophic haemorrhage control (pressure on external bleeding).
- A—airway maintenance, with cervical spine control.
- B—breathing and ventilation.
- C—circulation and haemorrhage control.
- D—disability (rapid assessment of neurological status).
- E—exposure (the patient is completely undressed to allow full examination).

With optimal staffing and direction, instead of considering each aspect sequentially ('A' to 'E'), aim for the team to address them simultaneously.

## Resuscitation phase

During this period, treatment continues for the problems identified during the primary survey. Further practical procedures (eg insertion of oro-/NG tube, chest drain, and urinary catheter) are performed. Occasionally, immediate surgery (damage control surgery) is required for haemorrhage control before the secondary survey.

## Secondary survey

A head-to-toe examination to identify other injuries should be accompanied by relevant imaging and other investigations. Monitor closely—any deterioration requires a repeat assessment. Adopting a high index of suspicion is essential to avoid missing occult injuries.

## Definitive care phase

The early management of all injuries is addressed, including fracture stabilization and emergency operative intervention.

# Investigations in major trauma

Select specific investigations according to the presentation, but most patients with major trauma require: group and save/cross-match, FBC, U&E, CT and/or X-rays, and VBG (including lactate and glucose levels).

### $SpO_2$

Attach a pulse oximeter on ED arrival, then monitor continuously.

### Blood tests

Check U&E, FBC, and glucose. If there is any possibility of significant haemorrhage, request a group and save or cross-match. Request a baseline clotting screen in patients with major haemorrhage or those at special risk (eg alcoholics or those on anticoagulants). Request FFP and platelets early for patients with major haemorrhage. Thromboelastography (TEG) and rotational thromboelastometry (ROTEM) may have a role in identifying trauma-induced coagulopathy.

### X-rays

The traditional approach of obtaining cervical spine, chest, and pelvis X-rays for blunt trauma has been largely replaced by CT.

### Urinalysis

Test the urine for blood if there is suspicion of abdominal injury. Microscopic haematuria is a potential marker of intra-abdominal injury.

### Arterial blood gas

ABG provides useful information about the degree of hypoxia, hypoventilation, and acidosis. In critically ill patients (especially those requiring ventilatory support or those destined for neurosurgery/ICU), repeat as necessary and consider inserting an intra-arterial line to continuously monitor BP.

### Electrocardiogram

Monitor all patients; record an ECG if >50y or significant chest trauma.

### Computed tomography scan

CT is the standard way to evaluate head, neck and truncal injuries—often in the form of a 'pan-scan' if there is injury to more than one body region. Co-location of the scanner within the ED helps issues of transfer and monitoring whilst scanning. Do not transfer patients with haemodynamic instability out of ED to a CT scanner.

### USS (FAST) and diagnostic peritoneal lavage

Focussed assessment with sonography for trauma (FAST) (see ➲ Focussed assessment with sonography for trauma (FAST) scan, p. 355) is an USS technique to identify free fluid (or gas) in the peritoneal, pleural, or pericardial cavities. It can be performed by a trained ED doctor, surgeon, or radiologist. Local policy and expertise will determine individual ED practice. Diagnostic peritoneal lavage (DPL) is rarely used now in developed countries, given the availability of FAST and CT. DPL still plays an important role if FAST and CT are not available.

### Other investigations

Angiography is indicated in certain specific circumstances (major pelvic fracture, aortic injury). Occasionally, other tests requiring specialist expertise (eg echocardiography) may prove to be useful.

## Trauma scoring

Trauma scoring is often used in research on the epidemiology and management of trauma. A basic understanding of the accepted system of trauma scoring may be of benefit to those treating injured patients.

### *Injury Severity Score*

The Injury Severity Score (ISS) is widely used to retrospectively score the anatomical injuries of an individual patient. The score is obtained by first scoring each individual injury using the Abbreviated Injury Scale (AIS), which attributes a score between 1 and 6 to each individual injury, as follows:

| | |
|---|---|
| AIS 1 = minor injury | AIS 4 = severe injury |
| AIS 2 = moderate injury | AIS 5 = critical injury |
| AIS 3 = serious injury | AIS 6 = inevitably fatal injury |

To calculate the ISS from an array of AIS scores for a patient, the three highest AIS scores in different body regions are squared, then added together. The ISS considers the body to comprise six regions: head/neck; face; chest; abdomen; extremities; and external (skin). Possible ISS scores range from 1 to 75. Any patient with an AIS = 6 is automatically given an ISS of 75. (See https://www.aast.org/Library/TraumaTools/InjuryScoringScales.aspx)

For example, see Table 8.1.

**Table 8.1** Example of scores of a patient after blunt trauma

| Injuries | AIS (body region) |
|---|---|
| Closed linear temporal skull fracture | AIS = 2 (head/neck) |
| Major aortic arch rupture at its root | AIS = 5 (chest) |
| Bilateral pulmonary contusions | AIS = 4 (chest) |
| Massive splenic rupture with hilar disruption | AIS = 5 (abdomen) |
| Multiple widespread superficial abrasions | AIS = 1 (external) |

ISS = $(5)^2 + (5)^2 + (2)^2 = 54$. The ISS is non-linear, and some scores (eg 15) are impossible. One accepted definition of 'major trauma' is an ISS of >15.

## The Revised Trauma Score

The Revised Trauma Score (RTS) is used to assess the physiological disturbance of a trauma patient. The score is calculated from the RR, systolic BP, and GCS. Each of these parameters are assigned a code (value) to which a weighting factor is applied. The three resultant scores are then added together to give the RTS. The RTS ranges from 0 (worst possible) to 7.84 (best).

### *TRISS methodology*

Combining the ISS with the RTS and adding a weighting factor according to the age of the patient, it is possible to calculate a 'probability of survival' (Ps) for each patient, based upon the national norm. Patients who survive with Ps of <0.5 are regarded as 'unexpected survivors', and patients who die with Ps of >0.5 as 'unexpected deaths'. By analysing the results of treating a large number of patients, the TRISS methodology may be used to compare 'performances' (eg of one hospital against the national norm).

# Airway obstruction: basic measures

Severely injured patients die rapidly unless oxygenated blood reaches the brain and other vital organs. Clear, maintain, and protect the airway; ensure that ventilation is adequate, and give $O_2$ as required. The most urgent priority is to clear an obstructed airway, but avoid causing or exacerbating any neck injury—instruct someone to hold the head and neck in a neutral position until the neck is satisfactorily immobilized.

When treating any seriously injured patient, always ensure that $O_2$, suction, and airway equipment are readily available. Get senior ED ICU/anaesthetic help early if a patient with a serious airway problem arrives or is expected.

## Causes of airway obstruction

- Coma from any cause can result in airway obstruction and loss of protective airway reflexes.
- Blood or vomit may block the airway.
- The airway may be disrupted by trauma of the face or larynx, or may be occluded by a haematoma or by oedema following burns.

## Assessment of airway obstruction

Talk to the patient and assess—a lucid reply shows the airway is patent, the patient is breathing, and some blood is reaching the brain. Do not move the neck until it has been checked and cleared of injury (see ➲ Major trauma: treatment principles, p. 330).

Look and listen to check how the patient is breathing. Complete airway obstruction in someone who is still trying to breathe results in paradoxical movements of the chest and abdomen, but no breath sounds. Gurgling, snoring, and stridor are signs of partial obstruction.

## Management of airway obstruction

- Look in the mouth and pharynx for FBs, blood, and vomit. The tip of a laryngoscope may be useful as an illuminated tongue depressor.
- Remove any FB with Magill's forceps, and suck out any liquid with a large rigid suction catheter. See if the patient responds and has a gag reflex, but beware of precipitating coughing or vomiting.
- If vomiting occurs, tilt the trolley head down and suck out any vomit.
- Lift the chin and use the jaw thrust manoeuvre (see ➲ Jaw thrust manoeuvre, p. 335) to open the airway, but do not flex or extend the neck.
- After any airway intervention, look, listen, and feel to reassess airway patency and efficacy of breathing.
- If the gag reflex is absent or poor, insert an *oropharyngeal airway* (see ➲ Insertion of oropharyngeal airway, p. 335). This helps to hold the tongue forward but can cause vomiting or coughing if there is a gag reflex. If the gag reflex is present or the patient's jaws are clenched, consider a *nasopharyngeal airway* (evidence of severe facial or head injury is a relative contraindication).
- If the airway is now patent and the patient is breathing, give high-concentration $O_2$ (15L/min via a non-rebreathing reservoir mask).
- If the airway is patent, but breathing inadequate, ventilate with an $O_2$ bag-and-mask device and prepare for tracheal intubation. Aim for one person to hold the mask on the face with both hands to ensure a good seal, whilst a second person squeezes the ventilation bag.

### Insertion of oropharyngeal airway

- Select the appropriate size of the airway.
- Hold an airway against the patient's face. Select the size based on the vertical distance between the incisors and the angle of the mandible. A large adult usually needs a size 4 airway; most men require a size 3, and some women need a size 2. An incorrectly sized airway may make the obstruction worse, rather than better.
- Open the patient's mouth and use a rigid suction catheter with high-power suction to suck out any fluid or blood from the oropharynx.
- Insert the oropharyngeal airway 'upside down' for 4–5cm (halfway), then rotate it 180° and insert it until the flange is at the teeth.
- In children, use a laryngoscope as a tongue depressor and insert the airway the 'correct way up' to avoid trauma to the palate.
- Recheck the airway and breathing and give high-flow $O_2$.
- Ventilate the patient if breathing is inadequate.

### Insertion of nasopharyngeal airway

- Select an appropriate airway, usually a 7.0mm for adult ♂ and a 6.0mm for adult ♀.
- Lubricate the airway and insert the tip of it into one nostril, then direct it posteriorly.
- The airway should slide easily into the nose until the flange abuts the nostril and the tip is just visible in the pharynx. Never force a nasopharyngeal airway into the nostril—any bleeding produced will markedly aggravate the airway problem.
- Recheck the airway and breathing, and give high-flow $O_2$.

### Jaw thrust manoeuvre

The aim of this is to open the upper airway with minimum movement of the cervical spine. Place the forefingers of both hands immediately behind the angles of the mandible and push the mandible anteriorly. This will lift the tongue anteriorly and thus away from the posterior pharyngeal wall.

### Tracheal intubation in trauma

An injured patient with no gag reflex needs intubation to maintain the airway and protect it against blood and vomit. Intubation may also be needed because of: apnoea (after initial ventilation with a bag–valve–mask), respiratory inadequacy, to prevent potential obstruction from facial burns, or to allow manipulation of ventilation in patients with ↑ ICP. Intubation in such circumstances requires emergency anaesthesia—suitable expertise, appropriate equipment, and assistance are essential (see ➲ Emergency anaesthesia and rapid sequence induction, pp. 322–3). An assistant holds the head to prevent neck movement during intubation, whilst another assistant provides cricoid pressure (as per local protocols).

Confirm correct tracheal tube placement by:

- Seeing the tube pass through the cords.
- Observing symmetrical chest movement.
- Listening over both axillae for symmetrical breath sounds.
- Confirming placement with end-tidal $CO_2$ monitoring.

If airway obstruction is complete, the obstruction cannot be relieved, and intubation is impossible, an urgent surgical airway is needed (see ➲ Airway obstruction: surgical airway, p. 336).

# Airway obstruction: surgical airway

A *surgical airway* is needed if the airway is obstructed by trauma, oedema, or infection and the trachea cannot be intubated. Emergency tracheostomy is not indicated in this situation because it is too time-consuming to perform and the necessary expertise may not be available.

## Surgical cricothyroidotomy

This technique (see Fig. 8.1) is not appropriate in children aged <12y.

- Feel the thyroid and cricoid cartilages and the cricothyroid membrane between them. If right-handed, stand on the patient's left.
- Clean the area and give LA (if the patient is conscious and time allows).
- Hold the thyroid cartilage with the non-dominant hand, and make a transverse incision through the skin and the cricothyroid membrane, then turn the blade through 90° with the sharp edge caudally. If unable to palpate the cricothyroid membrane, make a ~9cm vertical incision, then bluntly dissect with the fingers to the larynx.
- Slide a bougie alongside the blade into the trachea; remove the scapel, and railroad a lubricated 6.0mm cuffed tracheal tube into the trachea.
- Remove the bougie tube; inflate the cuff, and connect the tube to a catheter mount and ventilation bag.
- Confirm correct placement with end-tidal $CO_2$ monitoring.
- Ventilate the patient with $O_2$ and secure the tracheal tube.
- Examine the chest and check for adequacy of ventilation.

## Needle cricothyroidotomy

This is a rapid temporizing measure whilst preparation is made for a definitive airway (eg surgical cricothyroidotomy). Jet insufflation via a cannula placed through the cricothyroid membrane can provide up to 45min of oxygenation of a patient with partial airway obstruction (see Fig. 8.2).

- Use a large IV cannula-over-needle (adults: 12 or 14G; children: 16 or 18G) attached to a syringe. If right-handed, stand on the patient's left.
- Palpate the cricothyroid membrane between the thyroid and cricoid cartilages. Hold the cricoid cartilage firmly with the left hand.
- Pass the needle and cannula at a 45° angle to the skin in the midline through the lower half of the cricothyroid membrane into the trachea.
- Aspirate whilst advancing the needle. Aspiration of air confirms entry into the trachea. Withdraw the needle whilst advancing the cannula down into position in the trachea.
- Connect the cannula via a Y connector or $O_2$ tubing with a side hole to wall $O_2$ at 15L/min (in a child, the rate should initially be set in L/min at the child's age in years, ↑ if necessary until capable of causing chest movement). Hold the cannula firmly in position. Occlude the side hole or the end of the Y connector with the thumb for 1 in 5s to give intermittent insufflation of $O_2$.

Spontaneous breathing through the small airway of a cannula is very difficult, but the patient should be able to exhale partially in the 4s between jets of $O_2$. However, $CO_2$ retention occurs and limits the time that jet insufflation can be tolerated. Proceed immediately to a definitive airway (call a senior ENT or maxillofacial surgeon).

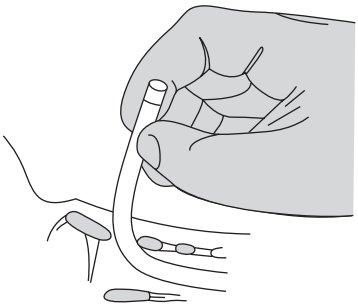

**Fig. 8.1** Surgical cricothyroidotomy.

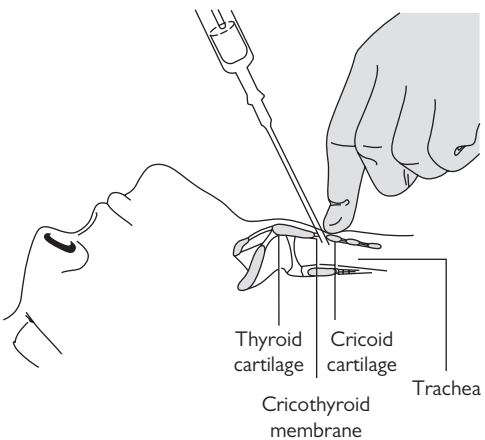


**Fig. 8.2** Needle cricothyroidotomy.

# Tension pneumothorax

Tension pneumothorax is a life-threatening emergency and requires prompt recognition and treatment. It occurs when gas progressively enters the pleural space but is unable to leave. ↑ pressure causes complete lung collapse on the affected side and ultimately pushes the mediastinum to the other side. Movement of the mediastinum leads to kinking of the great vessels, thereby ↓ venous return and cardiac output. Additional compromise results from compression of the lung on the other side, particularly in patients undergoing IPPV. The process leading to tension pneumothorax may occur very rapidly, culminating in cardiac arrest within minutes.

## Causes

Tension pneumothorax is seen most frequently following trauma, but it may also occur iatrogenically after attempted insertion of a central venous line (see ➲ Central venous access, pp. 58–9). A small (perhaps unsuspected) simple pneumothorax is particularly likely to become a tension pneumothorax when IPPV is commenced.

## Features

- Dyspnoea, tachypnoea, and acute respiratory distress.
- Absent breath sounds on the affected side.
- Hyper-resonance over the affected lung (difficult to demonstrate in a noisy environment).
- Distended neck veins (unless hypovolaemic), tachycardia, hypotension, and ultimately loss of consciousness.
- Trachea deviated away from the affected side (rarely clinically apparent).
- ↑ airway pressure in a patient receiving IPPV.

## Diagnosis

This is *essentially clinical*—do not waste time obtaining X-rays.

## Treatment

Apply high-flow $O_2$, then perform urgent decompression. Options are:

- If skills allow, perform finger thoracostomy decompression in the axilla in the fifth intercostal space, just anterior to the mid-axillary line, then follow this with a chest drain.
- Alternatively, immediately decompress by inserting an IV cannula (16G or larger) into the second intercostal space in the mid-clavicular line, just above the third rib (to avoid the neurovascular bundle; see Fig. 8.3). Withdraw the needle and listen for a hiss of gas. Tape the cannula to the chest wall, then insert an axillary chest drain on the affected side immediately (see ➲ Chest drain insertion, p. 346). Remove the cannula and apply an adhesive film dressing.

After decompression, check the patient and obtain a CXR.

*Note*: the risk of causing a pneumothorax by needle decompression in a patient who did not have one is ~10%. If the patient is very muscular or obese, consider using a longer cannula than normal (eg central venous line) to ensure that the pleural cavity is reached.

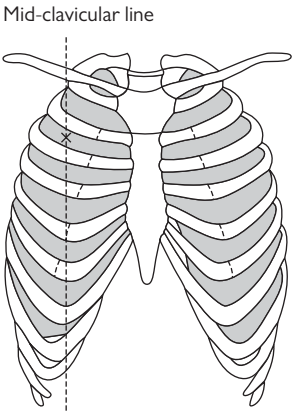


**Fig. 8.3** Site for needle decompression of right tension pneumothorax.

### Tension pneumothorax seen on X-ray or CT

Traditional teaching implies that tension pneumothorax is easy to diagnose on clinical grounds, and whilst this can be true, there are occasions when the diagnosis is less obvious—some of the signs are relatively subtle in a busy resuscitation room. Also, sometimes patients deteriorate whilst undergoing imaging, such that tension pneumothorax is first identified on a CXR or a CT scan. Once identified, decompress, according to the findings on the CXR or CT scan. Take particular care to ensure that the correct side is decompressed—there have been occasions when attention has been focussed on the wrong side (particularly with a pneumothorax seen on CT scan).

# Rib fractures

## Isolated rib fracture

A history of trauma with subsequent musculoskeletal pain suggests rib fracture. The diagnosis is confirmed by localized chest wall tenderness—the diagnosis of a single rib fracture is a clinical one. Check for features of pneumothorax (dyspnoea, ↓ air entry—see ➔ Traumatic pneumothorax, p. 344), secondary pneumonia, or multiple rib fractures, and if any is present, obtain a CXR.

*Treat* an uncomplicated isolated rib fracture with oral analgesia (eg co-codamol ± NSAID). Warn the patient that the rib may remain painful for ≥3 weeks and to seek medical advice if additional symptoms develop.

## Multiple rib fractures

Observe the chest wall carefully for a flail segment, and look for clinical evidence of pneumothorax or, in late presentations, pneumonia.

*Check* $SpO_2$ and ABG, and obtain a CXR. Note that up to 50% of rib fractures may not be apparent on CXR—adopt a low threshold for CT scan which may reveal the full extent of the chest injury.

*Treat* flail segment and pneumothorax as described in ➔ Flail segment, p. 342 and ➔ Traumatic pneumothorax, p. 344. Treat patients with uncomplicated multiple rib fractures according to the presence of other injuries and pre-existing medical problems as follows:

- In patients with other injuries requiring IPPV, discuss the potential need for chest drains with the ICU team (↑ risk of pneumothorax).
- Patients with pre-existing pulmonary disease and limited respiratory reserve require admission for analgesia and physiotherapy.
- Patients with chest infection often require admission for analgesia, monitoring, antibiotics, and physiotherapy, depending upon the past medical history and clinical and radiological findings.

## Chest wall injury pathways

Recognition of the morbidity and mortality associated with chest wall injury (especially in the elderly) has resulted in widespread adoption of chest wall injury pathways. These include a plan for management based on a scoring system—an example is shown in Table 8.2.

**Table 8.2** Chest wall injury score

| | |
|---|---|
| Age | +1 for each 10y aged >10y |
| Ribs | +3 for each fracture |
| Chronic lung disease | +5 if present |
| Anticoagulant or antiplatelet use | +4 (low-dose aspirin does not count) |
| $SpO_2$ | +2 for each 5% ↓ below 95% |

Scores can help to guide management:

- >10—requires admission.
- >20—refer to ICU/critical care.
- >30—consider referral to a regional major trauma centre.

# Sternal fracture

## Background

Fracture of the sternum frequently occurs during road traffic collisions, either due to impact against the steering wheel or seat belt. The injury may be associated with myocardial contusion, great vessel injury, and spinal injury.

## Features

Anterior chest pain with localized tenderness over the sternum may be accompanied by bruising and/or swelling.

## Investigations

- Place on a cardiac monitor.
- Record an ECG to exclude arrhythmias, MI (see ➲ ST segment elevation MI, pp. 74–5), or myocardial contusion (look for ST changes, particularly elevation). Consider further investigation with echocardiography.
- Check cardiac-specific enzymes (troponins) if there are ECG changes.
- Request CXR and lateral sternal X-ray—the latter will demonstrate the fracture (which is usually transverse), and the former associated injuries (see Fig. 8.4).

## Treatment

Provide analgesia and $O_2$, as required. Admit patients who have evidence of myocardial contusion or significant injuries elsewhere. Only consider discharging those patients who have an isolated sternal fracture, with a normal ECG, no associated injuries, and normal pre-existing cardiopulmonary function. Ensure that patients who are discharged receive oral analgesia (eg co-codamol ± NSAID) and GP follow-up, together with advice on when to seek urgent medical review if complications ensue.

*Note*: rarely, forced flexion of the chest causes a displaced sternal fracture, with wedge fractures of the upper thoracic vertebrae. Check the spine carefully; ask about pain, and look for kyphosis and tenderness (which may not be apparent). Lateral thoracic X-rays often fail to show injuries to the upper thoracic vertebrae, so if injury is suspected at this site, consider requesting a CT scan.

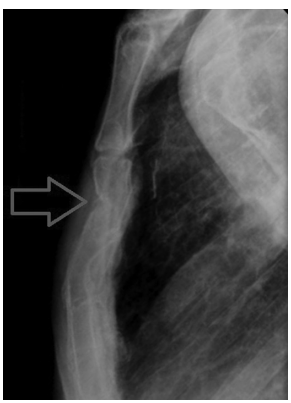

**Fig. 8.4** Fracture of the sternum in a 90y old man.

# Flail segment

Fracture of ≥3 ribs in two places allows part of the chest wall to move independently. This flail segment usually indicates significant injury to the underlying lung (typically pulmonary contusions). Large flail segments occur laterally when several ribs on one side fracture anteriorly and posteriorly. Similarly, an anterior flail segment is produced by bilateral fractures of all ribs anteriorly—in this case, the free portion comprises the sternum, the costal cartilages, and the medial parts of the fractured ribs (see Fig. 8.5).

## Presentation

The flail segment causes pain and moves paradoxically, compared with the rest of the chest wall, limiting the effectiveness of respiration. The diagnosis is a clinical one, but it can be difficult to make. Look tangentially at the chest for areas which move paradoxically (ie inward during inspiration and outward during expiration). There may be associated features of respiratory distress (cyanosis, tachypnoea). Check for pneumothorax or haemothorax (see ➲ Traumatic pneumothorax, p. 344).

## Investigations

Assess for respiratory compromise clinically, together with some tests:

- $SpO_2$ on pulse oximetry.
- ABG—the combination of hypoxia and respiratory acidosis (↑ $pCO_2$, ↑ $H^+$) indicates severe respiratory compromise.
- CT is more useful than CXR in demonstrating fractures and other injuries (eg pulmonary contusions, pneumothorax, haemothorax).

## Treatment

- Provide high-flow $O_2$, and treat associated life-threatening problems.
- Contact the ICU/anaesthesia team, and carefully consider the need for immediate or urgent tracheal intubation with IPPV.
- Carefully observe and monitor in an HDU or ICU.
- Prescribe regular oral analgesia. Selected patients may benefit from patient-controlled analgesia, an epidural, or regional anaesthesia.

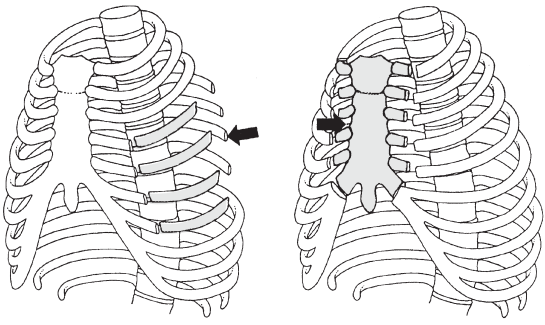


**Fig. 8.5** Lateral (left) and anterior (right) flail segments.

# Ruptured diaphragm

Left-sided ruptures predominate (75%).

### Major diaphragmatic rupture

Usually with associated herniation of abdominal contents into the chest, this is a severe injury resulting from a significant traumatic insult (often massive abdominal crushing). Depending upon the extent of the injuries, the patient may present hypoxic, with hypovolaemic shock and respiratory compromise. Note that a ruptured diaphragm may have some clinical features similar to those of a tension pneumothorax. Call a surgeon and an anaesthetist, as the patient will require urgent intubation and IPPV.

### Minor diaphragmatic rupture

Less dramatic herniation may present in a more subtle fashion and result from a penetrating injury. The diagnosis is difficult to identify on CT scanning and is frequently missed initially. However, it is important because:

- It is often associated with injury to both abdominal and thoracic contents.
- There are possible late complications (eg bowel herniation/obstruction).
- It does not heal spontaneously.

Suspect a ruptured diaphragm from the mechanism of injury and an abnormal or a high hemidiaphragm contour on erect CXR. Look for stomach or bowel loops in the chest—the gastric tube may be seen coiled in the intra-thoracic stomach. If a ruptured diaphragm is suspected, resuscitate and refer to a surgeon.

# Oesophageal rupture

Traumatic (non-iatrogenic) rupture of the oesophagus is uncommon but may follow a blunt or penetrating injury. Suspect it if the patient complains of chest and back/neck pain in the presence of a normal ECG. Check for surgical emphysema in the neck. Imaging (CXR or CT) may demonstrate pneumomediastinum (a layer of gas around the heart/mediastinum), a left-sided pleural effusion, or pneumothorax. Provide $O_2$ and IV analgesia, and start IV antibiotics (eg cefuroxime 1.5g). Resuscitate; treat other injuries, and refer to a cardiothoracic surgeon.

### Boerhaave's syndrome

'Spontaneous' rupture of the oesophagus is associated with overindulgence and vomiting. Patients are classically middle-aged and present with severe chest pain, signs of shock, and subcutaneous emphysema. CXR or (better) CT will confirm the diagnosis. If suspected, treat as outlined above for traumatic oesophageal rupture.

# Traumatic pneumothorax

## Background

Pneumothorax frequently results from a blunt injury, with associated rib fractures, or from a penetrating injury (knife stabbing or gunshot wound). It may also be iatrogenic, secondary to attempted insertion of a central venous line.

## Clinical features

Patients are likely to complain of symptoms relating to the associated injuries (eg rib fractures—see ➲ Rib fractures, p. 340). The degree of breathlessness resulting from a pneumothorax depends largely upon its size. Other features may be present, including surgical emphysema, cyanosis, and ↓ air entry over the affected lung. Severe dyspnoea and distended neck veins/hypotension suggest tension pneumothorax (see ➲ Tension pneumothorax, pp. 338–9). $SpO_2$ and ABG may reveal hypoxia.

*CXR* demonstrates the pneumothorax. Both inspiratory and expiratory X-rays are not required. Wherever possible, obtain an erect CXR. X-rays taken with the patient lying supine may not show a free lung edge, despite a considerable pneumothorax, because in this position, air tends to lie anteriorly in the pleural space (see Fig. 8.7). If there is no definite pneumothorax visible on a supine CXR, features which are suggestive of a pneumothorax are:

- Hyperinflation of the affected hemithorax, with a depressed hemidiaphragm.
- Double contour of a hemidiaphragm.
- Basal hyperlucency of the affected lung.
- Visualization of apical pericardial fat tags.

A *CT scan* obtained to assess other injuries will easily demonstrate a pneumothorax. Not infrequently, small pneumothoraces that are not apparent on a CXR are clearly apparent on CT.

*Point-of-care USS* is also good at identifying pneumothoraces.

## Treatment

Tension pneumothorax is an emergency requiring immediate needle decompression (see ➲ Tension pneumothorax, pp. 338–9) or thoracostomy. Provide $O_2$ and drain significant traumatic pneumothoraces using a chest drain and an open technique, as described in ➲ Chest drain insertion, p. 346.

There is ↑ experience with initially managing some patients who have an isolated chest injury and small traumatic pneumothoraces in a conservative fashion, using close observation and no chest drain. Patients who have multiple injuries and/or other injuries (particularly those requiring GA and IPPV) certainly require chest drain insertion in the ED.

# Haemothorax

Blood may collect in the pleural cavity, in association with a pneumothorax (haemopneumothorax) or without (haemothorax). A large amount of bleeding into the pleural space sufficient to produce hypovolaemic shock is termed *massive haemothorax*.

## Clinical features

The clinical presentation is similar to that seen in traumatic pneumothorax, except that there may be dullness to percussion over the affected lung and, with massive haemothorax, evidence of hypovolaemia.

*CXR* Blood from a haemothorax collects under the affected lung, showing up as ↑ shadowing on a supine X-ray, with no visible fluid level. It may be very difficult to distinguish a haemothorax from pulmonary contusions on a supine X-ray, but a haemothorax may produce blurring of the hemidiaphragm contour or of the costophrenic angles.

*CT scan* Will easily define a haemothorax and associated injuries (see Fig. 8.8).

## Treatment

Give $O_2$ and insert two large venous cannulae (sending blood for cross-matching). If hypovolaemic, give blood before inserting a large (≥32FG) chest drain. Although it is common practice to try to direct the chest drain towards the diaphragm, this seldom makes a difference in practice—it is more important to use a chest tube of sufficient calibre in order to minimize blockages due to blood clots.

# Chest drain insertion

Use the 'open' technique, as described below. Explain the procedure; obtain consent, and confirm that the patient has venous access, is breathing $O_2$, and is fully monitored. Ensure that all equipment is ready and a good light and assistance are available. Give adequate IV opioid analgesia to conscious patients, as this procedure can be painful.

- Abduct the ipsilateral arm fully.
- Don a sterile gown and gloves, plus goggles/face shield.
- Clean the skin with antiseptic and cover with sterile drapes.
- Identify the fifth intercostal space just anterior to the mid-axillary line (count down and across from the angle of Louis at the level of the second rib) (see Fig. 8.6, top).
- Generously infiltrate LA (1% lidocaine ± adrenaline) under the skin and down to the periosteum at the upper edge of the sixth rib.
- Prepare the chest drain; remove and discard the trocar (in adults, use size 28–32FG; in children, use the largest size that will comfortably pass between the ribs).
- Make a 2–3cm skin incision in the line of the ribs (see Fig. 8.6).
- Use blunt dissection with artery forceps to open the tissues down to the pleural space, just above the sixth rib.
- Puncture the pleura with the artery forceps.
- Taking care to avoid a finger injury from rib fractures, insert a gloved index finger into the pleural cavity to ensure there are no adhesions and that you are within the thoracic cavity (see Fig. 8.6).
- Insert the chest drain, ensuring that all drainage holes are inside the chest (typically ~15–20cm in adults).
- Connect the drain to an underwater seal and look for 'swinging'.
- Suture the drain securely in place (eg with heavy silk), and cover with an adhesive film dressing and adhesive tape (see Fig. 8.6). Whilst securing it, get an assistant to hold the drain, so that it does not inadvertently fall out. It is useful to insert two untied sutures at the site of exit of the chest drain, so that these can be later tied to close the exit site when the drain is removed.
- Check the underwater seal is 'swinging' in the tube with respiration.
- Listen for air entry and check the patient.
- Obtain a CXR to confirm placement—if the tube has been inserted too far (eg so that it is touching the mediastinum), pull it back slightly and re-suture and secure in place.
- Afterwards, keep the water seal drainage bottle below the level of the patient. Avoid clamping the tube.

*Referral to a thoracic surgeon* If the chest drain initially yields >1500mL of blood or subsequently drains >200mL/hr for 2hr, refer urgently to a thoracic surgeon for a possible urgent thoracotomy.

*Ruptured bronchus* Persistent, continuing bubbling of gas through the underwater drain may reflect a major rupture of the tracheobronchial tree, especially if the lung fails to re-expand. Bronchial rupture may also present with haemoptysis or tension pneumothorax. Involve a thoracic surgeon at an early stage.

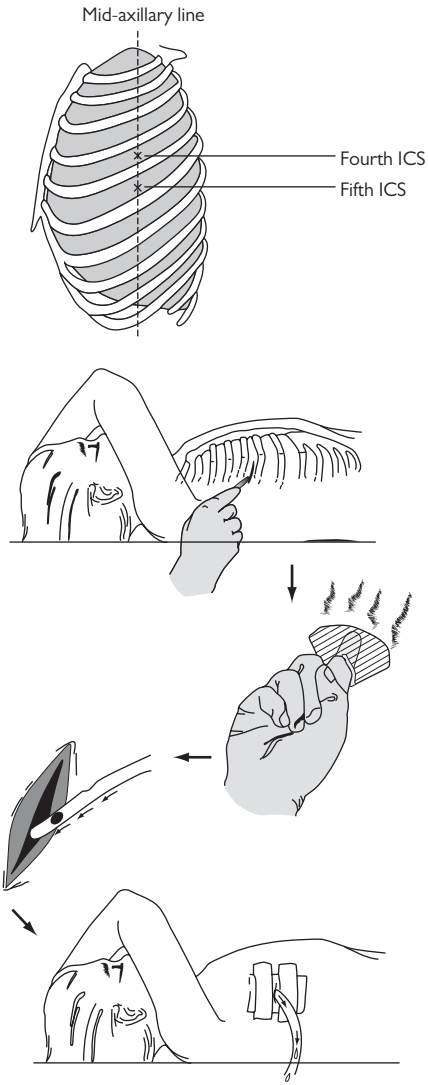


**Fig. 8.6** Chest drain insertion.

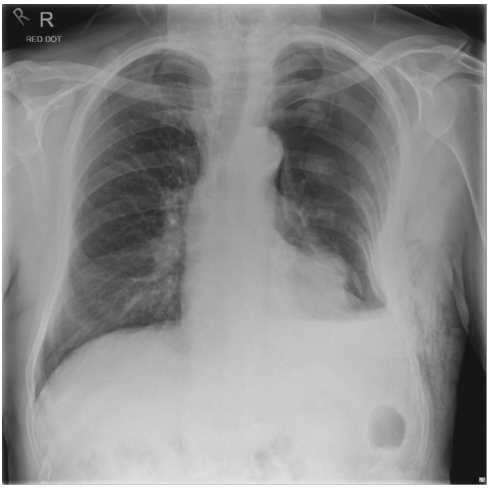


**Fig. 8.7** CXR in a patient with blunt chest trauma.

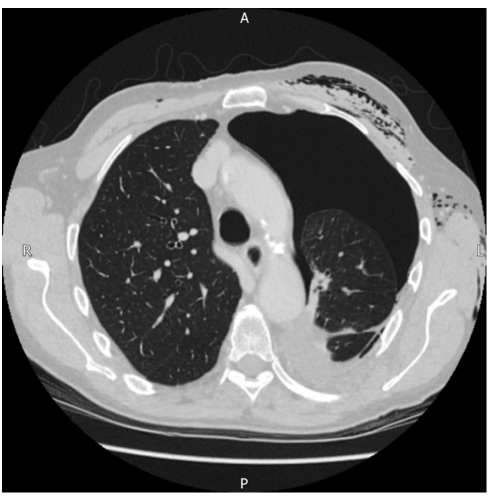


**Fig. 8.8** CT in the same patient shown in Fig. 8.7—blunt chest trauma, comprising rib fractures, subcutaneous emphysema, a left pneumothorax, and a small haemothorax.

# Pulmonary contusions and aspiration

## Pulmonary contusions

High-energy transfer during blunt injury (eg road traffic collisions or high falls) often causes pulmonary contusions. Suspect these in all patients with flail segments (see ➲ Flail segment, p. 342).

*Clinical features*

Pulmonary contusions produce V/Q mismatch, which may lead to hypoxia and respiratory distress, and ↑ the likelihood of ARDS.

*Radiological appearances*

Pulmonary contusions may be visible on initial CXR as patchy opacification. However, initial radiological appearances are non-specific and may be confused with those seen after pulmonary aspiration or haemothorax (see ➲ Haemothorax, p. 345). X-ray changes resulting from pulmonary contusions tend to be progressive and become more prominent with time.

*Management*

Provide high-flow $O_2$, and check ABG to help assess the need for GA, tracheal intubation, and IPPV. Involve ICU specialists early.

## Pulmonary aspiration

Inhalation of vomit and other foreign material may add considerably to the damage resulting from the initial injury (see ➲ Pulmonary aspiration, p. 116–17).

*Common associations*

- Inhalation of vomit after head injury with ↓ conscious level and impaired protective laryngeal reflexes—gastric contents are particularly irritant to the respiratory tract.
- Inhalation of blood and teeth after facial trauma.
- Inhalation of water and foreign matter in near drowning (see ➲ Drowing and near drowning, pp. 268–9).

*Presentation*

Suspect pulmonary aspiration from the history, associated respiratory signs, and the X-ray appearance. The CXR may show diffuse opacification affecting one or both lungs—the distribution depends upon the position at the time of aspiration.

*Management*

- Check $SpO_2$ and ABG, and obtain a CXR.
- Provide high-flow $O_2$ as required.
- Treat other injuries.
- Consider the need for GA, tracheal intubation, and IPPV. Bronchoscopy may be needed to remove large FBs from within the bronchial tree.
- Even if there is no urgent requirement for IPPV, remember that the respiratory problem is likely to worsen (with the development of infection/ARDS), so involve the ICU team early.
- Do not give routine antibiotics, unless there is a specific indication, such as immersion in sewage or in rat-infested water with the risk of developing leptospirosis (see ➲ Leptospirosis (Weil's disease), p. 249).

# Approach to penetrating chest injury

## Initial assessment and resuscitation

Do not be misled by seemingly innocuous wounds. The size of the external wound has no correlation with the potential for internal injury. Give $O_2$ if $SpO_2$ is low. Gain venous access (send blood for cross-matching or group and save) and resuscitate according to an evaluation of ABCDE. Remove the patient's clothes and log roll to check for wounds to the back and perineum. Particularly in gunshot injuries, perform an early check for evidence of spinal cord injury. Remember that a penetrating chest injury often involves the abdomen (and vice versa). During the initial assessment, aim to exclude or identify and treat:

- Tension pneumothorax (see ➲ Tension pneumothorax, pp. 338–9).
- Sucking chest wound (see ➲ Thoracotomy for cardiac arrest, p. 353).
- Cardiac tamponade (see ➲ Thoracotomy for cardiac arrest, p. 353).
- Massive haemothorax (see ➲ Traumatic pneumothorax, p. 344 and ➲ Haemothorax, p. 345).

Further management depends partially upon the haemodynamic status.

## Haemodynamically stable patient

Many patients present without overt evidence of significant injury:

- Provide $O_2$ and secure venous access, and send blood for group and save.
- Monitor $SpO_2$, pulse, BP, and RR.
- Administer minimal or no IV fluids.
- Perform a FAST scan, if possible (see ➲ Focussed assessment with sonography for trauma (FAST) scan, p. 355).
- Obtain a CT chest and abdomen or a CXR (ideally PA erect).
- Record an ECG.
- Provide IV analgesia as required (see ➲ Major trauma: treatment principles, p. 330).
- Consider tetanus status and the need for prophylactic antibiotics (eg cefuroxime 1.5g IV—according to local policy).
- Cover the chest wound with a sterile dressing.
- Drain any pneumothorax with a chest drain (having decompressed any tension pneumothorax—see ➲ Tension pneumothorax, p. 338–9). *Do not* insert the drain through the wound (this ↑ the risk of infection).
- Refer all patients for admission, observation, formal wound cleaning, exploration, and closure. If the patient remains stable overnight, with no clinical or radiological abnormalities, they may be safely discharged with arrangements for review.
- Document the size, position, and other features of the chest wound, remembering the potential medicolegal significance (see ➲ The approach to wounds, p. 410).

### The unstable patient

Haemodynamic instability may be due to tension pneumothorax, massive haemothorax, sucking chest wound, or cardiac tamponade. Treat each of these, as outlined in ➔ Thoracotomy for cardiac arrest, p. 353, involving a senior surgeon at an early stage.

*Indications for thoracotomy*

Thoracotomy in theatre will be required for significant haemorrhage, which typically means:

- >1.5L of free blood obtained by initial chest drainage, or
- >200mL of blood draining per hour via a chest drain.

## Open chest injury

An open wound between the pleural cavity and the outside may cause respiratory insufficiency. When the chest expands on inspiration, there is less resistance to air movement through the open chest wound than down the tracheobronchial tree. Air flow into the lungs is reduced, and the lung collapses as air enters the pleural space and produces a pneumothorax. Hypoxia develops rapidly.

### Features

Look for respiratory distress, tachypnoea, and cyanosis.

### Management

- Provide high-flow $O_2$.
- Ideally, cover with a one-way adhesive chest seal. If not available, it is traditional to occlude with a dressing on three sides (although the evidence for this is lacking), or alternatively cover with an occlusive dressing and insert an immediate chest drain. If signs of deterioration occur, consider a possible tension pneumothorax, in which case remove the dressing.
- Insert a chest drain (not through the wound) to drain the pneumothorax.
- Provide further resuscitation as necessary.
- Call a thoracic surgeon to arrange formal wound closure.

# Traumatic cardiac arrest

## Background

Cardiac arrest following trauma generally carries a poor prognosis, although there is a chance of recovery (especially following penetrating trauma), depending upon the exact situation. First, try to determine whether the cardiac arrest is likely to be due to the injurious event itself or to an underlying medical condition which subsequently has resulted in injuries.

## Action if cardiac arrest is likely due to the injury

If there is a high index of suspicion of a traumatic aetiology to the arrest, consider the possible likely reversible causes (usually associated with PEA). These are:

- Hypoxia: secure the airway with a tracheal tube and ventilate with $O_2$.
- Hypovolaemia: give blood and plasma (eg 4U of O-negative packed red cells stat—warmed via a blood warmer).
- Tension pneumothorax: perform immediate bilateral finger thoracostomies.
- Cardiac tamponade: if the above fail and/or fluid is suspected or seen on eFAST, perform a clam shell thoracotomy. Pericardiocentesis is often unsuccessful due to clots which have formed in the pericardial sac and which cannot be aspirated.

Do not perform chest compressions, as these are not likely to improve outcome, given the underlying aetiology. Remember also that sometimes peripheral pulses cannot be felt in very low output states. In the presence of some injuries, such as multiple displaced rib fractures, chest compressions carry the risk of causing further injury.

Similarly, avoid adrenaline as there is no evidence of a benefit for it in traumatic cardiac arrest.

## Return of spontaneous circulation after traumatic arrest

If there is ROSC, continue resuscitation and employ appropriate imaging (eg abdominal USS, CXR, pelvic X-ray, CT) to establish the exact nature of the underlying injuries and to guide management such as damage control surgery and interventional radiology.

# Thoracotomy for cardiac arrest

## Indications for resuscitative thoracotomy

Emergency thoracotomy can be performed in order to relieve cardiac tamponade, perform haemostasis of lung injury, compress the thoracic aorta if there is massive haemorrhage below the diaphragm, and perform internal cardiac massage.

Consider thoracotomy in the ED if there is refractory hypotension despite vigorous resuscitation or failure to regain output despite treatment of hypoxia, blood replacement, and bilateral thoracostomies. It is generally stated that thoracotomy can be considered after penetrating trauma with <15min of CPR and after blunt trauma with <10min of CPR.

## Procedure

- Summon expert help (ED consultant; cardiothoracic, general, or trauma surgeon; anaesthetist) and proceed immediately. Do not wait.
- Whilst the thoracotomy tray is being opened, don gloves, a face shield, and an apron; ensure that the patient is being ventilated with $O_2$ via a tracheal tube. Continue rapid IV blood via multiple lines, and obtain blood for transfusion.
- Perform bilateral thoracostomies.
- Connect the thoracostomies with an incision using a scalpel.
- Join the thoracostomies by cutting through the intercostal muscles using strong scissors (eg Tuff Cut®), then cut horizontally through the sternum using scissors or a Gigli saw.
- Open the chest with rib spreaders or ask a member of staff to pull the chest open.
- Open the pericardium with scissors (cut vertically and take care to avoid the left phrenic nerve).
- Remove any tamponade.
- Manage myocardial wounds by finger pressure or with a Foley catheter or a non-absorbable suture (eg interrupted 4/0 Prolene sutures, using Teflon or pericardial buttresses if necessary). Once sutures are in place, stop internal cardiac massage and check the cardiac rhythm and output. If the heart is fibrillating, defibrillate using internal defibrillation paddles, by placing a paddle over each side of the heart. Start with 5J energy initially, ↑ as necessary to a maximum of 50J. Use an external defibrillator if no internal paddles are available.
- Consider controlling massive pulmonary haemorrhage by collapsing the lung (eg using an incontinence pad).
- If massive bleeding below the diaphragm is suspected, compress the aorta using a fist.
- If internal cardiac compression is required, provide this using a bimanual technique, by compressing the heart between two flat hands, with the fingers placed over the defects.
- Once a pulse has been restored, ensure that hypovolaemia is corrected. Give cefuroxime 1.5g IV; insert an arterial line and a urinary catheter, and recheck U&E, glucose, FBC, and clotting.
- The surgical team will direct further surgical management.

# Aortic injury

The vast majority of aortic injuries (~90%) are sustained during high-energy blunt trauma (eg road traffic collisions, high falls). Only a small proportion of these patients reach hospital with signs of life. The usual site of rupture is just distal to the origin of the left subclavian artery, possibly caused by differential shearing forces between the mobile arch and the fixed descending thoracic aorta. An alternative proposed mechanism is that during rapid deceleration, the first rib and clavicle swing down and directly 'nip' the aorta (the 'osseous pinch' theory). The injury is relatively unusual in children, who are perhaps protected by having more elastic tissues.

## Features

Patients who reach hospital alive are most likely to have a partial or a contained rupture, with a haematoma confined by the aortic adventitia. They may complain of chest and back pain, and there may be a harsh systolic murmur, absent or ↓ pulses (with differential BP between the arms and legs), and evidence of hypovolaemic shock—features of other significant non-aortic injuries may predominate.

## Diagnosis

The diagnosis of aortic injury can be difficult—adopt a high index of suspicion. If there is suspicion of an aortic injury, obtain a CT scan. CXR features can be quite subtle.

*CXR features suggesting aortic injury*

- Widened mediastinum (>8cm on PA film).
- Abnormal aortic arch contour.
- Deviation of the trachea to the right side.
- Deviation of an orogastric/NG tube to the right side (such that it lies to the right of the T4 spinous process).
- Depression of the left main bronchus >40° below the horizontal.
- Left pleural cap or fractured first/second ribs are often quoted but are of little diagnostic value.
- The CXR may be normal!

## Management

Resuscitate and treat other injuries. Aortic injuries are associated with other severe chest injuries, eg flail segments, pulmonary contusions. As a minimum, provide $O_2$, insert two IV cannulae, start IV fluids, provide analgesia, and monitor vital signs and $SpO_2$. Check U&E, glucose, FBC, clotting, ABG, and cross-match. Insert a urinary catheter and an arterial line.

Involve a cardiothoracic surgeon or a vascular surgeon with expertise in aortic injury. Refer urgently for specialist investigation (CT and/or aortography). Involve an anaesthetist/ICU. Control BP (avoid over-infusion of IV fluids; use GTN IVI to maintain systolic BP (~90mmHg) prior to treatment. This may involve open surgical repair or an endovascular stent graft.

# Focussed assessment with sonography for trauma (FAST) scan

This is used in the ED resuscitation room to assess the chest and abdomen of acutely injured patients, especially those with shock. Do not perform FAST scanning before immediate CT or to determine the need for CT.

This can be performed by a trained ED doctor, surgeon, or radiologist.

## Advantages

- Can be done in the ED.
- Quick: takes 2–3min.
- Non-invasive.
- Repeatable if concerns persist or the patient's condition changes.

## Disadvantages

- Operator-dependent.
- Does not define the injured organ, but only the presence of blood or fluid in the abdomen or pericardium.

Ideally performed with a portable or hand-held USS scanner.
Looks at four areas for the presence of free fluid only:

- Hepatorenal recess (Morrison's pouch).
- Splenorenal recess.
- Pelvis (pouch of Douglas).
- Pericardium.

The scan is usually done in that order, as the hepatorenal recess is the first to fill with fluid in the supine position and is most easily identified.

If the indication for FAST scanning is to identify cardiac tamponade, the first view should be the pericardial view.
Free fluid appears as a black echo-free area:

- Between the liver and the right kidney.
- Between the spleen and the left kidney.
- Behind the bladder in the pelvis.
- Around the heart in the pericardium.

## A positive FAST scan

- One which identifies any free fluid in the abdomen or in the pericardium.
- Visible free fluid in the abdomen implies a minimum volume of ~500mL.

The finding of blood in the pericardium after trauma is an indication for an emergency thoracotomy, ideally in the operating theatre; however, perform this in the ED if the patient arrests.

Note that FAST scanning is very sensitive at detecting a pneumothorax. FAST scanning requires training prior to use on trauma patients; there is a significant false-negative rate in inexperienced hands.

# Blunt abdominal trauma

Blunt injury to the abdomen may be isolated or associated with injuries elsewhere. Evaluation of the abdomen may be particularly difficult in the latter situation. The mechanisms of injury responsible are diverse and include road traffic collisions, crushing injuries, high falls, and direct blows (eg kicks and punches). Remember that a lower chest injury may be associated with splenic or liver injuries.

## Examination

- *Assess* for hypovolaemia. Check pulse, BP, and capillary refill.
- *Look* for bruising (eg 'lap belt' imprint). (Measurements of the abdominal girth are unhelpful and unreliable as a means of assessing intra-abdominal haemorrhage.)
- *Feel* for tenderness and evidence of peritonism. Listening for bowel sounds is not helpful—their presence or absence is not discriminatory.
- *Check* for femoral pulses.
- *Log roll* to check for loin tenderness and back injury, but do not allow this to delay CT—defer until after the scan.
- *Examine the perineum* and consider a *rectal examination*, checking perineal sensation, anal tone, rectal integrity/blood, and in the ♂, the position of the prostate. A high-riding, 'boggy' or impalpable prostate may indicate urethral injury (see ➲ Urethral trauma, p. 360).

## Investigations

The choice of investigation depends upon individual circumstances, local policy, facilities, and expertise. Patients who are haemodynamically unstable or who have peritonism need immediate referral/laparotomy.

*Perform urinalysis* in all patients. A positive urinalysis is a marker for an intra-abdominal solid organ injury, not just a renal tract injury. Insert a urinary catheter in patients who present with haemodynamic disturbance or who are critically ill (unless there is evidence of urethral injury—see ➲ Urethral trauma, p. 360). Perform a pregnancy test in all women of child-bearing age.

*Serum amylase* does not discriminate between those with significant intra-abdominal injury and those without: it is unhelpful in the early stages of trauma resuscitation.

*Plain abdominal X-ray* is traditional, but rarely useful.

*FAST (USS)* provides a rapid, repeatable, non-invasive bedside test. It is operator-dependent. Haemoperitoneum is identified by scanning the hepatorenal and splenorenal recesses and the pelvis. The pericardium can also be scanned to look for tamponade (see ➲ Focussed assessment with sonography for trauma (FAST) scan, p. 355).

*CT scans* are extensively used to evaluate abdominal injuries, as well as identify injuries in other regions (eg retroperitoneum, brain, chest). The major advantage of CT is the ability to diagnose the injured organ(s) within the abdomen and to quantify injuries (minor laceration of the liver or spleen vs multiple deep lacerations with significant haemoperitoneum) (see Fig. 8.9). CT can help to guide management, particularly interventional radiology.

### Initial stabilization

(See ➔ Major trauma: treatment principles, p. 330.)

- Provide $O_2$.
- Treat airway and breathing problems.
- Insert two wide-bore (>16G) IV lines.
- Send blood for U&E, glucose, FBC, clotting screen, and cross-matching.
- Give blood as required, according to initial evidence of hypovolaemia and response to treatment.
- Provide IV analgesia as necessary (contrary to popular opinion, this does not compromise clinical abdominal evaluation).
- Consider the need for an orogastric/NG tube and a urinary catheter.
- Involve a surgeon at an early stage.
- Inform the senior surgeon, duty anaesthetist, and theatre staff if an urgent laparotomy is needed.

### Further evaluation and treatment

Once resuscitation is under way, tailor treatment according to the clinical situation.

*Haemodynamically unstable* Refer urgently to a senior surgeon for laparotomy. Inform the operating theatre and the duty anaesthetist immediately. There may be no need (or time) to attempt to define the intra-abdominal injury. Encourage damage control surgery to be urgently considered in unstable, acidotic, or cold patients.

*Clinical peritonism* Resuscitate as above; provide IV antibiotics (eg cefuroxime 1.5g) and refer urgently to a surgeon for laparotomy.

*Haemodynamically stable, no peritonism* Refer to a surgeon for further investigation and observation. FAST (USS) and abdominal CT scans are very useful in further assessment of these patients. Depending on local policy, others may be appropriately managed with regular observations and clinical re-examination.

*Possible abdominal injury in the multiply injured* These patients provide a diagnostic challenge—tailor investigations and management to individual circumstances. FAST (USS) is a rapid, simple, and useful tool to help to identify significant intra-abdominal haemorrhage in the multiply injured patient. CT has superior diagnostic accuracy, but it takes time and requires IV contrast and usually an internal transfer within the hospital. If the patient is haemodynamically stable, aim to perform a 'pan-scan' CT as soon as possible. The pan-scan CT will typically include the head, neck, chest, abdomen, pelvis, spine, and femoral shafts. The decision of whether or when to perform a CT scan on haemodynamically unstable patients needs to be taken by senior members of the trauma resuscitation team and will be shaped partly by expertise, resources, and transfer time to the CT scanner.

*Abdominal trauma in pregnancy* Involve a senior obstetrician and a gynaecologist at an early stage. USS can demonstrate fetal viability and look for signs of abruption and uterine rupture. Remember to check the rhesus/antibody status (see ➔ Trauma in pregnancy, pp. 612–13).

# Penetrating abdominal trauma

Most penetrating abdominal injuries are caused by knives or guns. The size of the external wound bears little relationship to the severity of intra-abdominal injuries. These injuries have medicolegal implications (see ➲ Interpersonal violence—medicolegal implications, p. 411).

## Initial approach

On receiving the patient, provide $O_2$; secure venous access, and resuscitate according to an initial assessment of ABCDE.

Obtain complete exposure at an early stage in order to check for additional wounds to the chest, back, loins, buttocks, and perineum.

## Evaluation of abdominal injury

Unless the patient presents with hypovolaemic shock, it may be difficult to decide the extent and severity of the abdominal injury on clinical grounds. In addition to standard monitoring and palpation of the abdomen, perform a digital rectal examination, and (especially in gunshot injuries) check carefully for spinal cord/cauda equina injury (see ➲ Approach to possible spinal injury, pp. 388–9).

## Investigations

*Urinalysis* Check the urine for blood.

*Blood* Check BMG, U&E, glucose, FBC, clotting, and group and save/cross-match.

*CT scan* Obtain a CT scan if the haemodynamic status of the patient allows. X-rays are much less useful—a CXR can show free gas under the diaphragm, and a supine abdominal X-ray can identify bullet fragments, etc.

*FAST (USS)* FAST scanning will rapidly identify the presence of free intra-abdominal fluid (see ➲ Focussed assessment with sonography for trauma (FAST) scan, p. 355).

## Management

- Give $O_2$: insert two IV cannulae, and send blood as outlined previously.
- In the unstable patient, give blood as necessary, but avoid excessive IV fluids—aggressive infusion worsens outcome. A systolic BP of ~90mmHg in a conscious patient is enough until the start of surgery.
- Provide IV analgesia (eg titrated increments of morphine) as required.
- Give IV antibiotics (eg cefuroxime 1.5g + metronidazole 500mg).
- Consider the need for tetanus prophylaxis (see ➲ Tetanus prophylaxis, p. 424).
- Cover the wound with a sterile dressing. Never probe or explore the wound in the ED to try and define the depth and possible peritoneal penetration. Involve the surgeon early to decide further management.
- Patients who are haemodynamically unstable, have gunshot wounds, or have obvious protruding bowel contents require urgent resuscitation and laparotomy at an early stage. Cover protruding omentum or bowel with saline soaked sterile swabs, but do not push it back into the abdomen.

# Renal trauma

Most renal injuries result from direct blunt abdominal trauma, the kidney being crushed against the paravertebral muscles or between the twelfth rib and the spine. Indirect trauma (eg a fall from a height) can tear the major blood vessels at the renal pedicle or rupture the ureter at the pelviureteric junction. Penetrating injuries are relatively rare. Many patients with renal trauma also have other important injuries, which may obscure the diagnosis of the renal injury.

*Children* are particularly prone to renal injuries. Trauma may uncover congenital abnormalities, hydronephrosis, or occasionally incidental tumours.

## Clinical features

Most patients provide a history of a blow to the loin or flank and have loin pain followed by haematuria (which may be delayed). The loin is tender and there may be visible bruising or abrasions. Worsening renal pain may indicate progressive renal ischaemia. Perinephric bleeding can cause loin swelling and a palpable mass. Haematuria *may be absent* in severe injuries in which there are renal vascular tears, thrombosis, or even complete ureteric avulsion.

## Investigations

Look for, and record, visible haematuria and test for microscopic haematuria. Get venous access, and send blood for FBC, U&E, glucose, clotting screen, and group and save.

- *CT*: urgent abdominal CT is needed if there is frank haematuria or if the patient was shocked (but is now stable) and has frank or microscopic haematuria. The surgical team should be involved before a CT scan is arranged. Patients should be haemodynamically stable for transfer to CT scanning. Intravenous urography (IVU) is unnecessary if contrast-enhanced CT is planned or has been done.
- *FAST (USS)*: this shows renal morphology and confirms the presence of two kidneys, but it does not demonstrate function. It may reveal intraperitoneal blood.
- *Selective angiography*: this is occasionally helpful.

Stable patients with isolated microscopic haematuria do not necessarily need urgent IVU or CT but require review and appropriate follow-up (eg repeat urinalysis at the GP in a few days' time).

## Management

Most *blunt renal injuries* settle with bed rest and analgesia. Give prophylactic antibiotics after consulting the surgical team and according to local policy. Repeat and record pulse, BP, and T°.

Patients with *penetrating renal injuries* and *severe blunt renal trauma* need urgent expert urological assessment ± emergency surgery—the warm ischaemic time of a kidney is only ~2hr. Resuscitate with blood and give IV analgesia and antibiotics.

# Bladder injury

The bladder most often ruptures into the peritoneal cavity, as a result of a direct blow to the lower abdomen. These injuries often occur in individuals with distended bladders. Bone fragments from a fractured pelvis may also penetrate the bladder (see Pelvic fractures, pp. 480–1).

### Clinical features

Lower abdominal tenderness ± peritonism may be associated with haematuria or an inability to pass urine. Look for perineal bruising, and check for fresh blood at the external urethral meatus. Perform a rectal examination to check for the position of the prostate and the integrity of the rectum.

### Investigations and management

CT will identify significant bladder injuries and any associated pelvic fractures. If there is no sign of urethral injury, pass a catheter to check for haematuria. Refer to the urology team. A cystogram will demonstrate extravasation from a bladder injury. Refer patients with intraperitoneal rupture for laparotomy and repair. Extraperitoneal ruptures may heal with catheter drainage and antibiotics.

# Urethral trauma

Posterior urethral tears are often associated with pelvic fractures. Urethral injury may also result (in the absence of fracture) from blows to the perineum (especially falling astride).

Look for perineal bruising and blood at the external urethral meatus, and perform a rectal examination (an abnormally high-riding prostate or an inability to palpate the prostate imply urethral injury).

If urethral injury is suspected, do not attempt urethral catheterization, but refer urgently to the urology team. Some urologists advocate a single gentle attempt at urethral catheterization. Other options are to perform a retrograde urethrogram to assess the extent of the urethral injury or to undertake suprapubic catheterization and subsequent imaging.

### Penile injuries

(See Penile problems and prostatitis, p. 543.)

# Scrotal and testicular trauma

## Scrotal injuries

Wounds involving the scrotal skin may need to be sutured (preferably with absorbable sutures)—most heal rapidly. Refer for investigation if there is complete scrotal penetration with the attendant risk of damage to the testis, epididymis, or vas deferens. If the testis is visible through the wound, refer for surgical exploration and repair in theatre.

## Testicular injuries

Blunt injury to the scrotum/testis may result in a scrotal haematoma or testicular haematoma or rupture. All of these are very painful—provide good analgesia. Further management depends upon the exact diagnosis. USS will help to distinguish between a haematoma and testicular rupture. Involve the urology team—haematomas may respond to conservative measures, but testicular rupture requires urgent surgical exploration and repair.

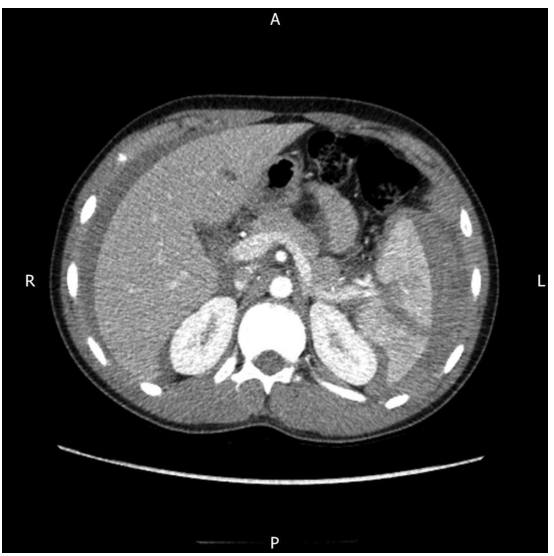


**Fig. 8.9** CT in blunt abdominal trauma showing splenic rupture with a large amount of free fluid in the peritoneal cavity.

# Head injury: introduction

## The size of the problem

Many patients with serious or fatal trauma have suffered a head injury. Additionally, minor head injuries are a frequent reason for attendance at an ED. Blunt injury is far more common than penetrating injury.

*Common causes of head injury*

- Road traffic collisions of all types.
- Falls.
- Assaults.
- Sporting and leisure injuries.
- Workplace injuries.

## Pathophysiology

Brain injury may be primary or secondary.

*Primary injury* Occurs at the time of the head injury. This takes the form of axonal shearing and disruption, with associated areas of haemorrhage. This primary damage may be widespread ('diffuse axonal injury') or localized (eg 'contre-coup' frontal contusions in a fall hitting the occiput).

*Secondary injury* Occurs later, due to various problems that commonly co-exist. Many of these are preventable or treatable and should thus be the focus during resuscitation:

- Hypoxia.
- Hypovolaemia and cerebral hypoperfusion.
- Intracranial haematoma with localized pressure effects and ↑ ICP.
- Other causes of ↑ ICP, including cerebral oedema and hypercapnia.
- Epileptic fits.
- Infection.

## The role of intracranial pressure

Once the skull sutures have fused, the cranium is a closed box. Thus, a small ↑ in volume (eg from swelling or haematoma) results in a large ↑ in ICP (see Fig. 8.10). As ICP ↑, cerebral perfusion pressure ↓, since:

$$\text{Cerebral perfusion pressure} = \text{Mean arterial pressure} - \text{ICP}$$

Once cerebral perfusion pressure falls <70mmHg, significant secondary brain injury may occur. Control of ICP and BP (including avoiding wild swings in BP) is an important treatment goal, especially as the normal cerebrovascular autoregulatory mechanisms are impaired after head injury. Cerebral arterioles remain sensitive to $pCO_2$, however, with an ↑ $pCO_2$ resulting in marked arterial vasodilatation and unwanted ↑ ICP. Controlling $pCO_2$ to within normal levels is therefore important.

↑ ICP produces a diminishing conscious level and causes herniation of the temporal lobe through the tentorial hiatus, compressing the oculomotor nerve, resulting in ipsilateral pupillary dilatation. This may progress to contralateral hemiparesis and brainstem compression, with cardiorespiratory arrest. ↑ ICP leads to a reflex ↑ in systemic arterial BP, together with bradycardia—this combination is the *Cushing response*.

### Indications for referral to hospital

Any one of the following criteria indicates the need for hospital assessment:

- Impaired conscious level at any time.
- Amnesia for the incident or subsequent events.
- Neurological symptoms (vomiting, severe and persistent headache, seizures).
- Clinical evidence of a skull fracture (CSF leak, peri-orbital haematoma).
- Significant extracranial injuries.
- Worrying mechanism (high-energy, possible NAI), possible penetrating injury).
- Continuing uncertainty about the diagnosis after the first assessment.
- Medical comorbidity (anticoagulant use, alcohol abuse).
- Adverse social factors (eg alone at home).

The following are highly recommended:

- The SIGN guideline on head injury is accessible at https://www.sign.ac.uk
- The NICE clinical guidelines on head injury updated in 2019 are accessible at https://www.nice.org.uk

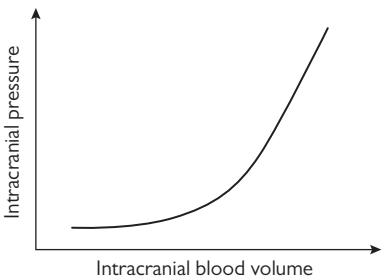


**Fig. 8.10** ICP according to intracranial blood volume.

# Head injury: triage and monitoring

## Triage

Every ED requires a system for the rapid initial assessment of head-injured patients. The exact system will depend upon local policy, expertise, and facilities. It must enable patients with significant injuries to receive immediate resuscitation and ensure urgent treatment of those patients liable to complications. Experienced nursing staff can quickly identify those patients in need of urgent attention, based upon:

- The mechanism of injury.
- The history from the ambulance crew.
- An assessment of vital signs.
- The conscious level according to the GCS (see ➔ Glasgow coma Score (adults), p. 369).
- Limb power.
- Pupil responses.
- BMG.

For patients who are *haemodynamically stable, alert, and orientated*, with no neurological deficit and an apparently minor head injury, it is appropriate to proceed to obtaining a full history, as outlined in ➔ Head injury: history, pp. 366–7.

For patients with *multiple injuries and/or a serious head injury*, there will be no time initially to obtain a full history. Instead, proceed rapidly to initial assessment and resuscitation. During the first few seconds, it is useful to obtain an impression of the severity of the head injury. One simple method (AVPU) classifies patients according to their response to stimulation:

- *A*lert.
- Responsive to *V*oice.
- Responsive only to *P*ain.
- *U*nresponsive.

If a patient is unresponsive or responds only to pain, call for senior ED help and an ICU specialist or anaesthetist, since expert airway care (RSI, tracheal intubation, and IPPV) will be needed.

## Monitoring

Ensure that every head-injured patient receives regular neurological observations. These should include measurements of the GCS, pupil response, limb power, pulse, BP, and RR on a standard chart, such as the one shown in Fig. 8.11. This monitoring is critical if complications such as intracranial haematomas, fits, and hypovolaemia from other injuries are to be detected and treated at an early stage. Any deterioration in GCS is an emergency—re-examine the patient and correct identifiable problems promptly, whilst obtaining urgent senior help.

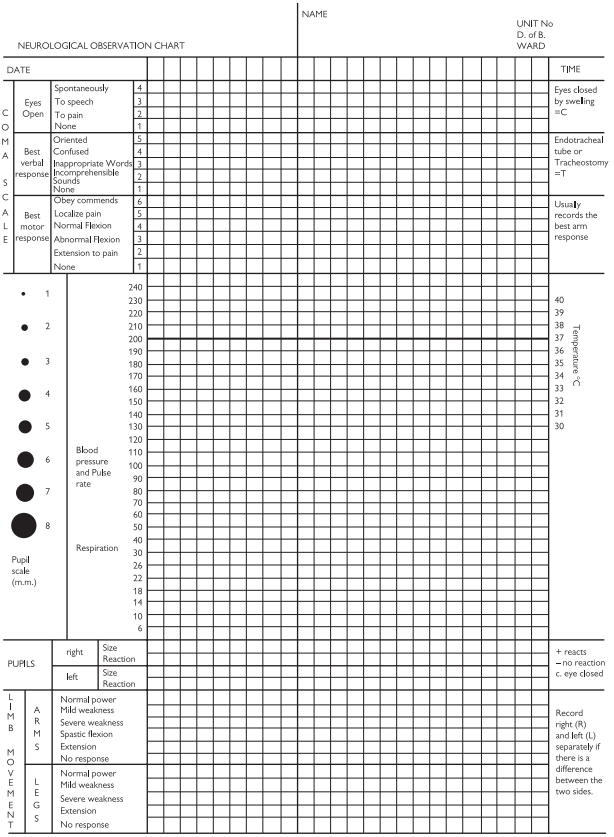


**Fig. 8.11** An example of a neurological observation chart.

# Head injury: history

It may be impossible to obtain a complete history of what happened from the patient, particularly if there was loss of consciousness and/or amnesia. Patients may incorrectly assume that they were rendered unconscious as a result of an injury if they cannot recall what happened. Use all available sources of information, including friends and family, other witnesses, and the ambulance crew.
Cover the following areas:

### Mechanism of injury

Eliciting the exact mechanism of injury will provide an impression of the nature of the forces involved and the risk of subsequent complications. Consider the possibility that the head injury may have been preceded and caused by another medical problem (eg arrhythmia, epilepsy, MI, diabetes).

### Time of injury

Although this information is useful, it may be difficult to establish exactly on occasions.

### Loss of consciousness/amnesia

A period of unconsciousness implies a head injury of at least moderate severity. It can be difficult to establish exactly how long unconsciousness lasted, particularly if there is associated amnesia. Document the length of amnesia (both before and after injury), but remember that the full extent of the amnesia may not become apparent until much later. Thirty minutes of amnesia for events before the injury is one of the criteria for obtaining a CT scan in adults (see ➔ Head injury: imaging, p. 370).

### Subsequent symptoms

Some symptoms are relatively common after head injury (eg headache and vomiting)—many patients will complain of these without being directly asked. There are a number of other symptoms, however, which the patient may not mention unless specifically asked. Enquire about the following symptoms:

- Headache.
- Nausea and vomiting.
- Limb weakness.
- Paraesthesiae.
- Diplopia.
- Rhinorrhoea.
- Otorrhoea.

### Past medical history

Document pre-existing illnesses and symptoms, particularly those that may have caused the head injury (eg cardiac arrhythmias, epilepsy, diabetes) or might make the consequences more severe (eg bleeding tendency, low platelets).

### Drug history

Ask particularly about recent alcohol and other drug ingestion and whether or not the patient is taking anticoagulant drugs (eg warfarin, rivaroxaban). This is very important, since patients with bleeding disorders and/or on anticoagulants have a much higher risk of intracranial problems after head injury and often require CT and hospital admission (see ➲ Minor head injury, pp. 374–5). Evidence is emerging that some antiplatelet drugs (eg clopidogrel) may also add extra risk of intracranial haemorrhage after a head injury—although not currently part of the standard criteria for obtaining a CT scan after head injury, adopt a lower threshold for requesting a CT brain scan in patients on clopidogrel (see ➲ Head injury: imaging, p. 370).

### Social history

Before contemplating discharge of any head-injured patient, establish if there is a responsible adult at home or if there is someone else with whom the patient could go and stay overnight.

### Tetanus status

If there are any wounds, consider the need for tetanus prophylaxis (see ➲ Tetanus prophylaxis, p. 424).

# Head injury: examination

Resuscitate according to problems identified in the primary survey. Follow an initial brief neurological examination (GCS, pupil reactions, limb weakness) with a definitive complete examination as follows.

### Cervical spine injury

Consider this possibility in all cases (see ➲ Major trauma: treatment principles, p. 330).

### Glasgow coma score

Determining the conscious level is a crucial part of the neurological examination. The adult score ranges from 3 to 15 and is calculated as shown in Table 8.3. Repeated GCS recordings underpin monitoring the head-injured patient. A fall in GCS indicates a potentially serious deterioration and mandates a search for correctable conditions.

### Vital signs

Record pulse, BP, and RR.

### BMG

This is essential in all patients with altered conscious level.

### Alcohol

Record if the patient smells of alcoholic drinks, but never assume ↓ GCS is due to alcohol.

### Eye signs

Document the pupil size (in mm) and reaction to light. Unilateral pupillary dilatation may reflect orbital injury or oculomotor nerve compression due to ↑ ICP (see ➲ The role of intracranial pressure, p. 362). A unilateral small pupil may indicate carotid artery dissection (Horner's syndrome). Check the range of eye movements and for diplopia or nystagmus. If there is any suspicion of eye injury, measure VA (see ➲ Approach to eye problems, pp. 550–1). In infants, check for retinal haemorrhages (see ➲ Head injuries, p. 760). Note that papilloedema is a late sign of ↑ ICP.

### Scalp, face, and head

Examine the cranial nerves, and search for abnormal cerebellar signs (nystagmus, hypotonia, intention tremor, dysdiadochokinesia). Carefully record scalp, ear, or facial injury (see ➲ Maxillofacial injuries: introduction, pp. 378–9).

### The limbs

Check limb tone, power, sensation, and reflexes. Abnormalities (eg hemiparesis) may result from the primary brain insult or spinal injury or be a consequence of a developing intracranial haematoma requiring urgent intervention. A stroke can cause a fall resulting in a head injury.

### Other injuries

The presence of a head injury can render the identification of non-cranial injuries difficult. Intra-abdominal injuries often coexist with serious head injuries and are difficult to detect: have a low threshold for FAST ± CT. In particular, relatively minor non-life-threatening orthopaedic injuries (eg finger dislocations, wrist fractures) are easily missed. Ensure full examination, including palpation of all limbs, for possible injury.

## Signs of base of skull fracture

This is often a clinical diagnosis. One or more of the following may be seen:

- Bilateral orbital bruising confined to the orbital margin ('panda eyes').
- Subconjunctival haemorrhage (no posterior margin of bleeding seen).
- Haemotympanum or bleeding from the auditory meatus.
- CSF otorrhoea or rhinorrhoea (± anosmia). Fluid mixtures containing relatively similar quantities of blood and CSF will separate into a 'double ring' when dropped onto blotting paper.
- Battle's sign: bruising over the mastoid process without local direct trauma follows a petrous temporal bone fracture but takes several days to appear.

## Glasgow coma score (adults)

The GCS assesses the level of consciousness by scoring three aspects of the patient's response and adding up the scores to reach a final score.

**Table 8.3** Glasgow coma score

| | | |
|---|---|---|
| Eye response | Open spontaneously | 4 |
| | Open to verbal command | 3 |
| | Open to pressure | 2 |
| | No response | 1 |
| Verbal response | Talking and orientated | 5 |
| | Confused/disorientated | 4 |
| | Inappropriate words | 3 |
| | Incomprehensible sounds | 2 |
| | No response | 1 |
| Motor response | Obeys commands | 6 |
| | Localizes pain | 5 |
| | Flexion/withdrawal | 4 |
| | Abnormal flexion | 3 |
| | Extension | 2 |
| | No response | 1 |
| Total (GCS) score | | Range 3–15 |

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*Notes*

- Record the GCS in shorthand, showing its component parts [eg GCS 10/15 (E3, V2, M5) means that the patient opens their eyes to verbal commands, speaks incomprehensible sounds, localizes a painful stimulus]. Similarly, when communicating with other health professionals, describe the total score (GCS) and list its components.
- Unconsciousness is generally taken to mean no eye response and GCS ≤8.
- 'Abnormal flexion' implies decorticate rigidity, and 'abnormal extension' implies decerebrate rigidity.
- The GCS is difficult to apply to small children but may be modified, as outlined on ➲ Glasgow coma score (children), p. 737.

# Head injury: imaging

Use of X-rays has been replaced by CT scanning. In the UK, the SIGN guidelines on early management of head injury (https://www.sign.ac.uk) were updated in 2009. In England and Wales, NICE guidance was published in 2014 and updated in 2017, and is available at https://www.nice.org.uk

## Role of CT scanning

CT scanning is used to identify and define brain injury, especially intracranial haematomas amenable to surgical treatment. Ensure adequate resuscitation before transferring for CT scanning. In many cases, this will include RSI, tracheal intubation, and IPPV. Always arrange for appropriately trained staff to accompany the patient to the CT scanner. When clinical features point strongly to an intracranial haematoma (eg emergence of focal signs or a deteriorating GCS), discuss promptly with a neurosurgeon the benefits of transferring the patient to a centre that has both CT scanning facilities and an emergency neurosurgical service.

## Indications for CT scan

Request CT scan for any of the following (https://www.nice.org.uk):

- GCS <13/15 on initial assessment in the ED.
- GCS <15/15 at 2hr post-injury.
- Suspected open or depressed skull fracture.
- Any sign of basal skull fracture.
- Post-traumatic seizure.
- Focal neurological deficit.
- >1 episode of vomiting (except in children <12y where clinical judgement is required).
- Amnesia for >30min of events before impact*.
- Loss of consciousness and/or amnesia, combined with one of: age >65y, coagulopathy (including clotting disorder, anticoagulant drug treatment), or dangerous mechanism* (eg pedestrian hit by car, fall >1m or five steps).

Most requests will be urgent (scan performed and interpreted within an hour), except for the two indications marked with an asterisk (*), which, if isolated, may allow a CT scan to be obtained less urgently (within 8hr), depending upon locally agreed policy.

## Interpretation of CT scan

CT scans must be assessed by someone with appropriate expertise.

- Skull fractures are usually obvious, as is the degree of depression of fragments.
- Intracranial haematomas may cause a midline shift and take several forms. Extradural haematomas (Intracranial haematoma, p. 373) appear as high-density (white), lens-shaped lesions (see Fig. 8.12). Subdurals conform more to the surface of the brain (Intracranial haematoma, p. 373) (see Fig. 8.13). Extradural and subdural haematomas can coexist.
- Cerebral contusions appear as patches of low or mixed attenuation.
- Cerebral swelling may take some time to develop, causing the ventricles to appear smaller than normal.

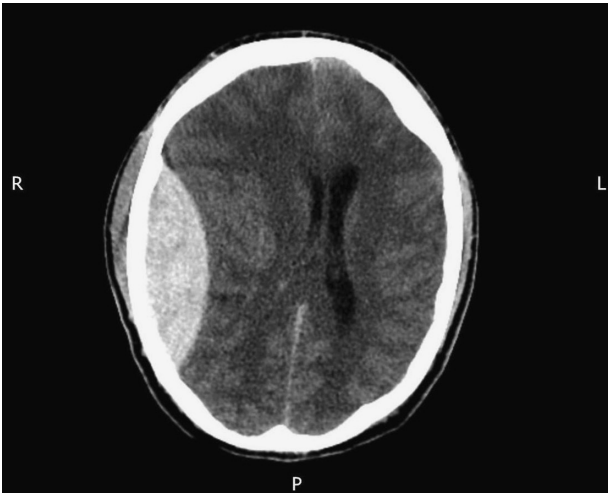


**Fig. 8.12** CT of acute right extradural haematoma with associated midline shift.

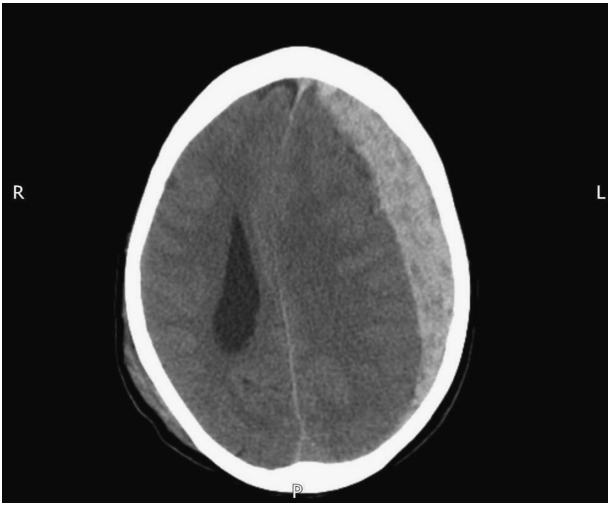


**Fig. 8.13** CT showing an acute left subdural haematoma with midline shift. Note the right scalp haematoma.

# Management of serious head injury

## Initial management

- Clear, establish, and maintain the airway; provide $O_2$ as required, and protect the cervical spine (➲ Major trauma: treatment principles, p. 330).
- Check and support breathing as necessary. Treat serious chest injury.
- Check BMG and treat hypoglycaemia if present (➲ Investigations in major trauma, pp. 332–3).
- Insert two large IV cannulae, and send blood for cross-matching, FBC, clotting screen, U&E, and glucose.
- Correct hypovolaemia, resuscitate, and treat other injuries.
- If GCS ≤8/15, arrange urgent airway protection with RSI, tracheal intubation, and IPPV (➲ Emergency anaesthesia and rapid sequence induction, pp. 322–3). Call for senior ED help and request help from ICU and/or anaesthesia. Check ABG and ventilate to $pCO_2$ of ~4.5kPa.
- Liaise early with an anaesthetist, ICU, and a neurosurgeon.
- Arrange CT with minimum delay (consider the need for 'pan-scan').
- In the multiply or seriously injured patient who will require a CT scan, concerns of opioids masking pupillary signs are less important than ensuring adequate analgesia. Give titrated IV opioid analgesia (➲ Major trauma: treatment principles, p. 330), after recording the GCS, pupil reactions, and basic neurological examination.
- Give IV antibiotics for compound skull fractures. Cefuroxime 1.5g IV is suitable, but be guided by local policy. Regional experts vary as to whether or not they advise prophylactic antibiotics for clinical base of skull fractures—follow local policy. Consider tetanus immunization.
- Liaise with the neurosurgeon early—discuss the need for transfer and other treatments such as prophylaxis against fits (eg levetiracetam).
- Give tranexamic acid if traumatic brain injury with GCS <13 within 3hrs of injury.
- Clean and close scalp wounds to control scalp bleeding, but do not allow this to unduly delay the CT scan or neurosurgical transfer.
- Insert a urinary catheter.
- Consider the need for an orogastric tube. Avoid using NG tubes in facial injury or any possibility of base of skull fracture.

## Treating complications

Early treatment of complications prevents secondary brain damage.

*Deteriorating conscious level*

Having corrected hypoxia, hypercapnia, and hypovolaemia, a diminishing conscious level is likely to reflect intracranial pathology, leading to ↑ ICP, mandating urgent investigation and treatment. Bradycardia, hypertension, and a dilating pupil are very late signs of ↑ ICP. Speed is of the essence. Liaise with a neurosurgeon who will advise on use of agents to ↓ ICP (eg a bolus of mannitol 0.5g/kg IV—typically 200mL of 20% for an adult). Mannitol is an osmotic diuretic which may temporarily ↓ ICP and 'buy time' to get the patient to theatre for drainage of an intracranial haematoma.

Hypertonic saline also acts as an osmotic agent to ↓ ICP and has the advantage of ↑ intravascular volume and not causing diuresis, so it is often used in polytrauma. The dose is ~100mg/kg over 10min (eg 3–5mL/kg of 3% saline or 3mL/kg of 5% saline).

*Seizures*

Check BMG, glucose, and ABG. Give IV lorazepam 4mg. Repeat this once if not initially effective. Start phenytoin IVI (loading dose 20mg/kg IV over 30min, with ECG monitoring) to prevent further fits (an alternative is levetiracetam). Fits which continue ≥10–15min or recur despite this require senior ED and ICU help, RSI, tracheal intubation, and IPPV.

*Other examples of deterioration requiring urgent reassessment*

- The development of agitation or abnormal behaviour.
- The development of severe or ↑ headache or persistent vomiting.
- New or evolving neurological symptoms/signs (eg limb weakness).

## Intracranial haematoma

Causes of neurological deterioration after head injury include hypoxia, hypovolaemia, seizures, cerebral swelling, and intracranial haematomas. Intracranial haematomas are important, as prompt surgery may save lives. Patients with bleeding disorders or on anticoagulants have a greatly ↑ risk of developing an intracranial haematoma after head injury. Reverse anticoagulation as soon as possible (use prothrombin complex concentrate and vitamin K to reverse vitamin K antagonists, see ➲ Managing major bleeding on anticoagulation, p. 179).

*Extradural haematoma*

Classically, extradural haematoma follows bleeding from the middle meningeal artery's anterior branch after temporal bone fracture (See Fig. 8.12) Texts describe head injury with initial loss of consciousness, then return to full consciousness, before neurological deterioration, as intracranial bleeding continues and ICP ↑. However, many patients deviate from the classical 'talk and die' descriptions—extradural haemorrhage may occur in non-temporal areas, with no skull fracture and no initial loss of consciousness. Refer urgently to the neurosurgical team and prepare to transfer.

*Subdural haematoma*

Bridging vein bleeding between the brain and the dura causes subdural haematoma. Unlike extradural haematoma (which is separated from the brain surface by the dura), subdural haematoma conforms to the brain surface. This helps distinguish extradural from subdural haematoma on CT. Subdural haematoma may be acute or chronic.

*Acute subdural haematoma* (see Fig. 8.13) is associated with severe brain insult.

*Chronic subdural haematoma* often occurs in the elderly and alcoholics (↑ risk due to cerebral atrophy, low platelets, bleeding tendency, or anticoagulant medication). Chronic subdural haematoma develops over several days, often presenting with fluctuating conscious level, sometimes with an obscure (or even no) history of head injury. On this basis, adopt a low threshold to request a CT scan for a middle-aged/elderly patient who presents with new unexplained confusion or ↓ GCS, especially if there are risk factors present. Discuss subdural haematomas with the neurosurgical team—unlike extradural haematomas, they are often managed conservatively, especially in the elderly with multiple comorbidities and when there is no mass effect apparent on the CT scan.

# Minor head injury

## Introduction

Assessment and management of patients who have sustained relatively minor primary brain insults can be difficult. This is especially true when assessment is rendered awkward by virtue of age, epilepsy, or drug or alcohol ingestion. In these circumstances, adopt a cautious approach and admit the patient for observation until the picture becomes clearer.

*Golden rules for managing head injury*

- Never attribute ↓ GCS to alcohol alone.
- Never discharge a head-injured patient to go home alone.
- Consider admitting patients with head injury and coexisting bleeding tendency (including those taking anticoagulant drugs).

## Differential diagnosis

Consider whether another condition could be principally responsible for the patient's symptoms. For example, small children who vomit after head injury may be suffering from otitis media or a throat infection. Otitis media may be responsible for both vomiting (with fever) and the head injury (by causing unsteadiness of gait, resulting in a fall).

## Considerations for admission

Consider the need for admission in patients who have:

- Abnormal findings on CT scan.
- ↓ GCS (ie <15/15), neurological deficit, or post-traumatic seizure.
- Significant neurological symptoms (severe headache, vomiting, irritability or abnormal behaviour, continuing amnesia >5min after injury).
- Significant medical problems, particularly bleeding tendency (including inherited diseases and anticoagulant drugs).
- Inability to assess due to epilepsy, consumption of alcohol, or drugs.
- Clinical or radiological evidence of skull fracture.
- No one available at home or no safe home to go to (including suspected NAI and domestic violence).

## Observation of those admitted

Ensure regular neurological observations (see ➔ Head injury: triage and monitoring, p. 364). Act promptly if conscious level ↓ or neurological deficit develops. Remember that the principal reason for admitting patients with apparently minor head injuries is to monitor for the development of intracranial problems. In these cases, resuscitate, liaise with a neurosurgeon, and obtain an urgent CT scan.

If after 12–24hr of observation, the patient is symptom-free and haemodynamically stable and has a GCS of 15/15, with no neurological deficit, consider discharge. Perform a CT scan (if not already imaged) on patients who do not fall into this category (ie symptomatic, ↓ GCS, or with neurological deficit).

### Discharging patients

Most patients who present with minor head injury can be safely discharged directly from the ED. Ensure that there is a responsible adult available to accompany them home and someone to stay with them for 24hr once they get home. Warn the patient and the accompanying adult of the potential problems following a head injury (see Box 8.1)—and what to do if any of these problems are experienced. Give advice regarding analgesia. Most EDs have standard written instructions which are given to the patient and accompanying adult. Examples of head injury warning instructions are shown in Box 8.1.

#### Box 8.1 An example of head injury warning instructions

**Adults**

- Ensure a responsible person is available to keep an eye on you for the next 24hr and show them this card.
- Rest for the next 24hr.
- Do take painkillers, such as paracetamol, to relieve pain and headache.
- DO NOT drink alcohol for the next 24hr.
- DO take your normal medication, but DO NOT take sleeping tablets or tranquillizers without consulting your doctor first.
- If any of the following symptoms occur, then you should return or be brought back to the hospital or telephone the hospital immediately. Tel (01***)****** (24hr):
  - Headache not relieved by painkillers such as paracetamol.
  - Vomiting.
  - Disturbance of vision.
  - Problems with balance.
  - Fits.
  - Patient becomes unrousable.

**Children**

- Your child has sustained a head injury, and following a thorough examination, we are satisfied that the injury is not serious.
- Your child may be more tired than normal.
- Allow him/her to sleep if he/she wants to.
- Give paediatric paracetamol for any pain or headache.
- Try to keep your child resting for 24hr.
- If your child should develop any of the following:
  - Headache not relieved by paediatric paracetamol.
  - Vomiting.
  - Altered vision.
  - Irritability.
  - Fits.
  - Becomes unrousable.

Bring him/her back to the hospital or telephone for advice immediately. Tel (01***)****** (24hr).

Alternative suggested written advice is available from NICE (https://www.nice.org.uk).

# Post-concussion symptoms

## Presentation

Post-concussion symptoms are common after head injury and cause much anxiety in patients and relatives. The most frequent complaints are: headache, dizziness, lethargy, depression, and inability to concentrate.

*Headaches* occur in most patients admitted to hospital after head injury—in ~30%, headaches persist for >2 months. They are usually intermittent and become worse during the day or on exertion. Some appear to be 'tension headaches' and are often not significantly helped by analgesics. Migraine may become more frequent or severe after head injury. Headaches that do not fit these patterns may reflect serious intracranial pathology.

*Non-specific dizziness* is common after concussion. Detailed questioning may distinguish dizziness from vertigo due to disturbance of the vestibular mechanisms. Dizziness may be caused by postural hypotension or by drugs (eg co-codamol and other analgesics) or alcohol (to which patients are often more sensitive after a head injury).

## Diagnosis

Post-concussion symptoms are diagnosed by exclusion of other problems or complications following head injury. Take a careful history, including questions about drowsiness, intellectual function, neck pain, photophobia, vomiting, and rhinorrhoea.

Examine for any specific cause of the symptoms and for any neurological deficit. Look for evidence of meningitis or an intracranial haematoma. Check for papilloedema.

Elderly or alcoholic patients or those on anticoagulants are prone to develop chronic subdural haematomas, which may cause confusion or intellectual deterioration, often without localizing signs. Adopt a low threshold for CT for these patients and for others who have worrying or worsening features.

## Treatment

After a careful history and examination, with appropriate investigations to exclude other problems, reassure and explain that the symptoms are likely to resolve gradually. Since symptoms may last for some time, arrange appropriate follow-up, usually with the GP.

## Concussion in sport

When assessing a patient with a head injury as a result of sport, follow the standard approach to head injury already described. Each sport has its own guideline on whether and when it is safe to return to sport, but in general, patients are advised to rest initially following a head injury, especially whilst symptomatic. Continuing symptoms may warrant specialist input and/or investigation (eg MRI). Advise patients who play high-level sport to take advice from within the sport about when it is safe to return. Returning to sport too early may risk a second injury, with more dramatic consequences (see https://www.headway.org.uk).

# Carotid/vertebral artery dissection

## Background

Cervical artery dissection includes carotid and vertebral artery dissection. It can occur spontaneously (typically in middle-aged individuals) or follow blunt trauma (eg after road traffic collisions). Dissection of both the carotid artery and the vertebral artery can be difficult to diagnose—adopt a high index of suspicion.

Pathophysiologically, dissection can result in ischaemic brain damage from reduced blood flow through the artery as a direct result of blood entering its wall, which can become aneurysmal. Thrombosis may occur at the site of the dissection, with subsequent embolism resulting in acute ischaemic stroke.

Occasionally, the dissection extends intracranially, in which case subarachnoid haemorrhage may ensue.

## Presentation

Carotid or vertebral artery dissection may present acutely as a stroke or in a more chronic fashion. There are often additional features which are unusual in a stroke, with symptoms including:

- Headache.
- Neck or facial pain (especially around one eye).
- Pulsatile tinnitus ('whooshing sound' heard in one ear).
- TIA.

### *Examination findings*

Physical signs are variable but may reflect an acute ischaemic stroke, with signs being determined by the brain territory affected (eg posterior circulation stroke from vertebral artery dissection). In addition, there may be other features, including Horner's syndrome and lateral medullary syndrome. Check for a carotid bruit.

## Investigations

CT angiogram is the standard investigation of choice. Doppler ultrasonography can also identify abnormal blood flow in the carotid artery.

## Management

Take advice from the regional neurosurgical centre. Standard initial treatment (NICE, 2019—see https://www.nice.org.uk) is with either an antiplatelet drug or an anticoagulant.

# Maxillofacial injuries: introduction

These injuries often look dramatic and can be life-threatening, as well as cause significant long-term morbidity. Common causes are assaults, road traffic collisions, and sport.

## Emergency resuscitative measures

- Perform a rapid initial assessment to look for, and treat, airway obstruction or major bleeding (see ➲ Major trauma: treatment principles, p. 330). Remember the possibility of an associated neck injury. Blood may rapidly accumulate in the pharynx, requiring anterior ± posterior nasal packing for control (see ➲ Epistaxis, p. 568).
- Management of airway obstruction is complex and intubation often difficult, and occasionally a surgical airway is required—obtain experienced ED and anaesthetic assistance early. Use jaw thrust, chin lift, and suction to establish a patent airway.
- With bilateral mandibular fractures, the tongue may fall backwards. Restore airway patency by pulling the fractured segment anteriorly or by inserting a large (0 silk) suture in the tongue and pulling anteriorly.
- Maxillary fractures may be displaced far enough backwards to compromise the airway by contact of the soft palate against the posterior pharyngeal wall. This can be relieved by hooking two fingers behind the hard palate and pulling forward and upward, but this can produce considerable bleeding.

## History

Important clues may be obtained from knowing the causative events both in relation to the facial injury itself and also of injury to the head, spine, etc. Drug history (eg anticoagulants or bleeding tendency) may be important.

## Examination

Inspect the face from the front, side, and above (by standing above and behind the patient). Look for:

- Asymmetry.
- Flattening of the cheek (depressed zygomatic fracture).
- 'Dish face' deformity (flattened, elongated face due to posterior and downward displacement of the maxilla).
- Nasal deviation or saddle deformity. Measure the intercanthal distance—if >3.5cm, suspect naso-ethmoidal fracture—see ➲ Naso-ethmoidal fractures, p. 381.
- Uneven pupillary levels (due to orbital floor fracture).
- CSF rhinorrhoea (causes 'tramline' effect with central CSF and blood on either side).
- Subconjunctival haemorrhage without a posterior border (suggests an orbital wall or anterior cranial fossa fracture).

Palpate the facial bones systematically. Start over the superior orbital margins. Work down, feeling both sides at the same time, checking for pain, deformity, crepitus, and movement. Feel specifically for steps in the inferior orbital margin and zygoma. Subcutaneous emphysema implies a compound fracture—often of the maxillary sinus.

Check for hypo-/anaesthesia of the cheek, side of the nose, and the upper lip (infra-orbital nerve injury) and for numbness of the upper teeth (anterior superior alveolar nerve in the infra-orbital canal) and lower teeth and lip (inferior dental nerve damage due to mandibular fracture).

Examine inside the mouth, checking for dental malocclusion (ie the teeth do not meet together properly when the mouth is closed), loose or lost teeth (this may need CXR), bruising, and bleeding.

Examine the eyes carefully (see ➔ Approach to eye problems, pp. 550–1)—assume any laceration below the medial canthus involves the lacrimal duct until proven otherwise.

## Investigations

In patients with multiple injuries, imaging of the chest, pelvis, and cervical spine will take precedence. Even with 'isolated' facial injuries, perform imaging of the cervical spine and head, where indicated, before facial X-rays or CT scanning.

Facial X-rays are often both difficult to perform (because of poor patient co-operation) and difficult to interpret. Get maxillofacial specialist advice regarding the views required and their interpretation. CT scanning is often required prior to definitive maxillofacial surgery.

The commonly required views include:

- Occipitomental 10°, 30°, and 45°.
- Lateral.
- Orthopantomogram (for the mandible).

## Treatment

Treatment of specific facial fractures is considered in ➔ Middle third facial fractures, pp. 380–1, ➔ Zygomatic, orbital, and frontal sinus fractures, pp. 382–3, and ➔ Mandibular injuries, pp. 384–5. Remember that even in the absence of a visible fracture on X-ray, patients in whom there is clinical suspicion of facial fracture (swelling, tenderness, asymmetry, numbness, etc.) require expert attention and/or follow-up (see Fig. 8.14).

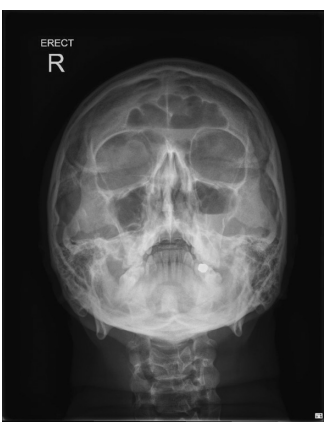


**Fig. 8.14** X-ray showing fluid levels in the left frontal and maxillary sinuses.

# Middle third facial fractures

## Dento-alveolar fractures

Involve only teeth and their bony support. Look for deranged occlusion and stepped malalignment of teeth, bruising of gums, and palpable fracture in the buccal sulcus.

## Le Fort facial fractures

These lie between the frontal bone, the skull base, and the mandible. They involve the upper jaw, teeth, nose, and maxillary and ethmoid air sinuses

- *Le Fort I* involves the tooth-bearing portion of the maxilla. Look for lengthening of the face due to the dropped maxillary segment. There may be movement or a split of the hard palate, a haematoma of the soft palate/buccal sulcus, and malocclusion (See Fig. 8.15).
- *Le Fort II* involves the maxilla, nasal bones, and the medial aspects of the orbits. Look for a 'dished-in' face, a step in the infra-orbital margin, infra-orbital nerve damage, malocclusion, and surgical emphysema. The maxilla may be 'floating'. Check for epistaxis, CSF rhinorrhoea, diplopia, and subconjunctival haematoma. Facial swelling occurs rapidly and is often severe. Later, bilateral peri-orbital bruising may be evident.
- *Le Fort III* involves the maxilla, zygoma, nasal bones, ethmoid, and small bones of the base of the skull (see Fig. 8.15). The entire midface is fractured from the base of the skull. Features include those of type II plus: flattened zygomatic bones (which may be mobile and tender), steps over the fronto-zygomatic sutures, movement and deformity of the zygomatic arch, and different pupillary levels. There is usually severe facial swelling and bruising. Pharyngeal bleeding may severely compromise the airway and cause hypovolaemic shock.

Le Fort fractures may be asymmetrical (eg Le Fort II on the right; III on the left).

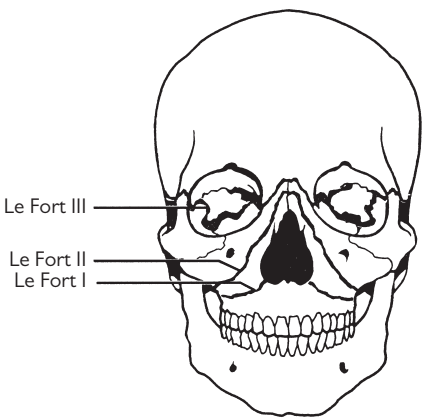


**Fig. 8.15** Le Fort classification of facial fractures.

### Naso-ethmoidal fractures

These produce a flattened nasal bridge, with splaying of the nasal complex, saddle-shaped deformity of the nose, traumatic telecanthus, peri-orbital bruising, subconjunctival haematoma, epistaxis, CSF rhinorrhoea, and supraorbital or supratrochlear nerve paraesthesiae.

### Management of middle third facial fractures

- Resuscitate and establish a clear airway, as described in ➲ Airway obstruction: basic measures, pp. 334–5.
- Refer dento-alveolar fractures for repositioning and immobilization with acrylic/metal splints ± wiring.
- Refer all patients with middle third or naso-ethmoidal fractures to the maxillofacial surgeons for admission. Continuing haemorrhage may need packing—leave this to the specialist. Tell the patient not to blow the nose (↑ subcutaneous emphysema and may drive bacteria into fracture sites and intracranially). Prophylactic antibiotics are often advised by maxillofacial surgeons. Ensure tetanus prophylaxis (see ➲ Tetanus prophylaxis, p. 424).
- Discuss patients with CSF leaks with the neurosurgeons.
- Clean and dress compound facial lacerations, but do not close them (unless actively bleeding); they may need formal debridement and they provide access to fractures for open reduction and internal fixation.

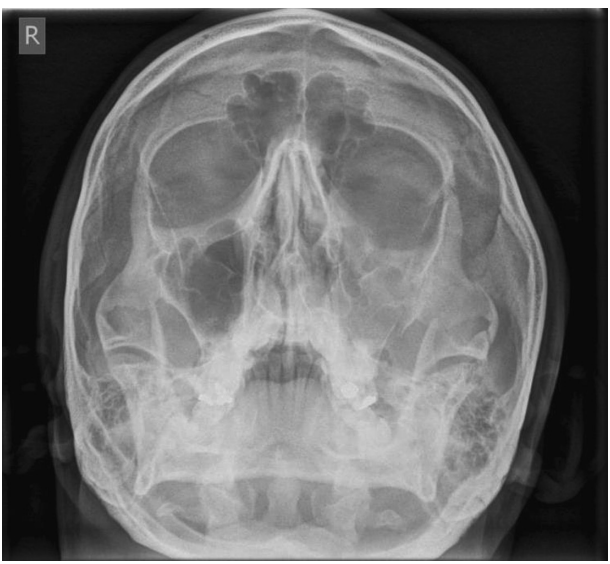


**Fig. 8.16** Left 'tripod' fracture following an assault, involving fractures of the left infra-orbital margin, the zygoma, and the lateral orbital wall.

# Zygomatic, orbital, and frontal sinus fractures

## Zygomatic (malar) fractures

These injuries are usually due to a direct blow and are frequently associated with severe eye injuries. 'Tripod fractures' (see Fig. 8.16) involve fractures through the zygomatico-temporal and zygomatico-frontal sutures and the infra-orbital foramen.

*Examination* Look for flattening of the cheek (often obscured later by swelling), a palpable defect in the infra-orbital margin, infra-orbital nerve damage, diplopia, and subconjunctival haemorrhage (especially if no posterior margin is seen). Isolated fractures of the zygomatic arch may be accompanied by a palpable defect over the arch and limited or painful jaw movement resulting from interference with the normal movement of the coronoid process of the mandible.

## Orbital 'blow-out' fractures

This is caused by a direct blow to the globe of the eye (commonly from a squash ball or a shuttlecock), resulting in a fracture of the orbital floor and prolapse of contents into the maxillary sinus.

*Examination* Check for diplopia due to inferior rectus entrapment ( patient cannot look up and medially), enophthalmos, and surgical emphysema. Carefully check the eye itself for injury (hyphaema, retinal detachment, glaucoma, blindness). Record the VA. Test infra-orbital nerve function. Fractures of the floor of the orbit may not be easily visible on X-ray but can often be inferred by the soft tissue mass in the roof of the maxillary sinus ('tear drop' sign), clouding of the sinus, and surgical emphysema.

*Management of zygomatic and orbital fractures*

- Tell the patient not to blow his/her nose.
- Refer all patients (including those in whom a fracture is clinically suspected but not evident on X-ray) to maxillofacial specialists who will advise regarding prophylactic antibiotics and will arrange further investigation (usually CT scanning) and treatment.
- Involve the ophthalmologists if the eye is also injured.

Patients with orbital emphysema who complain of sudden ↓ in vision may be suffering from a build-up of air under pressure which is compromising retinal blood flow. These patients need emergency decompression.

## Retrobulbar haemorrhage

Blunt orbital trauma may be complicated by the formation of retrobulbar haemorrhage. This may result in ↓ vision, limited eye movements, proptosis, and ↑ intra-ocular pressure. This is an ophthalmological emergency requiring lateral canthotomy and cantholysis.

### Frontal sinus fractures

Presenting features include supraorbital swelling, tenderness, and crepitus, occasionally with supraorbital nerve anaesthesia. CT scanning will determine whether or not there are fractures of simply the anterior wall or both anterior and posterior sinus walls (± depressed fragments). Give IV antibiotics and refer for admission and observation, which, in the case of depressed fragments, should be to the neurosurgical team.

## Major bleeding after facial trauma

Ongoing haemorrhage from the nasal and oral cavities following facial trauma may be difficult to control. Obtain senior expert help and try the following approach:

- Secure the airway with a tracheal tube.
- Reduce fractures into anatomical alignment.
- Use a rigid cervical collar to support the mandible.
- Insert bite blocks into both sides of the mouth, and position these between the posterior molars.
- Tamponade nasal bleeding by placing a 12G Foley catheter into the affected nostril. Insert it into the posterior nasal cavity, then inflate the balloon with 5–7mL of saline. Then pull the catheter slightly more anteriorly, and instil a further 5–7mL of saline, before securing it with a clamp.
- Pad the nose with gauze to ↓ the likelihood of necrosis.
- If there is persistent nasal or oral bleeding, try packing with Vaseline gauze dressings.
- If control of bleeding is not achieved, surgical options include arterial embolization and ligation of the external carotid artery.

# Mandibular fractures

Considerable force is required to fracture the mandible, so look for concurrent head or other injuries. The mandible may be fractured at a site distant from the point of impact (eg a fall on the chin may cause condylar fractures). There are often fractures at two or more sites (see Fig. 8.17). The temporomandibular joint may be dislocated or the condyle driven through the temporal bone, causing a skull base fracture.

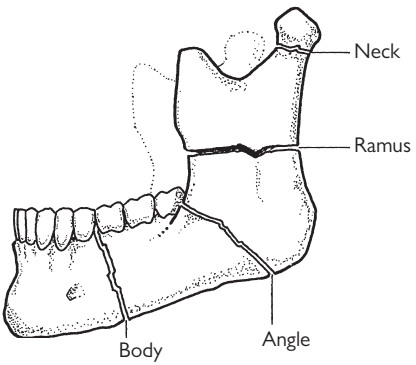


**Fig. 8.17** Common fracture sites of the mandible.

## Symptoms and signs

The patient usually presents with pain (aggravated by jaw movement or biting). Check for swelling, tenderness, or steps on palpation of the mandible. Look for malocclusion, loose or missing teeth, and intraoral bruising. Numbness of the lower lip indicates injury to the inferior dental nerve where it passes through the ramus of the mandible.

## X-rays

Request an orthopantomogram (OPG), which demonstrates most mandibular fractures well (see Fig. 8.18). Temporomandibular joint dislocation and condylar fractures are best shown on condylar views.

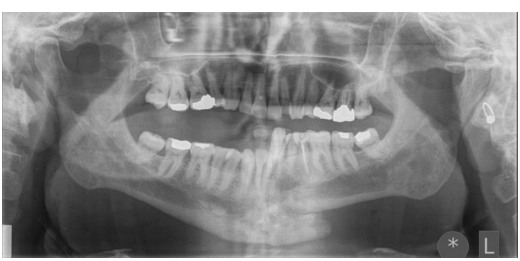


**Fig. 8.18** Displaced fractured mandible as seen on OPG.

### Management

Treat simple undisplaced single fractures not involving the teeth with analgesia, soft diet, prophylactic antibiotics (eg penicillin or co-amoxiclav), tetanus cover, and referral to the maxillofacial outpatient department. Refer displaced or multiple fractures to the on-call specialist.

Refer to the on-call specialist patients with bilateral condyle fractures or single fractures with malocclusion or deviation of the jaw on opening. Advise patients with unilateral asymptomatic fractures to take a soft diet, and arrange outpatient follow-up.

## Temporomandibular joint dislocation

This is almost invariably anterior but can be uni- or bilateral. It may be caused by a direct blow to the (often open) jaw or, in patients with lax joint capsule/ligaments, by yawning, eating, dystonic reactions, or intubation. The patient cannot close the mouth; the jaw protrudes anteriorly, and difficulty in swallowing leads to drooling of saliva. The pain is often over the temporal fossa, rather than in the temporomandibular joint itself. Only obtain X-rays if there is a history of direct trauma.

### Reduction technique

If seen shortly after dislocation, reduction can usually be achieved simply and without anaesthesia or sedation (see Fig. 8.19). Explain the process to the patient. Sit in front of him/her, and with your gloved thumb(s), protected by a gauze swab, press down and backwards on the lower molar teeth, whilst gently cupping and lifting the chin with the fingers. After reduction, advise the patient to take a soft diet and not to yawn (difficult!) or open the mouth widely for 24hr. Delayed presentations can be associated with muscle spasm, requiring anaesthesia and muscle relaxants.

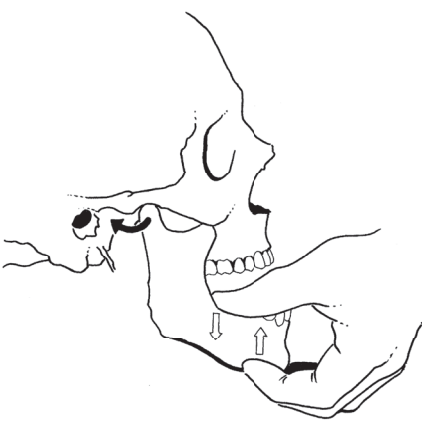

**Fig. 8.19** Reduction of dislocated temporomandibular joint.

# Penetrating neck trauma

In the UK, neck 'stabbings' and 'slashings' are not uncommon, but gunshot wounds to the neck are rare. The neck is divided into 'zones' when classifying wounds:

- *Zone 1*: extends from the clavicles to the cricoid cartilage.
- *Zone 2*: extends from the cricoid to the angle of the mandible.
- *Zone 3*: is the area from the angle of the mandible to the skull base.

## Initial assessment and resuscitation

Give $O_2$ as required; establish wide-bore venous access (send blood for cross-matching), and resuscitate according to an evaluation of ABCDE. Quickly check for evidence of spinal cord injury. Do not aim to raise the BP too high: a systolic of ~90mmHg is sufficient if the patient is conscious. Look for, and rapidly treat, the following:

- Direct airway injury—may need emergency surgical airway (see ➲ Airway obstruction: surgical airway, p. 336).
- Tension pneumothorax (see ➲ Tension pneumothorax, pp. 338–9).
- Major external haemorrhage—apply pressure to the wound.
- Massive haemothorax (see ➲ Haemothorax, p. 345).

Occasionally the open end of a cut trachea will be seen in extensive neck wounds; secure the airway temporarily by passing an ET or tracheostomy tube into the lumen and securing the tube carefully. Further management depends partially upon the haemodynamic status.

## The unstable patient

Haemodynamic instability may be due to tension pneumothorax or massive haemothorax. Persistent major bleeding from a neck wound (usually zone 2) associated with haemodynamic instability is an indication for emergency surgical exploration in theatre. Other indications for exploration include:

- Breach of the platysma (do not probe or explore the wound in the ED).
- Evidence of vascular injury (haemorrhage, expanding haematoma).
- Evidence of surgical emphysema (indicates laryngeal or oesophageal disruption which requires repair).

## The stable patient

Many patients are stable and have little evidence of significant injury.

- Provide $O_2$; secure venous access, and send blood for group and save.
- Monitor $SpO_2$, pulse, BP, and RR.
- Obtain a CXR (to exclude pneumothorax/haemothorax).
- Provide IV analgesia as required (see ➲ Major trauma: p. 330).
- Establish tetanus status and the need for prophylactic antibiotics (eg cefuroxime 1.5g IV—according to local policy).
- Investigate—consider CT neck. Occasionally four-vessel angiography or duplex USS (to exclude vascular injury) and a contrast swallow/oesophagoscopy (to exclude oesophageal injury) can help (usually zone 1 or 3 injuries).
- Refer to ENT or maxillofacial surgeons for admission, observation, formal wound cleaning, exploration, and closure.
- Carefully document the size, position, and other features of the neck wound, in view of the high medicolegal significance (see ➲ Interpersonal violence—medicolegal implications, p. 411).

# Silver trauma

## Background

The number of older individuals in the population is ↑ dramatically, and associated with this is a rise in the amount of serious injury in the elderly—which is sometimes referred to as 'silver trauma'.

The 2017 Trauma Audit and Research Network report shows that the most common mechanism of serious injury amongst adults aged >60y is a fall from standing (<2m), which compares with road traffic collisions in adults aged <60y. The elderly are more likely to sustain major injuries after less serious mechanisms, when compared with younger people. Further, the extent of these injuries is less likely to be recognized at presentation.

## Outcomes after trauma in the elderly

Falls indoors are an important cause of serious injury in the elderly, with falls down stairs being responsible for the majority of all fall deaths.

### *Age vs frailty*

Evidence suggests that outcomes after trauma in the elderly depend less upon the chronological age of a person than the 'physiological age', which is perhaps most easily expressed in terms of frailty (eg the 'clinical frailty score'—see ➲ Assessing the elderly patient, p. 22). The frail elderly trauma victim has less physiological reserve to cope with serious injury.

## Additional considerations in elderly trauma

- The prehospital alerting of potential major trauma from the ambulance service to the ED tends to underestimate injuries in the elderly—so adopt a high index of suspicion of major trauma in older patients who arrive unannounced.
- Initial assessment of the elderly following trauma is also more difficult—patients aged >65y are more likely than younger patients to have catastrophic bleeding, the source of which cannot be identified on primary survey.
- When interpreting initial vital signs, remember that mortality rates in the elderly with a systolic BP of <110mmHg are similar to those in a younger person with a systolic BP of <90mmHg.
- Pre-existing illness can impact dramatically upon presentation (eg prescribed β-blockers preventing tachycardia as a response to hypovolaemia) and upon treatment (eg pre-existing cardiac disease may predispose the patient to heart failure/MI). An initial venous lactate of >2.5mmol/L is a better predictor of mortality after trauma than altered initial vital signs. Think about the person being treated, particularly comorbidities and medication—consider generating a problem list (to include illnesses) following a 'silver survey'.
- Radiation issues are less of a concern in the elderly and injuries are more likely to be concealed, so adopt a lower threshold for CT.
- When performing CT head scans in the elderly after trauma, consider scanning the neck as well—some departments routinely scan down to the bottom of C2, taking into account the fact that 50% of neck fractures in the elderly affect C1 and C2.

# Approach to possible spinal injury

Consider spinal injury in every injured patient. Maintain a particularly high index of suspicion and provide spinal immobilization in:

- Major trauma.
- 'Minor' trauma with spinal pain and/or neurological symptoms/signs.
- Altered consciousness after injury.
- A mechanism of injury with a possibility of spinal injury (eg road traffic collision, high fall, diving, and rugby injuries).
- Pre-existing spinal disease (eg rheumatoid arthritis, ankylosing spondylitis, severe osteoarthritis, osteoporosis, steroid therapy), as serious fractures or dislocations may follow apparently minor trauma.

The most common sites of spinal injury are the cervical spine and the thoraco lumbar junction.

## Airway management and spinal immobilization

These two aspects demand immediate attention in any patient with possible spinal injury—manage them together. The neck is the most common site of cord injury. If immobilization is not achieved with unstable injuries, it is the site at which most additional cord or nerve root damage can be produced.

- Perform manual immobilization rapidly (without traction), keeping the head and neck in the neutral position, by placing both hands around the neck and interlocking them behind, with the forearms preventing head movement (see Fig. 8.20).
- Maintain manual stabilization/support with sand bags or blocks placed on either side of the head and tape or straps applied to the forehead and chin to prevent rotation. Hard collars are still used by some but remain controversial—evidence of the effectiveness of hard collars is limited and their use is associated with discomfort and other problems. Consider allowing fully conscious patients who present days after a neck injury to lie flat without formal immobilization until assessment/imaging.
- Ensure airway patency and adequate ventilation—hypoxia compromises an injured cord. Initially in an unconscious patient, jaw thrust and suction to the upper airway can be used. Remember that oropharyngeal stimulation can provoke severe bradyarrhythmias. Simple airway adjuncts such as oro- and nasopharyngeal airways often maintain upper airway patency, but sometimes tracheal intubation is required. This must be performed by an individual experienced in advanced anaesthetic techniques (usually RSI, rarely fibre-optic), with an assistant controlling the head/neck to limit cervical spine movement.
- Ventilation can deteriorate due to cord oedema/ischaemia, so look regularly for diaphragmatic breathing (the diaphragm is supplied by C3/4/5) and the use of accessory muscles of respiration. Use pulse oximetry and regular ABG analysis to confirm adequate oxygenation and ventilation. Tracheal intubation and controlled ventilation may be required.
- Patients may have been transported on a scoop stretcher or vacuum mattress—remove these as soon as the primary survey is completed and resuscitation commenced (see Fig. 8.21). Receiving patients onto a specially designed trauma mattress (with carrying handles) can help resuscitation, particularly transferring in and out of the CT scanner.

Suspect spinal injury in patients with ↓ consciousness if there is:

- Flaccid arreflexia.
- ↓ anal tone on PR examination.
- Diaphragmatic breathing.
- An ability to flex (C5/6), but not to extend (C6/7), the elbow.
- Response to painful stimulus above, but not below, the clavicle.
- Hypotension with associated bradycardia.
- Priapism.

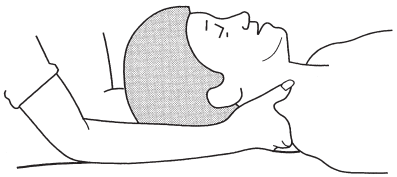

**Fig. 8.20** Manual immobilization of the neck.

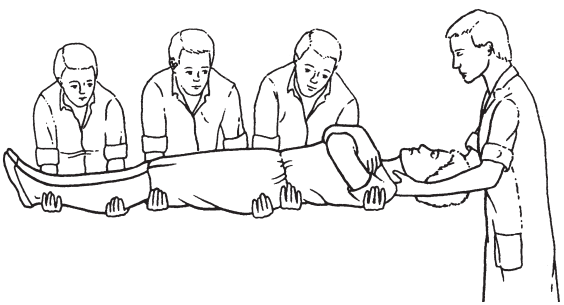

**Fig. 8.21** Co-ordinated four-person lift.

# Spine and spinal cord injury

## Managing the circulation

Monitor ECG and BP. Interruption of the cord sympathetic system causes loss of vasomotor tone, with vasodilatation, ↑ venous pooling, and ↓ BP. Flaccidity and arreflexia, together with the absence of reflex tachycardia or associated (inappropriate) bradycardia, are pointers to this, but before diagnosing 'neurogenic shock', exclude and treat other causes of hypotension (eg blood loss, tension pneumothorax). IV fluid usually corrects relative hypovolaemia, but consider inotropes if ↓ cardiac output persists despite adequate volume replacement and correction of bradycardia by atropine. CVP monitoring in neurogenic shock can help prevent fluid overload.

## Other considerations

Insert a urinary catheter to monitor urine output and prevent bladder distension. If there is no craniofacial injury, an NG tube will prevent gastric distension (ileus is common after cord injury) and ↓ the risk of aspiration.

Many patients with spinal cord injury from blunt trauma have other major injuries. Conscious patients can usually describe a sensory level and paralysis, with pain at the level of the vertebral injury. Adopt a high index of suspicion for thoracic/abdominal injury—clinical features may be obscured by sensory or motor deficits from the cord injury itself. Abdominal distension may occur and there may be no signs of peritonism. Consider the need for FAST or CT scan.

## Neurological examination

Carefully document the neurological findings, ideally on a specialist chart (eg American Spinal Injury Association chart available at https://www.asia-spinalinjury.org).

Check light touch and pinprick sensation, proprioception, muscle power, tone, co-ordination, and tendon reflexes. Evidence of distal, motor, or sensory function implies an incomplete lesion, and hence the possibility of recovery. The accuracy of this baseline examination is important, since cephalad progression of abnormalities is a sensitive marker of deterioration and, in the cervical region, may lead to respiratory failure.

Document muscle group strength in upper and lower limbs using the 0–5 grading system (see Table 8.4). It is standard practice to record the most caudal location which has intact (normal) motor and sensory function.

Examine the perineum, and perform a PR examination: look for voluntary contraction and anal tone. An intact bulbocavernosus reflex (squeezing the glans penis/contraction of the bulbocavernosus muscle—S2, 3, 4) and anal cutaneous reflex (scratching peri-anal skin/anal contraction—S4, 5) implies sacral sparing.

## Spinal examination

Log roll the patient. The person controlling the head and neck directs movement. Carefully examine for tenderness, step deformity, gibbus, widening of interspinous gaps, and prominence of spinous processes. There may not be overlying tenderness with vertebral body fractures. Remove any debris from under the patient. Keep the patient covered and warm, as ↓ sympathetic vasomotor tone can ↑ the risk of hypothermia.

**Table 8.4** Grading muscle strength

| Grading of muscle power | |
|---|---|
| 0 | Total paralysis |
| 1 | Palpable or visible contraction |
| 2 | Movement with gravity eliminated |
| 3 | Movement against gravity |
| 4 | Weaker than usual |
| 5 | Normal strength |
| **Muscles supplied by various nerve roots** | |
| C5 | Shoulder abductor (deltoid) |
| C6 | Wrist extensors (extensor carpi radialis) |
| C7 | Elbow extensor (triceps) |
| C8 | Middle finger flexor (flexor digitorum profundus) |
| T1 | Little finger abductor (abductor digiti minimi) |
| L2 | Hip flexors (iliopsoas) |
| L3 | Knee extensors (quadriceps) |
| L4 | Ankle dorsiflexors (tibialis anterior) |
| L5 | Big toe extensor (extensor hallucis longus) |
| S1 | Ankle plantar flexors (soleus, gastrocnemius) |

## Incomplete cord injury patterns

There are several recognized patterns of incomplete spinal cord injury. Although the resultant physical signs can be predicted from a detailed knowledge of neuroanatomy, bear in mind that some patients present with an atypical injury, and therefore an atypical pattern of injury.

*Anterior cord syndrome* Loss of power and pain sensation below the injury, with preservation of touch and proprioception.

*Posterior cord syndrome* Loss of sensation, but power preserved.

*Brown-Séquard syndrome* Hemisection of the cord, producing ipsilateral paralysis and sensory loss below the injury, with contralateral loss of pain and temperature. This syndrome occurs more frequently after a penetrating injury than after a closed injury.

*Central cervical cord syndrome* Typically seen in elderly patients following extension injuries to the neck, with degenerative changes being the only X-ray or CT abnormality. It is characterized by incomplete tetraparesis, affecting the upper limbs more than the lower limbs (as nerves supplying the upper limbs lie more centrally within the cord). Sensory deficits are variable.

## Spinal cord injury without radiographic abnormality

Some children with spinal cord injury have no X-ray or CT abnormality (MRI may help). The extent of both neurological deficit and recovery varies. Adults may have spinal cord injury due to traumatic herniation of an intervertebral disc, epidural haematoma, or ligamentous instability.

# Spine and spinal cord injury: imaging

## X-rays

For indications for cervical spine X-rays, see ➲ Soft tissue neck injuries, pp. 476–7. Note that spinal cord injury can occur without X-ray (or CT) abnormality. This may be due to ↑ soft tissue elasticity, allowing excessive movement (children), or cord compression from disc prolapse (younger patients), or vascular involvement or spondylosis (older patients).

*Cervical spine* Request *AP, lateral* (must show C7/T1 junction), and *open-mouth odontoid peg views* if CT of the cervical spine is not indicated. Displacement (subluxation/dislocation) and fractures of vertebral bodies, spinous processes, and the peg are best seen on lateral view. Unifacet dislocation causes anterior displacement ≤50% of the AP diameter of the vertebral body. Displacement >50% suggests bilateral facet dislocation. Look for swelling of prevertebral soft tissues.
*AP views* show injuries to the pedicles, facets, and lateral masses.
*Open-mouth odontoid views* usually demonstrate peg fractures.

*Thoracolumbar spine* Standard views are AP and lateral. In the thoracic region, overlapping structures may make interpretation difficult and necessitate other imaging. If X-rays are of diagnostic quality, visualization of compression or burst fractures and displacement is not difficult, but these have little relation to the degree of cord injury.

## Assessment of spinal X-rays

Interpreting spinal X-rays can be difficult. If in any doubt, get senior expert help. A systematic approach helps to prevent injuries from being missed:

- Check alignment of the vertebrae. The spine should be straight or follow gentle curves and should not exhibit any 'steps'. On the lateral X-ray, assess the alignment by checking in turn: anterior vertebral border, posterior vertebral border, posterior facets, anterior border of spinous processes, and posterior border of spinous processes. Look also at interspinal distances.
- Check alignment on the AP film by following the spinous processes and the tips of the transverse processes (see Fig. 8.22). Look for rotational deformity and asymmetry.
- Assess the integrity of each spinal vertebra, including the vertebral bodies, laminae, and pedicles.
- Be vigilant in assessing the odontoid peg view (see Fig. 8.22), looking for asymmetry/displacement of the lateral masses of C1. Distinguish fractures (limited to the bone area) from overlying soft tissue shadows (extend beyond the area of the bone). Note that the atlanto-odontoid distance should be ≤3mm in adults and ≤5mm in children.
- Look for indirect evidence of significant spinal injury (↑ prevertebral space). The normal soft tissue prevertebral thickness at the anteroinferior border of C3 (ie the distance between the pharynx and the vertebral body) is <0.5cm.

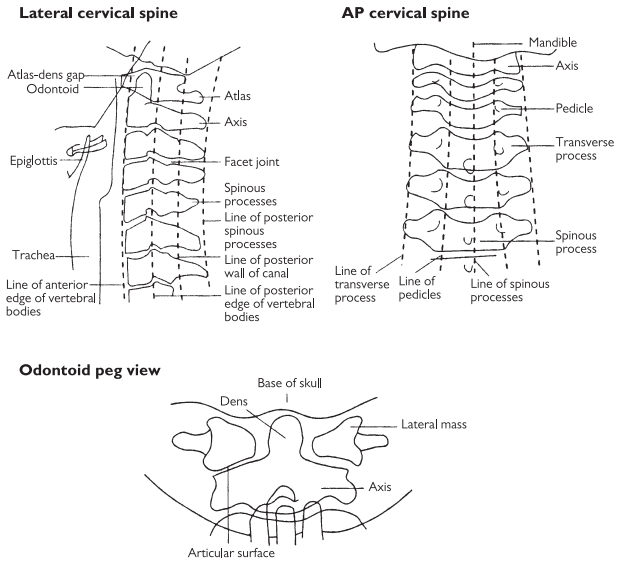


**Fig. 8.22** Interpretation of spinal X-rays.

## CT and MRI

CT delineates bony abnormalities and the extent of spinal canal encroachment. CT of the cervical spine (base of the skull to T4 level) is indicated for patients requiring CT brain for significant head injury or as part of a 'pan-scan' for multiple trauma (see SIGN and NICE guidelines). CT or MRI are useful for patients in whom there is clinical suspicion of injury (persistent pain, positive neurology) despite normal X-rays. CT is the first-line investigation of older patients (>65y) with suspected spinal injury, as X-rays are likely to be hard to interpret due to degenerative changes.

## Further treatment

Immobilize cervical injuries using a firm, well-fitting cervical collar (eg Philadelphia or Miami J), pending advice from the specialist spinal team. Skeletal traction using Gardner–Wells calipers or halo devices and pulley/weight systems may be undertaken by spinal specialists to reduce fracture–dislocations, improve spinal alignment, and decompress the cord.

Stable thoracolumbar fractures usually respond to analgesia and rest. If in doubt, discuss with the spinal team, as unstable injuries may be surgically fixed.

With penetrating injuries, if the object is still in place, arrange removal in theatre where the spinal cord/canal injury can be directly seen.

# Dermatomes

## Dermatomes front

(See Fig. 8.23.)

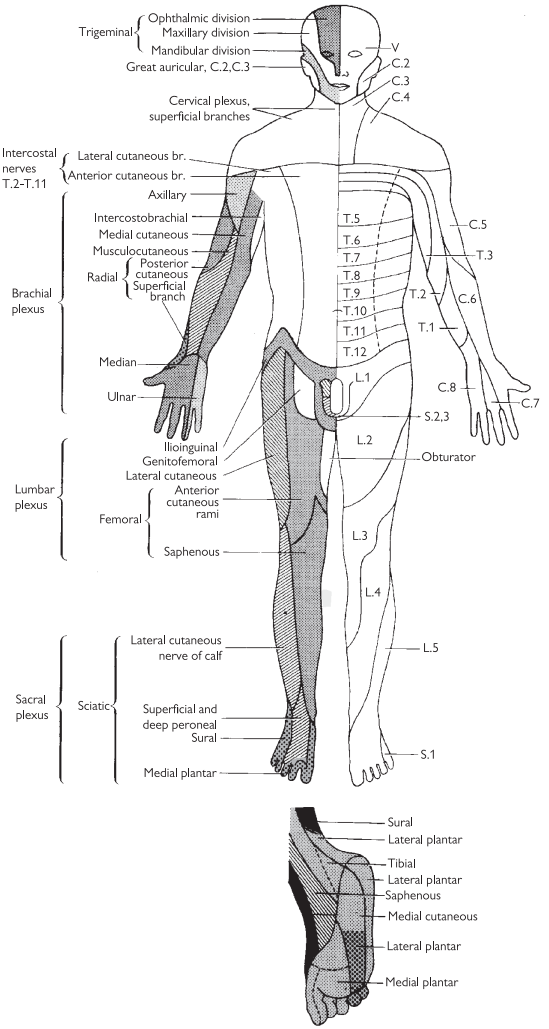


**Fig. 8.23** Dermatomes: front.

## Dermatomes back

(See Fig. 8.24.)

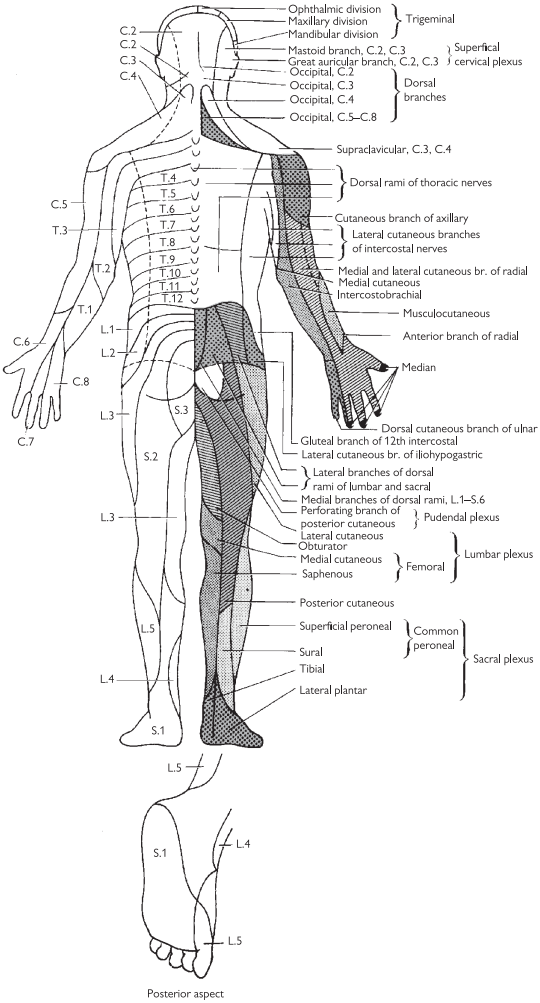


**Fig. 8.24** Dermatomes: back.

# Gunshot injuries

In the UK, inform the police as soon as possible whenever a patient presents with a gunshot wound. Wounds produced by bullets/missiles are determined by kinetic energy (KE) transfer, missile flight characteristics, and the tissue injured.

## Kinetic energy transfer

The KE of a missile is directly proportional to its mass and the square of its velocity ($KE = \frac{1}{2}mv^2$). Thus, tissue injury depends more upon the bullet's velocity than its mass. At velocities > speed of sound, the rate of dissipation of KE becomes proportional to the velocity$^3$ or even higher powers. Bullets travelling at >1000ft/s (300m/s) are 'high velocity'.

## The tissue itself

Tissue density affects a missile and the energy dissipation and tissue destruction. Bone involvement may cause additional retardation, whilst bony fragments cause secondary injuries.

## Cavitation

High-velocity bullets transmit energy to the tissues, compressing and accelerating them at right angles away from the track. This leads to cavity formation around the track. Over a few micro-seconds, the cavity enlarges and then collapses. Tissue elasticity perpetuates a process of cavity reformation and collapse, with rapidly ↓ amplitude of oscillations until all KE is expended. This causes highly destructive stretching, tearing, and shearing of tissues, causing injury many times the size of the bullet. Since the pressure in the cavity is sub-atmospheric, debris and organisms are sucked in.

## Clinical aspects

Follow standard principles to manage major trauma. Specific aspects are:

- Consider staff safety—involve the police and check the patient for weapons.
- The magnitude of the external wounds may bear little relationship to the severity of internal injury. Remove the patient's clothes (police evidence) and examine the entire body for entrance/exit wounds that are often missed in hairy areas (eg scalp, axillae, and perineum).
- Patients are often young and fit—signs of hypovolaemia may be delayed.
- Chest injuries are commonly associated with pneumothorax (see Traumatic pneumothorax, p. 344). PEA cardiac arrest should prompt rapid exclusion of tension pneumothorax, then immediate thoracotomy to relieve cardiac tamponade (see Thoracotomy for cardiac arrest, p. 353).
- Abdominal wounds are associated with a high incidence of internal injury and require laparotomy and antibiotic cover.
- Gunshot wounds are prone to anaerobic infection (especially tetanus and gas gangrene)—clothing/fragments spread widely through tissues distant from the wound track. Extensive surgical debridement (wide excision/fasciotomy) is often required to remove devitalized tissue and foreign material. All high-velocity injuries need delayed primary closure with grafting or suture at 3–5 days.
- Ensure tetanus cover and give prophylactic antibiotics.
- X-ray (AP + lateral) one body region above and one body region below any wound, as well as the region involved, to look for metallic FBs.

# Blast injuries

These may follow explosions involving domestic gas, industrial sites (eg mines/mills), or bombs. Several injurious mechanisms may coexist.

## Blast wave (primary blast injury)

This is an extremely short-lived pressure wave (lasting a few milliseconds only) which expands outwards from the explosive focus. It is produced by intense compression of air at the interface of rapidly expanding hot gases. The effects can be dramatically aggravated and reinforced by reflection from solid surfaces such as buildings. Blast wave injuries are caused by three mechanisms:

- Disruption at air/tissue interfaces (especially lungs and ears, producing blast lung and tympanic membrane rupture, respectively).
- Shearing injuries at tissue/tissue interfaces, causing subserous and submucosal haemorrhage.
- Implosion of gas-filled organs, leading to perforation of the GI tract and cerebral or coronary air embolism.

**Blast winds** These are fast-moving columns of air that follow the initial blast wave. Their destructive force can be immense, leading to traumatic amputation or even complete dismemberment. Blast winds also carry debris (masonry, glass, etc.), which act as secondary missiles causing fragmentation injuries.

**Fragmentation injuries** Objects from a bomb (eg nails, casing, nuts, and bolts) or flying debris (masonry, wood, glass) cause lacerations or penetrating injuries. This is classified as *secondary blast injury*.

**Flash burns** These are usually superficial, affecting exposed skin in those close to the explosion. Smoke inhalation may also occur.

**Tertiary blast injuries** These result from individuals being thrown by the blast wind, often causing severe multiple injuries.

**Quaternary blast injuries** These include all explosion-related injuries or illnesses not due to primary, secondary, or tertiary mechanisms listed above.

**Psychological** The psychological effects of blast injury are often severe, comprising acute fear, anxiety, and the potential for chronic sequelae.

## General aspects of treatment

The principles of blast injury treatment are the same as those of other causes of major trauma (see ➲ Major trauma: treatment principles, p. 330).

Clinical features in blast injuries may be delayed, in terms of both onset and development of clinical signs. This particularly relates to lung and intra-abdominal complications; therefore, observe all patients for at least 48hr.

Search for pneumothorax (may be tension), respiratory failure/ARDS, peritonitis, abnormal neurological signs (suggesting air embolism), eardrum perforation, and anosmia (direct olfactory nerve damage). Note that ventilation of patients with blast injuries is a highly specialized area, with potential risks of producing tension pneumothoraces and air embolism.

Ensure all the patient's clothes, belongings, and any missile fragments are carefully retained, bagged, labelled, and kept secure until given to the police.

# Burns: assessment

## Types of burns

- Thermal.
- Chemical.
- Electrical (see ➲ Electrical injuries, pp. 276–7).
- Radiation (see ➲ Radiation incidents, pp. 278–9).

## History

Determine the circumstances resulting in the burn to appreciate the nature of the insult and potential risks. Consider the following questions:

- Was there an explosion? (risk of blast injuries)
- Was the fire in an enclosed space? (CO poisoning, smoke inhalation)
- What was the burning material? (Burning plastics release cyanide.)
- When was the patient removed from the fire?
- How long was the patient exposed to fire and smoke?
- Was there a history of loss of consciousness?
- Did the patient fall or jump to escape the fire? (Look for other injuries.)
- What is the patient's past medical history and tetanus status?

## Initial assessment

This proceeds with resuscitation. *Check*: Airway, Breathing, and Circulation. Particular problems associated with burns are:

- *Airway burns*: suggested by hoarseness, stridor, dysphagia, facial and mouth burns, singeing of nasal hair, soot in nostrils or on palate.
- *Spinal injury*: particularly seen with blast injuries and in those who have jumped from buildings to escape fire.
- *Breathing problems*: contracting full-thickness circumferential burns ('eschar') of the chest wall may restrict chest movement.
- *Circulatory problems*: hypovolaemic shock is a feature of severe burns and may also result from other associated injuries.

## Assessing extent

See Mersey burns app, available at https://merseyburns.com

Estimation of the percentage of body surface area burnt is difficult for non-experts. Use Lund and Browder charts appropriate for the age of the patient (see Table 8.5 and Fig. 8.25). The palmar surface of the patient's palm (not including the fingers) represents ~0.75% of body surface area. Do not include simple erythema in calculating the burn percentage.

## Assessing depth

Burn depth varies with the temperature and duration of heat applied.

*Superficial (first- and second-degree) burns* Range from minor erythema (first-degree) through painful erythema with blistering to deep partial-thickness (second-degree) burns, which do not blanch on pressure.

*Full-thickness (third-degree) burns* May be white, brown, or black and look 'leathery'. They do not blister and have no sensation.

On the day of injury, it may be difficult to distinguish deep superficial (second-degree) burns from full-thickness (third-degree) burns, but correctly making this distinction does not alter the initial management.

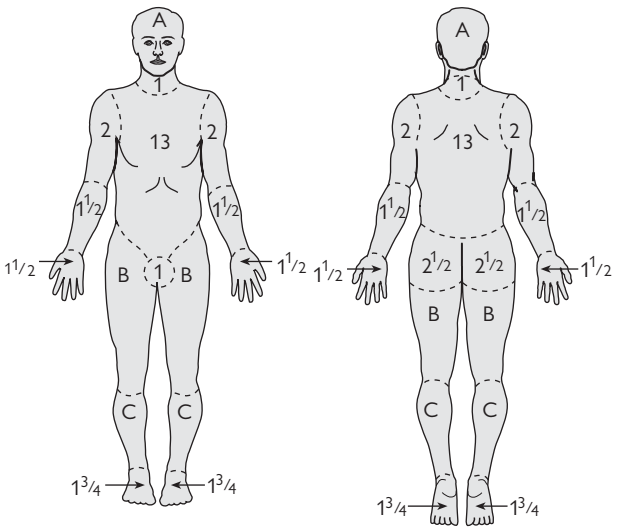


**Fig. 8.25** Assessing extent of burns—Lund and Browder charts.

**Table 8.5** Relative percentage of area affected by growth (age in years)

| | 0 | 1 | 5 | 10 | 15 | Adult |
|---|---|---|---|---|---|---|
| A: half of head | 9½ | 8½ | 6½ | 5½ | 4½ | 3½ |
| B: half of thigh | 2¾ | 3¼ | 4 | 4½ | 4½ | 4¾ |
| C: half of leg | 2½ | 2½ | 2¾ | 3 | 3¼ | 3½ |

| | | |
|---|---|---|
| Adults—rule of 9s: | Head | = 9% |
| | Each arm | = 9% |
| | Each leg | = 18% |
| | Front of trunk | = 18% |
| | Back of trunk | = 18% |
| | Perineum | = 1% |
| Infants—rule of 5s: | Head | = 20% |
| | Each arm | = 10% |
| | Each leg | = 20% |
| | Front of trunk | = 10% |
| | Back of trunk | = 10% |

# Major burns: resuscitation

## Prehospital first aid measures

- Ensure rescuer safety first—be guided by the fire crew.
- Remove the patient from the burning environment. If clothes are smouldering, apply cold water and remove them, unless adherent.
- Provide high-flow $O_2$. Cover burns in clean sheets.

## Airway and cervical spine protection

- Treat airway obstruction (see ➲ Major trauma: treatment principles, p. 330).
- Continue $O_2$ and immobilize the neck if there is any possibility of spinal injury—cervical spine imaging will be required subsequently.
- If there is any evidence of impending airway obstruction (stridor, oropharyngeal swelling—see ➲ Inhalation injury, pp. 402–3), call immediately for senior ED help and a senior anaesthetist. Urgent GA and tracheal intubation may be life-saving. Use uncut ET tubes to allow for swelling of lips and face.

## Analgesia

- Obtain IV access with two large peripheral cannulae.
- Send blood: cross-matching, FBC, COHb, U&E, glucose, and coagulation.
- Provide analgesia (IV morphine titrated according to response).
- Provide an antiemetic (eg IV cyclizine 50mg).

## Fluid resuscitation

(See the Mersey burns app, available at https://merseyburns.com)

- Give IV fluids. Start with isotonic crystalloid (eg 0.9% saline) at 2–4mL of crystalloid per kilogram body weight per percentage body surface area burnt, over the first 24hr following injury. Give half of this volume in the first 8hr.
- Check pulse, BP, and RR every 10–15min initially.
- Insert a urinary catheter and test the urine. Patients with myoglobinuria are at particularly high risk of ARF—reduce this risk by adequate fluid resuscitation. Use urine output to guide fluid therapy.
- Review the rate of IV volume replacement frequently and adjust it according to haemodynamic parameters, in order to maintain a satisfactory urine output (>50mL/hr in adults; 1–2mL/kg/hr in children).
- Some burns units prefer a colloid (eg Gelofusine® or albumin) to form a component of the initial volume replacement—follow local policy.
- Patients with full-thickness burns of a body surface area of >10% may require red cell transfusion, in addition to the above measures.

## Breathing

- Check COHb and ABG.
- Circumferential full-thickness chest burns restricting chest movement require escharotomy. Cut the burnt areas down to viable tissue to release the constriction. Cutting diathermy can be helpful to reduce the significant blood loss involved in extensive escharotomy.
- Obtain a CXR.

### The burn

- Measure the area of the burn as a percentage of the body surface area.
- Irrigate chemical burns with warmed water (see ➔ Management of smaller burns, pp. 404–5).
- Cover the burn with cling film or dry sterile sheets. Do not apply extensive burns dressings before assessment by a burns specialist.
- Involve a burns specialist at an early stage—in the UK, the National Burn Bed Bureau can help to locate a suitable bed (tel: 01384 679036), but clinicians are advised to liaise initially with their local specialized burns service.
- Ensure tetanus prophylaxis, but avoid 'routine' prophylactic antibiotics.

### The burnt patient in cardiac arrest

- Follow standard guidelines.
- Give a large bolus of IV fluid.
- If there is a strong possibility of cyanide poisoning (eg burnt plastic furniture in a house fire), give an appropriate antidote, eg dicobalt edetate (see ➔ Cyanide poisoning, p. 215).

### Vascular impairment to limbs and digits

Consider the need for longitudinal escharotomies. These are occasionally needed if ischaemia causes severe pain—get advice from a burns specialist.

### Liaison with specialist burns centres

Arrangements for liaison with burns centres vary according to the region. Many have systems in place whereby images can be transferred easily, using modern technology, within the law.

# Inhalation injury

The most common inhalation injury is smoke inhalation accompanying burns in house fires. Inhalation injury alone may be fatal, and it ↑ mortality for a given body surface area of burn. Smoke is a complex and unpredictably variable mixture of solid, liquid, and gas constituents.

## Common components of inhalation injury

- Direct thermal injury.
- Soot particles cause local injury to the cilia of the respiratory tract and obstruct small airways.
- ~85% of fire deaths are caused by CO (see ➲ Carbon monoxide poisoning, p. 216).
- *Gas products of combustion*: oxides of sulfur, nitrogen, ammonia, chlorine, hydrogen cyanide, phosgene, isocyanates, ketones, and aldehydes are highly irritative and cause laryngospasm. Some react with water in the respiratory tract, producing strong acids which cause bronchospasm, mucosal injury, and oedema.

The nature of the inhaled insult determines the site, severity, and systemic features. The upper respiratory tract can dissipate heat efficiently, so that direct thermal injury to the lower respiratory tract is rare unless steam or other hot vapours are inhaled. In the lower airway, toxic components, such as CO and oxides of sulfur, nitrogen, hydrogen cyanide, and hydrogen chloride, cause direct injury and may act as systemic poisons.

## Clinical features

Suspect smoke inhalation if any of the following features are present: exposure to smoke or fire in an enclosed space, confusion or altered/loss of consciousness, oropharyngeal burns, hoarseness/loss of voice, singed nasal hairs, soot in nostrils or sputum, wheeze, dysphagia, drooling or dribbling, and stridor.

## Investigations

*Peak flow rate* Determine this in all patients.

*ABG* Detection of hypoxia, hypercapnia, and acidosis may be helpful but does not correlate well with the severity of inhalation injury. Note that most pulse oximeters have limited value because of the difficulty in distinguishing between oxyhaemoglobin and COHb.

*CXR* Usually normal initially; later features of ARDS may develop.

*COHb* CO poisoning cannot be detected by physical examination, $SpO_2$, or $pO_2$. Either arterial or venous COHb can be measured. Clinical features correlate poorly with COHb levels. Use the nomogram shown in Fig. 8.26 to estimate COHb levels at the time of exposure. The management of CO poisoning is covered in ➲ Carbon monoxide poisoning, p. 216.

*ECG* CO binds to myoglobin three times more avidly than to Hb, and by affecting the myocardium, it may produce arrhythmias, ischaemia, or even MI.

*Fibre-optic bronchoscopy, xenon lung scanning, V/Q scans, or lung function testing* May be required subsequently to assess lung problems due to inhalational injury.

## Management

Signs of upper airway problems (facial burns, stridor, dysphagia, drooling, ↓ consciousness) indicate the need for *early tracheal intubation* by an experienced doctor with appropriate training. Mucosal swelling in the oropharynx and epiglottis can progress rapidly. A surgical airway (➲ Airway obstruction: surgical airway, p. 336) can be difficult due to burnt skin and loss of landmarks. Flexible bronchoscopy may help to assess thermal injury to the airway and help intubation. *Assisted ventilation with PEEP* may be indicated.

Give the highest possible concentration of humidified $O_2$. Hyperbaric $O_2$ may be indicated for CO poisoning but remains controversial (➲ Carbon monoxide poisoning, p. 216).

If bronchospasm occurs, give *nebulized β2-agonist* (salbutamol 5mg) via an $O_2$-powered nebulizer. ↑ in microvascular permeability leads to pulmonary oedema 2–3 days after the injury, and pneumonia after 7–14 days. Pulmonary fibrosis is common amongst survivors.

Inadequate IV fluid resuscitation is associated with greater pulmonary oedema. Burnt patients who have smoke inhalation need larger amounts of IV fluids to maintain cardiac and urine output.

Inhalation of hydrogen cyanide from smouldering plastics (eg polyurethane) results in rapid systemic absorption. Measurement of blood cyanide concentration is difficult and takes several hours. *Cyanide poisoning* may be suggested by severe metabolic acidosis, a high lactate level, and an ↑ anion gap. Consider cyanide antidotes (➲ Cyanide poisoning, p. 215), but they are potentially toxic, so do not use blindly. There is no proven benefit from steroid therapy.

## Nomogram of decay of COHb with time

This nomogram (see Fig. 8.26) allows back-calculation estimation of the likely peak COHb level. It will considerably under-read for children and patients who received a high prehospital $FiO_2$.

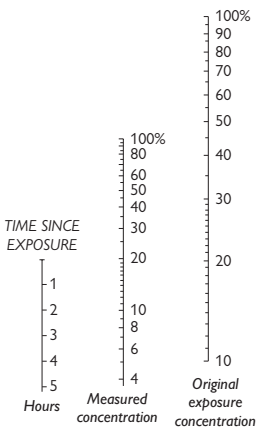


**Fig. 8.26** Nomogram of decay of COHb with time.

# Management of smaller burns

## Assessment

(See ➲ Burns: assessment, p. 398.)

## First aid measures

Separate the patient and the burning agent. Cool the affected area with copious quantities of cold water, but beware of hypothermia in infants and young children.

## Need for admission

Admit patients with large burns or significant smoke inhalation for IV fluids, resuscitation, and analgesia. In the UK, the National Burn Bed Bureau will search for an appropriate bed (tel: 01384 215576) after discussion with the local burns unit. Also refer for admission burns of suspected NAI origin and patients who would be unable to cope at home (eg an elderly person or if living in difficult social circumstances).

## Referral to a burns specialist

Refer patients with the following:

- Airway burns.
- Significant full-thickness burns, especially over joints.
- Burns >10%.
- Significant burns of special areas (hands, face, perineum, feet).

## The burn wound

- Leave *full-thickness burns* uncovered, and refer to a specialist.
- Do not de-roof *partial-thickness burns* with blistering—consider simple aspiration. Most can be cleaned and covered with an appropriate dressing (see below).

*Hand burns* Consider covering with soft paraffin inside a polythene bag or glove sealed at the wrist, changed after 24hr. Simple paraffin/tulle dressings are an alternative—follow local policy. Elevate to minimize swelling. Avoid silver sulfadiazine cream, except on specialist advice.

*Facial burns* Leave uncovered, or consider application of soft paraffin.

*Eye burns* Check VA, and refer to a specialist, with prior irrigation if chemical burns (see ➲ Corneal trauma, pp. 554–5).

*Perineal and foot burns* Burns in these areas should be referred for burns unit admission, as they require specialist nursing and wound care.

## Burns dressings

The ideal burns dressing is sterile and non-adherent and encourages wound healing in a moist environment. The diversity of dressings available reflects the fact that this ideal dressing remains elusive. Senior ED nursing staff will advise on local preference and policy. Accumulation of fluid means that many dressings need to be changed at ~48hr—often this is appropriately done at a GP surgery.

## Analgesia and tetanus

Unless there is a contraindication and/or if the patient is elderly, NSAID is appropriate and an effective analgesia for many burns which do not require admission. Ensure prophylaxis against tetanus.

## Burns in children and non-accidental injury

Unintentional burns are common in children—use the opportunity to offer advice regarding injury prevention. A minority of burns may result from NAI. Suspect NAI (see ➲ Child abuse: head injuries, wounds and burns, p. 760) and seek senior help in the following situations:

- When the explanation does not fit the burn.
- Late presentation.
- Other suspicious injuries.
- Stocking-and-glove distribution scalds (± sparing of the buttocks)—this implies forced immersion in hot water.
- Circular full-thickness burns of ~0.75cm in diameter may represent cigarette burns.

## Chemical burns

*Initial assessment* is notoriously difficult. Alkalis tend to produce more severe burns and can continue to penetrate, even after initial irrigation.

*Treat* chemical burns with copious irrigation with water, continued for at least 20min in alkali burns. Check skin pH and irrigate until it is normal.

### *Hydrofluoric acid burns*

Hydrofluoric acid is used industrially in a number of processes. Contact with the skin causes particularly severe burns, often with significant tissue damage and severe pain. This is because hydrofluoric acid rapidly crosses lipid membranes and penetrates the tissues deeply where it releases the highly toxic fluoride ion. Fluoride ions may gain access to the circulation and produce a variety of systemic problems by a variety of mechanisms, including interfering with enzyme systems and producing hypocalcaemia by binding to $Ca^{2+}$.

*Manage* hydrofluoric acid burns as follows:

- Provide copious lavage to the affected skin, then apply iced water (this provides better pain relief than calcium gluconate gel).
- Call a plastic surgeon at an early stage.
- Check serum $Ca^{2+}$ and $Mg^{2+}$, and U&E.
- Record an ECG and place on a cardiac monitor.
- Treat hypocalcaemia.

### *Cement burns*

Wet cement or concrete can cause chemical burns due to alkali contact. These are usually partial thickness but may be full thickness. They often occur when wet cement falls into a work boot, but the burn is not initially noticed. Involve a specialist at an early stage.

### *Phenol burns*

Phenol may be absorbed through the skin, resulting in systemic toxicity and renal failure. Get advice from NPIS (see ➲ National Poisons Information Service, pp. 188–9).

# Crush syndrome

## Background

Crush syndrome covers a spectrum of conditions characterized by skeletal muscle injury (rhabdomyolysis).

Causes include:

- Direct injuries and severe burns causing muscle damage.
- Compartment syndromes—'true' crush injuries produced by entrapment or 'self-crushing' (eg an unconscious individual from drug overdose or alcohol excess lying on a hard surface). A vicious cycle is established where ↑ muscle compartment pressure obstructs blood flow, the muscles become ischaemic and oedematous, and further ↑ compartment pressure and ↓ blood flow lead to more ischaemia and muscle cell death.
- Non-traumatic causes—metabolic disorders (diabetic states, ↓ $K^+$, ↓ $PO_4^{3-}$), myxoedema, neuroleptic malignant syndrome, myositis due to infection, or immunological disease.
- Exertional—from undue exertion, grand mal fitting, rave dancing (particularly associated with ecstasy or cocaine use), often complicated by hyperthermia.

## Clinical features

Adopt a high index of suspicion. Symptoms depend on the underlying cause, but muscle pain, tenderness, and swelling may not be present at the time of admission. In the lower limbs, the condition is commonly confused with DVT. The classic compartment syndrome with pain on passive muscle stretching and sensory deficits may take several days to develop and can pass unnoticed. The presence of distal pulses does not rule out compartment syndrome.

## Investigations

↑ creatine phosphokinase (CPK) levels reflect muscle damage. Check U&E, $PO_4^{3-}$, $Ca^{2+}$, and urate. 70% have myoglobinuria and pigmented granular casts (urinary stix tests do not differentiate between Hb and myoglobin). However, absence of myoglobinuria does not exclude rhabdomyolysis, as myoglobin clears rapidly from plasma and its presence in urine depends upon the release rate, the degree of protein binding, GFR, and urine flow. If DIC is suspected, check a coagulation screen.

## Treatment

*Local problems*

Refer urgently to the orthopaedic team if compartment syndromes are suspected. If the difference between intra-compartmental and diastolic pressures is >30mmHg, fasciotomy, excision of dead muscle, and even distal amputation may be required. These procedures may induce life-threatening electrolyte shifts, bleeding, local infection, and later generalized sepsis.

*Systemic complications*

Severe metabolic complications start after revascularization. Hyperkalaemia may be life-threatening (see ➲ Hyperkalaemia, pp. 170–1). Hypocalcaemia is common initially, but rarely symptomatic.

*Acute renal failure*

This can be produced by pre-renal, renal, and obstructive elements. Following restoration of circulation or release from entrapment, fluid leaks into damaged areas, ↓ circulating plasma volume. Intracellular muscle contents enter the circulation, and myoglobin and urate crystals can block the renal tubules. This process is aggravated by the ↓ intravascular volume and associated metabolic acidosis. DIC and drugs which inhibit intra-renal homeostatic mechanisms (eg NSAIDs and β-blockers) may also contribute.

Prompt correction of fluid deficits and acidosis (often with CVP monitoring) and establishing a good urinary flow are essential. Alkalinization of the urine may be required—early use of mannitol has been advocated but can cause pulmonary oedema if renal impairment is already present. If renal failure occurs, dialysis may be needed, but prospects for renal recovery are good.

Chapter 9

# Wounds, fractures, and orthopaedics

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# The approach to wounds

Wounds often have medicolegal implications—record notes thoroughly, legibly, and accurately (see ➲ Note keeping, pp. 6–7). Resuscitation is the initial priority for the seriously wounded patient. Stop bleeding by applying direct pressure.

## History

Key questions are:

- *What caused the wound?* (Knives/glass may injure deep structures.)
- *Was there a crush component?* (Considerable swelling may ensue.)
- *Where did it occur?* (Contaminated or clean environment?)
- *Was broken glass (or china) involved?* (If so, obtain an X-ray.)
- *When did it occur?* (Old wounds may need delayed closure + antibiotics.)
- *Who caused it?* (Has the patient a safe home to go to?)
- *Is tetanus cover required?* (See ➲ Tetanus prophylaxis, p. 424.)

## Examination

Consider and record the following:

- *Length*: preferably measure. If not, use the term 'approximately' in the notes.
- *Site*: use diagrams whenever possible (rubber stamps recommended). Consider taking digital photographs, particularly for open fractures, in order to ↓ the risk of infection by disturbing the wound as little as possible prior to surgery.
- *Orientation*: vertical, horizontal, or oblique.
- *Contamination*: by dirt or other FBs—may be obvious.
- *Infection*: either localized or spreading—is a feature of delayed presentations and is associated in particular with certain specific injuries (eg 'reverse fight bites'—see ➲ Bite wounds, pp. 420–1).
- *Neurological injury*: test and record motor and sensory components of relevant nerves. Be aware that complete nerve transection does not automatically result in complete loss of sensation—some feeling is likely to be preserved (particularly in the hand). Assume that any altered sensation reflects nerve injury.
- *Tendons*: complete division is usually apparent on testing. Partial tendon division is easily missed unless the wound is carefully examined—the tendon may still be capable of performing its usual function. Look in the wound whilst moving the relevant joint, and attempt to re-create the position of the injured part at the time of injury (eg clenched fist) to bring the injured structures into view.
- *Vascular injury*: check for distal pulses.
- *Depth*: wounds not fully penetrating the skin are 'superficial'. Do not try to judge the depth of other wounds before exploration. In some circumstances (eg neck wounds), exploration is not appropriate in the ED.
- *Type of wound*: inspection allows wounds to be described, helping to determine the mechanism of trauma (blunt or sharp injury), and hence the risk of associated injuries. The crucial distinction is whether a wound was caused by a sharp or blunt instrument. If in doubt, avoid any descriptive term and simply call it a 'wound'. This avoids inaccuracy and courtroom embarrassment! Use the terms as described in ➲ Forensic classification of wounds, p. 411.

# Forensic classification of wounds

Expert forensic evaluation of injury is outside the remit of the ED specialist, but a simple understanding helps to avoid incorrect use of terminology with associated confusion (and sometimes embarrassment).

**Incised wounds** Caused by sharp injury (eg knives or broken glass) and characterized by clean-cut edges. These typically include 'stab' wounds (which are deeper than they are wide) and 'slash' wounds (which are longer than they are deep).

**Lacerations** Caused by blunt injury (eg impact of the scalp against the pavement or an intact glass bottle), the skin is torn, resulting in irregular wound edges. Unlike most incised wounds, tissues adjacent to laceration wound edges are also injured by crushing and will exhibit evidence of bruising.

**Puncture wounds** Most result from injury with sharp objects, although a blunt object with sufficient force will also penetrate the skin.

**Abrasions** Commonly known as 'grazes', these result from blunt injury applied tangentially. Abrasions are often ingrained with dirt, with the risk of infection and, in the longer term, unwanted and unsightly skin 'tattooing'. Skin tags visible at one end of the abrasion indicate the edge of skin last in contact with the abrading surface and imply the direction in which the skin was abraded.

**Burns** See ➲ Burns: assessment, p. 398.

**Bruises** Bruising reflects blunt force (crush) injury to the blood vessels within the tissues, resulting in tender swelling with discoloration—sometimes localized bleeding collects to form a *haematoma*. The term '*contusion*' is sometimes used as an alternative for bruise—it has no particular special meaning (or value). Record the site, size, colour, and characteristic features of any bruising. It is impossible to determine the exact age of a bruise from its colour. However, yellow colour within a bruise implies (except in the neonate) that it is >18hr old.

**Scratches** These may comprise either a 'very superficial incision' or a 'long, thin abrasion'—leave the distinction to an expert.

## Interpersonal violence—medicolegal implications

Victims of violence frequently attend the ED for treatment of their injuries. Some patients (particularly those who have suffered domestic violence) may not provide an accurate account of how the injuries occurred and may not seek involvement of the police. Classical defence wounds include:

- Isolated ulnar shaft fracture as the arm is raised up for self-protection.
- Incised wounds on the palmar aspects of the palms and fingers sustained in attempts to protect against knife attack.

In cases where the police are involved and where injuries are serious or extensive, the police may arrange photographs and/or examination by a forensic physician (police surgeon) to document injuries. Most ED patients who have suffered violence do not see a forensic physician. Therefore, notes made by ED staff may be important medicolegally.

# Further assessment of skin wounds

## Investigations

*X-ray* if there is suspicion of fracture, involvement of joint, penetration of a body cavity, or an FB. Specify if an FB is being sought, to allow appropriate views and exposure. Most metal (except aluminium) and glass objects of >1mm diameter will show up on X-ray. Some objects (eg wood) may not—USS may demonstrate these.

- *Note: X-ray all wounds from glass that fully penetrate the skin.*
- During X-ray, use radio-opaque markers (eg paper clip) taped to the skin to identify the area of concern.
- *Wound swabs* for bacteriology are unhelpful in fresh wounds, but obtain them from older wounds showing signs of infection.

By far, the most important 'investigation' is:

### *Wound exploration under appropriate anaesthesia*

This allows full assessment and thorough cleaning of wounds that extend fully through the skin. Do not explore the following wounds in the ED:

- Stab wounds to the neck, chest, abdomen, or perineum.
- Compound fracture wounds requiring surgery in theatre.
- Wounds over suspected septic joints or infected tendon sheaths.
- Most wounds with obvious neurovascular/tendon injury needing repair.
- Other wounds requiring special expertise (eg eyelids).

Obtain relevant X-rays beforehand. Adequate anaesthesia is essential—in adults, LA (eg 1% plain lidocaine) is often suitable (see ➲ Local anaesthesia, pp. 292–3), but document any sensory loss first (if there is altered sensation, presume nerve injury and refer for formal exploration in theatre). Do not inject LA into the edges of an infected wound—it will not work in that acidic environment and it may spread the infection. GA or topical agents may be the preferred option for treating some wounds in young children.

Inspect wounds for FBs and damage to underlying structures. Most problems with wound exploration relate to bleeding. If it proves difficult to obtain a good view:

- Obtain a good light and an assistant. The assistant retracting on a stitch placed on either side of the middle of the wound allows full exposure.
- Press on any bleeding point for ≥1min, then re-examine. Lidocaine with adrenaline (see ➲ Lidocaine (previously known as 'lignocaine'), p. 293) may help with profusely bleeding scalp wounds.
- If bleeding continues, consider a tourniquet for up to 15min. Consider a sphygmomanometer BP cuff inflated above the systolic pressure (after limb elevation for 1min) on the limbs. Take extreme care if considering using digital tourniquets. Never leave a patient alone with a tourniquet on, lest it is forgotten. Ensure removal of the tourniquet afterwards. Record the time of application and removal.

If these measures fail, refer for specialist exploration in theatre. Do not blindly 'clip' bleeding points with artery forceps, for fear of causing iatrogenic neurovascular injury. Sometimes, small blood vessels in the subcutaneous tissues can be safely ligated using an appropriate absorbable suture (eg 4/0 or 6/0 Vicryl (braided polyglactin) or Dexon).

# Puncture wounds and foreign bodies

## Puncture wounds

Puncture wounds often involve the foot, after treading on a nail. Examine for neurovascular injury, then obtain an X-ray to look for FBs. If significant foreign material is present or there is associated fracture, tendon injury, or neurovascular deficit, refer for formal exploration and cleaning in theatre under a bloodless field. Otherwise:

- Irrigate and clean other wounds under LA where possible (consider nerve blocks). For wounds to the sole of the foot, this may be impractical. As a compromise, immersing the foot in warm antiseptic (eg povidone iodine solution) for 15min is traditional.
- Apply a dressing and advise review/follow-up with the GP as appropriate.
- Ensure adequate tetanus cover (see ➲ Tetanus prophylaxis, p. 424).
- Prescribe simple analgesia.
- Strongly consider prophylactic oral antibiotic cover (eg co-amoxiclav).

Some puncture wounds may become infected despite treatment. This may be due to retained foreign material in the wound. *Pseudomonas osteitis* is an uncommon, but recognized, complication of puncture wounds to the foot, particularly where a nail has gone through training shoes to cause the wound. Refer infected wounds for formal exploration and irrigation.

## Approach to foreign bodies

FBs within soft tissues can cause pain, act as a focus for infection, or migrate and cause problems elsewhere. Try to remove FBs from recent wounds where possible, particularly if lying near a joint (but if the FB is within a joint, refer to orthopaedics for formal exploration and removal). Finding FBs is frequently difficult without a bloodless field and good light. It may be appropriate to leave some FBs such as gunshot deeply embedded in buttock soft tissues (antibiotic cover advised). However, most FBs of any size not removed in the ED warrant specialist consideration.

### *Late presentation*

Patients not infrequently present with symptoms relating to (suspected) FBs (eg thorn) under scabbed or old healed wounds, days or weeks following the injury. USS may help to confirm the presence of an FB and determine its position (marking the skin can help later exploration, which is usually best performed by specialist teams).

## Fishhooks

Smaller fishhooks that are relatively superficially embedded can sometimes be pulled back and removed through the entry wound (advancing a hollow needle alongside the hook to cover the barb may help). In other cases, it may be necessary to push a fishhook onwards (under LA), and thus out through the skin—wire cutters can then cut through the hook below the barb and allow release. Wear eye protection when doing this.

Other methods, such as the 'string-yank' technique, carry potential risks of causing additional tissue damage—leave to experienced practitioners.

*Note*: do not attempt to remove fishhooks from the eyes in the ED—refer to the ophthalmologist.

# Wound cleaning and prophylactic antibiotics

## Wound cleaning

Thoroughly clean all wounds, irrespective of whether closure is contemplated, in order to ↓ the risk of infection. The standard agent used for wound cleaning is 0.9% (normal) saline, preceded, where appropriate, by washing using tap water—many studies have shown the benefits of thorough tap water irrigation. Aqueous chlorhexidine or 1% cetrimide solutions are sometimes used. Do not use hydrogen peroxide or strong povidone iodine solutions, as these carry other risks, including causing damage to healthy tissue. Wounds ingrained with dirt may respond to pressure saline irrigation (19G needle attached to a 20mL syringe) or may require to be scrubbed with a toothbrush (use goggles to ↓ the chance of conjunctival 'splashback'). Devitalized or grossly contaminated wound edges often need to be trimmed back (debrided), except on the hand or face. If dirt or other foreign material is visible despite these measures, refer to a specialist who may choose to leave the wound open.

## Antibiotic prophylaxis

Most wounds do not require prophylactic antibiotics. Thorough cleaning is the best way of preventing infection. After cleaning and closure, consider oral antibiotic prophylaxis for certain wounds—compound fingertip fractures and wounds in those at extra risk (eg valvular heart disease, post-splenectomy). Co-amoxiclav has activity against anaerobes and is appropriate for bites and heavily contaminated or infected wounds—leave these wounds open. Give oral antibiotics for penetrating injuries which cannot be properly cleaned (see ➲ Puncture wounds and foreign bodies, p. 413). Although the scientific basis is debatable, antibiotics are frequently used for wounds >6hr old and complex intraoral wounds and in workers at high risk (gardeners, farmers, fishermen).

There is no evidence of any benefit from the application of topical antibiotics or irrigation using antibiotic solutions.

### *Tetanus prophylaxis*

Consider prophylaxis against tetanus whenever assessing skin wounds (see ➲ Tetanus prophylaxis, p. 424).

# Wound closure

## Three recognized types of wound closure

*Primary closure*

- Surgical closure (by whatever physical means) soon after injury.

*Secondary closure*

- No intervention: heals by granulation (secondary intention).

*Delayed primary closure*

- Surgical closure 3–5 days after injury.

If there is no underlying injury or FB, aim to treat fresh wounds by primary closure as soon as possible. Accurate opposition of wound edges provides the best cosmetic outcome.

Wounds not usually suitable for primary closure in the ED include:

- Stab wounds to the trunk, perineum, and neck.
- Wounds with associated tendon, joint, or neurovascular involvement.
- Wounds with associated crush injury or significant devitalized tissue/skin loss.
- Other heavily contaminated or infected wounds.
- Most wounds >12hr old (except clean facial wounds).

## Methods of closure

*Sutures* If in doubt, sutures are usually the best option (see ➲ Sutures, p. 416).

*Steri-Strips™* Adhesive skin closure strips allow skin edges to be opposed with even distribution of forces. They are inappropriate over joints, but useful for pretibial lacerations where the skin is notoriously thin and sutures are likely to 'cut out'. Before application, make Steri-Strips™ stickier by applying tincture of benzoin to dry skin around the wound. Leave 3–5mm gaps between Steri-Strips™. (See also see ➲ Pretibial lacerations, p. 495.)

*Skin tissue glue* Particularly useful in children with superficial wounds and scalp wounds. After securing haemostasis, oppose the dried skin edges before applying glue to the wound. Hold the skin edges together for 30–60s to allow the glue to set. Ensure that glue does not enter the wound. Do not use tissue glue near the eyes or to close wounds over joints.

*Staples* Quick and easy to apply, these are particularly suited to scalp wounds. Staple-removers are required for removal, so consider giving a staple-remover to the patient to take to the nurse at the GP surgery.

# Sutures

*Sutures (otherwise known as 'stitches' or 'ties')* are the traditional and most commonly used method to achieve primary closure. Oppose the skin aiming for slight eversion of the wound edges, using strong non-absorbable inert monofilament suture material attached to curved cutting needles (eg Prolene, polypropylene, or nylon), with knots tied on the outside. Interrupted simple surgical knots tied using instruments are relatively easy and economical of thread and have a low risk of needlestick injuries. Specialized continuous sutures (eg subcuticular) are not appropriate for wounds in the ED. The size of thread used and the time to removal vary according to the site. Use absorbable sutures (eg Vicryl) on the lips and inside the mouth. Absorbable sutures may also be used to close subcutaneous tissues to ↓ the chance of haematoma and infection. Suture choice and time to removal are given in Table 9.1.

**Table 9.1** Suture choice and time to removal

| Part of body | Suture and size | Time to removal |
|---|---|---|
| Scalp | 2/0 or 3/0 non-absorbable† glue or staples | 7 days |
| Trunk | 3/0 non-absorbable† | 10 days |
| Limbs | 4/0 non-absorbable† | 10 days |
| Hands | 5/0 non-absorbable | 10 days |
| Face | 5/0 or 6/0 non-absorbable | 3–5 days* |
| Lips, tongue, mouth | Absorbable, eg 6/0 Vicryl/Dexon | – |

† One size smaller may be appropriate for children.

* Sutures may be replaced with Steri-Strips™ at 3 days.

## Key points when suturing

The technique of a basic instrument tie is shown in Fig. 9.1.

- Tie sutures just tight enough for the edges to meet.
- Do not close a wound under tension.
- Handle the skin edges with toothed forceps only.
- Avoid too many deep absorbable sutures.
- Mattress sutures are useful on some deep wounds, but avoid on the hands and face.
- Dispose of sharps as you use them—do not make a collection.
- Use strategic initial sutures to match up obvious points in irregular wounds.
- If a suture does not look right—take it out and try again.
- If it still does not look right—get help!

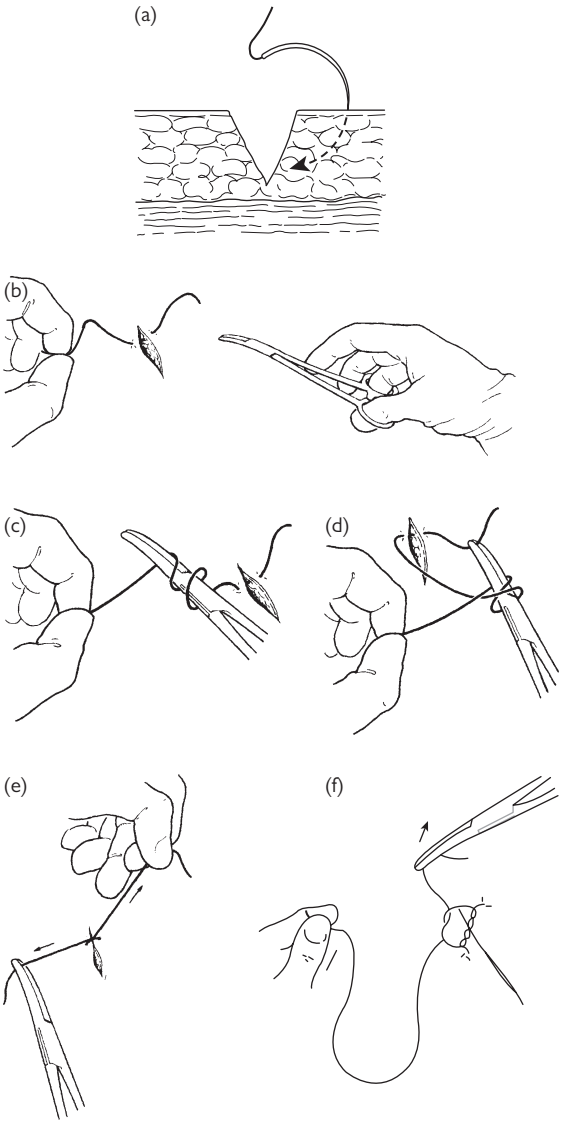


**Fig. 9.1** (a) to (f) Basic instrument tie technique.

# Wound aftercare

## Dressings

A large variety of dressings are available, with little scientific evidence to help choose between them—the choice depends upon personal preference/prejudice and local departmental policy. A dry, non-adherent dressing will protect most wounds from inadvertent contamination in the first few days. Dressings are not usually necessary for facial and scalp wounds. Beware circulatory problems resulting from encircling dressings/bandages applied too tightly to digits or other parts of limbs.

Burns dressings are considered in ➲ Management of smaller burns, pp. 404–5.

## General advice

Advise to keep wounds clean and dry for the first few days. Limb wounds require rest and elevation for the first 24hr. After this, restrict movements to avoid undue stress causing the suture line to open up (especially where the wound is over a joint). Warn all patients to return if features of infection develop (redness, ↑ pain, swelling, fever, red streaks up the limb). Approximate times to suture removal are shown in Table 9.1—adjust these according to the situation. For example, sutures over joints are sensibly left for 14 days to avoid dehiscence. Similarly, sutures may need to be left in for longer where wound healing may be delayed (eg diabetes mellitus, the elderly, malnourished, and those on steroids). Local policy will dictate where suture removal occurs (usually at the GP surgery). Ideally, discharge with illustrated instructions about wound care and suture removal. This may particularly help patients with memory impairment or those under the influence of alcohol.

## Specific advice

Provide advice about when to return to work. If there is a question of personal safety or safety of the public or work colleagues, advise return to usual duties only once the wound has healed and sutures removed. This particularly applies to food handlers and some machine operators.

## Review and delayed primary closure

Arrange review at ~36hr of heavily contaminated wounds, infected wounds not needing admission, and other wounds at particular risk. Evidence of infection are ↑ T°, wound discharge and erythema, ascending lymphangitis, and regional lymphadenopathy. Systemic symptoms or evidence of spreading infection despite oral antibiotics are indications for admission for wound toilet, rest, elevation, and IV antibiotics.

Treat other wounds deemed initially to be at less risk of infection, but not suitable for primary closure, with cleaning, light packing/dressing, and review at 3–5 days. At this stage, delayed primary closure after wound cleaning and debridement under appropriate anaesthesia can be considered.

Do not employ ‘loose closure’ to manage contaminated wounds. The technique has all the risks of infection combined with a poor cosmetic result.

# Infected wounds and cellulitis

## Wound infection after injury

Although prompt treatment with cleaning and primary closure will ↓ the risk, any wound may become infected. The risk of infection is ↑ by:

- Contamination (eg bites) and foreign material (including excess sutures).
- Haematoma.
- Devitalized tissue.
- Poor nutrition and ↓ immunity (eg steroid therapy).

Pain is usually the first clue to wound infection. Many soft tissue infections occur without an obvious wound (see ➲ Cellulitis and erysipelas, p. 545).

*Examination* Indicates the extent of the infection. Erythema and tenderness limited to the area around the wound suggest localized infection. Swelling and fluctuation are evidence of a collection of pus. Remove all sutures, together with pus and devitalized tissue, under appropriate anaesthetic. Send wound swabs for culture. Consider the possibility of a retained FB—X-ray/explore as appropriate. After thorough cleaning, leave the wound open, cover with a dressing, and arrange review with a dressing change in 36hr. Consider the need for antibiotics (eg co-amoxiclav), particularly for cellulitis, the immunocompromised, and patients at particular risk (eg those with prostheses and valvular heart disease).

*Consider admission* (For rest, elevation, analgesia, wound/blood cultures, and IV antibiotics) in patients with one or more of the following:

- A red line spreading proximally (ascending lymphangitis).
- Regional (sometimes tender) lymphadenopathy.
- Pyrexia >38°C.
- Systemic upset.

Soft tissue crepitus is ominous, suggesting gas-forming organisms (see ➲ Gas gangrene, p. 247).

## Infected hand wounds

A particularly common problem is an infected wound on the dorsum of the hand over a metacarpophalangeal joint (MCPJ) after a punch injury. These are often bite wounds, presenting late with infection in the region of the joint. Refer for exploration/washout in theatre and antibiotics (see ➲ Specific bites and stings, pp. 422–3).

## Infected facial wounds

Take infected wounds of the cheek very seriously. They pose a significant threat of sepsis spreading intracranially, resulting in papilloedema and ophthalmoplegia due to cavernous sinus thrombosis. Adopt a low threshold for referring for admission and IV antibiotics.

## Infected surgical wounds

Infection of a recent surgical wound after a planned procedure is a relatively common complication. In addition to the possible threat to life, wound infection can have disastrous implications as far as the success of the preceding operation is concerned (eg hernias may recur). Contact the team which performed the surgery as soon as possible, to allow the surgeon to treat the complication.

# Bite wounds

## Bites and infection

Bites cause contaminated puncture wounds, contaminated crush injuries, or both. All carry a high risk of bacterial infection, some also a risk of viral or other infections (eg rabies).

Bacterial infection is particularly likely in:

- Puncture wounds (cat/human bites).
- Hand wounds, wounds >24hr old.
- Wounds in alcoholics, diabetics, or the immunocompromised.

Bacteria responsible include: streptococci, *Staphylococcus aureus, Clostridium tetani, Pasteurella multocida* (cat bites/scratches), *Bacteroides*, and *Eikenella corrodens* (human bites).

## Approach

Establish what the biting animal was and how long ago and where the bite occurred. Obtain X-rays if a fracture, joint involvement (look for air), or a radio-opaque FB (tooth) is suspected.

## Management of bite wounds

### *Cleaning*

Explore fresh bite wounds under appropriate anaesthesia, and debride and clean thoroughly with 'normal' saline (or by washing using tap water). Refer significant facial wounds and wounds involving tendons or joints to a specialist.

### *Closure*

Cosmetic considerations usually outweigh the risks of infection for most facial wounds, so aim for primary closure. Elsewhere, choose between primary or delayed primary closure—the latter may be preferred in bite wounds affecting the limbs, due to an ↑ risk of infection. Do not close puncture bite wounds that cannot be satisfactorily irrigated.

### *Antibiotic prophylaxis*

Deciding whether or not to employ prophylactic antibiotics for bite wounds can be difficult and is controversial. Many practitioners advocate prophylactic antibiotics for all bite wounds. One approach is to give antibiotics for patients with any of the following:

- Puncture bites.
- Crush injuries with devitalized tissues.
- Bites to the hand, wrist, or genitals.
- Bites that are primarily closed.
- Bites from humans, cats, and rats.
- Bitten patients with immunocompromise (immunosuppressed, diabetes, post-splenectomy, rheumatoid arthritis) or prosthetic joints.

Co-amoxiclav is an appropriate broad-spectrum agent, effective against streptococci, staphylococci, *Pasteurella*, and *Eikenella*. Alternatives for adult patients allergic to penicillin/amoxicillin include doxycycline + metronidazole or (especially if pregnant) ceftriaxone alone. In children, azithromycin + metronidazole may be an option.

*Tetanus*

Bite wounds are tetanus-prone. Give prophylaxis accordingly (see ➲ Tetanus prophylaxis, p. 424).

*Rabies*

(Covered fully in ➲ Rabies, p. 257.)

Rabies results after the 'bullet-shaped' RNA rhabdovirus present in the saliva of infected animals is transmitted to humans via a mucous membrane or skin break. After thorough cleaning, refer all patients who might have been in contact with a rabid animal to an infectious diseases specialist. Obtain further help from the Virus Reference Department in London (tel: 0208 327 6017). The long incubation period of the rabies virus (14–90 days) allows successful post-exposure prophylaxis at even a relatively late stage, according to agreed guidelines.

*Hepatitis and HIV*

Consider the possible risks of hepatitis B, hepatitis C, and HIV in anyone who presents following a human bite, and treat accordingly (see ➲ Needlestick injury, p. 425). Quantifying risks can be difficult, particularly, for example, in 'reverse fight bites' (see ➲ Specific bites and stings, pp. 422–3) where the other person involved may be unknown. If in doubt, take a baseline blood sample for storage (to allow later testing, if necessary) and provide cover against hepatitis B.

## Treatment of infected bites

Most bacterial infections occur >24hr after injury and are due to staphylococci or anaerobes. Pain, inflammation, swelling ± regional lymphadenopathy within 24hr suggest *P. multocida* infection. Take wound swabs of all infected wounds, then treat with cleaning, elevation, analgesia, and antibiotics. Oral co-amoxiclav and outpatient review at ~36hr is appropriate for localized wound infection with no systemic symptoms and no suspected underlying joint involvement. Refer patients with spreading infection for IV antibiotics and admission.

Septicaemia is uncommon after bite injury but has been reported with the Gram –ve bacillus *Capnocytophaga canimorsus*, previously known as Dysgonic Fermenter 2 (DF-2). Infection produces severe illness with septicaemia and DIC, often in the immunocompromised (splenectomized individuals, diabetics, or alcoholics). Take wound swabs and blood cultures, then give IV antibiotics and refer.

## Prevention of dog bites

Injury prevention measures aimed at preventing children from being bitten include legislation relating to 'dangerous dogs' and education. Children may be taught the following:

- To treat dogs with respect.
- To avoid disturbing a dog that is sleeping, eating, or feeding puppies.
- To avoid shouting or running in the presence of a dog.
- Not to approach or play with unfamiliar dogs.

# Specific bites and stings

## Human bites and 'fight bites'

Many human bites occur 'in reverse', when an individual punches another in the mouth, causing wounds on the hand over the MCPJs. Underlying joint involvement is common and may progress to septic arthritis, unless treated aggressively with exploration, irrigation, and antibiotics. Refer all patients for this. Consider hepatitis B, hepatitis C, and HIV; give appropriate prophylaxis (see ➲ Needlestick injuries, p. 425), and arrange counselling.

## Tick bites

Ticks are recognized vectors of a number of exotic diseases worldwide. In the UK, patients often present with embedded sheep ticks. Remove ticks by gentle traction, with blunt forceps applied as close to the skin as possible. Avoid traditional folklore methods of removal, which may cause the tick to regurgitate, promoting infection. In areas where Lyme disease is endemic (see ➲ Ticks, p. 241), routine antibiotic prophylaxis is not recommended (℞ http://www.nice.org.uk), but there is evidence (in those aged ≥12y) that a single dose of doxycycline 200mg PO may ↓ the risk of developing Lyme.

## Insect bites

Minor local reactions are common. Treat with ice packs, rest, elevation, analgesia, and antihistamines (eg chlorphenamine 4mg tds PO or a non-sedating alternative such as loratadine 10mg od PO). Occasionally, insect bites may be complicated by cellulitis and ascending lymphangitis requiring antibiotics (see ➲ Infected wounds and cellulitis, p. 419).

## Wasp and honey bee stings

These may cause local reactions or anaphylaxis—treat promptly (see ➲ Anaphylaxis, pp. 44–5). Flick out bee stings left in the skin. Treat local reactions as for insect bites.

## Jellyfish stings and fish spines

*Most jellyfish* in UK coastal waters are relatively harmless. Wash the injured part in sea water or saline, and lift off any remaining tentacles with a towel or stick. Consider applying an ice pack as analgesia. Use of vinegar is no longer recommended.

*Fish spines* (typically Weever fish) produce a heat-labile toxin, which may be neutralized by immersion in hot water for 30min. Occasionally, tiny parts of the fish spines become embedded and cause long-term irritation. Localizing and removing these tiny FBs is difficult, so refer to an appropriate expert.

## Contact with other wild animals

Contact with rats' urine may cause leptospirosis (Weil's disease) ➲ Leptospirosis (Weil's disease, p. 249). Provide prophylactic doxycycline to anyone who presents following an episode of significant exposure (eg immersion in river water or sewage). Unusual bites may pose specific threats, which infectious disease specialists will advise about (eg monkey bites may cause herpes simplex infection—give prophylactic oral aciclovir). Bats may carry rabies (➲ Rabies, p. 257).

## Snake bites

The European adder (*Vipera berus*) is the only native venomous snake in the UK. It is grey/brown, with a V-shaped marking behind the head and dark zig-zag markings on the back. Most bites occur in summer. Venom is injected by a pair of fangs. The venom contains enzymes, polypeptides, and other low-molecular weight substances. Only 50% of bites cause envenomation.

*Features*

Envenomation causes pain and swelling—look for two puncture marks, 1cm apart. Vomiting, abdominal pain, diarrhoea, and hypotension may follow. A small proportion of patients develop severe systemic symptoms within minutes of the bite.

*Investigations*

Check urine; perform an ECG (check QTc), and take blood for: FBC, U&E, LFTs, coagulation screen, and D-dimer.

*Treatment*

- Prehospital: rest (and avoid interference with) the bitten part—do not try to 'suck out' fluid or apply tourniquets.
- Clean and expose the wound; give analgesia and IV fluids for hypotension.
- Treat anaphylaxis urgently according to standard guidelines (see ➲ Anaphylaxis, pp. 44–5).
- Ensure tetanus cover. Latest evidence suggests that prophylactic antibiotics are not usually required.
- Obtain specific advice from NPIS (see ➲ National Poisons Information Service, pp. 188–9)—a toxicology specialist will advise regarding the appropriate use/dose of antivenom.
- Use of antivenom does carry some risk but is indicated for anaphylaxis-like reaction to the venom, signs of systemic envenoming (abdominal pain and vomiting), hypotension for >10min, WCC >20 × $10^9$/L, ECG abnormalities, elevated CK, metabolic acidosis, pulmonary oedema, spontaneous bleeding, or significant limb swelling (eg past the wrist for bites on the hand or past the ankle for bites on the foot, within 4 hr).
- Observe for least 24hr all patients who have any symptoms after a snake bite.

# Tetanus prophylaxis

Spore proliferation and toxin production are likely in heavily contaminated wounds with devitalized tissue (see ➲ Tetanus, p. 246). However, any wound (including a burn) is a risk—always ensure tetanus prevention.

## Tetanus immunization programme

Standard active immunization involves an initial course of three IM or deep SC doses of 0.5mL of tetanus toxoid (formalin-inactivated toxin), given at monthly intervals, starting at 2 months of age, followed by booster doses at 4 and 14y. In the UK, combined tetanus/diphtheria/inactivated polio vaccine has replaced the previous tetanus/diphtheria vaccine for adults and adolescents. Inadequate immunity against tetanus is particularly likely in immigrants, the elderly, patients with ↓ immunity, and those who have refused vaccination.

**Anti-tetanus prophylaxis** (See https://www.gov.uk)
Follow the Department of Health guidelines. The need for tetanus immunization after injury depends upon a patient's tetanus immunity status and whether the wound is 'clean' or 'tetanus-prone' or 'high-risk, tetanus-prone': The following are regarded as 'tetanus-prone':

- Puncture wounds in a contaminated environment.
- Certain animal bites (especially non-domestic or farm animals).
- Wounds containing FBs.
- Wounds or burns with systemic sepsis.
- Open fractures.

'High-risk, tetanus-prone' wounds are 'tetanus-prone' wounds plus one of:

- Heavy contamination with soil/manure.
- Wounds/burns with extensive devitalized tissue.
- Wounds/burns requiring surgical intervention that is delayed by >6hr.

Do not give tetanus vaccine if there is a past history of a severe reaction—give tetanus immune globulin (TIG). Pregnancy is not a contraindication to giving tetanus prophylaxis.

*Children who are up-to-date and adults who have had booster within 10y*

Do not give any immediate vaccine or human TIG. Encourage children to complete a course in future, as planned.

*Initial course complete, but last booster >10y ago or children aged 5–10y who had an initial priming course, but no preschool booster*

No vaccine is required for clean wounds, but give a reinforcing dose of tetanus vaccine for tetanus-prone wounds. For high-risk, tetanus-prone wounds, give a booster and human TIG (250–500U IM) in a different site.

*Has not received an adequate priming course (of ≥3 vaccines or immunization status unknown or uncertain)*

Give an immediate reinforcing dose of vaccine for all wounds. For tetanus-prone wounds (whether high risk or not), give a dose of human TIG at a different site.

The TIG dose is usually 250U IM, but give 500U if >24hr have elapsed since the injury or if there is heavy contamination or following burns.

# Needlestick injury

Many infective agents have been transferred by needlestick: blastomycosis, brucellosis, cryptococcosis, diphtheria, Ebola fever, gonorrhoea, hepatitis B, hepatitis C, herpes zoster, HIV, leptospirosis, malaria, mycobacteriosis, mycoplasmosis, Rocky Mountain spotted fever, scrub typhus, sporotrichosis, *Staphylococcus aureus, Streptococcus pyogenes*, syphilis, toxoplasmosis, and TB.

In practice, the principal risks are hepatitis B and C and HIV. The risk of acquiring hepatitis B following a needlestick injury from a carrier has been estimated at 2–40%. All hospital workers should be immunized against hepatitis B. The risk of hepatitis C is believed to be 3–10%. In contrast, the risk of acquiring HIV after a needlestick injury with a HIV +ve source is much less (estimated at 0.2–0.5% but may be higher if significant volumes are injected). There is a small (~0.03%) risk of HIV transmission after mucocutaneous exposure (ie exposure of cuts, abrasions, and mucous membranes, including the eye). The (small) risk of acquiring HIV following a needlestick injury from a person with known HIV may be reduced further by post-exposure prophylaxis, but time is of the essence (see ➲ Management below). No proven post-exposure prophylaxis currently exists for hepatitis C. Preventing needlestick injuries and exposure to these viruses is therefore crucial.

### Management

- Wash the wound with soap and water.
- Ensure tetanus cover.
- Ensure hepatitis B cover—if not previously immunized, consider hepatitis B immunoglobulin and start an active immunization course (give the first vaccine in the ED, and arrange subsequent doses). If previously immunized, check antibody titres. If satisfactory, take no further action. If low, give a booster vaccine. If very low, both give the immunoglobulin and start a vaccine course. Many local needlestick policies advise obtaining informed consent from the source patient, prior to taking blood to check the hepatitis and HIV status. In practice, the identity of the source patient is not always clear—do not withhold hepatitis B prophylaxis if there is any doubt.
- If the source patient is known to be (or suspected of being) HIV +ve, follow local guidelines and/or refer immediately to an infectious diseases specialist to discuss post-exposure prophylaxis and follow-up. Follow guidance on 🖱 http://www.dh.gov.uk. Current combined prophylaxis therapy is one Truvada® tablet od (comprising 245mg tenofovir + 200mg emtricitabine) + one raltegravir tablet 400mg bd. It is most effective if started within an hour of exposure but may be worth considering up to 72hr. Prophylaxis has side effects, although raltegravir is better tolerated than the previously used Kaletra®. Involve both a health care worker and a local expert in deciding whether or not to start prophylaxis. Either way, advise the patient to use barrier contraception and not to give blood as a donor until subsequent HIV seroconversion has been ruled out.
- Take baseline blood for storing (serology for possible future testing), and in the case of a possible HIV source patient, also take blood for FBC, U&E, LFTs, and amylase.
- If the incident occurred in hospital, report it to occupational health.

# How to describe a fracture

Clear, precise, complete descriptions of fractures aid accuracy and save time when referring patients.

## System for describing fractures

- State the age of the patient and how the injury occurred.
- If the fracture is open, state this first (and the Gustilo type—see ➲ Open (compound) fractures, pp. 428–9).
- Name the bone (specify right or left and, for the hand, whether dominant).
- Describe the position of the fracture (eg proximal, supracondylar).
- Name the type of fracture (eg simple, spiral, comminuted, crush).
- Mention any intra-articular involvement.
- Describe the deformity (eg displacement, angulation) from the anatomical position.
- State the grade or classification of the fracture (eg Garden IV).
- State the presence of any complications (eg pulse absent, paraesthesiae, tissue loss).
- Other injuries and medical problems.

## Example using this system

'A 29y-old ♂ motorcyclist with an open fracture of the left humerus. It is a transverse fracture of the humeral shaft and is Gustilo type I open and minimally displaced with no neurovascular compromise … '

## Type of fracture

*Simple* Single transverse fracture of bone with only two main fragments.

*Oblique* Single oblique fracture with only two main fragments.

*Spiral* Seen in long bones as a result of twisting injuries, only two main fragments.

*Comminuted* Complex fracture resulting in >2 fragments.

*Crush* Loss of bone volume due to compression.

*Wedge* Compression to one area of bone resulting in a wedge shape (usually applied to vertebra).

*Burst* Comminuted compression fracture with scattering of fragments.

*Impacted* Bone ends driven into each other.

*Avulsion* Bony attachment of ligament or muscle is pulled off.

*Hairline* Barely visible lucency with no discernible displacement.

*Greenstick* Incomplete fracture of immature bone follows angulatory force, with one side of the bone failing in compression, the other side in tension.

*Torus/buckle* Kinking of the metaphyseal cortex follows axial compression.

*Pathological* Fracture due to underlying disease (eg osteoporosis, metastasis, Paget's disease).

*Stress* Certain bones are prone to fracture after repetitive minor injury.

*Plastic deformation* Deformation beyond the elastic limit, but below the fracture point, results in bending of bone ± microfractures (these are not apparent on X-ray) (see Fig. 9.2).

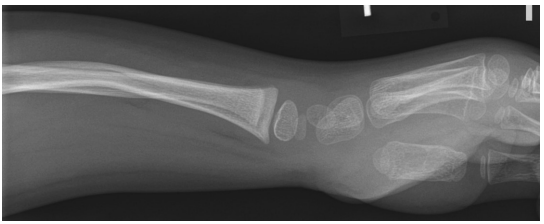

**Fig. 9.2** Plastic deformation of radius and ulna shafts in a 5y old.

*Fracture–dislocation* Fracture adjacent to, or in combination with, a dislocated joint.

*Deformity* Describe deformity using the terms displacement, angulation, and rotation.

*Displacement ('translation')* Describe the relative position of two bone ends to each other. Further describe the direction that the distal fragment is displaced from the anatomical position (eg volar, lateral). Also estimate the degree of apposition of the bone ends (eg 50%).

*Angulation* This is usually described in terms of the position of the point of the angle (eg posterior angulation means that the distal fragment is pointing anteriorly). This can sometimes be confusing. Although a little long-winded, one way to avoid confusion is to describe the direction in which the distal part points, relative to the anatomical position (eg a Colles' fracture may be described as a 'fracture of the distal radius in which the distal fragment points dorsally'). Try to measure the angle on X-ray.

*Rotation* Describe the degree of rotation from the anatomical position, in terms of the direction (eg external or internal rotation) in which the distal part has moved.

## Long bone anatomy

Each long bone has a shaft or diaphysis with an epiphysis at each end. Whilst the bone is growing, these are separated by an epiphyseal growth plate and this narrows down into the bone shaft. The transitional area of bone is the metaphysis. In addition to these landmarks, the femur and humerus have a ball-shaped head, a narrower neck, and, at the lower ends, a widened area consisting of, respectively, the medial and lateral condyles of the femur and the medial and lateral epicondyles of the humerus. Fractures proximal to these areas of the femur and humerus are termed supracondylar. Intercondylar fractures involve the central, distal, and juxta-articular portions. Fractures of the proximal femur between the greater and lesser trochanters are termed intertrochanteric.

# Open (compound) fractures

Open (or compound) fractures occur when a fracture is open to the air through a skin wound. They incur a risk of infection and can be associated with gross soft tissue damage, severe haemorrhage, or vascular injury. Treat as orthopaedic emergencies requiring rapid assessment and treatment.

## Classification of open injuries

The Gustilo classification of open injuries is as follows:

*Type I* Open fracture where the wound is <1cm long and appears clean.

*Type II* Open fracture where the wound is >1cm but is not associated with extensive soft tissue damage, tissue loss, or flap lacerations.

*Type IIIA* Either an open fracture with adequate soft tissue coverage of bone despite extensive soft tissue damage or flap laceration or any fracture involving high-energy trauma or bone shattering regardless of wound size.

*Type IIIB* Open fracture with extensive soft tissue loss, periosteal stripping, and exposure of bone.

*Type IIIC* Compound fracture associated with vascular injury needing repair.

## Management

Provide adequate fluid replacement, analgesia, splintage, antibiotics, and tetanus prophylaxis prior to surgical treatment. Rapidly complete the following steps whilst contacting the orthopaedic service:

- Treat life-threatening injuries before limb-threatening injuries. Do not be distracted from initial priorities by dramatic distal limb injuries.
- Control obvious haemorrhage by direct manual pressure whilst commencing IV fluids and/or blood replacement.
- Give analgesia in the form of incremental IV opioids (see ➲ Analgesics: morphine, p. 286).
- Once analgesia is adequate, correct obvious severe deformities with gentle traction and splint. Certain dislocations may require immediate correction. Remove obvious contaminants if possible (eg large lumps of debris or plant matter).
- 'Routine' wound swabs for bacteriological culture are no longer recommended. They do not alter management and are poor predictors of deep infection.
- If available, take digital photographs of the wound (this helps to avoid the need for repeated inspection by different clinicians).
- Irrigate with saline, then cover the wound with a sterile moist dressing (eg saline-soaked pads). Immobilize the limb in a POP backslab. Do not repeatedly inspect the wound, as this greatly ↑ the risk of infection. Once dressed and in POP, leave injuries covered until surgery.
- Give IV antibiotics (eg co-amoxiclav 1.2g or cefuroxime 1.5g according to local policy). Consider adding gentamicin or metronidazole if the wound is grossly contaminated.
- Give tetanus toxoid if indicated, and give human TIG if gross wound contamination is present (see ➲ Tetanus prophylaxis, p. 424).

*Record the presence/absence of distal pulses/sensation and recheck frequently.*

### Limb salvage or amputation

Orthopaedic surgeons often face a difficult decision as to whether or not a limb can be salvaged. Gustilo type IIIC injuries are associated with a high rate of amputation. The Gustilo classification alone is not always an accurate predictor of outcome—other tools have been developed to assist. For example, the Mangled Extremity Severity Score takes into account the extent of skeletal and soft tissue damage, the extent and severity of limb ischaemia, associated shock, and age.

## Dislocations (and subluxations)

A dislocation has complete loss of congruity between articular surfaces. A subluxation implies movement of the bones at the joint, but with some parts of the articular surface still in contact. Describe dislocations in terms of the displacement of the distal bone. For example, most shoulder dislocations are 'anterior', with the humeral head lying in front of the glenoid. Aim to reduce dislocations as soon as possible to prevent neurovascular complications, ↓ the risk of recurrence, and ↓ pain. In general, X-ray (to identify the exact dislocation ± associated fracture) before attempting a reduction. Exceptions to this principle are:

- Dislocations associated with considerable neurovascular compromise requiring urgent intervention (eg some ankle fracture–dislocations).
- Uncomplicated patellar dislocations.
- Uncomplicated mandibular dislocations.
- Some patients with (very) recurrent shoulder dislocations where there may be longer-term concerns regarding radiation exposure.
- Some patients with collagen disorders resulting in hypermobility (eg Ehlers–Danlos syndrome) and unusual/recurrent dislocations without significant trauma.
- 'Pulled elbow' in young children (see ➲ Subluxation of the radial head ('pulled elbow'), p. 750).

Use analgesia/sedation/anaesthesia appropriate to the dislocation and the individual circumstances. For example, patellar dislocations often reduce under Entonox®, finger proximal interphalangeal joint (PIPJ) dislocations with LA digital nerve blocks (see Fig. 9.3), and shoulder dislocations with IV analgesia ± sedation, whereas posterior hip dislocations typically require manipulation under GA. Unless there are exceptional circumstances, X-ray after manipulation to confirm adequate reduction and also to check for fractures which may not have been apparent on initial X-rays.

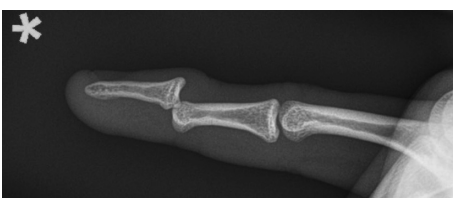

**Fig. 9.3** Dorsal dislocation of finger DIPJ.

# Casts and their problems

## Plaster of Paris (POP)

POP is cheap and easy to use and can be moulded. Usually applied in the form of a bandage or multiply folded as a supporting slab (see Fig. 9.4). Disadvantages are susceptibility to damage (POP rapidly disintegrates if wet) and it takes up to 48hr for larger casts to dry fully after application. Cut slabs to shape prior to use, and apply over wool roll and stockinette. Mould with the palms (not the fingertips) to avoid point indentation of plaster.

## Resin (fibreglass) casts

More costly, but lighter and stronger than POP and more resistant to water or other damage. Made of cotton or fibreglass impregnated with resin that hardens after contact with water. Sets in 5–10min, maximally strong after 30min. Most resin casts are more difficult to apply and remove. Being more rigid and harder to mould, there is an ↑ risk of problems from swelling or pressure necrosis. Remove/cover any sharp edges on the cast.

## Complications of casts

Give all patients discharged with casts clear written instructions (including a contact phone number) to return if they develop pain or other symptoms in the immobilized limb. Formal cast checks within 24hr are only required if there is particular concern about swelling. Simple swelling or discoloration of fingers or toes usually responds to elevation and simple exercises.

*Is the cast too tight?*

Act immediately upon suspicion of circulatory compromise from a cast. Look for the 'five Ps': pain, pallor, paraesthesiae, paralysis, and 'perishing cold'. If any of these are present:

- Elevate the limb.
- Cut wool and bandages of the backslab until the skin is visible along the whole length of the limb.
- Split full casts and cut through all layers until the skin is visible along the whole length of the limb.

Any undivided layers will continue to obstruct the circulation until released. If this action fails to completely relieve the symptoms, contact orthopaedic and vascular surgery staff immediately, as angiography and urgent surgical intervention may be required. Note that compartment syndrome may occur in the presence of normal pulses.

*Is the cast too loose?*

Test by trying to move the plaster longitudinally along the limb. Replace excessively loose or damaged casts, unless there is an outweighing risk of fracture slippage.

*Local discomfort*

If there is local pressure discomfort (eg over a malleolus), cut a window in the cast to allow direct inspection of the skin. Trim or replace plasters which restrict movement unduly.

### Cast removal

Standard POP and selected resin casts may be removed with plaster shears. Use a plaster saw only after instruction in its proper use. In both cases, be careful to avoid skin damage. Note that some newer resin materials enable casts to be removed by being 'unwrapped', which has the advantage of being less stressful for children and parents can remove the cast themselves after a specified period of immobilization.

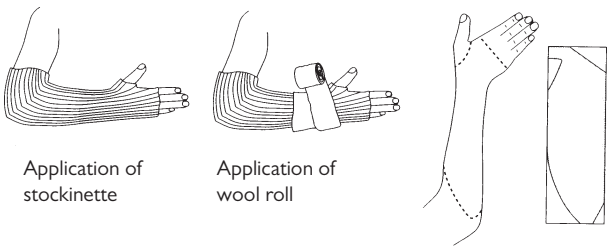


**Fig. 9.4** Application of a Colles' backslab POP.

## Fractures and osteoporosis

Osteoporosis is an important factor in a significant proportion of fractures seen in the ED. The following fractures are frequently (but by no means exclusively) associated with osteoporosis:

- Colles' fracture (see ➲ Colles' fracture, pp. 454–5).
- Fracture of the surgical neck of the humerus (see ➲ Humeral neck/head fracture, p. 472).
- Lumbar spine vertebral fracture.
- Fracture of the neck of femur (see ➲ Hip fractures, pp. 484–5).
- Pubic rami fracture (see ➲ Pelvic fractures, pp. 480–1).

Patients with post-menopausal osteoporosis may be treated with a bisphosphonate in an attempt to ↓ the risk of future fractures, but do not commence this treatment in the ED.

# Soft tissue injuries

## Sprains

These occur from overstretching and tearing of ligaments. Sprains vary from sparse fibrous tears to complete disruption of a ligament complex. The results are pain, tenderness, and swelling. Ligament sprains are traditionally graded into three types, although distinguishing clinically between them may be difficult:

- *First-degree sprains* involve minor tearing of fibres and are stable.
- *Second-degree sprains* are more severe partial sprains—there may be some resultant slight ligamentous laxity, but with a definite end-point on stressing.
- *Third-degree sprains* reflect completely torn ligaments causing significant laxity—patients sometimes report hearing a 'snap' at the time of injury.

Ligament sprains are very common, but there is a lack of reliable evidence about treatment. Prolonged immobilization seems to be detrimental to recovery, due to stiffness, muscle wasting, and loss of proprioception. Painful minor sprains respond well to traditional measures—ice, compression with elastic support/strapping, elevation, and progressive mobilization as soon as symptoms allow. Complete ligament rupture can be relatively painless, but, if associated with gross joint instability, may require surgical repair. Associated haemarthroses require orthopaedic appraisal, aspiration, and, often initially, protection and immobilization in POP.

## Strains

Indirect injury involving muscle–tendon units may be classified in a similar fashion to ligament sprains. Pain on palpation over the site of injury is also reproduced by passive stress or active contraction of the affected muscle unit. Sometimes, a palpable defect may be apparent in complete ruptures (which typically occur at the musculotendinous junction). Treat minor strains similarly to sprains; consider specialist review for complete ruptures, some of which may require surgical repair.

## Direct muscle injuries

These result from direct impact causing local pain, bruising, and soft tissue swelling. Note that associated bone contusions can occur such as in the perimeniscal areas of the knee (these are visible on MRI). Treat minor injuries with ice, analgesia, and early mobilization within the limits of symptoms. For more significant injuries, consider and treat according to possible risks of compartment and crush syndromes (with rhabdomyolysis) and large haematomas (see ➲ Haematomas, p. 432).

## Haematomas

Blood can accumulate as a result of traumatic disruption of the vascular structures in bone, muscle, or soft tissues. Deceptively large volumes of blood can be accommodated within the soft tissue planes of the chest wall or thigh. In the presence of massive visible bruising of the torso or a limb, check for shock and measure Hb and Hct. Perform a coagulation screen. Blood transfusion may be necessary. Treat minor haematomas with compression dressings and ice. Large haematomas or supervening infection requires selective surgical drainage, haemostasis, and antibiotics.

# Other soft tissue problems

## Myositis ossificans

After some muscle or joint injuries, calcification can occur within a haematoma, leading to restriction of movement and loss of function. Frequent sites include calcification within a quadriceps haematoma (eg following a rugby injury) where inability to flex the knee >90° at 48hr after injury indicates an ↑ risk of myositis ossificans. Other sites include the elbow and femur. Passive stretching movements of joints may be implicated in the development of myositis ossificans. This particularly applies at the shoulder, hip, and knee where passive exercises are performed for spasticity following paraplegia or head injury.

*Treatment* involves immobilizing the limb or joint for a period of weeks, under specialist supervision. Early excision is contraindicated, as it is invariably followed by massive recurrence, but delayed excision (after 6–12 months) can improve function.

## Tendonopathy (tendonitis/tenosynovitis)

This includes a wide range of conditions, some of which may have medicolegal implications ('overuse' or 'repetitive strain' injury). Examples include:

- *Classic tenosynovitis*: swelling along a tendon sheath, with pain on passive stretching or upon attempted active movement against resistance (eg de Quervain's tenosynovitis—see ➲ Soft tissue wrist injuries/problems, p. 459).
- *Chronic paratendonitis* (eg affecting Achilles tendon): swelling around the tendon with localized pain and tenderness.
- *Tendon insertion*: inflammation causes epicondylitis in adults (see ➲ Soft tissue elbow/arm problems, pp. 466–7) and traction apophysitis in children (➲ Osteochondritis, pp. 730–1).

Appropriate initial treatment usually includes rest, immobilization, and NSAID. Later, consider involving an appropriate specialist (eg physiotherapist or hand therapist).

## Bursitis

Inflammation of bursae most frequently affects the subacromial, olecranon, and prepatellar bursae. There is localized swelling and tenderness—generalized joint effusion and/or tenderness along the whole joint line suggests an alternative diagnosis. In many instances, bursitis is non-infective and responds to rest and NSAID. Significant warmth and erythema raise the possibility of an infective origin. In this case, consider aspiration for bacteriological culture, and provide antibiotics (eg flucloxacillin or clarithromycin).

## Other problems

Other causes of joint or limb pain with no specific history of trauma in the adult patient include stress fractures, cellulitis and other infections, osteoarthritis and other forms of acute arthritis, and nerve compression (eg carpal tunnel syndrome). Apparently atraumatic limb pain in children may present with limping—likely underlying causes vary according to the age (see ➲ The limping child, pp. 726–7).

# Physiotherapy in the ED

Most simply, 'physiotherapy' includes advice given following minor injury. At the other extreme, it encompasses assessment and treatment of selected patients by skilled, experienced physiotherapists. It is valuable for the ED to have close links with a physiotherapy unit, preferably with designated physiotherapists in the ED responsible for referrals.

## 'Everyday' physiotherapy

Minor soft tissue injuries are amongst the most commonly seen problems in EDs. Once bony injury has been excluded (clinically and/or radiologically), ensure that patients are discharged with clear, consistent advice:

- Be clear and specific about what the patient is to do.
- Prescribe/provide appropriate slings, boots, and bracings, with advice on how to manage these.
- Give additional written instructions for reinforcement (eg ankle sprains, minor knee injuries), as patients forget much verbal advice.
- Set a realistic time limit after which the patient should seek further attention if their symptoms are not improving.

*Protection/Rest/Ice/Compression/Elevation (PRICE)*

The traditional basic framework for treatment of acute soft tissue injury.

*Protection*

Protect the injured part (eg using crutches or a walking stick).

*Rest*

With most acute injuries, advise a period of 24–48hr rest after an injury.

*Ice*

Ice is often advocated both in immediate first aid of soft tissue injuries and in subsequent treatment. Crushed ice cubes wrapped in a damp cloth (to avoid direct contact with the skin) placed against an injured joint may ↓ swelling and pain. Do not apply for more than 10–15min at a time. Repeat every few hours initially. A cold pack or bag of frozen vegetables can be used (do not refreeze if for consumption!).

*Compression*

Despite a lack of evidence, injured joints (particularly the ankle) are often treated with compression. The easiest is an elasticated tubular bandage (eg Tubigrip®), either single or doubled over. If provided, advise not to wear it in bed and to discard as soon as convenient. If not provided, explain why or the patient may feel inadequately treated. Avoid support bandages for elbow and knee injuries—they can be uncomfortable and 'dig in', and in the case of the knee, they may affect venous return and ↑ the chance of DVT.

*Elevation*

Initially, advise elevation of injured limbs above horizontal to ↓ swelling and discomfort. This is particularly important in hand or foot injuries.

*Exercise*

Start gentle, controlled exercises for any injured joint as soon as symptoms allow. Demonstrate what is expected and confirm that the patient understands what to do and how to progress rehabilitation appropriately.

## Formal physiotherapy

Physiotherapists are trained in the rehabilitation and treatment of injury, based on a detailed knowledge of relevant limb and joint anatomy, biomechanics, and physiology. In the ED, physiotherapy staff are valuable in assessment and treatment of acute soft tissue injuries, patient education and advice, and the provision of appropriate mobility aids after injury (particularly in the elderly). Many EDs have adopted extended scope/advanced practitioner roles to further enable the therapist to deliver timely, appropriate, and cost-effective care—this includes triage, assessment, requesting/interpreting imaging, and managing soft tissue injuries and problems.

*Utilizing physiotherapists*

In order to make the best use of physiotherapy services, follow these guidelines:

- Refer early if required for acute injury. Ideally, aim for the patient to be seen for initial assessment the same day, so treatment needs can be properly planned. It should be acknowledged, however, that on occasions, it is necessary for the initial pain and swelling to settle before a definitive assessment can be made.
- Discuss the problem and treatment options with the physiotherapy staff prior to referral, where possible.
- Use the physiotherapy service for selected (rather than all) cases.
- Do not use the physiotherapy department to simply offload difficult or problematic patients.

Physiotherapists have a range of different treatments at their disposal, which typically focus upon regaining the range of movement and mobility and improving strength and proprioception.

# Fracture clinic and alternatives

Traditionally, any patient who was discharged from ED with a fracture and/or significant soft tissue limb injury was followed up in the fracture clinic (or hand clinic). This system resulted in a large number of patients being seen at the wrong time by the wrong person (and often having to wait a long time for a consultation). A ground-breaking reorganization has been pioneered by a team at Glasgow Royal Infirmary (http://www.fractureclinicredesign.org). The new, more efficient system is now widespread. Patients with a range of relatively minor self-limiting fractures and injuries are discharged with advice ± splints (see Injuries discharged with advice sheet and no follow-up, pp. 436–7). All other patients are not allocated specific fracture clinic appointments but are referred to a 'virtual fracture clinic' where follow-up is timed and tailored to meet individual needs (as outlined in Virtual fracture clinic, p. 436).

## Virtual fracture clinic

At the virtual fracture clinic, patients' notes and X-rays are reviewed and a decision is made about future management to suit their needs, which is then conveyed to the patient via a telephone discussion. This allows patients to have targeted treatment to suit their needs—to be discharged with advice, be brought back to see particular specialists (eg knee surgeon), have further tests (eg MRI), or return for review at a more appropriate time (rather than just attend the next available clinic).

## Injuries discharged with advice sheet and no follow-up

Provide all patients who are discharged with an advice sheet and no follow-up a telephone number to call if they are worried, need advice, or would like to be seen for hospital follow-up. Examples of advice sheets used in Glasgow may be found at http://www.fractureclinicredesign.org

*Torus/buckle wrist fracture* (See Forearm and wrist injuries, p. 751 and Fig. 9.5.)

Aim to discharge most children with minor torus/buckle wrist fractures with a removable splint to wear for 3 weeks, together with advice regarding avoidance of sport for subsequent 2 weeks.

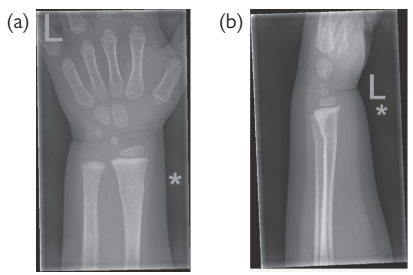


**Fig. 9.5** Buckle fracture in a 2y old.

*Undisplaced radial head/neck fracture* (See Elbow injuries, pp. 462–3.)

Discharge patients with undisplaced radial head/neck fractures with a sling to wear whilst there is significant pain and advice not to forcibly extend the elbow and to expect recovery within 3–6 weeks.

*Mallet finger injury* (See ➲ Mallet finger with fracture, p. 444)
In the absence of a large bony fragment, discharge patients with a mallet finger injury with an appropriate splint and advice sheet outlining the importance of wearing the splint and keeping the finger straight for 6–8 weeks.

*Clavicle fracture in children* (See ➲ Clavicle and AC joint injuries, p. 471 and Fig. 9.6.)
Aim to discharge children with undisplaced or minimally displaced clavicle fractures with a sling for 2 weeks and written advice to avoid contact sports for 6 weeks.

*Base of fifth metatarsal fracture* (See ➲ Foot fractures and dislocations, pp. 504–5 and Fig. 9.7.)
Discharge patients with a base of fifth metatarsal (MT) fracture with crutches, an appropriate walking boot for 3–5 weeks, crutches, and advice.

*Fifth metacarpal neck fracture* (See ➲ Hand fractures and dislocations, pp. 444–5 and Fig. 9.8.)
Discharge patients with a fifth metacarpal (MC) neck fracture and no or minor angulation with buddy strapping and an advice sheet, with an expectation of recovery within 6 weeks or so.

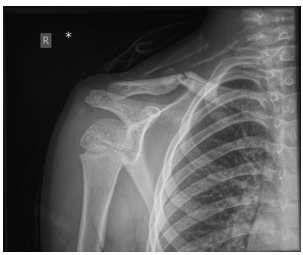


**Fig. 9.6** Clavicle fracture in 7y old.

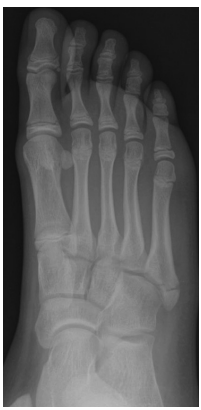

**Fig. 9.7** Base of fifth MT fracture.

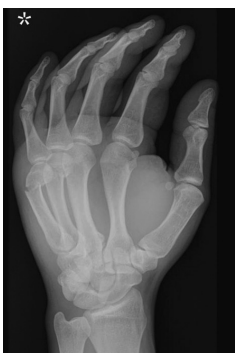

**Fig. 9.8** Angulated fracture of fifth MC neck.

# Approach to hand injuries

## History

Determine and record whether the patient is right- or left-handed and their occupation and social situation. These points may have treatment implications (eg an elderly person living alone with little social support may not cope at home after a dominant hand injury).

Suspect patients presenting with wounds on the dorsum of the hand over the index, middle, ring, or little finger MC heads of having sustained a human bite ('fight bite'), whatever history is provided (see ➲ Specific bites and stings, pp. 422–3).

## Terminology

To avoid confusion, always refer to fingers by name, not number (index, middle, ring, little).

*Use*: palmar (or volar), dorsal, radial, ulnar (not anterior, posterior, lateral, medial).

## Bones of the hand and wrist

There are 14 phalanges and five MCs. Name the MCs according to the corresponding fingers (ie thumb, index, middle, ring, and little)—this avoids confusion. There are eight carpal bones arranged in two rows. The proximal row (radial to ulnar) comprises the scaphoid, lunate, triquetral, and pisiform (see Fig. 9.9). The distal row (radial to ulnar) are the trapezium, trapezoid, capitate, and hamate.

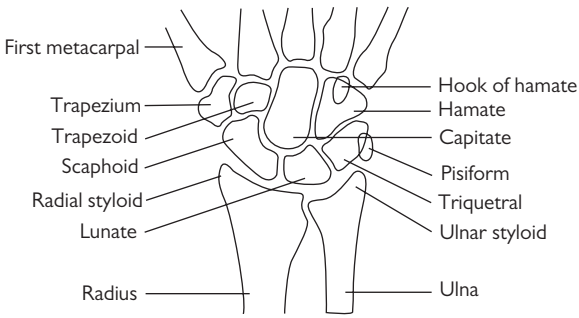


**Fig. 9.9** AP view of a normal wrist.

**Anatomy of finger extensor tendon** (See Fig. 9.10.)

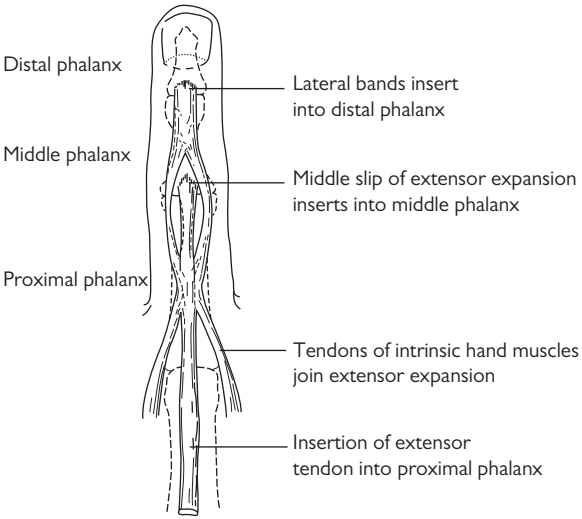


**Fig. 9.10** Anatomy of finger extensor tendon.

**Anatomy of finger flexor tendon** (See Fig. 9.11.)

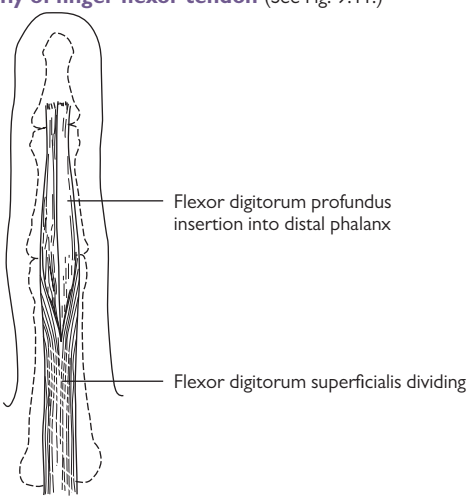


**Fig. 9.11** Anatomy of finger flexor tendon.

# Clinical signs of hand injury

## Examination of hand injuries

Injury to the hand's rich collection of nerves, blood vessels, and tendons results in considerable functional deficit. Assess carefully, taking into account the hand anatomy and clinical patterns of injury (see Figs 9.12 and 9.13).

### Specific signs of injury (See Table 9.2.)

**Table 9.2** Specific signs of injury

| | |
|---|---|
| Median nerve | ↓ sensation in the palm over radial three-and-half digits, unable to abduct thumb against resistance |
| Ulnar nerve | ↓ sensation in palmar and dorsal one-and-half fingers, little finger flexed (non-functioning lumbrical) |
| | Unable to cross index and middle fingers |
| | ↓ abduction/adduction, weak pinch grip (Froment's sign) |
| Radial nerve | ↓ sensation in dorsum first web space |
| | (No motor branches in hand, but proximal injury results in inability to extend wrist) |
| Digital nerve | ↓ sensation along radial or ulnar half of digit distally—note that some sensation is usually preserved, even with significant nerve injuries |
| Superficial flexor | Hold other fingers straight (immobilizing all deep flexors), then unable to flex PIPJ (unreliable for index finger). Also, ~10% of individuals do not have a flexor superficialis tendon to the little finger |
| Deep flexor | Unable to flex DIPJ |
| Extensors | Complete division prevents extension (at DIPJ causes mallet deformity) |
| | Central slip division causes Boutonnière deformity |
| | In recent trauma, hold PIPJ at 90° over table edge, and try to extend against resistance—DIPJ hyperextends in central slip division (Elson's test) |
| Deformity | A small amount of rotational deformity of a digit (typically associated with a spiral/oblique MC or finger fracture) can have a dramatic effect upon long-term hand function (see Fig. 9.14)—check carefully to ensure that there is no abnormal overlapping of fingertips in the palm on making a fist |

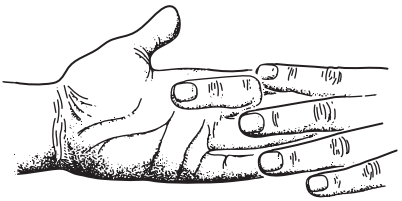

**Fig. 9.12** Testing superficial flexor finger tendon.

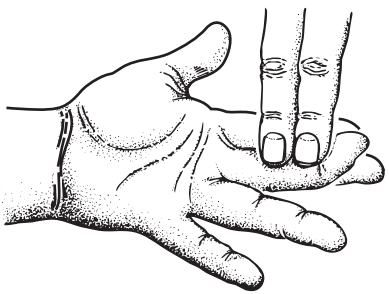

**Fig. 9.13** Testing deep flexor finger tendon.

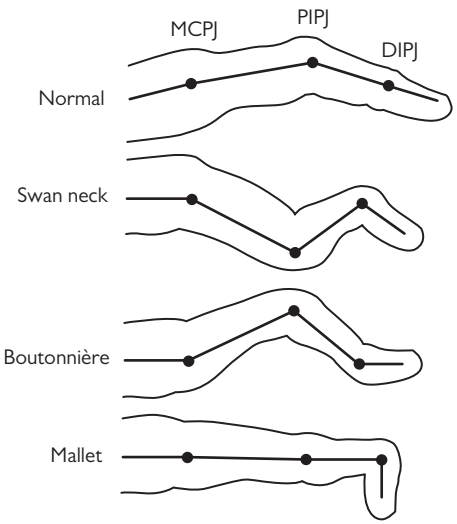


**Fig. 9.14** Finger deformities.

# Hand wounds and associated injuries

## General principles of treating hand wounds

- Remove rings as soon as possible after any hand or arm injury, as swelling can develop relatively rapidly. Try soap or water-based lubricant or alternatively, pass a string or 0/0 silk under the ring and wrap it firmly around the finger distally, allowing the ring to come off over the compressed tissues. Ring cutters are a last resort—note that tungsten rings cannot be cut but can be removed by crushing and cracking in a vice.
- Elevate to diminish swelling and pain.
- Avoid subcutaneous sutures.
- Consider admitting patients who are unco-operative due to excess alcohol consumption, to allow suturing with better co-operation later.
- X-ray any hand injury caused by glass.
- Remember to consider tetanus cover.

## Exploration under anaesthesia

If it is obvious that surgical intervention is required, do not explore the wound in the ED. This particularly applies to suspected nerve injuries where use of LA renders subsequent assessment difficult. Conversely, clinical assessment of tendon injuries can be misleading if the patient is reluctant to move due to pain. Exploration under anaesthesia is necessary in this situation and to exclude division of >50% of a tendon (where clinical examination may be normal, but repair is required). Use an appropriate LA nerve block (as outlined in ➔ Median and ulnar nerve blocks, pp. 306–7, Radial nerve block at the wrist, p. 308).

During exploration, consider the position of the hand at the time of injury—reproducing this may reveal injuries otherwise hidden. Therefore, put all mobile structures through their full range of movement.

## Extensor tendon injuries

More than 50% or complete division needs repair (eg 4/0 or 5/0 non-absorbable monofilament using Bunnell or Kessler stitch) by an experienced surgeon. This may be achieved under LA in the ED, depending on expertise. Immobilize after repair (eg volar slab-type POP with finger joints in full extension and slight flexion at the MCPJs). Treat <50% division by splintage in extension (eg POP slab as above) under the care of the hand surgeon.

## Flexor tendon injuries

Refer immediately for specialist repair.

## Nerve injuries

Complete nerve division may cause surprisingly little sensory loss, so take any altered sensation very seriously. Refer patients with suspected nerve injuries. Digital nerves can be repaired up to the level of the DIPJ, although it may be decided not to attempt to repair injuries distal to the PIPJ. It is functionally important to have intact sensation over the 'edges' of the hand (the thumb, the radial aspect of the index finger, or the ulnar aspect of the little finger). Patients sometimes present late after digital nerve injuries—repair can still be quite successful up to 2 weeks after injury.

### Reverse fight bites

Treat as outlined in ➲ Specific bites and stings, pp. 422–3. Consider transfer of blood-borne infection, as discussed in ➲ Needlestick injury, p. 425.

### Amputations

Refer patients with partial or complete digital amputation with bony loss. Recent proximal amputations without crush injury in fit young patients may be suitable for re-implantation—others may be treated with 'terminalization' or advancement flap. Let the hand surgeon decide. Meanwhile, dress, bandage, and elevate; give IV analgesia, tetanus cover, and broad-spectrum antibiotics (eg cephalosporin), and keep fasted. Wrap the amputated part in moist saline swabs, and place in a sealed plastic bag, surrounded by ice/water mix at 4°C. Do not freeze or place it directly in solution.

### Finger pad amputations

Skin loss of $<1cm^2$ without bony exposure may be allowed to heal with non-adherent dressings. Larger areas of tissue loss (particularly in adults) may require skin grafting or advancement flap, but some do heal satisfactorily with simple dressings.

### Ring avulsions

Refer all circumferential and significant degloving injuries.

### Open (compound) injuries

Wounds over dislocations or fractures usually require specialist attention. Distal open phalangeal fractures may be treated in the ED with wound cleaning, closure, review, and prophylactic antibiotics.

### Crush injuries

These frequently cause 'burst' injury fingertip wounds. Clean the wounds, and take into account the likely swelling when considering closure. Elevate, dress, give analgesia, and arrange review.

### Nail bed lacerations

Accurate repair (eg 6/0 Vicryl) may prevent nail deformity. Nailfold lacerations extending towards the nail bed require removal of the nail to allow suture. Consider replacing the nail after to act as a temporary dressing.

### Foreign bodies under the nail

Splinters and other FBs under fingernails are relatively common. Apply a digital block and remove with fine forceps. If the FB cannot be reached easily, cut away an appropriate piece of nail.

### Subungual haematomas

Blood collecting under the nail from a crush injury causes pain. If >50% of the nail is affected by a recent injury (within 48hr), trephine the nail distal to the lunula, using a red hot paper clip or battery-operated drill.

### High-pressure injection injuries

Industrial grease or paint guns may cause small skin wounds, which initially appear trivial, disguising a devastating injury, with a risk of permanent stiffness and tissue loss. X-rays may indicate the extent of foreign material. Refer to a hand surgeon for immediate exploration and debridement.

# Hand fractures and dislocations

## Distal phalangeal fractures

Treat closed fractures of the distal portion (tuft) of the distal phalanx with analgesia and elevation. Open (compound) burst injuries (from crushing injuries or hammer blows) require meticulous exploration, wound toilet/repair under LA, and follow-up—local policy will guide if this may be delivered in the ED or under the hand surgeon as an inpatient. Give antibiotics (not a substitute for primary surgical treatment).

## Mallet finger with fracture

The characteristic 'mallet finger' deformity (see Figs. 9.15 and 9.16) may be associated with a small fracture at the base of the distal phalanx at the point of attachment of the extensor tendon. Treat as for (the more usual) mallet finger injury without fracture by plastic mallet splint for ~6 weeks, advice, and follow-up (see details in ➲ Soft tissue hand injuries, p. 448). Refer patients with larger bony fragments (more than one-third of articular surface) with mallet deformity or those with subluxation for possible K-wire internal fixation.

**Fig. 9.15** Mallet finger deformity.

**Fig. 9.16** Mallet finger injury due to tendon rupture.

## Proximal and middle phalangeal fractures

Treat undisplaced fractures with elevation, neighbour strapping (see Fig. 9.17), and analgesia. Manipulate angulated proximal and middle phalangeal fractures under digital or wrist block. A useful tip for proximal phalangeal fractures is to use a needle-holder or a pencil placed adjacent to the web space as a fulcrum. Maintain reduction using neighbour strapping and a volar slab POP or flexible padded aluminium (Zimmer) splint, although the latter can be difficult to secure. If reduction is unsatisfactory or cannot be maintained, refer for surgical fixation.

## Index, middle, and ring metacarpal fractures

Check for displacement or rotational deformity, and refer if either is present. Treat with analgesia and elevation, and protect in a volar slab POP. Internal fixation may be considered for midshaft MC fractures with marked angulation but can be complicated by marked postoperative stiffness.

### Phalangeal dislocations

X-ray all dislocations prior to reduction for the presence of associated fractures. Reduce under digital or metacarpal nerve block (see ➲ Digital nerve block, pp. 304–5) or Entonox® by traction and gentle manipulation, then check the integrity of the collateral ligaments. Confirm reduction on X-ray, and immobilize the finger by neighbour strapping. Elevate the hand; provide oral analgesia, and arrange hand clinic follow-up.

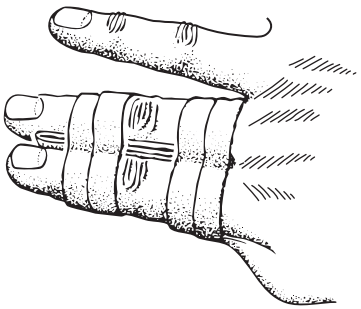

**Fig. 9.17** Neighbour (buddy) strapping.

### Little (fifth) metacarpal fractures

These commonly result from punching (see Fig. 9.8). Check for rotational deformity by gently flexing the fingers into the palm (they should point roughly to the thenar eminence and touch, but not overlap, adjacent fingers on flexion). Angulation is common with neck fractures and rarely requires correction, with even up to 40° being accepted. Apply neighbour strapping; elevate and give analgesia. Warn the patient that the fifth knuckle will be shorter than before. Traditional fracture/hand clinic follow-up for uncomplicated injuries are increasingly being replaced by written advice with no follow-up (unless problems arise—see ➲ Fracture clinic and alternatives, pp. 436–7). Whatever the follow-up arrangements, ensure that the patient is aware of the importance of appropriate hand exercises as soon as possible.

Refer to the orthopaedic team if there is rotational deformity or significant angulation, particularly with base and shaft fractures, which may need surgery. Also refer patients with associated wounds, remembering that these may be compound human bites ('reverse fight bites'—see ➲ Specific bites and stings, pp. 422–3).

### Little (fifth) metacarpal dislocations

Dislocations at the base of the fifth MC may be associated with a fracture. Refer for reduction and internal fixation.

# Thumb fractures and dislocations

## Dislocation at the metacarpophalangeal joint

(See Fig. 9.18.)

After X-rays and LA block, attempt reduction. If successful, assess and document the integrity of the collateral ligaments (see ➲ Soft tissue hand injuries, p. 448), then immobilize in slight (~15°) flexion in a POP and arrange follow-up in the fracture clinic. Reduction may be unsuccessful due to 'button-holing'—in this case, refer for open reduction.

## Gamekeeper's thumb with associated avulsion fracture

Most abduction injuries result in ulnar collateral ligament injury without fracture, but occasionally an avulsion fracture occurs at the point of ligament attachment instead (see Fig. 9.19). Treat this in a scaphoid POP and refer to the fracture clinic, unless the bony fragment is displaced by >2mm, in which case internal fixation will probably be required. If undisplaced, treat in scaphoid POP and refer to the fracture clinic, but if displaced, refer for internal fixation.

## Thumb dislocations

Dislocations usually follow falls onto the thumb or hyperextension injuries. They can occur at any level, including at the interphalangeal joint (IPJ), MCPJ, and carpometacarpal joint. Reduce dislocations by traction and local pressure under combined median and radial nerve blocks (see ➲ Median nerve block, p. 306), Radial nerve block, p. 308. Confirm reduction by X-ray; immobilize in a scaphoid POP, and arrange follow-up.

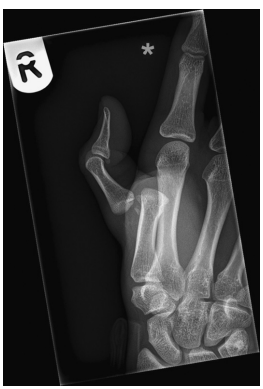

**Fig. 9.18** Dislocated thumb at the MCPJ.

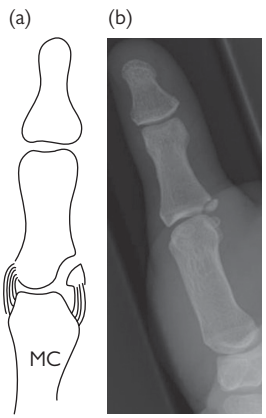


**Fig. 9.19** (a) Diagram of gamekeeper's thumb with avulsion; (b) X-ray of gamekeeper's thumb with avulsion.

### Bennett's fracture–dislocation

(See ➲ Eponymous fractures, pp. 514–18.)

This is a fracture through the base of the thumb (first) MC, with radial subluxation of the MC, leaving a small proximal fragment still joined to the trapezium (see Fig. 9.20). The injury results from a fall onto the thumb or from a fall/blow onto a fist closed around the thumb. Deformity and swelling occur over the base of the thumb and may be mistaken clinically for a scaphoid injury. This is an unstable injury requiring expert attention. If undisplaced, apply a Bennett's-type POP (similar to a scaphoid POP, but with the thumb abducted). If there is any displacement, refer for MUA/fixation. Maintaining reduction often requires the use of screw or Kirschner wire fixation.

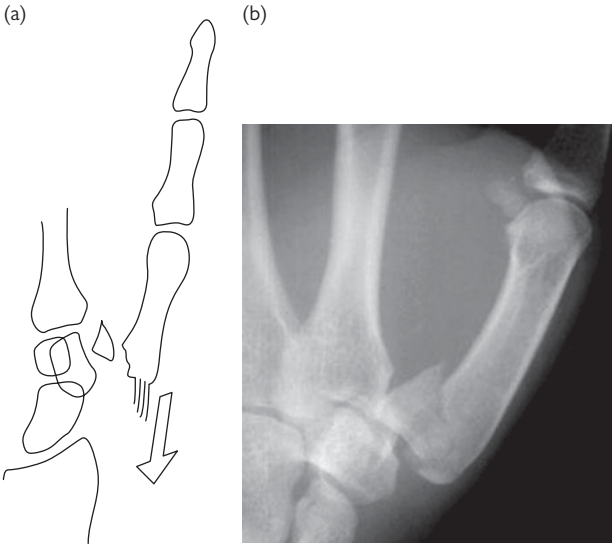


**Fig. 9.20** (a) Diagram of Bennett's fracture–dislocation; (b) X-ray of Bennett's fracture–dislocation.

# Soft tissue hand injuries

## Gamekeeper's thumb

The thumb's ulnar collateral ligament is crucial for stability and function. It is typically injured in hyperabduction injuries (eg falls whilst skiing). Complete rupture usually results in the two parts of the ligament being separated by the adductor aponeurosis (the 'Stener lesion'), so satisfactory healing cannot occur. If tender over the ulnar collateral ligament of the thumb MCPJ, obtain X-rays—if these demonstrate a fracture, do not stress the joint, but treat appropriately instead (see Thumb fractures and dislocations, pp. 446–7). If no fracture, assess stability of the ulnar collateral ligament by gentle abduction of the MCPJ (compare with the other hand). Examine the ulnar collateral ligament with the thumb slightly (15°) flexed. If pain precludes adequate examination, consider Entonox® (and/or LA injection) and repeat the examination. Significant (>30°) laxity implies complete rupture and the need for operative repair.

*Treat* uncomplicated sprains with analgesia, elevation, and either criss-cross adhesive strapping ('thumb spica') or a scaphoid POP if symptoms are severe, and arrange follow-up. Refer suspected or demonstrable ulnar collateral ligament rupture to consider primary surgical repair.

## Mallet finger

Injury to the extensor mechanism at the DIPJ is relatively common and results from forced flexion of the DIPJ or from a blow/fall directly onto the fingertip. In the elderly, it can follow minimal trauma. There is loss of full active extension at the DIPJ. Normal flexion is preserved.

X-ray to exclude associated fracture—treated as outlined in Hand fractures and dislocations, pp. 444–5.

In the absence of a large fragment, treat in a plastic (mallet) splint secured with tape for ~6wk (see Hand fractures and dislocations, pp. 444–5). Ensure the patient understands the need to wear the splint continuously and to keep the finger straight if the splint is removed for washing (eg to hold the finger against a flat surface until the splint is replaced). Warn that there may be a small degree of permanent flexion deformity. Consider initial follow-up at ~7–10 days, to ensure compliance with treatment and to reassess in case swelling has ↓ and a smaller splint is required.

## Volar plate injury

These are significant injuries, often with prolonged morbidity. Hyperextension at the PIPJ injures the *volar plate* at the base of the middle phalanx, with or without evidence of bony involvement. Examination shows fusiform swelling of the PIPJ, with tenderness over the volar aspect. Treat with 'buddy strapping' to adjacent fingers (or 'Bedford splint'), elevate, provide analgesia, and begin mobilization immediately. Arrange review to ensure full mobility is regained.

## A2 pulley injury

The finger flexor tendon sheath at the PIPJ is thickened as the A2 pulley. Occasionally (eg in rock climbers), the tendon cuts through the A2 pulley, causing bowstringing on flexion. There may be associated tendon injury. Treat with buddy strapping and elevation. Arrange hand specialist follow-up.

# Other soft tissue hand problems

## Pulp infections

Infection of the pulp space at the fingertip may reflect underlying FB or osteomyelitis, so X-ray to search for these and treat accordingly. If X-rays are normal, incise the pointing area under LA digital block. Send pus for bacteriology; apply a dressing, commence oral antibiotics (eg flucloxacillin 250–500mg PO qds), and arrange follow-up.

## Paronychia

Infection of the nailfold adjacent to the nail is common. In the early stages, oral antibiotics (eg flucloxacillin or clarithromycin) may cure.

Once pus has developed, drain this under LA digital block by an incision over the fluctuance (usually a small longitudinally orientated incision adjacent to the proximal nailfold suffices, but pus under the nail may require removal of a segment of the nail). Alternatively, incise immediately adjacent to and along the affected lateral nailfold. Once drained, do not give antibiotics, unless there is cellulitis or spreading infection or the patient is immunocompromised and/or has diabetes.

## Pyogenic flexor tenosynovitis

Infection of a finger flexor tendon sheath may follow a penetrating injury. Classically, the evidence is in the form of 'Kanavel's signs':

- Tenderness over the flexor tendon.
- Symmetrical swelling of the finger.
- Finger held in flexion.
- Extreme pain on passive extension.

Ensure tetanus prophylaxis, then refer urgently for exploration, irrigation, and IV antibiotics.

## Other infections

These include palmar space infections and septic arthritis—refer immediately for specialist treatment.

## Locked finger

Elderly patients with underlying osteoarthritis (OA) sometimes present with locking at a finger MCPJ. A fixed flexion deformity is present, such that the patient can flex but not fully extend at the MCPJ. There is usually no particular history of trauma—the underlying cause is entrapment of the palmar plate on an osteophyte. Refer for an early hand surgeon opinion—surgery may be required.

## Trigger finger/thumb

This is relatively common, but not particularly related to trauma. In young children, many resolve spontaneously, although some require surgery. Most cases in adults are satisfactorily treated by steroid injection into the flexor tendon sheath, but leave this to a specialist.

# Scaphoid fracture

## Background

Spanning the two rows of carpal bones, the scaphoid is at particular risk of injury. Scaphoid fractures occur from falling onto an outstretched hand or from 'kick-back' injuries (eg from a steering wheel in a car crash or a football goalkeeper making a save).

The combination of fractures being difficult to identify on X-ray and the risk of significant complications (non-union, avascular necrosis) demands a careful approach. The scaphoid mostly fractures through the waist, but sometimes through the tubercle (the latter does not give rise to significant complications).

## Clinical assessment

Assess and document whether there is evidence of scaphoid fracture in anyone who presents with a wrist injury. Pain and swelling over the radial aspect of the wrist may be accompanied by difficulty gripping.

*Examine for*:

- Tenderness in the anatomical snuffbox—compare both sides.
- Tenderness over the palmar aspect of the scaphoid (scaphoid tubercle).
- Scaphoid pain on compressing the thumb longitudinally.
- Scaphoid pain on gentle flexion and ulnar deviation of the wrist.

## X-rays

Fractures of the scaphoid may be difficult to see (or not be apparent at all) on initial X-rays. Request specialized scaphoid (not wrist) views if there is clinical suspicion of scaphoid fracture. Four views are usually taken (AP, lateral, right, and left obliques), as shown in Fig. 9.21.

## Treatment where a fracture is visible

If there is a visible fracture on X-ray, immobilize in a cast and arrange follow-up in the hand/fracture clinic. Although it is traditional to apply a scaphoid-type POP, evidence suggests that it is equally acceptable to use a Colles-style POP.

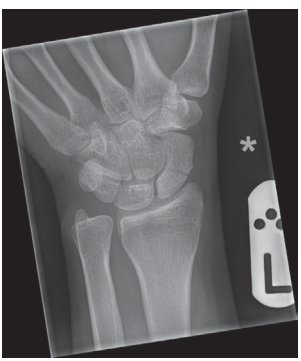


**Fig. 9.21** Scaphoid waist fracture.

## Clinically suspected scaphoid fracture

If there is clinical suspicion of a scaphoid fracture, but the X-rays look normal, apply a splint and arrange review (usually in 10–14 days). Most of these patients will turn out not to have a fracture, but failing to adequately immobilize those patients who do have a fracture may ↑ the risk of complications.

*Follow-up of clinically suspected scaphoid fractures*

Patients with clinically suspected scaphoid fractures (but normal X-rays) are often seen in the ED clinic. Aim to discharge patients if there is no clinical evidence of fracture at review at 10–14 days after injury. If, however, there is continuing pain and/or scaphoid tenderness, arrange further imaging. The choice includes repeat X-rays, MRI, and CT. Of these, MRI has the advantage of avoiding ionizing radiation and the potential to pick up other injuries not seen on X-ray (see Figs. 9.22a and b).

(a) (b)

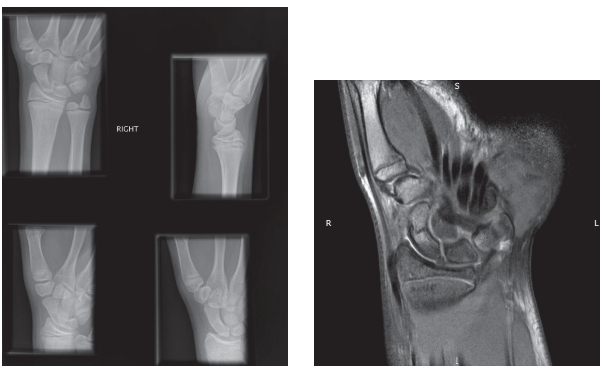


**Fig. 9.22** Scaphoid fracture in 15y old seen at 2 weeks on (b) MRI, but not (a) X-ray.

## Complications of scaphoid fracture

These include non-union, avascular necrosis, and OA. Sometimes patients present with symptoms relating to one or more of these, but often an abnormality is picked up as an incidental finding—the patient may not even have any recollection of previous injury (see Fig. 9.23). Arrange specialist follow-up (may be most appropriately achieved via the GP).

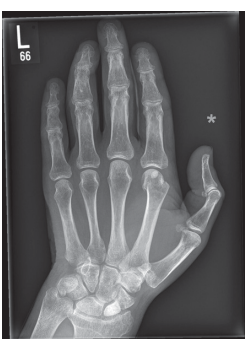


**Fig. 9.23** New index MC fracture with incidental old non-union of the scaphoid plus associated avascular necrosis of the proximal pole (which has a sclerotic appearance).

## Lunate dislocation

These injuries are rare, but important, as they are often missed, yet require urgent admission for MUA. Lunate dislocations usually follow falls onto an outstretched wrist and result in pain and swelling anteriorly over the wrist (see Fig. 9.24). Median nerve paraesthesiae may be a clue to the diagnosis. X-ray shows dislocation and rotation of the lunate, so that it is shifted in front of the carpus and its concave surface faces towards the palm instead of distally. The AP view may look relatively normal, so carefully scrutinize the lateral views. It may help to compare with normal X-rays (see Fig. 9.25).

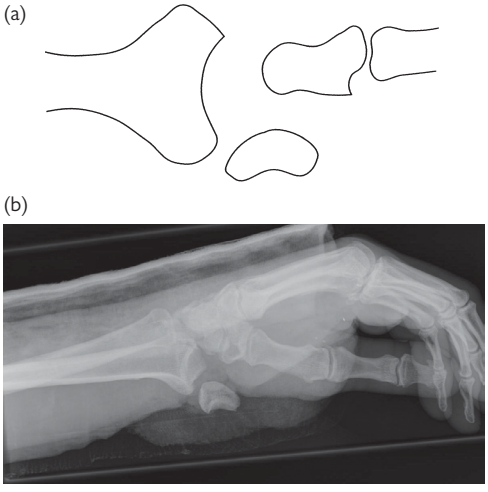


**Fig. 9.24** (a) Diagram of lunate dislocation. (b) X-ray of lunate dislocation (as part of a complex wrist injury).

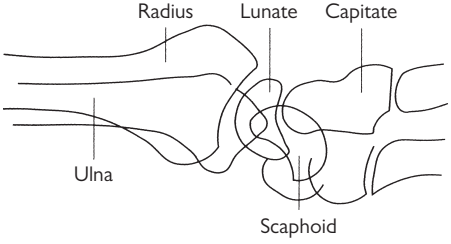


**Fig. 9.25** Wrist: normal lateral view.

# Other carpal injuries

## Perilunate dislocation

Isolated dislocations of carpal bones, apart from the lunate, can occur, but often injuries are more complicated and involve dislocations (and fractures) of one row of carpal bones (eg trans-scaphoid perilunate dislocation) (see Fig. 9.26). Surprisingly, perhaps, given almost inevitable significant swelling, these injuries can be missed. Give analgesia and refer for reduction by the orthopaedic team.

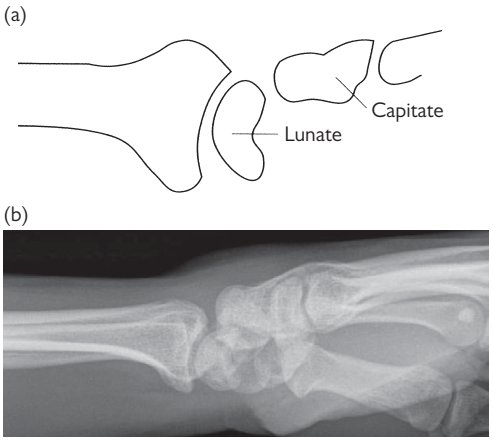


**Fig. 9.26** (a) Diagram of perilunate dislocation. (b) X-ray of perilunate dislocation.

## Fractured hook of the hamate

Local palmar tenderness may give rise to suspicion of a fracture of the hook of the hamate. Diagnosis can be difficult—specialized X-rays or CT may be required to demonstrate the fracture. Immobilize in POP and refer to the fracture clinic.

## Flake avulsion carpal fractures

Small avulsions from the dorsum of the carpus are often from the triquetrum (see Fig. 9.27). Treat with immobilization in a POP backslab or a wrist support splint and analgesia, and refer to the fracture clinic.

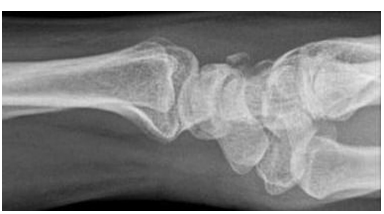

**Fig. 9.27** X-ray of triquetral avulsion fracture.

# Colles' fracture

## Presentation

This fracture affects the radius within 2.5cm of the wrist, such that the distal fragment is angulated to point dorsally. It usually results from a fall onto an outstretched hand. Osteoporosis contributes to an ↑ frequency in post-menopausal women. Colles' fractures produce characteristic clinical deformity (sometimes likened to a 'dinner fork'). Check for scaphoid tenderness, distal sensation, and pulses in all cases.

## Radiological features

X-ray appearances include one or more of the following:

- Posterior and radial displacement (translation) of the distal fragment.
- Angulation of the distal fragment to point dorsally (the articular surface of the distal radius normally has a 5° forward tilt on the lateral wrist X-ray) (see Fig. 9.28).
- Angulation of the distal fragment to point more radially (the articular surface of the distal radius is normally tilted 22° towards the ulnar side on an AP wrist X-ray) (see Fig. 9.29).
- Impaction, leading to shortening of the radius in relation to the ulna.

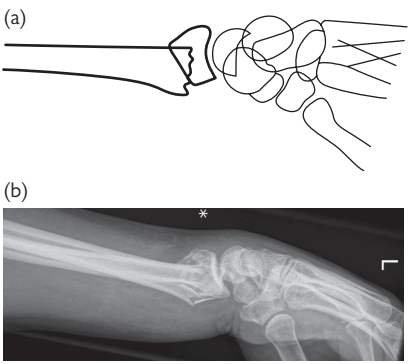


**Fig. 9.28** (a) Diagram and (b) X-ray of Colles' fracture, lateral view.

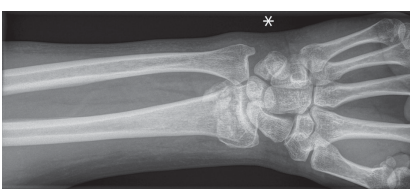

**Fig. 9.29** Diagram of Colles' fracture, AP view—there is comminution of the distal radius, with shortening of the radius in relation to the ulna.

## Treatment

Provide analgesia; immobilize in a backslab POP, and elevate with a sling. Discharge those with undisplaced fractures (if they will manage at home), and arrange fracture clinic follow-up. Advise the patient to keep moving the fingers, thumb, elbow, and shoulder.

### *Deciding if MUA is indicated*

MUA is required for:

- Grossly displaced fractures.
- Loss of normal forward radial articular surface tilt on lateral wrist X-ray. Neutral or minimal tilt may be acceptable in the very young or very old (particularly in the non-dominant limb). Seek senior advice if unsure.

### *Timing of MUA*

Patients with compound fractures and/or symptoms of nerve compression require urgent MUA. For many other patients, the timing of the procedure is less important. Many EDs undertake closed manipulation of Colles' fractures in adult patients at the time of initial presentation, whilst others arrange for the patient to return for the procedure within 1–2 days to a specific theatre list as a day case.

During manipulation of Colles' fractures, focus particularly upon reducing the angulation and displacement of the distal radius seen on the lateral view (see Fig. 9.30).

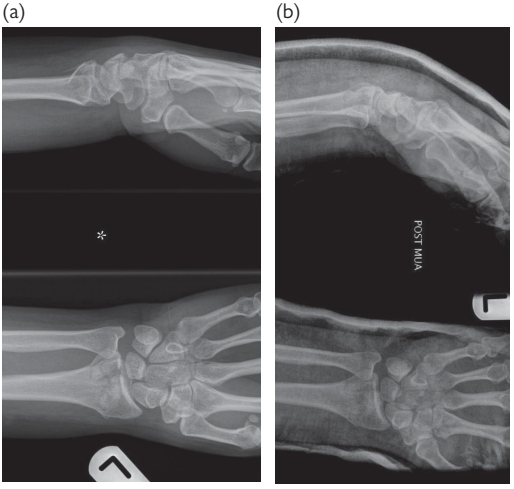


**Fig. 9.30** X-rays of angulated Colles' fracture before (a) and after (b) manipulation.

# Manipulating Colles' fractures

## Consent

Discuss the risks and benefits of the procedure. In particular, explain that, if left untreated, an angulated Colles' fracture may result in long-term stiffness and a significantly weaker grip. The principal risks of manipulation are:

- Tears to the skin on the dorsum of the wrist (especially in those with thin skin, eg patients on steroids, and/or significant swelling, eg those on warfarin).
- Late slippage of the bones requiring a further procedure.
- Risks of the anaesthesia employed.

## Choice of anaesthetic

The anaesthetic options available include: haematoma block (see ➔ Local anaesthetic administration, p. 299), IV regional anaesthesia (see ➔ Bier's block, pp. 300–1), IV sedation (see ➔ Approach to sedation, pp. 316–17), and GA (see ➔ General anaesthesia in the ED, pp. 320–1). The choice of anaesthetic will depend upon local protocols, as well as patient-related factors such as the type of fracture and extent of fasting. For example, a minimally angulated fracture in an elderly individual may be satisfactorily managed using a haematoma block, whereas a more dramatically angulated and displaced fracture may not. Evidence suggests that Bier's block is superior to haematoma block (see http://www.bestbets.org).

## Technique

Different individuals may employ different techniques, but the aim is to attempt to return the anatomy to its previous position. In particular, it is important to correct the dorsal angulation ('restore the volar cortex'). Many descriptions of reduction techniques involve initial traction and 'disimpaction' of the fragments, followed by wrist flexion and pronation, with pressure over the distal radial fragment(s). Some operators focus more upon gentle direct manipulation of the distal fragment, rather than indirect measures (traction, wrist flexion, etc.).

Following manipulation, apply a backslab POP, whilst maintaining the reduction, with the wrist slightly flexed and pronated (avoid excessive flexion as this can cause additional long-term problems). Satisfactory reduction can be confirmed by image intensifier/X-ray. If the reduction is not satisfactory, repeat the manipulation procedure.

## Medium- and long-term complications of Colles' fracture

Patients may present to the ED with later complications following Colles' fracture (and the treatment provided for it), including the following:

- *Stiffness of wrist and adjacent limb joints*: refer for physiotherapy.
- *Malunion and cosmetic problems*: refer to the GP/orthopaedic team.
- *Reflex sympathetic dystrophy (Sudeck's atrophy)*: refer for physiotherapy and GP/orthopaedic follow-up.
- *Carpal tunnel syndrome*: this may occur after Colles' fracture but also reflects other problems (eg lunate dislocation)—check original X-rays.
- *Extensor pollicis longus rupture*: this may occur some weeks after fractures with minimal displacement (see ➔ Soft tissue wrist injuries/problems, p. 459).

# Smith's fracture

This is an unstable distal radius fracture (sometimes referred to as a 'reverse Colles' fracture') where the distal fragment is impacted, tilted to point anteriorly, and often displaced anteriorly (see Fig. 9.31). It usually follows a fall onto a flexed wrist. Give analgesia; immobilize in a volar slab POP, and refer for MUA (often difficult to hold in position after reduction) or open reduction and internal fixation (ORIF) using a buttress plate (preferred in some orthopaedic centres).

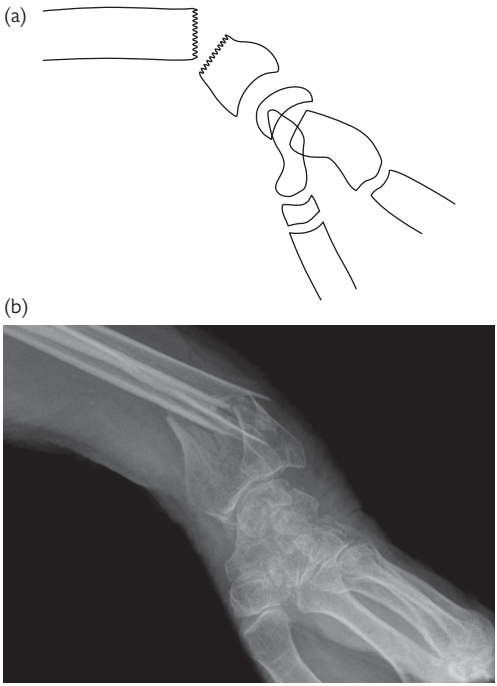


**Fig. 9.31** (a) Diagram of Smith's fracture (lateral view). (b) X-ray of Smith's fracture (there is also a fracture of the distal ulna).

## Barton's and reverse Barton's fracture

An intra-articular fracture involving only the dorsal or volar portion of the distal radius is called a Barton's fracture and reverse Barton's fracture, respectively (see Fig. 9.32), although describing them as 'dorsal Barton's fracture' and 'volar Barton's fracture' may avoid possible confusion. The resultant dorsal or volar fragment tends to slip, so the fracture is inherently unstable. Provide analgesia; immobilize in a POP backslab, and refer. Most patients require ORIF and plating.

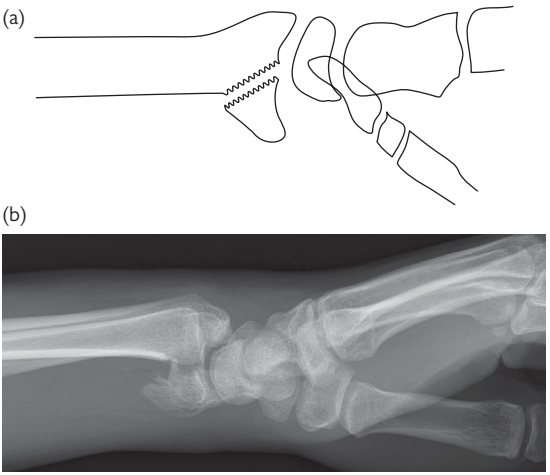


**Fig. 9.32** (a) Diagram of lateral view of a reverse (volar) Barton's fracture. (b) X-ray (lateral view) of a reverse (volar) Barton's fracture.

### Isolated radial styloid fracture

This is caused by similar mechanisms of injury as scaphoid fractures (ie falls onto an outstretched hand or kick-back injuries). It is sometimes referred to as a Hutchinson fracture (see ➲ Eponymous fractures, pp. 514–18). Treat with analgesia, backslab POP, and an elevation sling, and refer to the fracture clinic. Internal fixation is occasionally required.

# Soft tissue wrist injuries/problems

## Wrist sprain

Exclude scaphoid or other fracture (or dislocation) before considering the diagnosis of a 'simple wrist sprain' (triquetral avulsions are particularly easy to miss—see ➲ Flake avulsion carpal fractures, p. 453). Relatively minor damage to ligaments around the wrist can occur following hyperextension or flexion of the wrist, causing swelling and tenderness around the joint. Treat with a wrist splint or Tubigrip® (elasticated tubular) support, analgesia or NSAIDs, and progressive exercise. Continuing pain and problems arouse suspicions of more significant injury (possibly involving other structures such as the scapholunate ligament or triangular fibrocartilage complex). Refer for specialist investigation.

## Triangular fibrocartilage complex injury

The triangular fibrocartilage complex (TFCC) at the distal end of the ulna may be injured with associated structures. Often, these injuries only become apparent later, when what was diagnosed as a 'simple wrist sprain' fails to settle—pain and tenderness persist over the TFCC. Arrange specialist follow-up for further investigation (eg MRI) and treatment.

## Rupture of wrist/hand tendons

Rupture of tendons may occur without penetrating trauma. The most common rupture involves the extensor pollicis longus a few weeks after a (usually undisplaced) fracture of the distal radius. Rupture of other extensor (and occasionally flexor) tendons occurs in association with OA, rheumatoid arthritis (RA), scaphoid non-union, CRF, and SLE. Refer to a hand surgeon.

## Radial tenosynovitis ('intersection syndrome')

This typically follows unaccustomed repetitive activity such as gardening, DIY, or decorating. Over hours to days, a painful fusiform swelling develops over the radial aspect of the distal forearm. Movement of the wrist produces pain and palpable (occasionally audible) crepitus. Immobilize in a simple adjustable wrist splint, and unless contraindicated, prescribe an NSAID for 7–10 days. After this, allow gradual mobilization of the wrist and educate about eliminating the cause. Immobilize severe cases in a forearm POP for 2 weeks before beginning mobilization.

## de Quervain's tenosynovitis

Affects the tendon sheaths of the abductor pollicis longus and extensor pollicis brevis. Pain, swelling, and crepitus occur over the lateral (dorsoradial) aspect of the radial styloid. Symptoms can be reproduced by thumb or wrist movement. Finkelstein described grasping the patient's thumb and rapidly 'abducting the hand ulnarward', but probably more useful is pain on ulnar movement of the wrist with the thumb clenched in a fist. Treat with an NSAID and splintage for 7–10 days. A removable fabric wrist splint (including the thumb) may suffice, but consider a POP for severe pain. Persistent symptoms may respond to steroid injection of the tendon sheath using an aseptic technique.

# Forearm fractures and related injury

► *If one forearm bone is fractured, look for a fracture or dislocation of the other.*

Obvious deformity in an adult forearm indicates fracture of the radial and ulnar shafts. Initially treat with:

- Analgesia (eg increments of IV morphine + antiemetic until pain is relieved).
- Immobilization in backslab POP.
- If one or both fractures are compound, give IV antibiotics (see ➲ Open (compound) fractures, pp. 428–9) and tetanus cover, and dress the wound.

Always check distal pulses and sensation, and examine for associated injuries at the wrist and elbow. Only once this has been done and the patient is comfortable can he/she be sent for X-ray. Ensure X-rays demonstrate the whole lengths of the radius and ulna, including separate views of both the elbow and wrist joints.

### Fractures of both radial and ulnar shafts

Adult fractures, unlike those in children, may be markedly displaced, with little or no bony contact between the fragments. Rotational deformity is common. Check carefully for clinical evidence of neurovascular injury. Closed reduction is difficult and often fails or is complicated by late slippage. Treat fractures with analgesia/immobilization as described earlier, and refer for ORIF.

### Isolated ulnar shaft fracture

These usually occur from a direct blow to the outer edge of the forearm (it is typically seen as a defence injury) or from a fall striking the ulnar shaft. X-ray the whole ulna and radius to exclude associated fracture or dislocation of the radial head (see ➲ Monteggia fracture–dislocation, p. 461). If undisplaced, treat in an above-elbow POP, with the elbow flexed to 90° and the forearm in mid-supination. Refer all displaced or angulated fractures for ORIF.

### Galeazzi fracture–dislocation (See ➲ Eponymous fractures, pp. 514–18.)

This is defined as a fracture of the radius associated with dislocation of the distal radio-ulnar joint at the wrist (see Fig. 9.33). Always look for subluxation of the ulna in radial fractures. Treat with analgesia and immobilization in a temporary POP backslab. Refer for ORIF.

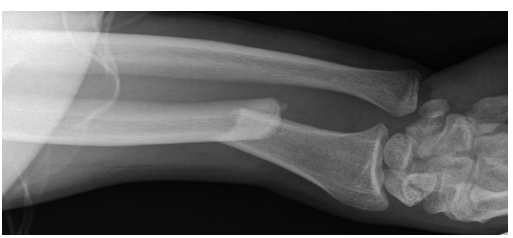

**Fig. 9.33** Galeazzi injury—fractured radius plus disrupted radio-ulnar joint.

### Monteggia fracture–dislocation

(See ➲ Eponymous fractures, pp. 514–18.)

This is defined as a fracture of the ulna associated with dislocation of the radial head. It occurs from forced pronation of the forearm (eg fall onto an outstretched, fully pronated forearm). It can also occur by a direct blow or fall onto the proximal ulna, displacing the head of the radius. Treat with analgesia and immobilization in a temporary above-elbow POP backslab. Refer to the orthopaedic team for ORIF (or, sometimes in children, for treatment with MUA and POP).

A related injury is the *Hume fracture* (see ➲ Eponymous fractures, pp. 514–18), in which anterior dislocation of the radial head is combined with an olecranon fracture. Refer for ORIF.

*Note*: Monteggia fracture–dislocations are not infrequently missed at initial presentation, due to attention being distracted by the ulna fracture (see Fig. 9.34). To avoid this:

- Request elbow and wrist X-rays in any patient with a forearm shaft fracture.
- Check all elbow X-rays carefully to ensure that the radial shaft is normally aligned and the radial head abuts the capitellum.

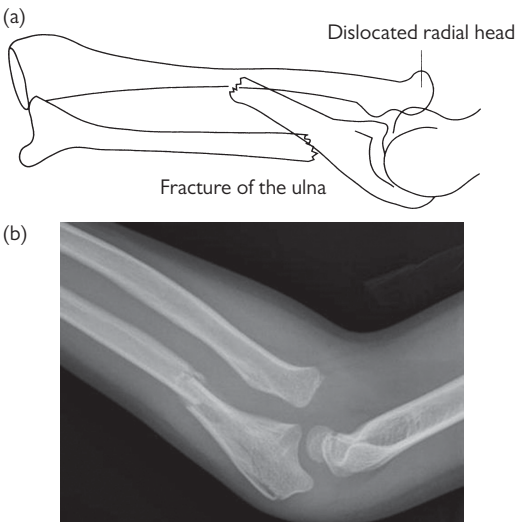


**Fig. 9.34** (a) Monteggia fracture–dislocation; (b) X-ray of Monteggia fracture–dislocation.

### Isolated radial shaft fracture

These are very uncommon. Always treat and assume that there is some associated damage to the distal radio-ulnar joint at the wrist.

# Elbow injuries

## Assessment

In any injured elbow, look specifically for:

- Elbow effusion (felt as a tense, bulging swelling halfway between the lateral epicondyle and the point of the olecranon).
- The normal relationship between the olecranon and the lateral and medial epicondyles—all should form an equilateral triangle with the elbow flexed.
- Range of movement—X-ray patients who cannot fully extend the elbow and flex to touch the shoulder tip.

### Elbow effusion, no visible fracture on X-ray

The presence of an effusion on X-ray (see Fig. 9.35) implies that a radial head/neck (or even supracondylar) fracture is present, even if none is visible. Provide analgesia, a collar and cuff sling, and advice regarding active movements and gradual return to normal activities. Formal follow-up is not necessary, but ensure the patient knows to return for review if the anticipated recovery timescale is delayed.

**Elbow fat pad sign** (See Fig. 9.35.)

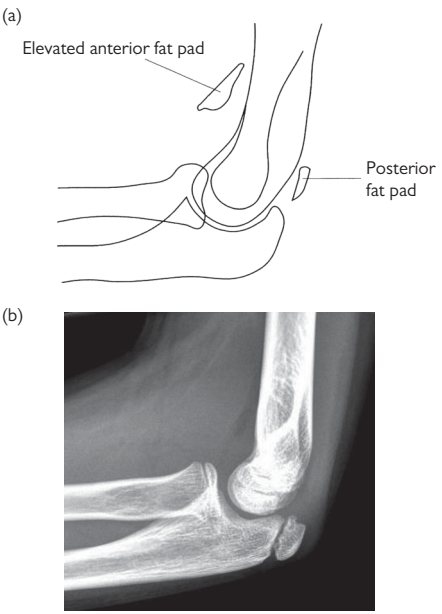


**Fig. 9.35** (a) Diagram of the elbow fat pad sign; (b) X-ray of the elbow fat pad sign in a 10y old child.

### Radial head/neck fracture

Follow falls onto an outstretched hand (the radial head impacts against the capitellum) or direct trauma to the elbow. It can sometimes occur in combination with a wrist fracture (a dramatic example is the Essex–Lopresti fracture–dislocation (see ➲ Eponymous fractures, pp. 514–18). Examine movements—extension and flexion are usually limited, but supination and pronation may be relatively normal. Check for an elbow effusion, and palpate for tenderness over the radial head whilst supinating/pronating the elbow. X-ray usually confirms elbow effusion, but fractures may be difficult to see (see Fig. 9.36). Treat fractures with analgesia and a collar and cuff sling. Discharge undisplaced fractures with written advice about exercises and likely recovery time, with advice to return if there are any problems (see ➲ Fracture clinic and alternatives, pp. 436–7). Arrange a fracture clinic review for displaced or comminuted fractures which may require surgery. If very painful, immobilize in an above-elbow POP backslab at 90°.

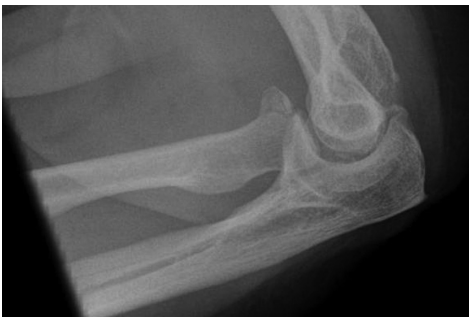

**Fig. 9.36** X-ray of radial head fracture.

### Olecranon fracture

Follow falls onto the point of the elbow. The olecranon fragment may displace proximally due to pull of the triceps. Swelling, tenderness, or crepitus are present on examination. In the young, the olecranon epiphysis may cause confusion on X-rays (see Fig. 9.37). Treat undisplaced or hairline fractures in an above-elbow backslab POP at 90°; provide analgesia, and arrange fracture clinic follow-up. Refer fractures that are displaced or involve the elbow joint for ORIF.

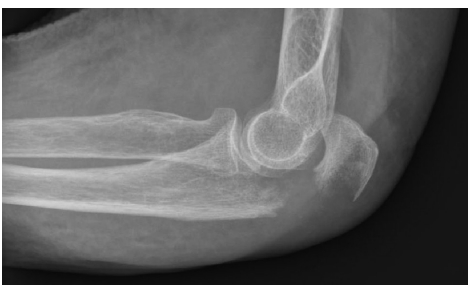

**Fig. 9.37** X-ray of displaced olecranon fracture.

# Dislocated elbow

## Assessment

Examination reveals loss of the normal triangular relationship between the olecranon and epicondyles. Check distal pulses and sensation as the brachial artery and the median and ulnar nerves may be damaged. Elbow dislocations may be classified according to the direction of dislocation and the presence of associated fractures (eg fractured coronoid). The most frequent injury is postero-lateral dislocation (ie movement of the distal part in a postero-lateral direction).

## Management

After analgesia and X-ray (see Fig. 9.38), most dislocations may be reduced in the ED under IV sedation with full monitoring (see ➲ Approach to sedation, pp. 316–17). However, GA is sometimes required.

*Reduction techniques for postero-lateral dislocations*

- Flex the elbow to 60° with countertraction on the upper arm. Pull on the fully pronated forearm at this angle. Slight flexion at the elbow may be necessary.
- Alternatively, lever the olecranon forward with both thumbs, whilst holding the elbow flexed and whilst an assistant provides traction on the forearm.

Reduction is usually confirmed by a 'clunk' and restoration of the normal triangular relationship of the elbow landmarks. Once reduced, recheck pulses and sensation; immobilize in an above-elbow POP backslab at 90°, and X-ray again (looking for associated fractures). Consider admission for analgesia and observation for possible significant limb swelling. If unable to reduce, refer for reduction under GA.

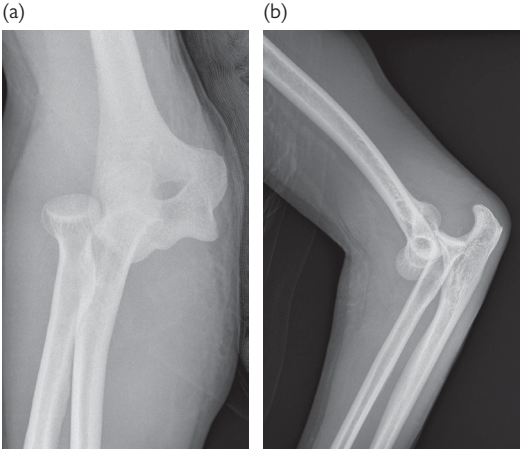


**Fig. 9.38** X-rays of postero-lateral elbow dislocation.

# Other elbow injuries

### Supracondylar fractures

These are most common in children (see ➲ Supracondylar humeral fracture, p. 748) but also occur in adults. The elbow may be grossly swollen and deformed, but the normal triangular relationship of the olecranon and epicondyles is characteristically preserved. Check distal pulses and sensation carefully, as the brachial artery, ulnar, median, and radial nerves can all be damaged. Immobilize in an above-elbow backslab POP, and give analgesia. Refer to the orthopaedic surgeon, as MUA/ORIF are usually required.

*Fractures of the capitellum* occasionally occur in isolation. If undisplaced, treat conservatively with analgesia and POP. Refer those with displaced fractures for specialist treatment (possibly ORIF).

### Medial collateral ligament injury

Instability on stress testing of the medial (ulnar collateral) ligament implies a significant injury. Treat in backslab POP, with the elbow flexed to 90° and supported in a sling. Arrange fracture clinic follow-up.

### Other elbow injuries

Elbow injuries are relatively common in children—see relevant pages:

- Supracondylar fracture (see ➲ Supracondylar humeral fracture, p. 748).
- Lateral and medial condylar injury (see ➲ Elbow injuries in children, p. 750).
- Pulled elbow (see ➲ Subluxation of the radial head ('pulled elbow'), p. 750).

# Shaft of humerus fracture

This results from a fall onto an outstretched hand or onto the elbow, or occasionally from excessive twisting (eg arm wrestling). The fracture may be obvious and palpable. Check distal pulses, the radial nerve, and the elbow joint. X-ray reveals a transverse, comminuted, or spiral (see Fig. 9.39) humeral shaft fracture.

*Provide* analgesia and support the fracture (eg in a POP U-slab from the axilla down to and around the olecranon and up the outside of the upper arm—apply with the elbow flexed to 90°, and hold in place with a bandage). Alternative treatment includes a 'hanging cast' POP (above-elbow POP at 90°—the weight of POP and the arm holds the fracture in a satisfactory position). Refer if displaced, comminuted, or angulated or if neurovascular complications are suspected. MUA and internal fixation are required in these cases.

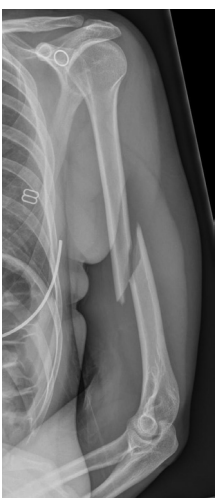

**Fig. 9.39** X-ray of spiral humeral shaft fracture.

# Soft tissue elbow/arm problems

## Injuries to the biceps and brachialis

Inflammation of the biceps and/or brachialis at the site of attachment at the elbow can cause persistent symptoms—treat with rest and NSAID. The biceps brachii can rupture either at its long head in the bicipital groove or near the elbow insertion. Distal ruptures are sometimes treated conservatively, but some benefit from repair—arrange orthopaedic review to consider this.

### *Ruptured long head of the biceps*

The long head of the biceps can rupture at its proximal insertion after lifting or pulling (see ➲ Soft tissue elbow/arm problems, pp. 466–7). This may follow little force (and with little pain) in the elderly. Look for the ruptured biceps muscle as a characteristic abnormal shape and low biceps bulge above the elbow on attempted elbow flexion against resistance. Treat with initial analgesia and support in a sling, followed by later exercises. Surgical repair is rarely indicated.

## Lateral epicondylitis

This is commonly called 'tennis elbow'. It follows repetitive or excessive stress to the origin of the forearm and hand extensor muscles at the lateral epicondyle. It can occur spontaneously but usually follows repetitive lifting, pulling, or sports (eg as a result of an incorrect backhand technique in tennis). Inflammation, oedema, and microtears occur within the extensor insertion.

- *Look for* localized swelling, warmth, or tenderness over the lateral epicondyle and immediately distal to it.
- *Examine movements*—dorsiflexion of the pronated wrist against resistance will reproduce symptoms.
- *X-ray* if the problem follows an acute injury. Refer to the orthopaedic surgeon if there is an avulsion fracture.
- *Treat* with analgesia (preferably an NSAID) and ice application. Support the arm in a broad arm sling and advise rest, followed by progressive exercise and avoidance of aggravating movements. If symptoms are recurrent or prolonged, refer as steroid injection, forearm clasp, physiotherapy, and occasionally surgery may help. Current evidence suggests that corticosteroid injection may provide short-term relief, but long-term benefit remains unproven.

## Medial epicondylitis

Often called 'golfer's elbow', this condition has a similar pathophysiology to lateral epicondylitis—it is frequently seen in racket sports and golf.

- *Examine for* localized tenderness and swelling over the forearm flexor insertion at the medial epicondyle. Flexion of the supinated wrist against resistance will reproduce symptoms. There may be ↓ grip strength, and ~60% of patients have some symptoms of associated ulnar neuritis.
- *Treat* as for lateral epicondylitis.

### Olecranon bursitis

Inflammation, swelling, and pain in the olecranon bursa may follow minor trauma or occur spontaneously. Other causes include bacterial infection (sometimes following penetrating injury) and gout. Elbow movements are usually not limited. Look for overlying cellulitis, wounds, and systemic symptoms, and check for ↑ T° (these suggest infection). Consider aspiration of the bursa under aseptic conditions—immediate microscopy for crystals or bacteria may confirm gout or bacterial infection. Aspirate using a small needle at a shallow angle, and try to aspirate the bursa completely.

*Non-infective bursitis* Provide analgesia and NSAID, and rest the arm in a broad arm sling. Consider compression and intermittent ice application. Symptoms should resolve with rest over a period of weeks. Rarely, persistent symptoms require surgical excision of the olecranon bursa.

*Gout bursitis* Treat as for non-infective bursitis. Arrange follow-up through the patient's GP.

*Infective bursitis* If there is evidence of an underlying infection, treat with rest and NSAID and start antibiotics (eg flucloxacillin or clarithromycin). Occasionally, infection requires referral to the orthopaedic surgeon for surgical drainage.

*Olecranon bursa haematoma* A history of blunt trauma to the olecranon, followed rapidly by 'golf ball-sized' swelling over the olecranon, but with a full range of elbow movement (and no evidence of fracture), implies a haematoma in the olecranon bursa. Treat conservatively—attempts at drainage may result in secondary infection.

### Nerve compression

Ulnar nerve entrapment at the elbow ('cubital tunnel syndrome') is the second most common upper limb nerve entrapment (median nerve compression in carpal tunnel syndrome is the most common). Refer these chronic conditions back to the GP.

*Acute radial nerve palsy* Above the elbow presents with sudden wrist drop, following a history of compression (eg crutch use, falling asleep with the arm over the back of a chair). The underlying injury is usually a neurapraxia, which has the potential to recover completely, given time, with conservative measures. It is crucial to ensure that flexion contractures do not develop in the meantime—provide a removable wrist splint; advise regular passive wrist exercises, and refer for physiotherapy and follow-up to ensure recovery.

### Osteochondritis dissecans

This can affect the elbow and cause locking of the elbow joint. X-rays may reveal a defect and/or loose body. Refer to the orthopaedic team.

# Anterior shoulder dislocation

This is a common injury, which typically results from forced external rotation/abduction of the shoulder. The humeral head usually dislocates to lie anteriorly and slightly inferiorly to the glenoid. Patients often present supporting the affected arm with the uninjured arm.

The diagnosis is usually obvious on examination. Look for:

- Step-off deformity at the acromion with a palpable gap below the acromion.
- Humeral head palpable anteroinferiorly to the glenoid.
- Evidence of complications—check especially for distal pulses and ↓ sensation over the lateral aspect of the shoulder (the 'regimental badge' area) supplied by the axillary nerve.

Give analgesia and support in a temporary sling. X-ray before reduction to exclude associated fractures. X-rays show loss of congruity between the humeral head and the glenoid. The humeral head is displaced medially and inferiorly on an AP shoulder X-ray.

## Treatment

Reduce under sedation/analgesia, with full monitoring, using one of the methods described in the next sections below. The choice of technique is personal and depends partly upon familiarity. Apply minimal force to prevent humeral fracture or further soft tissue damage. In patients with habitual recurrent dislocation (and in a significant proportion of other patients as well), reduction may be easily achievable with minimal use of drugs [eg Entonox® or methoxyflurane (Penthrox®) alone]. Take time and perform the manoeuvre slowly. Note that in situations where IV sedation cannot be used or needs to be avoided, intra-articular lidocaine is an option.

## External rotation method

This simple technique has a good rate of success. With the patient reclining at 45°, slowly and gently (without force) externally rotate the shoulder to 90°. If the dislocation has not yet reduced, forward flex (elevate) the shoulder slowly.

## Modified Kocher's method

Lie the patient back almost flat, and once sedation and analgesia are adequate:

- With the elbow flexed to 90°, slowly externally rotate the shoulder. Pause if there is any resistance and continue only when muscles relax.
- Slowly adduct the upper arm across the chest, with the shoulder still held in external rotation.
- Once adducted as far as possible, internally rotate the shoulder by flipping the forearm towards the opposite shoulder.

Reduction may occur at any time during the manoeuvre—success is more likely if the patient is relaxed (avoid traction) and if initial external rotation reaches 90°. A 'clunk' or return of the normal glenoid contour confirms success.

### Modified Milch method

Slowly abduct the straight arm to 110°. With the elbow extended, apply gentle steady traction to the arm, whilst an assistant controls movement of the humeral head back into the glenoid.

### Other techniques

*Scapular manipulation* With the patient lying prone, 'manipulate' the scapula onto the glenoid by pushing the inferior tip of the scapula medially and the superior part laterally.

*Stimson's technique* A more traditional method with the patient prone. Apply a weight strapped to the forearm/wrist of the affected side as it hangs down and await reduction.

*Hippocratic methods* Many techniques have been described over many centuries but are probably of historical interest only.

*Post-reduction* After reduction, recheck pulses and sensation (including axillary and radial nerves), and obtain a check X-ray. Immobilize in a collar and cuff and body bandage. Local policy sometimes includes shoulder immobilization webbing or braces as standard. Provide analgesia (eg co-dydramol) and arrange follow-up. If unsuccessful or difficult or if the shoulder has been dislocated for >24hr, refer for reduction under GA.

### Fracture–dislocation of the shoulder

Most involve fractures of the greater tuberosity associated with anterior dislocation of the shoulder (see Fig. 9.40). Reduce under sedation, as with uncomplicated dislocations—in most cases, the fracture will reduce satisfactorily, along with the dislocation. However, refer large or complex fracture–dislocations involving the humeral head, neck, or shaft.

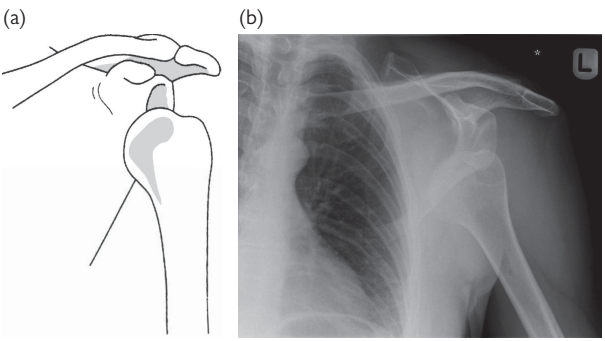


**Fig. 9.40** (a) Diagram of anterior dislocation of the left shoulder; (b) X-ray of anterior dislocation of the left shoulder.

# Posterior shoulder dislocation

This uncommon injury is easy to miss. It results from a blow onto the anterior shoulder or a fall onto the internally rotated arm. It may also occur during seizures or after an electric shock (when other injuries and medical problems may be partly responsible for it being initially overlooked). The patient presents with the shoulder internally rotated. AP shoulder X-ray may appear normal, but careful inspection reveals an abnormally symmetrical appearance of the humeral head ('light bulb sign') and loss of congruity between the humeral head and the glenoid (see Figs. 9.41 and 9.42). A modified axial shoulder X-ray (from above) or a translateral view confirms posterior dislocation. Manipulate under sedation—apply traction and external rotation to the upper limb at 90° to the body. If difficult, refer for reduction under GA. Treat and follow up as for anterior dislocation.

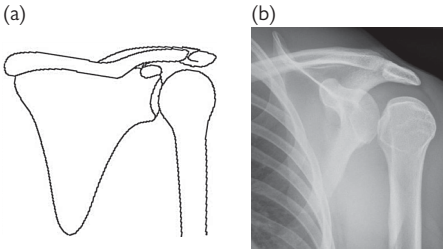


**Fig. 9.41** AP view of posterior shoulder dislocation—light bulb sign.

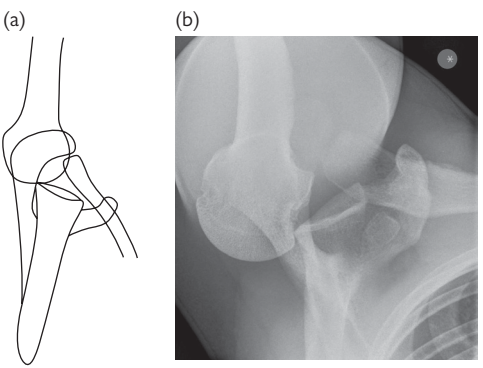


**Fig. 9.42** Modified axial view of posterior shoulder dislocation.

## Luxatio erecta

This is a rare inferior dislocation of the humeral head. The patient presents with the arm held abducted above the head. Check carefully for neurovascular complications. Reduce under sedation by traction in line with the abducted upper arm, followed by adduction of the shoulder. This may require reduction under GA. Treat and follow up as for anterior dislocation.

# Clavicle and AC joint injuries

**Clavicle fracture** (See ➲ Fracture clinic and alternatives, pp. 436–7.)

This common injury results from direct trauma or from falls onto an outstretched hand or point of the shoulder. Check carefully for neurovascular complications (these are rare, but potentially life-threatening).

*Treat* with analgesia and a broad arm sling, and arrange fracture clinic follow-up. The vast majority of fractures unite satisfactorily with conservative treatment. Rarely, grossly displaced fractures are internally fixed.

## Acromio-clavicular (AC) joint injury

These are common injuries which usually follow falls onto the shoulder. Look for tenderness, swelling, or a palpable step over the AC joint. X-rays show AC joint disruption (vertical subluxation of the AC joint by >1–2mm).

- *Grade I*: minimal separation. Only AC ligaments are involved.
- *Grade II*: obvious subluxation, but still some apposition of bony ends.
- *Grade III*: complete dislocation of the AC joint, indicating rupture of the conoid and trapezoid ligaments, in addition to the AC ligaments (see Fig. 9.43).

*Treat* with analgesia and support in a broad arm sling, and arrange follow-up for grade II and III injuries. These measures allow complete recovery in most cases. Occasionally, selected patients benefit from internal fixation.

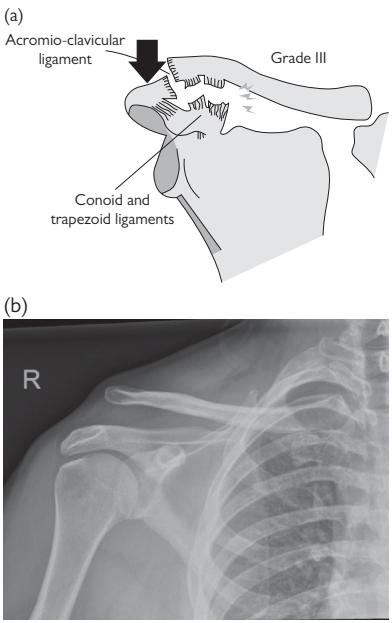


**Fig. 9.43** (a) Diagram of grade III dislocation of the AC joint; (b) X-ray of grade III dislocation of the AC joint.

# Humeral neck/head fracture

These result from direct trauma to the upper arm or from falls onto an outstretched hand. Examine for tenderness or swelling over the proximal humerus. Shoulder movements are usually limited by pain. X-rays typically reveal impacted or oblique fractures, with or without associated fractures of the greater and lesser tuberosities. Fractures may be classified as two-, three-, or four-part fractures, according to the number of fragments resulting (eg a fractured humeral neck combined with a fractured greater tuberosity will be a 'three-part fracture') (see Fig. 9.44).

*Treat* with a collar and cuff support, analgesia, and follow-up. Warn the patient to expect significant visible bruising to appear, extending down the arm towards the elbow (for this reason, it is helpful to document the lack of any clinical evidence of elbow injury at first presentation). Discuss with the orthopaedic team all comminuted, displaced, or markedly angulated humeral neck fractures as MUA and, occasionally, internal fixation/hemi-arthroplasty are indicated.

## Isolated greater tuberosity fracture

Occasionally, there is an isolated fracture through the greater tuberosity, with no fracture through the surgical neck (see Fig. 9.45). Provide analgesia; treat in a sling/collar and cuff, and arrange fracture clinic follow-up.

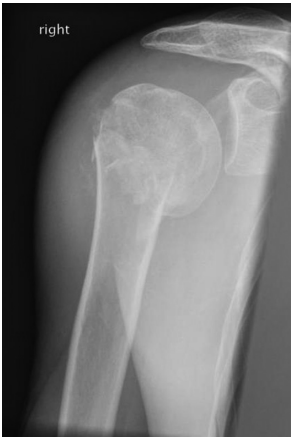


**Fig. 9.44** X-ray of three-part humeral neck fracture with displacement and angulation.

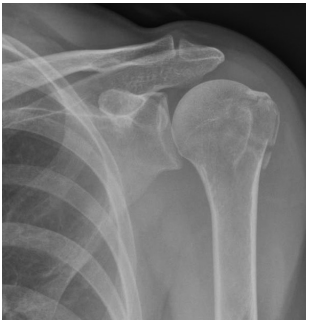

**Fig. 9.45** X-ray of isolated greater tuberosity fracture.

# Other shoulder injuries

## Rotator cuff tears

Acute tears of the rotator cuff (the supraspinatus ruptures most commonly) usually follow chronic rotator cuff disease in patients >40y. They may follow trauma (eg fall with hyperabduction or hyperextension of the shoulder). Examine for ↓ range of movement, weakness, crepitus, and tenderness over the cuff insertions and subacromial area. Check supraspinatus strength by testing resistance to abduction, but remember that pain from the acute injury may preclude a full assessment in the ED. Look for bony avulsions on X-ray (tensile strength of the cuff exceeds that of adjacent bone). Treat suspected rotator cuff tears conservatively initially with analgesia and support in a broad arm sling, followed by exercises/physiotherapy at ~10 days. Arrange follow-up for patients with significantly ↓ range of movement—complete tears (particularly in younger patients) may require surgical repair. Inability to actively abduct to 90° at ~10 days suggests a complete tear, which will be apparent on MRI.

## Scapular fracture

This usually results from direct trauma and implies a forceful mechanism of injury (see Fig. 9.46). Check carefully for associated injuries to the thorax such as rib fractures or haemo-pneumothorax.

*Treat* isolated fractures with a broad arm sling and analgesia, and arrange follow-up.

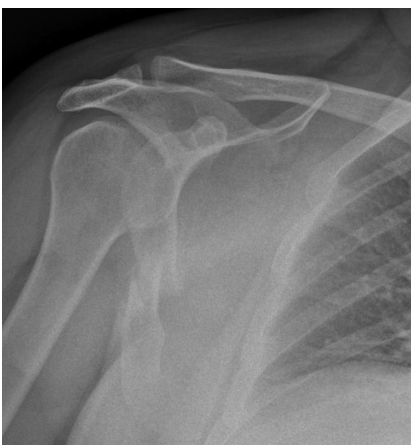

**Fig. 9.46** X-ray of right scapular fracture.

# Soft tissue shoulder problems

The extreme mobility of the shoulder is at the expense of stability which relies heavily on the rotator cuff. The rotator cuff comprises the supraspinatus (initiates abduction), infraspinatus and teres minor (externally rotate), and subscapularis (internally rotates). The rotator cuff may be injured acutely or damaged from a chronic degenerative process (eg impingement syndromes or RA).

## Impingement syndromes

The acromion process may compress or 'impinge' on the underlying subacromial bursa and rotator cuff during repetitive or strenuous shoulder use. The supraspinatus and its tendon are most commonly affected. Minor impingement is associated with inflammation, pain, and loss of function and is reversible with treatment. Rotator cuff tendonitis is more chronic and can lead to degeneration or tearing of the cuff. Although rotator cuff tendonitis and degenerative tears usually occur in later life, acute tears can occur in younger patients.

## Examination of the shoulder

Examine both shoulders for comparison with the patient sitting relaxed.

- *Look for* deformity of the clavicle or sternoclavicular joint, AC joint deformity (eg OA or injury), wasting of the deltoid muscle (axillary nerve damage), a step in the deltoid contour, or a gap below the acromion (subluxation or dislocation).
- *Feel for tenderness* over the sternoclavicular joint, clavicle, AC joint, subacromial area, rotator cuff insertion, and biceps tendon insertion.
- *Move the shoulder* gently in all directions to test passive movements. Test the strength of active movements.
- *Examine for* crepitus on movement, restriction, pain (note any painful arc), and weakness of particular movements.

Test sensation over the badge area (upper outer arm) supplied by the axillary nerve. Examine the cervical spine when shoulder examination does not reveal a cause for symptoms.

*In suspected impingement syndromes, consider*

- *Neer's impingement test*: fully abducting the straight arm will re-create symptoms.
- *Hawkin's impingement test*: hold the arm at 90° abduction and 90° elbow flexion. Rotating the arm across the body will re-create symptoms.

*LA* Injection of 10mL of 1% plain lidocaine into the subacromial bursa (approach just under acromion process from behind) should help the pain but will not affect the strength or range of movement, allowing assessment. Adding hydrocortisone, methylprednisolone, or triamcinolone to LA injection is useful for a first presentation of acute impingement. Note that symptoms may ↑ briefly after steroid injection. Repeated injection can precipitate tendon rupture.

### Subacromial bursitis

This is an early form of impingement in younger patients. It follows unaccustomed activity or exercise. Look for a painful arc of 60–100° abduction, with dull, aching pain worse on activity. The differential diagnosis includes gout, sepsis, or RA. Treat with analgesia, NSAID, and ice. Demonstrate simple exercises (eg gentle pendulum swings and circling movements of the arm, crawling fingers up a wall). LA injection will improve pain and movement, and help confirm the diagnosis. Consider steroid injection if first presentation.

### Rotator cuff tendonitis/tendinopathy

Usually a longer history and chronic pain (± sleep disturbance) in patients aged 25–40y. Examine for tenderness and crepitus over humeral insertions of the rotator cuff and ↓ active and passive shoulder movements. X-ray may show osteophytes or subacromial calcification. LA injection may ↓ pain but usually does not ↑ the strength or range of movement. Treat as for subacromial bursitis. In more severe cases, consider formal physiotherapy and orthopaedic referral.

### Calcific tendonitis

A poorly understood process of calcium deposition and resorption within the rotator cuff tendon. Commoner in women. May be related to degenerative change or follow minor trauma. Most common site is within supraspinatus 1–2cm proximal to humeral insertion. Acute pain (occurs during periods of calcium resorption, granulation, and healing) often starts at rest, worsens on movement and at night. Examine for tenderness at the rotator cuff insertion. There may be crepitus, painful limitation of movement or a painful arc. The calcium deposits may be evident on X-ray.

Most episodes spontaneously resolve in 1–2 weeks. Treat with analgesia, NSAID, and ice. Immobilize briefly in a broad arm sling, but start gentle exercises (as described earlier) once symptoms allow. Arrange orthopaedic follow-up—steroid injection and/or physiotherapy and, rarely, surgical treatment may be required.

### Adhesive capsulitis

A misleading term, since it is caused by generalized contracture of the shoulder capsule, not adhesions. Causes include immobilization, injury, or diabetes. More common in women and rare in those <40y or >70y. Insidious onset results in diffuse, aching pain (worse at night) and restricted active and passive shoulder movements. The cuff is usually not tender. X-rays exclude posterior dislocation (see ➔ Posterior shoulder dislocation, p. 470). Refer to orthopaedics for MUA, arthroscopy, and capsulotomy.

### Other causes of shoulder pain

These include referred pain from degenerative cervical spine, C5/6 disc prolapse, brachial plexus neuritis, axillary vein thrombosis, IHD, suprascapular nerve compression, Pancoast's syndrome, and a cervical rib.

# Soft tissue neck injuries

Neck injuries that do not involve fractures, dislocations, ligamentous laxity, or spinal cord damage are common. Most follow car crashes involving neck hyperextension. These injuries may be given various labels but are most simply 'neck sprains'. Patients with continuing symptoms are often referred to as having a 'whiplash-associated disorder'. MRI (which rarely changes management) reveals many to have significant soft tissue injuries.

## History

Neck pain and stiffness may not appear until 12hr after injury—symptoms are typically maximal at ~48hr. Ask about other symptoms (some are relatively common), which include: headache, shoulder pain, backache, and altered limb sensation. A range of other symptoms may also occur, including: dizziness, tinnitus, vertigo, and visual disturbance.

## Examination

Perform a neurological examination. In fully alert, neurologically intact patients, examine for any midline or paravertebral tenderness, muscle spasm, or deformity. If there is no midline tenderness, assess active neck movements. If there is localized bony tenderness, pain on active movements, or any neurological symptoms, immobilize fully and X-ray.

## X-ray[1]

Arrange cervical X-rays (AP, lateral, and odontoid peg views) in the presence of high-energy trauma, neurological symptoms or signs, ↓ conscious level, or serious injury elsewhere. In the absence of these, do not routinely X-ray if the patient is fully conscious, has no midline neck tenderness, and can rotate the neck by 45° to right and left (see Fig. 9.47).

Check for evidence of fracture or dislocation. The most common abnormality is loss of the normal cervical lordosis (neck 'straightening')—this implies neck muscle spasm and does not necessarily indicate cervical spine injury. If the patient has severe pain or any abnormal neurology, but the initial plain X-rays are normal, consider requesting a CT scan.

## Treatment

If there is any clinical or radiological suspicion of vertebral or spinal cord injury, refer urgently, maintaining cervical spine immobilization.

Treat patients in whom there is no suspicion of spinal cord or vertebral injury with initial analgesia (eg co-dydramol and/or ibuprofen) and advise GP follow-up. Leave referral to a physiotherapist for the GP to decide, based upon progression of symptoms. Avoid the use of a soft collar (the evidence is against it), but instead encourage early mobilization.

## Prognosis

The rate of resolution of symptoms after neck sprains is highly variable. Many patients (>40%) continue to complain of pain, stiffness, and other symptoms for many months. It is often difficult to make a long-term prognosis within 12 months of the injury.

[1] Available at: http://www.ohri.ca/emerg/cdr/cspine.html

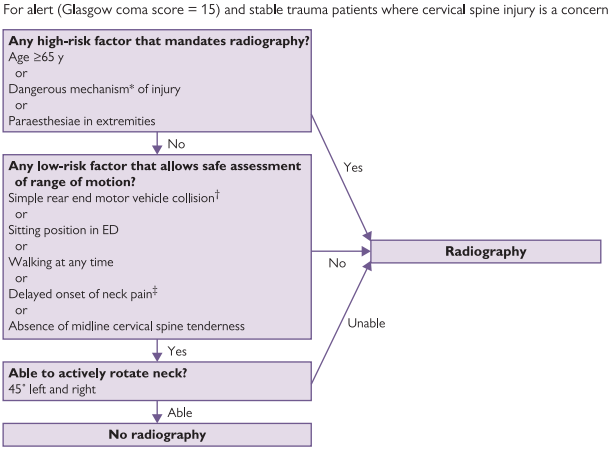


**Fig. 9.47** Canadian C-spine rule. Rule not applicable if: non-trauma cases, GCS <15, unstable vital signs, age <16y, acute paralysis, known vertebral disease, or previous surgery of cervical spine. * Fall from elevation ≥0.9m (3ft)/five stairs, axial load to head, eg diving, motor vehicle collision high speed, (>100km/hr), rollover, ejection, motorized recreational vehicles, bicycle struck, or collision. † Excludes: pushed into oncoming traffic, hit by bus or large truck, rollover, hit by high-speed vehicle. ‡ Not immediate onset of neck pain.

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## Non-traumatic neck pain

Neck pain without injury may result from a variety of causes:

- *Cervical disc herniations*: present with neck pain and sensory and motor signs. Even if X-rays are normal, refer for further investigation (such as MRI) and treatment.
- *Acute torticollis ('wry neck')*: reflects painful sternocleidomastoid spasm, which may occur on waking or after sudden neck movement. It responds to NSAIDs, local heat (eg heat pad or hot water bottle), and (in severe cases) physiotherapy.
- *Referred pain*: eg tonsillitis/quinsy (especially in children).
- *Dystonic reactions*: eg drug-induced (see ➲ Complications of psychiatric drugs, p. 635).
- *Cervical arthritis*: including both OA and RA.
- *Spinal infection*: if there is suspicion of spinal sepsis (neck pain with fever, ↑ WCC, and/or ↑ CRP), refer for urgent MRI.

# Facial wounds

(See also ➲ Maxillofacial injuries: introduction, pp. 378–9; ➲ Middle third facial fractures, pp. 380–1; ➲ Zygomatic, orbital, and frontal sinus fractures, pp. 382–3; and ➲ Mandibular fractures, pp. 384–5.)

## Cosmetic considerations

These are very important. The final appearance of a scar depends partly upon the orientation of the wound and its relation to natural skin lines (modified from Langer's description), but also upon initial management. Cleaning is crucial, but do not debride with tissue excision in the ED. Consider suturing facial dog bites (see ➲ Bite wounds, pp. 420–1) and non-contaminated facial wounds up to 24hr after injury (get senior advice first). Close facial wounds in layers, using 5/0 Dexon or Vicryl for deeper layers, with knots tied on the deep aspect. Aim to remove skin sutures (interrupted 6/0 non-absorbable monofilament) at 3 days and replace with Steri-Strips™ to minimize scarring. Consider GA to treat facial wounds in children.

## Damage to parotid duct/gland and facial nerve

This is particularly likely with incised wounds in the pre-auricular area. The facial nerve emerges through the parotid gland to supply the muscles of facial expression—unrepaired injury results in permanent disfigurement. The parotid duct runs transversely forward from the anterior portion of the gland, parallel and inferior to the zygomatic arch, before entering the mouth opposite the second upper molar (look for blood here, as this implies proximal duct injury). Refer for exploration in theatre if there is clinical suspicion of involvement of any of these structures.

## Associated head injury

Consider head or neck injury in all patients with a facial wound.

## Specific wounds

*Lip wounds* Oppose the vermilion border accurately (it is often easiest to do this first). Remember that even a 1mm mismatch will result in a permanent visible abnormality. Close in layers if the wound extends into subcutaneous or muscle layers.

*Tongue and oral wounds* Check the teeth—if any are broken or missing, consider obtaining soft tissue lateral X-rays of the lips to search for embedded fragments. Small superficial lacerations need not be closed, but close deeper ones in layers, using absorbable sutures (eg 4/0 or 5/0 Vicryl/Dexon for mucosal surfaces). Close through and through oral lacerations in layers (mucosa, muscle, subcutaneous tissue, skin).

*Eyebrow wounds* Do not shave the eyebrows. Exclude an underlying fracture by palpation (and X-rays, as appropriate).

*Eyelid wounds* Many may be sutured with 6/0 non-absorbable monofilament. Full eye examination, excluding an FB, is necessary. Refer wounds if there is involvement of the lid margin, loss of tissue, or lacrimal duct (medial canthus) or gland (superolateral) injury is suspected.

*Ears* Involvement of cartilage requires suturing with fine absorbable material (by an ENT specialist) prior to skin closure. Give prophylactic antibiotic cover (eg co-amoxiclav) if there is any contamination.

**Langer's lines** (see Fig. 9.48).

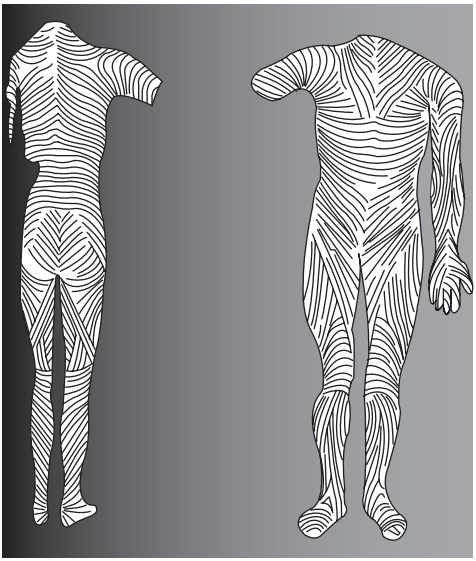

**Fig. 9.48** Langer's lines.

# Pelvic fractures

Major pelvic fractures result from very high-energy trauma and are true orthopaedic emergencies. Associated thoracic or abdominal injuries occur in 10–20%—the principal immediate risk is massive haemorrhage and exsanguination. Compound fractures of the pelvis have a mortality of >50%. Associated bladder or urethral damage is common. Rectal and vaginal injuries occur occasionally.

## Initial assessment and management

- Resuscitate as for any severely traumatized patient (see ➲ Major trauma: treatment principles, p. 330), and arrange a trauma pan-CT scan if major trauma is suspected; otherwise, request pelvic X-ray.
- Look carefully for evidence of hypovolaemia and treat appropriately.
- Examine the pubis, iliac bones, hips, and sacrum for tenderness, bruising, swelling, or crepitus. Do not try to 'spring the pelvis' to assess stability—this is unreliable and unnecessary, and may cause additional haemorrhage/damage. Similarly, avoid log rolling patients with obvious pelvic fractures.
- Apply a pelvic binder if not already applied prehospital.
- Look carefully for wounds, especially in the perineum.
- Defer PR examination until after emergency CT. PR examination may give information on anal tone and palpable fractures, and detect bleeding, rectal tears, and urethral damage (high-riding, boggy prostate).
- Insert a urinary catheter after CT scanning (one attempt only) and test the urine for blood.
- Look at X-rays carefully for disruption of normal pelvic contours (Shenton's lines), asymmetry, and widening of the pubic symphysis or sacroiliac joints.

## Classification of pelvic fractures

(See Table 9.3 and Fig. 9.49.)

**Table 9.3** Tile classification of pelvic injuries

| | |
|---|---|
| Type A | (Stable injuries) include avulsion fractures, isolated pubic ramus fractures, iliac wing fractures, or single stable fractures elsewhere in the pelvic ring |
| Type B | Rotationally unstable but vertically stable |
| B1 | 'Open book' antero-posterior compression fractures, causing separation of the pubic symphysis and widening of one or both sacroiliac joints |
| B2 | Ipsilateral compression causing the pubic bones to fracture and override |
| B3 | Contralateral compression injury resulting in pubic rami fractures on one side and compression sacroiliac injury on the other |
| Type C | Rotationally unstable and vertically unstable. The pelvic ring is completely disrupted or displaced at two or more points. Associated with massive blood loss and very high mortality. Subdivided into C1 (unilateral), C2 (bilateral), and C3 (involving acetabular fracture) |

## Treatment

Stable type A injuries require analgesia and bed rest until able to mobilize (usually 3–6 weeks). *Isolated pubic ramus fractures* are common and often missed in the elderly (particularly when the focus is on a potential fractured neck of femur). Refer to orthopaedics/elderly care team for analgesia, initial bed rest, and then mobilization.

*Unstable type B and C fractures are emergencies*

Resuscitate as for any major trauma (see ➲ Major trauma: treatment principles, p. 330). Correct hypovolaemia; anticipate coagulopathy, and ensure blood is rapidly available as massive transfusion may be required. Minimize movement, but support an obviously unstable pelvic fracture associated with severe haemorrhage using a pelvic binder or splint (eg SAM sling). Involve senior trauma specialists early to consider options which include reduction and immobilization using an external fixator, angiography with selective embolization, or surgical packing in theatre. Interventional radiology is very useful in patients with active arterial pelvic haemorrhage.

## Avulsion fractures around the pelvis

Avulsion fractures occur at attachments of various muscles as follows:

- *Anterior inferior iliac spine*—rectus femoris (typically results from a miskick into the turf—see Fig. 9.50).
- *Anterior superior iliac spine*—sartorius.
- *Ischial tuberosity*—hamstrings.

In most instances, symptomatic treatment based upon rest (consider crutches) and analgesia suffices. Larger avulsions (particularly of the ischial tuberosity) may require internal fixation (to avoid complications such as non-union).

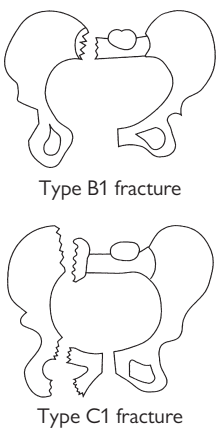


**Fig. 9.49** Examples of pelvic fractures.

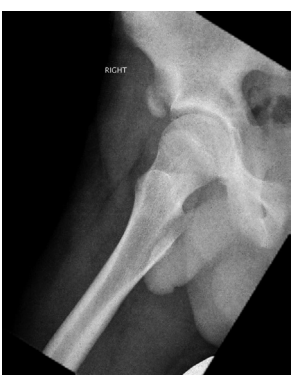


**Fig. 9.50** Avulsion of anterior inferior iliac spine in a 15y old.

# Hip dislocations and acetabular fractures

## Acetabular fracture

(See Fig. 9.51.) Often accompanies traumatic hip dislocation following violent injury. Posterior rim fractures are the most common. Complications include massive haemorrhage, sciatic nerve damage, myositis ossificans, and secondary OA. Resuscitate, give analgesia, and deal with priorities first. CT demonstrates the exact injury better than X-ray. Refer to orthopaedics.

*Central dislocation of the hip*

A serious acetabular fracture, which involves the head of the femur being driven through the acetabular floor following a fall or force directed along the length of the femur (eg car dashboard).

## Traumatic posterior dislocation of the hip

(See Fig. 9.52.) Implies major trauma, often with other critical injuries (eg dashboard knee injury in a car crash) or fractured posterior acetabulum. The limb is shortened and internally rotated, with the hip flexed and adducted. This appearance may be absent if there is also a femoral shaft fracture. Check for sciatic nerve damage—examine foot dorsiflexion and below-knee sensation. Complications: sciatic nerve injury, avascular necrosis of the femoral head (risk ↑ the longer the hip is dislocated), and secondary OA.

Resuscitate, give analgesia, and address ABC priorities. Refer for reduction under GA. In unconscious or anaesthetized patients, reduce in the ED.

*Reduction technique for posterior dislocation ('Allis technique')*

- It is easiest and safest to reduce dislocation if the anaesthetized patient is placed on the floor. If this is not possible, stand on the trolley. An assistant presses down on the patient's anterior superior iliac spines to hold down the pelvis.
- Flex the hip and knee, both to 90°, and correct adduction and internal rotation deformities.
- Grip the patient's lower leg between your knees, and grasp the patient's knee with both hands.
- Lean back and lever the knee up, pulling the patient's hip upward. A 'clunk' confirms successful reduction. X-ray to confirm reduction.

## Anterior dislocation of the hip

Less common. The leg is held abducted and externally rotated. Complications include damage to the femoral nerve, artery, and vein. Give analgesia and refer for reduction under GA.

## Dislocated hip prosthesis

Relatively common (affects ~3% of total hip replacements). It can follow minor (or even no) trauma—sometimes, crossing legs or flexing the hip to 90° can be enough. The patient presents in severe pain, unable to move the hip. Confirm posterior dislocation of the hip prosthesis by X-ray (see Fig. 9.53). Give IV opioid, and refer to orthopaedics for MUA (and assessment of prosthesis stability) under GA. Depending on protocols, expertise, and resources (especially for recovery), it may be possible to do this in the ED.

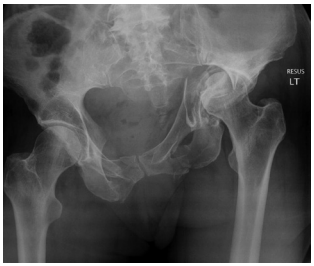


**Fig. 9.51** Major pelvic trauma involving fractures through all four pubic rami, the acetabulum, and the left sacroiliac joint.

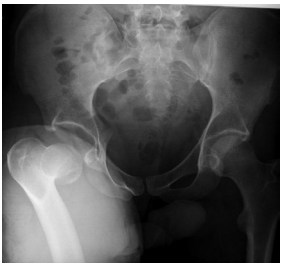

**Fig. 9.52** Posterior hip dislocation.

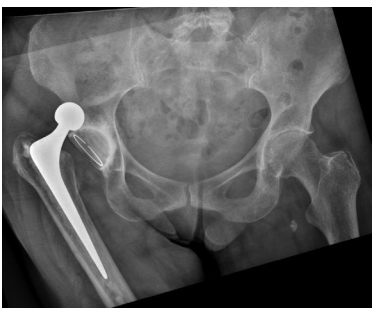

**Fig. 9.53** Dislocated hip prosthesis.

# Sacral and coccygeal fractures

## Fractures of the sacrum

Usually follows violent direct trauma such as falls. Damage to sacral nerve roots may occur. Check carefully for saddle anaesthesia, ↓ anal tone, lower limb weakness, or bladder dysfunction. Refer to the orthopaedic team.

## Fracture of the coccyx

Follows a fall onto the bottom. Do not X-ray routinely—the diagnosis is clinical. Perform a PR examination, and check for local coccygeal tenderness, palpable fractures, or rectal damage. Complications are unusual, but refer patients with rectal tears to the general surgeon and refer to the orthopaedic team if the coccyx is grossly displaced, as it may require manipulation under LA or even excision. Treat the rest (the majority) symptomatically (eg suggest a ring cushion and provide analgesia).

# Hip fractures

## Intracapsular fractures of the neck of femur

Can follow relatively minor trauma. Risk ↑ in the elderly, because of osteoporosis, osteomalacia, and ↑ rate of falls. These fractures can disrupt the blood supply to the femoral head, causing avascular necrosis.

Fractures around the hip in younger patients imply high-energy injury—the incidence of non-union or avascular necrosis may be as high as 20%.

### *Diagnosis*

Usually follows a fall onto the hip or bottom. Pain may radiate down towards the knee. The affected leg may be shortened and externally rotated. Check for hypothermia and dehydration (the patient may have been lying for hours). Look for tenderness over the hip, particularly on rotation. Suspect hip fracture in an elderly person who:

- Exhibits a sudden inability to weight-bear. There may be no history of injury, particularly in the presence of confusion or dementia.
- Is unable to weight-bear and has pain in the knee (the hip may not be painful).
- Has 'gone off her feet'.

### *X-rays*

Look closely for disrupted trabeculae/cortices and abnormal pelvic contours (Shenton's lines). Fractures of the femoral neck are not always visible on initial X-rays. Repeat X-rays, CT, or MRI may be required if symptoms continue. Intracapsular femoral neck fractures are graded according to the Garden classification (see Table 9.4 and Fig. 9.54).

**Table 9.4** Garden classification of intracapsular hip fractures

| | |
|---|---|
| Garden I | Trabeculae angulated, but inferior cortex intact. No significant displacement |
| Garden II | Trabeculae in line, but a fracture line visible from superior to inferior cortex. No significant displacement |
| Garden III | Obvious complete fracture line, with slight displacement and/or rotation of the femoral head |
| Garden IV | Gross, often complete, displacement of the femoral head |

### *Treatment*

- Obtain IV access and send blood for U&E, glucose, FBC, and cross-match.
- Start IVI if indicated (eg dehydration or shock).
- Give IV analgesia plus an antiemetic. Provide all analgesia IV in small increments every few minutes until pain is controlled.
- Perform a fascia iliaca compartment block (see ➔ Fascia iliaca compartment block, p. 312).
- Obtain an ECG to look for arrhythmias/MI, and consider the need for CXR.
- Arrange other investigations as indicated by the history/examination.
- Admit to the orthopaedic ward.

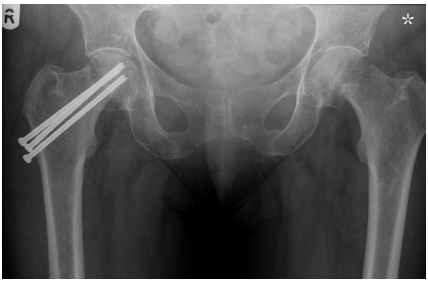

**Fig. 9.54** Garden IV left subcapital hip fracture with previous fixation to the right side.

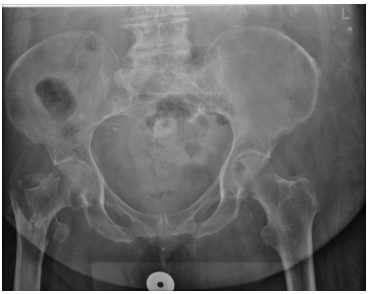

**Fig. 9.55** Comminuted intertrochanteric right hip fracture.

### Intertrochanteric fracture

These affect the base of the femoral neck and the intertrochanteric region (see Fig. 9.55). Initial management is identical to that for neck of femur fractures outlined previously.

### Isolated trochanteric avulsion fracture

Sudden force may avulse insertions of the gluteus medius (greater trochanter) or iliopsoas (lesser trochanter). Give analgesia; assess mobility with crutches, and refer for follow-up for gradual mobilization and symptomatic treatment.

### Hip pain after injury, but no fracture seen

Elderly patients who report hip pain and struggle to walk after a fall, and yet have no fracture of the hip or pubic rami on X-ray, may need assessment by an occupational therapist or physiotherapist before deciding if they can be safely discharged home with analgesia and appropriate walking aid. A small, but significant, proportion of such patients will turn out to have a hip fracture. In patients with significant pain and difficulty weight-bearing, consider requesting a CT (or MRI) scan at the time of initial presentation to show up a hip fracture not identified on plain X-rays.

For patients who have normal X-rays, are able to mobilize satisfactorily, and are being discharged, advise them to return for review and further imaging (CT or MRI) if the pain continues for >1 week—ideally, provide an advice leaflet to explain and reinforce this.

# Shaft of femur fractures

Enormous force is required to break an undiseased adult femoral shaft. Fractures are frequently associated with multisystem trauma. Treatment of immediately life-threatening injuries takes priority. Transverse, spiral, or segmental shaft fractures usually result from falls, crushing injuries, or high-speed road traffic collisions. There is often associated dislocation of the hip or other serious injury to the lower limb ± major trauma affecting the head, chest, abdomen, and pelvis.

## Complications

Closed fractures of the femoral shaft, even without obvious vascular injury, may be associated with marked blood loss. Up to 1.5L of blood may be lost without visible thigh swelling. Rarely, gross blood loss may occur from compound femoral fractures. Later complications include fat embolism/ARDS. The incidence of complications is ↓ by early splintage and early definitive treatment (usually closed intramedullary nailing).

## Diagnosis

The diagnosis is usually clear on examination, with deformity, shortening, external rotation, and abduction at the hip on the affected side. The fracture may be felt or even heard on movement of the lower limb. Carefully check for associated pelvic, knee, or distal limb injuries or for the presence of associated wounds. Document sensation and pulses in the limb, and recheck frequently.

## Treatment

Before X-rays, resuscitate, exclude life-threatening injuries, replace IV fluids, give adequate analgesia, and splint fractures as follows:

- Assess ABC, establish priorities, and resuscitate as for patients presenting with major trauma (see ➲ Major trauma: treatment principles, p. 330).
- Commence fluid replacement via two large-bore IV cannulae—start with 1000mL of 0.9% saline.
- Obtain blood for cross-matching, plus U&E, FBC, and coagulation screen.
- Administer IV analgesia—give small increments of opioid (with an antiemetic) until pain is controlled.
- Give IV tranexamic acid (see ➲ Major trauma: treatment principles, p. 330).
- Strongly consider femoral nerve block (see ➲ Femoral nerve block, p. 313) or fascia iliaca block (see ➲ Fascia iliaca compartment block, p. 312). As this starts to take effect (~5–10min), prepare splintage and immobilize in a traction splint (eg Kendrick).
- Arrange imaging of the femur—very often a trauma pan-CT scan of the head, neck, chest, abdomen, and pelvis can be appropriately extended down to involve the femoral shafts.

*Note*: there was a time when the August and February junior doctor job changeover in the UK took a terrible toll on sleep-deprived doctors who fell asleep whilst travelling long distances between hospitals. The doctor who sustained the fractures shown in Fig. 9.56 as part of multiple injuries was successfully resuscitated in the middle of the night by a senior ED doctor—both later joined forces to write the first edition of the *Oxford Handbook of Emergency Medicine*.

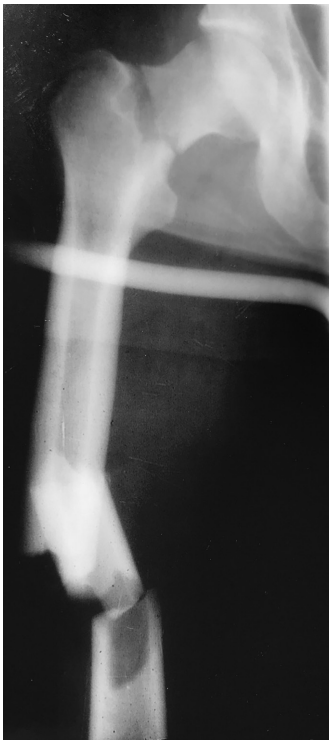

**Fig. 9.56** Comminuted right femoral shaft fracture with a 'butterfly fragment' plus a Garden II intracapsular hip fracture in a 26y old doctor involved in a high-speed road traffic collision with a lorry.

### Subtrochanteric fractures

These involve the most proximal part of the femoral shaft, at or just distal to the trochanters. They typically involve high-energy trauma in younger patients and are often associated with other serious injuries. They can also occur as isolated injuries following relatively minor trauma in those with osteoporosis or metastatic disease. Treat as for femoral shaft fractures.

### Supracondylar femoral fractures

Fractures of the distal third of the femur usually follow violent direct force. They are frequently comminuted and often intra-articular with associated damage to the knee joint. In adults, the distal femoral fragment tends to rotate due to pulling from the gastrocnemius. Treat as for femoral shaft fractures, but note that femoral nerve block may not be as effective.

# Approach to knee injuries

## History

Many knee injuries result from sports, particularly football and rugby. Carefully elicit the exact mechanism of injury, as it provides clues to the diagnosis. Valgus or varus stresses can damage the medial and lateral collateral ligaments, respectively. Flexed, twisting knee injuries are frequently associated with meniscal injuries. The anterior cruciate ligament (isolated or associated with medial collateral and/or medial meniscal injuries) may tear during forced flexion or hyperextension. Posterior cruciate injuries may follow falls or dashboard impact where the tibia is forced backwards violently (often associated with medial or lateral ligament injuries).

Rapid-onset tense swelling in a knee is usually an *acute haemarthrosis*. Swelling developing more gradually over several days is more likely to represent a reactive effusion. Ask about previous knee problems: swelling, clicking, locking, or giving way (the last two suggest underlying meniscal pathology). Document any previous knee surgery or the presence of other joint problems. In a hot, swollen, painful, and stiff knee without a history of significant trauma, consider and exclude septic arthritis.

## Examination

Examine both legs, with the patient lying supine. If there is much discomfort, consider giving oral analgesia and re-examine in 10–15min. Reassure him/her that you will not suddenly pull or move the leg without warning.

- *Observe* how the patient mobilizes (or not) to enter the examination cubicle.
- *Look for* bruising, swelling, redness, abrasions, or other wounds.
- *Feel* for warmth, crepitation, or the presence of a knee effusion.
- *Check straight leg raise*: the ability to do this against resistance virtually excludes quadriceps and patellar tendon rupture or transverse patellar fractures. If unable (possibly due to pain), ask the patient to kick forwards whilst sitting, with the affected leg dangling free.
- *Assess tone and bulk* of the quadriceps muscle, and compare sides.
- *Assess knee movement*: gentle encouragement or supporting the limb may be required, but do not use any force.
- *Assess the cruciate ligaments*: this may not be easy or possible soon after some acute knee injuries. If the patient is struggling to achieve 90° flexion, assess with slight flexion (~10°) (Lachman test).
- *Assess the collateral ligaments*: with the leg straight, gently apply a valgus stress to the knee joint (ie move the lower leg laterally), examining for laxity or pain in the medial collateral ligament. Next apply a varus stress (ie move the lower leg medially), examining for laxity or pain in the lateral collateral ligament complex. Repeat the procedure with the knee in ~20° flexion, as this will relax the cruciate ligaments. Compare both sides.

### X-rays for knee injuries

X-rays are the mainstay of initial imaging—CT and MRI may be indicated after specialist consultation. Obtain X-rays if there is suspected fracture or other significant injury. Use the Ottawa knee rules to assist the decision (in those aged between 18 and 55y) as to whether or not to X-ray.

X-rays are only required if any of the following are present:

- There is isolated bony tenderness of the patella.
- There is bony tenderness over the fibula head.
- The patient cannot flex the knee to 90°.
- The patient could not weight-bear (take at least four steps), both immediately after the injury and at the time of examination.

Adopt a lower threshold for obtaining X-rays in those aged <18 or >55y, patients intoxicated with alcohol, those suffering from bone disease (eg RA, documented osteoporosis), and those who re-attend the ED with the same injury (having not been X-rayed initially).

## Patella fracture

This may follow a direct blow or fall onto the patella or sudden violent knee flexion or contraction of the quadriceps muscle. Look for pain, swelling, crepitus, and difficulty extending the knee. Displaced transverse fractures result in an inability to straight leg raise (this is also a feature of rupture of the quadriceps tendon or patellar tendon—see ➲ Soft tissue knee injuries, p. 492). There may be associated haemarthrosis.

*X-rays* may be difficult to interpret, as the patella overlies the distal femur on the AP view and can obscure subtle fractures (see Fig. 9.57). Take care not to mistake a bipartite patella for a fracture (the accessory bone is typically in the upper, lateral part of the patella).

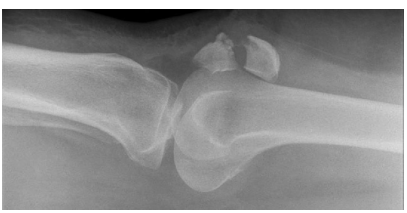

**Fig. 9.57** Lateral X-ray showing a transverse fracture of the patella with separation of the fragments.

### Treatment

- Treat vertical fractures with analgesia; immobilize in a non-weight-bearing above-knee POP backslab; supply crutches, and arrange orthopaedic follow-up.
- Transverse fractures tend to displace due to the pull of the quadriceps. Treat with analgesia and immobilization in a POP backslab, and refer to the orthopaedic team for probable ORIF (occasionally, the orthopaedic team may treat an undisplaced transverse fracture conservatively).

# Dislocations of the patella and knee

## Dislocation of the patella

The patella typically dislocates laterally. This often follows medial stress to the knee—the dislocation may reduce spontaneously. There may be a history of recurrent dislocation. The patient has a painful knee, held in flexion, with obvious lateral displacement of the patella. X-rays are not generally required prior to reduction of the dislocation. Reduction can usually be achieved using Entonox®—IV analgesia is seldom required. Stand on the lateral side of the affected limb and hold the affected knee gently. Using a thumb, lever the patella medially in one smooth, firm movement, whilst gently extending the knee at the same time. Successful reduction is obvious and should rapidly relieve symptoms. Once reduced, obtain X-rays, immobilize in a canvas ('cricket pad') back-splint or backslab cast POP, provide analgesia, and arrange orthopaedic follow-up. An MRI scan at follow-up may help to identify the extent of damage to the medial patellofemoral ligament—a knee specialist will decide about possible surgical repair.

*Spontaneous reduction/patella subluxation* The patient who has experienced spontaneous reduction and/or subluxation prior to arrival at the hospital will typically have maximal tenderness over the medial aspect of the upper patella, reflecting damage to the attachment of the vastus medialis. There may be 'apprehension' when gentle lateral pressure is applied to the patella. If clinical features are dramatic, rest in a splint (occasionally, cylinder POP may be needed); otherwise refer for physiotherapy and orthopaedic follow-up.

## Dislocation of the knee

Although rare, this injury indicates severe disruption of the ligamentous structures and soft tissues of the knee. Look carefully for associated injuries (eg femur or lower limb), and document distal pulses and sensation—the popliteal artery or nerve are often injured. Reduction requires adequate (IV opioid) analgesia and usually GA or sedation with full precautions. Reduce by simple traction on the limb, correcting the deformity. Check distal pulses and sensation after reduction; immobilize in a long leg POP backslab, and arrange orthopaedic admission. Check the circulation repeatedly, since popliteal artery damage may not become apparent for some hours—angiography is usually required. Compartment syndrome is another recognized complication.

# Tibial plateau fracture

Falls onto an extended leg can cause compression fractures of the proximal tibia. Valgus stresses crush or fracture the lateral tibial plateau. These injuries are commonly seen in pedestrians injured following impact with car bumpers. Varus injuries result in crushing or fracture of the medial tibial plateau and are usually associated with rupture of the opposite collateral ligaments. Examine for tenderness over the medial or lateral margins of the proximal tibia. Look for swelling, haemarthrosis, or ligamentous instability (also try to assess the cruciate ligaments—see ➲ Approach to knee injuries, pp. 488–9). Look carefully on X-rays for breaks in the articular surfaces of the proximal tibia, avulsions from the ligamentous attachments, or loss of height from the medial and lateral tibial plateaux, but beware this may be subtle (see Fig. 9.58). Adopt a low threshold to request a CT scan to clarify the nature and extent of the injury.

*Treat* with immobilization in a long leg POP backslab, following adequate analgesia, and refer to orthopaedic staff. Fractures of the tibial plateau often require elevation ± ORIF with bone grafting. Admit all patients with acute haemarthrosis. Treat small, isolated avulsions without haemarthrosis with immobilization, crutches, and analgesia, and arrange orthopaedic follow-up.

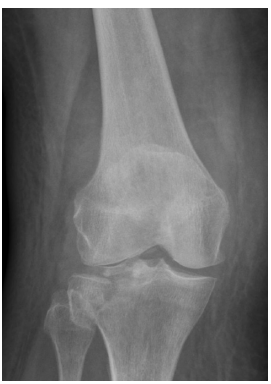

**Fig. 9.58** AP knee X-ray showing a displaced lateral tibial plateau fracture, with a large associated joint effusion.

### Postero-lateral corner injuries

The postero-lateral corner of the knee comprises a group of ligaments and muscles/tendons that add to the stability of the joint. Postero-lateral corner injuries often occur in association with other significant knee trauma (eg dislocations, rupture of anterior or posterior cruciate ligaments), but isolated injuries can occur. Suspect this injury when significant symptoms follow the application of varus force to the anteromedial aspect of the extended knee. Chronic instability can result. X-rays may be normal or show subtle avulsions or widening of the lateral joint space. Urgent MRI and orthopaedic referral will enable prompt treatment.

# Soft tissue knee injuries

## Acute haemarthrosis

Rapid-onset swelling following a knee injury, often warm, tense, and painful. Common causes include cruciate ligament rupture, tibial avulsion, and tibial plateau or other fractures. An acute haemarthrosis indicates serious injury. Refer for orthopaedic appraisal following splintage, analgesia, and appropriate X-rays. Aspiration of a haemarthrosis (advocated by some experts to provide analgesia) requires a strict aseptic technique.

## Cruciate ligament rupture

Pain and swelling can make it hard to elicit classical physical signs. An audible 'pop' at the time of injury is highly suggestive of anterior cruciate ligament rupture.

*Anterior cruciate ligament tears* often occur in association with tears of the medial collateral ligament and/or medial meniscus. Examine for the presence of haemarthrosis, abnormal ↑ anterior glide of the tibia ('+ve anterior drawer test'), and injuries to the medial collateral ligament or other structures. Look carefully at X-rays for avulsion of the anterior tibial spine (anterior cruciate insertion). Give analgesia, and refer to the orthopaedic surgeon.

In *posterior cruciate ligament tears*, the tibia may appear to sag back when the knee is flexed, so the tibia can be pulled into a more normal position, causing a 'false +ve' anterior drawer test. X-rays may reveal the relevant posterior tibial spine to be avulsed. Provide analgesia and refer.

## Collateral ligament injuries

Tenderness over the medial or lateral collateral ligament, with pain at this site on stress testing, indicates collateral ligament injury. Most injuries are isolated and have no associated haemarthrosis and no abnormality on X-ray. The degree of laxity on stress testing will help to guide treatment:

- Local tenderness with no laxity (or very slight laxity) implies a grade I injury. Treat with analgesia and physiotherapy (± crutches), with an expectation of full recovery in 2–4 weeks.
- Local tenderness with minor/moderate laxity, but with a definite end-point, implies a grade II injury. Provide analgesia, crutches, and instruction on quadriceps exercises, and refer for orthopaedic follow-up.
- Major laxity (ie the joint opening up >1cm) with no end-point implies complete rupture. Consider a POP cylinder (or splint), and provide crutches, analgesia, quadriceps exercises, and orthopaedic follow-up.

## Ruptured quadriceps

Complete rupture of the distal quadriceps insertion can result from a direct injury or from sudden, violent contraction of the quadriceps muscle. Examination reveals complete inability to straight leg raise—never assume this is just due to pain. There may be a palpable defect in the muscle insertion. Refer to the orthopaedic surgeon for repair.

## Ruptured patellar tendon

Examine for complete inability to straight leg raise and a high-riding patella, a palpable defect in the patellar tendon. There is frequently an associated avulsion of the tibial tuberosity. Refer to orthopaedics for repair.

# Other knee problems

## Acutely locked knee

A springy block to full extension (which varies from just a few degrees to much more) in the knee indicates an underlying meniscal injury or other loose body in the knee joint. Obtain knee X-rays (including a tunnel view), which may show a loose body. Do not attempt to unlock the knee by manipulation, as this is usually painful and futile. Give analgesia, and refer for arthroscopy.

## Prepatellar and infrapatellar bursitis

This results from inflammation of the fluid-filled bursa in front of or just below the patella, respectively, typically from unaccustomed kneeling. Treat with rest (which may involve the use of crutches), a short course of NSAID, and avoidance of the causative activity. Persistent symptoms may necessitate elective excision of the bursa. Infective bursitis may occur (↑ T° and cellulitis are clues to this)—aspirate fluid for culture and sensitivity, and start antibiotics (eg flucloxacillin).

## Other causes of knee pain

Patients present not infrequently with knee pain of variable duration and with no history of trauma.

*In adults*, causes include Baker's cyst, OA (especially in the elderly), and acute arthritic conditions, including septic arthritis (rare, but important). Also rare, but worthy of consideration, is osteosarcoma, which typically affects teenagers or young adults, producing pain and swelling.

*In children*, causes include sepsis (including both septic arthritis and osteomyelitis—see ➔ The limping child, pp. 726–7), Osgood–Schlatter's disease, osteochondritis dissecans, Johansson–Larsen's disease (see all in ➔ Osteochondritis, pp. 730–1), chondromalacia patellae, referred pain from the hip, and malignancy (eg leukaemic deposits).

# Tibial and fibular shaft fractures

Adult tibial fractures are usually a result of direct blows or falls onto the tibial shaft. Spiral fractures of the tibia or fibula follow violent twisting injuries, usually from sports (eg soccer, rugby, skiing). Displaced fractures typically involve both the tibia and the fibula. A large portion of the tibia has relatively little soft tissue covering—compound injuries are common. Displaced tibial shaft fractures may be complicated by injury to the popliteal artery and compartment syndromes (see ➲ Crush syndrome, pp. 406–7). Fractures of the proximal fibula may be associated with injury to the common peroneal nerve. Check (repeatedly) for distal pulses and sensation.

*Diagnosis* Is usually easy. Look for deformity, localized swelling, or tenderness. Regard all wounds near the fracture site as potential compound injuries.

*X-rays* Ensure X-rays show the whole length of the tibia and fibula. Examine closely for the presence of other injuries (eg around the knee or ankle).

Stress fractures can occur and may not be visible on initial X-rays. Refer if there are persisting symptoms suggestive of stress fracture.

## Tibial shaft fractures

Treat undisplaced transverse tibial shaft fractures with analgesia and long leg POP backslab. Spiral and oblique fractures also need immobilization but are potentially unstable, so refer to the orthopaedic team for admission. Immobilize displaced fractures in a long leg POP backslab, following IV analgesia, and refer (to consider MUA or closed intramedullary nailing). Badly comminuted or segmental fractures may require ORIF. Contact orthopaedics immediately if suspected vascular injury, sensory deficit, or gross swelling.

Treat compound fractures initially as described in ➲ Open (compound) fractures, p. 349, and refer to the orthopaedic surgeon for urgent wound toilet, debridement, and fixation (see http://www.boa.ac.uk).

## Fibular shaft fractures

These can occur in combination with a tibial fracture, as a result of a direct blow or from twisting injuries. The common peroneal nerve may be damaged in proximal fibular injuries. Examine specifically for weakness of ankle dorsiflexion and ↓ sensation of the lateral aspect of the forefoot.

*Treat* undisplaced proximal or fibular shaft fractures with analgesia and elevation. Consider support with a light bandage. If unable to weight-bear, use a below-knee POP for comfort, with crutches until weight-bearing is possible. Arrange follow-up in all cases. Refer displaced or comminuted fractures.

*Stress fractures of the fibula* are relatively common, typically affecting the fibular neck of military recruits and athletes following vigorous training. Treat symptomatically with rest and analgesia.

## Maisonneuve fracture

(See ➲ Eponymous fractures, pp. 514–18.)

Transmitted forces may fracture the proximal fibula following an ankle injury. This usually involves fracture of the medial malleolus and fracture of the proximal fibula or fibular shaft, and implies damage to the distal tibiofibular syndesmosis. Examine the proximal fibula in all ankle injuries, and X-ray if locally tender.

# Pretibial lacerations

Common in the elderly following relatively minor trauma. Most pretibial lacerations can be satisfactorily treated in the ED with adhesive strips ('Steri-Strips™'). Clean and irrigate to remove clots, and close using Steri-Strips™ under appropriate anaesthesia. Aim to leave gaps of ~0.5cm between the Steri-Strips™. Apply a non-adherent dressing and light compression bandage. Instruct the patient to elevate the limb whenever possible (see Fig. 9.59). Arrange follow-up (ED or GP) for 5 days' time for wound inspection and dressing change (but leave the underlying Steri-Strips™ until the wound is healed). Consider admission for patients with poor social support.

*Note*: suturing pretibial wounds is not usually recommended as the pretibial skin is friable and undue tension compromises wound healing.

*Complications* are likely in patients with large, distally based, and poorly viable skin flaps and in patients on steroids or anticoagulants (check clotting control). Refer to plastic surgeons large lacerations where skin edges cannot be opposed or where complications are likely.

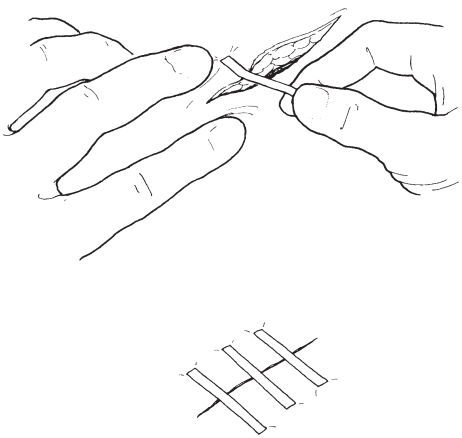

**Fig. 9.59** Application of Steristrips™.

# Calf and Achilles tendon injuries

## Calf muscle tears

Acute tears of the gastrocnemius muscle often occur during sports. They can also occur simply from stepping from a bus or kerb or from a sudden jump. Sharp or burning pain in the calf is followed by stiffness or pain on weight-bearing. Examine for localized tenderness and/or swelling over the calf muscle bellies. The medial head of the gastrocnemius is more commonly injured.

Carefully check the Achilles tendon for signs of rupture (see ➲ Achilles tendon rupture, pp. 496–7). Differential diagnosis includes DVT (see ➲ Deep vein thrombosis, pp. 122–3) or rupture of a Baker's cyst.

Treat with analgesia, NSAID, and initial ice application. Raising the heel with a pad may also help. Advise elevation of the leg and progressive weight-bearing, as guided by symptoms. Use of crutches may be required if symptoms are severe (in this case, arrange follow-up and early physiotherapy).

## Calf muscle bruising

Direct blunt calf trauma can result in haematoma formation and considerable swelling. Be alert to the possibility of compartment syndrome, particularly where there is a significant mechanism of injury (eg see ➲ Crush syndrome, pp. 406–7).

## Achilles tendon rupture

Achilles tendon rupture can occur without prior symptoms during sudden forceful contraction of the calf. Usually this occurs during sports (notoriously badminton). It also occurs in other situations (eg running for a bus or missing a step and landing heavily). Patients on ciprofloxacin or oral steroids or those with a history of steroid injection of the Achilles tendon area are at ↑ risk. The patient often describes a sudden sharp pain ('snap') behind the ankle. Patients may mistakenly initially believe they have sustained a blow to the back of the ankle. Examination may reveal swelling, pain, bruising, and often a (diagnostic) palpable defect (gap) in the tendon ~5cm above the calcaneal insertion. Plantar flexion against resistance will be weaker than on the uninjured side, but do not rely on this when making a diagnosis.

*Beware* plantar flexion (even standing on tiptoes) may still be possible due to action of the tibialis posterior and peroneal and toe flexor muscles.

*Calf squeeze test (Simmonds/Thompson's test)* Kneel the patient on a chair, facing the back, with the feet hanging free over the edge. Alternatively, position the patient to lie prone on a trolley, with the ankles over the end. Gently squeeze mid-calf, and look for normal plantar flexion of the ankle (see Fig. 9.60). To avoid confusion, do not describe the result as +ve or –ve—just state 'calf squeeze test normal' or 'abnormal'.

*Treatment* Remains controversial, so follow local policy. Options are:

- *Conservative management*: many ruptures are managed with crutches, analgesia, and immobilization for 6 weeks in a long leg (equinus) plaster, with the ankle in plantar flexion and the knee flexed to ~45°. This is followed by careful rehabilitation under the care of the orthopaedic team and physiotherapist.
- *Primary surgical repair*: often employed in young patients and athletes. Refer to the orthopaedic team to consider this.

*Note*: sometimes a 'partial' Achilles tendon rupture is suspected. In this instance, the safest initial treatment is immobilization in a non-weight-bearing below-knee POP (BKPOP) with ankle flexion, crutches, and orthopaedic follow-up. USS can help to determine the state of the tendon.

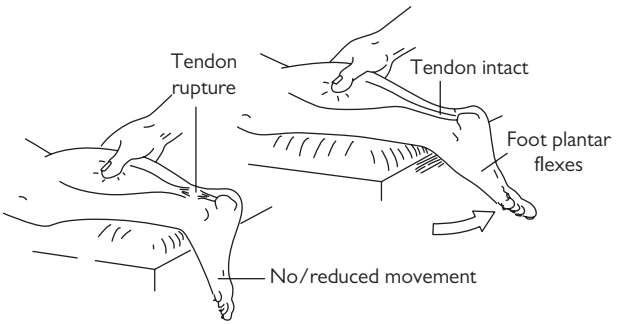


**Fig. 9.60** Calf squeeze test to check the integrity of the Achilles tendon.

### Achilles tendonitis/paratendonitis/tendinopathy

This frequently follows unaccustomed activity or overuse (eg dancing, jumping, running, or even walking) and may be associated with familial hypercholesterolaemia. There is usually a history of ↑ pain, aggravated by ankle movements. Examine for localized pain, swelling, and palpable crepitus over the Achilles tendon (the most common site is ~5cm from its insertion). The calf squeeze (Simmonds) test is normal. Check lipid profile.

*Treat* with analgesia, NSAID, and a brief period of rest (eg 1–2 days) before gradually returning to normal activities, as guided by symptoms. Occasionally, 1–2 weeks in a below-knee walking POP (BKWPOP) may be useful. A heel pad inserted into footwear may help. Athletes may benefit from removal of heel tabs from training shoes if implicated. Avoid local steroid injection, which may ↑ the risk of tendon rupture by impeding healing or by allowing premature resumption of activity.

### Calf/leg pain with no history of trauma

A variety of conditions may be implicated, including:

- *Shin splints*: a variety of pathophysiological processes have been suggested, including tibial periostitis. This condition is characterized by pain over the anterior distal tibial shaft after running on hard surfaces. Advise rest and NSAID.
- *Stress fractures*: can affect the tibia (as well as the fibula—see ➲ Tibial and fibular shaft fractures, p. 494). Treat with analgesia and POP, with orthopaedic follow-up.
- *Bursitis*: inflammation of the bursae around the insertion of the Achilles tendon—responds to conservative measures.
- *DVT* (see ➲ Deep vein thrombosis, pp. 122–3).
- *Cellulitis* (see ➲ Infected wounds and cellulitis, p. 419).
- *Ischaemia* (see ➲ Acute limb ischaemia, p. 538).
- *Ruptured Baker's cyst*.

# Approach to ankle injuries

Ankle injuries are amongst the most common problems presenting to the ED. Adopt a logical, consistent approach to identify which patients are likely to have a fracture and to avoid unnecessary X-rays in patients with uncomplicated sprains.

## History

Establish the exact mechanism of injury. Most are inversion injuries (where the sole of the foot turns to face medially as the ankle is plantar flexed) causing damage to structures around the lateral malleolus (most notably, the anterior talofibular ligament). Eversion injuries occur less commonly and damage the structures around the medial malleolus. Hyper-dorsiflexion and plantar flexion injuries occur less frequently.

The following are relevant in the initial assessment of ankle injuries:

- A fracture is more likely in patients who are unable to weight-bear immediately following the injury.
- A 'crack' or 'snap' may be heard and is not indicative by itself of a fracture.
- Ice, analgesia, and elevation may influence the appearance of an ankle injury.

## Examination

Examine from the knee down for tenderness over the:

- Proximal fibula.
- Lateral malleolus and ligaments.
- Medial malleolus and ligaments.
- Navicular.
- Calcaneum.
- Achilles tendon.
- Base of the fifth MT.

## Is an X-ray required?[1]

Follow the Ottawa ankle rules (see Fig. 9.61) for adults and X-ray the ankles if patients:

- Were unable to weight-bear for four steps both immediately after the injury and at the time of examination.
- Have tenderness over the posterior surface of the distal 6cm (or tip) of the lateral or medial malleolus.

Note that tenderness over the navicular, calcaneum, base of the fifth MT, or proximal fibula require specific X-rays to exclude fractures.

Adopt a lower threshold for X-ray in the very young, the elderly, and in patients who are difficult to assess (eg intoxicated).

[1] Source: data from Stiell IG (1993) Decision rules for the use of radiography in acute ankle injuries. Refinement and prospective validation. *J Am Med Ass* 269: 1127–32.

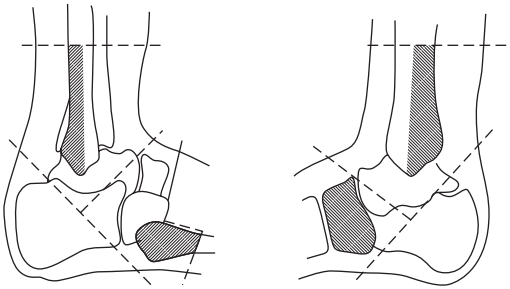

**Fig. 9.61** The Ottawa ankle rules. Guidelines for X-ray in a simple ankle injury. Bony tenderness over the points indicated requires an X-ray. X-ray is also required if the patient is unable to weight-bear immediately after the injury or to walk four steps in the ED. X-ray the ankle for malleolar tenderness and the foot for metatarsal/tarsal tenderness. If the patient is not X-rayed, then they are given instructions to return after 5 days if they have trouble weight-bearing.



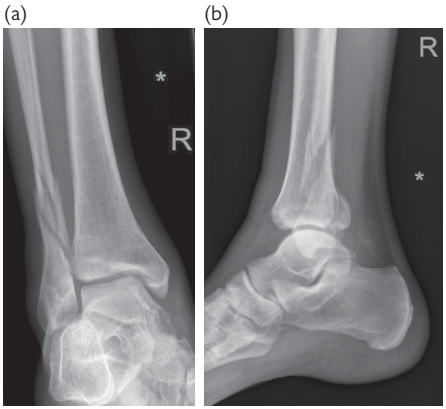


**Fig. 9.62** AP and lateral ankle X-rays showing a (Weber C) fracture of the distal fibula plus a fracture of the posterior distal tibia, but no obvious talar shift.

**Table 9.5** Weber classification of lateral malleolus fractures

| | |
|---|---|
| Weber A | Fracture below syndesmosis, usually stable |
| Weber B | Fracture at the level of syndesmosis, may be stable or unstable |
| Weber C | Fracture above syndesmosis, usually unstable |

# Ankle fractures and dislocations

## Ankle fractures

(See Table 9.5 and Figs. 9.62, 9.63, and 9.64.) Fractures around the ankle most commonly involve the malleoli—medial, lateral, and what is commonly referred to as the 'posterior malleolus' (the posterior part of the distal tibia). The ankle mortice joint allows very little rotation or angulation at the ankle joint, so forced twisting or angulation of the ankle joint causes fractures associated with ligamentous injuries and, in severe cases, disruption of the distal tibiofibular syndesmosis.

*Treatment* This depends upon a combination of clinical findings and X-ray appearances. Look carefully for talar shift.

- *Small avulsion fractures* essentially reflect ligament/joint capsule damage. Treat with rest, elevation, analgesia, and early mobilization, as for sprains.
- *Larger avulsion fractures* may require initial immobilization in a boot ± crutches and orthopaedic follow-up.
- *Undisplaced, isolated medial or lateral malleolar fractures* are usually stable and do well with conservative measures. Provide analgesia and crutches, and immobilize in a well-padded BKPOP cast. Advise limb elevation, and arrange orthopaedic follow-up. Note that an isolated 'high' lateral malleolus fracture may only be apparent on the lateral X-ray and may be associated with deltoid (medial) ligament injury with instability—some require ORIF.
- *Displaced fractures of the medial or lateral malleolus* require ORIF. Give analgesia and, as appropriate, IV sedation to allow reduction of talar shift. Immobilize the limb in a BKPOP slab, and refer to the orthopaedic team.
- *Bimalleolar or trimalleolar fractures* are unstable. Having attempted to reduce any significant talar shift (with appropriate sedation), place in a BKPOP, obtain fresh X-rays, and refer to the orthopaedic team.

## Ankle dislocation

Dislocation of the ankle is a true emergency. Treat promptly on diagnosis. Examination shows gross deformity of the ankle, severe stretching of the skin (resulting in fracture blisters, skin necrosis, or even converting the injury to a compound fracture), and often deficits in peripheral pulses or sensation. The ankle can dislocate in the absence of associated fractures, but this is uncommon.

*Treatment* Prompt closed reduction and immobilization in POP usually have to precede X-ray (unless available immediately). 'Prompt treatment' does not justify reduction without considering analgesia or sedation.

- Give Penthrox®, IV analgesia/sedation, as needed, with full precautions.
- Warn that there may be a brief ↑ in discomfort as the ankle reduces.
- With the knee flexed and supported, gently grasp the heel with one hand and support the patient's calf with the other.
- Pull smoothly on the heel—it may be necessary to slightly exaggerate the deformity in order to obtain reduction. Success is indicated by return of normal ankle contours, relief of skin tension, and often dramatic relief of pain.
- Once reduced, re-check pulses and sensation; immobilize in a POP slab, and arrange check X-rays.
- Refer the patient to the orthopaedic team immediately.

# Ankle X-rays

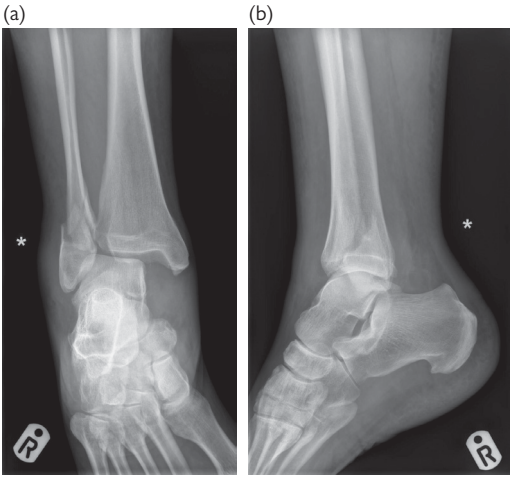


**Fig. 9.63** AP and lateral views of Weber B right lateral malleolus fracture with significant talar shift requiring MUA apparent on AP view.

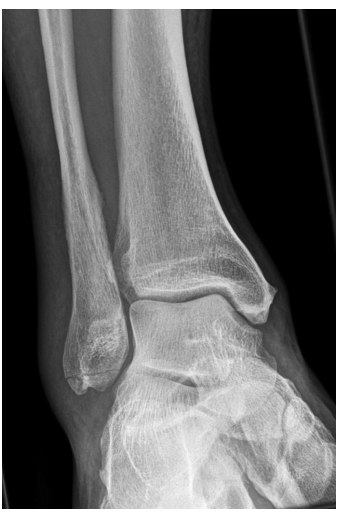

**Fig. 9.64** AP of undisplaced Weber A fracture in a 73y old patient.

# Ankle sprains

Clinical assessment and imaging after ankle injury are outlined in ➲ Approach to ankle injuries, p. 498. The structures most frequently injured in inversion injuries are the lateral joint capsule and the anterior talofibular ligament. ↑ injury causes additional damage to the calcaneofibular ligament and posterior talofibular ligament.

## Treatment

Historically, treatment of sprained ankles has been based upon 'RICE' (rest, ice, compression, elevation), but the scientific basis for all the elements of this is distinctly lacking!

Advise initial rest, elevating the ankle above hip level, and to consider applying ice intermittently during the first 2 days for periods of 10–15min. Begin to weight-bear as soon as symptoms allow, but elevate at all other times. An elastic support from toes to knee is traditional, but of no proven value (and may be harmful by ↑ pain without speeding recovery). If used, ensure that it is not worn in bed. Advise the patient to gently exercise the ankle in all directions and to use simple analgesia regularly until symptoms improve. Most patients with minor sprains can expect full recovery in ~4 weeks. It may be possible to resume sports gradually within 2 weeks, depending on progress.

The inability to weight-bear implies more severe injury. Provide crutches to those completely unable to weight-bear despite analgesia, with advice to elevate the ankle. Arrange review at 2–4 days—if still unable to weight-bear, consider 10 days' immobilization in a boot or below-knee cast, with subsequent outpatient follow-up. Other approaches include use of adhesive strapping or preformed ankle braces. These may be useful in selected cases. Patients can usually expect good functional recovery and should not regard the ankle as 'weak'. Long-term problems (eg weakness/instability whilst walking over rough ground) are often related to ↓ ankle proprioception following immobilization, so aim to mobilize as soon as possible.

## Long-term complications

Do not regard ankle sprains simply as trivial injuries—patients may suffer long-term morbidity (which often causes them to return to the ED):

- *Instability* often manifests itself by recurrent ankle sprains. Refer to physiotherapy (to include isometric exercises).
- *Peroneal tendon subluxation* reflects a torn peroneal retinaculum, allowing the peroneal tendons to slip anteriorly. The clinical presentation includes clicking and a sensation of something slipping. Movement of the foot/ankle (especially eversion) reproduces the subluxation. Refer for orthopaedic follow-up—surgery is an option.
- *Peroneal nerve injury* is relatively common, but not frequently sought for. Neurapraxia results from stretching of branches of the peroneal nerve at the time of injury, with subsequent ↓ sensation over part of the dorsum of the foot and ↓ proprioception at the ankle joint (reflecting injury to the articular branches).

# Venous thromboembolism prevention

Patients who are not fully weight-bearing and are immobilized in a cast or boot may be at risk of developing a DVT, so consider the need for providing prophylaxis in the form of LMWH. Most departments employ a venous thromboembolism (VTE) risk assessment tool to guide staff. An example scoring system is shown in Table 9.6 (adapted from Derriford Hospital).

**Table 9.6** An example VTE risk assessment score for adults not fully weight-bearing in cast or boot

| Risk factor | Score |
|---|---|
| Overweight (BMI >30kg/m$^2$) | 2 |
| Achilles tendon rupture or repair | 3 |
| Previous DVT or PE | 3 |
| Pregnant or within 6 weeks of delivery | 3 |
| Complex surgery of lower leg or pelvic surgery in past 6 weeks | 3 |
| Active cancer | 3 |
| History of DVT or PE in first-degree relative | 2 |
| Unable to walk before the injury | 2 |
| Age over 60y | 1 |
| Abdominal surgery within the last 6 weeks | 1 |
| Gross varicose veins | 1 |
| Taking oral contraceptive pill or hormone replacement therapy | 1 |
| Inflammatory bowel disease (Crohn's or ulcerative colitis) | 1 |
| TOTAL SCORE generated by adding individual scores | |

### Recommendation using the above scoring system

- Score 0–2 = no prophylaxis.
- Score 3 or more = LMWH daily until cast/boot removed and fully weight-bearing (unless contraindications—see ➲ Contraindications to LMWH, p. 503).

### Typical VTE prophylaxis regimes

- If estimated glomerular filtration rate (eGFR) is >30mL/min—dalteparin 5000U SC od.
- If eGFR is <30mL/min—enoxaparin 20mg SC od.

### Contraindications to LMWH

- Concurrent use of oral anticoagulation (eg warfarin, rivaroxaban).
- Acquired or inherited bleeding disorder (eg haemophilia).
- Thrombocytopenia (platelet count <75 × 10$^9$).
- Uncontrolled hypertension (>230/120mmHg).
- Active bleeding from any source.

# Foot fractures and dislocations

Crushing or other violent injuries to the foot can result in significant long-term disability. Delayed or inadequate treatment results in high rates of post-traumatic OA. Compartment syndromes (see ➲ Crush syndrome, pp. 406–7) or vascular injuries may occur. Amputations or severe mangling injuries of the foot are rarely suitable for reconstruction/re-implantation due to poor long-term functional results.

## Talar injuries

Falls onto the feet or violent dorsiflexion of the ankle (eg against car pedals in a crash) can result in fractures to the anterior body or articular dome of the talus. Displaced fractures and dislocations frequently result in avascular necrosis.

*Treat* with analgesia and immobilization in a backslab POP, and refer promptly for orthopaedic treatment (may require MUA and/or ORIF). Dislocations of the talus require prompt reduction under GA.

## Upper/midfoot dislocations

These injuries follow violent twisting, inverting, or everting injuries of the foot. *Peritalar/subtalar dislocations* involve the articulation between the talus and the calcaneum. Give adequate analgesia, and refer to orthopaedics for prompt reduction under GA. *Mid-tarsal dislocations* involve the mid-tarsal joint (comprising the calcaneum and talus posteriorly and the navicular and cuboid anteriorly) and are treated similarly. *Isolated dislocation* of the talus is rare and requires prompt reduction under GA.

## Calcaneal fracture

Calcaneal fractures most often follow a fall from height directly onto the heels. Always exclude associated injuries of the cervical and lumbar spine, pelvis, hips, or knees. Examine for swelling, bruising, and tenderness over the calcaneum, particularly over the sides. Examine both calcanei for comparison, remembering that fractures are commonly bilateral. Examine the Achilles tendon for injury (see ➲ Calf and Achilles tendon injuries, pp. 496–7). Request specific calcaneal X-rays and scrutinize carefully breaks in the cortices, trabeculae, or subtle signs of compression (reduction in Bohler's angle—see Fig. 9.65). Refer all fractures to orthopaedic staff. The majority will require admission for elevation, analgesia, and, in selected cases, ORIF following CT scanning.

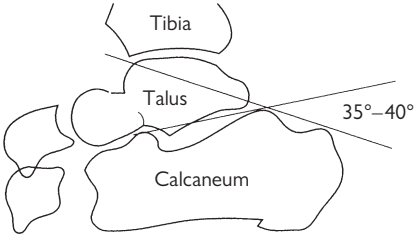


**Fig. 9.65** Bohler's angle (normally 35–45°).

### Clinically suspected calcaneal fracture, but X-rays normal

Sometimes, clinical suspicion of a calcaneal fracture is not confirmed by initial X-rays. Either arrange CT or treat clinically with analgesia, rest, elevation, and crutches, and arrange review at 7–10 days when consideration can be given to further imaging if symptoms persist.

### Metatarsal fractures and dislocations

Multiple MT fractures may follow heavy objects falling onto the feet or, more commonly, after being run over by a vehicle tyre or wheel. In all such cases, consider the possibility of tarso-metatarsal (Lisfranc) dislocation (see Fig. 9.66). This can be easily missed on standard foot X-rays, which do not usually include a true lateral view—look to check that the medial side of the second MT is correctly aligned with the medial side of the middle cuneiform. Check for the dorsalis pedis pulse. Support in a backslab POP, and refer if there are multiple, displaced, or dislocated MT fractures.

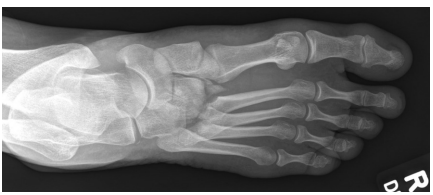

**Fig. 9.66** X-ray of Lisfranc fracture–dislocation, with significant widening between the first and second MTs and fractures at the base of the second MT.

### Isolated avulsion fractures of the fifth MT base

Inversion may avulse the base of the fifth MT by the peroneus brevis. Always examine this area in ankle injuries, and request foot X-rays if tender. Do not mistake accessory bones or the epiphysis (which runs parallel, not transverse, to the fifth MT base). Give analgesia, elevation, and support in a padded crepe bandage or, temporarily, in a boot if symptoms are severe. Discharge with advice or fracture clinic follow-up, according to local policy.

### Jones fracture (of the fifth MT)

This is a transverse fracture of the fifth MT just distal to the inter-MT joint. It is a significant fracture, as it is prone to non-union. Treat with analgesia, crutches, BKPOP or boot, and orthopaedic follow-up.

### Stress fractures of MTs

These typically follow prolonged or unusual exercise ('march fracture') but often occur without an obvious cause. The most common site is the second MT shaft. Examine for swelling over the forefoot (there may be none) and localized tenderness over the MT shaft. X-rays are usually normal initially—callus or periosteal reaction is seen at ~2–3 weeks.

Treat symptomatically with analgesia, elevation, rest, and modified daily activity, as required. Suggest a padded insole. Firm shoes or boots may be more comfortable than flexible trainers. Expect full recovery in 6–8 weeks. If unable to weight-bear, consider a brief period in a BKPOP or boot.

# Toe injuries

*Most toe injuries do not require X-ray.*
The treatment of isolated closed fractures of the toe phalanges without clinical deformity or other complicating factors is not altered by X-rays.
X-ray the following:
- Obvious deformity, gross swelling, or suspected dislocation.
- Suspected compound injuries.
- If any tenderness over the MT head or metatarsophalangeal joint (MTPJ).
- Suspected FB.

## Toe fractures

Treat uncomplicated phalangeal fractures with simple analgesia, elevation, and support with padded buddy strapping. Advise the patient to resume normal activities as soon as possible, but explain that some discomfort may be present for up to 4–6 weeks. Hospital follow-up is not normally required. Manipulate displaced fractures under LA digital block (as described for fingers in ➲ Digital nerve block, pp. 304–5). Angulated toe phalangeal fractures can be difficult to manipulate—a useful trick is to use a pen (or needle holder) placed between the toes as a fulcrum. Once satisfactorily reduced, buddy strap and confirm the position with X-rays.

## Dislocated toes

Untreated, toe dislocations may cause troublesome, persistent symptoms. Reduce promptly under LA digital block and splint by buddy strapping. Always confirm reduction by X-ray, and discharge with analgesia and advice on elevation and gradual mobilization.

## Compound toe injuries

Careful wound toilet, debridement, and repair are essential to ensure rapid healing and avoid infective complications. Ensure that there is adequate tetanus prophylaxis. Always clean wounds thoroughly under adequate anaesthesia (usually LA digital block); provide antibiotics and analgesia. Advise the patient to elevate the injured foot, and arrange follow-up according to local practice. More severe injuries will require exploration and repair under GA. Refer these cases to the orthopaedic team.

## Mangled or amputated toes

Functional results of attempted re-implantation of amputated toes or repair of badly mangled toes are often poor. Provide analgesia, and refer to the orthopaedic surgeon for wound management and amputation of unsalvageable toes.

# Soft tissue foot problems

## Puncture wounds to the foot

- *'Simple' puncture wounds*: see ➲ Puncture wounds, p. 413.
- *Weever fish injuries*: see ➲ Specific bites and stings, pp. 422–3.

## FBs embedded in the foot

Searching for small FBs in the sole of the foot has been likened to searching for a needle in a haystack. Follow the principles set out in ➲ Further assessment of skin wounds, p. 412. Nerve blocks (see ➲ Nerve blocks at the ankle, pp. 314–15) can be useful to allow exploration of foot wounds.

## Morton's metatarsalgia

A burning discomfort radiating to the toes may result from an interdigital nerve neuroma at the level of the MT heads. The nerve between the second and third MT heads is frequently affected. There is localized tenderness, which is also reproduced on compression of the MT heads together. Advise simple analgesia and GP follow-up to consider referral to a foot surgeon.

## Plantar fasciitis

Plantar fasciitis can occur spontaneously or as a chronic overuse injury. Inflammation develops in the plantar fascia, typically at its calcaneal insertion. This results in gradually ↑ burning pain in the sole of the foot and heel, which is worse on weight-bearing. Examine for localized tenderness over the calcaneal insertion of the plantar fascia and heel pad. X-ray may reveal a calcaneal spur, but this is not a useful diagnostic feature.

Advise NSAID, rest, and elevation for 1–2 days, with GP follow-up. A padded shoe insole or sorbothane heel pad may help. Severe, persistent cases are occasionally treated with local steroid injection or even surgical division of the plantar fascia.

## Osteochondritis dissecans

Osteochondritis of an MT head (usually the second—Freiberg's disease) causes gradual-onset pain on weight-bearing. The cause is often unclear, but it may follow minor injury. Examination may reveal local tenderness, but little else. X-ray for evidence of flattening, widening, or fragmentation of the MT head or narrowing of the MTPJ. (See ➲ Osteochondritis, pp. 730–1.)

Treat initially with simple analgesia. Refer persistent cases to orthopaedics to consider excision of the MT head or osteotomy.

## Ingrowing toenails

Refer back to the GP for elective treatment, unless there is evidence of infection. In this case, consider oral antibiotics (eg flucloxacillin) or, if there is acute paronychia, incision and drainage under LA. It is not usually appropriate to excise a wedge of nail under LA in the ED.

# Atraumatic low back pain

Low back pain is the most common cause of lost work days in the UK. The initial ED approach is to identify any patients who may have immediately life-threatening problems (eg leaking aortic aneurysm) and to sort the rest into:

- *Simple ('mechanical') back pain*: no investigations or referral required.
- *Nerve root pain*: referral and investigation needed if symptoms persistent or progressive.
- *Possible serious spinal pathology*: referral and investigation required.
- *Suspected cord compression*: immediate neurosurgical/orthopaedic referral mandatory.

Psychogenic back pain is not an ED diagnosis. If in doubt, refer.

## History

- *General*: note the onset and duration of symptoms, character, position and radiation of pain, and exacerbating or relieving factors. Precipitants include injuries, falls, heavy lifting, or unaccustomed activity.
- *Past history*: detail any previous back problems or surgery and other medical conditions (eg RA, OA, osteoporosis).
- *Drug history*: is the patient using analgesia (and has it helped?)? Ask about corticosteroids and contraindications to NSAIDs.
- *Social history*: ask about home circumstances, work, and stress.
- *Systemic enquiry*: weakness, altered sensation, weight loss, anorexia, fever, rigors, cough, sputum, haemoptysis, and bowel or urinary symptoms.

## Examination

*'Unwell' patient* Assess 'ABC', and look for shock, a pulsatile abdominal mass, peritonism, radial–femoral pulse discrepancies, or asymmetry.

*'Well' patient* Look for signs of weight loss, cachexia, anaemia, clubbing, or muscle wasting. Inspect the back for muscle spasm, scars, scoliosis, or other deformity. If possible, watch the patient walk, looking for spasm, abnormal posture, or limping. Palpate for tenderness over the spine, lower ribs, and renal angles. With the patient supine on a trolley, look for muscle wasting in the legs. Examine both sides:

- *Straight leg raise*: note the angle which reproduces pain (lumbar nerve root irritation).
- *Crossed straight leg raise*: nerve root symptoms reproduced by lifting the contralateral leg strongly suggests lumbar disc prolapse and nerve root entrapment.

*Perform a neurological examination* Check tone, power, sensation, and reflexes in the lower limbs:

- L4 covers sensation of the medial lower leg, quadriceps power, and knee jerk.
- L5 covers sensation of the lateral lower leg and great toe, extensor hallucis longus power, and hamstrings jerk.
- S1 covers sensation of the little toe and lateral foot, foot plantar flexor power, and ankle jerk. Always check perineal and peri-anal sensation.

Perform a rectal examination for anal tone, masses, or blood. Examine the abdomen for masses. Document peripheral pulses and perfusion.

## Investigations

Check T° and urinalysis. X-ray is indicated for some patients aged >55y or those who are systemically unwell with a history of trauma (except clinical coccyx fracture) or where malignancy, infection, or HIV is suspected. In the latter cases, also check CRP, FBC, and U&E.

*MRI scan for suspected cauda equina syndrome*

Request an urgent MRI if there is any suspicion of cauda equina syndrome due to a central lumbar disc prolapse:

- Pain radiating down both legs.
- Difficulty passing urine or urinary incontinence.
- Weakness and/or numbness in both legs.
- Altered perineal sensation/reduced anal tone.
- Loss of sensation of rectal fullness.
- New impotence or priapism.

## Treatment

*Refer urgently* patients with lower limb weakness, altered perineal or perianal sensation, and sphincter disturbance (this is strongly suggestive of *cauda equina syndrome* due to central lumbar disc prolapse). An MRI scan will confirm this diagnosis—in which case, urgent consultation with a neurosurgeon will allow emergency surgical decompression to be arranged as appropriate.

*Refer patients* with the following: age <20 or >55y, unremitting or ↑ symptoms, widespread neurological signs, weight loss, systemic illness, pyrexia, chronic corticosteroids, osteoporosis, or HIV +ve patients with thoracic pain.

*Treat simple 'mechanical' back pain* with regular simple analgesia and/or NSAID, and plan to discharge the patient. Avoid routine use of opioids. Small doses of benzodiazepines (eg diazepam 2–5mg tds) may be useful but tend to cause drowsiness. Advise the patient to aim to return to normal activity, even if some discomfort persists. Avoid bed rest. Expect recovery in 4–6 weeks. Nerve root symptoms mostly resolve over weeks to months with the above treatment, physiotherapy, or manipulation. In all cases, give written and verbal advice for immediate return if limb weakness, numbness, bladder, or bowel problems occur. Advise follow-up with the GP.

Occasionally, patients require admission to get pain under control, benefitting from input from the pain team and occupational therapist.

# Acute arthritis: 1

## Approach

Whenever a patient presents with a painful joint, try to distinguish whether the source of pain is articular or peri-articular. Painful joints of articular origin produce warmth, tenderness, and swelling about the entire joint, with painful movement in all directions. Pain of peri-articular origin (outside the joint capsule), such as bursitis/tendinitis, tends to result in tenderness and swelling localized to a small area, with pain on passive movement only felt in limited planes.

Consider a septic cause in every patient who presents with acute arthritis. Useful investigations include WBC, CRP (or ESR), and joint aspiration.

## Joint aspiration

The most important diagnostic test in patients presenting with acute arthritis is examination of the synovial fluid. When joint aspiration is performed, ensure that an aseptic technique is employed. Avoid joint aspiration through an area of cellulitis. Send fluid for Gram staining, culture, crystal examination, and cell count (see Table 9.7). Remember that the absence of bacteria on Gram staining does not exclude septic arthritis.

**Table 9.7** Joint aspirate findings

| | Normal | Reactive | Infectious |
|---|---|---|---|
| Colour | Colourless/pale yellow | Yellow | Yellow |
| Turbidity | Clear, slightly turbid | Turbid | Turbid, purulent |
| Cell count/mm | 200–1000 | 3000–10,000 | >10,000 |
| Predominant cell type | Mononuclear | Neutrophil | Neutrophil |
| Gram stain | None | None | +ve |
| Culture | –ve | –ve | +ve |

## Causes of polyarthritis

- RA.
- Ankylosing spondylitis.
- Reactive arthritis.
- Psoriatic arthritis.
- Arthritis associated with inflammatory bowel disease.
- Viral arthritis.
- Rheumatic fever.
- Gonococcal arthritis.
- Gout.

## Septic arthritis

Pyogenic infection usually reaches a joint via the bloodstream but may also develop from adjacent osteomyelitis or external skin puncture wounds. Sepsis may progress to complete joint destruction within 24hr.

*Infective agents* *Staphylococcus aureus, Gonococcus, Streptococcus*, TB, *Salmonella*. *Haemophilus* was the most common organism in babies before *Haemophilus* immunization but is now rare. There is an ↑ incidence in patients with RA, those taking steroids, the immunosuppressed, and those at the extremes of age. Do not overlook septic arthritis superimposed on a non-infectious joint (eg gout, rheumatoid joints).

*Presentation* Typically only one joint is affected and is red, painful, and swollen. No movement is usually tolerated (but steroids and analgesics can mask many of the common features of septic arthritis). The joint is held in the position of most comfort, usually in slight flexion. There may be fever, shaking, and rigors. Note that hip joint infection may not produce obvious external findings due to its deep location. Do not overlook a septic joint with signs obscured by concomitant antibiotic use. IV drug users may have involvement of uncommon joints of the axial skeleton (eg sacroiliac, vertebral, and sterno-clavicular joints).

*Investigations* FBC, CRP (or ESR), blood cultures, and joint aspiration (see Table 9.7). X-rays may be initially normal or show only soft tissue swelling, with displacement of capsular fat planes. Later, features of bone destruction occur.

*Treatment* Commence IV antibiotics (eg flucloxacillin + gentamicin), according to local guidelines. Refer urgently to the orthopaedic team for joint irrigation/drainage, analgesia, and splintage of the joint.

*Note: prosthetic joint infection* can be difficult to detect, but pain is typically constant and present at rest. Early infection (within 3 months of surgery) may cause obvious wound inflammation. This is less likely to be apparent in delayed or later infections. There may be little in the way of systemic symptoms. Suggestive radiological features include widening and lucency of the bone–cement interface by >2mm, movement of the prosthesis, periosteal reaction, and fractures through the cement, although X-rays may be normal. Adopt a low threshold for suspecting prosthetic joint infections and referral to the orthopaedic team.

## Traumatic arthritis

Joint pain, tenderness, ↓ range of movement, and haemarthrosis after injury imply intra-articular fracture. Note, however, that septic arthritis may occur in association with trauma, even in the absence of penetrating injury.

## Osteoarthritis

Elderly patients with known OA may suffer acute 'flare-ups'. Constitutional symptoms are not a feature. X-rays may show asymmetrical joint space narrowing, osteophytes, and subchondral cyst formation. Advise NSAID and/or paracetamol, plus graduated exercises.

# Acute arthritis: 2

## Acute gout

Most often affects the first MTPJ or knee. Precipitated by trauma, diet, diuretics, renal failure, myeloproliferative disease, and cytotoxics. Ask about previous renal stones. Look for tophi. Joint aspiration reveals negatively birefringent crystals. Septic arthritis can occur with gout—ensure aspirates are Gram-stained and cultured. X-rays may show punched-out lesions in periarticular bone. Serum uric acid may be ↑ but can be normal. Advise rest and NSAID (eg diclofenac 75mg bd), or if NSAIDs are contraindicated, consider colchicine (500mcg bd initially, slowly ↑ to qds, as needed, for symptoms, with GP review). Do not alter the treatment of patients already on long-term gout therapy. Oral steroids (eg prednisolone 30mg od for 5 days) may help those who are unable to tolerate NSAIDs or are resistant to other treatments.

## Acute pseudogout

Typically affects the knees, wrists, or hips of an elderly person, with arthritic attacks precipitated by illness, surgery, or trauma. Associated with: hyperparathyroidism, haemochromatosis, Wilson's disease, hypothyroidism, diabetes, and hypophosphataemia. X-ray shows calcification in joints, menisci, tendons, ligaments, and bursae. Aspiration reveals weakly +ve birefringent crystals on polarizing microscopy. Treat symptomatically with NSAID.

## Rheumatoid arthritis

*Presentation* Persistent symmetrical, deforming peripheral arthropathy typically starts with swollen, painful, stiff hands and feet, which gradually get worse, with larger joints becoming involved. Other modes of presentation are: persistent or relapsing monoarthritis of different large joints, systemic illness with minimal joint problems, sudden-onset widespread arthritis, and vague limb girdle aches.

*Hand signs* Include MCPJ and PIPJ swelling, ulnar deviation and volar subluxation at MCPJs, and boutonnière and 'swan neck' finger deformities. Extensor tendon rupture may occur.

*Neck problems* Degeneration of the transverse ligament of the dens carries the risk of subluxation and cord damage.

*Extra-articular features* Subcutaneous nodules, vasculitis, pulmonary fibrosis, splenomegaly, anaemia, pleurisy, pericarditis, scleritis, kerato-conjunctivitis.

*Rheumatoid factor* +ve in 70% of cases.

*X-rays* Show soft tissue swelling, peri-articular osteoporosis, joint space narrowing, and bony erosions/subluxation.

*Treatment* Refer patients who are systemically unwell. Others may benefit from NSAID, splintage, and rheumatology clinic referral.

## Viral arthritis

Rubella, hepatitis B, mumps, EBV, and enteroviruses may cause arthritis. In hepatitis B, arthritis usually affects PIPJs, MCPJs, or the knees and precedes the onset of jaundice. Rubella is associated with acute symmetrical arthritis and tenosynovitis.

**Rheumatic fever** (See ➔ Skin lesions in multisystem disease, pp. 688–9.)

This is a non-infectious immune disease which follows infection with group A β-haemolytic streptococci. Typically, migratory or additive symmetrical polyarthritis affects the knees, ankles, elbows, and wrists.

*Diagnosis* Based on revised Jones criteria: evidence of previous streptococcal infection (ie recent scarlet fever, +ve throat swab, or anti-streptolysin titre >200U/mL) plus two *major* or one *major* plus two *minor* criteria.

*Major criteria* Carditis (pericarditis, myocarditis, or endocarditis), migratory polyarthritis, chorea, subcutaneous nodules, rash (erythema marginatum).

*Minor criteria* ↑ ESR/CRP, arthralgia, fever, history of previous rheumatic fever (or rheumatic heart disease), ↑ PR interval on ECG.

*Investigations* Throat swab, ESR, CRP, and anti-streptolysin titre.

*Treatment* Refer for admission, rest, aspirin, benzylpenicillin, and splintage.

## Sero-negative spondyloarthropathies

These have the following common features: involvement of the spine and sacroiliac joints; inflammation, then calcification, of bony tendon insertions; peripheral inflammatory arthropathy; and extra-articular manifestations such as uveitis, aortic regurgitation, and pulmonary fibrosis.

*Ankylosing spondylitis* Usually presents with chronic low back pain in men aged 15–30y. Progressive spinal fusion ultimately results in a fixed kyphotic spine (which is particularly prone to fracture after injury), hyperflexed neck, and restricted respiration. Hips, shoulders, and knees may be involved. Other features are: iritis, apical lung fibrosis, plantar fasciitis, and Achilles tendonitis. There may be normochromic anaemia and ↑ CRP. X-rays show 'bamboo spine' (squared vertebrae), eroded apophyseal joints, and obliterated sacroiliac joints.

*Reactive arthritis* A triad of urethritis, conjunctivitis, and sero-negative arthritis may follow infection (urethritis, cervicitis, or dysentery). It may cause large joint monoarthritis of a weight-bearing leg joint. Other features include: iritis, keratoderma blenorrhagicum, circinate balanitis, plantar fasciitis, Achilles tendonitis, and aortic incompetence. Joint aspirate yields inflammatory cells, with –ve culture. WCC and CRP are ↑ .

*Psoriatic arthritis* Arthritis rarely precedes skin involvement.

*Enteropathic arthropathies* Inflammatory bowel disease is associated with spondyloarthritis and large joint mono-arthropathy. There may also be migratory polyarthritis.

*Gonococcal arthritis* May present with fever, migratory tenosynovitis and polyarthralgia, arthritis (knee, ankle, or wrist), and skin rash. Genital infection may be silent, especially in women. Take swabs with special culture media, and refer for investigation.

# Eponymous fractures

Correctly applied, the one or two words that comprise an eponymous injury convey succinctly an otherwise involved description of a complex fracture.

### Aviator's astragalus

Fractures of the neck of the talus, previously commonly observed amongst World War II pilots who crash-landed their damaged planes on returning from bombing raids. The injuries resulted from the upward thrust of the rudder bar, causing dorsiflexion forcing the talus against the anterior tibia.

### Bankart lesion

Avulsion of the joint capsule and glenoid labrum, resulting from anterior dislocation of the shoulder joint. It is implicated as a causative factor for recurrent dislocations.

### Barton's fracture

First described by Barton in 1839, this complex distal radial fracture is intra-articular. Displacement of the distal radial fragment allows subluxation of the carpal bones. A rare variety is called a Lentenneur's fracture.

### Bennett's fracture–dislocation

These intra-articular fractures of the base of the first MC are notorious for allowing the main MC fragment to slip into a poor position. If conservative treatment (POP) is preferred to internal fixation, careful follow-up will be needed to ensure a satisfactory outcome.

### Boutonnière deformity

Rupture of the central slip of the extensor tendon at the PIPJ allows the base of the middle phalanx to 'button-hole' through. The remaining two parts of the extensor expansion slip along the side of the finger and act as flexors at the PIPJ, whilst still extending the DIPJ. This produces the characteristic deformity.

### Boxer's fracture

Fracture of the neck of the little finger MC rarely occurs during formal boxing when gloves are worn. It is much more commonly seen following impromptu street or bar-room brawls—innocuous-looking overlying wounds are often compound human ('reverse fight') bites (see ➲ Specific bites and stings, pp. 422–3).

### Bumper fracture

The height of the average car bumper renders the adult pedestrian (who is unfortunate enough to be knocked down) particularly vulnerable to a fracture through the lateral tibial condyle into the tibial plateau. There is often an associated tear to the medial collateral knee ligament.

### Chance fracture

A horizontal fracture through a vertebral body, arch, and spinous process may follow a distraction and flexion injury. It typically involves the lumbar spine of car passengers restrained only by a lap belt in a crash.

### Clay-shoveller's fracture

Resistance against neck flexion may produce an avulsion of the tip of a spinous process of the lower cervical or upper thoracic spine. The lesion typically affects C7.

### Colles' fracture

Abraham Colles, Professor of Surgery in Dublin, described this common distal radial fracture in 1814. The classic dinner fork deformity results from posterior displacement and angulation of the distal fragment (see ➲ Colles' fracture, pp. 454–5).

### Dashboard dislocation

A high-speed head-on road traffic collision causing the dashboard to impact upon the flexed knee often results in posterior dislocation of the hip.

### Dupuytren's fracture–dislocation

A highly unstable ankle injury in which there is a fracture of the distal fibula shaft and disruption of the medial ankle ligament and posterior tibiofibular ligament. The result is gross diastasis and dislocation of the talus laterally.

### Duverney fracture

This is an isolated fracture of the pelvis, involving one iliac wing. Unlike a number of other fractures affecting the pelvis, it is usually a relatively stable injury.

### Essex–Lopresti fracture–dislocation

A heavy fall on an outstretched hand may cause a comminuted fracture of the radial head. It is associated with tearing of the interosseous membrane (diastasis), allowing subluxation of the distal ulna.

### Galeazzi fracture–dislocation

Describes the combination of a fracture of the distal radial shaft with dislocation of the distal radio-ulnar joint (see ➲ Forearm fractures and related injury, pp. 460–1). A Moore's fracture–dislocation is a similar injury, except that the radial fracture involves the distal radius, not the shaft.

### Gamekeeper's thumb

Rupture of the ulnar collateral ligament of the first MCPJ was originally described as an occupational injury amongst gamekeepers, sustained whilst breaking the necks of wounded rabbits. It is now most commonly seen as a result of skiing injuries, particularly on artificial slopes, when the thumb is caught in the diamond latticework matting. The injury requires prompt diagnosis and treatment in order to avoid the long-term complication of a weak pinch grip.

### Hangman's fracture

Although no longer a part of modern life in the UK, executions were previously achieved by hanging. The victim was allowed to fall several feet before being arrested by a noose. This produced rapid death by severing the cervical spinal cord. The mechanism of injury is a combination of distraction and extension, causing an unstable (hangman's) fracture of the pedicles of the axis (C2) and disrupting the intervertebral disc between C2 and C3. The fracture may also result from extension and axial compression and may occur without neurological damage.

### Hill–Sachs lesion

This is an impacted compression fracture of the humeral head, which occurs during anterior shoulder dislocation. It is produced by the recoil impaction of the humeral head against the rim of the glenoid as the former dislocates. It is believed by some to be an important causative factor for recurrent dislocation.

### Horse rider's knee

Frontal impact at the level of the proximal tibiofibular joint may result in posterior dislocation of the fibular head. Reduction usually requires an MUA.

### Hume fracture–dislocation

This refers to the combination of an olecranon fracture with dislocation of the radial head.

### Hutchinson fracture

Also known as a 'chauffeur' fracture, this name is sometimes given to a radial styloid fracture. It is classically caused by forced radial deviation of the wrist when the starting handle of an old-fashioned motor car 'kicks back'.

### Ice skater's fracture

Children aged 2–8y are susceptible to distal fibula stress fractures.

### Jefferson fracture

An unstable 'blow-out' fracture of C1 follows an axial load. One-third are associated with a C2 fracture.

### Jones fracture

This is a transverse fracture of the base of the fifth MT, just distal to the inter-MT joint. It is a more significant injury than an avulsion fracture at the insertion of the peroneus brevis, as it is prone to non-union (see ➲ Foot fractures and dislocations, pp. 504–5).

### Le Fort facial fractures

Experiments by Le Fort in 1901 were followed by descriptions of facial fractures and classification into three anatomical types (see ➲ Middle third facial fractures, pp. 380–1), including the Guérin fracture (Le Fort I).

### Lisfranc fracture–dislocation

Fracture–dislocation at the tarso-metatarsal joint is a significant injury. It is named after the surgeon who described the surgical operation of partial amputation of the foot at the level of the tarso-metatarsal joint.

### Luxatio erecta

First described in 1859, this is an uncommon shoulder dislocation (inferior glenohumeral dislocation). The term is derived from Latin and describes the erect hyperabducted position of the arm after dislocation. The injury follows a hyperabduction force, most often after a fall. Axillary nerve damage occurs in 60%. Reduction of the dislocation may follow overhead traction or conversion to an anterior dislocation to which conventional techniques can be applied.

### Maisonneuve injury

An unstable injury in which rupture of the medial ankle ligament is associated with diastasis and proximal fibula fracture.

### Malgaigne's fracture

An unstable injury in which the pelvic ring is disrupted in two places: anteriorly (through both pubic rami) and posteriorly (sacroiliac joint disruption or fracture of the ilium or sacrum).

### Mallet injury

Stubbing a finger may rupture the extensor tendon (or avulse its phalangeal attachment) at the DIPJ, causing a 'mallet deformity', in which the DIPJ is held flexed. The mechanism of injury is forced flexion of the extended DIPJ.

### March fracture

This refers to a stress fracture of the (usually second) MT shaft after strenuous and unaccustomed exercise. Traditionally, it was observed after heavy marching in new army recruits.

### Monteggia fracture–dislocation

Fracture of the proximal ulnar shaft is associated with dislocation of the radial head. The latter is relatively easy to miss. Never accept an ulnar fracture as an isolated injury without obtaining complete views of both forearm bones, including the elbow and wrist joints.

### Nursemaid's elbow

Alternative name for a 'pulled elbow' in a preschool child (see ➲ Subluxation of the radial head ('pulled elbow'), p. 750).

### Nutcracker fracture

Lateral force applied to the forefoot may cause the cuboid to be fractured, as it is compressed between the calcaneum and the base of the fourth and fifth MTs.

### O'Donahue's triad

A torn medial meniscus, ruptured anterior cruciate ligament, and ruptured medial collateral ligament combine to produce a significant knee injury.

### Pelligrini–Stieda's disease

Ossification of the medial collateral knee ligament may follow avulsion of the superficial part from its attachment to the medial femoral condyle.

### Pilon fracture

These intra-articular fractures of the distal tibia are uncommon but may also be subdivided into three types.

### Pipkin fracture–dislocation

This refers to a posterior hip dislocation in which part of the femoral head is avulsed by the ligamentum teres and remains attached to it within the acetabulum. The avulsed fragment is rarely large enough to be reattached.

### Pott's fracture

This term has come to be applied indiscriminately to any ankle fracture, which may be simply subdivided into 'uni-', 'bi-', or 'trimalleolar'.

### Rolando fracture

Essentially a comminuted Bennett's fracture, the classic description is of Y-shaped intra-articular fractures at the base of the first MC. Treatment is difficult.

### Runner's fracture

Stress fractures of the tibia are particularly common amongst runners who chalk up many miles of running on roads each week.

### Smith's fracture

The so-called 'reversed Colles' fracture' was first described by Smith in 1847.

### Straddle fracture

Falls astride classically produce bilateral vertical pubic rami fractures.

### Tillaux fracture

An avulsion fracture of the distal lateral tibia may occur due to the pull of the anterior tibiofibular ligament.

### Toddler's fracture

Undisplaced spiral fractures of the tibial shaft in children <7y often follow minimal trauma and may not be visible on initial X-ray. Subperiosteal bone formation is usually apparent radiologically by 2 weeks (see ➲ Toddler's fracture, p. 754).

# Chapter 10

# Surgery

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# Approach to abdominal pain

First identify patients requiring resuscitation or urgent treatment. The need for resuscitation is apparent in emergencies with associated hypovolaemic and/or septic shock. Less obvious, but equally important, is recognition of patients requiring urgent treatment with no clinical evidence of shock (especially ruptured abdominal aortic aneurysm).

## History

*The pain* Determine details of site, severity, radiation, shift, character, timing, precipitating and relieving factors.

*Vomiting* Record anorexia, nausea, and vomiting. Ask about the nature of vomit (blood, bile, etc.). Vomiting that follows the onset of abdominal pain tends to imply a surgical cause, whereas vomiting preceding pain is often non-surgical.

*Bowel disturbance* Enquire about recent change of bowel habit, particularly any rectal bleeding.

*Other symptoms* Do not forget that abdominal pain may be due to urological, respiratory, cardiovascular, or gynaecological disorders.

*Past history* Determine the nature of previous surgery, preferably by obtaining old notes.

## Examination

*Vital signs* Pulse, BP, RR, $SpO_2$, GCS, and T° may indicate the need for immediate intervention.

*Abdomen* Note distension and old scars. Check hernial orifices. Palpate gently for areas of tenderness. It is unnecessary and unkind to attempt to elicit rebound tenderness—tenderness on percussion is ample evidence of peritonitis. Perform PR/per vaginam (PV) examination only if necessary.

*General* Look for evidence of dehydration and jaundice. Examine the respiratory and cardiovascular systems.

## Investigations

Assessment of patients with abdominal pain in the ED usually depends upon history and examination, rather than sophisticated tests. However, the following investigations may prove useful:

- *BMG*: DKA may present with abdominal pain (see ➲ Hyperglycaemic crises, pp. 160–1).
- *Urinalysis*: abdominal pain may result from urinary stones or infection. Perform a urine pregnancy test on all women of child-bearing age.
- *Blood tests*: consider FBC, U&E, amylase, LFTs, CRP, coagulation screen, and cross-matching. If clearly unwell, check ABG (or venous gas), and lactate. Although FBC is frequently requested in patients with abdominal pain, the awaited WCC rarely alters initial management.
- *ECG*: especially in patients aged >55y who may be suffering from an atypical presentation of an acute medical problem, most notably acute MI.
- *Erect CXR*: can help to exclude chest pathology which may mimic abdominal conditions (eg congestive heart failure, basal pneumonia). It may also reveal free gas under the diaphragm.

- *Abdominal X-ray*: specific indications for abdominal X-ray include suspicion of intestinal obstruction, toxic megacolon, sigmoid volvulus, and GI perforation. X-rays are not indicated in patients with suspected uncomplicated appendicitis, UTI, 'simple' constipation, gastroenteritis, GI bleeding, and acute pancreatitis. They are not 'routinely indicated' in the investigation of abdominal pain. In severely ill patients requiring imaging, CT or USS is usually more appropriate than plain abdominal X-rays.
- *USS*: reveals gallstones, free peritoneal fluid, urinary stones, and aortic aneurysms. It is increasingly used in the ED but needs specific training.
- *CT scan:* may have a role in assisting with the diagnosis of certain conditions (eg acute appendicitis in less straightforward cases).

## Treatment

Prompt resuscitation and provision of analgesia are integral components of the management of serious abdominal conditions. Ensure that patients who are very sick and/or hypotensive receive early IV fluids (caution if aneurysm suspected) and full monitoring (including measuring urinary output via a urinary catheter). Follow the guidelines outlined in ➲ Sepsis, pp. 62–3.

The traditional belief that analgesia should not be given because it might mask a serious diagnosis is incorrect and cruel. Diagnosis is often easier when pain is relieved and the patient can give a better history and co-operate with examination. The most appropriate form of analgesia is usually IV opioid (eg morphine) ± IV paracetamol.

It can be difficult to decide if admission is needed for a patient with abdominal pain. Adopt a low threshold for seeking senior help. In general, if doubt exists, refer to the surgeon, who may decide that it is prudent to admit the patient for observation and investigation.

## Pitfalls

- Steroids, NSAIDs, or obesity may render physical signs less obvious.
- In elderly patients with peritoneal inflammation, a lack of abdominal musculature may make it difficult to elicit guarding.
- β-blockade may mask signs of shock.
- Absence of fever does not exclude infection, especially in the very old, the very ill, and the immunosuppressed.
- When severe abdominal pain is out of all proportion to the physical findings, consider mesenteric infarction, aortic rupture/dissection, acute pancreatitis, and torsion of an ovarian cyst.
- Splenic rupture may occur after relatively trivial trauma in patients with glandular fever or haematological disorders.
- Consider gynaecological causes of abdominal pain in any woman of child-bearing age—always perform a pregnancy test.
- WCC may be normal in established peritonitis/sepsis.
- Amylase may be normal in acute pancreatitis. Conversely, moderate amylase ↑ may occur in acute cholecystitis, perforated peptic ulcer, and mesenteric infarction.

# Causes of acute abdominal pain

There is a wide range of causes of abdominal pain. The cause is often initially unclear. Remember that a patient is much more likely to have a common condition (perhaps with an atypical presentation), rather than a very rare condition. Thus, a patient presenting with atypical abdominal pain is more likely to have acute appendicitis than tabes dorsalis, lead poisoning, or acute intermittent porphyria. A number of conditions are seen relatively frequently (see Table 10.1).

**Table 10.1** Causes of acute abdominal pain

| Surgical | Gynaecological | Medical |
|---|---|---|
| • Non-specific abdominal pain<br>• Acute appendicitis<br>• Cholecystitis/biliary colic<br>• Pancreatitis<br>• Peptic ulcer disease<br>• Ruptured aortic aneurysm<br>• Mesenteric infarction<br>• Diverticulitis<br>• Large bowel perforation<br>• Intestinal obstruction<br>• Ureteric calculi<br>• Urinary retention<br>• Testicular torsion<br>• Intussusception<br>• Cancer (especially of the colon) | • Ectopic pregnancy<br>• Pelvic inflammatory disease<br>• Rupture/torsion of ovarian cyst<br>• Endometriosis<br>• Mittelschmerz | • MI<br>• Pneumonia<br>• PE<br>• Aortic dissection<br>• Acute hepatitis<br>• DKA<br>• UTI<br>• Herpes zoster<br>• Irritable bowel syndrome<br>• Gastroenteritis<br>• Inflammatory bowel disease |

## No cause for pain found

Many patients get better without any definite cause being identified ('non-specific' abdominal pain). If symptoms have improved and there are no worrying signs, it may be reasonable to consider discharging some patients, especially if blood tests and urinalysis are normal. Obtain senior review of patients aged >70y before discharge—there is a relatively high incidence of important missed pathology (eg ruptured aortic aneurysm—see ➲ Ruptured abdominal aortic aneurysm, pp. 536–7).

When discharging a patient, provide appropriate advice about when to return if symptoms recur/worsen.

## Cancer causing abdominal pain

Unexplained abdominal pain in patients >50y may be caused by cancer, especially of the large bowel. The pain may result from transient or partial bowel obstruction. Ask about previous episodes of pain, weight loss, and change of bowel habit. If there is no indication for admission, consider referral to a surgical clinic for investigation or contact the GP to suggest this.

# Acute appendicitis

This common cause of abdominal pain in all ages is particularly difficult to diagnose in the extremes of age and in pregnancy. However, the diagnosis of acute appendicitis is often missed initially at all ages.

## History

The classic presentation is of central colicky abdominal pain, followed by vomiting, then shift of the pain to the right iliac fossa. Many presentations are atypical, with a variety of other symptoms (eg altered bowel habit, urinary frequency), partly depending upon the position of the tip of the inflamed appendix (retrocaecal 74%; pelvic 21%; paracaecal 2%; other 3%).

## Examination

In the early stages, there may be little abnormal; in the late stages, the patient may be moribund with septic shock and generalised peritonitis. Between these extremes, there may be a variety of findings, including ↑ T°, tachycardia, distress, and fetor oris. There is usually a degree of tenderness in the right iliac fossa (± peritonitis). Rovsing's sign (pain felt in the right iliac fossa on pressing over the left iliac fossa) may be present. PR examination may reveal tenderness high up on the right, with inflammation of a pelvic appendix.

## Investigations

The diagnosis of acute appendicitis is essentially clinical. Scoring systems (eg Alvarado score) have been developed but cannot be relied upon. X-rays are not helpful, but do perform urinalysis ± pregnancy test. WCC (and CRP) may be ↑, but this is not invariable. CT (or USS) may help the surgeon to make the diagnosis in certain circumstances.

## Differential diagnosis

Depending upon the presentation, the potential differential diagnosis is very wide—consider urinary, chest, and gynaecological causes.

## Treatment

Despite studies describing non-operative treatment of acute appenditicis with antibiotics, surgical intervention remains standard treatment.

- Obtain IV access and resuscitate as necessary. Commence IV fluids (eg 1000mL of 0.9% saline) if there is evidence of dehydration.
- Give IV opioid and antiemetic (eg slow IV metoclopramide 10mg).
- If acute appendicitis is likely, or even possible, keep 'nil by mouth' and refer to the surgeon. If appendicectomy is required, preoperative antibiotics (eg amoxicillin + gentamicin + metronidazole) ↓ infective complications.
- If a diagnosis of acute appendicitis seems very unlikely and the patient is going to be discharged (± after surgical review), ensure that (s)he is advised to return for review (or seek medical attention) if symptoms worsen.

## Appendix mass

Untreated, acute appendicitis may proceed to perforation, with generalized peritonitis, or may become 'walled off' to produce a localized right iliac fossa inflammatory mass. There are many other causes of such a mass, including: caecal carcinoma, Crohn's disease, ovarian mass, pelvic kidney, ileocaecal TB, psoas abscess, actinomycosis, and Spigelian hernia. Refer to the surgeon for further investigation and management.

# Acute pancreatitis

This is a relatively common serious cause of abdominal pain in the middle-aged and elderly, with an incidence of ~5 per 100,000/y.

## Causes

Most are due to gallstones or alcohol. Many are idiopathic. Other causes include: hypothermia, trauma, infection (glandular fever, mumps, Coxsackie, and infectious hepatitis), hyperlipidaemia, hyperparathyroidism, drugs (steroids, azathioprine, thiazides, and statins), PAN, pancreatic cancer, post-endoscopic retrograde cholangiopancreatography (ERCP), and scorpion stings.

## Symptoms

Typically, the complaint is of severe constant epigastric pain radiating to the centre of the back (possibly relieved by leaning forward), with associated nausea and vomiting.

## Signs

The patient may be distressed, sweating, and mildly pyrexial. Look for evidence of shock—there may be a need for urgent resuscitation. Abdominal tenderness is likely to be maximal in the epigastrium ± guarding. The oft-quoted, but uncommon, bluish discoloration in the loins (Grey–Turner's sign) only develops after several days.

## Investigations

- Check BMG and $SpO_2$.
- Serum amylase is likely to be grossly ↑ to >4 times upper limit of normal range (but if not diagnostically ↑, consider urinary amylase level). Note that mild ↑ amylase may occur in a wide range of other acute abdominal conditions.
- Serum lipase may be measured, instead of amylase.
- FBC may reveal ↑ WCC.
- U&E, $Ca^{2+}$, LFTs, glucose—hypocalcaemia is relatively common.
- Coagulation screen.
- CXR, ECG, ABG, including lactate.

## Treatment

- Provide $O_2$.
- Obtain IV access and resuscitate with IV crystalloid fluids as necessary.
- Give IV analgesia (eg morphine titrated according to response—see Approach to abdominal pain, pp. 520–1).
- Give an antiemetic (eg cyclizine 50mg or metoclopramide 10mg slow IV).
- Insert a NG tube.
- Insert a urinary catheter and monitor urine output.
- Consider early involvement of the critical care team and possible insertion of a central venous line to monitor the CVP and guide IV fluid therapy in the seriously ill, particularly the elderly.
- Contact the appropriate specialist(s) and transfer to HDU/ICU.

*Complications of acute pancreatitis*

Acute pancreatitis has significant mortality. Early complications include ARF, DIC, hypocalcaemia, and ARDS. Later, pancreatic abscess or pseudo-cyst may occur.

*Prognosis of acute pancreatitis*

The risk of death may be predicted according to the number of prognostic indicators present (Glasgow Imrie scoring system).

*Three or more* of the following on admission and subsequent repeat tests over 48hr constitute severe disease:

- Partial pressure of $O_2$ ($pO_2$) <7.9kPa.
- Age >55y.
- Neutrophils ↑ (WCC >15 × $10^9$/L).
- Corrected $Ca^{2+}$ <2mmol/L.
- Raised blood urea >16mmol/L.
- Elevated enzymes (serum LDH >600U/L; AST >100U/L).
- Albumin <32g/L.
- Sugar ↑: fasting glucose >10mmol/L.

## Chronic pancreatitis

The term chronic pancreatitis implies permanent pancreatic damage. The condition most often results from alcohol excess. Some patients with chronic pancreatitis present frequently to the ED requesting opioid analgesia (and, increasingly, cyclizine too). This can pose a difficult problem for the doctor who has not treated them previously. Patients with chronic pancreatitis can experience severe episodes of acute pancreatitis. Follow the approach outlined below and review previous hospital notes/letters early to help guide treatment. For patients with alcohol-related chronic pancreatitis, consider the need for IV thiamine (eg Pabrinex®) and diazepam or chlordiazepoxide (see ➲ Alcohol withdrawal, p. 640).

*Admission or discharge?*

Many patients with chronic pancreatitis can be successfully managed on an outpatient basis without admission to hospital. Refer for admission (usually under the medical team) patients with dehydration/shock, severe illness, uncontrolled pain, or vomiting.

# Biliary tract problems

Most emergency biliary tract problems relate to gallstones. Both solitary cholesterol and multiple mixed gallstones are common amongst the middle-aged and the elderly. Pigment stones comprise a small proportion—they occur in hereditary spherocytosis, malaria, and haemolytic anaemia.

## Complications of gallstones

Acute/chronic cholecystitis, biliary colic, obstructive jaundice, Mirizzi's syndrome, ascending cholangitis, mucocele, empyema, acute pancreatitis, gallstone ileus, gall bladder cancer.

## Acute cholecystitis

*History* Impaction of gallstones with acute inflammation of the gall bladder usually manifests itself by right hypochondrial pain radiating to the right side of the back ± vomiting.

*Examination* Look for features of an acute inflammatory process. Fever is frequently present, combined with right hypochondrial tenderness (particularly felt on inspiration—Murphy's sign). There may be a palpable mass—this is also a feature of mucocele and empyema (the latter causing high fever, extreme tenderness, and septic shock).

*Management*

- Provide IV analgesia and antiemetic (see ➲ Analgesics: morphine, p. 286).
- Check FBC (WCC often ↑), U&E, glucose, amylase, and LFTs.
- CXR, ECG (in case pain is due to an atypical presentation of MI).
- USS will confirm the diagnosis (tenderness on pressing the USS transducer over the area where the thickened gall bladder containing stones is located is called the ultrasonic Murphy's sign).
- Commence antibiotics (eg cefotaxime 1g IV) and refer to the surgeon.

## Biliary colic/chronic cholecystitis

Patients (sometimes with known gallstones) may present with short-lived recurrent episodes of epigastric/right hypochondrial pain ± radiation to the back. This pain of biliary colic/chronic cholecystitis may be difficult to distinguish from other causes, including peptic ulcer disease. If the pain has subsided and there are no residual abnormal physical signs, discharge the patient with arrangements for GP or surgical outpatient follow-up.

## Common bile duct stones

Stones in the common bile duct can cause problems, including acute pancreatitis (see ➲ Acute pancreatitis, pp. 524–5), obstructive jaundice, and ascending infection.

*Obstructive jaundice* Biliary obstruction results in ↑ jaundice with pale stools and dark urine (± pain). Acute hepatitis and cholangio-/pancreatic carcinoma may present in a similar fashion. A palpable gall bladder implicates pancreatic carcinoma as the more likely diagnosis (Courvoisier's law: 'In the presence of jaundice, if the gall bladder is palpable, the cause is unlikely to be a stone').

*Ascending cholangitis* Biliary stasis predisposes to infection, characterized by Charcot's triad (abdominal pain, jaundice, and fever). The patient may be very ill and require resuscitation for septic shock (see ➲ Shock, pp. 64–5) and combination IV antibiotics (eg amoxicillin, gentamicin, and metronidazole).

# Peptic ulcer disease

## Perforated peptic ulcer

*History* Perforation of a gastric or duodenal ulcer is usually a severely painful sudden event. It may occur in those without known peptic ulcer disease, although close questioning may reveal recent symptoms attributed to 'indigestion'. Sudden localized epigastric pain spreads to the remainder of the abdomen—the pain is worse on coughing or moving and may radiate to the shoulder tip.

*Examination* Although distressed, patients often prefer to lie still, rather than roll about. However, some patients in extreme pain writhe or roll in agony and are unable to keep still for examination or X-rays until analgesia is given. Absent bowel sounds, shock, generalized peritonitis, and fever develop as time passes.

*Investigations* An erect CXR will demonstrate free gas under the diaphragm in ~75% of patients with perforated peptic ulceration. In those cases where the diagnosis is suspected, but not proven by X-ray, consider a contrast CT scan.

Other relevant investigations are: U&E, glucose, amylase (may be slightly ↑), FBC (WCC typically ↑), $SpO_2$, ABG, and ECG/troponin (ensure symptoms do not reflect MI, rather than peptic ulcer disease).

*Treatment*

- Give $O_2$.
- Provide IV analgesia (eg morphine titrated according to response).
- Give an antiemetic (eg slow IV metoclopramide 10mg).
- Resuscitate with IV 0.9% saline.
- Refer to the surgeon and give IV antibiotics (eg cefotaxime 1g and, in late presentations, metronidazole 500mg as well).

## Other GI perforations

Perforations may affect any part of the GI tract, but the chief causes are peptic ulceration, trauma, diverticular disease, and colonic carcinoma. The emergency treatment principles are similar to those of perforated peptic ulcer (described in ➲ Perforated peptic ulcer, p. 527). Bowel perforation usually results in gas under the diaphragm on an erect CXR, but remember that there are other possible causes, including: recent surgery, peritoneal dialysis, gas-forming infections, and occasionally vaginal gas insufflation during waterskiing or oral sex.

## Other presentations of peptic ulcer disease

Peptic ulcer disease may also present with upper GI haemorrhage (see ➲ Upper gastrointestinal bleeding, pp. 126–7) or pain from oesophagitis, gastritis, or duodenitis. If the presentation suggests inflammation of the upper GI tract and there is no evidence of serious complications, consider discharging the patient with an antacid and GP follow-up. It is not usually appropriate to initiate therapy with PPIs in the ED without an accurate diagnosis. *Note*: some patients require an urgent endoscopy arranged by the GP to exclude cancer (eg older patients with chronic pain, weight loss, and anaemia).

# Mechanical intestinal obstruction

Intestinal obstruction may be *mechanical* or *paralytic* in nature. Causes of mechanical intestinal obstruction are shown in Table 10.2.

**Table 10.2** Causes of mechanical intestinal obstruction

- Adhesions (most common)
- Obstructed hernia (commonly: inguinal, femoral, para-umbilical, incisional; rarely: obturator, Spigelian, lumbar)
- Tumours (gastric, caecal, or most commonly sigmoid)
- Peptic ulcer disease
- Pelvic kidney
- Enlarged gall bladder
- Intussusception (see ➲ Intussusception, p. 721)
- Volvulus (gastric, caecal, or most commonly sigmoid colon—see ➲ Large bowel emergencies, pp. 532–3)
- Inflammatory mass (eg diverticular, Crohn's)
- Gallstone ileus
- Common iliac artery aneurysm

## History

Classic symptoms of intestinal obstruction are: abdominal pain, distension, vomiting, and constipation. The exact presentation depends upon the site of obstruction and the underlying cause. Ask about previous surgery. A history of severe pain suggests strangulation and developing ischaemia in a closed loop. The nature of the vomit (eg faeculent) may give a clue to the site of obstruction.

## Examination

Check T°. Look for evidence of dehydration or shock. Carefully examine the hernial orifices (an obstructed femoral hernia is otherwise easily missed). Inspect for scars from old surgery. Note any distension and areas of tenderness (peritonism implies the surgical problem is advanced). Bowel sounds may be tinkling or absent. PR examination may reveal an 'empty' rectum.

*Blood tests* U&E, glucose, amylase, FBC, LFTs, clotting, group and save.

*X-rays* Request CXR and supine abdominal X-rays. If there is no convincing evidence of obstruction on the supine view, but still a high index of clinical suspicion, consider requesting a CT scan. X-rays may demonstrate distended loops of bowel (with multiple fluid levels visible on an erect abdominal view). The site and nature of distended bowel loops may suggest the site of obstruction—small bowel obstruction results in dilated loops of bowel with valvulae conniventes completely traversing the lumen, whereas large bowel haustra do not completely cross the lumen.

Note that although gallstone ileus is rare, X-rays may be diagnostic—the fistula between the bowel and the gall bladder allows gas into the biliary tree, which shows up as an abnormal Y-shaped gas shadow in the right hypochondrium (see Fig. 10.1).

*ECG* Obtain this if the patient is middle-aged or elderly.

*ABG* If the patient is shocked, check $SpO_2$, ABG, and lactate.

*Old notes* Review previous hospital case notes/letters.

### Management of mechanical obstruction

- Insert an IV cannula and start IVI of 0.9% saline.
- If the patient is shocked, resuscitate with $O_2$ and IV fluids and insert a urinary catheter. Consider the need to insert a central venous line to guide resuscitation and involve ICU specialists at an early stage.
- Provide analgesia (eg IV morphine titrated according to response—see ➲ Analgesics: morphine, p. 286).
- Give an antiemetic (eg cyclizine 50mg).
- Insert an NG tube.
- Refer to the surgical team for ongoing care.

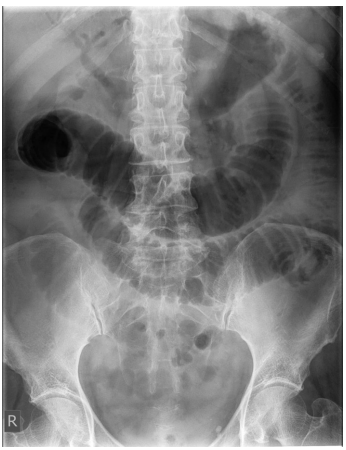


**Fig. 10.1** Gallstone ileus. Note the combination of small bowel obstruction with gas in the biliary tree (appearing as a prominent Y-shaped shadow in the right upper abdomen).

## Paralytic intestinal obstruction

This is relatively rare in the ED. Causes include postoperative ileus, electrolyte disturbance (eg hypokalaemia), and pseudo-obstruction.

### Intestinal pseudo-obstruction

This condition results from chronic impairment of GI motility. Many of the patients affected are elderly and taking tricyclic antidepressants or other drugs with anticholinergic actions. Although pseudo-obstruction may involve any part of the GI tract, it typically presents with colonic distension. On rare occasions, this may be sufficiently severe to rupture the caecum or cause hypotension by compressing the inferior vena cava and blocking venous return. There may be a diagnostic X-ray appearance showing gas in the bowel all the way to the rectum, whereas in a classical, more proximal obstruction, gas will be absent from the rectum. Treatment of acute colonic distension from pseudo-obstruction is by decompression using a colonoscope.

# Mesenteric ischaemia/infarction

## Acute mesenteric infarction

Abrupt cessation of the blood supply to a large portion of gut results in irreversible gangrene of the bowel in a relatively short space of time. This is associated with high mortality. Unfortunately, the diagnosis can be difficult to make—the challenge therefore lies with making it early.

### *Pathophysiology*

One or more of the following processes may be responsible:

- Mesenteric arterial embolism (often associated with AF).
- Mesenteric arterial thrombosis.
- ↓ mesenteric arterial blood flow (eg hypotension secondary to MI).
- Mesenteric venous thrombosis.

Most cases involve either arterial embolism or thrombosis.

### *History*

Acute mesenteric infarction usually occurs in middle-aged or elderly patients. It is often heralded by severe, sudden-onset, diffuse abdominal pain. Typically, the severity of the pain initially far exceeds the associated physical signs. The pain may radiate to the back.

Some patients have a preceding history of chronic mesenteric ischaemia, with pain after meals and weight loss. There is often an associated history of vascular disease elsewhere (eg intermittent claudication).

### *Examination*

Shock, absent bowel sounds, abdominal distension, and tenderness are late signs. Initially, there may be little more than diffuse mild abdominal tenderness. If the diagnosis is suspected, search carefully for evidence of an embolic source (eg AF, recent MI with a high risk of mural thrombus, aortic valve disease or valve prosthesis, recent cardiac catheter).

### *Investigations*

- U&E, BMG, and laboratory blood glucose.
- Amylase may be moderately ↑.
- FBC may demonstrate ↑ WCC.
- Coagulation screen.
- Group and save.
- ABG typically reveals severe metabolic acidosis, and lactate may be ↑.
- X-rays may show non-specific dilatation of bowel loops and, in advanced cases, gas within the hepatic portal venous system.
- ECG may demonstrate AF.
- CT angiography may reveal the exact nature of the underlying problem and help to guide treatment.

### *Management*

If the diagnosis is suspected:

- Resuscitate with $O_2$ and IV fluids.
- Provide analgesia (eg IV morphine titrated according to response).
- Consider broad-spectrum IV antibiotics.
- Refer urgently to the surgeon to consider heparin/revascularization.

### Ischaemic colitis

Chronic arterial insufficiency to the bowel usually affects the mucosa and submucosa, typically in the region of the splenic flexure (the junction of the territory supplied by the superior and inferior mesenteric arteries).

The patient presents with abdominal pain, starting in the left iliac fossa. Loose stools with blood may be passed. The patient may have had previous similar episodes and exhibit evidence of cardiovascular disease. Examination may reveal low-grade pyrexia, tachycardia, and colonic tenderness, with blood PR.

Check FBC, U&E, group and save, ECG, and CXR. Plain abdominal X-rays may show 'thumb-printing' (submucosal colonic oedema), typically at the splenic flexure (see Fig. 10.2). Provide analgesia and IV fluids, and refer to the surgical team.

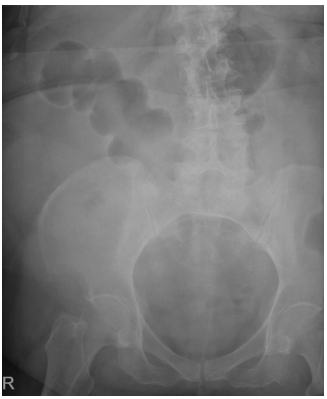


**Fig. 10.2** Abdominal X-ray showing dilated transverse colon with mural oedema.

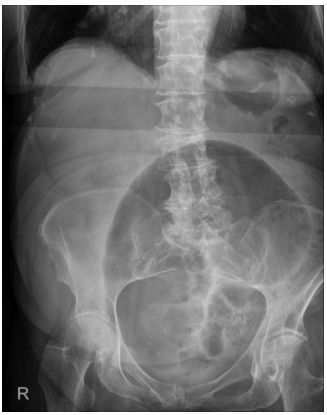


**Fig. 10.3** Abdominal X-ray showing sigmoid volvulus.

# Large bowel emergencies

## Sigmoid volvulus

Usually occurs in the elderly with initially intermittent cramping lower abdominal pain and progressive abdominal distension, which may be spontaneously relieved by passage of large amounts of flatus/faeces. Some patients progress to complete obstruction—marked distension progressing to fever and peritonitis suggests strangulation.

*Plain abdominal X-ray* typically shows a large single dilated loop of colon (a 'bent inner tube') on the left side, with both ends down in the pelvis (see Fig. 10.3).

*Refer* to the inpatient surgical team for sigmoidoscopy/insertion of a flatus tube (if not strangulated) or surgery (if strangulated).

## Caecal volvulus

Most common between the ages of 25 and 35y. Patients have symptoms of acute-onset small bowel obstruction.

*Plain abdominal films* usually show one large dilated segment of the colon in the mid-abdomen with distended small bowel loops and empty distal large bowel. Refer to the surgical team.

## Acute diverticulitis

Diverticulosis is common in the middle-aged and elderly, particularly affecting the sigmoid colon. Without significant complications, there may be a change in bowel habit with passage of mucus.

Acute diverticulitis results from inflammation ± perforation of a diverticulum and may be confined to the colonic wall by the serosa. If this perforates, then inflammation may remain localized (pericolic abscess) or spread (frank peritonitis). Symptoms and signs reflect the extent of the infection—there may be lower abdominal dull constant pain and low-grade fever, with tenderness, rigidity, and occasionally a mass in the left lower quadrant. The elderly (the group most at risk of diverticulitis and its complications) and those on immunosuppressants may not manifest the expected pyrexia and signs of peritonitis.

*Investigations* Check FBC, U&E, CRP, group and save, and blood cultures. Plain abdominal X-rays may show non-specific changes and help to exclude perforation/large bowel obstruction. An erect CXR often shows copious subdiaphragmatic gas in free perforations. CT is the best investigation to delineate the extent of the problem and associated complications.

*Treatment* Give analgesia and IV fluids; keep fasted, and refer to the surgeon. Start broad-spectrum antibiotics (eg cefuroxime + metronidazole).

*Complications*

- Perforation: may be localized and walled off (forming an abscess) or generalized.
- Intestinal obstruction: both large and small (due to adherent loops).
- Massive PR bleeding.
- Fistulae to adjacent structures: small bowel, uterus, vagina, bladder.
- Post-infective strictures.

### Ulcerative colitis

Severe acute colitis is characterized by the passage of >6 loose bloody motions per day, together with systemic signs (tachycardia, fever) and hypoalbuminaemia. There is a risk of haemorrhage, perforation, and toxic megacolon.

Resuscitate with IV fluids and give IV hydrocortisone (100–200mg), then refer to the inpatient gastroenterology service for aggressive medical therapy (IV and PR steroids, IV fluids) and joint review by medical and surgical teams. Surgery may be required for complications, especially toxic megacolon.

Suspect toxic megacolon if the colonic width is >5.5cm on abdominal X-ray (this sign is associated with a 75% risk of requiring colectomy).

Refer any patient who presents with suspected new-onset ulcerative colitis for investigation and control of the disease.

### Crohn's disease

Colonic Crohn's disease may present as colitis with bloody diarrhoea, urgency, and frequency, similar to ulcerative colitis. Fibrosis may cause diarrhoea or obstructive symptoms.

Peri-anal disease with chronic anal fissure may be the first presenting symptom. Emergency surgery is indicated in acute fulminating Crohn's colitis with bleeding, toxic dilatation, or perforation.

### Epiploic appendagitis

Primary inflammation of one of the hundreds of appendices epiploicae on the antimesenteric colonic border may present in a similar fashion to acute diverticulitis or appendicitis. However, T° and WCC are usually normal. Although often diagnosed at laparotomy, CT scan may be characteristic, allowing conservative treatment (including IV analgesia).

### Irritable bowel syndrome

Patients are usually aged 20–40y with a prolonged history of intermittent symptoms—altered bowel function (diarrhoea, constipation, or diarrhoea alternating with constipation). Typically, the abdominal pain is crampy/aching and localized in the lower abdomen over the sigmoid colon. The patient may report that the pain is eased by the passage of stool or flatus. Examination fails to reveal any worrying features. The diagnosis is one of exclusion—be vigilant for clues that may point to other organic disease.

### Mesenteric ischaemia/infarction

(See ➲ Mesenteric ischaemia/infarction, pp. 530–1.)

# Anorectal problems

Any PR bleeding requires surgical follow-up to exclude malignancy.

## Complications of haemorrhoids ('piles')

- *Bleeding*: haemorrhoids typically cause painless, bright red PR bleeding associated with defecation, but blood is not mixed with the stools. Check the abdomen and inspect the anus—if there is no prolapsed or external haemorrhoid, perform PR and arrange GP/surgical follow-up.
- *Prolapsed internal haemorrhoids (piles)* are acutely painful—treat conservatively with adequate analgesia (may need admission), bed rest, and stool softeners.
- *Thrombosed external pile* is due to rupture of a tributary of the inferior haemorrhoidal vein, producing a peri-anal haematoma. One or more dark blue nodules covered with squamous epithelium may be visible at the anus and a clot palpable. Refer to the surgeon to decide between incision and drainage under LA or conservative management.

## Anal fissure

Typically causes severe pain on defecation and for 1–2hr afterwards. There may be blood on the toilet paper, but usually bleeding is minimal. The fissure is located just inside the anal orifice and is usually associated with the passage of hard stools. Most are located posteriorly in the midline. PR examination may be impossible due to pain, but the fissure is often visible with traction of anal skin.

*Treatment* Prescribe analgesia and stool softeners. Most heal spontaneously, but the presence of significant ulceration, hypertrophied tissue, or a skin tag suggests chronicity and the need for surgical follow-up. Be suspicious of those fissures not in the midline and those that are multiple (the differential diagnosis includes chronic inflammatory bowel disease, anal cancer, and adenocarcinoma of the rectum invading the anal canal).

## Pruritus ani

Not strictly an emergency problem. There are numerous possible causes:

- Poor hygiene.
- Fissure, prolapsing piles, fistulae, rectal prolapse, anal cancer.
- Contact dermatitis due to local applications (especially LAs).
- Threadworms.
- Part of a general condition (eg obstructive jaundice, lymphoma, severe iron deficiency anaemia, uraemia, diabetes).
- Lichen sclerosis.
- STI (herpes, anal warts, HIV).

Treatment requires identification of the underlying problem—refer to the GP. In the meantime, advise avoidance of ointments and creams.

## Pilonidal abscess

An infected pit in the natal cleft causes pain and/or offensive discharge.

*Treatment* Refer to the surgical team. Treatment may involve initial incision and drainage, followed by healing, then elective excision of the sinus.

*Note*: fissures, tears, or bruising around the anus of a child arouse suspicion of abuse. Refer to a specialist and avoid rectal examination.

## Anorectal abscesses

Most begin with infection involving an anal crypt and its gland, from which it can spread between the external and internal sphincters to a variety of sites—these determine its symptomatology and mode of presentation.

Peri-anal and ischiorectal abscesses account for ~80% of cases. In 20%, there is a clear predisposing cause such as inflammatory bowel disease, anorectal cancer, or anal fissure (see Fig. 10.4).

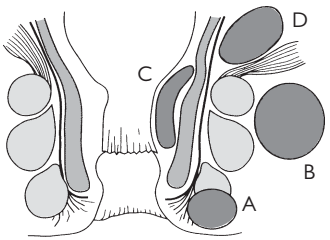


**Fig. 10.4** Four types of anorectal abscesses: (A) peri-anal; (B) ischiorectal; (C) submucous; and (D) pelvi-rectal.

*Clinical features* Pain is a prominent initial feature of peri-anal and superficial ischiorectal abscesses, followed by local signs of inflammation. Patients complain of persistent dull, throbbing pain, made worse by walking and sitting and prior to defecation. Such symptoms are less evident with deep infections, which tend to develop slowly with pyrexia and systemic upset. Peri-anal abscesses produce localized fluctuant red, tender swellings close to the anus. With ischiorectal sepsis, the findings are more diffuse and fluctuance is a late finding. Deeper infections are less obvious—PR examination may reveal a mass or a tender area of induration.

*Treatment* Provide analgesia and refer to the surgical team for incision and drainage under GA.

## Venereal proctitis

The organisms are similar to those transmitted by vaginal intercourse—assume >1 type of organism is present. Patients complain of pain, irritation, discharge, and bleeding. Consider gonococcus, *Chlamydia*, syphilis, and herpes simplex. Refer urgently to a GU specialist.

## Rectal foreign bodies

X-rays may demonstrate the position and shape of FBs. More especially, look for the presence of any free air—perforation of the rectum or colon is the most frequent and most serious complication, in which case give IV antibiotics (eg cephalosporin + metronidazole). Refer the patient for removal of the FB by the surgical team.

## Rectal prolapse

Complete prolapse of all bowel layers occurs particularly in elderly ♀ after excessive straining. If it is not reduced easily by gentle manipulation, refer to the surgical team.

# Ruptured abdominal aortic aneurysm

Rupture of abdominal aortic aneurysm causes a large number of deaths, many of which occur suddenly out of hospital. Even when the patient reaches hospital alive, there is significant mortality. The best chance of survival lies with early diagnosis, prompt resuscitation, and rapid transfer to theatre. Most aneurysms are saccular and occur in the infrarenal aorta—haemorrhage after rupture is usually into the retroperitoneum and may be initially contained locally. Aneurysm extension to involve the renal arteries renders surgery more difficult and ↑ the risk of postoperative complications.

## History

Presentation is highly variable, ranging from PEA cardiac arrest to painless sudden collapse of obscure origin, through to a classical history of central abdominal and lower back pain in a patient with a known aneurysm. Abdominal and/or back pain is usually a feature—typically sudden in onset and severe in nature.

## Examination

The seriously ill patient may present a characteristic picture—distressed, pale, sweating, tachycardic, and hypotensive, with mottled skin of the lower body and a tender, pulsatile abdominal mass. One or both femoral pulses may be absent.

## Diagnosis

Ruptured aortic aneurysm is not infrequently misdiagnosed as ureteric colic. Adopt a low threshold of suspicion in any middle-aged or elderly patient who presents with back pain, abdominal pain, or collapse. In some patients (eg the obese), it may be difficult to be certain about the presence of a pulsatile abdominal mass. In such cases, assume there is a ruptured abdominal aortic aneurysm and commence resuscitative measures, whilst appropriate experts are summoned and relevant emergency confirmatory investigations (eg USS or CT scan—see Fig. 10.5) are performed. It may be safer and quicker to perform USS in the ED, rather than transfer the patient for CT scan.

## Management

- Provide high-flow $O_2$.
- Obtain venous access with two large-bore venous cannulae.
- Send blood for FBC, U&E, glucose, baseline coagulation screen, LFTs, and emergency cross-matching, as per the hospital's massive transfusion protocol.
- Give IV analgesia (eg morphine titrated according to response).
- Provide IV antiemetic (eg cyclizine 50mg).
- Avoid excessive IV fluid resuscitation. Treat major hypovolaemia, but accept moderate degrees of hypotension (systolic BP >90mmHg). In general, patients who are conscious and passing urine require minimal IV fluid therapy until theatre. Ensure O-negative blood is available if needed.
- Obtain a CXR.
- Insert a urinary catheter and a radial arterial line, and record an ECG.
- Call the vascular team early to consider open or endovascular repair.
- Ensure that other relevant staff (eg ICU, emergency theatre staff) are informed.

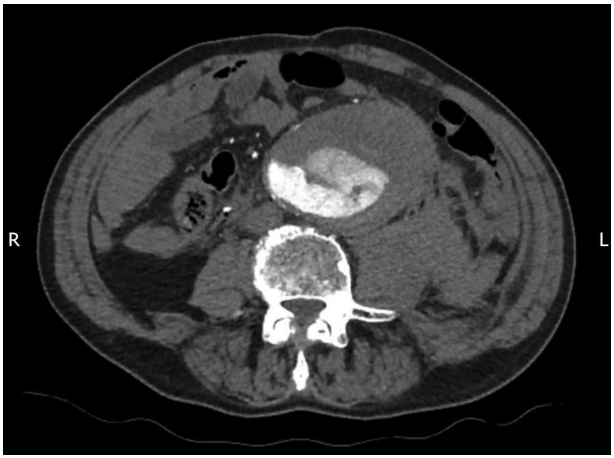


**Fig. 10.5** CT showing a leaking abdominal aortic aneurysm.

Note that in patients with very poor renal function, there may be understandable concern about administering contrast. In this situation, consider a non-contrast CT scan which can still give useful information.

## Pain after previous surgery for aortic aneurysm

If a patient presents with abdominal pain and has previously undergone aortic aneursym repair, consider complications of the surgery.

### *Infection of graft*

This can be difficult to diagnose. It may present with evidence of sepsis and/or anastomotic leak. Take blood tests, including FBC and CRP; obtain a CT scan, and involve the vascular surgeon.

### *Anastomotic leak*

A leaking anastomosis can produce a pseudoaneurysm and/or catastrophic haemorrhage. Pseudoaneurysms may produce a painful pulsatile mass—evaluate by CT, and involve the vascular surgeon.

### *Endoleak*

Patients who have undergone endovascular repair may develop an 'endoleak' whereby blood flows outside the graft (but within the original aneurysm sac). Although many endoleaks may be managed conservatively, there is a risk of external rupture of the aneurysm sac.

### *Fistula*

Aortoenteric and aortocaval fistulae can occur as primary problems associated with an untreated aortic aneurysm. An aortoenteric fistula may also develop after surgical repair, often with massive GI haemorrhage with an underlying associated graft infection.

# Acute limb ischaemia

## Clinical features

The cardinal features of acute limb ischaemia are shown in Table 10.3.

**Table 10.3** Cardinal features of acute limb ischaemia (6 Ps)

| Men | Women |
|---|---|
| • Pain | • Pulselessness |
| • Paraesthesiae (later anaesthesia) | • Paralysis (muscle damage) |
| • Pallor | • Perishing cold |

Where acute arterial occlusion occurs in a previously normal limb, the features of ischaemia will be ↑ because of the absence of a developed collateral circulation. In the absence of a traumatic cause (either direct arterial injury or indirect injury such as compartment syndrome—see ➲ Crush syndrome, pp. 406–7), the most common causes are embolism or thrombosis.

*Embolism* Cardiac sources account for >80% (AF, post-MI, prosthetic valves, atrial myxoma, vegetations, and rheumatic heart disease). Acute embolic events affect legs more often than arms (especially artery bifurcations).

*Risk factors* Diabetes, smoking, hypertension, hypercholesterolaemia.

*Past history* Ask about previous TIA, stroke, and MI.

*Examination* A clear demarcation between normal and ischaemic skin suggests an embolic cause of an acutely ischaemic limb. Look for sources of emboli (irregular pulse, abnormal heart sounds, murmurs, valve clicks). Check all pulses in both the affected and contralateral limbs. The presence of normal pulses in the contralateral limb suggests an embolic cause, whereas absent contralateral pulses makes thrombosis more likely.

*Investigations* ECG, CXR, U&E, CK, FBC, coagulation screen, ABG, urinalysis (for myoglobin), cross-match. Cardiac and/or abdominal USS may be required, and if thrombosis *in situ* is suspected, angiography is indicated.

*Thrombotic* Thrombosis may develop acutely around atheroma. Previous intermittent claudication/vascular impairment is likely. The other limb may also have chronic vascular insufficiency (muscle wasting, hair loss, ulceration).

## Treatment

- Give appropriate pain relief (usually IV opioid).
- Correct hypovolaemia and other causes of low-flow states.
- Revascularization is required within 6hr to avoid permanent muscle necrosis (and subsequent need for amputation) and metabolic effects (such as rhabdomyolysis and renal failure). If the cause is embolic, embolectomy is required. If thrombotic, angiography will define the site and extent of the lesion—thrombolysis ± reconstructive surgery is then undertaken.

# Complications of varicose veins

## Bleeding from varicose veins

Patients with chronic venous hypertension associated with varicose veins have a significant risk of haemorrhage from the dilated thin-walled veins which commonly surround the area of lipodermatosclerosis at the ankle. Haemorrhages may be profuse and sufficient to cause hypovolaemic shock. In extreme cases, this may even cause death.

### *Treatment*

Control bleeding by elevating the leg and applying a non-adherent dressing and pressing firmly. Follow with appropriate bandaging, unless there is evidence of occlusive arterial disease (varicose veins and arterial disease frequently coexist in the elderly). Some patients may require resuscitation with IV fluids.

Refer for admission those who were shocked at presentation, those who have subsequently bled through the bandaging, those with occlusive arterial disease, and those who live alone. All patients require surgical outpatient follow-up—advise patients who are discharged about first-aid measures in the event of a rebleed.

## Superficial thrombophlebitis

This occurs in those with varicose veins or prothrombotic states (eg underlying inflammatory/malignant conditions). It usually produces redness, tenderness, and induration along the course of the involved vein.

### *Treatment*

Bed rest, elevation, and analgesia (NSAID). Pain typically ↓ over 1–2 weeks, and the patient is left with a hard thrombotic cord. Superficial thrombophlebitis is only rarely associated with DVT, but occasionally thrombosis spreads from the long saphenous vein to involve the femoral vein. If there is any question of deep vein involvement, request an USS. If the thrombotic process involves the sapheno-femoral junction or the ilio-femoral system, refer for anticoagulation (see ➲ Patients on anticoagulants, pp. 178–9).

## Venous ulcers

Venous (varicose) ulcers tend to be chronic and recurrent. They typically occur on the medial side of the ankle. There is often associated dermatitis with surrounding brown discoloration, thickening of the skin, and leg oedema. There is often mixed venous and arterial disease, especially in the elderly. Although ischaemic ulcers tend to lie on the lateral aspect of the ankle, exclude ischaemic ulceration by checking the peripheral pulses (request Doppler in patients with oedematous legs). Look for areas suspicious of malignant change, which may rarely occur in chronic ulcers (Marjolin's ulcer).

### *Treatment*

Clean the ulcer with saline, and dress it with either paraffin gauze or colloidal dressing. Follow with firm bandaging (unless there is coexisting arterial disease). Advise leg elevation when resting. Avoid topical antibiotics and indiscriminate use of oral antibiotics. Prescribe oral antibiotics (eg flucloxacillin) only if there is cellulitis. Liaise with the GP about surgical outpatient follow-up and to arrange for redressing by the district nurse.

# Ureteric colic

►► *New-onset flank/back pain in the elderly may represent a leaking aortic aneurysm (even if haematuria is present).*

## Causes

Calculi or blood clots may cause ureteric (or 'renal') colic. Colicky pain is produced by ureteric obstruction, ↑ intraluminal pressure, and muscle spasm. Calculi most commonly consist of calcium oxalate and/or calcium phosphate. Less common are magnesium ammonium phosphate (associated with UTIs and urea-splitting organisms such as *Proteus*), urate, and cystine stones.

*Calculi* are associated with hypercalcaemia, hyperoxaluria, and hyperuricaemia. 'Staghorn' calculi in the collecting system predispose to infections.

Calculi may form throughout the length of the renal tract. They vary in size from tiny particles to large 'stones' in the bladder. They cause symptoms from local obstruction and infection, and rarely they may ulcerate through the wall of the structure in which they are present. Smaller stones (<5mm diameter) are likely to pass spontaneously.

## Clinical features

The most common presenting symptoms are pain from obstruction or UTI and/or haematuria. Constant dull, severe loin discomfort is associated with excruciating colicky pain, spreading to the respective iliac fossa, testis, tip of the penis, or labia. The pain may cause the patient to move, roll, or walk about. Nausea, vomiting, pallor, and sweating are common. There is frequently a previous history of stone disease—ask about this and whether there is any past history of renal disease. Ask about urinary and GI symptoms.

Apart from loin tenderness, abdominal examination is usually normal, but carefully check the haemodynamic status, pulses, bruits, and the abdominal aorta, as a ruptured aortic aneurysm can present in a similar fashion (see ➲ Ruptured abdominal aortic aneurysm, pp. 536–7). Pyrexia or rigors suggest associated infection. Microscopic (sometimes frank) haematuria is very common but not always present. Symptoms are usually relieved when the stone passes into the bladder, but larger calculi may then cause obstruction at the bladder neck or the urethra, producing acute urinary retention. Bladder calculi may present with symptoms of UTI and/or bladder irritation (frequency, dysuria, strangury, and haematuria).

## Investigations

- Urinalysis and MSU: blood on stix testing is present in >80% of patients with proven stones. A pH >7.6 implies an associated infection with urea-splitting organisms.
- U&E, creatinine, glucose, $Ca^{2+}$, $PO_4^{2-}$, urate, amylase.
- FBC—↑ WCC is associated with infection.
- The traditional 'KUB' (kidneys, ureters, bladder) X-ray with subsequent IVU has now been replaced by CT. Plain CT (without contrast) has a high sensitivity and specificity and has the advantage of assisting the diagnosis of other causes of abdominal and/or loin pain. In addition to establishing the size and position of stones, CT will also identify obstruction and hydronephrosis.
- USS has a role in the investigation of pregnant patients with suspected ureteric colic but is much less reliable at identifying smaller stones than CT scan.
- Take specialist advice when considering imaging for patients who present with problems which are related to recurrent/known stones.
- Send any stone which has passed and been collected for laboratory analysis.

## Treatment

See NICE guidance at https://cks.nice.org.uk/renal-or-ureteric-colic-acute

*Analgesia*

- Give diclofenac 75mg IM, repeated after 30min if necessary. If NSAIDs are not appropriate (eg allergy or renal impairment), give IV opioid titrated to effect (eg morphine 5–10mg).
- Consider adding IV paracetamol (1g).
- Provide an antiemetic (eg metoclopramide 10mg IM or IV).

*Admission criteria*

- Shock, fever, or other signs of systemic infection.
- ↑ risk of AKI (eg CKD, solitary kidney).
- Pregnancy.
- Dehydration and inability to take oral fluids due to vomiting.
- Uncertainty about the diagnosis.
- Continuing or early recurrence of severe pain.

*Discharge advice*

Aim to discharge patients (with arrangements for appropriate outpatient investigation) when symptoms have resolved or dramatically improved and in whom CT/IVU shows no obstruction. Provide NSAID analgesia (eg naproxen or diclofenac) plus antiemetic (eg metoclopramide). Advise the patient to maintain a normal oral fluid intake, with the aim of maintaining a normal urine colour. Explain that the stone may pass spontaneously—ideally, ask the patient to sieve the urine (eg using a tea strainer or nylon stocking) and retrieve the stone, so that its content can be analysed in the laboratory. Ask the patient to return to hospital if they develop severe pain, fever, or rigors. Ensure that the GP is informed in a timely fashion, so that further follow-up can be arranged as an outpatient.

# Retention of urine

## Causes of acute urinary retention

The common causes are shown in Table 10.4—men are affected more than women, most usually due to benign prostatic hyperplasia.

**Table 10.4** Common causes of urinary retention

| Men | Women |
|---|---|
| • Prostatic hyperplasia/cancer | • Retroverted gravid uterus |
| • Urethral stricture | • Atrophic urethritis |
| • Postoperative | • MS |

Other causes include: acute urethritis, prostatitis, phimosis, urethral trauma, bladder blood clot, urethral calculus, prolapsed intervertebral disc/spinal cord syndromes, drugs (alcohol, antihistamines, anticholinergics, antihypertensives, tricyclics), faecal impaction, and anal pain.

## Presentation

The diagnosis is usually obvious—the patient complains of inability to pass urine, combined with bladder discomfort. Remember, however, to consider the diagnosis in those patients unable to describe their symptoms (eg those unconscious after trauma).

Examination reveals a tender, enlarged bladder, with dullness to percussion well above the symphysis pubis. Search for the causes listed above. In particular, look for evidence of prolapsed disc/cord compression by checking lower limb power/reflexes and perineal sensation. Perform PR examination to assess anal tone and the prostate.

## Initial management

Provided there is no contraindication (eg trauma or urethral stenosis), decompress the bladder by urethral catheterization. Use an aseptic technique. If urethral catheterization is impossible or contraindicated, consider a suprapubic catheter, but this requires a doctor experienced in the technique.

After bladder drainage, record the volume of urine obtained, then re-examine the abdomen for pathology that might have been previously masked. Test the urine and send an MSU for culture and sensitivity.

Check U&E and FBC. (Prostate-specific antigen is unreliable in retention.)

## Further management

If blood tests are normal and there is no complication (eg bleeding) after urinary catheterization, discharge the middle-aged/elderly man with likely underlying benign prostatic hyperplasia, with plans to return to a Trial WithOut Catheter (TWOC) clinic, typically a week later.

Take specialist advice on starting an α-blocker (eg tamsulosin 400mcg daily), as this may ↑ the chance of successful removal of the catheter, although there are significant side effects and drug interactions.

# Penile problems and prostatitis

## Paraphimosis

Paraphimosis occurs when the foreskin is left retracted, thereby causing swelling of the glans, which results in difficulty replacing the foreskin to its proper position. Untreated, tissue necrosis may develop. Paraphimosis may be iatrogenic, occurring after urethral catheterization.

*Treatment* Initially attempt reduction by manual decompression, which may require the use of Entonox®, IV sedation, or LA (a small amount of topical 1–2% lidocaine gel or injection of 10mL of plain 1% lidocaine around the base of the penis). Digital pressure may allow the glans to ↓ in size, prior to the foreskin being delivered back into its usual position. If unsuccessful, refer to the surgical team for reduction under GA or dorsal slit of the prepuce followed by later circumcision.

## Priapism

Persistent (and usually painful) penile erection has a number of causes:

- Iatrogenic (following intra-cavernosal injection of one or more of: papaverine, alprostadil, vasoactive intestinal polypeptide, and phentolamine for impotence).
- Others: leukaemia, myeloma, sickle-cell disease, spinal injury, drugs [eg sildenafil (Viagra®), phenothiazines, cannabis, cocaine), renal dialysis.

*Management* Priapism is a urological emergency. Refer urgently to the urology team. Initial emergency treatment of a prolonged (>6hr) artificial erection (ie following an intra-cavernosal drug injection or oral sildenafil) is to aspirate 50mL of blood from each corpus cavernosum through a 19G butterfly needle into a 50mL syringe with a Luer lock.

## Urethritis

This usually presents with dysuria/urinary frequency, reflecting underlying STI. Refer for appropriate investigation, treatment, and follow-up.

## Prostatitis

Inflammation of the prostate may be acute or chronic and presents in a variety of ways (fever, urgency, frequency, perineal pain, urethral discharge). PR examination reveals a tender prostate. Urinalysis demonstrates protein. Refer for further investigation and treatment.

## Penile trauma

*Minor superficial tears* Relatively common. Most involve the frenulum. Patients report pain and bleeding following sexual intercourse. Bleeding usually responds to local pressure (if not successful, consider tissue glue or refer to the surgical team). Once bleeding has stopped, advise abstinence from sexual activity for ~10 days to allow healing to occur and prevent recurrence.

*Fracture of the penis* This occurs infrequently. It involves injury to the tunica albuginea of the erect penis. The result is penile tenderness and swelling. Refer to the urologist for urgent surgical exploration, evacuation of haematoma, and repair.

# Testicular problems

Any pain of testicular origin may be initially referred to the abdomen.

## Testicular torsion

Testicular torsion is most frequently seen in children and young adults. Any suspicion of testicular torsion should prompt immediate referral. The condition is covered fully in ➲ Inguinal and scrotal swellings, pp. 722–3.

## Acute epididymitis

*Causes* For those aged <35y, infection with *Chlamydia* or gonococcus is commonly responsible. Acute epididymitis in those aged >35y is usually secondary to UTI and associated with underlying urinary tract pathology.

*Clinical features* There is typically a gradual onset of progressive testicular ache, with subsequent swelling of the epididymis and testis. There may be a history of dysuria or urethral discharge. The patient may be pyrexial. The epididymis is acutely tender, with the testis lying low in the scrotum. Advanced late cases may have progressed to abscess formation.

*Investigations* Send an MSU. Leave taking urethral swabs to the GU clinic.

*Management* The chief initial concern is to ensure that testicular torsion is not being missed—if there is any possibility of this (see ➲ Inguinal and scrotal swellings, pp. 722–3), refer urgently. Treatment of acute epididymitis comprises antibiotics (eg ciprofloxacin for 2 weeks), analgesia, and rest. Some patients require admission; others may be managed on an outpatient basis. Urology investigation and follow-up will be required, so involve the urologist early. For patients with suspected *Chlamydia* or gonococcus infection, refer to the GU clinic for appropriate advice, swabs, treatment, and contact tracing of sexual partners.

## Orchitis

Orchitis may present as epididymo-orchitis, an extension of bacterial epididymitis (see ➲ Acute epididymitis, p. 544). Orchitis of viral origin may also occur—typically mumps, following ~5 days after parotitis. Mumps orchitis may be unilateral or bilateral and can occur in the absence of overt parotitis. Rarely, orchitis is secondary to TB or syphilis.

*Treatment* All patients with orchitis require analgesia and follow-up. If there is any possibility of bacterial infection, antibiotics are indicated (see ➲ Acute epididymitis, p. 544).

## Scrotal/testicular lumps

Causes include: hydrocele, inguinal hernia, epididymal cyst, epididymitis, orchitis, and testicular tumour. Many patients require referral back to the GP or clinic. Be wary of an apparent epididymo-orchitis which failed to respond to antibiotics—it could be an atypical presenting testicular tumour.

## Testicular trauma

See ➲ Scrotal, and testicular trauma, p. 361.

# Cellulitis and erysipelas

## Cellulitis

(See ➲ Infected wounds and cellulitis, p. 419.)

Cellulitis reflects bacterial skin infection (usually streptococcal, occasionally staphylococcal). It can occur in association with a skin wound acting as a portal of entry for infection (eg athlete's foot), but it may also occur without any obvious breach in the skin. Ascertain whether or not there is evidence of systemic upset or any background problems such as immunodeficiency, diabetes, or steroid therapy.

The area of affected skin is red and warm to touch, with poorly defined margins. Check T° and look for lymphangitis and/or lymphadenopathy.

Mark the limits of the cellulitis with a permanent marker pen to enable future judgements on improvement/deterioration.

### *Treatment*

Treatment depends upon the nature and extent of clinical findings, as follows (see https://cks.nice.org.uk):

- Treat patients who have localized limb infection and no evidence of systemic upset with oral antibiotics (either flucloxacillin ± phenoxymethylpenicillin or co-amoxiclav or clarithromycin), and arrange follow-up in 36–48hr. Advise patients to return sooner for review if symptoms significantly worsen.
- Admit patients who are systemically unwell or have spreading infection (eg lymphangitis extending above the knee from an area of cellulitis on the foot). Obtain venous access; take blood cultures and start IV antibiotics (either benzylpenicillin + flucloxacillin or co-amoxiclav).
- Consider admission for IV antibiotics for patients who are systemically well but have a significant comorbidity (eg chronic venous insufficiency, morbid obesity, peripheral arterial disease). Note, however, that where systems exist to support it, some of these patients ('Eron class II') may be safely managed in the community by IV antibiotics and regular review.
- Admit patients with cellulitis of the face (particularly around the eye), unless very minor. They are at risk of significant intracranial complications (notably cavernous sinus thrombosis)—start IV antibiotics, and refer for admission to the ophthalmology team.

## Erysipelas

This streptococcal infection is limited to the more superficial parts of the skin, resulting in an area of redness and heat, with clearly defined margins. Treat with antibiotics, as outlined for cellulitis above, except that phenoxymethylpenicillin alone (500mg PO qds for 7 days) suffices in most cases.

## Necrotizing fasciitis

(See ➲ Streptococcal infections, p. 244.)

# Abscesses

An abscess is a localized collection of pus, resulting in a painful soft tissue mass that is often fluctuant, but surrounded by firm granulation tissue and erythema. The cause is usually bacterial, resulting from minor trauma to the epithelium/mucosa or blockage of apocrine glands. A history of a previous lump at the site suggests infection of a sebaceous cyst. Check BMG in all patients.

For patients with recurrent abscesses, check for signs of hidradenitis suppurativa, diabetes, inflammatory bowel disease, and malignancy. Ask about steroid use.

## Treatment

*Incision and drainage* A general surgical principle is that a collection of pus requires drainage. On occasions, depending upon local policy, it may be appropriate to do this in the ED. Some abscesses (eg face, breast, perineum, paediatric) require specialist attention. Regional, parenteral, or general anaesthesia may be needed to supplement LA, which works poorly in this situation.

*Technique* Incise along the length of the fluctuance and deep enough to enter the cavity. An elliptical incision will prevent premature closure and re-accumulation of pus. Send pus for culture. Ensure that loculi in the cavity are gently broken by the use of a curette. Consider inserting a loose antiseptic wick in the cavity to ensure drainage and prevent premature closure.

*Antibiotics* Not indicated in patients with normal host defences as long as the abscess is localized. Evidence of surrounding or spreading infection may warrant antibiotics (eg co-amoxiclav or penicillin + flucloxacillin) and, on occasions, admission (see below).

*Refer the following*

- Those who are systemically unwell (pyrexia, tachycardia, rigors), the immunocompromised, and those not responding to treatment.
- Abscesses secondary to IV drug misuse.
- Those with infection in certain anatomical sites: face (↑ risk of cavernous sinus thrombosis), those potentially involving the airway (sublingual abscesses, Ludwig's angina), and axillary, groin, retropharyngeal, perineal, and breast abscesses.
- Those with extensive or progressing cellulitis/lymphangitis.

These patients may require IV antibiotics (eg flucloxacillin + penicillin or co-amoxiclav IV), analgesia, and surgical drainage. Take blood for FBC, clotting studies, and blood culture. Treat sepsis/septic shock where necessary (see ➲ Sepsis, pp. 62–3 and ➲ Shock, pp. 64–5).

## Breast infection

*Lactational breast abscess* These are usually peripherally located and due to *Staphylococcus aureus*. Local discomfort proceeds to painful swelling. Overlying skin may be red. Extreme cases may undergo necrosis and spontaneous discharge.

*Technique* If seen prior to frank abscess formation, consider antibiotic treatment alone—prescribe a penicillinase-resistant antibiotic. If there is any suspicion of an abscess, refer for needle aspiration—if pus is found, drainage will be needed. Encourage the infant to feed from the contralateral breast whilst the affected side is emptied of milk manually or by a breast pump.

*Non-lactational breast abscess* Typically affects the 30–60y age group, usually peri-areolar, recurrent, and related to duct ectasia/periductal mastitis. Refer for needle aspiration, culture, and antibiotics (metronidazole and flucloxacillin). Note that inflammatory breast cancer may mimic septic mastitis and breast abscess. Incision of neoplastic lesions may have disastrous results.

## Perineal abscesses

(See ➲ Anorectal problems, pp. 534–5.)

# Complications after surgery

All surgical procedures carry risks of complications. The most common and serious risks of a specific procedure are discussed with patients prior to the operation, as part of obtaining informed consent. Many patients experiencing problems soon after surgery will get in touch directly with the surgical team that performed the surgery or the ward staff that looked after them. However, many patients simply attend (or are advised to attend) the ED (sometimes at a different hospital), especially out of hours.

## Approach

- Consider surgical complication as the cause of symptoms in any patient presenting within a few days of surgery.
- Try to establish exactly what surgery the patient has undergone.
- Remember that, irrespective of the nature of the operation, most surgeons will be very keen to be informed if a patient on whom they have operated develops a complication.
- Aim to involve the surgical team (or at least inform them) when treating a surgical complication.

## Specific surgical complications

### *Haemorrhage relating to skin wounds*

Apply pressure to a skin wound which is bleeding externally, and consider whether a further suture is needed and/or surgical referral. Haemorrhage developing deep to skin wounds can result in significant haematomas—manage according to the site, size, and situation. For example, significant haemorrhage relating to recent plastic surgery (eg 'facelift') may jeopardize the result, requiring urgent surgical intervention.

### *Internal haemorrhage*

This may not be easy to identify—adopt a high index of suspicion.

### *Infection*

Wound infection is usually apparent by localized pain, redness, and tenderness. Deeper infection may present in an atypical fashion—if there is any suggestion of this, check T°, WCC, and CRP and refer to the surgical team. Consider the possibility of intra-abdominal infection/collection in patients who have undergone recent laparotomy/laparoscopy.

### *Wound dehiscence*

This may be superficial or deep and is often associated with infection. Cover any exposed viscera with saline-soaked swabs, and refer to the surgical team.

## Misplaced gastrostomy tubes

(See ➲ Gastrostomy tube problems, p. 129.)

Inadvertent ('accidental') removal of percutaneous endoscopic gastrostomy (PEG) tubes requires a replacement tube to be inserted as soon as possible, so that the track does not close off. A temporary tube (eg lubricated urinary catheter) inserted gently will serve this purpose until a definitive replacement may be inserted. Refer to the relevant surgical/radiological interventionalist.

# Chapter 11

# Ophthalmology

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# Approach to eye problems

## History

Always take a full ophthalmic history. Which eye is affected (are both)? What is the disturbance? Are there flashing lights or floaters? How quickly did the symptoms come on? How does it affect the patient's lifestyle (job, reading, watching TV)? Ask about prior ophthalmic/optician treatment, and take a full medical and drug history. Family history of glaucoma may also be relevant.

*Always measure visual acuity* in anyone presenting with an eye problem. Pointers to potentially serious pathology include those with:

- Sudden visual loss.
- Significantly ↓ VA.
- Penetrating eye injuries.
- Chemical burns of the eye (these require immediate treatment and specialist referral).

Have a low threshold for involving an ophthalmologist if a patient who is already blind in one eye presents with a problem with the 'good eye'.

## Examination

*Visual acuity* This is the key to eye examination—measure this first.

*Failure to document VA may constitute negligence.*

Use a Snellen chart, and read at 6m for each eye separately. Allow patients to use glasses, if available; if not, employ a pinhole (made using a needle through a piece of card). Use of a pinhole eliminates refractive error.

VA is expressed as:

- *Distance from the chart in metres/number of lines on the chart (normal vision is 6/6),* eg a patient whose VA is recorded as 'Right eye 6/5; left eye 6/60' can read the bottom line with the right eye, but only the top line with the left eye. If patients read additional letters of the line below, record using + number of extra letters (eg 6/12 + 2).
- Bring patients unable to read the chart at 6m forward until they can read the chart (eg 3/60 = top line read at 3m). Very poor vision: try counting fingers or detecting hand movement at 1m, or light perception.
- A hand-held chart at 30cm is an alternative if a full Snellen chart is unavailable—ability to read small print implies normal VA for that eye. For patients who are illiterate, there is an alternative chart with various different versions of the letter 'E'—ask the patient to state in which directions the three limbs of the letter point.

*Pupils* Record pupil size, shape, and direct and consensual responses to light and accommodation.

*Eye movements* Check full range and for diplopia. Look for nystagmus.

*Visual fields* Check carefully in patients with visual loss.

*Fundoscopy* In a darkened room, first check for the red reflex. A lost or ↓ red reflex is abnormal, typically caused by vitreous haemorrhage, cataracts, or major corneal abrasions. Assess the optic discs and look for retinal haemorrhages and vessel abnormalities. Sometimes, there may be a poor view—leave the use of drops to dilate the pupil (eg tropicamide) to the ophthalmologist.

*Direct assessment* Under a bright light, look for inflammation or FBs.

*Subtarsal examination* If there is a possibility of an FB, evert the upper eyelid by pressing down lightly over the upper lid with a cotton bud or orange stick and rotating the lid upwards over it. Ask the patient to look down throughout.

*Slit lamp examination* Learn how to use a slit lamp. It allows a detailed view of the conjunctiva, cornea, and anterior chamber. Fluorescein staining reveals corneal abnormalities, particularly when viewed under blue light when abrasions appear yellow/green. Fluorescein is available either in drop form or dried onto a strip. Remember that fluorescein can permanently stain clothes and contact lenses.

*Intra-ocular pressure* Digital assessment is unreliable. Formal measurement of intra-ocular pressure is useful but requires training and is left to the eye specialist in many departments.

*Temporal arteries* Palpate for tenderness if temporal arteritis is a possibility.

### LA drops to aid examination

Sometimes, blepharospasm prevents satisfactory examination. Consider LA drops (one or two drops of 1% amethocaine/tetracaine or 0.4% oxybuprocaine; 0.5% proxymetacaine causes less stinging and is useful in children). *Never discharge patients with a supply of LA drops.*

### Notes on ophthalmological treatments

*Antibiotic ointment and drops* Apply to the lower fornix (between the lower eyelid and the sclera), then ask the patient to keep the eye shut for 1–2min. Ointment has the advantage over drops in that it lasts longer, eg chloramphenicol ointment needs to be given four times a day, whereas drops need to be given every 2hr initially. Theoretical concerns about aplastic anaemia are not well founded (see *BNF*).

*Eye pads* These were previously recommended following the administration of LA drops and for patients with corneal abrasions; they tend not to be useful.

*Driving* Advise patients not to drive until their vision has returned to normal (this particularly applies after the use of mydriatic agents). In addition, advise patients not to drive whilst wearing an eye pad. Document the advice given in the notes.

# Blunt eye injury

Blunt injury to the face may result in injury to the orbit or its bony margins. Compression of the eye in an antero-posterior direction (eg squash ball or fist) can cause a *'blow-out' fracture* of the floor of the orbit.

## Retrobulbar haematoma

This may lead to orbital compartment syndrome and blindness. Unless diagnosed and treated as an emergency, optic nerve ischaemia develops and the patient can lose sight in the affected eye within a few hours. Proptosis, reduced eye movements, reduced VA, and pain all point to a retrobulbar haematoma. There may be an afferent pupillary defect.

## Assessment

Swelling around the eye can make assessment difficult, and this situation may worsen as swelling ↑, so try to examine the eye at the earliest opportunity.

- Look for proptosis.
- Check VA.
- Check pupillary reflexes.
- Check for enophthalmos and ↓ infra-orbital nerve sensation, both found in a blow-out fracture.
- Document the range of eye movements, looking, in particular, for entrapment of the extra-ocular muscles.
- Look for a hyphaema (a horizontal fluid level in the anterior chamber when the patient is upright). It can cause pain, photophobia, and blurred vision and can ↑ the intra-ocular pressure, causing nausea and vomiting.
- Stain the cornea and examine using the slit lamp for corneal abrasions.
- Ophthalmoscopic examination may reveal lens dislocation, hyphaema, and vitreous, subhyaloid, or retinal haemorrhage. Sometimes retinal oedema ('commotio retinae') may be seen as white patches with diffuse margins on the posterior pole of the eye.

X-ray if there is bony tenderness or clinical evidence of orbital or facial bone fracture.

## Treatment

Any patient suspected of a retrobulbar haematoma requires an emergency lateral canthotomy and cantholysis. This should be performed by an ophthalmologist or a trained emergency physician, under LA in the ED, and it reduces the retro-orbital pressure.

Nurse patients with an obvious globe injury head up at 45°. Refer urgently. Consider prophylactic oral antibiotics (eg co-amoxiclav) for uncomplicated facial or orbital fractures, according to local policy, and arrange for maxillofacial follow-up, with advice to avoid nose-blowing in the meantime.

# Penetrating eye injury

Suspect an *intra-ocular FB* if there is a history of hammering or work involving metal on metal. Establish if protective glasses were worn—remember that standard glasses (without a seal to the skin) do not provide full protection against an FB. Ascertain whether a small FB travelling at speed may have penetrated the orbit (eg during grinding, hammering, chiselling). Failure to suspect and diagnose these injuries can have serious consequences.

## Assessment

- Check VA.
- Look for pupil irregularity.
- Look for puncture/entry wounds on both aspects of the eyelids, the cornea, and the sclera. Corneoscleral wounds are often situated inferiorly, due to upturning of the eyeball as the patient blinks.
- Examine the anterior chamber. There may be a shallow anterior chamber, air bubbles, a flat cornea, a deflated globe, and a positive Seidel's test (dilution of fluorescein by aqueous humour leaking from the anterior chamber).
- Look for a hyphaema.
- Look for vitreous haemorrhage on fundoscopy.

*X-ray all patients with possible globe penetration (consider also CT or USS).*

Give analgesia, tetanus prophylaxis, and IV antibiotics (eg cefuroxime 1.5g), and refer all patients with penetrating eye injuries immediately to an ophthalmologist, even if there are other major injuries needing attention at the same time.

Do not manipulate or try to remove embedded objects (eg darts).

## Eyelid wounds

Lacerations (and incised wounds) to the eyelids may be a pointer to the presence of associated (more significant) globe damage. Most eyelid wounds can be closed under LA using small (6/0 or 7/0) interrupted nylon sutures—in most instances, this is more appropriately undertaken by a specialist.

# Corneal trauma

## Conjunctival FB

The typical history is of dust or grit blown into an eye by the wind. The FB usually gravitates into the lower fornix—remove it with a cotton bud, then check for associated subtarsal/corneal FBs.

## Subtarsal FB

FBs may not gravitate into the lower fornix but may remain stuck under the upper eyelid. The patient reports pain on blinking. Fluorescein staining reveals characteristic vertical corneal abrasions (the cornea has been likened to an 'ice rink'). Evert the upper eyelid and remove the FB with a cotton bud. Discharge with topical antibiotic (eg chloramphenicol ointment qds or fusidic acid eye drops).

## Corneal abrasions

These often result from a newspaper or fingernail in the eye. Irritation, photophobia, and lacrimation occur. Use LA drops and fluorescein staining to examine the cornea. Exclude FB or penetrating injury. Prescribe regular antibiotic ointment (eg chloramphenicol) and oral analgesia. Only consider an eye patch if the abrasion is very large (>1cm diameter). If the patient is very uncomfortable, instilling a drop of 1% cyclopentolate to dilate the pupil (this reduces iris spasm) or a drop of 0.1% diclofenac may help. Advise the patient not to drive until vision has returned to normal. Advise also to return for review if symptoms continue beyond 36hr.

## Corneal FB

Instill LA and attempt removal with a cotton bud (moistening it and rolling it over the FB to pick it up may help). If unsuccessful, remove with a blue (23G) needle introduced from the side (ideally using a slit lamp). Ensure that the patient's head is firmly fixed and cannot move forward onto the needle—it can also help to attach the needle to a syringe and for the operator's hand to rest lightly on the patient's cheek to help to keep it steady. After complete removal of the FB, check that the anterior chamber is intact; instill and prescribe antibiotic ointment, and advise the patient to return if symptomatic at 36hr. Refer patients with large, deep, or incompletely removed FB or if a rust ring remains afterwards.

## Arc (welder's) eye/'snow blindness'

Exposure to ultraviolet light can cause superficial keratitis. Climbers/skiers, welders, and sunbed users who have not used appropriate protective goggles develop pain, watering, redness, and blepharospasm several hours later. LA drops allow examination with fluorescein staining, revealing multiple punctate corneal lesions. Consider instilling a drop of 1% cyclopentolate or 0.1% diclofenac into both eyes. Discharge with an eye pad, oral analgesia, and advice not to drive until recovered. Anticipate resolution within 24hr. Do not discharge with LA drops.

### Chemical eye burns

Chemical burns from alkali or acid are very serious. Triage urgently ahead; check TOXBASE® (https://www.toxbase.org), and irrigate the eye immediately with lukewarm normal saline for at least 20min or until the pH of tears has returned to normal (~7.4). A 1L bag of 0.9% saline with standard IV tubing is ideal. LA may be needed to enable full irrigation. Consider the need for protective clothing during irrigation. Try to identify the substance involved, and contact the Poisons Unit. Refer alkali and acid burns immediately to the ophthalmologist.

### Superglued eyelids

Wash with warm water. The eye will open within 4 days. If the patient reports an FB sensation, this may represent a lump of glue, which may cause an abrasion if untreated—refer to the ophthalmologist.

Note that despite precautions, occasionally eyelids are glued together during the application of tissue glue to close a forehead wound (see Skin tissue glue, p. 415). If the eyelids remain closed despite simple measures, contact the ophthalmologist ± TOXBASE® (https://www.toxbase.org).

## Contact lens problems

Contact lenses may be 'soft' (more comfortable) or 'hard'. Avoid using fluorescein with contact lenses, as permanent staining may occur.

### 'Stuck lens'

Most contact lens users are adept at removing their lenses. New users, however, can experience difficulty. Moisten soft lenses with saline, then remove by pinching between the finger and the thumb. Special suction devices are available to help remove hard lenses.

### 'Lost lens'

Patients may present concerned that they are unable to find their contact lens and cannot remember it falling out. Check under both eyelids carefully (evert the upper lid if the lens is not immediately apparent) and remove the offending lens, if present.

### Hypersensitivity and overuse

Preservatives in lens-cleaning fluid cause itching and may evoke a reaction. Advise to stop using the lenses; give antibiotic ointment, and arrange ophthalmological follow-up.

### Acanthamoeba keratitis

This protozoal infection of the cornea occurs in contact lens users, associated with poor lens hygiene or swimming whilst wearing contact lenses. The eye becomes painful and red. Corneal oedema and ulceration develop. If suspected, refer immediately for ophthalmological care.

### Other problems related to contact lenses

Treat and refer conjunctivitis, corneal abrasions, or ulcers apparently related to contact lenses, as outlined in The red eye, pp. 558–9. Advise avoidance of use of both contact lenses until the problem has resolved, and arrange appropriate follow-up with the GP or an ophthalmologist.

# Sudden visual loss

Sudden visual loss requires emergency assessment and treatment.

## Amaurosis fugax

The patient describes temporary loss of vision in one eye, like a 'curtain coming down', with complete recovery after a few seconds to minutes. The cause is usually a thrombotic embolus in the retinal, ophthalmic, or ciliary artery, originating from a carotid atheromatous plaque, but it can also be a feature of giant cell arteritis (see ➲ Giant cell (temporal) arteritis, p. 557). Treat as for TIA (see ➲ Transient ischaemic attacks, p. 155), and involve the ophthalmology team (to exclude other 'eye' pathology).

## Central retinal artery occlusion

The central retinal artery is an end artery. Occlusion causes an ischaemic stroke of the retina. It is usually embolic (check for AF and listen for carotid bruits), causing sudden painless ↓ VA to counting fingers or no light perception. The patient may have a history of amaurosis fugax. Direct pupil reaction is sluggish or absent in the affected eye, but it reacts to consensual stimulation (afferent pupillary defect). Fundoscopy reveals a pale retina, with a swollen pale optic disc and a 'cherry red macula spot' (the retina is thinnest here and the underlying choroidal circulation is normal). Retinal blood vessels are attenuated and irregular—there may be 'cattle-trucking' in arteries.

*Treat* by digitally massaging the globe (with the eye closed) for 5–15s, then release and repeat to dislodge the embolus, whilst awaiting urgent arrival of an ophthalmologist.

If there is any delay in the patient being seen by the ophthalmologist, discuss other options such as giving acetazolamide 500mg IV (to ↓ intra-ocular pressure and ↑ retinal blood flow). Note that current evidence does not support the use of thrombolytic agents in this situation.

Do reconsider the diagnosis. In particular, consider whether or not giant cell (temporal) arteritis (see ➲ Giant cell (temporal) arteritis, p. 557) is a possibility—ask about jaw claudication, headaches, and scalp tenderness.

## Central retinal vein occlusion

This is a more frequent cause of sudden painless visual loss than arterial occlusion. Predisposing factors include: old age, chronic glaucoma, arteriosclerosis, hypertension, and polycythaemia. Examination reveals ↓ VA, often with an afferent pupillary defect. Fundoscopy reveals a 'stormy sunset' appearance—hyperaemia with engorged veins and adjacent flame-shaped haemorrhages. The disc may be obscured by haemorrhages and oedema. Cotton wool spots may be seen. Although the outcome is variable and there is currently no specific treatment, refer urgently as the underlying cause may be treatable, thus protecting the other eye.

### Giant cell (temporal) arteritis

Inflammation of the posterior ciliary arteries causes ischaemic optic neuritis and visual loss. It is relatively common in those aged >50y and is associated with polymyalgia rheumatica. The other eye remains at risk until treatment is commenced. Rapid and profound visual loss may be preceded by headaches, jaw claudication, general malaise, and muscular pains—often these symptoms can worsen over several weeks or months. The temporal arteries are characteristically tender to palpation. Retinal appearances have been termed 'pale papilloedema'—the ischaemic disc is pale, waxy, and elevated and has splinter haemorrhages on it. If suspected, give oral prednisolone 60mg immediately; check ESR (typically >>40mm/hr but can be normal), and refer urgently.

### Vitreous haemorrhage

This occurs in diabetics with new vessel formation and in bleeding disorders and retinal detachment. Small bleeds may produce vitreous floaters with little visual loss. Large bleeds result in painless ↓↓ VA, an absent red reflex, and difficulty visualizing the retina. Refer urgently. Meanwhile, elevate the head of the bed to allow blood to collect inferiorly.

### Retinal detachment

This occurs in myopes, diabetics, and the elderly and following trauma. The rate of onset is variable—patients may report premonitory flashing lights or a 'snow storm', before developing cloudy vision. There may be a visual field defect. Macular involvement causes ↓ VA. The affected retina is dark and opalescent but may be difficult to visualize by standard ophthalmoscopy. Refer urgently for surgery and surgical reattachment or retinal laser photocoagulation.

### Optic neuritis

This usually presents in a young woman. Optic nerve inflammation and demyelination cause visual loss over a few days. Pain on eye movement may occur. An afferent pupillary defect is associated with ↓ VA, ↓ colour vision (the colour red looks faded), and a normal/swollen optic disc. Most recover untreated; later some develop MS. Refer to the ophthalmologist (possibly to discuss steroid treatment—currently controversial and not proven).

### Other causes

Patients with chronic visual loss due to a variety of conditions may present acutely (senile macular degeneration, glaucoma, optic atrophy, cataract, choroidoretinitis). Drugs which can cause painless visual loss include methanol (see Methanol poisoning, p. 211) and quinine (in overdose). Refer immediately all patients in whom an acute visual loss cannot be excluded.

# The red eye

Refer all patients with new findings of ↓ VA, abnormal pupil reactions, or corneal abnormalities.

## Orbital and preseptal cellulitis

(See ➲ Cellulitis, p. 545.)

This is a major infection of the orbital tissues. The infection most frequently spreads from the paranasal sinuses (ethmoid sinusitis), facial skin, or lacrimal sac. Occasionally, the infection follows direct trauma to the orbit or from haematogenous spread. Patients present with fever, eyelid swelling, erythema, and proptosis. Assess for signs of severe sepsis (see ➲ Sepsis, pp. 62–3 and ➲ Shock, pp. 64–5), and resuscitate as necessary. Obtain venous access; take blood for cultures, and commence IV antibiotics (eg co-amoxiclav) and fluids. Refer urgently to the ophthalmologist. Some aggressive infections may require surgical treatment. Cavernous sinus thrombosis and meningitis are potential complications.

## Acute iritis (acute uveitis)

A relapsing condition of the young/middle-aged, associated with ankylosing spondylitis, ulcerative colitis, sarcoid, AIDS, and Behçet's syndrome.

*Symptoms* Include acute-onset pain, photophobia, 'floaters', blurred vision, and watering.

*Signs* ↓ VA, tender eye felt through the upper eyelid, circumcorneal erythema, small pupil (may be irregular due to previous adhesions). Shining a light into the 'good' eye causes pain in the other. Pain ↑ as the eyes converge and the pupils react to accommodation (Talbot's test). Slit lamp examination may reveal hypopyon and white precipitates on the posterior cornea.

*Refer* Refer urgently to the ophthalmologist for steroid eye drops, pupil dilatation, analgesia, investigation, and follow-up.

## Acute closed angle glaucoma

Long-sighted middle-aged or elderly individuals with shallow anterior chambers are at risk. Sudden blocked drainage of aqueous humour into the canal of Schlemm causes intra-ocular pressure to ↑ from 10–20mmHg up to 70mmHg. This may be caused by anticholinergic drugs or pupil dilatation at night (reading in dim light).

*Symptoms* Include preceding episodes of blurred vision or haloes around lights due to corneal oedema. Acute blockage causes severe eye pain and nausea/vomiting.

*Signs* ↓ VA, hazy and oedematous cornea with circumcorneal erythema, and a fixed semi-dilated, ovoid pupil. The eye feels tender and hard through the upper eyelid. Measure the intra-ocular pressure if this facility is available.

*Treatment* Instill a 4% pilocarpine drop every 15min to produce ciliary muscle contraction and aqueous humour drainage. Apply prophylactic 1% pilocarpine drops into the other eye also. Give analgesia (eg morphine IV with antiemetic). Arrange an emergency ophthalmology opinion—consider giving acetazolamide 500mg IV (to ↓ intra-ocular pressure) in the meantime and/or mannitol 20% up to 500mL IVI over 1hr.

### Conjunctivitis

This is caused by bacteria (*Streptococcus pneumoniae* or *Haemophilus influenzae*), viruses (adenovirus), or allergy. The sensation of FB may involve both eyes. The conjunctiva is red and inflamed, sometimes with eyelid swelling. VA and pupils are normal. Bacterial infection classically produces sticky mucopurulent tears, and viral infection copious watery tears (associated with photophobia and pre-auricular lymphadenopathy in the highly contagious adenoviral 'epidemic keratoconjunctivitis'). It is not possible to clinically distinguish viral from bacterial cases.

Advise not to share towels or pillows. Most cases settle relatively quickly with symptomatic measures—advise patients to see the GP if not better in 4 days or to return if significantly worse. Reserve a course of antibiotic eye drops or ointment (eg fusidic acid, chloramphenicol, or gentamicin) for patients whose symptoms last >5 days.

### Ulcerative keratitis

Corneal ulceration causes pain with photophobia. It is apparent on fluorescein staining under a slit lamp.

- Hypopyon (pus in the anterior chamber) implies bacterial infection.
- Vesicles in the ophthalmic division of the trigeminal nerve occur with herpes zoster infection.
- Dendritic branching ulcers suggest herpes simplex. If misdiagnosed and steroid eye drops are given, ulceration can be disastrous.

Whatever the infective agent, refer corneal ulceration cases immediately.

### Episcleritis

Inflammation beneath one area of the conjunctiva is usually associated with a nodule and a dull aching discomfort. VA, pupils, and the anterior chamber are normal. Prescribe oral NSAIDs, and advise GP review ± outpatient follow-up to consider steroid eye drops if there is no resolution.

### Blepharitis

This chronic problem is quite common. Eyelashes are matted together and itchy. Ensure that there is no associated corneal ulceration; provide topical antibiotics (eg chloramphenicol), and refer for GP follow-up.

### External hordeolum (stye)

Treat staphylococcal infections of eyelash roots with warm compresses.

### Internal hordeolum (chalazion)

A chalazion is an inflammatory reaction in a blocked meibomian (tarsal) gland, which may get secondarily infected. Treat infected tarsal glands with topical antibiotics (eg chloramphenicol) plus oral antibiotics (eg co-amoxiclav). Refer patients who develop an abscess or nodule affecting vision.

### Dacrocystitis (lacrimal sac infection)

This may follow nasolacrimal duct obstruction. Treat early infection with oral antibiotics (co-amoxiclav); later, refer for drainage.

### Subconjunctival haemorrhage

This usually presents as a painless, well-defined area of haemorrhage over the sclera. It may result from vomiting or sneezing. Following trauma, consider orbital or base of skull fracture, and treat accordingly. Reassure the patient that subconjunctival haemorrhage will resolve in time.

# Pupillary abnormalities

Pupillary examination may yield valuable information (see Table 11.1).

**Table 11.1** Syndromes relating to examination of pupils

| | Description | Common causes |
|---|---|---|
| *Abducens nerve palsy* | Diplopia, inability to look outwards | Trauma, tumour, Wernicke's syndrome |
| *Adie pupil* | Pupil is dilated and responds abnormally slowly to light | Commonly part of Holmes-Adie syndrome—cause unclear |
| *Anisocoria* | Unequal pupils | May be physiological or reflect disease process (eg third nerve palsy or Horner's syndrome) |
| *Argyll Robertson pupil (usually bilateral)* | Small pupils that accommodate, but do not constrict, to light | Pathognomonic of tertiary syphilis |
| *Horner's syndrome (sympathetic nerve damage)* | Miosis, ptosis, and anhidrosis (usually unilateral) | Lung cancer, trauma, lateral medullary syndrome, carotid artery dissection |
| *Hutchinson's pupil* | (Ipsilateral) pupil dilates and is unreactive to light | Compression of the oculomotor nerve can follow trauma or tumour often due to temporal lobe herniation |
| *Marcus Gunn pupil (relative afferent pupillary defect)* | Light shone in affected eye causes slow direct and consensual pupil reactions (light in normal eye gives brisk direct and consensual reactions) | Optic neuritis, giant cell arteritis, optic nerve trauma, retinal detachment |
| *Miosis* | Excessive pupillary constriction | Drugs such as opioids (bilateral miosis), Horner's syndrome (unilateral miosis) |
| *Mydriasis* | Excessive pupillary dilatation | Drugs, post-cardiac arrest |
| *Oculomotor nerve palsy* | Ptosis, mydriasis, and movement of the eyeball downward and outward (unopposed action of fourth and sixth nerves) | Trauma, tumour, aneurysm, demyelinating disorders, cavernous sinus thrombosis |
| *Trochlear nerve palsy* | Vertical diplopia; inability to look down and in | Trauma, haemorrhage, infarction |

# Chapter 12

# Ear, nose, and throat

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# Ear, nose, and throat foreign bodies

## Ear foreign bodies

Various FBs may become lodged in the external auditory canal, including beads, insects, and vegetable matter. FBs may cause pain, deafness, discharge, or, in the case of live insects, an irritating buzzing in one ear.

*Diagnosis* Depends upon direct visualization with the auroscope. In children, as with FBs elsewhere, there may be no history of FB available.

*Removal* (See Fig. 15.13 for positioning of the child.)

- Many FBs can be removed under direct vision with hooks or crocodile forceps. Manipulate gently to avoid damage or further impaction.
- Suction with a small soft plastic catheter may be successful.
- Drown live insects in 2% lidocaine first.
- Do not try to syringe out vegetable matter with water, as this may cause swelling and pain.
- If there is difficulty (especially with an unco-operative child), refer to ENT to consider removal under GA. Removing beads using an orange stick with a tiny amount of superglue on the end can work but carries some obvious dangers and requires complete patient co-operation.
- Check the tympanic membrane is intact after FB removal.

*Embedded earrings* The 'butterfly' piece of an earring may become embedded in the posterior part of the earlobe, causing inflammation or infection. The earrings are usually easily removed once adequate analgesia has been established—render the ear anaesthetized with a greater auricular nerve block (see ➲ Nerve blocks of forehead and ear, p. 310–11), or directly infiltrate LA into the lobe, remembering that this is a highly sensitive area. To release the butterfly, apply pressure in a posterior direction. Occasionally, forceps and a small posterior skin incision may be required to open up the track. If there is evidence of infection, prescribe oral antibiotics (eg flucloxacillin), and arrange GP follow-up. Advise the patient not to wear earrings until the symptoms have settled.

## Nasal foreign bodies

These usually affect children, who present with offensive unilateral nasal discharge. They also occur in adults with psychotic illness or learning disabilities.

*Removal* Remove easily accessible anterior nasal FBs in the ED. However, there is a small risk of aspiration with any nasal FB, particularly in unco-operative patients. Refer such patients to an ENT surgeon for removal with airway protection. If co-operative, instruct the patient to blow their nose whilst occluding the unaffected nostril. If unsuccessful, consider attempting removal with a nasal speculum, hook, and forceps, as appropriate. A fine-bore tracheal suction catheter attached to wall suction can also work. One technique which can work in co-operative children is to ask a parent to blow into the child's mouth ('parent's kiss'), having first ensured a good seal and also occluded the normal nostril.

Nasal *button batteries* (see ➲ Button batteries, p. 220) can cause significant damage, so refer to ENT.

### Inhaled foreign bodies

Aspiration causing complete upper airway obstruction is an emergency, requiring immediate intervention (see ➲ Airway obstruction: basic measures, pp. 334–5). FBs lodged in the larynx or tracheobronchial tree cause persistent coughing. There may not be a clear history—suspect an inhaled FB if a child presents following an episode of coughing/spluttering. Auscultation of the chest is often normal but may reveal wheezes or localized absence of breath sounds.

*CXR* May be normal or show a radio-opaque FB, with distal consolidation or hyperinflation (FB acting as a ball valve). A CXR in expiration may demonstrate this more clearly. Refer to a cardiothoracic surgeon.

### Ingested foreign bodies

Various FBs, both radio-opaque (eg coins, rings) and non-radio-opaque (eg plastic pen tops, aluminium ring pulls) are frequently swallowed by children and by adults with psychiatric disorders. Provided the FB reaches the stomach, it is likely to pass through the rest of the GI tract without incident. Button batteries are an exception (see ➲ Button batteries, p. 220).

A hand-held metal detector may confirm a swallowed coin is below the xiphisternum and avoid the need for X-ray. For radio-opaque FBs, confirm with lateral neck X-ray and CXR that the FB is not impacted in the oesophagus. If there is doubt as to whether an FB is radio-opaque, consider X-raying a similar object if one is available (possibly by placing it on the patient's shoulder during the CXR).

Refer patients who are symptomatic, have impacted FBs, or have swallowed potentially dangerous items (button batteries, razor blades, open safety pins). Note that magnets can be dangerous if two or more are ingested, since they can attract each other through tissues and cause pressure necrosis/perforation of the bowel. Only discharge patients who are asymptomatic (with advice to return if they develop abdominal pain and/or vomiting), and arrange suitable follow-up. Unless the ingested FB is valuable or of great sentimental value, examination of the stools by the patient for the FB is unnecessary. It may take weeks to pass.

### Impacted fish bones

Fish bones often become stuck in the pharynx or oesophagus. Direct visualization with a good light (head torch useful) and a wooden spatula as tongue depressor may reveal fish bones lodged in the tonsil or base of the tongue—remove with Tilley's forceps. If no FB is seen, obtain soft tissue lateral neck X-rays (look for prevertebral soft tissue swelling/fish bone, remembering not all are radio-opaque), then refer to ENT for endoscopy. A fish bone can scratch the pharynx, causing a sensation of FB to persist.

### Oesophageal food bolus obstruction

This usually involves a lump of meat. Patients with complete obstruction are unable to swallow solids or liquids (including saliva). There may be retrosternal discomfort. Consider sips of fizzy drinks if obstruction is <24hr old and not complete. Refer patients with persistent obstruction for endoscopy. Glucagon and hyoscine butylbromide (eg Buscopan®) are used by some experts, but there is a lack of evidence to support their use and they carry a risk of side effects (vomiting, oesophageal rupture).

# Facial nerve palsy

The facial (VII) nerve supplies the muscles of facial expression. Examination reveals whether VII nerve palsy is an upper or lower motor neurone type.

*Upper motor neurone paralysis* is usually due to a stroke (see ➲ Stroke, pp. 150–1), resulting in unilateral facial muscle weakness, but with sparing of the muscles of the forehead. If stroke is the cause, there may be additional evidence elsewhere (eg hemiparesis affecting the limbs).

*Lower motor neurone paralysis* results in weakness of the muscles of one side of the face. The facial nerve arises from its nucleus in the pons and emerges from the pons to travel past the cerebello-pontine angle, through the petrous part of the temporal bone, to emerge from the stylomastoid foramen and thence into the parotid gland where it divides into branches. During its passage through the petrous temporal bone, the facial nerve is accompanied by the chorda tympani (carrying taste fibres from the anterior two-thirds of one half of the tongue) and gives off the *nerve to the stapedius*. Lesions of the facial nerve in the temporal bone therefore produce loss of taste and hyperacusis (noise is distorted and sounds loud) on the affected side.

Causes of lower motor neurone facial palsy are shown in Box 12.1.

**Box 12.1** Causes of lower motor neurone facial palsy

- Bell's palsy (most common cause)
- Pontine tumours
- Vascular events
- Acoustic neuroma
- Ramsay-Hunt syndrome
- Trauma
- Middle ear infection
- Cholesteatoma
- Sarcoidosis
- Parotid tumours, trauma, infection
- HIV

## Bell's palsy

This is the most common cause of sudden-onset isolated lower motor neurone facial nerve palsy. Most follow a viral infection, producing facial nerve swelling in the temporal bone ± hyperacusis and loss of taste in the anterior two-thirds of one half of the tongue. The absence of involvement of other cranial nerves is a reassuring feature, helping to secure this clinical diagnosis.

*Treatment* Most recover completely over several months without treatment—a few are left with permanent weakness. Recovery is quicker if prednisolone is started within 72hr of onset of symptoms (prednisolone 60mg daily for 5 days, then 10mg less each day, total of 10 days of treatment OR 10 days of prednisolone 25mg bd). Antivirals are controversial and do not appear to help. Advise the use of artificial tears and an eye patch at night, to prevent corneal drying, and refer for GP follow-up.

## Ramsay-Hunt syndrome

This is due to herpes zoster infection of the geniculate ganglion. Clinical features of Bell's palsy are present, together with (painful) herpetic vesicles present in the external auditory meatus and, occasionally, also the soft palate. Refer to an ENT specialist for aciclovir and follow-up.

# Ear examination

### Scope of the examination

Full ear examination includes assessment of the vestibulocochlear nerve and an auroscope examination. Check for mastoid or pinna tenderness. Look at the external ear canal for discharge or swelling, and examine the tympanic membrane for colour, translucency, bulging, and the cone of light.

### Assessing hearing

Hearing can be assessed by asking the patient to place one finger in their ear. Stand a foot behind the patient's unoccluded ear, and whisper a two-syllable word. Ask the patient to repeat the word.

### Weber's and Rinne's tests

*Weber's test* Strike a 512Hz tuning fork and place in the centre of the forehead. In conductive deafness, the sound localizes to the deaf ear; with sensorineural deafness, the sound localizes to the good ear.

*Rinne's test* Strike a 512Hz tuning fork and place it on the mastoid process. Ask the patient to tell you when they no longer hear the sound, then immediately place the tuning fork in front of the auditory meatus. In a normal ear, air conduction is heard for twice as long as bone conduction. In conductive deafness, bone conduction is heard for longer than air conduction. In sensorineural deafness, air conduction is heard longer than bone conduction.

### Nystagmus

To complete the assessment of the vestibulocochlear nerve, examine for nystagmus. All forms of nystagmus can be associated with intracranial lesions, as well as peripheral causes; however, downbeat and upbeat nystagmus, in particular, signify a central cause. Tinnitus or deafness tends to suggest a peripheral cause. Peripheral nystagmus is exacerbated by gazing towards the side of the fast phase (Alexander's Law). Central nystagmus may change direction, depending on the side of gaze.

# Cochlear implants

Cochlear implants consist of an implanted radio receiver and decoder package containing a magnet (above and behind the ear), together with a removable external microphone/radio transmitter. X-rays and CT do not damage this device, provided that the external microphone/transmitter is first removed and switched off. MRI can cause significant damage to the device and the patient. If there are concerns relating to a cochlear implant, refer to ENT. In particular, refer patients with:

- Significant direct trauma, including exposure by a scalp wound.
- Suspected otitis media of the implanted ear.

# Earache

## Otitis externa

Often caused by *Pseudomonas, Staphylococcus aureus, Streptococcus pneumoniae*, and *Escherichia coli*. Common in swimmers/surfers and after minor trauma. This causes intense itching and pain, which gradually ↑. Discharge and hearing loss may be present (profuse discharge implies middle ear disease). On examination, the external canal is inflamed and oedematous. Oedema and debris may obscure the tympanic membrane. Pain is induced by pressing on the tragus or pulling the pinna.

*Management* Prescribe topical antibiotics and topical steroids; advise against swimming, and arrange GP follow-up. In severe cases (eg if the drum is not visible), refer to an ENT surgeon for aural toilet to remove debris from the auditory canal.

## Cellulitis or furunculosis of the ear canal

Cellulitis of the ear canal may be caused by scratching or by infection of hair follicles (furunculosis). *S. aureus* is the usual organism. Itching and a feeling of pressure are followed by pain in the ear, with deafness if the ear canal is occluded by swelling. Examination shows swelling and inflammation of the ear canal, with tenderness over the tragus and pain on movement of the ear.

*Treatment* Analgesia (eg NSAID) and antibiotics (eg flucloxacillin 500mg PO qds for 5 days). Arrange GP follow-up (or ENT in severe cases).

## Acute otitis media

Most common in children aged 3–6y and may follow an URTI. The most common pathogens are *S. pneumoniae* and *H. influenzae*.

*Presentation* Earache may be accompanied by fever, deafness, irritability, and lethargy. Typically, hearing loss precedes pain. Examination of the tympanic membrane shows evidence of inflammation with loss of the light reflex and bulging of the drum. Eventual perforation results in purulent discharge, with some relief of pain. Look for associated swelling/tenderness over the mastoid—this implies secondary mastoiditis (see ➲ Acute mastoiditis, p. 567).

*Treatment* Prescribe oral analgesia. The use of antibiotics remains very controversial. Oral antibiotics (eg a 5-day course of amoxicillin or clarithromycin) are of questionable value but are frequently given. Prescribe oral antibiotics if the patient is very systemically unwell and/or is immunosuppressed or has significant comorbidities. Otherwise, consider antibiotics if there is no improvement within 72hr, or earlier if there is deterioration or perforation. If perforation has occurred (often heralded by a sudden ↑ in pain), arrange ENT follow-up and advise not to swim. Otherwise, arrange GP follow-up.

### Acute mastoiditis

This is an uncommon, but important, diagnosis to make, because of the risk of intracranial spread of infection. Mastoiditis is a complication of acute otitis media—consider it if there is no response to therapy (eg discharging ear for >10 days). Suspect mastoiditis if there is pain, redness, swelling, or tenderness over the mastoid process. The pinna may be pushed forwards/outwards—swelling may mean that the drum is not visible. Refer urgently to the ENT surgeon for admission and IV antibiotics.

### Cholesteatoma

This erosive condition affects the middle ear and mastoid. A cholesteatoma can result in life-threatening intracranial infection. There may be an offensive discharge, with conductive hearing loss, vertigo, or facial nerve palsy. Tympanic membrane examination shows granulation tissue and/or perforation with white debris. Refer to the ENT team.

### Traumatic tympanic membrane rupture

This may result from direct penetrating injury, blast injury (see ➔ Blast injuries, p. 397), or basal skull fracture (see ➔ Signs of base of skull fracture, p. 369). Pain is associated with ↓ hearing. Perforation is visible on examination.

*Treatment* Most heal spontaneously with conservative measures and advice to keep out of water. Arrange GP (± ENT) follow-up and give prophylactic oral antibiotics according to local policy. Note that gentamicin or neomycin eardrops may cause sensorineural deafness because of ototoxicity when the tympanic membrane is ruptured.

### Barotrauma

Sudden changes in atmospheric pressure with a blocked Eustachian tube can result in pain and hearing loss. This usually affects aircraft passengers and divers, especially if they have a cold (viral URTI). Pain is often relieved by the Valsalva manoeuvre (breathing out with the mouth closed, whilst pinching the nose). Decongestant nasal spray may help if the problem does not resolve spontaneously. Give analgesia (NSAID). Arrange ENT follow-up if the pain persists.

### Referred pain causing earache

Earache may result from referred pain, as shown in Table 12.1.

**Table 12.1** Causes of earache from referred pain

| Dental origin | Non-dental origin |
|---|---|
| Dental caries or abscess | Cervical spine spondylosis |
| Impacted molar tooth | Ramsay-Hunt syndrome |
| Pharyngeal infection or tumour | Temporomandibular joint dysfunction |

# Epistaxis

Nasal bleeding may be idiopathic or follow minor trauma (eg nose picking). Haemorrhage can be severe when associated with hypertension and coagulation disorders. Epistaxis may follow nasal fracture and major facial injury.

## Site of bleeding

Most nasal bleeding is from the anterior nasal septum in, or close to, Little's area. A few patients have posterior nasal bleeding, which may be brisk.

## Equipment

Direct visualization of the anterior nasal cavity is aided by a headlamp (eg battery-operated head torch), a fine soft suction catheter, and a nasal speculum. Wear goggles to avoid blood splashes in the eyes.

## Initial approach

*Associated facial injury* Assess ABC (especially pulse and BP), and resuscitate as necessary. Treat haemorrhagic shock (see ➲ Shock, pp. 64–5).

*No associated injury* Check airway, pulse, and BP. Treat hypovolaemia. Check coagulation status of patients on anticoagulants and treat appropriately (see ➲ Patients on anticoagulants, pp. 178–9). Sit the patient up, and instruct them to compress the fleshy part of their nose between their finger and thumb for 10min. If bleeding stops, the patient may be discharged after 30min observation.

## Continuing bleeding after pressure

*Adults* Apply a cotton wool pledget soaked in lidocaine with phenylephrine. Then, with a headlamp and a nasal speculum, try to identify the bleeding point. Treat small anterior bleeding points with cautious cautery by applying a silver nitrate stick for 10–15s. Avoid excessive cautery, and never cauterize both sides of the septum—this may cause septal necrosis. If cautery stops the bleeding, observe for 15min, and discharge with GP follow-up. Advise avoidance of sniffing, picking, or blowing the nose in the meantime.

*Children* Applying a nasal antiseptic cream (eg Naseptin®) is as effective as cautery in stopping bleeding. The cream is relatively easy to apply, but avoid if there is a history of peanut, soya, or neomycin allergy.

## Continuing bleeding despite cautery

Insert a compressed nasal tampon (eg Merocel®) or an inflatable pack (Rapid Rhino®)—follow specific product instructions on how to insert/inflate. Alternatively, pack traditionally with 1.25cm-wide ribbon gauze soaked in an oily paste (eg bismuth iodoform paraffin paste). Once packed, refer to ENT for observation, as the pack may dislodge and obstruct the airway.

## Continuing bleeding despite packing

Refer to ENT. The bleeding site is likely to be posterior and can cause hypovolaemic shock. In this case, insert two large venous cannulae; send blood for FBC, coagulation screen, and cross-matching, and start an IVI. Posterior nasal bleeding usually responds to tamponade with a Foley catheter. Remove the nasal tampon, and insert a lubricated, uninflated Foley catheter through the bleeding nostril into the nasopharynx. Inflate the balloon with air, and gently withdraw the catheter, thus tamponading the bleeding site. Tape the catheter to the cheek, then re-insert the anterior tampon.

# Nasal fracture

The prominent exposed position of the nose, combined with the delicacy of its bones, renders it relatively prone to injury.

Remember that the nose is part of the head, so nose injury = head injury (and potentially cervical spine injury also).

## History

The nose is commonly broken by a direct blow (eg from a punch) or following a fall onto the face. Nasal fracture is usually accompanied by bleeding. Search for a history of associated facial/head injury (diplopia, loss of consciousness, etc.).

## Examination

This is a clinical diagnosis based upon a history of injury with nasal swelling and tenderness. Having made the diagnosis, assess whether there is nasal deviation—it is useful to ask the patient to look in a mirror. Check and record whether the patient can breathe through each nostril. Look for an associated septal haematoma—this will appear as a smooth bulging swelling, which may obstruct the nasal passage. Children are at particular risk of septal haematoma, which predisposes to secondary infection and septal necrosis.

Assess for additional injuries to the head or face (eg tender mandible, diplopia, tender maxilla). Injury to the bridge of the nose may result in persistent epistaxis and/or CSF rhinorrhoea.

## Investigations

Do not X-ray to diagnose a nasal fracture—the diagnosis is a clinical one. Obtain appropriate X-rays (eg OPG or facial views) if there is clinical suspicion of other bony injuries. Nasal fractures are often apparent on facial X-rays or CT scans.

## Treatment

- Resuscitate and treat for associated head injury.
- Continuing nasal haemorrhage is uncommon—refer to an ENT surgeon to consider urgent MUA to stop the bleeding; meanwhile, insert a nasal tampon.
- Refer urgently to an ENT surgeon if there is a septal haematoma—this will require incision and drainage in order to prevent septal necrosis.
- Clean and close overlying skin wounds—Steri-Strips™ often allow good skin apposition. If there is significant contamination of the wound, start a course of prophylactic oral antibiotics (eg co-amoxiclav—one tablet PO tds for 5 days).
- Provide oral analgesia (eg ibuprofen 400mg PO tds).
- If the nose is deviated/distorted, or if there is too much swelling to judge, arrange for ENT follow-up at 5–7 days, so that MUA may be performed within 10 days. It is particularly important to ensure accurate reduction of fractures in children.
- Discharge with head injury instructions to the care of a relative.

# Sore throat

## Tonsillitis

*Causes* Acute pharyngo-tonsillitis may be caused by various agents:

- *Viral*: EBV, herpes simplex virus, adenoviruses.
- *Bacterial*: group A β-haemolytic *Streptococcus* (most common bacterial cause), *Mycoplasma, Corynebacterium diphtheriae*.

*Features* A sore inflamed throat often accompanies fever, headache, and mild dysphagia. Look for pus on the tonsils, and check for enlarged cervical lymph nodes—these are found in a variety of infections, but generalized lymphadenopathy (sometimes also with splenomegaly) may indicate glandular fever (infectious mononucleosis).

*Diagnosis* It is difficult to distinguish clinically bacterial from viral causes. Use the Centor criteria to help identify patients with a high chance of β-haemolytic streptococcal infection—three or more of: tonsillar exudate; tender anterior lymphadenopathy/lymphadenitis; absence of cough; and history of fever.

*Investigations* Consider throat swabs and anti-streptolysin titre in severe cases. If glandular fever is suspected, send blood for FBC and Paul–Bunnell (or Monospot) test.

*Treatment* Unless contraindicated, give paracetamol (1g PO qds PRN) and/or ibuprofen (400mg PO tds PRN) and discharge to the GP. Although frequently prescribed, oral antibiotics are rarely of benefit—a sensible approach is to limit their use for patients with any of the following: a history of valvular heart disease, immunosuppression, diabetes, marked systemic upset, peritonsillar cellulitis, known or suspected β-haemolytic streptococci (three or more Centor criteria—see above). In this case, prescribe phenoxymethylpenicillin (penicillin V) 500mg PO qds for 10 days (or clarithromycin 500mg PO bd for 5 days if allergic). Avoid ampicillin, amoxicillin, and co-amoxiclav, which cause a rash in patients infected with EBV.

Occasionally, patients with acute tonsillitis may be unable to swallow fluids (this is more commonly a feature of peritonsillar or retropharyngeal abscess). In this case, refer for IV antibiotics and IV fluids.

*Complications* Otitis media, sinusitis, retropharyngeal abscess, peritonsillar abscess.

## Peritonsillar abscess (quinsy)

Typically preceded by a sore throat for several days, the development of a peritonsillar abscess is heralded by high fever, pain localized to one side of the throat, and pain on swallowing. Difficulty swallowing can result in drooling. Trismus may make inspection difficult, but if visualized, there is a tense, bulging tonsil, pushing the uvula away from the affected side. Group A β-haemolytic streptococci are frequently implicated.

*Treatment* Insert an IV cannula and give IV benzylpenicillin 1.2g (clarithromycin 500mg if allergic to penicillin), and refer immediately to an ENT surgeon for aspiration or formal drainage.

### Retropharyngeal abscess

Spread of infection from adjacent lymph nodes may occasionally cause a retropharyngeal abscess, particularly in children aged <3y.

It is characterized by a sore throat, difficulty swallowing, fever, and dehydration. In children, cough is typically absent from the history (unlike in croup and other viral causes of upper airway obstruction). There may be evidence of airway compromise (stridor, neck hyperextension, signs of hypoxia). The differential diagnosis includes acute epiglottitis (see ➲ Acute epiglottitis, p. 693). Consider lateral neck X-ray which may show soft tissue swelling (preferably X-ray in the resuscitation room, rather than moving the patient to the X-ray department). Further imaging (CT or MRI) may be requested by the specialist team, especially if there is doubt about the diagnosis.

*Treatment* Get senior ED, ENT, and anaesthetic help. If the patient is a child with evidence of respiratory distress, do not upset them further. Airway obstruction may be precipitated by examination of the throat, so avoid this until appropriate staff and equipment are ready to cope with airway problems. The child can sit on mum's knee in the resuscitation room. On suspicion of a retropharyngeal abscess in an adult, insert an IV cannula; take bloods and blood cultures, and give IV fluids and IV antibiotics (eg ceftriaxone 2g IV daily + metronidazole 500mg IV tds OR if penicillin-allergic, clindamycin 600mg IV tds + gentamicin IV); refer immediately to an ENT surgeon.

### Paranasal sinusitis

Bacterial infection of the paranasal sinuses (maxillary, ethmoid, frontal, and sphenoid) may result from direct spread from infected tooth roots or (more usually) be secondary to viral URTI. Most cases resolve spontaneously.

*Clinical features*

- Clear nasal discharge becoming purulent.
- Pain in (and often also tenderness over) the affected sinus.
- Fever.
- Headache and/or toothache.

Note that occasionally, sinusitis may become fulminant and spread to involve adjacent bones and soft tissues.

*Management* Provide analgesia, and suggest warm face packs and nasal decongestant (eg 1% ephedrine). Consider oral antibiotics (eg amoxicillin, doxycycline, or clarithromycin) if there is significant comorbidity or if symptoms are severe (eg high fever, severe localized unilateral pain, and purulent discharge) (see https://cks.nice.org.uk). Advise GP follow-up.

In severe cases, refer to ENT for IV antibiotics and admission.

### Pharyngeal burns after cocaine use

Smoking cocaine can result in dangerous burns of the throat, since the drug acts as an LA. Swelling of the epiglottis may result in airway obstruction.

# Vertigo

Vertigo is the impression or illusion of movement when there is none. It is often accompanied by nausea and/or vomiting. Distinguish vertigo from 'dizziness', which is often used to describe a feeling of light-headedness.

## Causes of vertigo

*Peripheral (ear) causes* Typically sudden onset, severe, lasting for seconds or minutes (sometimes hours or days) and may be worsened by change in position and may be accompanied by auditory symptoms (hearing loss, tinnitus).

*Central (brain) causes* Typically result in milder nystagmus which is little affected by change in position, is accompanied by neurological findings, but not associated with auditory findings.
Causes of vertigo are shown in Table 12.2.

**Table 12.2** Causes of vertigo according to origin

| Peripheral | Central |
|---|---|
| • Benign positional vertigo | • Infection: meningitis, brain abscess |
| • Labyrithitis/neuronitis | • Post-traumatic |
| • Ménière's disease | • Subclavian steal syndrome |
| • Otitis media | • Vertebrobasilar insufficiency |
| • Wax or FB in the ear | • Stroke/cerebellar haemorrhage |
| • Acoustic neuroma | • MS |

Note that whilst patients are often worried that their symptom of vertigo may be due to a stroke, this is not often the underlying cause, especially in the absence of other neurological signs.

## Examination

- Perform a careful examination of the neurological system, including a search for cerebellar signs.
- Check the ears, including inspection of the tympanic membranes.
- Look for the characteristics of nystagmus—precipitation by changes in position strongly suggests a peripheral cause.
- Perform a Hallpike test—supporting the patient's head, with the eyes open, turn the head 45° whilst sitting upright, then lower the patient to lie supine, with the head lowered to 'hang' below the end of the trolley—ask if the manoeuvre has caused vertigo and check for nystagmus. Sit the patient back up. Repeat the test with the patient's head turned to the other side. A positive test results in vertigo (after a latent period of 5–10s), with nystagmus towards the affected side on lying down and with further transient vertigo (± nystagmus) on sitting up again.

## Vertigo and driving

Advise patients not to drive whilst experiencing vertigo. Document this advice in the notes, and communicate it to the patient's GP.

**Benign (paroxysmal) positional vertigo** Common. Mostly results from posterior semicircular canal otoliths and can follow head injury. Sudden-onset vertigo, positional in nature, lasting for seconds or minutes at a time, but recurring, sometimes into the long term. There may be associated nausea and/or vomiting. Diagnosed with Hallpike's test (see Vertigo, p. 572). Drug treatment is not usually effective, but vestibular exercises or the Epley manoeuvre may work (see Fig. 12.1).

**Acute labyrinthitis/vestibular neuronitis** Usually follows a viral URTI. Vertigo is usually severe, positional in nature, and often accompanied by nausea/vomiting and sometimes hearing loss. Refer to ENT if there is hearing loss; otherwise treat symptomatically with cyclizine (50mg PO tds). Anticipate recovery within days/weeks.

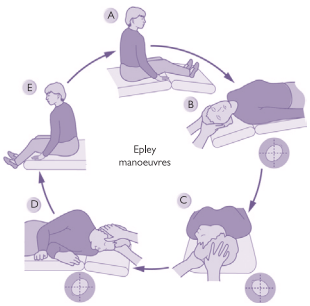


**Fig. 12.1** Epley manoeuvre.
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**Ménière's disease** Characterized by the triad of vertigo, tinnitus, and deafness. Recurrent vertigo typically lasts for hours at a time. There may be nausea/vomiting. Try oral cinnarazine or buccal prochlorperazine. Refer to the ENT team.

**Acoustic neuroma (or vestibular schwannoma)** Presents with slow-onset deafness and tinnitus. Vertigo and cranial nerve lesions (V, VI, IX, X) develop over time.

**Vertebrobasilar insufficiency** May be associated with headache and/or neurological symptoms/signs (eg diplopia, weakness, ataxia, dysarthria). Refer to the medical team.

**Stroke/cerebellar haemorrhage** Sudden onset of headache, vertigo, ataxia, and/or other cerebellar signs.

**Multiple sclerosis** May present with vertigo ± nausea, vomiting, and eye signs.

**Cause unclear** The cause may be unclear, in which case refer to the medical/ENT team as appropriate.

# Salivary gland problems

Saliva is a mixture containing water, various ions, mucin, and amylase, produced by the parotid, submandibular, and sublingual salivary glands. The problems most commonly affecting the salivary glands are infection and calculous disease.

## Acute bilateral parotitis

Painful swelling of both parotid glands in children is most frequently due to mumps infection (see ➲ Childhood infectious diseases, pp. 230–1). In adults, painless bilateral parotid swelling may be due to Sjögren's syndrome, sarcoidosis, hypothyroidism, lymphoma, and drugs (eg oral contraceptive). In each of these cases, there are often other features, which will help in the diagnosis.

## Acute unilateral parotitis

Painful unilateral parotid swelling may occur as part of mumps infection, but also in other circumstances (eg poor oral hygiene, postoperatively). Refer to an ENT surgeon for admission and IV antibiotics. Chronic painless unilateral parotitis is often neoplastic (mostly benign) in origin.

## Calculous disease

Mechanical obstruction of the flow of saliva is most commonly due to salivary gland stones, usually affecting the submandibular gland, although they sometimes occur in the parotid duct system. Obstruction may also occur from neoplasms or strictures.

### *Features*

Blockage of a salivary duct causes pain and swelling of the affected gland on eating. Bimanual palpation of the floor of the mouth may reveal a stone—occasionally, this may be visible intraorally at the duct orifice. If there is superimposed infection, it may be possible to express pus from the duct.

### *Investigations*

Obtain X-rays of the floor of the mouth. If the patient presses down with the tongue when the X-ray is taken, the stone may be seen more easily below the mandible on a lateral view or OPG.

### *Treatment*

Refer to an oral or ENT surgeon. If an immediate consultation is not available, discuss the use of antibiotics in the meantime (these are often reserved for situations where there is evidence of salivary gland infection).

# Dental anatomy

Primary teeth erupt between 6 months and 2y and are replaced by permanent teeth which first appear at ~6y (see Table 12.3). There are 20 primary and 32 permanent teeth. The permanent teeth are made up of four quadrants of eight teeth. Each quadrant comprises two incisors, one canine, two premolars, and three molars (including the 'wisdom tooth') (see Figs. 12.2 and 12.3).

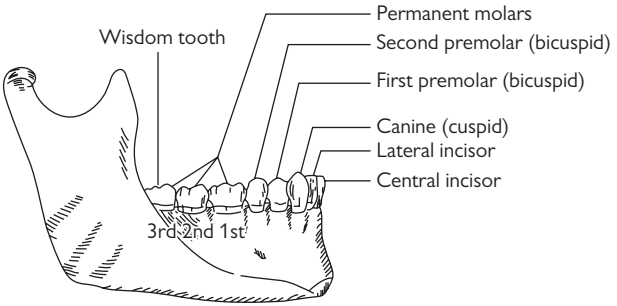


**Fig. 12.2** Dental anatomy: lower jaw lateral view.

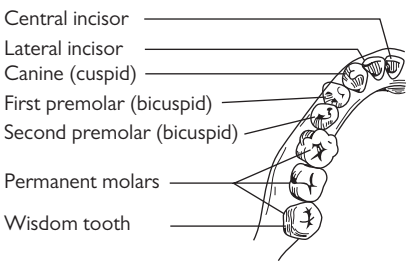


**Fig. 12.3** Dental anatomy: upper jaw—view from below.

**Table 12.3** Tooth eruption

| | Deciduous | Permanent |
|---|---|---|
| *Incisors* | 6–10 months | 7–8y |
| *Canine* | 16–20 months | 11y |
| *Premolars* | | 11–13y |
| *Molars* | 10–24 months | 6–25y |

# Dental emergencies

## Damaged teeth

*Chipped teeth and crowns which have become dislodged* Do not require immediate attention—redirect the patient instead to their dentist. Specialist 'sensitive teeth' toothpaste rubbed over the broken area of tooth may ↓ pain.

*Tooth fractures which involve the pulp* Present with a small area of bleeding and are exquisitely tender to touch. Refer to the on-call dentist.

*Mobile teeth after trauma* Need to be stabilized as soon as possible—advise the patient to avoid manipulating the tooth and to refer to the dentist.

## Simple classification of tooth fractures

(See Fig. 12.4.)

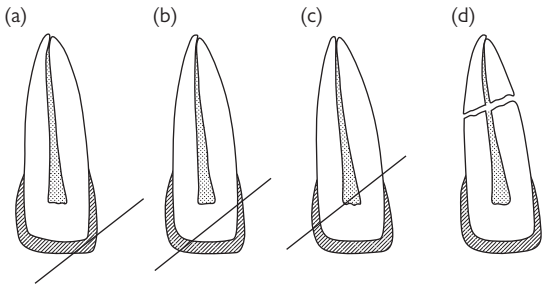


**Fig. 12.4** Simple classification of tooth fractures. (a) Enamel only; (b) enamel and dentine; (c) enamel, dentine, and pulp; and (d) root fracture.

## Avulsed teeth

*Missing teeth* Need to be accounted for (especially in the unconscious patient) in order to exclude the possibility of aspiration. Obtain a CXR to search for both the tooth and secondary problems such as pulmonary collapse and air trapping distal to the obstruction. Ensure adequate tetanus prophylaxis.

*Avulsed permanent teeth* Brought to the ED may be suitable for re-implantation. Avulsed primary teeth are usually not suitable. A history of rheumatic fever, valvular heart disease, or immunosuppressive treatment are contraindications to re-implantation. Milk is the best easily available transport medium to advise a patient to bring a tooth in. The best chance of success lies with early re-implantation (within the first few hours). Handle the tooth as little as possible. Hold it by the crown to clean it gently with 0.9% saline. Orientate the tooth, and then replace it within the socket using firm pressure (this may be easiest after LA—see ➲ Dental anaesthesia, p. 309). Refer immediately to the on-call dentist for stabilization and prophylactic antibiotics (eg clarithromycin). Ensure tetanus prophylaxis.

## Post-extraction problems

*Haemorrhage after tooth extraction* May respond to simple measures. Ask the patient to bite on a rolled-up piece of gauze placed over the socket for 10min. If this is unsuccessful, consider stopping the bleeding by inserting a horizontal mattress suture (eg using 'Vicryl'), placed under LA using lidocaine with adrenaline (see Fig. 12.5). If bleeding continues despite these measures, apply direct pressure; send a coagulation screen, and refer to the on-call dentist.

*Dry socket pain* May follow tooth extraction (typically 3–8 days later) when bone is exposed in the empty socket. Gently irrigate the socket with warm saline. Prescribe oral antibiotics (eg penicillin or erythromycin) and analgesia, and refer to the dentist.

**Fig. 12.5** Horizontal mattress suture in tooth socket.

## Dental infection

Toothache is most often due to dental caries. In the absence of associated local or systemic symptoms/signs, pain usually responds to analgesia (eg ibuprofen 400mg PO tds with food ± paracetamol/codeine as necessary). Add antibiotics (eg penicillin or clarithromycin) if there is suspicion of local infection. Advise follow-up with a dentist.

Toothache with associated swelling, trismus, dysphagia, or systemic evidence of infection requires immediate referral to a maxillofacial surgeon for IV antibiotics and surgical drainage.

## Temporomandibular dysfunction

Chronic pain and/or clicking in a temporomandibular joint is a relatively common problem but rarely presents to the ED. If there is no dislocation/fracture/infection, direct the patient back to the GP to consider elective referral to the maxillofacial team.

# Chapter 13

# Obstetrics and gynaecology

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# Gynaecological problems

## The history

For gynaecological problems, always take a proper gynaecological history. This involves asking personal and sometimes sensitive questions, so privacy and confidentiality are of utmost importance. It is often best to interview the patient without other family members being present.

- Ask about the presenting problem. Always ask about abdominal pain, dyspareunia, and vaginal discharge.
- Take a detailed menstrual history, including the date of the last menstrual period, length of cycle, and description of the bleeding pattern.
- Obtain a full obstetric history, asking about children, pregnancies, miscarriages, terminations, and infertility treatment.
- Remember to ask about sexual activity and the type and number of partners in the past year. Also establish what form of contraception has been used.
- Ask if she has ever been treated for a sexually transmitted infection (STI).
- Find out when her last smear test was and what the result was.
- Consider differentials—ask about bladder and bowel function, plus any previous history of surgery (especially appendicectomy).

## Examination procedure

Prior to performing a vaginal examination, explain the procedure to the patient and ensure you are in a private room. Allow the patient privacy to undress. Examine the patient in an unhurried manner, in the presence of a chaperone, who might usefully 'guard' the door to prevent sudden inadvertent interruption. Use a chaperone even when the patient is being examined by a ♀ member of staff. Document the name of the chaperone in the medical record. Full examination includes inspection of the external genitalia, digital bimanual palpation, and speculum vaginal examination, as well as taking necessary swabs. In certain circumstances (eg patients with painful vulval ulcers), this may not be appropriate in the ED. Vaginal examination in young children may require GA and should be undertaken by an expert. Adopt a low threshold for referring such patients. The appearance of the normal cervical os depends upon parity (see Fig. 13.1).

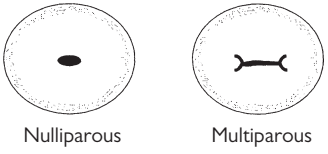



**Fig. 13.1** Appearance of the cervical os.

## Vulvovaginal pain

Distinguish between dysuria, dyspareunia (pain on vaginal penetration), and constant vulvovaginal pain/irritation. The latter is often associated with infection or ulceration. Enquire about other symptoms (abdominal pain, vaginal discharge, and bleeding).

*Vulval ulcers*

- *Herpes simplex virus* is sexually transmitted and usually due to type II virus but is increasingly due to type I virus (responsible for cold sores). Primary infection is extremely painful, lasting up to 3 weeks and sometimes causing urinary retention. Look for shallow yellow vulvovaginal or perineal ulcers with red edges. Cervical ulcers may also be present, although pain may prevent speculum examination. Liaise with the GU team (or the Gynaecology team out of hours) to treat primary infections immediately with aciclovir and analgesia and to exclude coexisting infection. Recurrent infections are less severe but may last up to a week. Treat with topical and oral aciclovir (200mg five times a day for 1 week), and arrange GU follow-up with advice to avoid sexual contact in the meantime. Leave the prescription of aciclovir in pregnancy to specialist teams and arrange for an obstetric opinion—the diagnosis may affect the fetus, as well as the mode of delivery.
- *Other STIs* may cause ulceration: syphilis (non-tender, indurated ulcers ('chancres') and lymphadenopathy), chancroid (painful ulcer caused by the streptobacillus *Haemophilus ducreyi*), lymphogranuloma venereum, and granuloma inguinale (see ➲ Sexually transmitted infections, p. 248). Refer to the GU clinic, and advise to abstain from sexual contact until treated.
- *Squamous carcinoma* causes indurated ulcers with everted edges, especially in the elderly. Refer urgently to gynaecology team.
- *Consider also*: Behçet's syndrome (arthritis, iritis, genital/oral ulceration), TB, and Crohn's disease.

## Painful lumps

- *Bartholin's abscess*: infection of vestibular (Bartholin's) cyst/gland at the posterior part of the labium majus is usually due to staphylococci, streptococci, or *Escherichia coli*, but it may be due to *Neisseria gonorrhoeae*. Refer for incision and drainage and a full GU screen—likely all managed without admission unless there are signs of sepsis.
- *Infected sebaceous cysts*: may also require incision and drainage.
- *Urethral carbuncle*: this small, red, painful swelling at the external urethral meatus is due to urethral mucosal prolapse. It may cause dysuria. Refer to an appropriate clinic to consider excision or diathermy.

## Pruritus vulvae

Vulval irritation may be caused by a generalized pruritic skin disorder (eg eczema), infection (particularly candidiasis), long-term skin conditions (eg lichen sclerosus), other causes of vaginal discharge (see ➲ Vaginal discharge, p. 582), urinary incontinence, threadworms, and vulval warts. Genital warts (including condylomata accuminata) are usually sexually transmitted and caused by human papillomavirus 6. Other STIs may coexist. Refer to the GU clinic.

# Vaginal discharge

May be physiological or due to atrophic vaginitis, infections including STIs, cervical and endometrial carcinoma, a variety of fistulae, and FBs.

## Physiological

A creamy/white discharge is normal. Variation in its consistency and amount occurs with puberty, pregnancy, OCP use, ovulation, and immediately prior to menstruation.

## Atrophic vaginitis

A profuse, sometimes bloody, yellow discharge may result from vaginal epithelial thinning due to ↓ oestrogen levels associated with the menopause. This responds well to local topical or oral oestrogens, most appropriately prescribed by the patient's GP.

## 'Thrush'

*Candida albicans* is a common vaginal infection. A white discharge accompanies a red, painful vulvovaginitis. It occurs in pregnancy, after or whilst taking a course of oral antibiotics, as well as with HIV and diabetes—check for glycosuria. Treatment options include clotrimazole pessaries, oral fluconazole, and topical application of live yoghurt. Advise for GP follow-up of any continuing symptoms or recurrent episodes.

## Bacterial vaginosis

Caused by a variety of organisms, including *Gardnerella vaginalis*. Classically produces a yellow brown offensive ('fishy') discharge. Refer to the GP/GU clinic to consider oral metronidazole.

## Other infections

Refer patients suspected of the following STIs to the GU clinic, and advise abstinence from sexual contact in the meantime:

- *Neisseria gonorrhoeae* may be asymptomatic and causes urethritis (dysuria), cervicitis (classically a green vaginal discharge), or pelvic inflammatory disease (PID—see ➲ Gynaecological pain, pp. 588–9).
- *Trichomonas vaginalis* infection results in a smelly, profuse yellow discharge and a 'cherry red' cervix.
- *Chlamydia trachomatis* causes chronic cervicitis, reactive arthritis, and PID. It may be asymptomatic. ~50% also have gonorrhoea.
- *Syphilis* causes painless genital ulceration ('chancre').

## Cervical and endometrial carcinoma

Classically presenting with bleeding between periods, these may cause discharge (see ➲ Vaginal bleeding, pp. 590–1). Refer to a gynaecologist.

## Fistulae

Colovaginal fistulae may follow diverticulitis or locally invasive colorectal carcinoma. Other fistulae (including vesicovaginal and ureterovaginal) may occur after pelvic surgery. Refer for admission and investigation, ideally to the team that performed the original surgery. Fistulae are sometimes the first presentation of a carcinoma.

## Foreign bodies

Tampons, condoms, and various other items may be 'lost' or forgotten about in the vagina. Removal with forceps under direct vision should cure the offensive vaginal discharge. If a condom has been removed, ascertain whether or not post-coital contraception is required (see ➲ Contraceptive problems, pp. 584–5). Consider hepatitis B/HIV prophylaxis and GU referral for STI screen, depending upon the circumstances.

Vaginal tampons (particularly highly absorbent ones which have been left *in situ* for many hours) are associated with 'toxic shock syndrome' (see ➲ Toxic shock syndrome, p. 583). Discuss with a paediatrician/gynaecologist if any girl under 16y presents with a vaginal FB—GA may be required to remove it.

Occasionally patients with mental health problems present having deliberately inserted a FB into the vagina—take care if the FB is sharp-edged (eg broken glass). Take a psychiatric history and involve the psychiatry team.

## Toxic shock syndrome

Tampons used during menstruation have been implicated in many cases of 'toxic shock syndrome'. First described in 1978, it is caused by exotoxin produced by *Staphylococcus aureus* (usually TSS toxin 1) or occasionally *Streptococcus*. Multi-organ failure may follow.

*Features* High fever, headache, vomiting, diarrhoea, myalgia, altered conscious level, hypotension, and a widespread erythematous macular rash (with subsequent desquamation 1 week later, especially of the palms and soles). There may also be abnormal vaginal bleeding or discharge.

*Examination* Look for clinical evidence of shock. Perform abdominal, bimanual, and speculum examinations—remove any tampon which remains in the vagina.

*Diagnosis* This is based upon clinical findings. Recent menstruation and the above features should prompt suspicion.

*Investigations* Includes vaginal examination. U&E, LFTs, clotting screen, FBC, blood lactate, blood cultures and vaginal swabs, ECG, and CXR.

*Treatment* Manage the patient in the resuscitation room. If due to a tampon, remove it. Follow guidelines for severe sepsis (see ➲ Sepsis, pp. 62–3 and ➲ Shock, pp. 64–5). Obtain venous access: give broad-spectrum IV antibiotics and start crystalloid. If hypotension is refractory, involve the ICU to consider measuring CVP, placing an arterial line, and starting inotropic support.

# Contraceptive problems

## Missed pill

Exact advice depends on the type of OCP the patient takes (combined, combined low-dose oestrogen, or progesterone-only). Refer to the NHS website (https://www.nhs.uk) which gives specific advice for each type of (missed) pill—consider printing this out and give it to the patient. The following is a summary.

*Missed combined pill*

- One missed pill: take it straightaway and continue taking the pack as usual. Emergency contraception is usually not required.
- Two or more missed pills: take one (missed) pill immediately and continue the pack as usual. Use additional contraception for 7 days. If there has been any unprotected sex in the past 7 days, then emergency contraception is likely to be required.

*Progesterone-only pill*

- If the pill is <3hr late (12hr if taking desogestrel), take it as soon as possible, then take the next one as normal. The patient is protected against pregnancy.
- If the pill is >3hr late (>12hr if taking desogestrel), take it as soon as possible, then take the next one as normal. The patient is not protected against pregnancy—advise her to use additional contraception for 2 days. Unprotected sex during this time may require emergency contraception.

If a woman vomits within 2hr of taking any OCP, advise her to take another pill as soon as she is able to.

If a woman has diarrhoea, the OCP will only be less effective if there is severe diarrhoea for >24hr.

## Emergency contraception

Women may attend the ED requesting emergency contraception (sometimes known as 'post-coital contraception') after:

- Isolated unprotected sexual intercourse.
- Burst or lost condom.
- Missed OCP.
- Complete or partial expulsion of intrauterine contraceptive device (IUD).
- Rape.

In the UK, pharmacists can sell the progesterone-only emergency contraception without prescription. This may be the preferred option if the patient presents within 72hr of unprotected intercourse, but it can be given up to 120hr. The risk of pregnancy following unprotected intercourse is greatest during 5 days around ovulation but exists at other times also. Patients given post-coital contraception require assessment and treatment, including counselling and follow-up—usually this will be with the GP and/or family planning clinic. Options include levonorgestrel, ulipristal, and insertion of IUD (see Intrauterine contraceptive device, p. 585).

*General advice for emergency oral contraception*

- Exclude contraindications.
- Advise the patient to return if she vomits shortly after taking the medication—give a replacement dose if vomiting occurs within 2hr of taking it.
- Explain that there is a chance of failure.
- Arrange follow-up (usually with the GP) in 3 weeks to confirm that menstruation has occurred.
- Advise alternative contraception (eg condoms) in the meantime and discuss future contraception plans.
- Discuss (± signpost to) potential use of IUD.
- Document exactly what advice has been given.

*Levonorgestrel (previously called 'the morning-after pill')*

This is most effective if given within 72hr of intercourse (95% effective if taken <12hr, 58% effective at 72hr). After appropriate checks (as above), give levonorgestrel 1.5mg (Levonelle-2®).

*Ulipristal acetate*

This progesterone receptor modulator must be taken within 120hr of intercourse (95% effective if taken <12hr, ~95% effective at 120hr).

Note: hormonal emergency contraception is less effective if the patient is already taking enzyme-inducing drugs—take specialist advice. Options include an IUD or ↑ dose of levonorgestrel to 3mg (see *BNF*). After appropriate checks (as above), give ulipristal acetate 30mg (ellaOne®).

*Intrauterine contraceptive device (IUD)*

This is particularly useful for patients who wish to use IUD long-term and/or for those presenting between 3 and 5 days after unprotected intercourse. Failure is very rare. Insertion is uncomfortable and requires appropriate training—refer to the sexual health team or GP. Exercise caution when considering use of an IUD for a patient at high risk of STI or with symptoms.

## Prescribing to patients on OCP

Both progestogen-only oral contraceptives and (combined) OCP may fail if enzyme-inducing drugs are prescribed. These include: rifampicin, rifabutin, carbamazepine, phenytoin, topiramate, griseofulvin, phenobarbital, and primidone. Patients need alternative or additional contraception if these drugs are started.

*Antibiotics and the OCP*

(Refer to *BNF*.) Latest guidance advises that the only antibiotics that are thought to interact with hormonal contraception are rifampicin-like antibiotics. Rifampicin and rifabutin are such potent enzyme-inducing drugs that contraceptive precautions need to continue for at least 4 weeks, even after a short course of rifabutin or rifampicin (eg as used for prophylaxis of meningococcal infection).

# Genital injury, assault, and female genital mutilation

The history may be misleading. Combine a high index of suspicion with a full examination to exclude significant injury.

*Blunt genital injury* may result from falls astride. Most resultant vulval haematomas settle with rest and ice packs. Refer very large haematomas for evacuation in theatre.

*Penetrating injury* may follow assault, FB insertion, or migration/perforation of an IUD (particularly during insertion). Abdominal pain associated with a vaginal wound may be due to peritonitis. Obtain venous access, an erect chest X-ray (for free gas), an abdominal X-ray (for FB), and group and save; give antibiotics and refer. Refer other vaginal tears without peritonitis for exploration and repair.

## Rape and sexual assault

Rape is defined in the UK as vaginal, anal, or oral penetration by the penis without consent. Rape and other sexual assaults are believed to be grossly under-reported. Those who do report it have special requirements. Privacy is essential—ideally, in a specially equipped room devoted to assessment of women who have been sexually assaulted. Ensure that a ♀ member of staff is present throughout. Document findings legibly and meticulously. Established protocols allow prompt and thorough investigation and treatment. Usually, ED staff provide emergency treatment and resuscitation, but most of the other aspects, including collection of forensic evidence, are managed by a forensic physician (police surgeon), ideally in a specialized Sexual Assault Referral Centre. Sometimes, women initially decline police involvement—full assessment and documentation may prove useful if there is a change of mind.

### *History*

Establish the type, date, time, and place of the assault. Ask what occurred (vaginal/anal penetration, oral sexual activity, other injuries). Ask about contraception use, and enquire about last menstrual period (LMP). Find out what is known about the assailant(s) and their risk of HIV and hepatitis B. In particular, are they injecting drug users, do they originate from sub-Saharan Africa, and are they homosexual?

### *Examination*

Look for evidence of vaginal, oral, or anal injury (the forensic physician will take swabs). Record any other injuries such as bites, bruising, or skin wounds (photographs of non-genital injuries may be useful—taken by the police with the patient's consent).

### *Investigations*

Obtain written informed consent. The police will be keen to retain clothing, loose hairs, fingernail clippings, and tampons for evidence. Similarly, the forensic physician will take appropriate swabs (vaginal, oral, anal). Perform a pregnancy test. Take and store blood for future DNA and viral testing.

*Treatment*

- Resuscitate as necessary. Refer urgently the 1% of patients who have significant genital injuries (eg vaginal tears) requiring surgical intervention.
- Consider the need for emergency (post-coital) contraception (see ➲ Emergency contraception, pp. 584–5).
- If the patient is not immunized, give hepatitis B immunoglobulin and start an accelerated active immunization course (see ➲ Needlestick injury, p. 425).
- Assess the risk of HIV. If the assailant is known to have HIV or is from an at-risk group, discuss the risk of disease transmission with the patient and consider the need for post-exposure prophylaxis (see ➲ Needlestick injury, p. 425).
- Assess tetanus vaccine requirements.
- Arrange follow-up to exclude STI. Consider antibiotic prophylaxis against STI if the patient is unlikely to attend follow-up—liaise with the GU team.
- Provide initial counselling and ensure a safe place to stay (a social worker may arrange this).
- Arrange future counselling. Inform the patient about independent local advice available (eg Rape Crisis Centre).
- Ascertain from the patient if she wishes her GP to be informed.

### Telephone advice

Women may telephone the ED for advice after being raped. Advise them to inform the police immediately and then to attend the police station or the ED. Discourage them from washing, changing clothes, using a toilet, or brushing teeth before being examined.

### Female genital mutilation

Around 137,000 women in the UK are affected by the illegal practice of female genital mutilation (FGM), sometimes known as 'female circumcision'. The term refers to procedures that are performed with the intention of causing injury or alterations to female genital organs for non-medical reasons. Consider FGM if a woman presents with a genital injury. All FGM data are now recorded nationally. Recently updated legislation sets out some important principles (see 🔗 https://www.rcog.org.uk/globalassets/documents/guidelines/gtg-53-fgm.pdf):

- FGM is illegal unless it is a planned surgical operation.
- It is illegal to arrange or assist in arranging a UK resident to be taken overseas for the purpose of FGM.
- It is illegal for those with parental responsibility to fail to protect a girl from FGM.
- If FGM is confirmed in a girl under the age of 18y, it is mandatory to report to the police.

The patient may present with bleeding, infection, or urinary retention.

Clearly document findings and refer to the gynaecology team. Involve the safeguarding team and social services if the patient is a child.

# Gynaecological pain

Gynaecological disorders with abdominal pain may be difficult to distinguish from other disorders. Obtain a full history of the pain—sudden onset of severe colicky pain follows ovarian torsion and acute vascular events; more insidious onset and continuous pain occur in infection and inflammation. Radiation of the pain into the back or legs suggests a gynaecological origin. Other clues in the history include coexisting symptoms of vaginal discharge, vaginal bleeding, or missed LMP.

Abdominal and pelvic pain in early pregnancy may be due to ectopic pregnancy or threatened miscarriage (see ➲ Miscarriage, pp. 598–9)—both occur in patients who do not realize that they are pregnant or who deny the possibility of pregnancy due to embarrassment. Perform a urine pregnancy test on every woman of child-bearing age who presents with abdominal pain.

## Pain related to the menstrual cycle

Consider first—could any associated vaginal bleeding be from an ectopic pregnancy or a threatened miscarriage?

*Physiological dysmenorrhoea* Pain regularly preceding menstruation and peaking on the first day of a period may be physiological. Suggest NSAID and refer to the GP.

*Endometriosis* Growth of functional endometrial tissue in the pelvis outside the uterus may produce cysts and adhesions. Patients often present aged ~30y with dysmenorrhoea and menstrual problems, infertility, and dyspareunia. Symptoms are usually chronic and recurrent in a cyclical fashion and are appropriately followed up by the GP. Occasionally, an endometrial cyst (endometrioma) may rupture and bleed severely into the pelvis, presenting in a similar fashion to a ruptured ectopic pregnancy. Resuscitate for hypovolaemia, and refer urgently for admission and a transvaginal USS ± diagnostic laparoscopy.

*Rupture of a corpus luteum cyst* This occurs premenstrually but may also cause significant haemorrhage, requiring resuscitation and referral.

*Mittelschmerz* Mid-cycle extrusion of an ovum from a follicular cyst can cause abdominal pain, which seldom requires admission or any investigation.

## Uterine problems

*Perforation* Seen especially in the presence of recent IUD/IUS insertion. This is diagnosed on transvaginal USS or X-ray if USS is not available. All intrauterine contraceptives can be detected on X-ray.

*Fibroids ('leiomyomas')* May undergo torsion (sudden, severe colicky pain with a tender uterus) or may infarct ('red degeneration'), particularly during pregnancy. Provide analgesia and refer such suspected problems for specialist investigation.

## Ovarian problems

*Torsion* Causes sudden-onset, sharp unilateral pain and usually involves an already enlarged ovary (cyst, neoplasm, endometrioma). There may be tenderness on abdominal and PV examination. Clinical diagnosis is difficult—if suspected, refer for transvaginal USS with Doppler to assess for ovarian blood supply and free fluid. Provide analgesia as required. Note that torsion can sometimes be partial and managed conservatively, but definitive management is with laparoscopy—let the specialist decide.

*Bleeding into an ovarian cyst* May present similarly and requires investigation if the patient is unstable or has pain which is not controlled.

## Pelvic inflammatory disease

This term includes infection which has spread from the external genitalia to the cervix (cervicitis) to the uterus (endometritis), Fallopian tubes (salpingitis), ovaries (oophoritis), or adjacent peritoneum (peritonitis). Severity ranges from chronic low-grade infection (with relatively mild symptoms) to acute infection (with severe symptoms) which may result in abscess formation.

*Causes* 90% are sexually transmitted—sexually active women aged 15–20y are at particular risk. Most of the remaining 10% follow pregnancy terminations or dilatation and curettage. Note that women undergoing surgical termination of pregnancy are now routinely given prophylactic antibiotics for pelvic infection.

*Organisms* *Chlamydia trachomatis* is the most common cause, with an estimated 50% having a concomitant *Neisseria gonorrhoeae* infection. *Mycoplasma hominis* and *Ureaplasma urealyticum* may also be responsible.

*Features* Bilateral lower abdominal tenderness, vaginal discharge, fever >38°C, abnormal vaginal bleeding, deep dyspareunia, cervical motion tenderness, and adnexal tenderness all point to PID.

*Management*

- *Shocked patients*: resuscitate with IV fluids. Check urinalysis and send high vaginal swab and cervical swab, and blood for FBC, CRP, clotting, and group and save. Try to arrange an urgent transvaginal USS. Refer and start antibiotics (eg ceftriaxone 2g od IV, metronidazole 500mg tds IV, and doxycycline 100mg bd PO).
- *Stable (not shocked) patients*: if PID is suspected, but the patient is stable and well, discharge (after taking swabs) on oral antibiotics (eg ofloxacin 400mg bd PO plus metronidazole 400mg bd PO for 14 days) and refer to the GP for follow-up. If there are any signs of sepsis, refer to the gynaecology team for inpatient management.

If pregnant, discuss the antibiotic regime with an obstetrician.

*Sequelae* Ectopic pregnancy (five times ↑ risk) or infertility—therefore, adopt a low threshold for empirical treatment (see http://www.rcog.org.uk).

# Vaginal bleeding

(See ➲ Vaginal bleeding in pregnancy, pp. 596–7.)

Triage ahead patients with severe bleeding or evidence of hypovolaemia. Resuscitate first ($O_2$, cross-match, and obtain Rh status, start IV fluids) and ask questions later. Most patients with vaginal bleeding, however, do not require resuscitation. Take a careful menstrual history and ask about associated symptoms. Attempt to assess the amount of bleeding. Interpreting a patient's description is notoriously difficult, but useful pointers are clots and the rate of tampon/towel use. Always consider pregnancy—remember that a ruptured ectopic pregnancy can present before a period is missed (see ➲ Ectopic pregnancy, pp. 600–1). Ask about medications—menorrhagia is not uncommon in women who have recently started taking an anticoagulant for other pathology.

Examine for evidence of hypovolaemia and abdominal masses/tenderness. Depending on circumstances, speculum and bimanual vaginal examinations may be required—local policy determines who performs this.

## Menorrhagia

*Dysfunctional uterine bleeding* This is a diagnosis of exclusion. Heavy and/or irregular periods without obvious pelvic pathology may result from hormonal imbalance. It is particularly common at menarche. Most settle without treatment or with simple measures (eg tranexamic acid 1g PO qds plus mefenamic acid 500mg PO tds after food). Refer to the GP, unless bleeding is very heavy, in which case refer to the gynaecology team.

*Uterine fibroids (leiomyomas)* Benign growths of the uterine cavity (usually smooth muscle, but sometimes containing fibrous tissue) often cause menorrhagia. They may present with a painful complication such as torsion or infarction; these are more common in pregnancy—refer.

*Other causes*

- *Endometriosis*, in which endometrial-like tissue is found outside the uterine cavity, is commonly associated with pain and bleeding problems.
- *PID*—see ➲ Pelvic inflammatory disease, p. 589.
- *IUD/IUS*—recent insertion can cause erratic and heavy bleeding, but most women are encouraged to leave the IUD/IUS *in situ* for at least 6 months.
- *Hypothyroidism* is believed to cause menorrhagia as a consequence of dysfunctional uterine bleeding.
- *Coagulation problems*—including von Willebrand's or recent oral anticoagulation.

## Bleeding unrelated to pregnancy or periods

*Trauma* The history may be elusive.

*Postoperative*

Bleeding is a risk of any operation. Resuscitate and refer.

*Hormonal contraception problems*

Endometrial hyperplasia may cause unscheduled bleeding. Exclude vaginal/cervical lesions and refer to the GP for review.

*Cervical ectropion (erosion)*

Occurs due to changes in the epithelium of the cervical canal. It may produce a mucoid discharge with a small amount of post-coital or intermenstrual bleeding. The cervix appears red. Arrange/obtain a cervical smear if due and arrange follow-up.

*Cervical polyp*

These are mostly benign but can cause post-coital or intermenstrual bleeding. Refer to the gynaecologist.

*Cervical cancer*

90% are squamous carcinoma. Strongly associated with human papillomavirus (mainly HPV 16 and 18). Suspect in anyone presenting with post-coital or intermenstrual bleeding. Always ask about smear tests—whether and when she has had them, and if she has ever had an abnormal result and/or been referred to the gynaecology team.

Speculum examination reveals nodules, ulcers, or erosions, which may bleed to touch. Advanced disease may present with pyometra, ureteric obstruction, or retrovaginal fistula. Arrange urgent gynaecology review for any patient with an abnormal-looking cervix.

*Uterine carcinoma*

Mostly adenocarcinoma, >90% of cases are in women aged over 50y. Risk factors include obesity, nulliparity, and late menopause. Classically presents with post-menopausal bleeding, but otherwise normal examination. Arrange urgent assessment for transvaginal USS and diagnostic pipelle biopsy with the gynaecologist.

# The pregnant patient

Pregnant patients presenting with emergency problems create understandable anxiety. There are two patients—one may be suffering unseen. Maintaining fetal oxygenation is crucial—call the obstetrician (or gynaecologist) early (depending on gestation).

## Terminology

The 40 weeks of pregnancy are divided into three trimesters. Traditionally, problems in the first trimester (weeks 1–12) are considered 'gynaecological' and are managed by gynaecologists, and weeks 22–40 by obstetricians.

- *Gravidity* = total number of pregnancies (eg a woman in first pregnancy is a 'primigravida').
- *Parity* = number of pregnancies after 24 weeks + number before (eg a woman who has had one child and two spontaneous miscarriages is described as 1 + 2; gravidity = 3).
- *Miscarriage* is fetal death before 24 weeks; stillbirth is fetal death after 24 weeks.

## Important aspects of history taking

Given the sensitive nature of questions which need to be asked, privacy and confidentiality are of utmost importance. Do not assume that the person with the patient is the partner, nor that the partner is the biological father of the baby. In addition to standard questions, establish:

- Gravidity and parity.
- Whether the pregnancy was natural or assisted (relevant as patients with assisted conception are at higher risk of antenatal complications).
- The expected date of delivery (calculated from LMP or by checking their dating scan).
- What was found at the dating scan (between 8 and 12 weeks) and anomaly scan (~20 weeks).
- Results of antenatal blood tests, particularly Rh status.
- If the patient is multiparous (has had a previous pregnancy), were there any previous antenatal, intrapartum, or postnatal complications.
- Any social concerns, including domestic violence and/or FGM.

## Diagnostic imaging in pregnancy

Try to avoid X-rays and CT scans. Excessive radiation exposure risks congenital malformation, growth retardation, and neoplasia. However, do not withhold necessary X-rays in life-threatening illness. Most head, neck, and extremity X-rays can be obtained without fetal risk by appropriate lead screening. When requesting X-rays, ensure the radiographer is aware the patient is pregnant. USS has not been shown to have adverse effects. If in doubt, discuss imaging requests with a radiologist.

'All pregnant women attending (accident and) emergency departments with anything other than minor complaints should be seen quickly and in conjunction with an obstetrician or senior midwife.' (See https://www.rcog.org.uk)

## Progression of pregnancy

(See also Fig. 13.2.)

Peristalsis and ciliary action carry the fertilized ovum to the uterus, which it reaches as a blastocyst ~5 days after ovulation. The blastocyst implants in the endometrium—the inner part forms the embryo, and the outer part the membranes and the placenta. Trophoblastic tissue produces human chorionic gonadotrophin (HCG), (peaks in the first trimester) acting on the corpus luteum, enabling it to release progesterone and maintain the pregnancy. This is essential until around 12 weeks' gestation when the placenta takes over hormonal control, producing several hormones, including oestrogen and progesterone. Levels of HCG then ↓, whereas oestrogen and progesterone ↑.

### *Symptoms of pregnancy*

Amenorrhoea, breast tenderness and fullness, polyuria, tiredness, nausea (appear by ~6 weeks). Vomiting is common, occasionally severe enough to cause dehydration and weight loss ('hyperemesis gravidarum'—see ➲ Hyperemesis gravidarum, p. 595).

### *Signs of pregnancy*

Not obvious in early pregnancy—uterine enlargement (see Fig. 13.2), breast changes.

### *Pregnancy testing*

See ➲ Pregnancy testing, p. 597.

## Maternal physiological changes

### *Cardiorespiratory*

Cardiac output ↑ by 30%, peripheral vascular resistance ↓, and BP (especially diastolic) ↓ by 10–20mmHg during the first and second trimesters but tends to return to the pre-pregnancy level in the third trimester. Systolic flow murmurs are common. Water retention occurs, which can cause ankle oedema and carpal tunnel syndrome. Tidal volume ↑ and the patient may feel breathless, but the RR does not change.

### *Haemodynamics*

Blood volume ↑ by 30%, plasma volume ↑ by 45%, and Hb ↓ slightly due to the dilutional effect of an ↑ circulatory volume. Platelets ↓, but their function is maintained. Pregnancy is a hypercoagulable state, with most clotting factors ↑ (especially fibrinogen), but clotting times remain unchanged. ↑ pressure in the pelvis may result in varicose veins and haemorrhoids.

### *Gastrointestinal and urinary tracts*

↓ tone of lower oesophageal sphincter predisposes to heartburn; ↓ gut motility can cause constipation. There is a significant ↑ in alkaline phosphatase (maximal in the third trimester) as it is produced by the placenta. Kidney size ↑ by 1cm in length from early pregnancy, with marked dilatation of the renal pelvis until 6 weeks post-partum.

### *Other changes*

- Backache is common and skin pigmentation changes are often seen (eg melasma).
- Platelets, ESR, cholesterol, and fibrinogen ↑; albumin ↓.

(See Table 13.1 for normal values in pregnant and non-pregnant women.)

# Prescribing in pregnancy

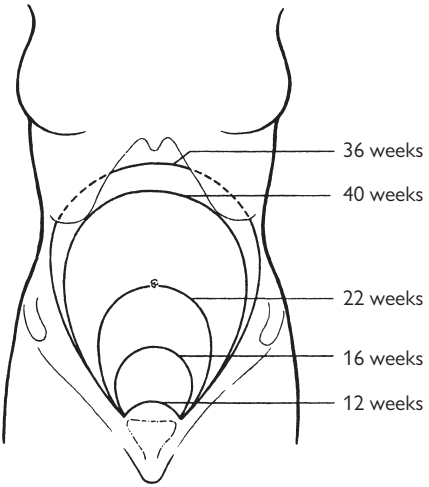



**Fig. 13.2** Uterine size in pregnancy.

**Table 13.1** Normal values in pregnant and non-pregnant women

| Value | Non-pregnant | Pregnant |
|---|---|---|
| Haematocrit | 0.37–0.47 | 0.32–0.41 |
| Haemoglobin (g/L) | 115–160 | 110–150 |
| WCC (/L) | $4.0–11.0 \times 10^9$ | $5.0–16.0 \times 10^9$ |
| Platelets (/L) | $150–400 \times 10^9$ | $134–400 \times 10^9$ |
| ESR (mm/hr) | (age in years + 10)/2 | 44–114 |
| Fibrinogen (g/L) | 2–4 | 4–6 |
| Albumin (g/L) | 35–50 | 28–40 |
| Urea (mmol/L) | 2.5–6.7 | 1.6–6.0 |
| Creatinine (mmol/L) | <110 | 38–90 |
| $pCO_2$ (kPa) | 4.5–6.0 (34–46mmHg) | 3.6–4.2 (27–32mmHg) |
| $pO_2$ (kPa) | >10.6 (>80.6mmHg) | >10.6 (>80.6mmHg) |
| $HCO_3^-$ (mmol/L) | 24–28 | 18–23 |

Consult the *BNF* before prescribing drugs in pregnancy or during breast-feeding. The following are generally considered safe in pregnancy: penicillin, cephalosporins, nystatin, paracetamol, chlorphenamine, and cimetidine.

Avoid: trimethoprim, tetracyclines, streptomycin, warfarin, thiazides, and sodium valproate.

# Hyperemesis gravidarum

## Background

Nausea and vomiting in early pregnancy can be normal and are found in ~70% of pregnancies.

Excessive vomiting causing medical problems, in the form of hyperemesis gravidarum, is relatively rare, affecting ~1 in 1000 pregnancies.

*At-risk groups* are women with a multiple pregnancy or molar pregnancy (due to excessive β-HCG production).

## Clinical features

Persistent vomiting in early pregnancy may be accompanied by dehydration, weight loss, electrolyte disturbances, and/or behavioural changes. Occasionally, vomiting results in haematemesis (Mallory–Weiss tear—see ➲ Upper gastrointestinal bleeding, pp. 126–7).

## Investigations

- Check urine pregnancy test.
- Send blood for FBC, U&E, LFTs, and β-HCG. Note that TFTs can be misleading in hyperemesis—at high levels, β-HCG can stimulate TSH receptors to cause a hyperthyroid picture.
- Transvaginal USS performed under the direction of the specialist team excludes multiple or molar pregnancies (if the patient has not already had this test in this pregnancy).

## Management

- If the patient is not tolerating oral fluids, keep her nil by mouth, gain venous access, and start IV replacement therapy.
- Give IV or IM antiemetics (cyclizine or prochlorperazine are first choices). Second-line options include PO or IM metoclopramide.
- Refer to the gynaecology team for longer-term management (which may include IV hydrocortisone and/or high dose folic acid and thiamine). Red flag signs are: impaired renal function, severe electrolyte disturbance, cognitive impairment or neurological symptoms consistent with Wernicke's encephalopathy or central pontine myelinolysis, and suicidal ideation. Research suggests that the presence of ketonuria does not necessarily correlate with the severity of hyperemesis gravidarum.

## Complications

*Maternal risks* include renal and liver failure, hyponatraemia, hyperkalaemia, and Wernicke's encephalopathy secondary to thiamine deficiency. There is also a risk to mental health from persistent symptoms.

*The fetus* is theoretically at risk of intrauterine growth retardation.

# Vaginal bleeding in pregnancy

Vaginal bleeding in pregnancy causes understandable maternal distress. The likely underlying cause varies according to the stage of pregnancy.

**Table 13.2** Causes of vaginal bleeding in pregnancy

| | Pregnancy-related | | Non-pregnancy-related |
|---|---|---|---|
| First trimester | Miscarriage | At any stage | Infection |
| | Ectopic pregnancy | | Vaginal ulcers |
| | Trophoblastic disease | | Vaginal inflammation |
| | Placental implantation | | Cervical erosions |
| | Chorionic haematoma | | Cervical polyps |
| | | | Coagulation disorders |
| | | | Trauma |
| Second trimester | Miscarriage | | |
| | Trophoblastic disease | | |
| | Placental abruption | | |
| | Placenta praevia | | |
| Third trimester | Placental abruption | | |
| | Placenta praevia | | |
| | 'Show' of pregnancy | | |
| | Vasa praevia | | |

## Anti-D immunoglobulin

A Rh –ve mother exposed to Rh +ve fetal blood during pregnancy may develop antibodies. These IgG antibodies may cross the placenta during subsequent pregnancies and cause Rh haemolytic disease of the (Rh +ve) newborn. The production of maternal antibodies may be prevented by appropriate use of anti-D Ig. Consider this every time that there is possible feto-maternal bleeding (ruptured ectopic pregnancy, spontaneous abortion, trauma, antepartum haemorrhage, labour, and delivery). Guidelines have been produced for the use of anti-D Ig (see https://www.rcog.org.uk). Check the Rh and antibody status of all women with bleeding in pregnancy. If a patient is <12 weeks, only give anti-D Ig (250U IM) to those who are Rh –ve and non-sensitized and have an ectopic pregnancy or uterine evacuation. If the patient is >12 weeks, give anti-D Ig to all women with bleeding (250U if <20 weeks; 500U if >20 weeks). Perform a Kleihauer test—this will give an indication of the extent of any feto-maternal haemorrhage; the blood transfusion service and/or obstetrician will advise.

## Approach to vaginal bleeding in early pregnancy

*Pregnancy testing*

Even if the patient denies pregnancy and there is no history of amenorrhoea, consider pregnancy. Most pregnancy tests look for β-HCG produced by the developing trophoblast. Serum β-HCG levels rapidly ↑ in the first trimester, so that pregnancy may be confirmed by serum tests within days of implantation and remain +ve until 20 weeks (and for 7–14 days post-miscarriage). Urine tests have improved considerably in recent years, but do not rely upon them to definitely exclude pregnancy. Transvaginal USS demonstrates most pregnancies by 5 weeks' gestation.

*Miscarriage or implantation bleeding—when to refer*

Many women attend the ED with bleeding in early pregnancy. Assume that bleeding in early pregnancy is from a miscarriage until proved otherwise. Try to establish the extent of the blood loss—this will guide management. Find out if the patient has had a scan in this pregnancy, as this can help to rule out trophoblastic disease and ectopic pregnancy. In some pregnancies, the woman may have been told after a scan that the pregnancy is likely to fail.

Bleeding in early pregnancy can occur normally when the embryo implants to the uterine wall at ~7–8weeks ('implantation bleed'). If the patient has normal vital signs, with a normal Hb and no abdominal pain, consider sending them home with a referral to the local early pregnancy unit for a review in the next 24hr. If the patient is unstable and has abdominal pain or continuing bleeding, then resuscitate as appropriate and refer to the gynaecology team.

*When to perform a speculum examination*

A patient with altered vital signs and ongoing bleeding is at risk of developing cervical shock. Cervical shock presents as hypotension with reflex bradycardia due to the products of conception passing partially through, and becoming stuck in, an open cervical os. In this case, perform a speculum examination in the ED as soon as IV access has been obtained. Removing the products usually resolves the shock. Ensure that the speculum examination is performed in a private examination room with a chaperone present. Once the speculum is inserted into the vagina, aim to visualize the whole cervix. Use sterile gauze on the end of sponge-holding forceps to swab away any blood in the vagina to see if there is active bleeding. If products are seen in the os, remove them with the sponge-holding forceps, then refer to the gynaecology team.

## Disposal of fetal products of conception

The Human Tissue Authority has issued guidance for England, Wales, and Northern Ireland (there is different guidance in Scotland) on the disposal of products of conception of any pregnancy tissue prior to 24 completed weeks (see https://www.hta.gov.uk).

Women have options regarding the disposal of their pregnancy remains, including cremation, burial, or incineration. Liaise with the gynaecology team for advice.

# Miscarriage

## Terminology

Use the term 'miscarriage' (not 'spontaneous abortion') with patients. Both refer to fetal loss before 24 weeks. *Spontaneous miscarriage* is common and affects >20% of pregnancies. *Threatened miscarriage* refers to vaginal bleeding through a closed cervical os; 50% proceed to miscarry. If the cervix dilates or products of conception are passed, miscarriage is inevitable. *Inevitable miscarriage* becomes *complete miscarriage* if all products are passed (as confirmed by transvaginal USS). Retained products of conception is an *incomplete miscarriage* which may become infected, causing a *septic miscarriage*. Alternatively, products may be retained as a *missed miscarriage*, which carries a risk of DIC.

## Aetiology

Mothers may feel guilty, but the causes are largely beyond their control. Risk factors include:

- Chromosomal anomalies (>50%).
- First pregnancy, maternal disease, and age >30y.
- Uterine abnormalities.
- Drugs (especially teratogens such as isotretinoin).
- Cervical incompetence, immunological factors, and trauma.

## Approach

Establish the gestation. Think—is this a ruptured ectopic pregnancy? Vaginal bleeding in spontaneous miscarriage ranges from spotting to flooding. Severe bleeding with hypovolaemia may occur in inevitable miscarriage. Abdominal pain is associated with a lower chance of fetal survival. Any pain with threatened miscarriage tends to be light and crampy. Severe pain and bleeding with hypotension and bradycardia implies 'cervical shock' (see ➲ When to perform a speculum examination, p. 597). Vaginal examination provides other important clues—look for cervical dilatation (the external os of a multigravida usually accepts a fingertip) and products in the os. Cervical tenderness suggests an alternative diagnosis (ectopic pregnancy, septic miscarriage, or PID).

## Investigations

Transvaginal USS may exclude ectopic pregnancy and indicate fetal viability—local policy determines who performs this. Urine pregnancy tests remain +ve for several days/weeks after fetal death. Check Rh status and baseline serum β-HCG. Cross-match and obtain FBC if shocked.

## Treatment

Resuscitate if significant pain or haemorrhage, and refer urgently. If cervical shock is present, remove products of conception from the cervical os using sponge forceps. If severe bleeding continues, give ergometrine 500mcg IM. Unfortunately, no intervention appears to alter fetal survival in threatened miscarriage. Patients with light bleeding, no abdominal pain, and a closed os (threatened miscarriage) may be allowed home after USS and gynaecology review. Reassure, emphasize that it is not her fault, and advise bed rest and abstinence from sexual intercourse until gynaecology follow-up in 2 days. Provide Rh anti-D Ig 250U IM if Rh –ve and non-immune.

### Septic miscarriage

Sepsis may follow spontaneous, surgically induced, or 'backstreet' abortion.

*Organisms* *Staphylococcus aureus, Clostridium welchii, Bacteroides, Escherichia coli*, streptococci, *Clostridium sordelli*.

*Features* Vaginal bleeding, offensive discharge, ↑ T°, ↓ BP, uterine tenderness, cervical excitation, peritonitis. Note that pyrexia is not invariable—particularly with *C. sordelli* which can result in a severe infection with high mortality.

*Obtain* FBC, clotting screen, blood cultures, blood lactate, vaginal swabs, cross-match, Rh status, erect CXR (to look for free gas).

*Resuscitate* With IV fluids, give co-amoxiclav 1.2g IV; follow the severe sepsis guidelines (see ➲ Sepsis, pp. 62–3 and ➲ Shock, pp. 64–5), and refer urgently. Monitor urine output and consider central and arterial lines.

### Missed miscarriage

Very occasionally presents several weeks or months after fetal death with no expected features of pregnancy, a negative pregnancy test and DIC. Resuscitate, and involve senior obstetrician and haematologist.

### Recurrent miscarriage

If a patient has had three or more consecutive spontaneous miscarriages in the first trimester with the same biological father, then her GP will arrange referral for fertility investigations.

### Retained products of conception

This relatively common gynaecological problem classically presents, following conservative or medical management of miscarriage, but can also occur after normal delivery or surgical management of miscarriage. Women often present with persistent bleeding and a positive pregnancy test (due to the presence of trophoblastic tissue retained in the uterus).

*Features* Vaginal bleeding ongoing for ≥3 weeks post-delivery, offensive discharge, ↑ T°, ↓ BP, uterine tenderness, cervical excitation, +ve pregnancy test.

*Obtain* FBC, group and save including Rh status. If there are signs of sepsis, then also take blood for U&E, CRP, and clotting screen.

*Management* Most women are managed in an outpatient setting, with involvement of the gynaecology team—a transvaginal USS will confirm the diagnosis and help to determine management. Most patients are treated by surgical evacuation of retained products of conception, although some may be managed conservatively or medically.

Start oral antibiotics (co-amoxiclav 625mg tds is safe in breastfeeding) if there is clinical concern about an infection. If there is evidence of sepsis from infected retained products, resuscitate and start IV antibiotics (eg co-amoxiclav 1.2g IV tds), and refer for inpatient treatment.

# Ectopic pregnancy

Gestational sac implantation outside the uterus is the main differential diagnosis for any pregnant woman with abdominal pain who has not had a scan to confirm an intrauterine pregnancy. Its incidence has ↑ and now occurs in 1–2% of all pregnancies in the UK. 96% implant in the Fallopian tube, 2% in the interstitial part of the uterus, 1.5% intra-abdominally, and the remainder in the ovary, previous scars, or cervix. The risk of heterotopic pregnancy (combined intrauterine and ectopic pregnancy) is ~1 in 4000.

## Importance

Ectopic pregnancy is the most common cause of maternal mortality in the first trimester. The diagnosis is frequently missed. Consider it in any young woman presenting with abdominal pain or vaginal bleeding, especially when combined with an episode of syncope.

Although many women with an ectopic pregnancy can be reviewed by the gynaecology team in a timely manner, a proportion are at risk of deterioration and need emergency surgery.

## Risk factors

These include anything which delays or limits normal transit of the fertilized ovum to the uterus: PID, pelvic surgery/adhesions, previous ectopic, endometriosis, assisted fertilization, IUD/IUS, progesterone-only pill, congenital anatomical variants, and ovarian and uterine cysts/tumours. Note that although pregnancy is unusual after tubal ligation, when it does occur, there is a relatively high chance (~1 in 6) of it being an ectopic pregnancy.

## Pathology

Implantation of a gestational sac in the Fallopian tube may have three results:

- Extrusion (tubal abortion) into the peritoneal cavity.
- Spontaneous involution of pregnancy.
- Rupture through the tube, causing pain and bleeding.

Implantation in a uterine horn is particularly dangerous—pregnancy may reach 10–14 weeks before rupture. Exceptionally, intraperitoneal pregnancies may proceed almost to term.

## Symptoms

Ectopic pregnancy may present with sudden, severe lower abdominal pain with collapse or fainting and vaginal bleeding. There is usually (but not always) a history of amenorrhoea. Haemorrhage may cause shoulder tip pain (from blood irritating the diaphragm) and features of hypovolaemia. Nausea and vomiting are common.

Many patients have more chronic symptoms, with recurrent abdominal pain and slight irregular vaginal bleeding, which may be fresh or dark (like 'prune juice'). Pain may have continued for >1 week before presentation, occasionally as long as 4 weeks. The pain may be worse on defecation. Some patients have no vaginal bleeding.

Enquire about symptoms of pregnancy (eg breast tenderness) and possible risk factors for ectopic pregnancy.

### Signs

Look for hypovolaemic shock, and if present, ensure that volume replacement accompanies full assessment. Abdominal tenderness is variable, ranging from mild to severe with peritonism. Cullen's sign (discoloration around the umbilicus) is of historical interest only. Bimanual vaginal examination reveals tender adnexae, and sometimes a mass, but may be better deferred to a specialist. Speculum inspection may show vaginal bleeding.

### Investigations

Must not delay resuscitation and referral.

*Pregnancy test* This is almost always +ve, but serum β-HCG levels are usually lower than expected for normal pregnancy.

*Transabdominal USS* This is useful if it demonstrates an intrauterine pregnancy, free fluid in the pouch of Douglas, and/or an adnexal mass. Frequently, it is inconclusive. Transvaginal USS is the gold standard for pelvic imaging.

### Differential diagnosis

- *Threatened miscarriage*: bleeding is usually more severe and can present with cervical shock (see ➲ Miscarriage, pp. 598–9).
- *Ruptured corpus luteum cyst*: the corpus luteum supports pregnancy for the first 6–8 weeks. Rupture causes sudden peritoneal irritation but rarely bleeds significantly.
- *PID* (see ➲ Pelvic inflammatory disease, p. 589): note that ectopic pregnancy can cause mild pyrexia and a raised WCC, which may easily be misinterpreted as evidence of pelvic infection.
- *Trophoblastic disease* (see ➲ Vaginal bleeding in later pregnancy, pp. 602–3).
- *Acute appendicitis* (see ➲ Acute appendicitis, p. 523).

### Treatment

Give $O_2$ as required; insert two large (12 or 14G) venous cannulae, and cross-match 6U of blood. Request Rh and antibody status—anti-D Ig may be needed (see ➲ Anti-D immunoglobulin, p. 596). Resuscitate initially with crystalloid IV fluids as necessary. Keep the patient nil by mouth. If ectopic pregnancy is suspected, refer urgently to the gynaecology team since sudden deterioration may occur. Significant haemorrhage requires urgent surgery. Alert the anaesthetist and theatre team early.

Patients who are not haemodynamically compromised are sometimes treated medically (eg with methotrexate), rather than with surgery.

# Vaginal bleeding in later pregnancy

Vaginal bleeding in the second or third trimester may indicate serious illness which threatens the life of both fetus and mother—refer urgently to the obstetrician. See Table 13.2 for causes of bleeding in pregnancy. Note that *antepartum haemorrhage* is defined as bleeding from the genital tract in pregnancy of ≥24 weeks' gestation, before the onset of labour.

## Key points

- Do not perform a speculum or digital examination until placenta praevia has been ruled out.
- Attempt to estimate the amount of bleeding (remembering concealed abruption).
- Try to establish if a fetal heart can be heard.
- Remember non-pregnancy causes of bleeding.
- Refer early to the obstetric team.

## Gestational trophoblastic disease

Occasionally, a fertilized ovum may form abnormal trophoblastic tissue, but no fetus. The pathological spectrum ranges from benign hydatidiform mole to invasive choriocarcinoma. Choriocarcinoma is relatively rare, affecting ~1 in 40,000 pregnancies. Trophoblastic disease is often diagnosed at the dating scan (at around 8 weeks).

*Presentation* Usually vaginal bleeding at 12–16 weeks, with passage of tissue, which may resemble frogspawn. Often accompanying abdominal pain and sometimes pre-eclampsia or eclampsia. The uterus may be much larger than expected for dates. DIC may occur.

*Investigations* USS shows 'snowstorm' and no fetus. Serum HCG is grossly ↑. Note β-HCG can be ↑ in multiple pregnancies.

*Management* Obtain venous access and blood for serum β-HCG, FBC, group and save; give IV fluids/resuscitation, and refer to gynaecology.

## Placental abruption

Premature separation of the normally situated placenta affects ~1% of pregnancies. It causes haemorrhage which may risk the fetus, depending on the extent of placental involvement and rapidity of separation.

*Risk factors* Pre-eclampsia, previous abruption, trauma (see ➲ Trauma in pregnancy, pp. 612–13), smoking, ↑ parity, cocaine.

*Presentation* In 80% of cases, there is some vaginal bleeding ('revealed haemorrhage'), but occasionally bleeding is limited to the confines of the uterus ('concealed haemorrhage'). In either case, there may be much more utero-placental bleeding than is immediately apparent. There may be abdominal pain and tenderness or back pain. Placental abruption may precipitate labour. A large bleed can cause DIC and fetal demise.

### Placenta praevia

The placenta is situated wholly or partly over the lower uterine segment and cervical os in ~1% of pregnancies. If a patient has a low-lying placenta at her anomaly scan (~20 weeks), a third trimester scan will be arranged by the obstetric team to assess the placental site prior to the onset of labour. Delivery is likely to be by Caesarean section if the placental edge is <2mm from the internal os. Major placenta praevia is defined as when the placenta overlies the os.

*Risk factors* Mother aged >35y, high parity, previous placenta praevia, twins, uterine abnormalities (including previous Caesarean section).

*Presentation* Most present with bright red, painless vaginal bleeding in the third trimester; 15% present in labour.

### Vasa praevia

Rarely, an abnormal fetal blood vessel may be attached to the membranes over the internal os, below the presenting fetal part.

*Risk factors* Multiple pregnancy, low-lying placenta, *in vitro* (IVF) pregnancy.

*Presentation* Often presents following rupture of membranes with massive vaginal bleeding, which may cause fetal exsanguination.

### Management of antepartum haemorrhage

- Call an obstetrician immediately and admit to hospital.
- Give $O_2$ and keep nil by mouth.
- Obtain venous access (two large-bore cannulae), and resuscitate with IV fluids as necessary.
- Send blood for U&E, FBC, blood glucose, cross-match, Rh and antibody status, Kleihauer test, and clotting screen.
- Monitor the fetus (cardiotocography or Doppler).
- USS locates the placenta, demonstrates the fetus, and may show concealed haemorrhage.
- Give anti-D Ig as advised by the blood transfusion service if Rh –ve (see ➲ Vaginal bleeding in pregnancy, pp. 596–7).

### Intrapartum bleeding

Heavy bleeding in labour is most commonly due to intrapartum abruption, placental problems, or uterine rupture. Secure venous access, resuscitate, and get expert help (obstetrician, anaesthetist, neonatologist).

### Post-partum haemorrhage

*Primary post-partum bleeding* >500mL from the genital tract within 24hr of delivery is usually due to uterine atony or genital tract trauma, but can be caused by retained products of conception or a coagulopathy.

*Secondary post-partum bleeding* Defined as occuring between 24hr and 6 weeks post-partum. It is most commonly due to infection (endometritis) or retained products of conception. Resuscitate as necessary and get expert help (obstetrician, anaesthetist). If there is any suspicion of sepsis, start IV antibiotics (eg co-amoxiclav 1.2g tds).

# Abdominal pain in pregnancy

## Approach

Attempting to deduce the cause of abdominal pain can ordinarily be quite difficult—in pregnancy, it is even more so. Some possible underlying diseases may be causing unseen fetal distress and can produce rapid maternal deterioration. Therefore, triage ahead, contact the obstetrician, and resuscitate vigorously. Initial investigations usually include BMG, urinalysis, blood tests (including β-HCG in early pregnancy), and USS. Vaginal bleeding accompanying abdominal pain implies a gynaecological or obstetric problem. Remember, however, that the reverse is not necessarily true—a ruptured ectopic pregnancy and concealed haemorrhage in placental abruption may present without vaginal bleeding. In later pregnancy, even if there is doubt as to whether the principal problem is obstetric or not, involve the obstetrician at an early stage.

## Pregnancy-related causes of abdominal pain

The following are considered elsewhere:

- Ectopic pregnancy (see ➲ Ectopic pregnancy, pp. 600–1).
- Miscarriage (see ➲ Miscarriage, pp. 598–9)
- 'Red degeneration' of a fibroid (see ➲ Uterine problems, p. 588).
- Gestational trophoblastic disease (see ➲ Vaginal bleeding in later pregnancy, pp. 602–3).
- Placental abruption (see ➲ Vaginal bleeding in later pregnancy, pp. 602–3).
- Onset of labour (see ➲ Emergency normal delivery, pp. 608–9).

### *Torsion, rupture, or haemorrhage into an ovarian cyst*

This may involve the corpus luteum of pregnancy or a pre-existing cyst. Sudden-onset lower abdominal pain results. Transvaginal or abdominal USS may demonstrate the problem. Refer to the obstetrician—management depends on cyst size, nature, and clinical state (typically as an outpatient if the cyst is <5cm and the patient is stable with minimal pain).

### *Acute polyhydramnios*

Excessive amniotic fluid may complicate pregnancy involving uni-ovular twins or a singleton pregnancy. Pain and vomiting are accompanied by a large abdomen for gestation and an unusually mobile fetus. Refer to obstetrics (who will check for fetal abnormality, infections, including CMV, or fetal macrosomia).

### *Pre-eclampsia*

Abdominal pain (particularly right upper quadrant pain) in pregnancy may reflect pre-eclampsia (see ➲ Medical complications of pregnancy, pp. 606–7). Check BP and urinalysis and refer urgently.

### *Obstetric cholestasis*

Abdominal pain (particularly epigastric or right hypochondrial) in pregnancy can reflect obstetric cholestasis, especially if associated with pruritus, but no rash. Check BP, urinalysis, and bloods (FBC, LFTs, bile acids, clotting), and refer urgently to an obstetrician.

## Non-obstetric causes of abdominal pain

*Urinary tract infection/pyelonephritis*

UTI is relatively common in pregnancy due to urinary stasis. Women are at particular risk if they have had previous UTI. Abdominal/loin pain and pyrexia with rigors indicate acute pyelonephritis. Send MSU, FBC, and blood cultures, and refer for IV antibiotics. Treat patients with mild UTI or cystitis without evidence of pyelonephritis with oral antibiotics (eg cefalexin 500mg PO tds), and arrange GP follow-up when the MSU result will be available. When prescribing antibiotics in pregnancy, take care to avoid those drugs which are contraindicated (eg trimethoprim, tetracyclines—see *BNF*).

*Acute appendicitis*

Presentation in early pregnancy may be as classically described but can be confused with ectopic pregnancy or rupture/torsion of an ovarian cyst. In later pregnancy, the point of maximal tenderness in acute appendicitis rises towards the right hypochondrium. Check BMG, serum amylase, and urinalysis. Give analgesia and refer if suspected. Remember that it is normal to have a slightly raised WCC in pregnancy, but not a raised CRP.

*Gallstones*

Pain from gallstones not infrequently presents for the first time in pregnancy due to ↑ stasis. The presentation of biliary colic and cholecystitis is similar to that in the non-pregnant patient (see ➲ Biliary tract problems, p. 526). USS reveals stones and associated pathology. Give analgesia and refer (usually to the general surgeons, with involvement of the obstetrician)—if possible, the patient will be treated conservatively initially and operated on in the postnatal period if necessary.

*Acute pancreatitis*

This is usually related to gallstones. There is a significant risk to mother and fetus. Presentation and treatment are as described on ➲ Acute pancreatitis, pp. 524–5. Involve both surgical and obstetric teams.

*Perforated peptic ulcer*

If suspected, obtain erect CXR with a lead shield for the fetus. Resuscitate and refer to the surgical team (see ➲ Peptic ulcer disease, p. 527).

*Intestinal obstruction*

Often follows adhesions from previous surgery. The diagnosis may not be immediately obvious—pain, vomiting, and constipation may be initially attributed to pregnancy. These symptoms, together with abdominal tenderness and high-pitched bowel sounds, suggest the diagnosis. An abdominal X-ray will confirm it, but this should only be requested by a specialist.

# Medical complications of pregnancy

## Pre-eclampsia

This poorly understood vasospastic utero-placental disorder affects ~5% of pregnancies. It results in widespread systemic disturbance involving the liver, the kidneys, and the coagulation and cardiovascular systems. Placental infarcts may occur and compromise the fetus.

Pre-eclampsia is diagnosed as two or more of: hypertension (>140/90), proteinuria, and oedema.

*Variant presentation*: haemolysis, elevated LFTs, low platelets (the 'HELLP syndrome').

*Risk factors* Previous pre-eclampsia, aged <18y or >40y, multiple pregnancy, ↑ BMI, primiparity, pre-existing medical problems (such as hypertension), diabetes, or renal disease.

*Symptoms* Frontal headache, right upper quadrant abdominal pain, visual disturbance, oedema, nausea and vomiting.

*Management*

- Refer all patients with BP >140/90mmHg or proteinuria and oedema.
- Obtain FBC, U&E, LFTs, uric acid, clotting screen, ECG, and fetal monitoring.
- Restrict fluids to a total of 80mL/hr or 1mL/kg/hr (because of the risk of pulmonary oedema).

Progression to eclampsia (see ➲ Eclampsia, p. 611) is heralded by: confusion, headache, tremor, twitching, and ↑ reflexes. Visual disturbance and/or abdominal pain may occur.

## Diabetes mellitus

Pregnancy encourages hyperglycaemia. Women are screened for gestational diabetes in pregnancy—risk factors include previous gestational diabetes, a family history of diabetes, BMI >30kg/m$^2$, and a previous baby with macrosomia.

Type 1 diabetes in pregnancy may be more difficult to control and is associated with an ↑ insulin requirement. DKA occurs relatively easily (see ➲ Hyperglycaemic crises, pp. 160–1).

## Disseminated intravascular coagulation

DIC may complicate a variety of obstetric problems: placental abruption, intrauterine death, missed abortion, amniotic fluid embolism, eclampsia, sepsis, and trophoblastic disease.

*Clinical picture* Widespread haemorrhage and microvascular occlusion.

*Obtain* FBC, cross-match, clotting screen, fibrin degradation products, fibrinogen, U&E, and LFTs.

*Treatment* Resuscitate with $O_2$, IV fluids (according to CVP), blood transfusion, and FFP. Refer urgently and consider urgent delivery and treatment of underlying disease. Involve the haematologist.

## Thromboembolic disease

VTE is one of the leading causes of maternal mortality.

*Risk factors* Caesarean section, previous DVT/PE, thrombophilia, family history of DVT/PE, and bed rest.

*Diagnosis* Clinical probability scoring for DVT or PE is difficult as all derived scores exclude pregnant women. Therefore, use imaging, rather than relying upon clinical probability assessment, combined with D-dimer (as levels ↑ during pregnancy).

*DVT in pregnancy* Presents similarly to that in a non-pregnant woman, with unilateral leg swelling and tenderness (see ➲ Deep vein thrombosis, pp. 122–3). USS is the safest initial investigation for DVT. Remember that to exclude DVT with USS requires either one normal complete scan (calf, popliteal fossa, and thigh) or two normal thigh and popliteal scans, 1 week apart (see ➲ Deep vein thrombosis, pp. 122–3).

*PE in pregnancy* Presents with pain or dyspnoea (see ➲ Pulmonary embolism, pp. 124–5). Unfortunately, these are not infrequent symptoms during pregnancy. A normal $SpO_2$ on air will not exclude PE. Investigation for PE starts with *bilateral leg USS* (no risk to fetus). If these are normal, request CXR (with lead shield covering the abdomen) and V/Q scan or CTPA (see ➲ Pulmonary embolism, pp. 124–5). A CXR may identify other diagnoses, eg pneumothorax.

*Management of DVT/PE* During investigation for DVT or PE, commence treatment with LMWH. Liaise with the obstetric team, as the dose of LMWH is often different in pregnancy.

Thrombolysis has been used successfully in peri-arrest pregnant women with a clear clinical picture of PE. If the patient is not in peri-arrest, always endeavour to obtain diagnostic imaging, given the risk that thrombolysis poses to the fetus.

Warfarin is teratogenic in the first trimester and may cause fetal or placental bleeding in later pregnancy, so avoid it in pregnancy. Note that rarely, in special circumstances, warfarin is prescribed by experts in pregnancy—see *BNF*.

## Other problems

Women with pre-existing medical conditions such as epilepsy or thyroid or cardiac disease are usually managed from pre-conception and have clear plans from the maternal medicine team (found in their pregnancy notes).

If a pregnant woman with complex medical history presents to the ED, contact the obstetric team (who may have easy access to relevant maternity records).

Thyrotoxicosis presents not infrequently in pregnancy. Pre-existing heart disease worsens as blood volume and cardiac output ↑—involve a specialist early. Although rare, consider aortic dissection in any pregnant patient with unexplained severe chest, back, or neck pain (see ➲ Aortic dissection, pp. 96–7).

# Emergency normal delivery

Sometimes even the best laid plans for controlled delivery on the labour ward go awry and patients present in an advanced stage of labour and deliver in the ED. This is most likely in very rapid ('precipitate') labour.

## Labour

At onset of labour, painless and irregular (Braxton Hicks) contractions are replaced by painful uterine contractions, with cervical dilatation (>3cm) ± 'show' (mucus/blood discharge). There may be rupture of membranes.

## Presentation

In the ED, only 'OA' (occiput anterior) vertex presentations are likely to proceed so fast that delivery occurs before specialist help arrives.

## Stages of labour

*First* Onset of labour until cervix is fully dilated (10cm). Usually lasts >6hr. The upper part or 'segment' of the uterus contracts, and the lower segment (including the cervix) dilates. Contractions ↑ in frequency (every 2min) and duration (last 1min). The head starts to descend.

*Second* Full dilatation until the baby is born. Should last <3hr in primigravida, and <2hr in multigravida. Contraction of the upper segment, abdominal muscles, and diaphragm cause the head to descend, then to rotate (usually to lie OA). An overwhelming desire to push helps expel the baby.

*Third* The placenta and membranes deliver and the uterus contracts.

## Assessment of a patient in labour

Check pulse and BP, and palpate the abdomen. Listen for fetal heart sounds with a fetal stethoscope or Doppler probe (rate should be 110–160/min). Gently examine the perineum. Do not fully examine the vagina unless the head is crowning and birth is imminent. Instead, transfer to the labour ward.

## Management of delivery

(See Fig. 13.3.)

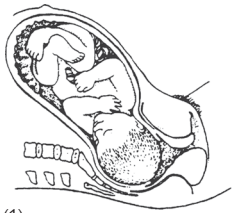
- Call obstetric/neonatology/anaesthetic help, and encourage the partner to stay.
- Don sterile gloves; stand on the patient's right, and offer Entonox®.
- As the head crowns, discourage bearing down—advise rapid shallow breaths.
- Use the left hand to control head escaping (to prevent perineal tearing).
- Press gently forwards, with the right thumb and fingers on either side of the anus.
- Once the head is delivered, allow it to extend.
- Feel for the cord around the neck—slip it over the head or, if impossible, clamp and divide.
- Allow the anterior shoulder to deliver first (with mother pushing if needed).
- Deliver the baby; wrap him/her up, and resuscitate as necessary.

## Management of the cord

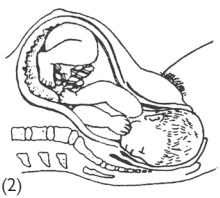
Once the baby cries and cord pulsation ceases, hold the baby level with the mother and clamp the cord twice (15cm from the umbilicus). Divide between clamps. Place a plastic Hollister crushing clamp 1–2cm from the umbilicus and cut 1cm distally. Check that two normal arteries and one vein are present in the cord.

## Management of the third stage

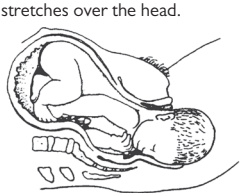
After the cord is cut, give oxytocin 5U IM plus ergometrine (Syntometrine®) 500mcg IM, unless there has been any maternal hypertension at any stage, in which case omit ergometrine (contraindicated due to risk of maternal stroke). A few minutes after delivery, regular contractions begin again, causing the placenta to detach. The cord may move down, accompanied by a small gush of blood. The Brandt–Andrews technique helps removal—pull gently down on the cord whilst exerting upward pressure on the uterus (preventing inversion). Give Rh anti-D Ig if Rh –ve (see ➲ Vaginal bleeding in pregnancy, pp. 596–7).



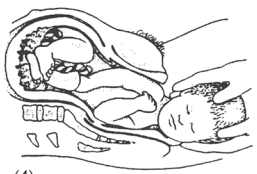
(1)
First stage of labour. The cervix dilates. After full dilatation, the head flexes further and descends further into the pelvis.



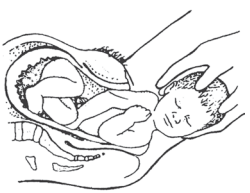
(2)
During early second stage, the head rotates at the level of the ischial spine, so the occiput lies in the anterior part of the pelvis. In late second stage, the head broaches the vulval ring (crowning) and the perineum stretches over the head.



(3)
The head is born. The shoulders still lie transversely in the mid pelvis.



(4)
Birth of the anterior shoulder. The shoulders rotate to lie in the antero-posterior diameter of the pelvic outlet. The head rotates externally. Downward and backward traction of the head by the birth attendant aids delivery of the anterior shoulder.



(5)
Birth of the posterior shoulder is aided by lifting the head upwards whilst maintaining traction.

**Fig. 13.3** Management of delivery.

# Obstetric emergencies

Emergencies around the time of delivery are a very definite domain of the obstetric team—involve them as soon as possible. The following emergencies are included for the sake of completeness and to cover the instance of an obstetrician not being immediately available.

## Imminent perineal tear

The risk of perineal tearing may be minimized by controlled delivery. An extensive tear risks the integrity of the external (or even internal) anal sphincter. If a tear is imminent, perform an episiotomy (see Fig. 13.4). Infiltrate 10–20mL of 1% lidocaine postero-laterally from the posterior fourchette. Cut the perineal tissues postero-laterally, using straight scissors with blunt points (see Fig. 13.4), avoiding large veins. After delivery, carefully examine the episiotomy wound, which needs to be closed in layers using absorbable (usually continuous) sutures.

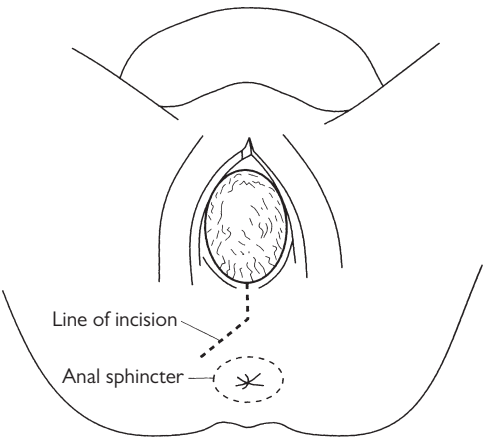



**Fig. 13.4** Performing an episiotomy.

## Cord prolapse

The situation where the umbilical cord lies below ('prolapsed') the presenting part of the fetus is an obstetric emergency demanding urgent attention and an emergency Caesarean section. Untreated, it can result in cord compression and vasospasm, which can risk the life of the fetus.

### *Management*

Whilst waiting for the obstetric team, minimize handling of the cord (this causes further vasospasm and fetal compromise). Place the woman on all fours in a knee-to-chest position, with her head against the bed and her back up in the air. Try to carefully reduce the cord into the vagina, and keep it in place with a warm swab to prevent further expulsion.

### Meconium-stained liquor

(See ➲ CPR of the newborn, pp. 660–1.)

### Difficulty in delivering the shoulders (shoulder dystocia)

After delivery of the head, the shoulders usually rotate to lie in an AP direction, so the first one can be delivered anteriorly. Failure of this rotation is known as shoulder dystocia.

*Maternal complications* Post-partum haemorrhage, genital tract trauma.

*Fetal complications* Hypoxia, intracranial haemorrhage, fracture, brachial plexus injury.

*Management* Request emergency obstetric help. Place the woman's legs into the McRobert's position (hyperflex the hips, so the knees are up towards the woman's ears); apply gentle suprapubic pressure in order to rotate the shoulder (having first established which side the fetal back is on). If these measures fail, then the obstetric team will attempt internal manoeuvres to deliver an arm/shoulder (eg hooking a finger into the axilla of the anterior shoulder of the fetus to bring it down).

### Eclampsia

(See also ➲ Pre-eclampsia, p. 606.)

Eclampsia is defined as the onset of fits with pre-eclampsia after 20 weeks' gestation. It is a serious condition, with maternal mortality of 2% and perinatal mortality of 15%.

*Management*

(See 🖱 http://www.rcog.org.uk)

- If there is evidence of impending eclampsia or the patient starts to fit—call the obstetrician and anaesthetist.
- Check BMG.
- Control the airway.
- Consider moving the patient into the left lateral position.
- Give magnesium sulfate 4g slowly IV over 10min, followed by maintenance magnesium sulfate 1g/hr IVI for 24hr.
- Treat recurrent fits with further magnesium sulfate 2g IV over 10min.
- Follow local advice regarding control of hypertension (eg labetalol 10mg slow IV bolus, followed by an IVI, starting at 1–2mg/min, ↑ as required).
- Urgent delivery is a priority in eclampsia (if the woman is still pregnant) both for mother and fetus.

# Trauma in pregnancy

## Background

The principal causes are similar to those in the non-pregnant woman: road traffic collisions, falls, and assaults. Contrary to popular opinion, the use of seat belts does ↓ the risk of serious injury in pregnancy. The 'lap' belt should lie over the anterior superior iliac spines.

## Anatomical considerations

The following are worthy of consideration:

- As the uterus enlarges, it rises out of the pelvis with the bladder—both are at ↑ risk of injury.
- The size of the uterus and stretching of the peritoneum make abdominal assessment difficult.
- The bony pelvis is less prone to fracture, but retroperitoneal haemorrhage may be torrential due to ↑ vascularity.
- The pregnant uterus may obstruct the inferior vena cava, causing supine hypotension and ↑ bleeding from lower limb wounds.
- The diaphragm is higher in pregnancy.
- The pituitary doubles in size and is at risk of infarction in untreated hypovolaemic shock.

## Physiological considerations

Pregnancy is associated with dramatic changes in physiology:

- Pregnant patients may tolerate up to 35% loss of blood volume before manifesting classic signs of hypovolaemic shock, largely at the risk of the utero-placental circulation.
- ↓ functional residual capacity and ↑ $O_2$ requirement result in hypoxia developing more quickly.
- There is an ↑ risk of regurgitation of gastric contents.
- Coagulation may be deranged or rapidly become so.

## Injuries to the uterus, placenta, and fetus

*Fetal injury* Both blunt and penetrating trauma may damage the fetus. It is, however, more likely to suffer as a result of maternal hypoxia/hypovolaemia or placental abruption.

*Placental abruption* Deceleration forces in blunt trauma may shear the inelastic placenta from the elastic uterus. Haemorrhage (maternal and fetal) may be significant and result in DIC. This may present with vaginal bleeding (much may be concealed internally), uterine tenderness, or fetal distress.

*Uterine rupture* This is relatively uncommon. Major rupture causes severe bleeding. The uterus and fetus may be felt separately.

*Amniotic fluid embolism* Rare and carries a poor prognosis. Presents with sudden collapse, dyspnoea, ↓ BP, fitting, and bleeding (from DIC).

## Approach to the injured pregnant patient

Follow that outlined in ➲ Major trauma, pp. 329–407, with additional specific points.

*History*

Determine gestation and any problems in this and previous pregnancies.

*Examination*

- Involve an obstetrician early—examine the vagina for bleeding or rupture of membranes.
- Palpate for fundal height (mark the skin), abdominal tenderness, and uterine contractions.
- Listen for fetal heart sounds and the rate using a fetal stethoscope (Pinard) or Doppler probe.
- Remember that head injury may mimic eclampsia, and vice versa.

*Investigations*

- Check BMG, coagulation screen, Rh/antibody status, and Kleihauer test.
- Consider CVP monitoring (remembering CVP is lower in pregnancy).
- Monitor the fetal heart (cardiotocograph)—the rate should be 110–160/min.
- USS investigates fetal viability, placental injury, gestational age, and free peritoneal fluid.
- Do not withhold essential X-rays and CTs, but do consider early USS to look for free intra-abdominal fluid and fetal viability. Seek senior advice. Remember that the greatest risks from X-rays to the fetus are in early pregnancy. In later pregnancy, risks to the fetus may be outweighed by failure to identify injuries by not obtaining X-rays.
- DPL has been superseded by USS (FAST scan) (see ➲ Focussed assessment with sonography for trauma (FAST) scan, p. 355).

*Treatment*

- Give $O_2$ and summon senior obstetric, ICU, and surgical help early.
- If chest drains are required, insert 1–2 intercostal spaces higher than usual.
- Decompress the inferior vena cava by manually displacing the uterus to the left or by using a 15° right lateral (Cardiff) wedge (or where possible, tilt the trolley/bed), or if neck injury has been excluded, by nursing in the left lateral position.
- Treat fluid losses with aggressive IV fluid replacement.
- An NG tube ↓ the risk of regurgitation and aspiration.
- Remember tetanus prophylaxis (see ➲ Tetanus prophylaxis, p. 424).
- Consider anti-D Ig if the patient is Rh –ve.
- Even if there is no overt maternal injury, refer for fetal monitoring for 4hr.
- Abdominal tenderness, hypovolaemia, or fetal distress may require urgent laparotomy.
- If the patient has a cardiac arrest, perform an emergency Caesarean section if the patient is >24 weeks pregnant and 5min have elapsed without output (see ➲ Cardiac arrest in pregnancy, pp. 614–15).

# Cardiac arrest in pregnancy

## Rate

Estimated in late pregnancy at ~1 in 30,000.

## Causes

- *Obstetric*: massive obstetric haemorrhage, sepsis, pre-eclampsia/eclampsia, cardiac failure (from cardiomyopathy of pregnancy), amniotic fluid embolism.
- *Medical/surgical*: stroke, PE, anaesthetic problems and drug reactions, underlying heart disease. IHD is rarely implicated—the underlying rhythm is more commonly PEA than VF—unfortunately, this is reflected in the poor prognosis.

## Remember the following physiological factors

- The airway is difficult to control (large breasts, full dentition, neck oedema, and obesity). Ventilation may be difficult, and intubation technically challenging.
- ↑ aspiration risk (↓ lower oesophageal pressure, ↑ intragastric pressure); therefore, securing a definitive airway early is essential.
- ↑ $O_2$ requirements, yet harder to ventilate (↓ chest compliance).
- Chest compression is awkward (flared ribs, raised diaphragm, obesity, breast hypertrophy).
- Gravid uterus >20 weeks compresses the inferior vena cava, ↓ venous return.
- There are two patients: mother and fetus.

## Approach to resuscitation

Follow the resuscitation guidelines for managing adult cardiac arrest (see ➲ Cardiac arrest, p. 48). The special situation of pregnancy means some additional points apply. If there is advanced warning, think ahead. In addition to the usual team needed for airway control, IV access, and chest compressions, organize:

- An anaesthetist for the airway, an obstetrician to perform a Caesarean section, and a neonatologist to resuscitate the baby.
- The neonatal resuscitation equipment (overhead warmer, suction, airway equipment, and $O_2$).
- A member of staff to apply cricoid pressure at the beginning of resuscitation and until the airway is secured.
- A member of staff to manually displace the uterus to the left (a Cardiff wedge is not helpful in this situation).

It may take time for help to arrive and there may be no warning prior to patient arrival. In the meantime:

- Call the obstetrician, neonatologist, and ED consultant immediately.
- Apply cricoid pressure (Sellick manoeuvre) at the beginning of resuscitation and until the airway is secured.
- Try to secure the airway with a cuffed tracheal tube at an early stage.
- Decompress the inferior vena cava by manual displacement of the uterus to the left.
- Consider and treat the cause (remember that hypovolaemic shock from unseen haemorrhage may respond to a large IV fluid challenge).
- If there is no return of spontaneous circulation within 5min, perform a Caesarean section (if the patient is >24 weeks pregnant).

## Emergency Caesarean section

*Rationale*

After several minutes of maternal cardiac arrest, the best chance of survival for the fetus is to be removed from the now hostile hypoxic environment of the uterus. A Caesarean section also benefits the mother by decompressing the inferior vena cava, resulting in ↑ venous return. It has not yet been established whether it is beneficial to deliver a fetus before 24 weeks—there are less obvious haemodynamic benefits (and minimal aortocaval compression at this time).

*Procedure*

- Continue closed chest compression and ventilation throughout the procedure.
- Make a midline skin incision from the pubic symphysis to the epigastrium (level of the fundus of the uterus).
- Carefully dissect through the anterior abdominal wall and peritoneum, taking care to avoid the bladder or bowel.
- Incise the underlying uterus vertically, starting 6cm above the bladder peritoneal reflection. Continue the uterine incision upwards to the fundus, through an anteriorly placed placenta if necessary. Speed is essential.
- Deliver the baby, holding his/her head down and below the level of the mother's abdomen.
- Clamp and cut the umbilical cord.
- Resuscitate the baby (see ➲ Resuscitation of the newborn, p. 658).
- Close the uterus and abdominal wall to avoid hypovolaemia.

# Post-partum problems

## Physiology of the puerperium

Within 24hr of delivery, uterine involution results in the fundus being level with the umbilicus. By 2 weeks, the uterus should be impalpable. Uterine discharge ('lochia') gradually ↓ but may last up to 6 weeks. An initially bloody discharge becomes yellow within 2 weeks. The external cervical os gradually closes, so that after 1 week, it no longer accepts a finger. Speculum examination now reveals the typical parous os (see Fig. 13.1).

## Post-partum haemorrhage

(See ➲ Post-partum haemorrhage, p. 603.)

## Pyrexia

Treat according to the underlying cause, which includes: pelvic infection (see below), UTI, mastitis, DVT, retained products of conception (see ➲ Retained products of conception, p. 599), and other causes unrelated to pregnancy or delivery.

## Pelvic infection

Involves a significant threat—may be complicated by septicaemia, necrotizing fasciitis, DIC, or septic PE. There is an ↑ risk with: surgical procedures in labour, prolonged membrane rupture, internal fetal monitoring, and repeated examinations.

*Features* Uterine tenderness and subinvolution, pyrexia, offensive lochia, peritonitis.

*Send* Vaginal swabs for culture, FBC, group and save, clotting screen, and blood cultures.

*Resuscitate with* $O_2$ and IV fluids, and refer. For septic shock, follow severe sepsis guidelines; give IV co-amoxiclav (1.2g) and IV metronidazole (500mg); monitor CVP, and consider inotropes and ventilation.

## Infected episiotomy wound

Refer to obstetrician.

## Mastitis and breast abscess

Mastitis is commonly due to *Staphylococcus* or *Streptococcus*. Send milk for culture, and commence oral antibiotics (eg co-amoxiclav 625mg PO tds). Instruct the patient to continue to breastfeed from both breasts. Arrange GP follow-up.

Refer patients with abscesses for surgical review.

## Psychiatric illness

Rapid hormonal swings are responsible for elation being frequently replaced by tearfulness and anxiety ('fourth day blues'). Less commonly (0.5% pregnancies), puerperal psychosis occurs. Those with a previous psychotic illness are at particular risk and should be known to the maternity teams. Exclude sepsis, and refer for psychiatric help. The patient may need to be compulsorily detained (see ➲ Compulsory hospitalization, p. 644).

## Thromboembolic disease

A major cause of maternal mortality throughout pregnancy and the puerperium. Adopt a high index of suspicion, and refer for investigation (see ➲ Pulmonary embolism, pp. 124–5).

# Chapter 14

# Psychiatry

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# Approach to psychiatric problems

Psychiatric presentations comprise ~1–2% of ED new attendances. These patients are sometimes considered unwelcome because they are seen as complex, heavy consumers of staff time and energy, and not infrequently exhibit aggressive and/or disturbed behaviour. A careful systematic approach to patients presenting with psychiatric emergencies produces an accurate diagnosis in most cases. If this is not possible, the information gained will at least assist referral to the appropriate service, allowing management of the problem.

## Liaison psychiatry service

Most EDs have close links with liaison psychiatrists and specialist psychiatric nurses. These individuals are often embedded in the department, working both in and out of hours. They are expert at managing a variety of psychiatric problems, including overdose and physical self-harm.

## Potential points of conflict

The ED is not the ideal environment for the assessment of potential psychiatric illness. Bear the following in mind:

- The vast majority of aggressive, violent, or bizarrely behaving patients in the ED are not suffering from a formal psychiatric illness. Many require input from the police, rather than psychiatric services.
- Admission is not mandatory simply because a psychiatric illness has been diagnosed.
- The presence of alcohol or drug intoxication makes assessment of mental state very difficult and, in many cases, impossible—do not assume that this, in itself, reflects an acute psychiatric problem.
- Acute alcohol withdrawal is a medical emergency, with significant mortality—refer to the medical team, not to the psychiatric service.
- Delirium (acute confusional state—see ➲ Acute confusional state (delirium), pp. 140–1) is usually organic, rather than psychiatric, in origin; investigate with this in mind.
- An emergency Section form must be signed by the examining doctor, but this does not have to be a psychiatrist.

Similarly, *psychiatric staff within the ED* need to consider the following:

- EDs are under pressure to manage large numbers of patients in a timely fashion, so it may be difficult for ED staff to spend large amounts of time with any single patient.
- Overcrowding and unavailability of appropriate interview facilities may make it necessary to compromise patient privacy, rather than the safety of staff.
- A psychiatric referral can be appropriate in a patient who has consumed alcohol, if there is a significant psychiatric history ('dual diagnosis' or 'co-occurring disorders').

## General approach to psychiatric problems

Adopt the same approach of history taking and examination as with other general medical problems. Do not dismiss psychiatric patients as 'mad, and therefore, the psychiatrist can sort them out'—this can result in misdiagnosis and inappropriate referral.

# Glossary of psychiatric terms

**Concrete thinking** Impairment of abstract or symbolic thinking (eg interpretation of proverbs, explanation of similes).

**Delirium** An organic syndrome characterized by rapid-onset global disturbance of cognition and disturbed consciousness.

**Delusion** A firm, usually false, belief unshakeable by logical argument or contrary experiences and which is out of keeping with the patient's social or cultural norms.

**Flight of ideas** Thoughts rapidly cycled, linked by chains of ideas or verbal associations or sounds resulting in disjointed or, in extreme cases, incomprehensible speech.

**Hallucination** A false perception not due to a sensory distortion or misinterpretation, but which occurs at the same time as real perceptions. Hallucinations can occur in each of the sensory modalities. Auditory hallucinations are most commonly associated with psychiatric illness. Visual and other hallucinatory phenomena suggest organic aetiology.

**Ideas of reference** A feeling that others are talking about, or looking at, the patient for some reason. Insight is usually retained, which is not the case in delusions of reference.

**Obsession** Recurrent, persistent, and intrusive thoughts, impulses, or mental images that the individual usually tries to resist, finds unpleasant, and recognizes as senseless.

**Passivity** An experience of being under external control either physically, emotionally, or intellectually. Suggests schizophrenia.

**Perseveration** Repetition of an idea, thought, speech, or an action beyond the point of relevance (eg giving the answer of an initial question in response to subsequent unrelated questions). Usually caused by organic brain disease.

**Pressure of speech** Rapid or hurried speech, often occurs with flight of ideas.

**Psychosis** Extreme disorders of thinking and perception, often involving delusions and hallucinations, with loss of insight.

**Thought blocking** A feature of schizophrenia in which a train of thought stops abruptly and, following a pause, a new line of conversation begins.

**Thought broadcasting** More than simply feeling others can read personal thoughts. An experience of thoughts spilling out beyond personal control or that thoughts are being relayed from external sources.

**Thought insertion** Thoughts that are not the patient's own are put in their mind from outside.

**Thought withdrawal** The feeling that thoughts have been removed or stolen by an external influence.

# The psychiatric interview

Interview usually follows initial clinical contact at triage (see ➲ Australian Mental Triage Scale, p. 628), which will have established the basic background to the presentation. In addition, triage notes will include an initial assessment of the level of distress of the patient and record the appearance of the patient/clothes worn in case they leave the department and need to be searched for.

## Setting and safety

Conduct the interview in a quiet, relatively private, and specially designed setting. Most EDs have specific consultation rooms (with an alarm, two doors that open out/both ways, safe comfy furniture which cannot be used as a weapon/missile, and no ligature points). Whatever the state of the department, never allow the need for privacy to compromise staff safety! Make sure other staff are easily available and can be summoned *immediately* if necessary. If this is not possible, either conduct the interview within the main ED (in a cubicle or side-room) and/or ensure that other staff are present during the interview. During the interview, position yourself between the patient and the exit.

## Approach in the initial interview

- Listen supportively and obtain a history of the presenting problem.
- Assess the mental state, emotions, and attitudes of the patient—with particular reference to potential risk (to themselves or others).
- Make a formulation (identify key factors of the illness, list probable causes, consider why the patient became ill, and plan treatment).

*Initial history* Take a rapid, thorough history, concentrating upon the following:

- What is the presenting complaint?
- What factors have caused the patient to present here and now?
- Is there a past history of psychiatric illness or medication?
- What does the patient want (advice, treatment, or admission)?
- Are the patient's wishes appropriate?

## Ethnic minorities

Be aware of the different communities living in the area, and remain sensitive to their needs. Assessment of mental health problems needs to take into account the relevant cultural and religious issues.

*Language* It can be a real challenge to assess the mental health of patients who do not speak English. Consider the following solutions:

- Assessment may be performed by an ED or mental health professional who speaks the patient's language (the ideal result).
- A health professional from another discipline may act as interpreter.
- An interpreter who is not a health professional but who is trained in mental health issues may be used.

Do not use children as interpreters for patients with mental health problems. Similarly, try not to rely upon family members to interpret.

## Taking a full psychiatric history

The main components of a psychiatric history are described below. A detailed psychiatric history is often taken in the ED by the psychiatry liaison service, rather than ED doctors.

*Presenting complaint* List the principal complaints, and try to detail the course and severity of each. Ask about the effect of each problem on the person's life and work. Carefully determine how he came to be referred or why he presented here and now. When was he last well?

*Past psychiatric history* Ask about previous psychiatric or physical illness, hospital admissions (particularly if compulsory), and any outpatient contact (eg community psychiatric nurse), day hospital, day centres, or crisis intervention groups. Record psychiatric or other medications.

*Personal and family history* Obtain an outline of the patient's life history: birth, childhood, circumstances of upbringing (including parental relationships—marital disharmony, separation, violence, adoption, single parent, brought up by a grandparent, etc.). Ask about education, academic achievements, and relationships with family or friends. Ask if there has been any recent bereavement and what effect this has had.

*Work history* Is the patient employed? If not, ask about any previous jobs. Ask about the impact of any loss, change, or failure in work on the patient's life or mental status, and conversely determine if psychiatric or other illness has had any effect on employment.

*Sexual/marital history* Gently enquire about relationships and sexual experiences only where relevant. This may reveal important information about the patient's personality and relationships to others. It may form a major part of the presenting complaint (eg recent ending or change in a relationship or a history of sexual abuse). A more detailed account of sexual aberration or fantasy may be required in a forensic examination.

*Substance misuse* Try to estimate alcohol, tobacco, drug, or other substance misuse by the patient. Although it may be difficult to obtain accurate information, do not assume that patients always underestimate their consumption of such substances.

*Forensic history* Record any previous criminal charges, convictions, or contact with the police, including the dates on which they occurred. Ask if the patient has any present charges or court actions pending against him.

*Social circumstances* Determine where the patient lives and if he shares accommodation with others. Enquire about income and how he is coping financially. Ask if there are any dependants or any outstanding debts, and if he is receiving any form of social support or monetary assistance.

*Personality* Try to describe the patient's usual and present mood. How does he feel about himself and about other people? How does he enjoy himself and how does he react to good, bad, or stressful events?

*Corroboration* Extremely important information can be gathered from close relatives, GPs, community, or social services, which can verify or enhance information obtained directly from the patient.

# Mental state examination

Having taken an appropriately thorough history, make an assessment of the patient's mental state. If the patient is violent, disturbed, or, for some other reason, unable to provide background history, then the information or observations gathered whilst assessing the mental state become even more crucial to diagnosis.

### Appearance and behaviour

Gather information from the moment the interview begins. Is the patient appropriately dressed? Is he clean and tidy, or neglected? Does his general posture, body movement, and facial expression suggest fear, anxiety, aggression, withdrawal, detachment, or low mood? Does he maintain eye contact? Does he respond appropriately to external stimuli or is he easily distracted? Does he appear to be hallucinating or responding to no obvious stimuli? Are there any abnormal movements, tics, grimaces, or dystonic movements? Note whether behaviour is steady and consistent, or labile and unpredictable.

### Speech

Describe the rate, volume, intonation, and spontaneity of speech. Note the presence of dysarthria or dysphasia. Record any examples of invented new words (neologisms), unusual phrases, perseveration, or garbled speech verbatim. Note vagueness, over-preciseness, or sudden switching to new themes or subjects (flight of ideas).

### Mood

Taking cues from the appearance and behaviour, enquire about the patient's prevailing mood, opinion of himself, and view of the future. Enquire about suicidal thoughts and thoughts of harm to others. Ask about disturbances in sleep, appetite, libido, concentration, and mood variations during a typical day. Ask about irritability or memory disturbance (particularly of short-term memory).

### Thought abnormalities

Record these as they are found during the interview (eg thought blocking or flight of ideas). Test for concrete thinking by asking the patient to interpret a simple proverb. Ideas of reference or persecutory delusions may require direct enquiry to be revealed (eg asking about neighbours, electrical devices). Similarly, passivity phenomena may require specific questioning to be elicited (eg 'Is anyone making you think or move without you wanting to?').

### Hallucinations/perceptive abnormalities

Record the presence of any hallucinations, including their nature and specific content. Visual, olfactory, gustatory, and tactile hallucinations should prompt suspicion of organic, rather than psychiatric, disease.

### Insight and mental capacity

Does he believe he is ill? Does he think he requires treatment, and would he be willing to accept it? Does he have mental capacity (see ➲ Mental health assessment issues, p. 631)?

### Cognitive assessment

Although the psychiatric interview will, in general, reveal information about a patient's cognitive abilities, a formal evaluation of higher mental function is essential. Failure to do this can lead to organic brain disease being falsely labelled as a 'functional' or purely psychiatric illness, resulting in inappropriate treatment. Assess the following:

- Level of consciousness (eg alert, hyperalert, withdrawn, or comatose).
- Orientation.
- Attention and concentration.
- Registration of new information.
- Recall of recent and distant memories.
- Ability to interpret instructions and carry out tasks.

### The Mini-Mental State Examination

The Mini-Mental State Examination was designed as a screening tool for the assessment of cognitive function in the elderly. It is in widespread use, but note that, as with many psychological tests, it is subject to copyright.

### Assessment of risk

Consider whether the patient and/or others are at any risk of harm. Ask if the patient has any thoughts of self-harm and/or harm to other individuals. Establish if there is any past history of self-harm or violence. Try to decide if the patient is at risk of abuse/neglect, and consider whether he may be a 'vulnerable adult'—such concerns should trigger a Safeguarding Alert.

*Children at risk*

Find out if there are any children in the patient's household and, if so, whether or not there are satisfactory arrangements in place to care for them. Concerns should prompt consideration of involvement of social services and/or child protection referral.

### Physical examination

A physical examination completes any psychiatric evaluation. Specifically check for evidence of those physical illnesses which can be associated with psychiatric disturbance (eg thyroid disease, substance withdrawal, head injury, epilepsy, cerebrovascular disease, or other intracranial pathology). Carefully examine for focal neurological signs, meningism, organic confusional states, intoxication, and injury. In acute psychological disturbance, perform and record the following basic observations and investigations (this may prove to be very difficult in violent or aggressive individuals):

- Baseline pulse, RR, BP, and $SpO_2$.
- T°.
- BMG/blood glucose.
- Urinalysis.
- Breath alcohol (if available).

Undertake other investigations, such as U&E, FBC, blood alcohol level, CXR, or CT scanning, if clinically relevant. Urine drug screening, TFTs, or electroencephalogram (EEG) may be indicated in some situations, but the results are rarely available acutely.

# The aggressive patient: background

A significant (albeit small) proportion of patients exhibit aggressive behaviour towards staff (and others) who are attempting to help them. Sometimes this amounts to physical violence. All ED staff need appropriate training in this area, bearing in mind that recognition and prevention of aggression is just as important as knowing how to manage it when it occurs.

## Underlying causes

### *Medical illness*

Recognize that a patient's agitation and/or aggression may reflect an underlying treatable acute medical condition. Such conditions may be compounded (as well as being potentially caused) by the use of *alcohol and/or illicit drugs*:

- Hypoglycaemia (see ➲ Hypoglycaemia, pp. 158–9).
- Head injury.
- Hypoxia (any cause).
- Distended bladder.
- Post-ictal confusional states (epilepsy or drug overdose).
- Organic brain syndromes (eg delirium/acute confusional states—see ➲ Acute confusional states (delirium), pp. 140–1).

### *Psychiatric illness*

Most violent, aggressive, or bizarre patients in the ED are not mentally ill. Violence resulting directly from psychiatric illness, which needs urgent treatment, is relatively uncommon. It is restricted to a small number of patients and tends to be associated with the following:

- A past history of violent behaviour.
- Schizophrenia and other psychoses (eg mania or paranoid disorders), especially when there are delusions or hallucinations that focus upon one particular individual.
- Personality disorder, particularly sociopathic, impulsive, or explosive disorders.
- Learning disability.

## Warning signs of impending violence

Violent episodes can often be predicted and prevented. The experienced practitioner may be able to spot the signs of approaching trouble at an earlier stage. Warning signs include the following:

- Angry facial expressions, gestures, and posture (aggressive body language).
- Restlessness, overt irritation, discontentment, pacing about, overarousal (dilated pupils, tachycardia, ↑ RR).
- Prolonged eye contact.
- Loud speech and changes in tone of voice.
- Verbally threatening and/or reporting feelings of anger/violence.
- Repeating behaviour which has previously preceded violent episodes.
- Blocking escape routes.

# Safe consultations with potentially violent patients

## Planning before the consultation

*Physical design issues*

Most EDs have specially designed facilities (eg interview room door designed to open outwards in order to allow rapid, easy exit). Regard any loose items as potential weapons (eg telephones, chairs, lamps).

*Safety first*

Safety comes first—ensure that patients are not allowed to harm themselves, other patients, or staff. Aim to conduct the consultation in a quiet, comfortable, and preferably non-clinical area. However, compromise privacy, rather than safety, so if there are concerns, it may be necessary to undertake the consultation in a standard cubicle. Consider having another member of staff present during the consultation. Before consulting with any potentially violent patient, ensure the following:

- Other staff know where you are and with who you are.
- You know how to get help (a 'panic button' or other personal alarm).
- Staff know to respond immediately.
- Staff know what to do if there is a problem.

*Information gathering*

Get as much information as possible beforehand. Useful sources include relatives, police, social services, GP, and other health professionals.

## The consultation

The outcome of the consultation depends heavily upon how it is conducted:

- Ensure that your own body language does not provoke the situation.
- Remain calm and sympathetic, maintaining a reassuring and non-judgemental manner.
- Listen to any immediate complaint or grievance, with minimum interruption.
- Engage in conversation, with continuing reassurances that you are there to help.
- Adopt an attentive, but relaxed, posture.
- Speak slowly and clearly, keeping your voice low.
- Avoid excessive eye contact.
- Sit between the patient and the door. Do not directly face the patient (this may appear confrontational and provides a larger target).
- Do not turn your back on the patient, especially when leaving the room.

## Documentation and debrief following any violent episode

After any episode of verbal aggression or physical violence, make detailed notes, complete local incident forms, and arrange a debrief. Report it to a senior member of staff ± the police (as appropriate). Subsequently, when dealing with the same patient, do not purposely avoid him or treat him obviously differently, as this will merely emphasize concepts of his own unacceptability and may lead to further aggression.

# Managing aggression

Violent behaviour is unusual if a calm, sensible approach is followed. If violence does occur, focus upon preventing the patient from harming other patients/relatives, staff, or themselves (see https://www.nice.org.uk).

## Approach to the aggressive patient

Get immediate help from police/security officers and other staff. Avoid physical confrontation. Position yourself so as not to block escape. Take note of where the alarm buttons are. Continue de-escalation techniques.

- Find out what the problem is, establish a rapport, and encourage reasoning.
- Show concern and stay attentive.
- Avoid patronizing comments. Never insult the patient or make promises or commitments that cannot be kept.
- Remember that direct body contact can be misinterpreted.
- Do not engage in prolonged eye contact.
- Bear in mind that psychotic patients have different perceptions of personal space and may feel threatened by staff coming into what would otherwise be a normal and non-threatening distance.
- Try to maintain a calm atmosphere with a non-critical, non-domineering approach.

## Management of physical violence

If physical violence occurs, safety of staff, other patients, and relatives takes priority. Concern for property is secondary—it can be replaced. Even during a violent act, a calm approach with talking and listening often prevents escalation of the event and the need for physical confrontation.

*Physical restraint* Avoid physical intervention, if at all possible. Where physical restraint is required, use the minimum degree of force, applied for the minimum length of time (ideally <10min) in order to control the episode. Apply it in a manner that attempts to calm, rather than provoke, further aggression. This will require sufficient members of staff to control the event without injury to anyone involved.

Restrain by holding clothing, rather than limbs. If limbs have to be grasped, hold near a major joint to reduce leverage and the possibility of fracture or dislocation. Remove the patient's shoes or boots. In exceptional circumstances (eg when a patient is biting), the hair may have to be held firmly. Never apply pressure to the neck, throat, chest, back, or abdomen, and do not deliberately inflict pain. If the patient has to be placed on the floor, the supine position is preferable to the prone position.

Do not attempt restraint unless sufficient staff/expertise is available. Put one person in charge to ensure airway and breathing are not compromised and vital signs are monitored. Only ↓ restraint once it is certain that the risk has ↓—this may mean use of medication.

*Weapons* Ask for any weapon to be placed in a 'neutral' position, rather than handed over. Do not attempt to remove it from an aggressor.

# Emergency tranquillization

Pharmacological restraint using sedative drugs is a last resort only used on the advice of senior and experienced staff. Emergency sedation carries significant dangers. Sedative drugs may mask important signs of underlying illness, eg an intracranial haematoma requiring urgent treatment. Normal protective reflexes (including airway reflexes such as gag and cough response) will be suppressed. Respiratory depression and the need for tracheal intubation and IPPV may develop. Adverse cardiovascular events (eg hypotension and arrhythmias) may be provoked, particularly in a struggling, hypoxic individual. Finally, staff need to be aware of medicolegal implications of the carrying out any restraint.

## Oral tranquillization

If possible, give sedative drugs PO, rather than by injection. However, PO treatment may not be feasible in a violent and disturbed patient.

- Give *lorazepam* (1–2 mg PO) if there is no psychotic context.
- Give *lorazepam* (1–2 mg PO) + antipsychotic (eg *haloperidol* 1.5–3mg PO) if there is a psychotic context.
- Allow sufficient time for response before considering a second dose.

## IM (rapid) tranquillization

If PO therapy is inappropriate (refused, failed, or not indicated), choose between IM lorazepam OR IM haloperidol + promethazine.

In most instances, IM lorazepam is the most appropriate first choice:

- Give lorazepam (2–4 mg IM) and allow sufficient time for it to work.
- Consider giving a further dose of IM lorazepam if there is a partial response, but if there is no response, consider IM haloperidol (5–10mg) + IM promethazine hydrochloride (25–50mg) instead.

Do not use IM haloperidol + promethazine if there is any history of cardiovascular disease (including long QTc) or if there is no normal ECG recorded.

Monitor the patient's vital signs closely after any IM tranquillization—record observations every 15min if the patient:

- Is asleep/sedated.
- Has taken illicit drugs or alcohol.
- Has any pre-existing physical health problem.
- Has experienced any injury from restrictive intervention.
- If the *BNF*'s maximum dose has been exceeded.

## IV tranquillization

Senior staff will only consider using IV drugs (eg benzodiazepine) in truly exceptional circumstances, when immediate tranquillization is essential (see 2015 NICE Guidance 10 on 'Violence and aggression', available at: https://www.nice.org.uk/guidance/ng10).

# Self-harm

The term 'deliberate self-harm' is no longer used. Psychiatric symptoms are often associated with self-harm but tend to be transient and predominantly related to social or emotional factors. Psychiatric illness is relatively uncommon (~5–8% of cases, mostly depression). ~90% of self-harm involves self-poisoning, and the remainder physical self-injury (eg cutting). Most self-harm episodes are impulsive (considered for <1hr beforehand). Associated alcohol consumption is common and may have precipitated the event. However, assess carefully—1% of self-harm patients do commit suicide within a year. It is often prudent to admit patients with self-harm to an ED observation ward, allowing alcohol to wear off until the situation can be properly assessed. Useful guidance on the treatment and management of self-harm in EDs has been published by NICE (http://www.nice.org.uk) and the Royal College of Psychiatrists (http://www.rcpsych.ac.uk).

## Triage

Following an episode of physical self-harm and/or overdose, perform a rapid initial assessment (triage) to establish the degree of urgency of the situation, mental capacity, willingness to stay, distress levels, and presence of mental illness. Factors that may render the situation more urgent include:

- Need for urgent treatment for physical injury and/or overdose.
- Immediate risk of violence to others.
- Immediate risk of further self-harm.
- Need for treatment, but the patient is threatening to leave.

### *Australian Mental Health Triage Scale*

This combined physical and mental health triage scale is recommended by NICE and can be adapted for easy use (see http://www.rcpsych.ac.uk). Some features are summarized in Table 14.1.

**Table 14.1** Australian Mental Health Triage Scale

| Triage category | Features |
|---|---|
| 1 Extremely urgent | Violent, possessing a weapon, or further self-harm in the ED |
| 2 Very urgent | Extremely agitated/restless, aggressive, confused/unable to co-operate, or requiring restraint |
| 3 Urgent | Agitated/restless, bizarre behaviour, psychotic symptoms, severe depression, and/or anxiety |
| 4 Less urgent | Symptoms of anxiety and/or depression without suicidal ideation |
| 5 Least urgent | Compliant, co-operative, and communicative |

The system in place should ensure self-harm patients are checked upon at least every hour—a change in triage category may require more urgent assessment.

## Management plan

Offer all patients who present to the ED after self-harm a psychosocial assessment of the needs and risk by an appropriately trained individual. Some units continue to admit all patients with deliberate self-harm for psychiatric appraisal once medically fit, but sheer numbers can make this difficult. A selective approach distinguishes patients with underlying psychiatric pathology and/or true suicidal intent—both requiring formal psychiatric evaluation. Many centres have developed psychiatric liaison services with medical and nursing mental health specialists who can offer expert input in a timely fashion.

*Assessment*

Involve family/carers, whenever possible, with the patient's consent.
Focus upon:

- Events and circumstances leading up to the episode of self-harm.
- Preparation, concealment, and true intention of a self-harm act.
- Outcome of the act (eg unintended danger or accidental discovery).
- Current stresses and financial, legal, or interpersonal problems.
- Alcohol or substance misuse.
- Previous self-harm or psychiatric illness.

Decide about psychiatric referral using this information. If in doubt, refer. Also refer immediately any child or adolescent who presents with self-harm. Some EDs run a system whereby patients who are not deemed to be at immediate risk can return the following day for an appointment with a psychiatric liaison nurse/specialist for psychosocial assessment. Ensure that the patient's GP receives written communication about the patient's ED attendance and discharge.

## Factors suggesting suicidal intent

- Careful preparation (eg saving tablets) and/or significant premeditation.
- Final acts (eg organizing finances, insurance, or a will).
- Performing self-harm alone, secretly, or when unlikely to be discovered.
- Not seeking help following self-harm.
- A definite, sustained wish to die.

Suicide notes can be important but are sometimes left for dramatic effect and so are not always reliable indicators.

Take all self-harm acts by individuals aged >65y seriously—consider them to be evidence of suicidal intent until proved otherwise.

## Risk of further self-harm

*Recurrence* Is most likely if there have been repeated previous episodes (eg habitual self-cutters or recurrent overdoses).

*Socio-demographic predictors* Include being single or separated, aged 25–54y, and unemployed or social class V.

*Other factors* Include drug or alcohol dependence, a history of criminal behaviour, previous psychiatric treatment, or the presence of a personality disorder.

# Assessment of suicide risk

Prevention of suicide is a primary aim in assessing patients who self-harm. Certain factors are common amongst completed suicides and are significant if found in a patient who self-harms:

- ♂.
- Elderly (particularly ♀).
- Living alone.
- Separated, divorced, or widowed.
- Unemployed or retired.
- Physical illness (eg painful, debilitating, or terminal conditions).
- Psychiatric illness (especially schizophrenia and depression).
- Alcoholism.
- Sociopathic personality disorder.
- Violent method of deliberate self-harm (eg hanging, shooting, drowning, or high fall).

## Modified Sad Persons Scale

It can be difficult for clinicians without psychiatric training to assess suicide risk. The modified 'Sad Persons Scale' attempts to assist non-psychiatrists with this task. Previously, it was stated that patients with scores of <6 may be discharged (depending upon circumstances), but latest guidance advises against the use of scores to assess suicide risk (http://www.nice.org.uk). However, the scale serves as a guide regarding risk factors and as a useful prompt for areas to consider (see Table 14.2).

**Table 14.2** Modified Sad Persons Scale

| | Score |
|---|---|
| Sex ♂ | 1 |
| Age <19 or >45y | 1 |
| Depression or hopelessness | 2 |
| Previous suicide attempts or psychiatric care | 1 |
| Excessive alcohol or drug use | 1 |
| Rational thinking loss (psychotic or organic illness) | 2 |
| Separated, widowed, or divorced | 1 |
| Organized or serious attempt | 2 |
| No social support | 1 |
| Stated future intent (determined to repeat or ambivalent) | 2 |

## Repeat self-harmers

Consider referral to local organizations to help those who self-harm repeatedly, as well as national organizations (eg Samaritans—a listening service). Specific advice for people who repeatedly self-injure includes advice and instruction on harm minimization issues, self-management of superficial injuries, and dealing with scar tissue. The risk management plan within the overall care plan will help patients face the future. NICE guidance CG133, 'Information for the public' section (available at: http://www.nice.org.uk) may be useful.

# Mental health assessment issues

Patients who present with self-harm can pose difficult problems that are not often a feature of patients who do not have mental health problems. The management of some of these issues is addressed by NICE (http://www.nice.org.uk) and summarized below.

## Timing of psychosocial assessment

The ideal is to offer psychosocial assessment of patients with self-harm as soon as possible. There are occasions when this assessment needs to be delayed, including the following:

- Life-saving treatment for physical injuries is needed.
- The patient is unconscious and/or significantly under influence of alcohol/drugs, and therefore not capable of being properly assessed.

## Patient threatening to leave the department

Sometimes patients state that they wish to leave the department before psychosocial assessment. Very often, it is possible to persuade them to stay. Perform an assessment of the patient's mental capacity and mental illness to decide whether it is necessary to detain him/her under the Mental Capacity Act or Mental Health Act if he/she attempts to leave.

### *Diminished mental capacity and/or significant mental illness*

If there is diminished mental capacity and/or significant mental illness, refer for urgent mental health assessment and prevent the patient from leaving the department. If the patient does manage to leave the department despite best efforts, contact the police in order to try to bring him/her back.

### *No reduction in mental capacity and no significant mental illness*

If there is no reduction in mental capacity and no significant mental illness and the patient leaves, pass the information on to his/her GP and to the relevant mental health services as soon as possible, to enable rapid follow-up.

## Physical treatments

Management of poisoning is the focus of ➲ Chapter 4. Note that it is sensible to measure paracetamol levels in any patient who presents with a history of overdose of paracetamol and/or other drugs.

Try to close superficial skin wounds <5cm long with tissue adhesive strips. Employ standard assessment and treatment for deeper skin wounds or those >5cm in length (see ➲ Wound closure, p. 415), taking into account the preferences of the patient.

## Concerns about children and other dependants

Always analyse a patient's presentation following an episode of self-harm in the context of the family and social setting. In particular, consider whether the self-harm behaviour places children or other dependants at home at risk (eg patient self-harms whilst sole carer for a child). Make referrals to social services to protect children and other vulnerable persons as appropriate—this can be a tricky area, so if in doubt, discuss with a senior.

# Depression

The lifetime risk of depression is ~10% for men and ~20% for women. General population prevalence is 3–6% (↑ with age). Coexisting psychiatric or physical illness can make depression difficult to diagnose. Conversely, depression may be the presenting feature of physical illness (eg hypothyroidism, Cushing's syndrome, or malignancy). ~15% of those with recurrent affective disorder eventually commit suicide. Persisting suicidal ideation or recent self-harm, even if trivial, is highly significant in the presence of a diagnosis of depression.

## Background

Mood disorders are more common in relatives of depressives. Life events involving loss (partner, friend, health, job, status) can precipitate depression. Loss of a parent in childhood, unemployment, and lack of a confiding relationship with a partner ↑ vulnerability.

## Presentation and symptoms

Depressed patients almost always have persistent low mood, loss of interest and enjoyment (anhedonia), and lack of energy. Mood is unaffected by circumstances. Look for common features (see Table 14.3).

**Table 14.3** Common features indicating a person's mood

| Common symptoms | Somatic or vegetative symptoms |
|---|---|
| ↓ self-esteem and self-confidence | Sleep disturbance |
| ↓ concentration and attention | ↓ appetite |
| Memory disturbance (especially short-term) | Weight loss |
| Bleak and pessimistic views of the future | Constipation |
| Ideas of self-harm or suicide | Amenorrhoea |
| Feelings of guilt or worthlessness | Loss of interest or enjoyment |

Look for self-neglect. Does the patient exhibit psychomotor retardation (slow movements and speech) or is he agitated? Is eye contact maintained? Are there deficits of short-term memory and cognition that improve with ↑ effort? Psychotic symptoms occur in very severe cases (eg hallucinations or delusions). These are mood-congruent: derogatory voices, ideas of poverty, guilt, nihilism (patient believes he has no bowel, no clothes, no life, etc.). Anxiety can be a feature of depression.

*Atypical depression* can involve reversal of usual somatic symptoms, leading to ↑ appetite, ↑ weight, hypersomnia, and reversed diurnal mood variation.

## Treatment

Arrange psychiatric assessment for patients with *severe depression*, suicidal ideation, or psychotic features. Most respond to antidepressants, but do not start these in the ED. Some patients also require antipsychotics or electroconvulsive therapy (ECT). In cases with psychotic features or where there is a high risk of death from suicide or profound self-neglect, ECT is effective.

*Mild/moderate cases* may respond to psychological therapy. Counselling can help specific problems (eg bereavement or marital difficulties).

# Mania

*Mania and hypomania* are less common than other mood disorders but more often require compulsory hospital admission. Pathologically elevated mood combines with over-activity, irrationality, poor judgement, and lack of insight (see Table 14.4 for primary and other features). This leads to severe disruption of relationships, employment, or finances. Untreated, high rates of divorce, debt, violence, or suicide occur. Onset may be acute or insidious. Manic disorders can arise spontaneously or follow depressive illness, stress, surgery, infection, or childbirth. Antidepressant medication, ECT, steroids, and amphetamines can all precipitate mania, as can lithium withdrawal.

**Table 14.4** Primary and other features of mania

| Primary features | Other features |
|---|---|
| Over-cheerfulness | Irritability |
| Over-talkativeness | Flight of ideas |
| Over-activity | Distractibility |
| | Grandiosity |
| | ↓ requirement for sleep |
| | Delusions (mood-congruent) |
| | Hallucinations |
| | Impaired judgement |
| | Irresponsibility and impetuousness |
| | Gambling and promiscuity |

*Hypomania* denotes an intermediate state without delusions, hallucinations, or complete disruption of normal activities.

## Differential diagnosis

Schizophrenia can present with disorganized behaviour, violent excitement, delusions, and incomprehensible speech. The content of delusions (ie bizarre, rather than mood-congruent) will help distinguish this from mania.

## Approach to the patient

Stay calm and non-confrontational. Beware infectious optimism, which can easily lead to underestimating the severity of illness or the requirement for admission. Seek additional information from relatives. Irritability can be the dominant symptom of mania and may be expressed as a savage, highly detailed catalogue of the interviewer's shortcomings. Irritable patients can become angry or violent in the face of even minor frustrations.

## Treatment

Manage overt manic illness in hospital to avoid behaviour harmful to the patient or others. Insight is often ↓ or absent, so compulsory admission may be required. Liaise with the psychiatrist before commencing drug treatment, as this may adversely affect assessment. Lithium is traditional and effective, both as acute treatment and as prophylaxis. However, increasingly, other drugs (eg valproate) are now being used.

# Schizophrenia

This affects all areas of personal function, including thought content and process, perception, speech, mood, motivation, and behaviour. A common pattern is acute exacerbation, with ↑ residual deficit between episodes. 30% of those who suffer a first episode never have another. Another 30% develop chronic symptoms requiring frequent admission or long-term care. The lifetime risk is 1/100.

## Clinical features

No single symptom is pathognomonic—hallucinations or delusions simply confirm psychosis.

*Schneider's first rank symptoms* Originally suggested schizophrenia in the absence of organic disorder. It is now acknowledged that they can occur in mania and other conditions:

- *Auditory hallucinations*: ≥2 voices discussing the subject in the third person or giving a running commentary on his/her thoughts/behaviour.
- *Thought withdrawal, insertion, or broadcasting*.
- *Somatic passivity*: sensations, emotions, or actions are externally imposed or controlled.
- *Delusional perception*: a genuine perception takes on abnormal significance for the subject and is the basis of their delusional system.
- *Gedankenlautwerden*: voices repeating the subject's thoughts out loud or anticipating the subject's thoughts.

## Diagnosis

Mental state examination will help to exclude organic and affective disorders, remembering:

- Non-auditory hallucinations are more common in organic conditions.
- Delusions in depression and mania are mood-congruent.

*Differential diagnoses*

- *Organic causes*: temporal lobe epilepsy, drug-induced states, alcoholic hallucinosis, cerebral tumour, encephalitis, head injury.
- *Psychiatric*: affective psychoses, schizo-affective disorder, psychogenic psychosis, delusional disorder (eg infestation), personality disorder.

## Management

*Patients not known to have schizophrenia*

Refer to the psychiatric team who will advise about the need for urgent antipsychotic treatment.

*Patients known to have schizophrenia*

Schizophrenics frequently present to the ED with mental health issues and problems. It can be difficult to formulate a management plan unless relevant background information is available. Liaise with relevant individuals (including the community psychiatric nurse and psychiatrist) to decide whether to treat in hospital or in the community and what form any treatment should take.

# Complications of psychiatric drugs

## Antipsychotic drugs

*Acute dystonic reactions* (grimacing, facial and masseter spasm, deviated gaze, torticollis, limb rigidity, and behavioural disturbances) frequently present to the ED. They follow ingestion of antipsychotics (eg phenothiazines or haloperidol) and/or other drugs (eg metoclopramide), even in therapeutic dosages. Reactions can occur up to 1 week after ingestion. Acute dystonia can dislocate the mandible. Dystonia can be mistaken for malingering, as symptoms can be briefly interrupted by voluntary actions. Once diagnosed, *treat with* procyclidine 5–10 mg IV bolus, repeated as necessary after a few minutes.

Dramatic resolution of symptoms occurs within minutes, confirming the diagnosis. Symptoms may recur—treat with procyclidine 5mg PO every 8hr. Large doses of procyclidine cause euphoria and fixed dilated pupils, hence, its abuse by some patients. Diazepam also works but is less effective and carries risks of excessive drowsiness or respiratory depression.

## Clozapine

This is an atypical antipsychotic used in treatment-resistant schizophrenia. Agranulocytosis occurs in 3% of patients. For this reason, all patients are enrolled with the Clozaril Patient Monitoring Service (telephone 0845 7698269) which supervises regular blood screening. Check FBC for neutropenia in any patient presenting with fever, sore throat, or other infection.

## Monoamine oxidase inhibitors

MAOIs (eg phenelzine, tranylcypromine) irreversibly block enzymes responsible for oxidative metabolism of 5HT, noradrenaline, tyramine, and other amines. Once discontinued, enzyme inhibition continues for up to 2 weeks, during which time other drugs should not be introduced. Newer, reversible inhibitors of monoamine oxidase A (RIMAs), eg moclobemide, cease to have effects after 24–48hr. MAOIs cause postural hypotension, but acute hypertensive reactions follow ingestion of amine-rich foods (eg Bovril™, Marmite™, cheese, red wine). Noradrenaline release causes vasoconstriction, tachycardia, and hypertension that can, in severe cases, lead to intracerebral or subarachnoid haemorrhage. Similar hypertensive crises can be caused by concurrent use of levodopa, sympathomimetics, and amphetamine, or drinking certain low-alcohol beers or wines.

## Lithium

Lithium toxicity presents with severe nausea, vomiting, cerebellar signs, or confusion. SSRIs (eg fluoxetine), anticonvulsants, antipsychotics, diuretics, methyldopa, and calcium channel blockers can all precipitate toxicity.

- Look for tremor, cerebellar ataxia, muscular twitching (myoclonus), spasticity, choreiform movements, upgoing plantar responses, incoordination, slurred speech, impaired concentration, drowsiness, and coma.
- Check serum lithium (plain, not lithium heparin tube!) and U&E immediately. Serum lithium levels correspond poorly with clinical signs (toxicity can occur within the therapeutic range), so toxicity is a clinical diagnosis. Stop lithium and treat according to severity of toxicity (see ➲ Lithium poisoning, p. 205).

# Munchausen's syndrome

Also known as 'hospital hopper', this is characterized by recurrent admissions with factitious symptoms and signs of physical illness. Other basic components are a morbid attraction to the sick role, pathological lying, and pleasure from deceiving medical staff. The incidence is unknown, but it is probably underestimated. There may be an underlying personality disorder, but true psychiatric illness is rare. Origins are uncertain—excessive dependency, inability to form trusting relationships, attention-seeking, childhood hospitalization, and resentment of doctors for previous treatment have all been suggested.

## Presentation

Common presentations involve detailed and convincing descriptions of cardiac chest pain, abdominal pain (especially pancreatitis), haematemesis, haemoptysis, rectal bleeding, haematuria, or pyrexia. More rarely, patients present with artefactual dermatitis or with a dramatic history of trauma (eg fall or pedestrian knockdown). Distinguish Munchausen's syndrome from:

- *Malingering*: fabricating illness for definite gain (eg stealing drugs, avoiding court appearance, faking symptoms to obtain opioids).
- *Somatoform disorders*: physical symptoms or signs without organic cause, but not under voluntary control.
- *Fabricated and induced illness*: see Fabricated or induced illness, p. 760.

## Suspicious features

- Incomplete or inconsistent disclosure of personal details and past history.
- Patient a long way from home area for unclear reasons.
- Recent dramatic history of surgery, MI, or complications elsewhere.
- Excellent knowledge of finer details of past treatment and/or complications.
- *Multiple scars*: laparotomies, sternotomy, venous cutdowns.
- Elaborate history of allergy (eg allergic to all painkillers, except pethidine).
- Unconvincing claims of medical or paramedical occupation.
- Unusual/demanding behaviour and/or avoidance of eye contact.
- No ascertainable organic cause for the symptoms.

## Management

Early recognition is important, but first exclude genuine illness. Consider admission to make the diagnosis, even though this achieves the patient's aim. If suspicions are aroused, discreetly check past history. Once discovered, most patients self-discharge, often noisily, but rarely violently.

*Avoid a 'showdown'*. Simply state that deception is at an end and that no retribution is planned, and offer to help the patient with their problem.

*Do not use placebos* to uncover fabricated illness—they can work equally well on genuine symptoms!

*Once discovered*, record events carefully, particularly the medical history given, background details, appearance, and scars. Circulate details to other EDs.

## Factitious disorder in health care workers

The Clothier report (Department of Health, 1994) advised that patients with severe personality disorder (by inference, factitious disorder) should be prevented from working in health-related disciplines. Detection of factitious disorder in health care workers has serious implications. If suspected, discuss immediately with the ED consultant.

# Medically unexplained symptoms

## Background

A significant proportion of patients who attend the ED have symptoms for which no cause is found. Some of these patients manage to build up a significant volume (or volumes!) of medical records.

## Terminology

There is a potentially confusing range of terms in use.

*Somatization*

Physical symptoms with presumed psychological origin.

*Somatoform pain disorder*

Persistent, severe unexplained pain, which is attributed to psychological disorders.

*Conversion (dissociative) disorders*

Loss or disturbance of normal motor or sensory function, which is attributed to a psychological origin (thoughts/memories to the conscious mind are 'converted' into physical symptoms, eg amnesia).

*Factitious symptoms*

Symptoms which are intentionally produced, with the aim of receiving a medical diagnosis—when there is secondary gain (eg legal compensation, obtaining opioid drugs), it is known as *malingering*.

*Medically unexplained symptoms*

This is an umbrella term, which makes no assumptions about the cause of the symptoms.

## Differential diagnosis

Patients who present acutely with medically unexplained symptoms may be suffering from a range of problems, including: anxiety, depression, psychosis, 'functional somatic illness', conversion disorders, factitious disorders, malingering, and uncommon medical syndromes that have not yet been diagnosed.

## Approach to patients with medically unexplained symptoms

The Royal College of Psychiatrists (℔ http://www.rcpsych.ac.uk) has published some useful recommendations. Consider the following:

- Try to obtain past medical and psychiatric records/summaries (computerized records may assist in this process) and/or speak to the GP. Helpfully, frequent attenders with medically unexplained symptoms sometimes have a care plan which can guide management when they attend the ED.
- If the patient's medical complaints are known to be unexplained (or part of a psychiatric illness), then further investigations may be inappropriate.
- Investigate judiciously—do not underestimate the ability to cause iatrogenic harm.

# Alcohol abuse

Alcohol-related problems account for up to 15% of ED workload in the UK. Alcoholics have ↑ rates of heart disease, malignancy, and stroke but often succumb to injuries. Excessive alcohol consumption is a feature of 30% of road traffic fatalities, 25% of fatal work injuries, 30% of drownings, and 50% of burn deaths. Alcohol is involved in ~30% of suicides, ~60% of homicides, and most assaults. Suspicion is the key to detecting alcohol problems.

## Units

The number of 'units' (10mL of pure alcohol) is included on packaging. A bottle of wine contains ~10U, and a bottle of spirits ~30U. Current advice is a 'safe' limit of 14U/week (♂ and ♀).

## Alcohol absorption, metabolism, and elimination

Alcohol is absorbed from the small intestine and, to a lesser extent, the stomach. The rate of absorption depends on the nature of the drink and any associated food consumed. Alcohol is absorbed more slowly from dilute drinks (eg wine), compared with more concentrated fortified sherry or port. Alcohol is water-soluble, so distributes throughout the body. It is mostly metabolized in the liver by an enzymatic process involving alcohol dehydrogenase, which converts it to acetaldehyde and then acetic acid. A relatively small amount of alcohol is excreted unchanged in the urine (and, to a lesser extent, in breath and sweat).

*Clearance of alcohol* The rate of clearance of alcohol from blood varies enormously between individuals, with typical quoted values of 10–20mg/dL/hr in most adults, although higher values occur in some chronic alcoholics.

## Assessing alcohol problems

A history of alcohol consumption is notoriously unreliable when taken from heavy drinkers and chronic alcoholics, who may significantly under-report the extent of their drinking and its effect upon their lives. The actual amount of alcohol consumed is less important than the consequences of drinking to the patient. Cover the following areas:

*Biological* GI upset/bleeding, withdrawal fits, blackouts, peripheral neuropathy.

*Psychological* Low mood, hallucinations, delusions, memory problems.

*Social* Marital, work, driving, debt, criminality.

Significant features include compulsion to drink and loss of control.

## The CAGE questionnaire

- Have you ever felt you should *Cut down* your drinking?
- Have people *Annoyed you* by criticizing your drinking?
- Have you ever felt *Guilty* about your drinking?
- Have you ever had a drink first thing in the morning to steady your nerves or to get rid of a hangover (*Eye opener*)?

Any single +ve answer is significant, and >1 +ve answer is probably diagnostic of chronic alcohol dependence.

# Acute alcohol intoxication

## Effects of intoxication

Alcohol depresses the nervous system—initial euphoric effects are due to suppression of inhibition by the cerebral cortex. Effects vary between individuals and Table 14.5 is a very rough guide. Behaviour, including propensity to violence, is influenced by the environment and social setting. Although death may occur at levels of >350mg/100mL, the risk of a harmful or fatal event ↑ at any level—especially road traffic collisions, work, and home injuries and assaults (including sexual assault). The current UK blood alcohol legal limit for driving is 80mg/mL.

**Table 14.5** Effects of various concentrations of alcohol

| Blood alcohol concentration (mg/100mL = mg/dL) | Effects |
|---|---|
| 30–50 | Measurable impairment of motor skills |
| 50–100 | Reduced inhibitions, 'excitant effect' |
| 100–150 | Loss of co-ordination and control |
| 150–200 | 'Drunkenness', nausea, ataxia |
| 200–350 | Vomiting, stupor, possible coma |
| 350+ | Respiratory paralysis, possible death |

Alcohol intoxication is characterized by slurred speech, inco-ordination, unsteady gait, nystagmus, lethargy, and facial flushing. The differential diagnosis is extensive: head injury, hypoglycaemia, post-ictal confusional states, hepatic encephalopathy, meningitis, encephalitis, or intoxication with other drugs. In most patients, these conditions can be excluded by examination and simple investigations (although some not infrequently coexist with acute alcohol intoxication—especially head injury and hypoglycaemia).

## Management

Aim to discharge conscious, ambulant patients who exhibit uncomplicated acute alcohol intoxication if accompanied by a responsible adult.

*Violent patients* Who appear intoxicated require examination prior to escort from the ED by the police. As a minimum, perform a brief neurological examination, simple observations, and BMG.

*Comatose patients* Are a medical emergency. Protect the airway and anticipate vomiting (recovery position may be useful). Exclude hypoglycaemia and other metabolic causes of coma. Exclude head or neck injury, and adopt a low threshold for CT. Observe closely.

*Alcohol-induced hypoglycaemia* Particularly affects chronic alcoholics and children. It also occurs in binge drinkers who present with alcoholic ketoacidosis. Hypoglycaemia can occur during intoxication and up to 24hr after. In children, fits may result.

*Coagulation disorders* Often occur in alcoholics with liver damage. They often have ↓ platelets (direct effect of alcohol on bone marrow). Consider this in patients presenting with GI haemorrhage or head injury.

# Alcohol withdrawal

## 'Simple' alcohol withdrawal

Uncomplicated alcohol withdrawal is common, usually starting within 12hr of stopping (or reducing) alcohol intake. Withdrawal symptoms often start before alcohol is completely cleared from blood. Features include anxiety, restlessness, tremor, insomnia, sweating, tachycardia, and ataxia. Simple withdrawal can be managed on an outpatient or day-patient basis. Consider commencing treatment in the ED for uncomplicated withdrawal (eg diazepam 5–10mg PO or chlordiazepoxide 10–30mg), but leave decisions about continuing treatment to inpatient teams. Inpatient detoxification is indicated for those with a history of withdrawal seizures, delirium tremens, or withdrawal symptoms who are being admitted for other problems. The revised Clinical Institute Withdrawal Assessment for Alcohol (CIWA-Ar) Score assesses ten clinical signs of withdrawal and may help to guide treatment (🔗 https://www.agingincanada.ca/CIWA.HTM). *Note*: alcoholics admitted with 'simple' withdrawal may be thiamine-deficient, so use this as an opportunity to give parenteral and/or PO thiamine.

## Delirium tremens

Occurs in a small minority of alcoholics who undergo withdrawal but carries significant mortality. Typically starts >48hr after stopping drinking. As well as 'simple' withdrawal, there may be significant autonomic hyperactivity, with tachycardia, hyper-reflexia, hypertension, fever, visual or tactile hallucinations, sinister delusions, disorientation, and confusion. Deaths occur from arrhythmias (secondary to acidosis, electrolyte disturbance, or alcohol-related cardiomyopathy), infection, fits, or cardiovascular collapse. Monitor closely, check BMG, offer oral lorazepam, and refer to the medical team/HDU. If oral lorazepam is declined and/or symptoms persist or worsen (especially fits), give IV lorazepam and involve ICU. Experts may use haloperidol or olanzapine (see 🔗 http://www.nice.org.uk).

## Alcohol withdrawal seizures

Typically self-limiting grand mal fits, occurring hours or days after the last alcoholic drink. Check BMG and treat ongoing fits with IV lorazepam (see ➲ Seizures and status epilepticus, pp. 156–7)—phenytoin is not usually indicated. Examine for possible head injury. In patients who have recovered from a short-lived fit, consider oral lorazepam to prevent further fits.

## Alcoholic ketoacidosis

Can occur when an alcoholic stops drinking, vomits repeatedly, and does not eat. Ketoacidosis develops from fatty acid breakdown, complicated by dehydration from vomiting. The patient usually presents 1–2 days after the last binge with vomiting, signs of chronic alcohol abuse, and a high anion gap metabolic acidosis. ABG may reveal ↓ $pCO_2$, ↓ $HCO_3^-$, and normal $pO_2$. pH is variable because metabolic acidosis may be altered by metabolic alkalosis from vomiting and possibly respiratory alkalosis. Plasma ethanol is low or absent. Give IV 0.9% saline with 5% glucose and thiamine supplementation, whilst monitoring U&E, glucose. Refer to the medical team and consider HDU/ICU.

# Alcohol-related brain injury

Wernicke–Korsakoff syndrome develops in problem drinkers who are thiamine-deficient. Autopsy analysis suggests that the syndrome may occur in as many as 12.5% of chronic alcohol misusers. Make a presumptive diagnosis of Wernicke–Korsakoff syndrome in patients with a history of alcohol misuse and one or more of the following unexplained symptoms: ataxia, ophthalmoplegia, nystagmus, confusion, memory disturbance, reduced conscious level, hypotension, and/or hypothermia.

### Wernicke's encephalopathy

This is characterized by degenerative changes around the third ventricle and aqueduct, particularly the mammillary bodies. It presents with an acute confusional state, nystagmus, ophthalmoplegia, ataxia, and polyneuropathy. Ataxia typically affects the trunk and lower extremities. Clinical abnormalities may develop acutely or evolve over several days.

*Initially treat* with parenteral thiamine (eg Pabrinex® 2–3 pairs tds—10mL as an IVI in 100mL of 0.9% saline over 30min). This may occasionally cause anaphylaxis, so ensure resuscitation facilities are available. Refer to the medical team for continuing IV thiamine, unless Wernicke's is excluded.

### Korsakoff's psychosis

This is an amnesic state with profound retrograde and anterograde amnesia, but relative preservation of other intellectual abilities. It typically develops after Wernicke's encephalopathy, but some patients develop a combined syndrome from the outset with memory loss, eye signs, and unsteadiness, but without confusion. Treat with parenteral thiamine and admission, as for Wernicke's encephalopathy.

# Help for alcoholics

The relatively regular contact between those with alcohol problems and EDs may be viewed as an opportunity to offer intervention. There is good evidence to suggest that brief interventions may reduce alcohol consumption and the risk of physical harm. Consider the 1min Paddington Alcohol Test to help patients who present with alcohol-related problems (see http://www.rcem.ac.uk). Warn alcoholics about the risks of a sudden very dramatic reduction in alcohol intake.

The following organizations may help patients and relatives:

- Alcoholics Anonymous (see http://www.alcoholics-anonymous.org.uk), plus local networks and telephone numbers.
- Al-Anon for relatives (tel: 0800 0086 811) (see http://www.al-anonuk.org.uk).

### Medically assisted alcohol withdrawal

Aim to offer admission for medically assisted withdrawal to those in acute alcohol withdrawal who are <16y, vulnerable (frail, multiple comorbidities, learning difficulties, lacking social support), or at high risk of developing withdrawal fits or delirium tremens (see http://www.nice.org.uk).

# Drug and substance abuse

Drug users present to the ED at times of crisis (eg acute intoxication, overdose, withdrawal, or other medical complications of drug use). Do not assume all drug users present to the ED simply to obtain drugs. Find out about local addiction services and how referrals are made. Direct those seeking help with a drug problem to the appropriate services. Know the local preferred drugs of abuse and the preferred methods of taking them. Find out what terminology is used locally for each substance.

*Do not supply drugs* of dependence to users. Prescriptions are carefully controlled by addiction services and pharmacists. Elaborate tales of lost or stolen drugs/prescriptions are invariably false.

*Manage painful conditions* in drug users as for other patients. Do not withhold analgesia if in obvious pain. For minor complaints, simple analgesia is as effective as in non-drug users. Do not dismiss symptoms simply because the patient is a drug user. Even drug users get acute appendicitis and other common acute illnesses.

## Intoxication

As with alcohol, mild cases require little intervention. Observation by a responsible adult or briefly on a ward usually suffices. Discharge patients when ambulant and fully orientated, having excluded serious problems.

*Glue and solvents* Users may smell of substances or have them on their clothes or skin. There may be a perioral rash. Intoxication produces euphoria, agitation or drowsiness, slurred speech, and unsteady gait.

*Benzodiazepines and CNS depressants* Mild intoxication is similar to that with alcohol. ↑ intoxication produces nystagmus, diplopia, strabismus, hypotonia, clumsiness, and moderately dilated pupils.

*Amphetamines, ecstasy, cocaine, and mephedrone* Produce hyperstimulation, restlessness, pyrexia, and sympathomimetic effects. Cocaine effects occur more rapidly. Severe cases exhibit paranoia, violent behaviour, or seizures. Cocaine may also cause chest pain, arrhythmias, or even MI. Ecstasy can cause an idiosyncratic reaction similar to malignant hyperthermia (see ➲ Recreational drugs, pp. 222–3).

## Overdose

Protect the airway, provide $O_2$ as required, and exclude hypoglycaemia or serious injury in all cases.

*Opioid overdose* Is often inadvertent, either from use of unusually pure drugs or after a period of abstinence (tolerance is ↓). Characteristic signs are coma with pinpoint pupils and respiratory depression (see ➲ Opioid poisoning, p. 196). Pulmonary oedema, hypothermia, and rhabdomyolysis can occur. Hypoxia may cause dilated pupils. If opioid overdose is suspected, give naloxone 0.4–0.8mg IV, repeated according to response (for further detail regarding treatment, see ➲ Antidotes for poisons, pp. 194–5). Remember to ensure that the patient is observed for at least 6hr after the last dose of naloxone.

*Intentional overdose* Requires assessment of suicide risk (see ➲ Assessment of suicide risk, p. 630) and mental capacity in case the patient threatens to leave against advice.

### Skin complications

SC drug injection ('skin popping') can cause cellulitis, abscesses, extensive skin necrosis, necrotizing fasciitis, tetanus, botulism, and anthrax. Refer for formal exploration, drainage, and follow-up by the surgical team for all but the most minor infections. Apparently 'simple' abscesses may extend deeply into muscle or form part of a false aneurysm! Needle fragments rarely require removal unless they embolize (eg to the lungs).

*Anthrax in drug users* After an outbreak of anthrax in heroin users in Scotland in 2010, Health Protection Scotland (http://www.hps.scot.nhs.uk) advised doctors to suspect anthrax in a drug user presenting with any of the following:

- Severe soft tissue infection and/or signs of severe sepsis/meningitis.
- Clinical features of inhalational anthrax (see Anthrax, p. 243).
- Respiratory symptoms + features of meningitis or intracranial bleeding.
- GI symptoms (eg pain, bleeding, nausea, vomiting, diarrhoea, ascites).

*Approach* Get expert help early to advise on management (ICU, surgeons, microbiology, public health, hospital infection team). Start IV antibiotics according to advice (eg combination of ciprofloxacin, clindamycin + penicillin, or if there is soft tissue infection: ciprofloxacin, clindamycin, penicillin, flucloxacillin + metronidazole). Experts will advise on whether to use anthrax immune globulin IV (human) antitoxin.

### Vascular complications

IV injection ('mainlining') of drugs causes phlebitis, DVT, and bacterial endocarditis. Chronic injectors may resort to neck or groin vessels (the femoral artery being commonly damaged). Arterial injection can cause false aneurysms, fistulae, or peripheral emboli. Occasionally, IV drug users present with massive and devastating blood loss from an injection site (particularly the groin)—apply firm pressure, resuscitate with IV fluids ± blood, and call for the surgical team.

Inadvertent arterial injection of poorly soluble preparations causes severe limb pain, skin pallor, and mottling with paraesthesiae in the presence of palpable (often bounding) peripheral pulses. Diffuse soft tissue damage may result in compartment syndromes, rhabdomyolysis, renal failure, and irreversible limb damage necessitating amputation.

### Orthopaedic complications

Injecting drug users who present with acutely painful joints (especially hip joints) may have septic arthritis. Clinical and radiological evidence may be minimal, so adopt a high index of suspicion. Provide analgesia, take blood cultures and FBC, and admit for joint aspiration and IV antibiotics.

### Drug withdrawal states

Sometimes drug users present with overt evidence of drug (± alcohol) withdrawal. It can be difficult to judge if the problem is drug intoxication, drug-related (eg stimulant-induced psychosis, withdrawal), or coexisting disease. Observe and monitor closely—treat symptomatically (eg with small doses of oral benzodiazepines), and refer to the medical team.

# Compulsory hospitalization

Compulsory detention of patients in the UK requires the patient to be *both*:

- Suffering from a mental disorder (mental illness or handicap).
- Requiring emergency hospital admission to protect the health or safety of the patient or for the protection of others.

Emergency detention under mental health legislation does not allow treatment for psychiatric illness. Emergency treatment of psychiatric or physical illness is carried out under *common law*. In this situation, there must be an immediate threat to life or serious danger to the patient or others if treatment is not given. For this reason, mental health legislation cannot be used to impose emergency treatment without patient consent. Note that ED patients are not legally inpatients until they go to a ward.

## Detention of psychiatric emergencies in the ED

### *England and Wales*

Section 2 is used most commonly in the ED. It requires recommendations from two doctors to be accepted by an approved social worker and allows detention for up to 28 days for assessment and treatment.

### *Scotland*

The Mental Health (Care and Treatment) (Scotland) Act 2003 came into effect in 2005 (see http://www.mwcscot.org.uk). Part 5 of the Act enables a fully registered medical practitioner to grant an *emergency detention certificate* that authorizes managers of a hospital to detain someone for 72hr. Before granting an emergency detention certificate, the doctor needs to consult and gain consent of a mental health officer, unless impracticable. The patient is then examined by a psychiatrist, who, if not satisfied that relevant criteria are met, cancels the certificate.

### *Northern Ireland*

Mental Health (Northern Ireland) Order 1986, Part II, Article 4—admission for assessment of mental disorder.

- Requires two or three doctors, including the responsible medical officer (RMO)—in charge of the patient's treatment.
- Application by the nearest relative or an approved social worker.
- Lasts 7 days, renewable up to 14 days.
- Lasts until discharge by the RMO board or nearest relative or until detained under Article 12.

## Section 136 (England)

This allows a police officer to detain someone in a public place when he/she appears to be mentally disordered and is causing disturbance. The police officer's responsibility is to take the detained person to a 'place of safety' (usually a police station or a psychiatric ward) where he/she is assessed by a psychiatrist and an approved social worker.

The *Mental Health Act (Scotland) 2003* provides police officers in Scotland with similar powers—a police constable may remove a person to a place of safety from a public place if a mental disorder is suspected and it is also suspected that the person needs immediate care and treatment.

# Mental Capacity Act

The Mental Capacity Act (MCA) 2005 offers a comprehensive framework for decision-making on behalf of adults aged >16y lacking capacity to make decisions on their own behalf. It only applies in England and Wales.

## Defining capacity

A person lacks capacity if, when a decision needs to be made, they are unable to make or communicate the decision because of an 'impairment or disturbance of the mind or brain'. There is a 2-stage test of capacity:

- Is there an impairment of, or disturbance in the functioning of, the person's mind or brain? If so,
- Is the impairment or disturbance sufficient that the person lacks the capacity to make that particular decision?

## Five statutory principles

- Capacity must be assumed unless it is established to be lacking.
- A person is not being treated as unable to make a decision unless all practicable steps to help him do so have been taken without success.
- A person should not be treated as unable to make a decision merely because he makes an unwise decision.
- A decision made, or an action performed, for or on behalf of a person who lacks capacity must be taken in his/her best interests.
- Before a decision is made or an action performed, consideration must be given to whether the purpose for which it is needed can be as effectively achieved in a way that is less restrictive of the person's rights and freedom of action.

## Assessment of capacity

A person lacks capacity if he/she fails:

- To understand the information relevant to the decision.
- To retain the information relevant to the decision.
- To use or weigh the information.
- To communicate the decision (by any means).

## Admission and treatment

Patients can be admitted and treated under the MCA 2005 only if six qualifying safeguards are met:

- The person is at least 16y old.
- The person has a mental disorder.
- The person lacks capacity to decide whether to be in hospital or a care home for the proposed treatment or care.
- The proposed deprivation of liberty is in the person's best interests and it is necessary and a proportionate response to the risk of harm.
- The person is not subject, or potentially subject, to specified provisions of the Mental Health Act in a way that makes them ineligible.
- There is no advance decision or decision of an attorney or deputy which makes the proposed deprivation of liberty impossible.

In other circumstances, consider using the Mental Health Act 1983 to admit a mentally disordered patient who lacks capacity to consent.

# Chapter 15

# Paediatric emergencies

**Paediatric problems also covered elsewhere in this book**


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- Poisoning pp. 187–225
- Incubation periods of infectious diseases pp. 228–9
- Childhood infectious diseases pp. 230–1
- Meningitis pp. 232–3
- Gastroenteritis/food poisoning pp. 236–7
- Infestations pp. 240–1
- Analgesia in specific situations pp. 290–1
- Nasal diamorphine p. 291
- Local anaesthesia in children p. 297
- Sedation p. 319
- Instructions after minor head injury p. 375
- Tetanus prophylaxis p. 424
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# The paediatric environment

## Caring for children

Children are not little adults. They differ from adults anatomically, physiologically, emotionally, and in the spectrum of pathological conditions to which they are susceptible. It is natural for those hospital staff who have not previously dealt with children to be slightly apprehensive about treating them, particularly when they are distressed or seriously unwell. Be guided by more experienced staff, who are often adept at dealing with children as patients (and very often as parents as well). Such staff are particularly good at recognizing children who are seriously unwell—listen carefully to what they have to say. There is no substitute for experience, but practical courses aimed at managing emergencies in children (eg Advanced Paediatric Life Support (APLS)) are highly recommended. These courses deservedly devote much time to the recognition of seriously ill or injured children, according to whether or not they are physiologically deranged. Consider each child according to expected 'normal' physiological values (see Table 15.1).

Children do not always respond in the same way to illness as adults. They are particularly likely to be frightened of doctors, nurses, and hospitals. Do not waste the opportunity to make important observations (RR, pattern and effort, behaviour, conscious level, colour, and parental interaction). Spend time talking to children to reassure them and win their confidence before starting any examination or performing any procedure (unless, of course, they require emergency resuscitation). Lowering yourself to their physical level will make you less intimidating. Involve the parents from the start (see ➲ Interacting with parents, p. 648). Where appropriate, allow children to relax and play with toys. Play therapists can be particularly helpful by providing distraction during procedures.

## Interacting with parents

Parents are patients too. They are likely to be understandably upset and worried. Take time to explain to the parents exactly what is happening to their children at all stages. Obtain appropriate consent, but do not delay life-saving measures. For the sake of both parents and children, try to allow parents to remain with their children as much as possible. This is especially important during resuscitation—allocate an experienced member of nursing staff to look after and explain to parents what is happening. If the presence of the parents is impeding the progress of the resuscitation, consider gently asking them to step outside.

## Analgesia

Make an assessment of the extent of pain (see ➲ Assessment of pain in children, p. 282). Physiological differences between adults and children do not diminish the need to provide adequate analgesia for children. Reassurance is often an important component, but be honest and do not be tempted to tell to a child that a painful procedure (eg emergency insertion of an IV cannula) will not produce any pain or discomfort—this will simply cause the child to lose confidence in the ED team.

## Weight estimation

Try to weigh children who need treatment. This is important as most drug doses are based on weight. However, it is not always possible to weigh a sick child. In an emergency, use a chart, Broselow tape, or one of the formulae for estimating children's weight.

A baby typically weighs around 3.5kg at birth, becoming 4.5kg at 1 month, and 7.5kg by 6 months.

The following formula estimates a child's weight, based upon age (between 1 and 10y):

$$\text{Weight in kg} = (\text{age in years} + 4) \times 2$$

So a 6y old child will weigh:

$$(6 + 4) \times 2 = 20\text{kg}$$

This formula often underestimates the weight of many children in the UK or other developed countries, but it may overestimate the weight of children from other regions. A formula using mid-arm circumference (MAC) has been developed in Asia and is:

$$\text{Weight in kg} = (\text{mid-arm circumference in cm} - 10) \times 3$$

So a child with a 15cm MAC will weigh: $(15 - 10) \times 3 = 15$kg.

## Drug doses

Do not estimate 'rough doses' of drugs for children based on knowledge of adult doses. Instead, use the weight and age of a child, together with a reference source [eg *BNF for Children* (*BNFC*), which is available at: https://bnfc.nice.org.uk] to determine the appropriate dose.

## Preparation for resuscitation

Find out where the paediatric resuscitation equipment is kept and how it works. Learn the paediatric resuscitation guidelines and practise BLS and other procedures on manikins. Knowledge of normal (expected) physiology at various ages helps to evaluate sick children in the ED.

**Table 15.1** Normal (expected) physiological values at different ages*

| Age (y) | RR | Heart rate | Systolic BP (mmHg) |
|---|---|---|---|
| <1 | 30–40 | 110–160 | 70–90 |
| 1–2 | 25–35 | 100–150 | 80–95 |
| 2–5 | 25–30 | 95–140 | 80–100 |
| 5–12 | 20–25 | 80–120 | 90–110 |
| >12 | 15–20 | 60–100 | 100–120 |

Expected systolic BP = 80 + (age in years × 2) mmHg.

* Adapted from APLS.

# Primary assessment and resuscitation

## Preparation

Caring for a sick child is a daunting task. Get experienced help early—call for senior ED, paediatric, and ICU/paediatric intensive care unit (PICU) help if you are alerted that a sick or injured child is being brought to the ED. Many hospitals run team response systems (eg 'PERT'—Paediatric Emergency Response Team).

A pre-alert from the ambulance service provides time to prepare, including allocating roles and working out in advance the likely doses of drugs and amounts of fluid that will be required. If the child's details are available, it may also be possible to obtain background information, most particularly past medical history and allergies, which is very useful when children with complex problems present.

## ABCDE approach

Perform a rapid primary assessment of airway, breathing, circulation, disability, and exposure to quickly identify and treat life-threatening problems as they are found. This will help to maintain vital functions before disease-specific therapies are started. Early recognition and treatment of these problems will help to avoid cardiorespiratory arrest with its poor outcome.

## Airway

*Assess* Patency by looking, feeling, and listening.

*Resuscitate* If there is no air movement, perform chin lift or jaw thrust. If there is still no evidence of air movement, give rescue breaths using an appropriately sized bag–valve–mask device. If the child is breathing, listen for stridor (implies airway obstruction), and look for recession.

## Breathing

*Assess the effort of breathing* By measuring the RR, looking for intercostal recession and accessory muscle use, and listen for gasping, stridor, wheeze, and grunting. Note that a child's RR can vary according to a number of factors (eg being upset or distressed), so interpret a single value cautiously—instead, look for the trend.

*Assess the efficacy of breathing* By looking for chest expansion, auscultation of the chest, and measuring $SpO_2$. A 'silent chest' is a very worrying sign.

*Assess the effects of respiratory failure* By assessing mental status, measuring the heart rate (↑ with hypoxia, but bradycardia is a pre-terminal sign), and examining skin colour (hypoxia causes pallor, and cyanosis is a late sign). Reduced breathing effort and gasping may indicate exhaustion (a pre-terminal sign), cerebral depression, or neuromuscular disease.

*Resuscitate* Give high-flow $O_2$ to any child with respiratory difficulty or hypoxia. If respiration is inadequate, support with basic airway care and bag–valve–mask ventilation and get senior ED/ICU/PICU help to provide a definitive airway (tracheal intubation and IPPV).

### Circulation

*Assess* Heart rate (bradycardia is a late sign of cardiovascular failure), pulse volume, capillary refill, BP, and skin T°. Remember that BP is usually maintained until shock is advanced, so hypotension is a pre-terminal sign. Look for the effects of circulatory failure: tachypnoea, mottled cold skin, poor urine output (defined as <2mL/kg/hr in infants or <1mL/kg/hr in children aged >1y), agitation, and drowsiness.

Be alert for signs which might suggest a cardiac cause for the shock: cyanosis despite $O_2$, ↑ JVP, heart murmurs, and enlarged liver.

*Resuscitate* Give high-flow $O_2$ to all shocked patients. Gain IV/IO access; take blood samples, and give 20mL/kg of crystalloid. Reassess and repeat if necessary.

### Disability

Any problem with 'ABC' can affect 'D'.

*Assess* Conscious level. Initially, categorize according to AVPU scale:

A—Alert

V—responds to Voice

P—responds to Pain

U—Unresponsive

Check pupil size, reaction, and equality.

Assess GCS (or children's equivalent—see ➔ Assessing injuries in children, pp. 736–7) and posture (floppy, decerebrate, decorticate, etc.). Check BMG.

*Resuscitate* A child who does not respond to voice has an urgent need to have the airway secured. Treat hypoglycaemia and fits, and get senior help urgently.

### Exposure

Check T°. Expose to look for rashes (eg meningococcal disease, anaphylaxis) and for bruising/injuries.

### Reassessment

- After initial evaluation and intervention, follow this up with a more detailed approach to identify specific problems.
- Reassess ABCDE frequently to assess progress and detect deterioration.
- Undertake a secondary assessment by obtaining a full history (from the parents, paramedics, teachers, and witnesses) and undertaking a detailed physical examination.

# Standard immunization schedule

The UK Department of Health actively encourages immunization for children according to the standard schedule shown in Table 15.2 (see also https://www.gov.uk or *BNFC*). The recommended timing of the early immunizations is a compromise between trying to protect children whilst they are at most risk and delaying it until immunization is likely to be most effective. Children who have completed a course of immunization against a particular disease are obviously less likely to present with that disease.

Unfortunately, a significant proportion of children are still not receiving standard vaccines. Carefully enquire exactly which immunizations the child has received (information is often available from the child's GP or health visitor). Failure to follow the recommended schedule may result in the child presenting with an otherwise unusual disease.

**Table 15.2** Standard childhood vaccines

| Age | Vaccine |
|---|---|
| 2 months | Diphtheria, tetanus, pertussis, polio, Hib, meningococcal group B, rotavirus, hepatitis B |
| 3 months | Diphtheria, tetanus, pertussis, polio, Hib, rotavirus, hepatitis B, pneumococcal |
| 4 months | Diphtheria, tetanus, pertussis, polio, Hib, meningococcal group B, hepatitis B |
| 12–13 months | Measles, mumps, rubella, (MMR), pneumococcal, Hib, meningococcal groups B and C |
| 2–8y | Influenza |
| 3–5y | Diphtheria, tetanus, pertussis, polio, MMR |
| 11–14y | HPV (two doses 6–24 months apart) |
| 13–15y | Meningococcal groups A with C and W135 and Y vaccine |
| 13–18y | Tetanus, diphtheria, polio |

## Reactions to immunizations

Vaccination is frequently wrongly blamed for symptoms caused by an incidental viral illness. However, mild reactions, such as swelling and erythema at the injection site, are relatively common following administration of a variety of immunizations. These respond to symptomatic treatment and an expectant approach. Severe anaphylactic reactions involving airway obstruction or circulatory collapse are uncommon but require prompt and aggressive treatment (see Anaphylaxis, pp. 44–5).

### Immunization in other countries

If a child who normally lives outside the UK attends the ED, enquire carefully about their vaccination history. Likewise, if you are working outside the UK, make sure you know the local immunization schedule as this can have a significant impact on the type of communicable disease seen, particularly in children.

### Immunization in the ED

If a child attending the ED has not been immunized against diphtheria, tetanus, and pertussis and needs tetanus immunization, give the 'triple vaccine' (DPT) to avoid repeated injections. Inform the GP about any immunizations given.

## Expected child development

It is useful to know the standard paediatric milestones (see Table 15.3) in order to judge whether or not child development is at the expected level.

**Table 15.3** Paediatric milestones* (after allowance for preterm delivery)

| | |
|---|---|
| 2 months | Eyes follow movement. Smiles and makes noises when talked to |
| 3 months | Holds object placed in hand |
| 3–4 months | Turns head to sound |
| 6 months | Sits on floor with hands forwards for support<br>Transfers object from one hand to the other |
| 9–10 months | Crawls |
| 12 months | Walks with one hand held; says two or three words with meaning |
| 13 months | Walks unaided |
| 18 months | Makes tower of two or three bricks |
| 21–24 months | Joins two or three words together to make sentence |
| 2y | Can build a tower of six or seven bricks |
| 2½y | Knows full name and gender; can stand on tiptoes |

* See: Illingworth RS. *The Normal Child*, tenth edn. Churchill Livingstone, Edinburgh, 1991.

# Venous access and venepuncture

## Venepuncture

Needles frighten children. Topical anaesthetic cream (eg tetracaine—see Topical anaesthesia, p. 298) is useful whenever the need for blood sampling is not urgent. 4% tetracaine (Ametop®, amethocaine) anaesthetizes the skin and ↓ pain but should be applied for 30min before venepuncture and for 45min before cannulation. Identify prominent veins at two separate sites, apply cream and cover with an adhesive film dressing, then let the child play.

As in adults, if an IV cannula is inserted, it should be possible to obtain samples of blood via this—even if aspiration fails, blood will often drip out. The amount of blood sampled depends upon the size of the child and laboratory requirements, remembering that total blood volume is only 80mL/kg. Check requirements and obtain the appropriate bottles before attempting venepuncture.

*Neonates* FBC and U&E can be performed on capillary samples obtained from heel pricks. Ask an assistant to hold the foot and ankle firmly to encourage venous engorgement, then smear white soft paraffin on the heel and prick it with a lancet. Collect drops of blood into prepared capillary sample tubes.

*Toddlers and infants* Aspirate via a 23G butterfly needle in the hand or forearm. This allows the needle to stay in the vein, despite the child moving. Samples of 1mL are usually required.

*Older children* Use a 21G butterfly needle.

## IV cannulae

The route chosen to obtain venous access will depend upon the available veins and urgency of the problem. First attempt to insert an IV cannula percutaneously into an upper limb vein. Once inserted, flush the cannula, then secure it with adhesive tape, a splint, and bandage. In general, the following sizes of cannulae are appropriate:

- *24G (orange/yellow)*: neonates and infants.
- *22G (blue)*: toddlers and small children.
- *20G (pink) or 18G (green)*: older children.

Smaller cannulae are designed so the needle does not protrude much beyond the end of the cannula. This means that once a 'flashback' is obtained, the tip of the cannula may already be within the vein—advancing the needle further may puncture the other side of the vein and exit it. If attempts to insert a cannula into the hand or arm fail, it may be possible to use veins in the feet, ankle, or the scalp (useful in neonates, but first ensure that the intended target is not the superficial temporal artery). In an emergency, allow a maximum of 90s, and if still unsuccessful, then gain *IO access* (see Intra-osseous infusion, pp. 656–7), which is quick, easy, and reliable. Other venous access routes (eg central, femoral) require specialist training, are time-consuming, and are associated with significant complications (see Other routes of venous access, p. 655). Give fluids by infusion pump or paediatric infusion set to avoid over-transfusion.

## Other routes of venous access

*Femoral lines*

The femoral vein lies medial to the artery in the groin. It allows rapid venous access to be obtained and is particularly useful in cardiac arrest where physical constraints (eg several resuscitating staff) restrict access to the neck. Complications include sepsis (use strict aseptic technique), ↑ risk of thrombosis, and damage to other structures, including the hip joint. Femoral venous access may be achieved with or without USS guidance—the use of USS will depend upon resources, expertise, and the individual situation but may help to minimize the risks of complications.

*External jugular vein cannulation*

This is an option in children in whom spinal injury is not a concern. Place the child 15–30° head down and turn the neck to one side. The vein runs superficially and caudally over the sternocleidomastoid at the junction of its middle and lower third. Ask an assistant to compress the vein distally to distend and immobilize it.

*Central venous access*

The techniques (and complications) are similar to those in adults (see ➔ Central venous access, pp. 58–9), except that smaller equipment is needed. Safe insertion of central lines in children requires considerable experience and training, and is best achieved with USS guidance, which is sometimes unavailable in a resuscitation situation. Other routes may be more appropriate during initial resuscitation.

*'Cut-downs'*

The traditional site for cut-downs is the long saphenous vein at the ankle. Although this may remain an option during resuscitation, it can be time-consuming and so is rarely used, unless IO needles are not available. In infants, the long saphenous vein is located half a finger breadth superior and anterior to the medial malleolus. For children, it is situated one finger breadth superior and anterior to the medial malleolus.

*Umbilical venous access*

This can be useful in newborn resuscitation (see ➔ Venous access—the umbilical vein, p. 661).

# Intra-osseous infusion

If urgent venous access is required, but not obtained within 90s by percutaneous venous puncture, strongly consider using the IO route. Fluid and drugs given into the medullary cavity of long bones rapidly reach the central venous circulation. Gaining IO access is reasonably easy and can be performed quickly. It is particularly useful in young children but may be used in all ages, including adults.

## Indications

These include major burns, trauma, cardiac arrest, and septic shock.

## Contraindications

These include infection or fracture at (or proximal to) the insertion site, ipsilateral vascular injuries, multiple unsuccessful attempts, osteogenesis imperfecta, and osteopetrosis.

## Equipment

IO needles are usually of 16–18G and have a central metal stylet attached to a handle. A battery-powered mechanical driver (EZ-IO®) with paediatric or adult IO needles is available and can be used to insert the needle to a specific depth, possibly ↓ complications such as compartment syndrome. It can be used in babies weighing >3kg.

## Site of insertion

First choice is the proximal tibia 2.5cm below the tibial tuberosity on the flat anteromedial surface, thus avoiding the epiphyseal growth plate (see Fig. 15.1). If this route is not available, because of local infection or trauma, other options are: to use the distal femur (3cm above the lateral lower femoral condyle on the antero-lateral surface), the distal tibia (proximal to the medial malleolus), or the antero-lateral proximal humerus at the greater tuberosity.

## Manual intra-osseous needle insertion technique

- Support the limb on a pad or blanket.
- Sterilize the skin and use an aseptic technique. A small skin incision may be needed.
- Firmly grasp the handle and use a twisting motion to advance the needle and stylet through the cortex of the bone. (Note that some IO needles are designed with a thread and so require a rotatory, not an oscillatory, action.)
- Aim at 90° to the bone surface, or slightly away from the epiphyseal growth plate. Stop when the slight 'give' of the medullary cavity is felt.
- Remove the stylet and try to confirm correct placement by aspirating bone marrow (use this to check BMG or cross-match).
- If aspiration is not possible, the needle may still be correctly positioned—attach a primed 3-way tap with an extension set, and flush the needle and 3-way tap with 10mL of 0.9% saline, ensuring that there is no swelling of the surrounding soft tissues.
- It is most effective in children to give drugs and fluid by boluses using 20mL syringes and a 3-way tap.
- If necessary, immobilize with a POP backslab applied carefully to the posterior leg (eg for transport to PICU).

### Mechanical intra-osseous needle insertion technique

- Support the limb on a pad or blanket.
- Use a 15mm needle for patients weighing 3–39kg, a 25mm needle for patients weighing >39kg, and a 45mm needle for obese adult patients.
- Identify the insertion point and place the needle, loaded into the mechanical driver (drill), at the insertion point.
- Push the needle perpendicularly through the skin until reaching bone without drilling. Ensure that at least one 5mm marker line is visible above the skin. Press the button on the drill to turn it on, and push until a loss of resistance is felt.
- Remove the driver and stylet; attach a primed 3-way tap with an extension set, and flush the needle and 3-way tap with 10mL of 0.9% saline.
- Attach the giving set and secure as for manually inserted IO needles.

Use blood taken from an IO needle for BMG and culture or cross-matching, but not for FBC (automated blood counters may give spurious results). If using an IO needle in responsive patients, administer 0.5mg/kg of 2% lidocaine slowly into the needle before beginning the infusion to minimize pain.

### Complications of intra-osseous access

- Extravasation of fluid and compartment syndrome.
- Infection (cellulitis or osteomyelitis).
- Iatrogenic fracture.
- Fat or bone micro-emboli.
- Fractures and/or epiphyseal growth plate injury.

Ensure that IO needles are removed within 24hr to minimize the risk of infection and other complications. Aim to secure conventional IV access as soon as possible after IO needle insertion.

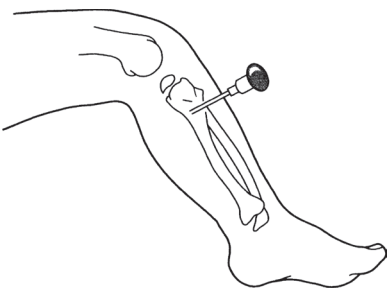

**Fig. 15.1** Tibial intra-osseous access.

# Resuscitation of the newborn

Neonatal resuscitation is usually undertaken by neonatologists, but unexpected deliveries require other personnel to initiate resuscitation. Fortunately, most newborn babies do not need resuscitation. The discomfort of being born into a hostile environment provides the major initial stimulus to breathe. Treat any baby requiring resuscitation in a warm room with an overhead heater. Call urgently for experienced help and start the clock.

## Approach (See Fig. 15.2.)

Make sure the umbilical cord is securely clamped, then dry the baby, remove wet towels, wrap the baby in dry towels, and put a hat on the baby. Assess breathing by chest movement (auscultation at birth is unreliable), heart rate (best assessed by a stethoscope placed over the apex), and tone in the limbs. Repeat assessments every 30s during resuscitation. The Apgar scores (ranging from 0 to 10, based upon assessment of heart rate, respirations, muscle tone, reflex irritability, and colour) at 1 and 5min are traditional, but do not delay resuscitation to calculate the score.

*A healthy baby* has good tone, cries within a few seconds of delivery, has a heart rate of 120–150/min, and becomes rapidly pink in the first 90s.

*A less healthy baby* has poorer tone and slower heart rate, and may not establish adequate respiration by 90–120s. The most sick are pale, floppy, apnoeic, and bradycardic.

## Airway

Open the airway by placing the baby's head in the neutral position (with the neck neither flexed nor extended). Because of the large occiput, a towel under the shoulders of the baby may help. Avoid hyperextension of the neck as this can occlude the pharyngeal airway. Consider a jaw thrust in very floppy babies. Remove visible meconium or secretions using a paediatric Yankauer sucker.

## Breathing

If the baby is not breathing adequately by 90s, give five inflation breaths (pressures of 30cm$H_2O$ for 2–3s). Aim to ventilate with air, but be guided in the use of $O_2$ by the 'pre-ductal' $O_2$ saturation (eg right hand) over time.

If the heart rate ↑, this indicates successful ventilation of the lungs. If the baby is apnoeic, continue ventilation at 30–40 breaths/min until self-ventilating. If the heart rate does not ↑, then the most likely cause is that the lungs have not been inflated. Recheck the head position and consider a jaw thrust and longer inflation time. Repeat the five inflation breaths and look for chest movement. If the heart rate remains <60/min or absent despite good chest movement, start chest compressions.

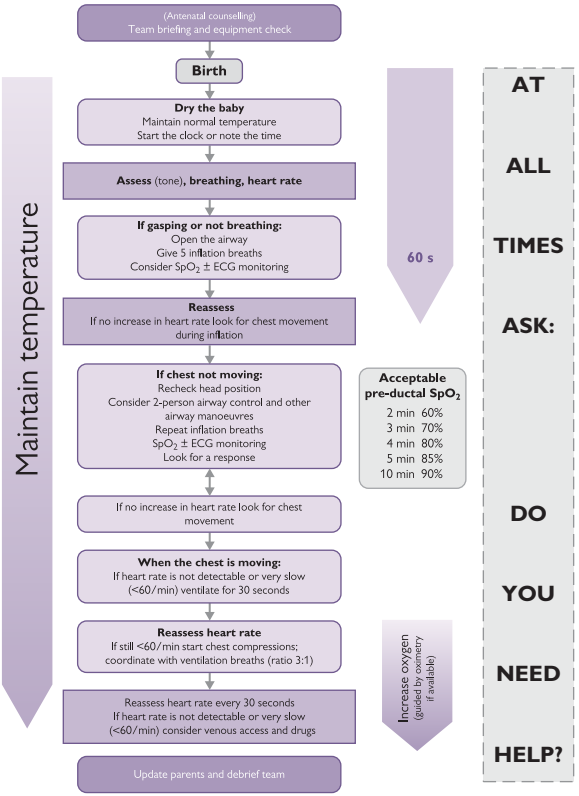


**Fig. 15.2** Algorithm for newborn life support 2015.

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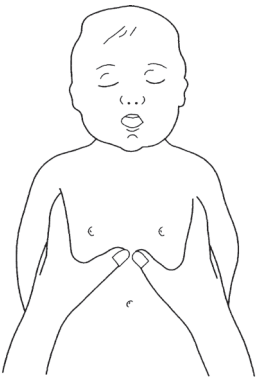
# CPR of the newborn

## Chest compressions

(Only start after successful lung inflation.)

Grip the chest in both hands in such a way that two thumbs can press on the lower third of the sternum (just below the intermammary line), with the fingers over the spine at the back (see Fig. 15.3). Overlapping thumbs may give better compressions but is more tiring to perform. Aim to depress the AP diameter of the chest by a third.

Use a chest compression to inflation *ratio of 3:1*. Aim for a rate of 120 events (90 compressions + 30 ventilations) per minute.



Using encircling fingers

**Fig. 15.3** Method of CPR in the newborn.

## Tracheal intubation

Treat continuing apnoea with tracheal intubation using a 3mm tube (2.5mm in premature babies). Precede intubation by pre-oxygenation with bag–valve–mask ventilation for 30s.

## Meconium

Meconium (green) staining of liquor is quite common, but true meconium aspiration is actually quite rare. The previous practice of focussing upon aggressively trying to clear meconium from the airway at an early stage has not been shown to be of benefit and might be detrimental by delaying basic resuscitation efforts. Reserve attempts to visualize the oropharynx and aspirate meconium for those patients where meconium appears to be so thick as to block the airway.

## Drugs

Only use drugs if there is no significant cardiac output despite effective lung inflation and chest compression. Give drugs IV via an umbilical vein catheter or an IO needle.

- *Give adrenaline* 10mcg/kg (0.1mL/kg of 1 in 10,000) if there is no initial response; if this is ineffective, consider ↑ dose to 30mcg/kg (0.3mL/kg of 1 in 10,000).
- *Consider giving sodium bicarbonate* 1–2mmol/kg (2–4mL of 4.2% solution/kg) when there is no cardiac output despite all resuscitative efforts or in profound and unresponsive bradycardia.
- *Hypoglycaemia*: a potential problem for all newborns, and BMG may be unreliable when reading <5mmol/L. Take blood sample to confirm, and treat immediately with a bolus of 2.5mL/kg of 10% glucose.
- *Suspect hypovolaemia if* very pale baby, PEA, history of antepartum haemorrhage, placenta praevia or vasa praevia, or unclamped cord. Give 10mL/kg of 0.9% saline, followed by O –ve (and CMV –ve) blood, repeated as necessary.
- *Atropine and calcium* have no role in newborn resuscitation.

## Venous access—the umbilical vein

The easiest and fastest method of obtaining venous access in the newborn is to cannulate the umbilical vein. Identify the umbilical vein in the cut umbilical stump—it is the single large dilated vessel adjacent to the two constricted arteries (see Fig. 15.4). Prepare a 5F gauge catheter with 0.9% saline, and insert it 5cm into the umbilical vein. Suture and secure in place.

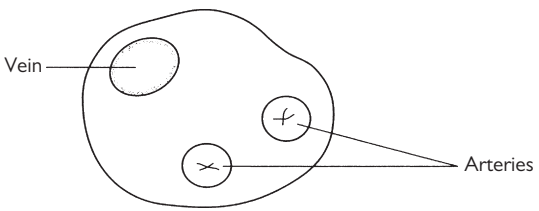



**Fig. 15.4** Diagram of a cross-section of the umbilicus.

## Stopping resuscitation efforts

If there are no signs of life after 10min of continuous and adequate resuscitation efforts, then discontinuation of resuscitation may be justified. Senior ED and neonatal staff will be involved in making this decision.

# Paediatric Basic Life Support

Follow the algorithm (see Fig. 15.5). For choking, see ➲ Choking from a foreign body, p. 664.

### Evaluate responsiveness

Gently stimulate and ask loudly, 'Are you alright?' If the child does not respond, shout for help ± get someone to go for assistance.

### Open airway

Open the airway by head tilt and chin lift. Desirable degrees of tilt are neutral if <1y and 'sniffing the morning air' if >1y. Do not press on the soft tissues under the chin, as this may block the airway. If it is still difficult to open the airway, try a jaw thrust. If there is any suspicion that there may have been a neck injury, instruct a second rescuer to manually immobilize it, and use either chin lift or jaw thrust alone. If this is unsuccessful, add the smallest amount of head tilt needed to open the airway.

### Check breathing

Whilst keeping the airway open, look, listen, and feel for breathing for 10s. If the child is not breathing or is making infrequent irregular breaths, carefully remove any obvious obstruction, and give five initial rescue breaths (with the rescuer taking a breath between each rescue breath).

### Rescue breaths

*For children (>1y)* Whilst maintaining head tilt and chin lift, give breaths mouth-to-mouth, pinching off the nose. Blow steadily for ~1s, watching for the chest to rise. Take your mouth away; watch the chest fall, and repeat this sequence five times.

*For infants (<1y)* Ensure the neutral position of the head and apply chin lift. Give mouth-to-mouth and nose breaths, ensuring a good seal. Blow steadily for ~1s, watching for the chest to rise. Take your mouth away; watch the chest fall, and repeat this sequence five times.

Difficulty achieving an effective breath suggests airway obstruction. Reposition head tilt/chin lift, and try again. If still unsuccessful, attempt with a jaw thrust instead. Try up to five times to give effective breaths. If still unsuccessful, consider the possibility of FB airway obstruction.

### Check pulse

Over the next 10s, check for signs of life—any movement, coughing, or normal breathing, and check for a pulse (use the carotid for >1y and the brachial for those <1y). If there are no signs of life and/or no pulse or pulse <60/min with poor perfusion or you are unsure—start chest compression.

### Chest compression

*For infants (<1y)* Perform chest compressions (100–120/min) by placing both thumbs flat, side by side, on the lower third of the sternum, with the tips pointing towards the infant's head. Encircle the rib cage with the tips of the fingers supporting the infant's back. Press down with the thumbs at least one-third of the depth of the chest (or by ~4cm).

*In children (>1y)* Using the heel of one hand, compress the lower half of the sternum by at least one-third of the depth (or by ~5cm) of the chest at a rate of 100–120/min. Use two hands, if necessary, to achieve this.

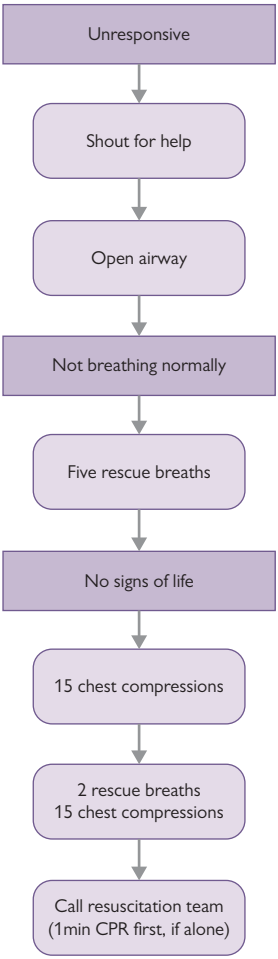



**Fig. 15.5** Paediatric Basic Life Support 2015 (health care professionals with a duty to respond).

# Choking from a foreign body

Despite preventative measures (eg making pen tops with holes in them), children continue to die each year from airway obstruction due to FB impaction. FB aspiration produces a sudden-onset airway problem, which helps to distinguish it from other causes of airway obstruction (epiglottitis, bacterial tracheitis—see ➲ Stridor: upper respiratory tract infections, pp. 692–3), which may be worsened by the basic measures described below.

The majority of choking events in children are witnessed and occur during play or whilst eating. FB airway obstruction is characterized by sudden onset of respiratory distress associated with coughing, gagging, or stridor, with no other signs of illness. If the child is coughing effectively (fully responsive, loud cough, able to take a breath before coughing, crying or verbal response to questions), encourage coughing and observe for the cough becoming ineffective.

## Conscious, but ineffective cough

If conscious with an ineffective cough, give five back blows. In the infant, support in a head-downward prone position, and in the child, aim for a head-down or forward-leaning position. Deliver five sharp *back blows*, with the heel of one hand centrally between the shoulder blades. If ineffective, turn to the supine position and give five *chest thrusts* to infants (using the same landmarks as for CPR), but thrusts are sharper and delivered at a slower rate, and *abdominal thrusts* to children >1y. Perform *abdominal thrusts* from behind the child, placing your fist between the umbilicus and the xiphisternum and grasping it with your other hand, then pulling sharply inwards and upwards—repeat up to five times.

Following chest or abdominal thrusts if the object has not been expelled and the victim is still conscious, then repeat the sequence of back blows and chest (for infant) or abdominal (for children) thrusts.

Do not use abdominal thrusts for infants.

## Unconscious from foreign body airway obstruction

If a child with FB airway obstruction is or becomes unconscious, place him on a flat surface, then open the mouth and look for any obvious object. If one is seen, use a single finger sweep to remove it. It may be possible to remove the FB with Magill's forceps under direct laryngoscopy. Do not attempt blind or repeated finger sweeps. Open the airway and attempt five rescue breaths. If a breath does not make the chest rise, reposition the head before making the next attempt. If there is no response whilst attempting the five rescue breaths, proceed to chest compression with ventilation using a ratio of 15:2. Each time the airway is opened, check for a FB, and if visible, try to remove it (see Fig. 15.6).

If the obstruction appears to have been relieved, open and check the airway. If the child is not breathing, deliver rescue breaths. If initial measures prove unsuccessful and the child is hypoxic, consider a *surgical airway* (eg needle cricothyroidotomy if aged <12y, surgical cricothyroidotomy if older—see ➲ Airway obstruction: surgical airway, p. 336).

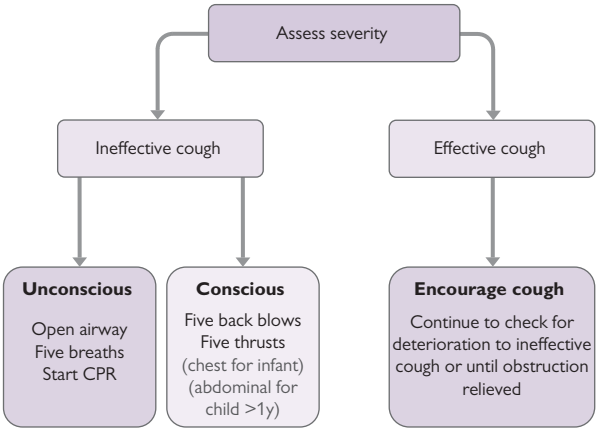



**Fig. 15.6** Paediatric foreign body airway obstruction treatment 2015.
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# Anaphylaxis in children

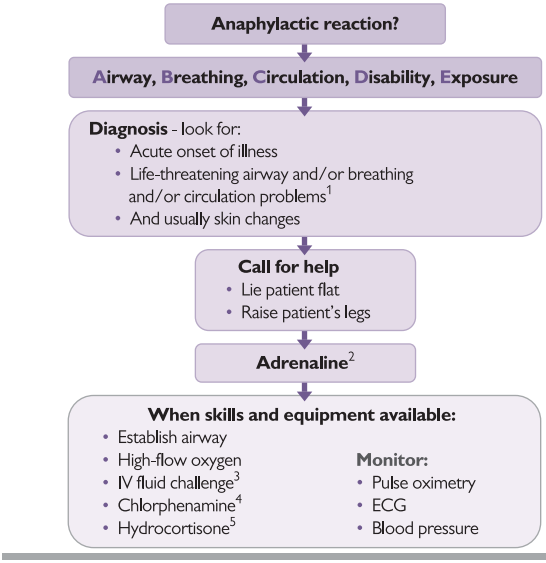
The background, causes, and pathophysiology of anaphylaxis in children is similar to that in adults (see ➲ Anaphylaxis, pp. 44–5). Treat according to the 2015 UK Resuscitation Council algorithm, shown in Fig. 15.7. After initial treatment, admit the child for observation in case of a delayed or biphasic reaction.

## Notes for anaphylaxis algorithm

1 IM adrenaline is the agent of choice in anaphylaxis and should be administered without delay.
2 If profound shock is judged immediately life-threatening, consider giving a slow bolus of 1mcg/kg of IV adrenaline as a *1 in 100,000* solution (= 10mcg/mL solution). *This is hazardous* and is recommended *only* for experienced specialists who can also obtain IV access without delay. Note that a different dilution of adrenaline is required for IM, compared to IV, use. Adrenaline can also be given via the IO route in the same dose as the IV route.
3 An inhaled $\beta_2$-agonist, such as salbutamol, may be used as an adjunctive measure if bronchospasm is severe and does not respond rapidly to other treatment.
4 For children who have been prescribed an EpiPen®, 150mcg can be given instead of 120mcg, and 300mcg can be given instead of 250mcg or 500mcg.
5 A crystalloid may be safer than a colloid.
6 Do not use the SC route for adrenaline. It has no role in anaphylaxis because its absorption is appreciably slower than IM adrenaline.

Consider taking blood samples for mast cell tryptase testing as soon as possible after starting treatment if the cause is thought to be venom-related, drug-related, or idiopathic (see 🔗 https://www.nice.org.uk/cg134):

- A sample as soon as possible after emergency treatment has started.
- A second sample ideally within 1–2hr (but no later than 4hr) from the onset of symptoms.




**1 Life-threatening problems:**

| | |
|---|---|
| **Airway:** | Swelling, hoarseness, stridor |
| **Breathing:** | Rapid breathing, wheeze, fatigue, cyanosis, $SpO_2$ <92%, confusion |
| **Circulation:** | Pale, clammy, low blood pressure, faintness, drowsy/coma |

**2 Adrenaline** (*give IM unless experienced with IV adrenaline*)
IM doses of 1:1000 adrenaline (repeat after 5 min if no better)

| | |
|---|---|
| • Adult: | 500 micrograms IM (0.5mL) |
| • Child more than 12 years: | 500 micrograms IM (0.5mL) |
| • Child 6–12 years: | 300 micrograms IM (0.3mL) |
| • Child less than 6 years: | 150 micrograms IM (0.15mL) |

Adrenaline IV to be given **only by experienced specialists**
Titrate: Adults 50 micrograms; Children 1 microgram/kg

**3 IV fluid challenge:**
Adult—500–1000mL
Child—crystalloid 20mL/kg

Stop IV colloid
if this might be the cause of anaphylaxis

| | **4 Chlorphenamine** (IM or slow IV) | **5 Hydrocortisone** (IM or slow IV) |
|---|---|---|
| Adult or child more than 12 years: | 10mg | 200mg |
| Child 6–12 years: | 5mg | 100mg |
| Child 6 months to 6 years: | 2.5mg | 50mg |
| Child less than 6 months: | 250 micrograms/kg | 25mg |

**Fig. 15.7** UK Resuscitation Council algorithm 2015.
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# Paediatric Advanced Life Support

Overall, cardiac arrest in children has a worse outcome than in adults, because the causes are different. Early recognition of deterioration is crucial to prevent cardiac arrest.

## Initial approach

Follow the Resuscitation Council (UK) guidelines (https://www.resus.org.uk) in Fig. 15.8. Assess and treat according to ABCDE. Establish BLS (see Paediatric Basic Life Support, p. 662). Ventilate with $O_2$ using a bag–valve–mask, then deliver compressions at a rate of 100–120/min, with a compression:ventilation ratio of 15:2. Perform tracheal intubation if expertise allows (with minimal interruption to chest compressions)—this will secure the airway and allow uninterrupted compressions (except during pulse checks and defibrillation) and ventilation at 10–12/min. Capnography can help to reassure that the tracheal tube is in the tracheobronchial tree and may provide information about the quality of CPR. A sudden ↑ in end-tidal $CO_2$ ($ETCO_2$) may provide an early indication of ROSC.

## Non-shockable rhythms: PEA and asystole

Focus on giving good-quality BLS with high-concentration $O_2$ and minimal interruptions. Give adrenaline IV/IO 10mcg/kg (0.1mL/kg of 1 in 10,000), repeated every 3–5min (every other loop) whilst in arrest.

Search carefully for reversible causes of the arrest—in particular, exclude tension pneumothorax and consider hypovolaemia. Septic shock, haemorrhage, and dehydration are implicated relatively frequently, so consider an initial IV fluid bolus of 20mL/kg of 0.9% saline at an early stage in the resuscitation. Follow this with further IV fluid and/or blood as appropriate.

## Shockable rhythms: VF and pulseless VT

VF is uncommon in children but occasionally occurs in children with congenital heart disease. Ensure delivery of good-quality CPR—only interrupt chest compressions and ventilation for defibrillation. When selecting the energy level to use during defibrillation, if the defibrillator can only deliver certain predetermined 'stepped' shocks, choose the nearest higher 'step' to that required. Paddles (or pads) for children are 8–12cm in size (4.5cm in infants). After shock delivery, resume CPR immediately without checking for a pulse and continue for 2min unless there is a response from the patient to indicate ROSC. If VF/pulseless VT persists, give IV adrenaline 10mcg/kg and IV amiodarone 5mg/kg (diluted in 5% glucose) after the third shock. Continue shocks every 2min if a shockable ryhthm continues; give further IV adrenaline every 3–5min, and consider a further dose of IV amiodarone (5mg/kg) after the fifth shock.

## Parents present during resuscitation

Whenever possible, give parents the chance to stay with their child during resuscitation. If parents wish to be present, ensure a dedicated staff member stays with them throughout and explains what is happening.

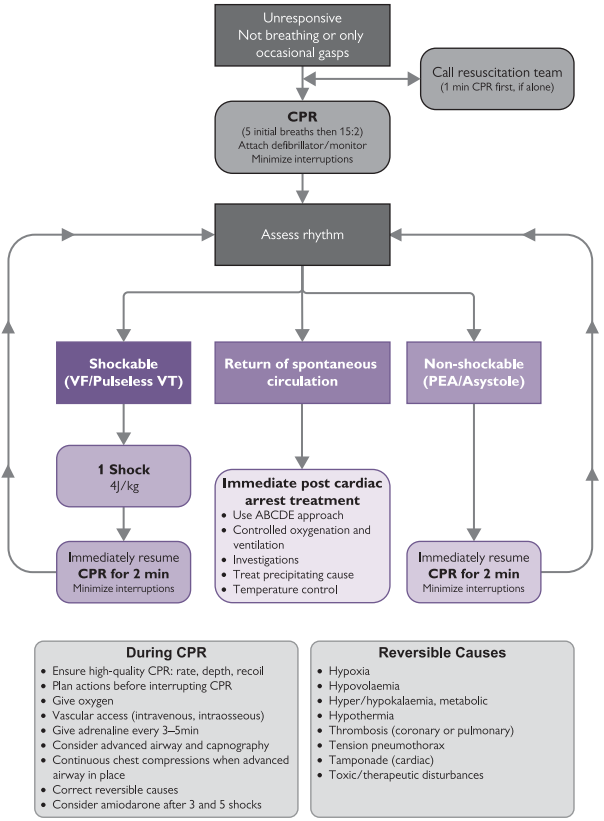


**Fig. 15.8** Paediatric Advanced Life Support 2015.

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# Notes on paediatric ALS

## Airway

*$O_2$* Give high-flow $O_2$ (use a well-fitting mask with a reservoir).

*Suction* Use a rigid suction catheter to aspirate pharyngeal contents.

*Oropharyngeal airway* An airway may help when ventilating with a bag–valve–mask device, whilst personnel and equipment are prepared for tracheal intubation. Size the airway by matching its length to the distance between the central incisor teeth and the angle of the mandible. Use a tongue depressor or laryngoscope to displace the large tongue, and insert the airway the 'right way up' in order to avoid trauma to the palate.

*Bag–valve–mask ventilation* Attach high-flow $O_2$ to a self-inflating bag–valve–mask device. Use a 500mL (up to age 1y) or 1600mL bag (>1y).

*Tracheal intubation* This method of securing the airway requires experience and practice. Call for senior help. Always use a capnograph. Follow the same technique as that described for adults (see ➲ Airway obstruction: basic measures, pp. 334–5), except:

- Use a straight-bladed laryngoscope in infants (<1y).
- Use the correct size of ET tubes in children, either cuffed or uncuffed.
- Correct size of ET tube:

$$\text{Internal diameter (mm)} = (\text{age in years}/4) + 4$$

## Equipment sizes, drugs, and doses

Become familiar with, and use, the Broselow tape (see Box 15.1 for key formulae).

*Venous access* First attempt to secure peripheral venous access. If this is not obtained within 90s, attempt IO access (see ➲ Intra-osseous infusion, pp. 656–7).

*High-dose adrenaline* Not recommended and may be harmful.

*Atropine* 20mcg/kg (minimum dose 100mcg, max 600mcg)—may be used for patients with bradycardia related to ↑ vagal tone. There is no evidence of efficacy for atropine.

*Magnesium* Indicated for polymorphic VT or documented hypomagnesaemia—give 25–50mg/kg over several minutes to a max of 2g.

*Calcium chloride* 0.2mL/kg of 10% solution—given for hypocalcaemia, hyperkalaemia, and clinically severe overdose of calcium channel-blocking drugs. Do not give in the same IV/IO line as bicarbonate.

*Sodium bicarbonate* Not recommended routinely, but consider it in prolonged arrest, hyperkalaemia, and arrhythmias associated with tricyclic antidepressant overdose. The dose is 1–2mL/kg of 8.4% solution IV/IO. Ensure adequate flushing after giving it. Avoid mixing with other agents (it inactivates adrenaline and precipitates out calcium).

*Glucose* Treat hypoglycaemia with IV glucose (2mL/kg of 10% glucose).

*IV fluids* Give a 20mL/kg IV normal saline bolus where cardiac arrest is secondary to hypovolaemia or sepsis.

## Discontinuing resuscitation

Resuscitation efforts are unlikely to be successful if there is no ROSC at any time after 30min of life support and in the absence of recurring or refractory VF/VT. Prolong resuscitation for patients who are hypothermic or who may have been poisoned.

## Paediatric resuscitation chart (See Fig. 15.9.)

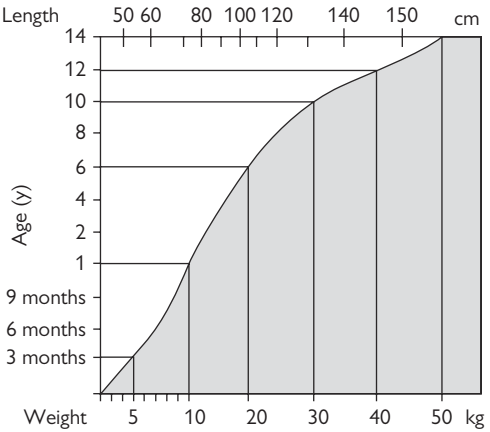



**Fig. 15.9** Paediatric resuscitation chart.

### Box 15.1 Key resuscitation formulae

Assume a birthweight of 3.5kg, reaching 10kg by the end of the first year.

- Weight in kg = (age in years + 4) × 2 [works for ages 1–10y]
- Tracheal tube internal diameter in mm = (age/4) + 4
- Tracheal tube length (oral) in cm = (age/2) + 12

Laryngeal mask sizes for children:

- Size 1 for weight up to 5kg.
- Size 1.5 for weight 5–10kg.
- Size 2 for weight 10–20kg.
- Size 2.5 for weight >20kg.

Defibrillation for VF/pulseless VT = 4J/kg.

### Drug doses

- *Glucose in hypoglycaemia*: 2mL/kg of 10%.
- *Adrenaline IV in cardiac arrest*: 0.1mL/kg of 1 in 10,000.
- *Lorazepam IV for seizures*: 0.1mg/kg.
- *Diazepam PR for seizures*: 0.5mg/kg.
- *Midazolam buccal for seizures*: 0.5mg/kg.
- *Phenytoin IVI for continuing seizures*: 20mg/kg IVI over 30min.
- *Morphine for pain*: IV 0.1–0.2mg/kg (titrated according to pain).

# Children with tachyarrhythmias

Arrhythmias are uncommon in children—*obtain expert advice* at an early stage. Children may present with poor feeding, heart failure, shock, or palpitations. Sinus tachycardia may be as fast as 220/min in infants and 180/min in children. Consider undiagnosed congenital heart disease in infants.

## SVT in children

Distinguish SVT from sinus tachycardia. In sinus tachycardia, the heart rate is <200/min, P waves are upright in ECG leads I and $aV_F$, there is beat-to-beat variation in rate, and the history is consistent with shock.

### *SVT with no shock*

In the *absence of clinical evidence of shock*, try *vagal stimulation*: immersion of the face in iced water, or Valsalva manoeuvre, or unilateral carotid massage. If this is unsuccessful, give adenosine 100mcg/kg IV rapidly, followed by a saline flush, followed, if still unsuccessful, by further dose(s) at 200mcg/kg, then 300mcg/kg. If this fails, seek expert help and consider IV amiodarone or propranolol or synchronized DC shock with appropriate anaesthesia.

### *SVT with shock*

If *clinically shocked*, but responsive, try vagal manoeuvres and obtain expert help to give synchronized shocks (starting at 1J/kg, ↑ if unsuccessful to 2J/kg) under anaesthesia. If there is any delay, try adenosine IV, as outlined for haemodynamically stable patients.

## Ventricular tachycardia in children

Until proved otherwise, initially consider wide-complex tachycardia in children to be VT (as opposed to SVT with bundle branch block).

### *Causes of VT in children*

These include: hyperkalaemia, long QT syndrome, congenital heart disease, myocarditis, and cardiomyopathy. Tricyclic poisoning (see ➲ Tricyclic antidepressant poisoning, pp. 202–3) often produces tachycardia and wide QRS resembling VT.

### *Management*

Address ABCDE, then treat according to the clinical condition:

- *If the child with VT is clinically shocked, but conscious*: arrange urgent anaesthesia, followed by synchronized DC shocks, starting at 2J/kg (followed, if necessary, by 4J/kg).
- *If the child is not clinically shocked*: involve a (paediatric) cardiologist and consider amiodarone 5mg/kg IVI over 30min.
- *Torsades de pointes*: treat with magnesium sulfate IVI 25–50mg/kg (up to a maximum of 2g), but seek expert guidance.

# Children with bradyarrhythmias

## Background

Heart rates of <60/min in children are usually seen as part of a pre-terminal sequence of events in response to profound hypoxia and ischaemia. However, bradycardia can occur in children due to ↑ ICP or poisoning (eg β-blockers or digoxin). In addition, some very athletic fit older children may normally have low resting heart rates.

## Management

Look for, and treat, the underlying cause. If the bradycardia is the result of vagal stimulation (eg tracheal intubation or tracheal suctioning), give IV/IO atropine 20mcg/kg (minimum dose 100mcg, maximum dose 600mcg).
If the child is shocked and hypoxic and has a heart rate of <60/min:

- Get expert help.
- Assess ABCDE.
- Give $O_2$ and ventilate as necessary.
- Give fluid bolus IV 20mL/kg and repeat as required.
- If these steps are ineffective, give adrenaline 10mcg/kg IV, and if the response to this is not satisfactory, start an adrenaline IVI of 0.05–2mcg/kg/min.

# Sudden infant death syndrome

Sudden infant death syndrome (SIDS) (also called SUDI—sudden unexplained death in infancy; previously known as 'cot death' or 'crib death') is ↓ in incidence, but each death is a tragedy. A senior doctor (consultant) should manage distressed parents (and staff). It remains a leading cause of infant death (1 in 2000 live births), with 90% occurring between 1 and 6 months of age. Most hospitals now have detailed SUDIC (sudden unexpected death in infancy and childhood) protocols, which should be followed in this situation.

## Definition

Sudden death in infancy with no cause identified after autopsy.

## Aetiology

Although the aetiology is unknown, a variety of theories have been proposed, including prone sleeping position, airway obstruction, apnoea, viral illness, and overheating.

## Risk factors

Passive smoking, ♂, winter months, sleeping prone, premature babies, twins, apnoeic spells in first week of life, lower socio-economic groups, maternal illicit drug abuse in pregnancy, sibling with SIDS, and co-sleeping (especially if parent has been drinking alcohol).

## Prevention

- Avoid overheating (aim for ambient T° of 16–20°C).
- Avoid duvets and excess bedding in infancy.
- Place infant's feet at cot end to prevent migration under blankets.
- Sleep supine (unless Pierre–Robin, scoliosis, or oesophageal reflux).
- Consider apnoea alarm.
- Avoid infant sharing bed with parent.

## Approach

- Take the infant into the resuscitation room, and continue resuscitation as for cardiac arrest unless there is post-mortem staining or rigidity.
- Call the ED consultant and consultant paediatrician.
- Ensure that a named senior nurse stays with the parents.
- Immediately after death is declared, prepare yourself, then inform the parents in the presence of the senior nurse. Use the techniques described in ➲ Breaking bad news, pp. 26–7. Refer to the child throughout by their first name.
- Some hospitals have dedicated bereavement counsellors—involve them early.
- Allow the parents to see and hold the baby, and suggest that they keep a lock of their hair.
- Take digital or polaroid photographs of the baby—give them to the parents and file copies in the notes.
- Explain further procedures (eg autopsy) to the parents and provide written information, eg '*A guide to the post mortem examination procedure involving a baby or child*' (Department of Health, ref 29768/A).

- Offer to request a minister of religion and involve a social worker.
- Careful documentation, including general appearance, state of nutrition, weight, rectal T°, marks from procedures, rashes, any visible injuries, and appearance of the retinae. Inform the GP to arrange to visit the parents and discuss whether to suppress lactation with bromocriptine if the mother is breastfeeding.
- Retain clothes and bedding (stored in a paper bag, not polythene), and inform the police and coroner (Procurator Fiscal in Scotland) in all cases.
- Ensure blood, urine, and skin specimens will be obtained (looking for infection and inborn errors of metabolism).
- Arrange a further appointment for the parents with the same consultant paediatrician.
- Suggest The Lullaby Trust (https://www.lullabytrust.org.uk) which offer bereavement support for families (tel: 0808 802 6868), as well as advice regarding 'safer sleeping' and training and help for professionals.
- Advise about preventative measures for siblings. If the baby was a twin, recommend admission of the surviving twin with the mother for monitoring and investigation.
- Cancel any hospital outpatient appointments and vaccination appointments for the child.
- Inform the parents that the police will visit them as a matter of course.
- Finally, consider yourself and your colleagues.

### Staff have feelings too

All staff involved with the child and family (ambulance staff, police, GP, nurses, and doctors—including you) will be traumatized by the experience. Those who are themselves parents with young children may be particularly distressed. At the very least, a debriefing session over a cup of coffee will be required.

### 'Near miss sudden infant death syndrome' (apparent life-threatening event)

Refer to the paediatrician for admission and monitoring any infant whose parents report an apparently life-threatening event ('ALTE'): apnoea, colour change, tone change, cyanosis, choking, and gagging. Note that the term Brief Unresolved Unexplained Event (BRUE) is an alternative way of describing this. The patient may appear well at the time of presentation. Liaise with the paediatric team, and take blood (to include FBC, U&E, glucose, $Ca^{2+}$, $Mg^{2+}$, and phosphate) and admit for apnoea monitoring.

The *differential diagnosis* includes arrhythmias and congenital heart disease, child abuse, gastro-oesophageal reflux, meningitis and sepsis, seizures, and metabolic disorders. In 50% of cases, no cause is found. Despite parental anxiety, short apnoeic episodes (<15s) may, in fact, be entirely normal. Theophylline, home monitoring devices, and counselling have all been used for infants believed to be at risk.

# Problems of neonates and infants

## Neonatal cephalohaematoma

This haematoma results from birth trauma and overlies a single skull bone (usually parietal). It resolves spontaneously—do not attempt to aspirate.

## Umbilical cord sepsis

The dried cord separates at 1 week. If the stump develops signs of infection (becoming moist and red), refer to the paediatrician.

## Breast swelling

Neonatal breasts commonly swell, due to exposure to maternal hormones. Occasionally, these breasts lactate ('witch's milk') and very occasionally become infected, requiring parenteral antibiotics.

## Neonatal jaundice

Jaundice within 24hr of birth is highly abnormal. Neonates who develop jaundice after 24hr mostly have 'physiological jaundice' (typically in the first week, especially premature babies) or 'breast milk jaundice' (typically in second week—self-limiting, breastfeeding can usually continue). Refer all patients to exclude serious underlying disorders: Rh haemolytic disease, ABO incompatibility, congenital spherocytosis, glucose-6-phosphate dehydrogenase (G6-PD) deficiency, CMV infection, hypothyroidism, and biliary atresia. The paediatrician will check: serum bilirubin (including ratio of conjugated:unconjugated), FBC, blood film, U&E, LFTs, direct antiglobulin test, TFTs, and infection screen.

## Neonatal conjunctivitis

A watery/sticky eye in the first few days of life may be due to an unopened tear duct, or occasionally due to gonococcal or chlamydial infection acquired from the mother's genital tract. Therefore, take a swab for Gram staining for gonococci and culture for *Chlamydia*. Refer the baby and mother if organisms are demonstrated; otherwise arrange GP follow-up.

## Sepsis

Potentially life-threatening sepsis (eg meningitis) may present in a non-specific manner in infants (this is especially true of neonates). Classic presentations are replaced by: feeding problems, irritability, drowsiness, jaundice, hypotonia, poor weight gain, petechiae or skin rash, apnoea, bradycardia, and cyanotic episodes. Neonates at ↑ risk are those with low birthweight, those previously ventilated, and those with congenital abnormalities.

*Treatment* Give $O_2$ and IV fluids (20mL/kg). Refer for admission and urgent investigation: BMG, urine culture, FBC, blood cultures, TORCH screen (*Toxoplasma*, rubella, CMV, herpes), CXR, abdominal X-ray (if necrotizing enterocolitis suspected), and LP. Commence 'blind' antibiotics (see *BNFC*).

### Crying babies

It is quite normal for babies to cry. The amount of crying varies enormously, as does the ability of parents to cope with it. With more acute onset of irritability and crying, exclude an acute cause (eg otitis media, incarcerated hernia, testicular torsion, intussusception, fractured limb), before reassuring and counselling the parents. Parents who are driven to despair may benefit from a self-help group (eg CRY-SIS, tel: 08451 228 669, https://www.cry-sis.org.uk) or follow-up with a paediatrician.

### Feeding difficulties

Parents bring their babies to the ED with a variety of feeding problems. The underlying causes vary widely and range from acute life-threatening sepsis to chronic parental anxiety or overfeeding. Obtain a careful feeding history and watch the baby feed. Babies normally require at least 15mL of milk/kg/day on day 1, ↑ to ~150mL/kg/day by day 7. Plot the weight, height, and head circumference on centile charts. Take weight loss or failure to satisfactorily gain weight seriously—it may be due to a significant underlying disorder (eg pyloric stenosis). Remember that newborn babies lose up to 10% of their birthweight in the first week but should regain it by 2 weeks. Arrange for the health visitor to advise. Refer chronic feeding problems to the GP or paediatrician.

### Bilious vomiting

Occasionally neonates and infants present with bilious vomiting, a sign of serious pathology. The most important differential diagnosis is intestinal malrotation (volvulus) secondary to peritoneal bands, which requires emergency laparotomy to avoid total small bowel infarction (see ➲ Abdominal pain in children, pp. 720–1). Consult a paediatric surgeon urgently. Other differential diagnoses include an obstructed hernia, Hirschsprung's disease, and sepsis.

### Metabolic diseases (inborn errors of metabolism)

Occasionally neonates present to the ED days after birth with coma or seizures with no obvious cause (infection, trauma, hypoglycaemia, etc.). These infants may have an inborn error of metabolism (e.g. maple syrup urine disease, urea cycle disorders, and hyperammonaemia), and they require urgent specialist paediatric care, often at a tertiary children's hospital. If an older child presents to the ED with a previously diagnosed metabolic disease, seek expert advice by referring to the paediatrician early. Remember that the parents are almost certain to know more about the disease than the doctors and nurses in the ED.

*Treatment* Give $O_2$ and IV fluids (20mL/kg), and treat hypoglycaemia and sepsis. Refer for admission and urgent investigation by a paediatrician. Emergency treatment protocols for this challenging group of patients are available in an easily accessible form at the British Inherited Metabolic Diseases Group website (http://www.bimdg.org.uk).

# Skin problems in infants

Minor skin problems are common in infants. The combination of a skin rash and an ill infant should arouse suspicion of serious illness (eg ➲ Meningococcal disease, pp. 682–3) and prompt urgent referral. *Do not discharge an infant with an undiagnosed rash*—obtain an expert opinion.

## Neonates

Multiple tiny white papules (*milia*) seen on the face of neonates are superficial epidermal inclusion cysts. Erythematous lesions with central white vesicles are common in the first days of life—*erythema toxicum* ('neonatal urticaria'). Both are harmless and disappear spontaneously within days.

## Peeling skin

Peeling skin is a common feature of post-mature babies and should be distinguished from scalded skin syndrome and Kawasaki disease (➲ Skin lesions in multisystem disease, pp. 688–9).

## Scalded skin syndrome ('toxic epidermal necrolysis')

This staphylococcal infection results in red, peeling skin, sometimes with blistering. Refer for admission and IV antibiotics.

## Eczema

Usually managed most appropriately by the GP and outpatient department with emollients ± topical corticosteroids, but if very severe, refer for a period of inpatient treatment. Sometimes the scratched skin becomes secondarily infected, requiring admission for IV antibiotics.

## Impetigo

Any breach in the skin (eg eczema, nappy rash, scabies) may develop impetigo. Staphylococcal or streptococcal infection results in an ulcerative, erythematous area, which forms a golden brown crust that spreads rapidly. If the infection is localized and the child is well, treat with topical fusidic acid (if extensive—PO flucloxacillin); arrange GP follow-up, and advise the parents to isolate the child from other children until it has resolved. If the child is unwell, refer for IV antibiotics.

## Nappy rash ('ammoniacal dermatitis')

Erythema with some ulceration in the nappy area, but sparing the flexures, is usually the result of excessive moisture contact with the skin. Treat by exposure to fresh air as much as practicable and frequent changing of nappies. Consider barrier creams (see *BNFC*).

## Monilial infection

Nappy rash may become infected with *Candida albicans*, leading to erythema of the flexures. Give nystatin cream, and advise regular changing.

## Seborrhoeic dermatitis

This erythematous, greasy rash commonly involves the nappy area, the occipital region, and behind the ears. It may become infected with *Candida albicans*—treat with nystatin and refer to the GP.

# Febrile illness in preschool children

Febrile illness is extremely common in childhood. Search for serious causes requiring treatment (meningitis, encephalitis, pneumonia, UTI, arthritis, Kawasaki disease). It can be difficult to assess the severity of illness in younger children, so adopt the NICE suggested 'traffic light' system as summarized in Fig. 15.10 (https://www.nice.org.uk), updated 2017.

| | Green – low risk | Amber – intermediate risk | Red – high risk |
|---|---|---|---|
| Colour (of skin, lips, or tongue) | • Normal colour | • Pallor reported by parent/carer | • Pale/mottled/ashen/blue |
| Activity | • Responds normally to social cues<br>• Content/smiles<br>• Stays awake or awakens quickly<br>• Strong normal cry/not crying | • Not responding normally to social cues<br>• No smile<br>• Wakes only with prolonged stimulation<br>• Decreased activity | • No response to social cues<br>• Appears ill to a health care professional<br>• Does not wake or if roused does not stay awake<br>• Weak, high-pitched, or continuous cry |
| Respiratory | | • Nasal flaring<br>• Tachypnoea:<br>– RR >50 breaths/minute, age 6–12 months<br>– RR >40 breaths/minute, age >12 months<br>• Oxygen saturation ≤95% in air<br>• Crackles in the chest | • Grunting<br>• Tachypnoea: RR >60 breaths/minute<br>• Moderate or severe chest indrawing |
| Circulation and hydration | • Normal skin and eyes<br>• Moist mucous membranes | • Tachycardia:<br>– >160 beats/minute, age <12 months<br>– >150 beats/minute, age12–24 months<br>– >140 beats/minute, age 2–5<br>• CRT ≥3<br>• Dry mucous membranes<br>• Poor feeding in infants<br>• Reduced urine output | • Reduced skin turgor |
| Other | • None of the amber or red symptoms or signs | • Age 3–6 months, temperature ≥ 39°C<br>• Fever for ≥5 days<br>• Rigors<br>• Swelling of a limb or joint<br>• Non-weight-bearing limb/not using an extremity | • Age <3 months, temperature ≥38°C*<br>• Non-blanching rash<br>• Bulging fontanelle<br>• Neck stiffness<br>• Status epilepticus<br>• Focal neurological signs<br>• Focal seizures |

CRT, capillary refill time; RR, respiratory rate
* Some vaccinations have been found to induce fever in children aged under 3 months
**This traffic light table should be used in conjunction with the recommendations in the NICE guideline on fever in under 5s.**

**Fig. 15.10** Traffic light system to assess children with fever. © NICE (2019) NG143 Fever in under 5s: assessment and initial management. Available from <www.nice.org.uk/guidance/ng143>. All rights reserved. Subject to Notice of rights, <https://www.nice.org.uk/terms-and-conditions#notice-of-rights>. NICE guidance is prepared for the National Health Service in England. All NICE guidance is subject to regular review and may be updated or withdrawn. NICE accepts no responsibility for the use of its content in this product/publication.

## Management based upon level of risk

If a diagnosis is reached, treat accordingly. If the cause of fever is unclear (no diagnosis is reached), the traffic light system may help to guide:

- If the child has any 'red' feature, refer for admission.
- If 'amber' features are present, consider discharge with a safety net plan (eg verbal/written advice on what to watch out for and what to do; arrangements for follow-up or admission directly if concerns).
- If there are only 'green' features, aim to discharge, with advice on what to do in the event of deterioration.

Adopt a low threshold for admitting febrile infants aged <3 months, as they are difficult to assess. For other children, a period of observation can help to reassure parents and staff that there is no obvious serious underlying cause. Similarly, when deciding about discharge, take into account family and social factors, as well as parental wishes/concerns. If a child presents with a fever and a non-specific/unusual rash, one option is to admit for observation for a few hours.

# The sick febrile child

If there is evidence that a child with fever is seriously ill or has severe sepsis, act quickly and decisively.

### Approach

- Assess the Airway, Breathing, and Circulation of the child to identify and treat life-threatening problems as they are found, in order to maintain vital functions before disease-specific therapies are started (see Primary assessment and resuscitation, pp. 650–1).
- Involve senior ED staff, PICU, and senior paediatric staff as soon as a child is suspected of being critically unwell.
- Measure and record T°—use an electronic thermometer in the axilla if <4 weeks old; if older than 4 weeks, use an electronic or chemical dot axillary thermometer or infra-red tympanic thermometer.
- Specifically search for an impaired conscious level and lack of recognition of parents/carers. Check BMG in all sick children.
- Early recognition and treatment of respiratory failure and shock are essential to avoid deterioration and subsequent cardiorespiratory arrest.
- Administer $O_2$ to maintain $SpO_2$ >94%.
- Give a bolus of 0.9% saline 20mL/kg IV/IO if there are any signs of shock (tachycardia, CRT >2s, mottled skin, purpuric rash, ↓ conscious level).
- If there is any suspicion or sign of meningococcal disease, administer parenteral benzylpenicillin or cefotaxime as soon as possible.
- If the child is <3 months old, check FBC, blood cultures, CRP, and urine; if unwell or WCC <5 or >15 × $10^9$/L, or <1 month old, perform LP and give parenteral antibiotics. If <3 months old, add ampicillin to cover *Listeria*.
- If the child is >3 months old, check FBC, blood cultures, CRP, and urine; get CXR if T° >39°C or WCC >20 × $10^9$/L or clinically unwell.
- Check U&E, ABG, and lactate if clinically unwell or drowsy.
- Consider LP if clinically unwell and febrile at any age, especially <1y.
- If the child is drowsy or has a ↓ conscious level (especially in the presence of focal neurological signs or focal seizures), consider adding IV aciclovir to cover the possibility of herpes simplex encephalitis.
- Resuscitate aggressively with repeated IV fluid boluses, inotropes, and early ventilation for children with ↓ conscious level.
- In older children, do not forget rarer causes of fever and impaired conscious level, including illicit drugs such as MDMA ('Ecstasy') or other amphetamines or ketamine.
- Try to obtain a detailed history of the illness from parents and carers at the earliest opportunity. Remember to include the vaccination history and any recent travel, or recent illness in the child's family or school.

# Purpuric rashes

The development of a purpuric rash is greeted with understandable parental alarm, due to the well-publicized association with meningococcal disease. History, examination, and FBC help to identify the cause.

## Causes of purpuric lesions

- Meningococcal disease (see ➲ Meningococcal disease, pp. 682–3).
- Henoch–Schönlein purpura.
- Thrombocytopenia.
- Immune thrombocytopenia.
- Leukaemia.
- Septic shock.
- Aplastic anaemia.
- Some viral illnesses.
- Trauma.
- Forceful coughing or vomiting may cause petechiae of the face.

## Meningococcal disease

(See ➲ Meningococcal disease, pp. 682–3.)

Presume that an ill child (particularly an infant) who develops a purpuric rash has meningococcal meningitis/septicaemia, and treat urgently for this.

## Henoch–Schönlein purpura

This vasculitic process affects small arteries in the kidneys, skin, and GI tract. It is relatively common in 4–11y olds and appears to follow a viral or bacterial infection. Erythematous macules develop into palpable purpuric lesions, which are characteristically concentrated over the buttocks and extensor surfaces of the lower limbs, although the distribution can be atypical in younger children. Associated symptoms include abdominal pain, testicular pain, and joint pains (arthritis in the ankles and knees). Nephritis may occur, producing micro- or macroscopic haematuria and proteinuria. Very occasionally, this progresses to renal failure.

*Check* BP, urinalysis, urine microscopy, FBC (platelets are normal), U&E.

*Refer* To the paediatrician.

## Immune thrombocytopenia

Probably results from an autoimmune reaction to preceding viral infection. Presents with a purpuric rash, mucous membrane bleeding, conjunctival haemorrhage, and occasionally GI bleeding. Check FBC (platelets are $<30 \times 10^9$/L). Refer for investigation and follow-up. In the presence of lymphadenopathy or splenomegaly, consider alternative diagnoses.

*Treatment* Is usually expectant, since the natural course is for most cases to resolve spontaneously over 3 months. Occasionally, life-threatening haemorrhage occurs—obtain expert help; resuscitate with $O_2$ and IV fluids, and give platelets.

## Acute leukaemia

This may present acutely to the ED with purpura associated with thrombocytopenia. Look for hepatomegaly, splenomegaly, and lymphadenopathy. FBC/blood film reveals anaemia with blast cells, ↓ platelets, and ↑ WBC.

*Refer* For admission.

# Meningococcal disease

(See Fig. 15.11; see also https://www.meningitis.org and https://www.nice.org.uk)

Meningococcal disease is unpredictable. Most children present acutely febrile and may not have a rash in the early stages.

## Septicaemia

Children presenting with septicaemia may have:

- A history of fever/rigors but be afebrile at the time of presentation.
- Isolated severe limb pain in the absence of any other physical signs.
- Abdominal pain, diarrhoea, and vomiting are common in septicaemia.
- Alertness until late in the illness.

Septic shock without meningitis at presentation has the worst prognosis.

## Meningitis

Young children may present with fever, vomiting, irritability, and confusion. Those aged <2y are less likely to have neck stiffness or photophobia. Take parental concerns about a child's responsiveness and alertness seriously. Older children typically present more classically with fever, vomiting, headache, stiff neck, and photophobia. However, teenagers may present atypically with a change of behaviour (eg confusion, aggression), which may be falsely attributed to alcohol or drugs.

## Rash

Underlying meningococcal disease may be very advanced by the time a rash appears. This may initially be blanching, macular, or maculopapular. Children without a rash or with a blanching rash can still have meningococcal disease. The classic rapidly evolving petechial or purpuric rash may be a very late sign and can carry a poor prognosis.

Urgent treatment and experienced help are essential. Perform CT scanning of the brain if there is impaired conscious level or focal neurological signs or signs of ↑ ICP. CT scans must not delay treatment. LP has no immediate role in ED care of the critically ill child and can be fatal. LP is contraindicated if there is extensive purpura, shock, impaired consciousness, coagulopathy, local infection, or ↑ ICP on CT or clinically.

Give antibiotics (IV ceftriaxone in children aged >3 months; IV cefotaxime + amoxicillin or ampicillin if aged <3 months) immediately to:

- All children with fever and petechial/purpuric rash.
- Children in shock with or without a rash.
- Children with clinical meningitis, but LP contraindicated.

Add vancomycin if the child has travelled outside the UK recently or has had prolonged or multiple exposure to antibiotics (in the past 3 months). If meningoencephalitis is suspected, give aciclovir.

Take any haemorrhagic rash in a febrile child very seriously. Although many children with fever and petechiae have viral illnesses, there is no room for complacency. Ensure that all have their vital signs measured and are carefully checked for signs of meningitis or septicaemia.

### Airway and ventilation

Intubate and ventilate:

- If impaired conscious level or ↑ ICP clinically.
- Prior to CT scanning if critically ill.
- If fluid resuscitation requirement is >40mL/kg.

Seek expert help for rapid sequence induction/intubation (RSI)—haemodynamic collapse is common. Consider using IV ketamine for induction if experienced in its use.

### Fluid resuscitation

Vast quantities of IV fluids are required in meningococcal septicaemia—often up to 100mL/kg. Some UK authorities recommend 4.5% human albumin solution (HAS) for fluid resuscitation, but give crystalloid (0.9% saline) if HAS is not immediately available.

### Inotropes

- Dopamine or dobutamine at 10–20mcg/kg/min. Make up 3× weight (kg) mg in 50mL of 5% glucose and run at 10mL/hr (= 10mcg/kg/min).
- These dilute solutions can be used via a peripheral vein.
- Start adrenaline via a central line only (seek expert help) at 0.1mcg/kg/min. Make up 300mcg/kg in 50mL of saline at 1mL/hr (= 0.1mcg/kg/min).

*Hypoglycaemia (glucose <3mmol/L)*

A 10% glucose bolus 2mL/kg IV and then a glucose infusion at 80% of maintenance requirements over 24hr.

*Correction of metabolic acidosis (pH <7.2)*

Sodium bicarbonate ($NaHCO_3$) 1mmol/kg IV = 8.4% $NaHCO_3$ 1mL/kg over 20min or 4.2% $NaHCO_3$ 2mL/kg in neonates.

*If $K^+$ <3.5mmol/L*

Give potassium chloride 0.25mmol/kg IV diluted in saline or glucose over 30min, with ECG monitoring. Caution if anuric.

*If total $Ca^{2+}$ <2mmol/L or ionized $Ca^{2+}$ <1.0mmol/L*

Give 10% calcium chloride (0.7mmol/mL) 0.1mL/kg over 30min IV (max 10mL) or 10% calcium gluconate (0.22mmol/mL) 0.3mL/kg over 30min (max 20mL).

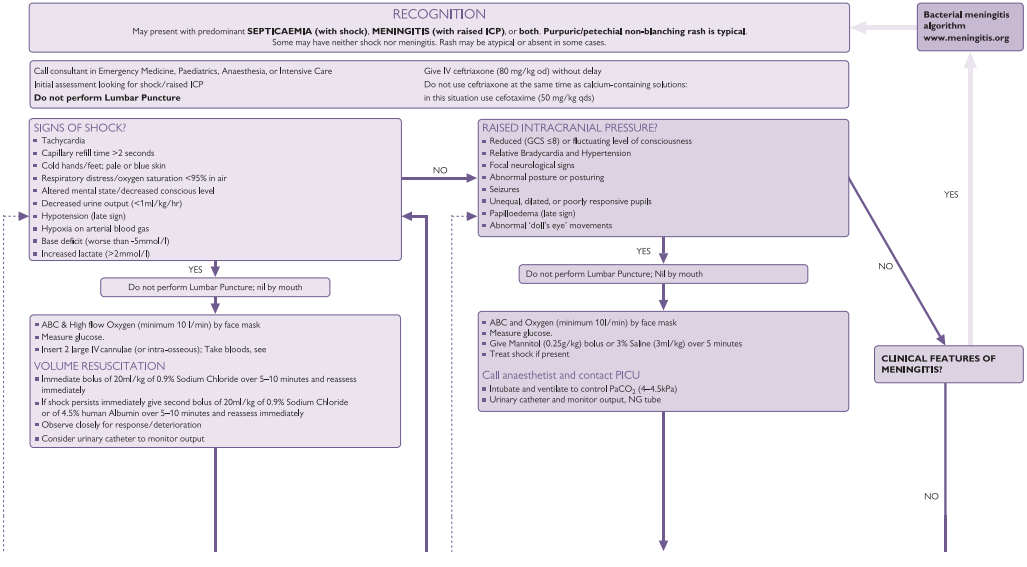
*If $Mg^{2+}$ <0.75mmol/L*

Give 50% magnesium sulfate 0.2mL/kg IV over 30min (max 10mL).

### Steroids in bacterial meningitis

NICE advises giving dexamethasone (0.15mg/kg to max of 10mg, qds for 4 days) for children aged >3 months with suspected or confirmed bacterial meningitis if LP reveals any of the following: frankly purulent CSF, CSF WCC >1000/microlitre, ↑ CSF WCC with protein concentration >1g/L, and bacteria on Gram stain.

Do not give steroids if TB meningitis is suspected.


RECOGNITION
May present with predominant SEPTICAEMIA (with shock), MENINGITIS (with raised ICP), or both. Purpuric/petechial non-blanching rash is typical.
Some may have neither shock nor meningitis. Rash may be atypical or absent in some cases.
Bacterial meningitis algorithm www.meningitis.org
Call consultant in Emergency Medicine, Paediatrics, Anaesthesia, or Intensive Care
Initial assessment looking for shock/raised ICP
Do not perform Lumbar Puncture
Give IV ceftriaxone (80 mg/kg od) without delay
Do not use ceftriaxone at the same time as calcium-containing solutions:
in this situation use cefotaxime (50 mg/kg qds)
SIGNS OF SHOCK?
Tachycardia
Capillary refill time >2 seconds
Cold hands/feet; pale or blue skin
Respiratory distress/oxygen saturation <95% in air
Altered mental state/decreased conscious level
Decreased urine output (<1ml/kg/hr)
Hypotension (late sign)
Hypoxia on arterial blood gas
Base deficit (worse than -5mmol/l)
Increased lactate (>2mmol/l)
NO
YES
Do not perform Lumbar Puncture; nil by mouth
ABC & High flow Oxygen (minimum 10 l/min) by face mask
Measure glucose.
Insert 2 large IV cannulae (or intra-osseous); Take bloods, see
VOLUME RESUSCITATION
Immediate bolus of 20ml/kg of 0.9% Sodium Chloride over 5–10 minutes and reassess immediately
If shock persists immediately give second bolus of 20ml/kg of 0.9% Sodium Chloride or of 4.5% human Albumin over 5–10 minutes and reassess immediately
Observe closely for response/deterioration
Consider urinary catheter to monitor output
RAISED INTRACRANIAL PRESSURE?
Reduced (GCS ≤8) or fluctuating level of consciousness
Relative Bradycardia and Hypertension
Focal neurological signs
Abnormal posture or posturing
Seizures
Unequal, dilated, or poorly responsive pupils
Papilloedema (late sign)
Abnormal 'doll's eye' movements
YES
NO
Do not perform Lumbar Puncture; Nil by mouth
ABC and Oxygen (minimum 10l/min) by face mask
Measure glucose.
Give Mannitol (0.25g/kg) bolus or 3% Saline (3ml/kg) over 5 minutes
Treat shock if present
Call anaesthetist and contact PICU
Intubate and ventilate to control $PaCO_2$ (4–4.5kPa)
Urinary catheter and monitor output, NG tube
CLINICAL FEATURES OF MENINGITIS?
YES
NO

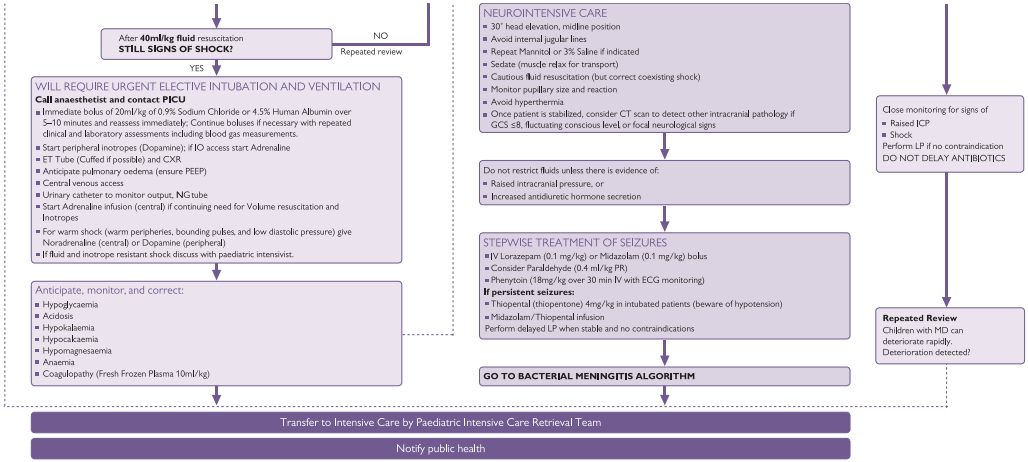



**Fig. 15.11** Management of meningococcal disease in children and young people. Take blood for glucose, FBC, coagulation screen, U&E, $Ca^{2+}$, $Mg^{2+}$, $PO_4^{3-}$, blood cultures, ABG, lactate, cross-match, and PCR for *Neisseria meningitidis*.

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# Lumbar puncture

In the context of infectious disease, an LP can help to confirm a diagnosis of meningitis and to identify the organism responsible and its antibiotic sensitivities.

## Contraindications to LP

If LP is performed in the presence of significantly ↑ ICP, there is a risk of 'coning' occurring. Take senior advice before performing an LP. The following are contraindications to performing an LP:

- Prolonged or focal seizure.
- Focal neurological signs.
- Purpuric rash.
- GCS <13/15.
- Pupillary dilatation.
- Impaired oculocephalic reflexes.
- Bradycardia.
- Coagulopathy and/or low platelets.
- Papilloedema.

## Performing an LP

- Confirm that there is no contraindication.
- Prepare the parents, set up the equipment, and enlist help from an experienced nurse.
- Position the child to be lying curled up into a ball, lying on the side (see Fig. 15.12).
- Mark the skin with a pen in the midline at the level of the iliac crests.
- Scrub and don a sterile gown and gloves.
- Clean the skin with antiseptic solution, and cover with sterile drapes.
- Consider LA for the skin using 1% lidocaine solution.
- Slowly insert the 21G LP needle, aiming towards the umbilicus.
- If this causes much pain, withdraw the needle and use more lidocaine LA (but <3mg/kg—see ➲ Analgesia in specific situations, pp. 290–1.)
- If no CSF is obtained, withdraw the needle and reassess its direction, then try again.
- Collect four drops of CSF in each of three bottles and send for: microscopy and Gram staining, culture, and sensitivity; cell counts, glucose, and protein; and PCR.
- If a bloody tap is obtained, send the clearest sample for cell count analysis.

**Fig. 15.12** Positioning for a lumbar puncture.

# Skin lesions in multisystem disease

The appearance of the skin may provide a valuable clue to an underlying disease process. If suspected, refer all of the following diseases to a paediatrician.

## Kawasaki disease (mucocutaneous lymph node syndrome)

This disease, believed to be related to a viral infection, was first reported in Japan in 1967 and has now spread worldwide. It is not contagious.

Most cases affect children aged <5y. Fever is often the first symptom and this usually lasts ≥5 days. Extensive skin and mucosal changes occur, including an erythematous rash, which may affect the palms and soles and desquamate. Conjunctivitis, uveitis, fissured lips, and a strawberry tongue may be seen. Other features include acute cervical lymphadenopathy, arthritis, and diarrhoea.

Coronary artery aneurysm (and subsequent thrombosis) is a significant complication, but the risk of this developing is ↓ for children who receive treatment (IV immunoglobulin), underlining the importance of making the diagnosis.

If Kawasaki disease is suspected, check FBC, ESR, and viral titres, and refer to a paediatrician.

## Dermatitis herpetiformis

This is the skin manifestation of coeliac disease. Vesicles and papules occur over the knees, elbows, and buttocks. The lesions are very itchy and produce much scratching. Dapsone is effective treatment—refer to a paediatrician.

## Erythema multiforme

Target lesions, often with pale, blistered centres, are symmetrically distributed, particularly over the extensor surfaces of the limbs, sometimes including the hands and feet. The skin lesions, combined with fever, systemic illness, and oral and genital ulceration, comprise the Stevens–Johnson syndrome.

*Causes* Include infection (herpes, *Mycoplasma*, TB) and drugs (sulfonamides, barbiturates).

## Erythema nodosum

Painful red skin nodules or plaques on the anterior surfaces of both shins may be associated with fever, lethargy, and arthralgia. Erythema nodosum may occur in children and adults at any age but is most common between the ages of 12 and 25y. It may be due to streptococcal infection, TB, sulfonamides, ulcerative colitis, or sarcoid. Sometimes, no cause is found. If suspected, refer to the paediatric team for investigation and follow-up.

## Erythema marginatum

A transient erythematous rash with raised edges occurs in 20% of cases of *rheumatic fever* (see ➲ Rheumatic fever, p. 513). Rheumatic fever is an autoimmune disease which follows infection with group A streptococci. Once common, it is now unusual in the UK.

Diagnose using the revised Duckett–Jones criteria (two or more major, or one major and two minor, plus evidence of preceding streptococcal infection, eg throat swab, ↑ anti-streptolysin O titre):

*Major criteria* Erythema marginatum, carditis, polyarthritis, Sydenham's chorea, subcutaneous nodules.

*Minor criteria* Fever, arthralgia, ↑ ESR, ↑ WCC, previous rheumatic fever, prolonged PR interval.

## Erythema (chronicum) migrans

(See ➲ Infestations, pp. 240–1.)

The characteristic skin rash of Lyme disease begins as a red papule, which spreads to produce erythematous lesions with pale centres and bright edges. Lyme disease is a multisystem disorder resulting from tick-borne infection. It initially manifests with one or more of a variety of symptoms, including fever, headache, malaise, arthralgia, and myalgia. The rash is present in most cases. The diagnosis can be elusive, but consider it if there has been any history of travel to an affected area.

## Identifying skin lesions

(See Table 15.4.)

*Description*

- Impalpable coloured lesion <1cm diameter = macule.
- Impalpable coloured lesion >1cm diameter = patch.
- Palpable lump <0.5cm diameter = papule.
- Palpable lump >0.5cm diameter = nodule.
- Palpable fluid-filled lesion <0.5cm diameter = vesicle.
- Palpable fluid-filled lesion >0.5cm diameter = bulla.

**Table 15.4** Skin lesions and possible causes

| Feature | Causes |
|---|---|
| Peeling skin | Toxic epidermal necrolysis ('scalded skin syndrome'), Kawasaki disease |
| | Streptococcal infection |
| Blistering lesions | *Staphylococcus* (impetigo and toxic epidermal necrolysis), scabies, chickenpox, herpes zoster, herpes simplex, Stevens–Johnson, pompholyx, Coxsackie A16 (hand, foot, and mouth disease), dermatitis herpetiformis, epidermolysis bullosa, drugs |
| Lesions on palms and soles | Coxsackie A16, Kawasaki disease, erythema multiforme, scables, pompholyx |
| Pruritus | Eczema, urticaria, psoriasis, chickenpox, scabies, lice, insect bites, dermatitis herpetiformis |

# Paediatric ENT problems

## Background

Due to frequent infections and large concentrations of active lymphoid tissue, certain ENT problems are very common in general and paediatric practice. For example, acute suppurative otitis media (see ➔ Earache, pp. 566–7) has an incidence of 20% amongst preschool children; secretory otitis media ('glue ear') has a prevalence of 5% amongst all children. Rhinorrhoea from coryza and rhinitis is even more common.

## Approach

Although many ENT diseases are usually considered as primary care problems, children often present to the ED suffering from them. It is obviously important to examine the ears and throat of any child presenting with a fever. Remember, however, that the ill, septic child with large red tonsils may also have a significant septic focus elsewhere (eg meningitis or pneumonia).

## Examination

Examination of the ears and throat is generally disliked by children and, as a result, can sometimes prove to be rather a struggle to undertake. It is therefore sensible to leave this part of the full examination of a child until last. Help from parents can be invaluable in enabling examination of the slightly unco-operative toddler or younger child. Sit the child on a parent's lap for examination of the ears and throat, as shown in Fig. 15.13.

*The difficult examination*

Despite attempting a variety of manoeuvres, it can be very difficult to adequately visualize the throat of a child who adamantly refuses to open their mouth. A useful trick is to draw the face of a 'Smiley Man' on the end of a wooden spatula. The child may then consent to the 'Smiley Man' having a look at their throat (preferably with the ink side up!).

## Presentation and treatment

The presentation, diagnosis, and treatment of specific ENT diseases in both children and adults are described in ➔ Chapter 12.

- See ➔ Ear, nose, and throat foreign bodies, pp. 562–3.
- See ➔ Earache, pp. 566–7.
- See ➔ Epistaxis, p. 568.
- See ➔ Nasal fracture, p. 569.
- See ➔ Sore throat, pp. 570–1.

*Examining a child's ear* In an infant, pull the pinna back and down (rather than up) for the best view.

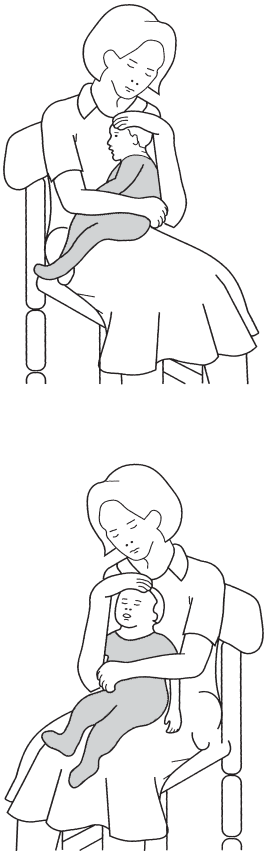

**Fig. 15.13** Examining a child's ear and throat.

# Stridor: upper respiratory infections

The upper airway may be blocked by: distortion (eg tongue falling back in coma), extrinsic compression (eg haematoma), swelling of its wall (eg burns, croup, epiglottitis, diphtheria), or FB within (see Table 15.5).

- *Signs of upper airway obstruction*: stridor, marked dyspnoea, drowsiness, subcostal/suprasternal recession, drooling of saliva, difficulty speaking, and cyanosis. Any of these warn of impending obstruction.
- *Stridor* is a high-pitched inspiratory noise. It occurs in croup, acute epiglottitis, inhaled FB, laryngeal trauma, laryngomalacia ('congenital laryngeal stridor'), and angioneurotic oedema.

## Acute croup (laryngotracheobronchitis)

Viral (para-influenza in >80%) and common between 6 months and 5y. Spring and autumn epidemics occur. Illness lasts ~3–5 days. Coryzal symptoms usually precede harsh stridor, a barking cough ('seal's bark'), with hoarseness ↑ over several days. T° is only mildly ↑. Leave the child in a comfortable position, preferably in the arms of a parent, who can hold an $O_2$ mask near the child. Look for signs of significant airway obstruction, but do not examine the pharynx as this may precipitate laryngospasm or obstruction. If any signs are present, or if $SpO_2$ is <92% on air, refer urgently—intubation may be required. Use the modified Westley croup score by adding individual values as follows:

- *Stridor*: none = 0, only when upset or agitated = 1, at rest = 2.
- *Retractions*: mild = 1, moderate = 2, severe = 3.
- *Air entry*: normal = 0, mild ↓ = 1, marked ↓ = 2.
- *$SpO_2$ <92% on air*: none = 0, with agitation = 4, at rest = 5.
- *Level of consciousness*: normal = 0, altered conscious level = 5.

Admit moderate (score 3–5) or severe (score 6–11) croup or impending respiratory failure (score >11).

Give dexamethasone 0.15mg/kg PO or, if vomiting or severe respiratory distress, nebulized budesonide (2mg in 5mL of 0.9% saline). If severe (score >5), give nebulized adrenaline driven by $O_2$ at 8L/min (0.4mL/kg of 1:1000, max 5mL; repeat as required). Refer severe cases to PICU (<1% of croup is severe).

Consider discharging mild croup (score 0–2) from the ED after a brief period of observation—let an experienced clinician decide. Discharge in the evening may be inadvisable, as croup can worsen overnight.

## Diphtheria

Although rare in the UK, the exotoxin of *Corynebacterium diphtheriae* may produce serious organ damage (especially myocarditis) and upper respiratory tract obstruction. The non-immunized child may present with pyrexia, sore throat, and dysphagia due to an adherent pharyngeal exudate. Cervical lymphadenopathy causes a 'bull neck' appearance. (Note that infectious mononucleosis may present similarly—see ➲ Infectious mononucleosis (glandular fever), p. 231.)

*Treat* With $O_2$, obtain ECG and venous access, send blood for FBC and blood culture, and obtain a throat swab. Refer for antitoxin (20,000U IM after a test dose) and IV antibiotics (eg erythromycin).

## Acute epiglottitis

Increasingly uncommon, due to widespread Hib vaccination. Rapidly progressive airway obstruction may result. Children aged 2–7y are most usually involved, although it can affect older children and adults. Unlike croup, stridor is usually soft and may even be absent. Onset is typically acute. The child is systemically unwell with pyrexia >38.5°C, but little or no cough. In severe cases, the child may be ominously quiet and unable to speak, sitting upright drooling saliva in a 'sniffing position'.

*Management* Do not try to visualize the throat as this may precipitate total airway obstruction. Let the child adopt the most comfortable position; give humidified $O_2$ and call urgently for anaesthetic and ENT help. Nebulized adrenaline (0.4mL/kg of 1:1000, max 5mL) may 'buy time'. Defer blood tests (FBC, blood cultures) and treatment with IV cefotaxime until an anaesthetist has assessed the child. Lateral neck X-rays are unnecessary and potentially hazardous. Intubation, if required, may be very difficult to perform. A safe approach is for an experienced anaesthetist to use a gaseous induction in the presence of a surgeon who is prepared for a surgical airway. Airway swelling may require a smaller than expected diameter (and thus uncut) ET tube. If visualization of the tracheal orifice is difficult at laryngoscopy due to oedema, ask an assistant to squeeze the chest and look for an air bubble emerging from the trachea.

*Loss of the airway* If this happens, summon help and attempt to ventilate with $O_2$ using a bag and mask. If ventilation proves impossible, obtain a surgical airway (needle cricothyroidotomy if <12y, surgical cricothyroidotomy if ≥12y—see ➲ Airway obstruction: surgical airway, p. 336).

## Bacterial tracheitis

May be due to *Staphylococcus aureus, Streptococcus*, or *Haemophilus influenzae*. The presentation of 'croup', plus moderate/severe pyrexia and production of copious secretions, suggests the diagnosis. If suspected, refer and treat as for acute epiglottitis (intubation is often required). Bacterial tracheitis can cause rapid onset of septic shock.

**Table 15.5** Clinical presentations of upper airway obstruction

| | Croup | Epiglottitis | Bacterial tracheitis | FB |
|---|---|---|---|---|
| Age | 1–2y | 2–6y | Throughout childhood | Throughout childhood |
| Onset | 1–2 days | <24hr | <24hr | <24hr |
| History | Coryza, barking cough | Sore throat, dysphagia | Rattling cough, sore throat | |
| Signs | T° <38.5°C, non-toxic, harsh stridor, hoarseness | T° >38.5°C, toxic, upright position | T° >38.5°C, toxic, mucopurulent secretions, soft/absent stridor | Afebrile, non-toxic |

# Severe acute asthma in children

## Assess

Conscious level, degree of breathlessness, degree of agitation, use of accessory muscles, amount of wheezing, pulse rate, and RR. Attempt to measure peak flow if age >5y (see Fig. 15.14 for normal peak flow).

Follow the 2019 BTS/SIGN guidelines based on age and severity (https://www.brit-thoracic.org.uk). Investigations, including blood gas estimations, rarely alter immediate management.

## Cautions

Children with severe asthma attacks may not appear distressed. Wheeze and RR correlate poorly with severity of airway obstruction. ↑ tachycardia denotes worsening asthma, and a fall in heart rate in life-threatening asthma is pre-terminal.

Assessment in the very young (<2y) may be difficult—get expert help.

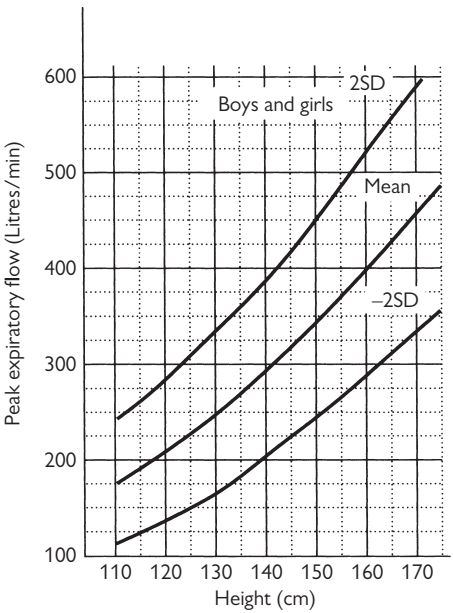


**Fig. 15.14** Normal peak expiratory flow in children aged 5–18y.

## Management of acute asthma in children aged >2y

(See Figs 15.15 and 15.16.)

- Summon senior ED/PICU/paediatric help if asthma is severe.
- Provide high-flow $O_2$ via a face mask (or nasal cannulae).
- Give an inhaled β-agonist. In mild or moderate asthma, use a metered-dose inhaler with a spacer, and 2–10 puffs of salbutamol.
- In severe or life-threatening asthma, use an $O_2$-powered nebulizer with salbutamol 2.5–5mg or terbutaline 5–10mg.
- Give oral prednisolone (20mg for children aged 2–5y; 30–40mg if aged >5y). If already taking maintenance steroids, give 2mg/kg (max 60mg). In children who vomit, give IV hydrocortisone 4mg/kg.
- Add ipratropium bromide 0.25mg if there is poor initial response to nebulized β-agonist.
- Repeat β-agonist and ipratropium every 20min up to 2hr as needed.
- Consider salbutamol (15mcg/kg) given IV over 10min in severe cases with a poor response to initial nebulized salbutamol and ipratropium bromide. Refer to PICU urgently and check $K^+$ levels.
- Consider an IVI of magnesium sulfate 40mg/kg over 20min.
- Aminophylline is not recommended in children with mild to moderate asthma. In severe or life-threatening asthma unresponsive to maximal doses of bronchodilators and systemic steroids, take specialist advice and consider IV aminophylline (5mg/kg over 20min; maintenance IVI at 1mg/kg/hr; omit loading dose if already receiving oral theophyllines).
- Do not give 'routine' antibiotics.

*Note*: if possible, repeat and record peak flow 15–30min after starting treatment. If the patient is not improving, give further nebulized β-agonist. Pulse oximetry is helpful in assessing response to treatment. An $SpO_2$ of ≤92% on air after initial bronchodilator therapy usually indicates the need for more intensive inpatient care usually in PICU. CXR is indicated for severe dyspnoea, focal chest signs, or signs of severe infection.

*Consider the need for anaesthesia/intubation/IPPV and PICU transfer*

- Deteriorating peak flow or worsening or persistent hypoxia or normal/↑ $pCO_2$ levels on ABG.
- Exhaustion, feeble respiratory effort, confusion, or drowsiness.
- Coma or respiratory arrest.

## Management of acute asthma in children aged <2y

Assessing acute asthma in early childhood is difficult—get specialist help (see https://www.sign.ac.uk). Intermittent wheezing attacks are usually due to viral infection. Differential diagnosis includes: aspiration and other pneumonias, bronchiolitis, tracheomalacia, and complications of underlying conditions (eg congenital abnormalities, cystic fibrosis). If there is no response to inhaled bronchodilators, review the diagnosis:

- Use a metered-dose inhaler with a spacer to give β-agonist therapy.
- Consider systemic steroids early in the management of moderate to severe asthma in infants (10mg of soluble prednisolone).
- Consider adding inhaled ipratropium bromide (0.25mg) to inhaled β-agonists for more severe symptoms.


**Fig. 15.15** Management of acute asthma in 2–5y olds (see: https://www.brit-thoracic.org.uk and https://www.sign.ac.uk).

This figure is reproduced from SIGN 158: *British guideline on the management of asthma*, by kind permission of the Scottish Intercollegiate Guidelines Network.


**Fig. 15.16** Management of acute asthma in children aged >5y (see: https://www.brit-thoracic.org.uk and https://www.sign.ac.uk).

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# Acute bronchiolitis

Viral infection of the small airways results in inflammation, oedema, and excessive secretions, presenting with signs of obstructive airways disease. Acute bronchiolitis is common, particularly in the winter months and predominantly involves infants (typically 3–6 months). Those at particular risk are the very young (aged <6 weeks), the premature (born <35 weeks), and those with chronic respiratory conditions, congenital heart disease, immunodeficiency, or neurological problems. Parental smoking ↑ the risk of bronchiolitis. Breastfeeding for >2 months appears to have a protective effect. Most infants recover completely within 2 weeks.

## Agents responsible

75% are caused by respiratory syncytial virus (RSV). Other causes include influenza, para-influenza, and adeno- and enteroviruses.

## Presentation

Coryza, rhinorrhoea, and mild fever progress to respiratory distress with dyspnoea, dry cough, feeding difficulties, and wheeze (variable). Some children may present with apnoea. Inspection may reveal cyanosis, dehydration, tachypnoea (>50/min), nasal flaring, grunting, and subcostal and intercostal recession. The chest is usually visibly hyperinflated in bronchiolitis. There may be tachycardia and prolonged expiration (± wheeze), with fine end-inspiratory crepitations.

## Complications

These include feeding difficulties, apnoeic spells, and respiratory failure (hence, adopt a low threshold for admission). Secondary bacterial infection can occur but is uncommon. Long-term airway damage may occasionally occur (obliterative bronchiolitis).

## Investigations

- Apply a pulse oximeter, and check the pulse and CRT.
- Do not do routine blood tests unless the infant is febrile or an alternative diagnosis, such as pneumonia or sepsis, is more likely.
- Consider CXR and ABG/capillary gas only for those with progressive, atypical, or severe illness. Do not obtain a CXR routinely.
- Fluorescent antibody tests on nasopharyngeal aspirate to demonstrate the presence of RSV are recommended; these help with cohorting and isolation arrangements on the wards (see ➲ Avoiding cross-infection, p. 699), particularly during the annual epidemic season in winter.
- Assess feeding difficulties by offering a bottle feed.

*CXR* Shows hyperinflation, with downward displacement of the diaphragm due to small airway obstruction and gas trapping. There may also be collapse or consolidation (usually upper lobe) or perihilar infiltrates hard to distinguish from pneumonia.

*Differential diagnoses for bronchiolitis* Include congenital heart disease, asthma, pneumonia, cystic fibrosis, inhaled FB, and septicaemia.

## Treatment

(See NICE guideline, published in 2015, available at: https://www.nice.org.uk)

Emergency treatment is largely supportive, comprising one or more of:

- Providing humidified $O_2$ if $SpO_2$ is <92%.
- Performing nasal suctioning if the presentation is with apnoea.
- Ensuring adequate hydration—give fluid by NG or orogastric tube if unable to take enough PO; give IV fluid if unable to tolerate NG or orogastric fluids or there is impending respiratory failure.
- Calling for expert help and considering CPAP for impending respiratory failure.

Do not give antibiotics for bronchiolitis, but consider for severe illness suggestive of coexisting pneumonia or septicaemia. There is no benefit from using ipratropium, salbutamol, montelukast, PO or inhaled steroids, or nebulized adrenaline—do not use these therapies in acute bronchiolitis.

### *Hospital admission/discharge*

Refer for admission all infants with respiratory distress, feeding difficulties (50–75% of usual fluid intake in previous 24hr), $SpO_2$ <94% on air, apnoeic episodes, or dehydration. When considering discharge, consider the family and social situation and the ability of parents to be able to identify and respond to deterioration. Provide advice to parents of children being discharged on how to recognize deterioration (eg apnoea, cyanosis, ↑ work of breathing/exhaustion, fluid intake ↓ to 50–75% of usual, or no wet nappy for 12hr) and how to seek help if needed.

### *PICU referral and ventilatory support*

This is indicated for those with recurrent apnoea, persistent acidosis with pH <7.25, infants with ↓ conscious level, poor chest wall movement, and low $SpO_2$ (<92%) despite $FiO_2$ >60%, and those with hypercapnia.

### *Avoiding cross-infection*

This is important during epidemics. Ensure all persons entering a cubicle containing a child with bronchiolitis clean their hands before and after seeing the patient, and use gloves and plastic aprons.

## Prevention

Palivizumab is a humanized monoclonal RSV antibody, which is used as a prophylactic agent to reduce the severity of RSV disease in at-risk infants. It can be considered for use on a case-by-case basis in infants who:

- Were born prematurely (<35 weeks' gestation).
- Have acyanotic congenital heart disease.
- Have chronic lung disease.
- Have severe congenital immunodeficiency.

Infants are selected for this treatment by a local lead paediatric specialist.

# Whooping cough[ND]

Caused by *Bordetella pertussis*, whooping cough is a notifiable disease, with an incubation period of 5–14 days (see ➲ Incubation periods, pp. 228–9). It is common (particularly in autumn) in children not immunized against it. A similar disease may also occur with other viral infections (*Bordetella parapertussis* and adenoviruses).

## Presentation

Coryza is followed by ↑ cough (typically worse at night and tending to occur in bouts, often culminating in vomiting). Severe coughing bouts may result in conjunctival haemorrhages. The characteristic 'whoop' is an inspiratory noise produced after a coughing bout. It is not present in all infants with whooping cough. The cough may persist for several weeks.

## Complications

Illness is often prolonged. There is a risk of neurological damage and bronchiectasis. Infants are at particular risk of death from apnoeic episodes.

## Investigation

Take cultures by nasopharyngeal/per nasal swabs. Send blood for viral titres, *Mycoplasma* antibodies, and FBC (usually reveals markedly ↑ lymphocytes). CXR may be normal or show a 'shaggy' right heart border.

## Treatment

(See NICE CKS, available at: https://cks.nice.org.uk)

*Criteria for admission*

- Infants aged <6 months (due to risk of apnoea).
- Significant breathing problems (apnoeic episodes, cyanosis, or severe paroxysms).
- Other complications (eg seizures, pneumonia).

*Management of those discharged*

If the child is fit for discharge, inform the infectious diseases consultant and prescribe a 7-day course of PO clarithromycin, provided the onset of the cough was within the past 21 days. Suggest simple analgesics (paracetamol or ibuprofen). Advise that children should be kept off school or nursery until 48hr of antibiotics have been taken (or until 21 days after onset of symptoms if not treated). Explain that even with treatment, whooping cough is likely to result in a prolonged (non-infectious) cough, lasting for several weeks. Arrange GP follow-up, and give PO clarithromycin as prophylaxis to unimmunized infant siblings.

## Prevention

Encourage immunization.

## Pulmonary tuberculosis[ND]

TB is being seen increasingly frequently again (see ➲ Tuberculosis, p. 242). It is more common in visitors from overseas or in HIV +ve children. TB may present in a variety of ways in children: persistent cough and fever, growth retardation, meningitis, pleural effusion, monoarticular arthritis, lymphadenopathy, back pain, and hepatosplenomegaly.

*Investigations* CXR.

*Treatment* Refer suspected cases for specialist evaluation, including Mantoux (0.1mL of intradermal tuberculin), and treatment.

## Cystic fibrosis

Recurrent respiratory infections in neonates and infants raise the possibility of cystic fibrosis, tracheo-oesophageal fistula, cleft palate, or a defect in immunity. Cystic fibrosis is an autosomal recessive disorder, affecting 1 in 2000 children. It may present neonatally with meconium ileus or later with respiratory infections (± finger clubbing), failure to thrive, rectal prolapse, and steatorrhoea. Once diagnosed, a child will remain closely monitored and treated by both the GP and a specialist cystic fibrosis respiratory team. Involve this team at an early stage if a child with cystic fibrosis presents with respiratory infection.

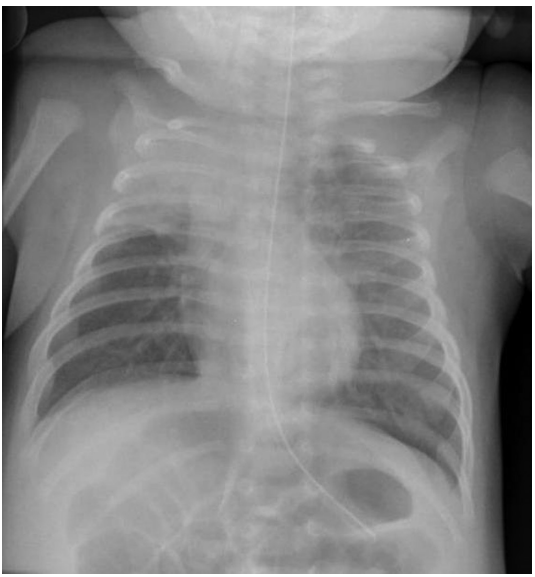


**Fig. 15.17** CXR of an infant with right upper lobe consolidation.

# Pneumonia

Pneumonia is relatively common at all ages throughout childhood, but the infective agents likely to be responsible vary considerably (see Table 15.6). Viruses are most commonly found as a cause in younger children. In older children, when a bacterial cause is found, it is most commonly *Streptococcus pneumoniae*.

**Table 15.6** Infective agents responsible for pneumonia

| Age | Common causes |
|---|---|
| Neonates | *Escherichia coli*, β-haemolytic *Streptococcus, Chlamydia trachomatis, Listeria monocytogenes*, CMV |
| Infants and toddlers | RSV, para-influenza viruses, *S. pneumoniae, Haemophilus influenzae, Mycoplasma* |
| Older children | S. *pneumoniae, H. influenzae, Mycoplasma* |

## Symptoms

Often an URTI is followed by ↑ fever, cough, dyspnoea, lethargy, feeding difficulties, and dehydration. Pleuritic chest pain, abdominal pain, and neck stiffness may occur.

The combination of headache, abdominal pain, maculopapular rash, and joint pains suggests *Mycoplasma* infection.

## Signs

- The child is usually dyspnoeic, pyrexial, and unwell.
- Classic signs of consolidation (see ➲ Pneumonia, pp. 114–15) are often absent, especially in infants and younger children (so if suspected, adopt a lower threshold for obtaining a CXR).
- Look for evidence of dehydration and of infection elsewhere (including the ears and throat).
- If wheeze is present in a preschool child, bacterial pneumonia is unlikely, although it does occur occasionally with mycobacteria in older children.

## Investigations

- Check $SpO_2$.
- Take throat swabs.
- Obtain blood samples for FBC, cultures, viral titres, and *Mycoplasma* antibodies.
- CXR may demonstrate widespread bronchopneumonia or lobar consolidation (see Fig. 15.17)—there may be an accompanying pleural effusion. The presence of cavitation suggests staphyloccocal pneumonia or TB.

## Treatment

- If $SpO_2$ is <93%, give $O_2$.
- Treat dehydration with IV fluids.
- Refer for admission and antibiotics—PO is often sufficient.
- IPPV is rarely required.

*ICU treatment*

Refer to ICU those children who have one or more of:

- Inability to maintain $SpO_2$ >93% with 60% $O_2$.
- Signs of shock.
- ↑ RR/pulse rate with respiratory distress and exhaustion.
- Slow, irregular breathing or recurrent apnoea.

*Choice of antibiotic*

This depends upon the likely infective agent and local/national protocols (see BTS guidelines, available at https://www.brit-thoracic.org.uk, see also Box 15.2).

### Box 15.2 Antibiotic treatment for suspected bacterial pneumonia

#### Uncomplicated community-acquired pneumonia

- *Neonate*: benzylpenicillin and gentamicin.
- *Neonate and child under 6 months*: cefuroxime or co-amoxiclav (or benzylpenicillin if lobar pneumonia or *S. pneumoniae* suspected).
- *Child 6 months to 5y*: PO amoxicillin or PO clarithromycin.
- *Child 5–18y*: PO clarithromycin (or PO amoxicillin if *S. pneumoniae* suspected).

Add flucloxacillin if *Staphylococcus* is suspected, eg in influenza or measles. Use clarithromycin if atypical pathogens are suspected or penicillin allergy.

#### Severe community-acquired pneumonia of unknown aetiology

- *Neonate*: benzylpenicillin and gentamicin.
- *1 month to 18y*: cefuroxime or co-amoxiclav (or benzylpenicillin if lobar pneumonia or *S. pneumoniae* suspected).

Use clarithromycin if atypical pathogens, such as *Mycoplasma* (more common in children over 5y) or *Chlamydia*, are suspected or penicillin allergy. Add flucloxacillin if *Staphylococcus* is suspected.

# Fits in children

A careful history is crucial and may take some time to piece together. Epileptic fits may take many forms:

**Grand mal (tonic/clonic)** Loss of consciousness and shaking of all limbs.

**Petit mal ('absences')** Child pauses in speech or other activity and is unaware of episode.

**Focal fit** Involves one part of body (progression to grand mal = Jacksonian march).

**Myoclonic fit** May be violent and includes drop attacks.

**Infantile spasm (Salaam attack)** May involve truncal flexion and cause a fall.

**Temporal lobe epilepsy** Numerous bizarre presentations.

## The fitting patient

(See ➲ Status epilepticus, p. 705.)

The child who is still fitting on arrival to hospital is likely to have had a prolonged fit, so provide immediate attention:

- Give $O_2$.
- Secure the airway. If teeth are clenched, do not try to prise them open to insert an airway. Instead, if the airway is obstructed, try a nasopharyngeal airway (see ➲ Insertion of nasopharyngeal airway, p. 335).
- Give IV lorazepam (0.1mg/kg) or if venous access is unsuccessful, buccal midazolam (0.5mg/kg, max 10mg) or PR diazepam (0.5mg/kg).
- Check bedside strip measurement of venous/capillary BMG, and treat hypoglycaemia with glucose IV 0.2g/kg (2mL/kg of 10%).
- Treat fever >38°C with PR paracetamol.
- If fits continue, follow the algorithm for status epilepticus (see ➲ Status epilepticus, p. 705).

## After the fit has finished

Reassess Airway, Breathing, and Circulation. Continue $O_2$ and place in the recovery position until consciousness is regained. Check for any injuries sustained as a result of the fit, and perform regular observations.

*First fit*

Refer for investigation of possible causes. U&E, blood glucose, $Ca^{2+}$, $Mg^{2+}$, FBC, and urinalysis will be required.

*Subsequent fit*

If appropriate, check serum anticonvulsant level and arrange for follow-up at the GP/outpatient clinic to receive the results and adjust the dose appropriately. Allow home those patients with known epilepsy who have fully recovered and have no obvious underlying medical cause for the fit needing treatment (eg meningitis, hypoglycaemia).

# Status epilepticus

**Definition** A fit (or consecutive fits without complete recovery in between) lasting >30min. The duration of the seizures is often underestimated because the intensity of the jerking diminishes with time and small-amplitude twitching may be easily missed.

Status epilepticus usually involves tonic–clonic fits and, as in adults, is associated with significant mortality (~4%) and morbidity (up to 30% have long-term neurological damage). Prompt treatment with termination of the fit is crucial to ↓ these risks.

*Causes* Meningitis, head injury, altered drug therapy or non-compliance in known epileptic child, metabolic disturbances, encephalopathy (including Reye's syndrome), 'febrile status', poisoning.

**Managing the fitting child** (See *APLS Manual*, sixth edition, 2016.)

- Open and maintain airway, and give $O_2$.
- Do not prise open clenched teeth—consider a nasopharyngeal airway.
- Rapidly obtain venous access, and check BMG.

*If convulsion continuing at 5min*

- If the fit has lasted for 5min, give lorazepam 0.1mg/kg IV/IO over 30–60s, or if venous access is unsuccessful, give buccal midazolam (0.5mg/kg, max 10mg) or PR diazepam (0.5mg/kg).
- Treat hypoglycaemia with glucose 2mL/kg IV of 10%.
- Apply pulse oximeter and send blood for investigations (see ➲ Investigations, p. 705).
- Check T°—if >38°C, give paracetamol 15mg/kg PR.

*If convulsion continuing after a further 10min*

- Repeat lorazepam 0.1mg/kg IV/IO over 30–60s. Do not give >2 doses of benzodiazepines, including prehospital treatment.
- Get senior help and call for senior ED/anaesthetic/PICU help.

*If convulsion continuing after a further 10min*

- Start phenytoin 20mg/kg IVI over 20min (monitor BP and ECG), or if already on phenytoin, consider instead phenobarbital (20mg/kg IV over 20min) or levetiracetam or sodium valproate.
- Whilst preparing to give phenytoin IVI, consider giving a dose of PR paraldehyde (0.4mL/kg) mixed with an equal volume of olive oil (thus making a total volume of 0.8mL/kg of the paraldehyde + oil mixture).

*If convulsion continuing after a further 20min*

- Paralyse, intubate, and ventilate using IV thiopental (induction dose 4mg/kg), and consider a thiopental infusion. Alternatively, consider midazolam IVI (0.1–1mg/kg/hr)—if this fails to control the fit, use thiopental.
- Transfer to ICU/PICU.

*Investigations* BMG and blood glucose, U&E, $Ca^{2+}$, $Mg^{2+}$, $PO_4^{3-}$, LFTs, FBC, ABG/capillary gas, blood cultures, coagulation screen, CXR. If taking anticonvulsant(s)—check serum level(s). Obtain brain CT scan if intracranial disease is suspected (unless clinically meningitis, in which case treat immediately—see ➲ Meningococcal disease, pp. 682–3).

# Febrile convulsions

## Definition

Grand mal seizures lasting <5min and secondary to pyrexia of febrile illness. By definition, children already diagnosed as epileptic do not have febrile convulsions, but 'further fits'.

## Background

Febrile convulsions are the most common cause of convulsions in children aged between 6 months and 5y. They affect 3% of children. Although 30% recur in childhood, only 1% go on to develop epilepsy in adult life.

When the patient first presents to ED either still having a fit or post-ictal, it is often not immediately apparent that the underlying problem is a febrile convulsion.

## Management

- Treat patients who arrive having a convulsion with $O_2$, airway care, and IV lorazepam, PR diazepam, or buccal midazolam, as described in ➲ The fitting patient, p. 704.
- Check T°.
- Check BMG and treat hypoglycaemia.
- Give PR (or, if conscious, PO) paracetamol (15mg/kg).
- Examine thoroughly for a source of infection (throat, ears, chest, and particularly for meningitis).
- Consider the need for an infection screen: U&E, FBC, blood cultures, MSU, CXR, and LP.

## Admission or discharge

Aim to discharge children aged >2y with a second or subsequent febrile convulsion and an obvious benign and treatable cause for pyrexia, with appropriate treatment. Liaise with the GP to consider arranging for parents to administer PR diazepam or buccal midazolam to terminate future febrile fits.

Refer for admission children with one or more of the following:

- Age <2y.
- A first febrile fit.
- Underlying serious infection.
- An unknown cause of pyrexia.

# Funny turns

Only a minority of reported 'funny turns' are epileptic fits. Most require referral and investigation. The history is crucial—the likely underlying causes vary according to the age of the child.

## Infants

Irregular and varying depth of respiration during sleep is normal but can cause parental alarm. Self-limiting apnoeic or cyanotic episodes may be due to: fits, inhaled FBs, near-miss cot death, gastro-oesophageal reflux and laryngeal spasm, or arrhythmias (eg SVT).

## Toddlers

Breath-holding attacks commonly accompany frustration in toddlers. They may cause the toddler to turn blue, lose consciousness, and even have a brief fit. Reflex anoxic episodes ('pallid syncope') are due to excess vagal stimulation in illness or after injury. Bradycardia, pallor, and loss of consciousness are occasionally accompanied by a short fit.

## Older children

Syncope on exertion is suggestive of a cardiac cause—consider aortic stenosis, SVT, coarctation, or hypertrophic cardiomyopathy. Vasovagal episodes and hyperventilation also cause 'collapse'. Atypical or unheralded collapse or fits may be a feature of inherited long QT syndrome and is associated with torsades de pointes. Obtain an ECG in any child who presents with collapse or 'first fit'.

The decision to refer/admit or discharge depends upon the exact circumstances, including the past history of similar episodes.

# Diabetic ketoacidosis

DKA usually presents in a child who is known to have diabetes, but occasionally it can be the first presentation of diabetes.

Features include: altered conscious level, polyuria, polydipsia, nausea, vomiting, and abdominal pain. Children with DKA can die from cerebral oedema (unpredictable but has 25% mortality), aspiration pneumonia, or hypokalaemia. All of these are potentially avoidable with appropriate treatment.

Be careful not to misdiagnose the abdominal pain of DKA as a 'surgical abdomen' or to dismiss the child as 'hyperventilating' (the ↑ RR reflects profound metabolic acidosis). Call senior ED and paediatric staff when DKA is suspected.

## Causes

First presentation of diabetes in a previously well child. In a child with known diabetes, lack of insulin, change of therapy, and intercurrent viral illness can cause DKA. Fever suggests sepsis (it is not part of DKA).

## Initial assessment and management

(See https://www.bsped.org.uk and https://www.nice.org.uk for detailed guidance.)

- Open and maintain airway if not fully conscious.
- Give high-flow $O_2$.
- Weigh the child if possible.
- Consider inserting an NG tube if unconscious or vomiting to ↓ the risk of aspiration.
- Attach a cardiac monitor (look for tall T waves) and record the CRT/BP.
- Rapidly obtain venous access and check BMG (remember BMG often underestimates blood glucose in DKA), and estimate the weight.
- Take blood for glucose, U&E, FBC, and VBG (and ketones if available).
- Only if evidence of shock (tachycardia, prolonged CRT, hypotension), give 10mL/kg of 0.9% saline IV as a bolus; discuss with senior/expert if repeated boluses are required. Consider sepsis if there is any of: fever, hypothermia, hypotension, refractory acidosis, and lactic acidosis.

### *Confirm the diagnosis of DKA*

Check the history with the child and parents: polyuria, polydipsia, vomiting, abdominal pain, drowsiness, and ↑ RR.

Biochemical diagnosis is: glucose >11mmol/L, acidosis (pH <7.3), bicarbonate <15mmol/L, and capillary blood ketones >3mmol/L.

### *Assess severity/dehydration*

Make a clinical assessment of the degree of dehydration.

The degree of dehydration/severity of DKA can be determined by pH:

- pH ≥7.1 implies mild or moderate DKA(~5% dehydration).
- pH <7.1 implies severe DKA (~10% dehydration).

The major concern is cerebral oedema—aim for slow metabolic correction over 48hr.

### *Involve senior paediatric ± PICU staff*

Involve PICU if aged <2y, severe acidosis (pH <7.1), severe dehydration, or ↓ conscious level (↑ risk of aspiration and cerebral oedema).

## Management

*Oral or IV fluids*

- Treat DKA with PO fluids and SC insulin only if the child is alert, not vomiting, and not dehydrated.
- Treat with IV fluids and IV insulin if the child is not alert, is dehydrated, or has nausea/vomiting.
- Only give an IV fluid bolus (10mL/kg) if shocked. Only give further boluses on expert advice (and if >20mmol/L is given, subtract any additional bolus volume from the 48hr total fluid calculation).

*IV fluid management*

Calculate the total fluid requirement for the first 48hr by adding the esimated fluid deficit to the maintenance requirement. Assume a 5% fluid deficit if pH is ≥7.1, and a 10% deficit if pH is <7.1.

Calculate maintenance fluid requirement using the 'reduced volume' rules:

- If weight is <10kg, give 2mL/kg/hr maintenance.
- If weight is 10–40kg, give 1mL/kg/hr maintenance.
- If weight is >40kg, give a fixed maintenance volume of 40mL/hr.

The fluid therapy calculator on https://www.bsped.org.uk is useful.

- Replace the deficit evenly over 48hr to ↓ the risk of cerebral oedema.
- Start replacing with 0.9% saline + 40mmol/L KCl (unless renal failure), but any initial fluid bolus should be 0.9% saline without KCl.
- Change fluid to 0.9% saline + 40mmol/L KCl + 5% glucose once plasma glucose level falls to below 14mmol/L.
- If plasma glucose falls <6mmol/L, ↑ glucose concentration of IVI—if there is persisting ketosis, continue insulin IVI at least 0.05U/kg/hr.
- Consider stopping IV fluids if ketosis is resolving and the child is alert and able to take PO fluids without nausea/vomiting.

*Insulin*

- Do not start insulin until IV fluids have been running for at least 1hr—earlier insulin ↑ risk of cerebral oedema. Give 0.05–0.1U/kg/hr (adding 50U of insulin to 50mL solution of 0.9% saline provides a concentration of 1U/mL (so 0.1U/kg/hr = 0.1mL/kg/hr).
- Do not administer bicarbonate.

*Monitoring*

- Perform hourly observations (pulse, BP, RR, T°, GCS).
- Monitor fluid balance (input/output chart).
- Monitor ECG whilst receiving IV therapy (look for U waves).
- Check blood glucose hourly and blood ketones every 2hr.
- Check U&E after 2hr of treatment.

*Cerebral oedema*

Suspect cerebral oedema if headache, agitation, ↓↓ pulse, and ↑ BP develops. Get expert help. Give mannitol (20%, 0.5–1g/kg over 15min) if ↓ GCS, pupil dilatation or inequality, oculomotor palsy, and respiratory pauses.

*Hypokalaemia*

If $K^+$ drops to <3mmol/L, get expert help and consider temporarily suspending insulin IVI.

# Urinary tract infection

UTI in children requires prompt investigation, since progressive renal failure and hypertension may occur insidiously. 35% have proven vesico-ureteric reflux—early treatment may help to prevent renal failure. UTI may present in a variable and non-specific fashion. Consider and exclude UTI as part of the initial approach to any ill child presenting to the ED.

## Presentations

Older children typically present with lower abdominal pain, dysuria, frequency, offensive urine, haematuria, or fever. However, dysuria and frequency do not always reflect UTI. Children <3y old often present unwell with fever and irritability, but no specific signs. Infants may present with poor feeding, vomiting, and failure to thrive.

## Examination

Always check the BP, and feel for loin tenderness (pyelonephritis) and abdominal masses (polycystic kidneys). Check T° and assess as for ➲ The sick febrile child, p. 680.

## Investigation

Obtain a clean-catch specimen of urine for urinalysis, microscopy, and culture and sensitivity. This can be difficult, but try the following approaches.

*Neonates and infants*

- Clean the perineum with sterile water, then tap with two fingers (or rub the skin gently with a gauze swab soaked with cold water) just above the symphysis pubis (ideally 1hr post-feed) and catch the urine which is forthcoming, trying to avoid the first few millilitres.
- Clean the perineum as above and use a urine collection pad according to the manufacturer's instructions.
- Suprapubic aspiration is useful if the baby is seriously ill. Clean the skin with antiseptic solution, then using sterile gloves and an aseptic technique, insert a 21G needle in the midline 2.5cm above the pubic crest and aspirate urine.

*Toddlers and older children*

- Co-operation will enable an MSU to be obtained [in the ♂, gently retract the foreskin (if possible) and clean the glans first; in the ♀, separate the labia and clean the perineum front to back first].
- If the child is unco-operative, try a urine collection pad or bag.

Dipstick urinalysis at the bedside will reveal the presence of blood, protein, sugar, bilirubin, ketones, or nitrite. A positive nitrite test is accepted as good evidence of UTI. Urine pH is not usually helpful, for although pH <4.6 or >8.0 may reflect infection, it may also be due to various acid–base disorders. Urinalysis may be normal, despite bacteriuria. Urinary leucocyte esterase may also help to identify UTI. Urine microscopy allows a search for pyuria and bacteriuria (highly suggestive of UTI) and an accurate assessment of other constituents (see Table 15.7). Perform FBC, U&E, blood glucose, and blood cultures if septicaemic, loin pain, or ↑ T°.

## Treatment

(See NICE guideline CG54, updated 2018, available at: https://www.nice.org.uk)

- *Children with suspected pyelonephritis or who appear toxic*: resuscitate as necessary with IV fluids (see The sick febrile child, p. 680), and refer for admission and IV antibiotics (eg cefuroxime) (see *BNFC*). Consider children who have a T° >38°C or those who present with loin pain/tenderness and bacteriuria to have pyelonephritis.
- *Symptomatic children with abnormal urinalysis (+ve nitrite, proteinuria, or haematuria)*: start a 3-day course of antibiotics PO (eg trimethoprim or cefalexin—dose according to age; refer to *BNFC*). Encourage plenty of PO fluids and complete voiding of urine. Offer advice to the child and parents (eg avoid tight underwear, use toilet paper wiping from front to back).
- *Organize paediatric or GP follow-up to receive results of MSU and to arrange subsequent investigations*: this may include U&E, blood glucose, USS, and a variety of other tests (eg isotope renography and micturating cysto-urethrography), according to local policy.
- Recurrent UTIs with anogenital signs may be due to sexual abuse.

**Table 15.7** Urine microscopy findings and their significance

| | |
|---|---|
| Red cells | Normally <3/mm$^3$ |
| White cells | Normally <3/mm$^3$ |
| Epithelial cells | Present normally—shed from urinary epithelium |
| Bacteria or fungi | Always abnormal, reflecting infection or specimen contamination |
| Casts | Hyaline casts: comprise Tamm–Horsfall protein—may be normal, but ↑ in fever, exercise, heart failure, and after diuretics<br>Fine granular casts—may be present normally, eg after exercise<br>Coarse granular casts—abnormal, seen in various renal disorders<br>Red cell casts—imply glomerular disease and glomerular bleeding<br>White cell casts—occur in glomerulonephritis and pyelonephritis<br>Epithelial casts—usually reflect tubular damage |
| Crystals | Phosphate, urate, and oxalate crystals may not be pathological but are also seen in *Proteus* UTI and hyperuricaemia |

# Haematuria

## Background

Dark or discoloured urine is frightening for both the child and parents. Although it may reflect haematuria, it may reflect other causes: very concentrated urine, beetroot, porphyria, conjugated hyperbilirubinaemia, and free Hb or myoglobin (usually black, as seen in rhabdomyolysis and malaria). Certain drugs or foods may discolour the urine (see Table 15.8).

**Table 15.8** Possible alternative causes of discoloured urine

| Drug/food | Colour |
|---|---|
| Rifampicin | Orange/pink |
| Desferrioxamine, senna, rhubarb | Brown |
| Methylthioninium chloride (methylene blue) | Green |

If haematuria is confirmed by urinalysis, obtain a detailed history, remembering to ask about preceding illnesses and trauma, foreign travel, drug history, and family history of renal or bleeding disorders.

A full relevant examination includes BP and a careful check for abdominal masses and oedema.

## Causes of macroscopic haematuria

- UTI (including schistosomiasis).
- Glomerulonephritis.
- Trauma.
- Wilm's tumour.
- Bleeding disorder.
- Urinary tract stones.
- Drugs (warfarin, cyclophosphamide).
- Factitious.

*Microscopic haematuria* may be associated with exercise or hypercalciuria or can be familial.

## Investigations

Send MSU and obtain USS of the urinary tract if there is abdominal pain suggesting stones (relatively rare). Check U&E, blood glucose, FBC, clotting screen, and, if significant bleeding (or if haematuria follows trauma), cross-match. Further tests may be required (throat swab, urine and serum osmolalities, viral titres, anti-streptolysin O, antinuclear antibodies, complement levels), but do not assist emergency treatment.

## Management

Severe haematuria with clots requires resuscitation with IV fluids (± blood) but is uncommon in children, except after trauma. Treat associated severe hypertension or hyperkalaemia associated with renal failure as described in ➲ Acute kidney injury, pp. 714–15. Refer children with haematuria of non-traumatic origin to the paediatrician.

### Glomerulonephritis

Glomerulonephritis in children is often an immune reaction following an URTI due to β-haemolytic streptococcal infection 2–3 weeks previously. It may present with haematuria, oliguria ± hypertension and uraemia. Refer for admission and further investigation.

A similar presentation can occur with Henoch–Schönlein purpura (see ➔ Purpuric rashes, p. 681), SLE, or Berger's disease (mesangial IgA nephropathy).

# Acute kidney injury

## Causes

*Pre-renal* Hypovolaemia (bleeding, dehydration, sepsis), heart failure, nephrotic syndrome.

*Renal* Haemolytic uraemic syndrome, glomerulonephritis, acute tubular necrosis, drugs.

*Post-renal* Obstruction following trauma or calculi.

## Presentation and investigation

Presentation varies according to the cause. Emergency investigations include MSU for microscopy, culture and sensitivity, urine and plasma osmolality, U&E, blood glucose, FBC, albumin, LFTs, clotting screen, and ECG monitoring.

## Treatment

Get expert help early. Pre-renal failure from hypovolaemia (urine:plasma osmolality ratio usually >5) should respond to treatment of the underlying condition and an IV fluid challenge (20mL/kg of 0.9% saline ± blood products, depending on the cause). Urinary catheter and close monitoring may help to assess fluid status. Urgent USS can assess for obstruction of the urinary tract, the presence of stones, and vascular filling status. ED treatment of renal failure focusses on hyperkalaemia and hypertension.

### *Hypertension*

Hypertension related to volume overload in renal failure may require IV nitrate therapy (± diuretic) in the ED (as for pulmonary oedema), but otherwise seek expert help for further intervention.

### *Hyperkalaemia*

Children presenting with hyperkalaemia ($K^+$ >7) in advanced renal failure may require emergency measures prior to dialysis.

Adopt the following approach to manage hyperkalaemia:

- Obtain expert help.
- Place the child on a cardiac monitor and obtain an ECG.
- If there are ECG changes (widened QRS complexes or tall T waves), give 0.5mL/kg of 10% calcium gluconate over 5min to stabilize the myocardium. This will not significantly alter the blood $K^+$ level.
- Give nebulized salbutamol (2.5mg if <3y; 5mg if 3–7y; 10mg if >7y). This redistributes and forces $K^+$ into cells within 30min and may be repeated after 2hr.
- Recheck $K^+$ and if falling after salbutamol, give calcium resonium 1g/kg PO or PR. If $K^+$ remains high after salbutamol, assess the pH—if pH <7.34, give sodium bicarbonate 1–2mmol/kg IV; if pH >7.34, give 10% glucose 5mL/kg/hr IV and insulin 0.05U/kg/hr.
- Plan dialysis as necessary.

Note that this approach is also appropriate for other causes of hyperkalaemia (eg adrenal insufficiency, acidosis, cell lysis).

### Nephrotic syndrome

Most cases of oedema, heavy proteinuria, and hypoalbuminaemia (± hypercholesterolaemia) are idiopathic ('minimal change nephropathy'). The presentation is diverse and includes: anorexia, lethargy, frothy urine, mild diarrhoea, abdominal pain, ascites, oliguria, and peri-orbital or genital oedema. The prognosis is generally good, but peritonitis and renal or cerebral venous thrombosis may occur.

Check U&E, albumin, LFTs, FBC, complement, cholesterol, and lipids. Refer for further investigation/treatment.

### Haemolytic uraemic syndrome

Micro-angiopathic haemolytic anaemia, thrombocytopenia, and renal failure of haemolytic uraemic syndrome typically affect infants/toddlers following a diarrhoeal illness (*Escherichia coli* O157, verocytotoxin, or *Shigella*). The disease is also associated with SLE, HIV, and various tumours. The child may present oliguric or anuric, with ↓ conscious level due to encephalopathy. Mortality is >5%.

FBC reveals anaemia with visible RBC fragments, thrombocytopenia, and leucocytosis. Coombs' test is –ve. Urea and creatinine levels are usually ↑, and there may be electrolyte disturbances.

Treat life-threatening hyperkalaemia as above, and refer for possible dialysis and transfusion.

# Poisoning in children

Paediatric poisoning may take many forms:

- Neonatal poisoning from drugs taken by the mother prior to birth (eg opioids, benzodiazepines).
- 'Accidental' (unintentional) poisoning is the most common form of poisoning. It largely involves toddlers and preschool children (boys > girls), who are at particular risk because of their innate curiosity and considerable indiscretion in putting things in their mouths. Children may be poisoned by any drugs that they can get their hands on, but also mushrooms, berries, plants, household items (eg disinfectant), and other objects misinterpreted as drink, food, or sweets (eg button batteries).
- Inadvertent self-poisoning with recreational drugs (including alcohol and volatile agents).
- Iatrogenic poisoning by administration of the wrong dose ± wrong drug can happen with frightening ease. Paediatric dosage charts, calculators, obsessional checking, attention to detail, and automatic checks via electronic prescribing should help to prevent this.
- Deliberate self-poisoning in an apparent suicide attempt occurs in (mostly) older children.
- Intentional poisoning by a parent, guardian, or carer is a sinister aspect of child abuse, which includes fabricated or induced illness (see ➲ Fabricated or induced illness, p. 760). The child may present in a bizarre fashion, with a non-specific illness, for which the diagnosis is not immediately apparent.

## Approach

Follow the general guidelines described in ➲ Poisons: background, pp. 188–9; ➲ Diagnosis of poisoning, p. 190; ➲ Poisons: supportive care, p. 191; ➲ Reducing absorption of poison, pp. 192–3; and ➲ Antidotes for poisons, pp. 194–5 to treat poisoned patients, with initial attention to oxygenation (airway), ventilation (breathing), and circulation. The National Poisons Information Service (🖱 https://www.toxbase.org) provides advice for specific poisonings (see ➲ Poisons: background, pp. 188–9). With some notable exceptions (eg paracetamol, opioids, iron, and digoxin), there are few 'antidotes' available—treatment is often largely supportive.

Try to elicit the substance(s) ingested, the amount involved, and the time since ingestion. The majority of ingestions are unintentional and the time to presentation is often short.

### *Gastric emptying*

Procedures designed to empty gastric contents (eg gastric lavage) are rarely indicated—consider only if advised by TOXBASE®. Do not use 'ipecac' (ipecacuanha), which is ineffective in ↓ drug absorption and can be dangerous. *Never* try to empty the stomach following ingestion of petrol or corrosives (see ➲ Petrol and paraffin poisoning, p. 213).

### *Charcoal*

The role of charcoal (dose 1g/kg PO in infants; 15–30g in older children) in paediatric poisoning is limited by its lack of palatability. Attempts are currently being made to make charcoal more palatable, yet remaining effective.

# Prevention of paediatric poisoning

## Background

Poisoning in children is very common. More than 40,000 children present to hospital in the UK each year, many of whom are admitted for observation. Thankfully, relatively few (10–15/y) die. More than 75% of paediatric unintentional ingestions involve drugs and poisons in the home that are plainly visible to the child. Poisoning is particularly likely to occur at times of 'stress' (eg arrival of new baby, disturbed parental relationships, moving house) when there may be ↓ supervision and disruption of the usual routine. Perhaps partly for this reason, children who present with a first episode of poisoning are at ↑ risk of further episodes. It is therefore important to advise the parents of ways of preventing poisoning in children (see list in ➲ Advice for parents (consider providing a leaflet), p. 717).

## Official measures: packaging of drugs

Legislation has been introduced to try to tackle the problem of poisoning in children. Perhaps the most successful has been the widespread adoption of child-resistant drug containers. Unfortunately, it is not yet mandatory for these containers to be used for liquid drugs or potentially dangerous household items such as bleach. Some drugs are presented in 'strip packaging', in the hope that an impulsive child would lose interest before gaining access to a significant quantity.

## Advice for parents (consider providing a leaflet)

- Provide adequate supervision for toddlers and young children, particularly when visiting friends and relatives.
- Keep all medicines locked out of reach in a cupboard.
- Only purchase those drugs presented in child-resistant containers.
- Dispose of out-of-date drugs and those no longer required.
- Never refer to drugs as 'sweets' in an attempt to encourage the child to take them.
- Take medicines out of sight of the child to help prevent imitation.
- Keep all alcohol, perfumes, cosmetics, detergents, and bleaches out of reach.
- Ensure that all turpentine, paints, and weed killers are securely locked and inaccessible.
- Give away all toxic plants.
- Keep ashtrays and waste baskets empty.

# Gastroenteritis in children

(See also an overview in ➲ Gastroenteritis/food poisoning, pp. 236–7.)

*A baby's parents* may seek advice about diarrhoea when, in fact, the stools are normal. Breastfed babies almost always have loose stools, which may be yellow or green and very frequent, often after every feed. However, gastroenteritis is relatively rare in fully breastfed babies. In children aged >6 months, normal stool frequency ranges from one stool on alternate days to three stools daily.

## Assessment of dehydration

*Clinical evidence of mild dehydration (<5%)*

- Thirst.
- ↓ urinary output (in a baby <4 wet nappies in 24hr).
- Dry mouth.

*Clinical evidence of moderate dehydration (5–10%)*

- Sunken fontanelle in infants.
- Sunken eyes.
- Tachypnoea (due to metabolic acidosis).
- Tachycardia.

*Clinical evidence of severe dehydration (>10%)*

- ↓ skin turgor on pinching the skin.
- Drowsiness/lethargy or irritability.

## Admission decision

It can be difficult to decide whether or not to admit a child to hospital for treatment. Admit if the child looks seriously ill, is clinically >5% dehydrated, has not passed urine for >12hr, or has a high fever, or there is doubt about the diagnosis or the family are unlikely to cope at home.

Refer for admission children with bloody and/or mucoid diarrhoea—to exclude *Escherichia coli* O157 infection, which may ↑ the risk of developing haemolytic uraemic syndrome.

Babies aged <3 months may be difficult to assess and can deteriorate rapidly—refer for admission.

In children who are less seriously ill, consider making the decision about admission based upon the response to oral rehydration therapy.

## Management

Treat severely dehydrated (>10%) children with immediate IV fluids, initially 0.9% saline (10mL/kg over 5min, repeated as necessary).

Consider IV fluids for children with moderate dehydration, especially if they are vomiting and unable to keep oral fluids down.

Give a trial of oral rehydration therapy to children with mild dehydration, with the aim of discharging them if the trial feed is successful (see ➲ Oral rehydration therapy, p. 719).

## Oral rehydration therapy

(See NICE guidance—https://www.nice.org.uk)

If a child with mild dehydration makes a satisfactory response to a test feed, consider discharge with oral rehydration therapy. Standard products (eg Dioralyte®) contain glucose, $Na^+$, $K^+$, $Cl^-$, and citrate (details in *BNF*). Glucose is important to enhance absorption of $Na^+$ and water.

Rehydrate according to age:

- Children aged ≤5y: give 50mL/kg of oral rehydration therapy for fluid deficit replacement over 4hr, as well as maintenance fluid (see Table 15.9).
- Children >5y: give 200mL of oral rehydration therapy after each loose stool, in addition to maintenance fluid (see Table 15.9).

*Advice for parents*

- Give oral replacement therapy frequently and in small amounts, and seek urgent medical advice if the child vomits repeatedly or is unable to drink.
- If the child is slow to recover, give 5mL/kg of oral rehydration therapy after each large watery stool.
- Avoid solid food until dehydration has been corrected, then reintroduce the usual diet, but avoid fruit juice and fizzy drinks until diarrhoea has stopped, as these often have high osmolarity and may worsen diarrhoea.

*Additional treatments*

Do not prescribe anti-diarrhoeal agents, probiotics, or antiemetic drugs for children with gastroenteritis. Similarly, do not give antibiotics without specialist advice (eg proven *Salmonella* in immunocompromised or young babies).

**Table 15.9** Daily maintenance fluid requirements in children

| Child weight | Daily maintenance fluid volume |
|---|---|
| 0–10kg | 100mL/kg |
| 10–20kg | 1000mL + 50mL/kg for every kg over 10kg |
| >20kg | 1500mL + 20mL/kg for every kg over 20kg |

# Abdominal pain in children

The approach to the initial assessment and management of children presenting with abdominal pain is similar in many ways to that in adults (see ➲ Approach to abdominal pain, pp. 520–1). Beware underlying 'medical' causes (eg DKA, pneumonia). Disease processes may progress with great rapidity in children, so adopt a low threshold for referring children with abdominal pain to the surgical team. Whilst many of the common causes of abdominal pain are the same in children as in adults (eg ➲ Acute appendicitis, p. 523), be aware of causes that are typically paediatric (eg intussusception). Likewise, certain causes of intestinal obstruction are seen almost exclusively in children. Avoid performing PR examination.

## Paediatric causes of intestinal obstruction

- Congenital (eg oesophageal/duodenal atresia, Hirschsprung's disease).
- Meconium ileus.
- Hypertrophic pyloric stenosis.
- Intussusception.
- Hernia (inguinal, umbilical).

## Hypertrophic pyloric stenosis

*Features*

Relatively common, this typically presents with effortless vomiting at 2–10 weeks. It occurs more often in boys than girls and in first-born children. Vomiting becomes projectile, with progressive dehydration and constipation. The vomit is not bile-stained. After vomiting, the baby appears hungry and keen to feed again. In advanced cases, there may be profound hypochloraemic alkalosis, with associated hypokalaemia.

*Diagnosis*

Look for visible peristalsis. Abdominal palpation confirms the diagnosis if an olive-sized lump is felt in the epigastrium (most prominent during a test feed). If the diagnosis is suspected, but not proven clinically, resuscitate (as below) and arrange USS.

*Management*

Once diagnosed, keep the infant nil by mouth. Insert an IV cannula and send blood for U&E, glucose, and FBC. Start fluid resuscitation under senior guidance and refer to the surgeon—operative treatment will be delayed until dehydration and electrolyte abnormalities have been corrected (this may take >24hr). Defer insertion of an NG tube for appropriately experienced staff.

## Volvulus

This is associated with congenital malrotations but may occur in other circumstances also (eg Meckel's diverticulum, adhesions from previous surgery). It can present with abdominal pain and other features of intestinal obstruction (vomiting, distension), sometimes with a palpable mass. Obtain an abdominal X-ray and refer promptly to the surgical team in order to maximize the chance of intervening to preserve bowel.

### Intussusception

Telescoping of one segment of bowel into another may affect the small or large bowel, but most cases are ileocolic. This typically affects children aged between 6 months and 4y. The child may suddenly become distressed, roll up into a ball, and appear unwell. Vomiting may develop and the child may pass a 'redcurrant jelly' stool. These features, however, together with pyrexia and a palpable mass, are not invariably present—sometimes the presentation is shock without an obvious cause. X-rays may be normal or reveal an absent caecal shadow.

If intussusception is suspected, refer urgently to the surgical team. The diagnosis may be confirmed by air or barium enema, which may also be curative, by reducing the intussusception. A barium enema characteristically reveals a 'coiled spring' sign or sudden termination of the barium but is contraindicated if there is evidence of perforation.

### Acute appendicitis

(See ➲ Acute appendicitis, p. 523.)

Consider this diagnosis in any child presenting with abdominal pain. Acute appendicitis can occur in children of all ages. It can be a difficult diagnosis to make, especially in the very young. 'Atypical' clinical presentation (eg diarrhoeal illness) is often associated with delayed diagnosis and an ↑ rate of perforation. Do not perform a PR examination—in the unlikely event of this being considered essential, leave it to the surgical team.

### Abdominal mass

There are many causes of abdominal masses in children, many of which may be relatively benign and asymptomatic:

- Full bladder.
- Full colon.
- Enlarged liver and/or spleen.
- Pregnancy in older children.
- Hydronephrosis.
- Hypertrophic pyloric stenosis (see ➲ Hypertrophic pyloric stenosis, p. 720).
- Appendix mass.
- Intussusception.
- Volvulus.
- Neuroblastoma.
- Nephroblastoma (Wilm's tumour).

### Intra-abdominal malignancy

Neuroblastoma and nephroblastoma may reach a large size before causing symptoms (eg haemorrhage into the tumour).

*Neuroblastomas* Arise most commonly from the adrenal glands but may occur at any point along the sympathetic chain.

*Nephroblastomas (Wilm's tumours)* Arise from the kidneys and may present with haematuria.

All children with suspected malignant abdominal masses require CT scan and/or USS investigation—refer urgently to the surgical team.

# Inguinal and scrotal swellings

## Painless groin and scrotal lumps

The parent or child who discovers a lump may become very concerned. The absence of pain is, to some extent, reassuring, in that an acute surgical problem is unlikely. Ascertain when the swelling appeared, whether it changes in size or disappears, or whether there are any other symptoms.

### *Reducible inguinal hernia*

Inguinal hernias in childhood result from a persistent patent processus vaginalis and are therefore indirect in nature. They are more common in boys than girls and often bilateral. The history is typically of an intermittent swelling, which appears with coughing or straining. If the swelling can be demonstrated, it will be impossible to get above it. If it cannot be demonstrated, a thickened spermatic cord may be palpated (sometimes known as the 'silk sign'). Refer neonatal hernias for admission and surgery, and refer infants and older children to a surgical clinic for elective surgery.

### *Painless irreducible inguinal hernia*

Refer all irreducible inguinal hernias for admission and surgery (preceded by gallows traction in the infant).

### *Hydrocele*

This transilluminable painless scrotal swelling has a similar aetiology to inguinal hernia. It appears gradually, rather than suddenly, and does not empty or reduce on palpation. Refer to a surgical clinic. An encysted hydrocele of the cord may be impossible to distinguish from an irreducible inguinal hernia and therefore requires surgical exploration.

### *Undescended, retractile, or ectopic testis*

Complete descent of the testis has yet to occur in 3% of term infants and 30% of premature infants. Arrange surgical follow-up if the testis cannot be brought down to the fundus of the scrotal sac—orchidopexy will be required if the testis fails to descend by 4y.

### *Inguinal lymphadenopathy*

This is on the list of differential diagnoses of painless inguinal swellings. Look for a potential source of infection in the leg and for involvement of any other lymph node groups.

### *Idiopathic scrotal oedema*

An obscure allergic condition of the scrotal skin is possibly a variant of angioneurotic oedema. Redness, mild tenderness, and oedema are not limited to one hemiscrotum. The testis is normal. The condition settles spontaneously, a process helped by antihistamines (eg chlorphenamine PO, doses: child 1–2y require 1mg bd; 2–5y require 1mg qds; 6–12y require 2mg qds). If in doubt—refer.

## Painful groin and scrotal lumps

*Painful irreducible inguinal hernia*

Likely to contain obstructed or strangulated small bowel. Confirm clinical suspicion of intestinal obstruction (pain, vomiting, and abdominal distension) by X-ray. Resuscitate as necessary with IV fluids; give analgesia, and refer for surgery.

*Testicular torsion*

Most common in the neonatal period and around puberty. In the neonatal period, torsion is extravaginal in nature and often diagnosed late. Later in childhood, torsion of a completely descended testis is intravaginal due to high insertion of the tunica vaginalis. Undescended testes are also at particular risk of torsion. Classical presentation is with sudden-onset severe pain and vomiting. Occasionally, the pain is entirely abdominal. Examination reveals a red, tender, swollen testis. The opposite testis may be seen to lie horizontally, rather than vertically (Angell's sign). Fast and refer all suspected torsions for urgent surgery: exploration, untwisting, and bilateral orchidopexy.

The diagnosis of testicular torsion is not always clear-cut—USS can sometimes be helpful in making the diagnosis, but do not allow this to delay referral to the surgical team.

*Torsion of the hydatid of Morgagni*

This remnant of the paramesonephric duct on the superior aspect of the testis is prone to undergo torsion, causing pain and vomiting. The pain is typically less severe than in testicular torsion, with a more gradual onset. A discrete, tender (~3cm) nodule may be palpable near the upper pole of the testis—the classic description is of it transilluminating as a blue dot, but this is rarely seen in practice. In contrast to testicular torsion, the remainder of the testis is not tender.

Refer to the surgical team and consider an urgent USS. If testicular torsion is excluded and a definite diagnosis of torsion of the hydatid of Morgagni is made, the surgical team can choose between conservative treatment (analgesia and rest) or surgical excision of the hydatid.

*Epididymo-orchitis*

Relatively unusual in the paediatric age group but may be associated with UTI. A painful, swollen red testis and epididymis usually develop over a longer period of time. Treatment is with antibiotics (eg ciprofloxacin), but it may be difficult to distinguish from testicular torsion, so refer for an urgent surgical opinion.

*Mumps orchitis*

The diagnosis is usually apparent because of parotitis (see ➲ Childhood infectious diseases, pp. 230–1). Refer if there is doubt or symptoms are severe.

*Henoch–Schönlein purpura*

Occasionally, testicular pain may be one of the initial presenting complaints of Henoch–Schönlein purpura (see ➲ Purpuric rashes, p. 681).

# Foreskin problems and zip entrapment

## Phimosis

The foreskin may normally remain non-retractile up to age 5y. Foreskin that remains non-retractile after this, which 'balloons' on micturition, or is associated with recurrent balanitis may benefit from surgery (preputial stretch or circumcision). Advise the parents to see their GP to discuss referral to a paediatric surgeon.

## Balanitis

Balanitis produces redness, swelling, and even pus. Take a swab; check for glucose in the urine and send an MSU. Treat with PO flucloxacillin or clarithromycin, and suggest GP follow-up. If redness and swelling involve the whole penis, refer for IV antibiotics.

## Paraphimosis

An irreducible, retracted foreskin results in pain and swelling of the glans. As in the adult, cold compresses and lubricating jelly may allow manual reduction to be performed. If this is not successful, refer for reduction under GA.

## Penile zip entrapment

Unfortunately, underpants do not completely protect boys (and sometimes men) from catching their foreskins in trouser zips. On many occasions, the entrapment will be released quickly by the child or parent. On others, the child will present to the ED.

*The optimal method to achieve release depends upon the entrapment*

- 15% of entrapments follow the foreskin moving through the moveable part of the zip, so that it is simply caught between the teeth of the zip alone. In this case, achieve easy release by cutting transversely through the zip below the entrapment.
- 85% of entrapments involve the foreskin being caught between the teeth and the moveable part of the zip. LA (either injection using plain lidocaine or topical gel) may allow manipulation and release. If this fails, the least traumatic option is to divide the moveable part of the zip into two parts by dividing the central section ('median bar' or 'bridge') using bone cutters or wire cutters (use gauze to protect against parts of the zip flying off) (see Fig. 15.18). Older children and adults may tolerate this in the ED, but in younger boys referral for release under GA is sensible. Circumcision is rarely required.

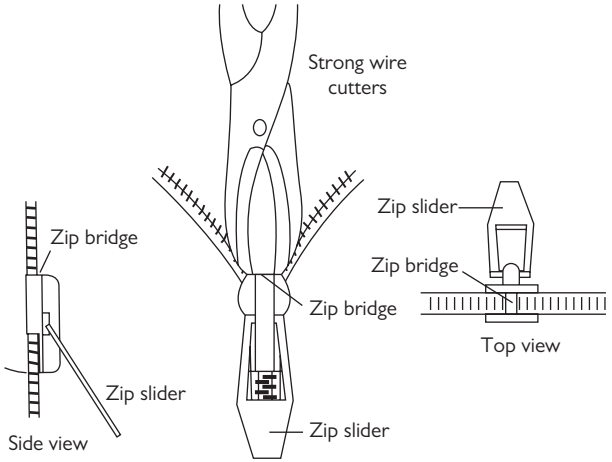



**Fig. 15.18** Method to achieve release from zip entrapment.

# The limping child

This common problem can cause diagnostic difficulty, particularly in the young child who cannot provide a history and is difficult to examine. Start by searching for, and trying to, exclude some causes of a limp that will require urgent treatment.

## Consider the following

- Trauma (fractures, soft tissue injury, FB in foot, NAI).
- Specific hip problems (Perthes', slipped epiphysis, irritable hip—see ➔ The painful hip, pp. 728–9).
- Infection (osteomyelitis, septic arthritis).
- Arthritis (juvenile idiopathic arthritis, juvenile ankylosing spondylitis).
- Osteochondritis (see ➔ Osteochondritis, pp. 730–1).
- Referred pain from an inflammatory process elsewhere.
- Malignant disease (Ewing's sarcoma, leukaemia).
- Sickle-cell crisis (see ➔ Sickle-cell disease, pp. 184–5).

Adopt the following approach.

## History

Ascertain whether the problem developed suddenly (eg after trauma) or gradually. Enquire about recent illness and other symptoms, including joint pains elsewhere.

## Examination

Check T°. If the child is walking, assess the gait. Carefully inspect all of the painful leg for erythema, swelling, and deformity, and note the position adopted. Exclude a relatively simple problem such as a FB embedded in the foot. Note any skin rashes. Palpate the limb for tenderness, joint effusions, and range of movement (compare with the other side). If the child will not walk but can crawl without any apparent discomfort, this localizes the problem to below the knee (thereby avoiding the need to request 'routine' X-rays of the hips).

## Investigation

If the child can walk and looks well, and there is no abnormality apparent on examination, consider providing analgesia and arranging to review after a couple of days, rather than undertaking all of the following investigations immediately. Ensure the parents are told that they should return earlier if the limp gets worse.

X-ray the tender or swollen part, particularly if there is a history of injury. If there is no obvious tenderness, X-ray the pelvis to include both hips. If the X-rays do not reveal a fracture, check WCC and CRP (or plasma viscosity/ESR). If the hip is implicated, but X-rays are normal, request USS of the hip (some experts prefer to use USS as the initial investigation). MRI is emerging as having a potentially useful role. Follow local ED protocols where available.

## Management

Treat according to the cause (see below; see also ➔ The painful hip, pp. 728–9; ➔ Osteochondritis, pp. 730–1).

### Trauma

Treat according to the cause, which may include a FB in the soft tissues. There may not always be a clear history of injury—this particularly applies to toddler's fracture (see ➲ Tibial fractures in children, p. 754). However, the abrupt onset of a limp in a toddler may be a clue to an underlying traumatic cause.

### Osteomyelitis

Acute osteomyelitis usually results from blood-borne spread of a distant pathogen (eg from the respiratory tract). *Staphylococcus aureus* is usually responsible, with almost invariable involvement of the metaphysis of a long bone (most commonly proximal or distal femur, or distal tibia).

*Features* ↑ T°, lethargy, localized tenderness (which may be misdiagnosed as trauma). Septic shock may occur (especially in infants).

*Investigations* ↑ WCC, ↑ CRP, ↑ ESR >50mm/hr (but all may be normal initially). Send blood cultures which may help to guide later antibiotic use. X-ray changes occur after ~10 days. MRI may enable an earlier diagnosis.

*Treatment* If suspected, refer for admission, IV antibiotics ± surgical drilling/drainage.

### Septic arthritis

Most commonly, *S. aureus* infection in the hip or knee, particularly affecting preschool children. Occasionally secondary to penetrating injury, but usually haematogenous spread from a distant site. Constitutional symptoms, fever, and joint pain occur. Joint movement is likely to be severely impaired. A joint effusion may be clinically evident (and confirmed on USS). Investigations may reveal ↑ WCC, ↑ CRP, and ↑ ESR. Refer for urgent confirmatory joint aspiration and treatment.

Traditionally, the four Kocher criteria (T° ≥38.5°C; non-weight-bearing on the affected side; ESR >40mm/hr; WCC >12 × $10^9$/L) have been used to estimate the chance of a child having septic arthritis of the hip. The presence of three criteria is associated with a >90% chance of septic arthritis.

### Non-septic arthritis

Multiple painful joints are more likely to be due to a juvenile arthritic process (eg juvenile idiopathic arthritis or ankylosing spondylitis) than septic arthritis. Pain felt in several joints frequently accompanies a variety of infections and other diseases (eg rubella, rheumatic fever, Lyme disease, Henoch–Schönlein purpura). Refer to the paediatrician for further investigation.

# The painful hip

The limping child may be able to localize pain to the hip, but hip pain may be referred to the knee. Hip problems causing a limp include trauma, infection, and other disorders, as described in ➲ The limping child, pp. 726–7. Specific hip problems include the following.

## Perthes' disease (Legg–Calvé–Perthes' disease)

Aseptic necrosis of the upper femoral (capital) epiphysis presents with a painful limp in children aged 3–10y. Boys are affected more than girls (♂:♀ = 4:1); 15% are bilateral. Aetiology is unclear, but Perthes' disease is often grouped with osteochondritides (see ➲ Osteochondritis, pp. 730–1). Often ↓ range of hip movement due to pain. FBC, CRP, ESR, and blood cultures are normal.

*X-ray changes* Reflect the stage of disease and are progressive (as shown in Fig. 15.19):

1 ↑ joint space on medial aspect of capital epiphysis (compare sides).
2 ↑ bone density in affected epiphysis (appears sclerotic).
3 Fragmentation, distortion (flattening), and lateral subluxation of upper femoral epiphysis (leaving part of the femoral head 'uncovered').
4 Rarefaction of the adjacent metaphysis in which cysts may appear.

*Treatment* Refer for specialist assessment and treatment. Most cases respond satisfactorily to conservative therapy.

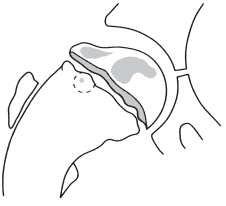


**Fig. 15.19** Changes in the hip in Perthes' disease.

## Irritable hip ('transient synovitis')

Common cause of sudden painful hip and limp in children of all ages. Aetiology is unclear, but many cases may follow viral illness. Presentation varies from a slight limp to great difficulty weight-bearing. X-rays are normal. USS may show hip effusion and allow aspiration for microscopy and culture. (Apply tetracaine cream over the hip before USS.) Pyrexia, ↑ WCC, ↑ CRP (and/or ↑ ESR/plasma viscosity) suggest infection.

*Treatment* If significant physical signs (significant pain, ↓ movement, difficulty weight-bearing) or there is evidence suggesting infection, refer to the orthopaedic team for admission for rest, traction, and further investigation. If physical signs are not dramatic and X-rays and blood tests are normal, discharge with NSAID, advise rest, and review within a few days.

### Slipped upper femoral (capital) epiphysis

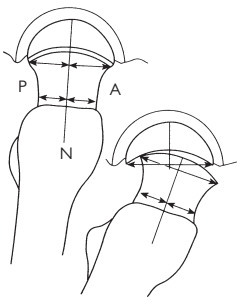
Sometimes occurs during puberty and has been attributed to hormonal imbalance (see Fig. 15.20). It occurs in children (particularly boys: ♂:♀ = 3:1) who have one of two body types: obese with underdeveloped genitalia or tall, thin, rapidly growing adolescent with normal sexual development. It may present with knee (not hip) pain.

*Presentation* A child aged 10–16y develops a painful limp suddenly or gradually. Often there is a history of trauma. The leg may be slightly adducted, externally rotated, and shortened. Movement of the affected hip is ↓, compared with the other side (especially abduction and internal rotation).

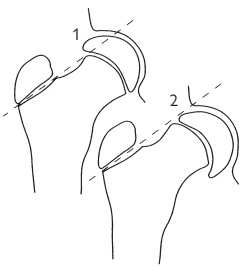
*X-ray* Obtain AP pelvis and lateral hip views ± 'frog leg' views. Subtle slips may only be seen on the lateral view. Larger slips will be obvious on all views. Look for Trethowan's sign—a line drawn along the superior border of the femoral neck normally cuts through the epiphysis (see Fig. 15.21).

*Treatment* Refer to orthopaedics for internal fixation ± manipulation.

*Complications* Avascular necrosis, chondrolysis, and osteoarthritis.




Lateral view showing normal (left) and abnormal (right)




AP view: Trethowan's sign; (1) normal, (2) abnormal hip

**Fig. 15.20** Diagrams of slipped upper femoral epiphysis.

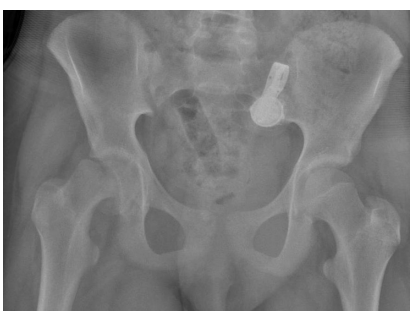


**Fig. 15.21** X-ray showing slipped right upper femoral epiphysis.

# Osteochondritis

This term is applied to a heterogenous array of non-infectious disorders affecting various epiphyses. They may be divided into three groups, according to the proposed aetiology (see Table 15.10).

## Crushing osteochondritis

Apparently spontaneous necrosis of an ossification centre occurs at a time of rapid growth. This is followed by new bone formation.

*Perthes' disease* See ➲ The painful hip, pp. 728–9.

*Scheuermann's disease* Fragmentation of low thoracic/upper lumbar vertebral epiphyseal plates of adolescents results in chronic back pain and a 'round-shouldered' kyphotic appearance. X-rays show anterior wedging of vertebral bodies, with sclerotic notches (Schmorl's nodes) on inferior or superior vertebral borders. Diagnostic criteria are >50° of kyphosis and wedging in three adjacent vertebrae. Treat symptomatically with NSAID and refer for orthopaedic follow-up.

*Kohler's disease* Avascular necrosis of the navicular affects children (particularly boys) aged 3–5y. A painful limp develops, with tenderness on the dorsum of the foot over the navicular. The sclerotic fragmented navicular seen on X-ray is also seen in many asymptomatic children. Treat symptoms with rest, NSAID, and orthopaedic follow-up. If symptoms are severe, consider a BKPOP.

*Kienbock's and Freiberg's disease* Usually affect young adults.

## Traction apophysitis

The pull of a strong tendon causes damage to the unfused apophysis to which it is attached.

*Osgood–Schlatter's disease* Traction apophysitis of the tibial attachment of the patellar tendon is especially seen in boys aged 10–15y. Symptoms may start after a period of excessive activity. Anterior knee pain after exercise is characteristic. The tibial tuberosity is prominent and tender. The pain may be reproduced by attempted extension against resistance.

X-rays are not always needed but may show an enlarged and sometimes fragmented tibial tuberosity. Treat symptomatically with rest, NSAID, and orthopaedic follow-up. Most settle with conservative measures, although children may find this process to be frustrating.

*Johansson–Larsen's disease (Sinding Larsen's disease)* Traction apophysitis of the lower pole of the patella in young adolescents results in local tenderness. Treat with rest, NSAID, and orthopaedic follow-up.

*Sever's disease* Traction apophysitis of the calcaneal attachment of the Achilles tendon occurs in 8–14y olds. The resulting limp is associated with local calcaneal tenderness. X-rays may reveal a fragmented sclerotic calcaneal apophysis. Treat with rest, NSAID, a heel raise, and orthopaedic follow-up.

## Osteochondritis dissecans

A piece of articular cartilage and adjacent bone become partially or completely separated as an avascular fragment. The cause is believed to be an osteochondral fracture from repeated minor trauma. The lateral aspect of the medial condyle of the distal femur is the most commonly affected site (see Fig. 15.22). Intermittent pain, swelling, and joint effusion result. If the fragment becomes detached as a loose body, locking or giving way may occur.

*X-ray* Demonstrates a bony fragment or defect. Often the small bony fragment is accompanied by a larger piece of cartilage (which is not seen on X-ray). MRI will demonstrate both.

*Treatment* Refer the locked knee immediately. Treat the remainder with rest; consider crutches, and arrange orthopaedic follow-up.

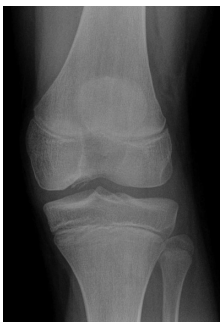


**Fig. 15.22** X-ray of osteochondritis dissecans in a 13y old.

**Table 15.10** Classification of osteochondritis

| Type of osteochondritis | Bone affected | Eponym |
|---|---|---|
| Crushing osteochondritis | Femoral head | Perthes' disease (see ➲ The painful hip, pp. 728–9) |
| | Vertebrae | Scheuermann's disease |
| | Second metatarsal head | Freiberg's disease |
| | Navicular | Kohler's disease |
| | Lunate | Kienbock's disease |
| | Capitulum | Panner's disease |
| Osteochondritis dissecans | Medial femoral condyle | |
| | Talus | |
| | Elbow | |
| | Metatarsal | |
| Traction apophysitis | Tibial tuberosity | Osgood–Schlatter's disease |
| | Lower pole of patella | Johansson–Larsen's disease |
| | Calcaneum | Sever's disease |

# Major paediatric trauma

## Background

Trauma is the largest single cause of death in children: ~500 deaths per year in the UK (see Table 15.11). As in adults, blunt injury in children is far more common than penetrating injury. The number of deaths in children after trauma is dwarfed by the number who sustain serious injuries. Most serious injuries result from road traffic collisions.

**Table 15.11** Causes of trauma deaths in children

| | |
|---|---|
| Road traffic collisions | 48% |
| Fires | 15% |
| Drowning | 12% |
| Hanging | 8% |
| Falls | 8% |
| NAI | 5% |
| Other | 4% |

More than 70% of paediatric trauma deaths occur in the prehospital setting. Most of these children are either dead when found or have sustained overwhelming injuries. The greatest potential for reducing trauma deaths clearly lies with injury prevention. However, there is enormous potential to reduce the number of permanently disabled children by early identification of injuries and expert treatment. The best outcome results from involvement of senior and experienced staff at an early stage. Prompt recognition of the seriously injured child is crucial to this.

## Pattern of injuries

Anatomical and physiological differences mean that the pattern of injuries in children differ considerably from those in adults. Compared with adults, children have: smaller physical size, a relatively larger head, more compliant bones, a higher ratio of surface area to body weight, and epiphyses. Experience and an awareness of the patterns of paediatric injury will assist resuscitation efforts. The smaller size and physical proximity of internal organs frequently result in the dissipated forces causing injuries to multiple structures (multiple injuries). The compliance of the bony thoracic cage in children allows significant underlying organ injury without rib fractures. Similarly, certain injuries not uncommon in adults (eg rupture of the thoracic aorta) rarely occur in children.

# Injury prevention

## Terminology

The term 'accident' implies an unforeseen unintentional event, one which occurs by chance. The implication is that 'accidents' cannot be prevented. However, there is much evidence that 'accidents' are far from random events but are relatively predictable and amenable to prevention. For this reason, experts now prefer to avoid use of the terms 'accidents' and 'accident prevention' and refer to 'injury prevention' instead. Similarly, 'accident and emergency deparments' have become 'emergency departments'.

## Background

Injuries to children tend to occur more frequently in certain groups and at certain times:

- Boys sustain more injuries than girls.
- Injuries are associated with social deprivation.
- Injuries often occur at times of family stress and change (including marital disharmony, moving house, and holidays).

## Prevention theory

Prevention of injury does not simply refer to physical injuries, but poisonings also. Injuries and/or the effects of injuries may be prevented in a number of different ways:

*Primary prevention measures* Stop injuries occurring, eg installing fences around domestic swimming pools may reduce drowning and locked medicine cabinets might prevent inadvertent poisoning.

*Secondary prevention measures* Reduce the extent of harm caused by an injurious event. The most obvious examples are helmets, seat belts, and air bags in the context of road traffic collisions.

*Tertiary 'prevention'* Includes first aid and hospital treatment, and aims to limit the effect of an injury after it has already happened (eg surgery to stop intra-abdominal haemorrhage, antidotes for certain poisons).

## Prevention strategies and the role of ED staff

The focus of staff treating injured patients has understandably always been the injuries themselves ('tertiary prevention'). In addition to any possible issues of NAI, ED staff need to consider how future injuries to children might be prevented (eg by discussing with parents the benefits of bicycle helmets). In the context of an individual child, it may sometimes be appropriate to contact the GP/health visitor with a view to seeing if interventions might prevent future injuries to a particular child and siblings.

More general interventions include:

- Leaflets and posters in the waiting room to target a captive audience.
- Media involvement (eg to minimize risks of fireworks and sparklers).

Further details of children's injuries and injury prevention are available from the Royal Society for the Prevention of Accidents (https://www.rospa.org.uk) and the Child Accident Prevention Trust (https://www.capt.com).

# Resuscitation of the injured child

The priorities in managing major paediatric trauma (Airway, Breathing, Circulation) are the same as in adults (see ➲ Major trauma: treatment principles, p. 330). Staff accustomed to treating adults may have difficulty with equipment sizes and drug doses. Establish/estimate the child's weight (see ➲ Weight estimation, p. 649). Call for help as soon as a seriously injured child arrives (or is expected) in the ED—senior ED doctor, ICU/PICU doctor, and surgeon. It is often very helpful to seek the help of a paediatrician to assist with vascular access and calculation of drug doses, particularly for preschool children.

Apply pressure to stop catastrophic external haemorrhage, and give tranexamic acid slow IVI (15mg/kg, up to max 1g) as soon as possible.

## Airway with cervical spine control

Clear and secure the airway (suction and adjuncts), and provide $O_2$ as required. If the airway is obstructed, use jaw thrust (not head tilt/chin lift), and call for expert help (senior ED/PICU/ICU) as intubation may be required. Ensure manual immobilization of the cervical spine is maintained whilst a patent airway is being obtained. When the airway is secure, consider the possibility of neck injury and the need for tape and sandbags until injury to the cervical spine has been excluded.

## Breathing

Quickly exclude and treat life-threatening chest injuries. Children are prone to swallow air, placing them at risk of massive gastric dilatation (can cause ↓ BP and subsequent aspiration)—consider an orogastric tube.

## Circulation with haemorrhage control

Hypotension is a late sign of hypovolaemia. Look for other evidence: tachycardia, tachypnoea, agitation, lethargy, and pale cold skin, with ↓ CRT (best elicited on the sternum). Get venous access (consider IO, as described in ➲ Intra-osseous infusion, pp. 656–7). Treat hypovolaemia by stopping haemorrhage (splinting fractures, applying pressure to wounds, prompt surgery) and giving IV blood. The approach to treat haemorrhage in trauma has changed in recent years—instead of giving large amounts of IV crystalloid, it is better to replace blood with blood. If blood products are not immediately available, give warmed 0.9% saline 10mL/kg IV.

If the child remains shocked, give 5mL/kg boluses of warmed packed red cells and FFP, aiming for a red cells:FFP ratio of 1:1.

Request a major haemorrhage pack (packed red cells, FFP, and platelets) and transfuse as required, monitoring Hb (aim no higher than 120g/L).

After blood products 40mL/kg, give platelets 10–15mL/kg IV—aim to keep the platelet count >100 × $10^9$/L.

After blood products 40mL/kg, give calcium chloride 0.1mL/kg IV—aim to keep ionized $Ca^{2+}$ >1mmol/L.

Discuss the need for cryoprecipitate (10mL/kg) and activated factor VII with the haematologist.

### Disability

Make a rapid assessment of the child's neurological status, using the 'AVPU' system (see ➔ Head injury: triage and monitoring, p. 364).

### Exposure

Early complete inspection is mandatory, but subsequently cover the child as much as possible in order to ↓ anxiety and prevent excessive heat loss.

### Analgesia

(See ➔ Analgesia in specific situations, pp. 290–1.)

Analgesia is often forgotten or not considered early enough, even with major injuries. Prompt and adequate analgesia given to injured children will gain their confidence, enhancing assessment and treatment. Give IV analgesia titrated according to response. Do not use IM or SC analgesia.

In *severe pain*, give morphine IV:

- Up to 100mcg/kg over 5min if 6–12 months.
- Up to 200mcg/kg over 5min if >12 months.

Certain fractures are amenable to LA nerve block techniques (eg femoral nerve block for femoral shaft fractures—see ➔ Femoral nerve block, p. 313). Nasal diamorphine (see ➔ Analgesia in specific situations, pp. 290–1) and Entonox® (see ➔ Analgesics: Entonox® and ketamine, p. 287) may also be useful for analgesia before IV access is available.

### Further history

After completing the primary survey and initial resuscitation, gain a more detailed history of how the injury occurred, together with the personal history, including:

- Allergies.
- Medication.
- Past medical history (and immunizations).
- Last mealtime.
- Events and environment relating to the injury.

### Imaging

A whole body ('pan') CT scan is the quickest way to determine the nature and extent of major injuries, but this needs to be balanced against the risk of a relatively large dose of radiation in a young person. Therefore, the team leader will aim to target CT to minimize radiation exposure. X-rays still have a role to play in some situations (see https://rcr.ac.uk).

### Parents

Remember the parents' needs—allocate a member of staff to this task (see ➔ Interacting with parents, p. 648). Children who have suffered a traumatic event are at risk of developing post-traumatic stress disorder—inform the parents or guardians about this. Briefly describe possible symptoms (sleep disturbance, nightmares, difficulty concentrating, and irritability). Suggest to the parents/guardians that they contact the child's GP if symptoms persist beyond 1 month (see NICE guideline NG116, published 2018, available at: https://www.nice.org.uk).

# Assessing head injuries in children

The principles of head injury management in children are the same as in adults (see ➲ Head injury: imaging, pp. 370; ➲ Management of serious head injury, pp. 372–3; ➲ Minor head injury, pp. 374–5), but there are some important differences (including the assessment of conscious level in small children).

## Background

Of those children who die from trauma, most succumb to head injuries. Anatomical differences are relevant. In infants, unfused sutures allow the intracranial volume to ↑ with intracranial haemorrhage, causing relatively large bleeds and even shock. Similarly, scalp wounds in infants and young children may bleed profusely and can result in significant hypovolaemia.

## Causes of head injury

Most head injuries in children are due to falls, but few of these cause serious injury. Severe head injury is often the result of a child running out in front of a vehicle. Some deaths are caused by NAI (see ➲ Head injuries, p. 760), especially in babies who have been shaken violently, dropped, or thrown.

## Assessment of a head-injured child

### *History*

Assessment of children may prove to be difficult. An isolated episode of vomiting after minor head injury is a frequent occurrence.

Record details of the injurious event, the time it occurred, and the condition of the child before and after the injury. Ascertain if the child was previously well. In particular, elicit any history of fits or bleeding disorder. An infection can render a child prone to falls and also cause subsequent symptoms—a small child who vomits after a fall may be suffering from otitis media, rather than the effects of a head injury.

Determine the condition of the child immediately after injury—if he cried at once, he did not lose consciousness. Record if he was unconscious, confused, or drowsy (and for how long), and whether he vomited or was unsteady or dizzy. Ask about headache. Remember to take into account the fact that a child might normally be asleep at the time he is examined.

### *Examination*

To assess level of consciousness, use the standard GCS (see ➲ Glasgow coma score (adults), p. 369) for children aged ≥4y.

Do not use the standard adult GCS in children aged <4y—instead use the adapted scale (see ➲ Glasgow coma score (children), p. 737).

Exclude hypoglycaemia. Note whether the child looks well and is behaving normally. Measure pupil size and check reactivity. Examine the head for signs of injury, but also look for injuries elsewhere, particularly the neck. Check T°, and consider coexisting illness such as ear, throat, or urinary infections, or occasionally meningitis.

## Glasgow coma score (children)

The 'Eye' and 'Motor' components of the GCS are similar as for adults (see ➲ Glasgow coma score (adults), p. 369), but a modified 'Verbal' score is used in small children. The paediatric version of the GCS is shown in Table 15.12 (see https://www.nice.org.uk). Assessment of the best verbal response is likely to require assistance from the parent/guardian/carer.

**Table 15.12** Paediatric version of the Glasgow coma score

| **Best eye response** | **Score** |
|---|---|
| Eyes opening spontaneously | 4 |
| Eyes opening to verbal command | 3 |
| Eyes opening to pain | 2 |
| No eye opening | 1 |
| **Best verbal response** | **Score** |
| Alert, babbles, coos, words, or sentences to usual ability | 5 |
| Less than usual ability and/or spontaneous irritable cry | 4 |
| Cries inappropriately | 3 |
| Occasionally whimpers and/or moans | 2 |
| No vocal response | 1 |
| **Best motor response** | **Score** |
| Obeys commands or has normal spontaneous movements | 6 |
| Localizes to painful stimuli or withdraws to touch | 5 |
| Withdrawal to painful stimuli | 4 |
| Abnormal flexion to pain (decorticate) | 3 |
| Abnormal extension to pain (decerebrate) | 2 |
| No motor response to pain | 1 |
| **Total** | **3–15** |

In pre-verbal or intubated patients, the 'best grimace response' may be used in place of the 'best verbal response', as shown in Table 15.13.

**Table 15.13** 'Best grimace response'

| | **Score** |
|---|---|
| Spontaneous normal facial/oro-motor activity | 5 |
| Less than usual spontaneous ability and/or only responds to touch | 4 |
| Vigorous grimace to pain | 3 |
| Mild grimace to pain | 2 |
| No response to pain | 1 |

# Managing head injuries in children

## Investigation

When faced with a child with severe injuries, summon senior help and follow standard resuscitation guidelines (see ➔ Management of serious head injuries, pp. 372–3). If there is any suspicion of NAI, involve the paediatrician at an early stage (see ➔ Management of child abuse, pp. 762–3).

*Indications for immediate CT scan*

(See https://www.nice.org.uk)

- Suspicion of NAI.
- Post-traumatic seizure, with no history of epilepsy.
- GCS <14 on initial assessment or paediatric GCS <15 in infants <1yr.
- GCS <15 at 2hr after the injury.
- Suspected open or depressed skull fracture or tense fontanelle.
- Clinical evidence of base of skull fracture.
- Focal neurological signs.
- For children <1y: bruise, swelling, or laceration >5cm on the head.

*If no indication for immediate CT, assess for risk factors*

- Witnessed loss of consciousness >5min.
- Abnormal drowsiness.
- ≥3 discrete episodes of vomiting.
- Dangerous mechanism (high-speed road traffic collision, fall >3m, high-speed injury from object).
- Amnesia (antegrade or retrograde) lasting >5min (hard to assess in children <5y).

*Action after assessing risk factors*

- If >1 risk factor is present, obtain a CT scan.
- If one factor is present, observe for ≥4hr post-head injury—obtain a CT scan if, during observation, the child drops GCS to <15 and has further vomiting or further episodes of abnormal drowsiness.
- If no risk factor is present, no imaging is required, unless the child is taking an anticoagulant, in which case obtain a CT scan within 8hr of injury.

## Management

Discuss children with abnormal CT scans with the neurosurgical team and treat accordingly.

Admit and observe children with continuing symptoms or signs, or an abnormal CT. When contemplating discharge, ensure adequate supervision from a responsible adult is available. Provide the parents with a verbal explanation and a written advice sheet (see ➔ Discharging patients, p. 375) (see also https://www.sign.ac.uk or https://www.nice.org.uk).

*Transient cortical blindness after head injury*

Occasionally, children present with blindness immediately or soon after an apparently minor head injury. The mechanism is unclear, but in most cases, blindness resolves spontaneously within a few hours. In the meantime, arrange a CT scan to exclude intracranial haematoma.

# Spinal injury in children

## Background

Cervical spine injury is relatively uncommon in children, but keep the spine immobilized until history, examination ± imaging exclude injury. Injuries in children tend to involve upper (C1–3 level), rather than lower, cervical spine. Rotatory subluxation may cause significant cervical spine injury without fracture—the clue is the combination of injury, neck pain, and torticollis. Interpretation of cervical spine X-rays in younger children is frequently complicated by pseudo-subluxation of C2 on C3 and of C3 on C4.

## Imaging the cervical spine in children

In a child with a head injury, obtain an urgent cervical spine CT scan if any of the following criteria is present (🔊 https://www.nice.org.uk):

- GCS <13/15 on initial assessment.
- The child has been intubated.
- Focal peripheral neurological signs.
- Paraesthesiae in the arms or legs.
- A definitive diagnosis is required urgently (eg prior to surgery).
- Multiple injuries affecting >1 body region.
- Strong suspicion of injury despite normal X-rays.
- X-rays show a significant bony injury.
- X-rays are technically difficult or inadequate.

If there is neck pain/tenderness, but no indication for CT, get X-rays if there is a dangerous mechanism (eg high fall >1m or five stairs, high-speed crash, rollover, ejection). Perform an assessment of the spine if safe to do so and the patient has one 'low-risk' factor (also applies to adults):

- A crash involving a 'simple' rear-end collision.
- Comfortable in a sitting position in the ED.
- Ambulatory at any time since injury.
- There is no midline cervical tenderness.
- There is a delayed onset of pain.

On assessment of the spine, if the patient can actively rotate the neck 45° right and left, no imaging is needed; if unable to do this, obtain X-rays.

### *Spinal cord injury without radiological abnormality (SCIWORA)*

The paediatric spine is inherently more elastic, so momentary intersegmental displacement may endanger the cord without disrupting bones or ligaments. This can result in spinal cord injury without radiological abnormality. Usually there are objective signs of injury, but these can be delayed. Therefore, if children present with transient neurological symptoms after neck injury, ensure careful assessment.

# Considerations in paediatric trauma

## Chest injury

Children have little respiratory reserve and can desaturate quickly. Significant thoracic visceral injuries may occur without rib fractures. There is a relatively high incidence of pulmonary contusion. In children with major trauma, obtain a CT scan; otherwise, if there is an isolated chest injury, consider obtaining a CXR (and then a CT scan only if that is abnormal).

If a chest drain is needed to treat a pneumothorax or haemothorax, use a size appropriate for the size of the child (as indicated by chart/tape).

## Abdominal and pelvic injury

Check for hypovolaemia. Abdominal palpation cannot yield useful information until the child's co-operation and confidence are gained. Restrict any PR and PV examinations to the senior surgeon. Consider a CT scan if there is abdominal injury with hypovolaemia, abdominal bruising, tenderness or distension, or bleeding PR or via the NG tube. FAST scanning is not of proven benefit in paediatric abdominal injury, but formal USS may help (eg to help exclude an isolated splenic injury).

Gastric tubes can help to treat air swallowing and gastric dilatation prevalent in injured children. Insert an appropriately sized urinary catheter if urine cannot be passed spontaneously or if accurate output measurement is required (eg after severe burns).

## Burns

Burns and smoke inhalation from house fires still cause death in many children each year. Even more frequently, children present with scalds from hot or boiling liquids. Most of these result from simple incidents in the home—ensure that treatment includes injury prevention advice for parents (see ➲ Injury prevention, p. 733). Remember that some (occasionally characteristic) burns may reflect NAI.

Assessment and treatment of the burnt child follow similar lines to those in adults; urgent priorities include securing the airway (with an uncut ET tube) and adequate analgesia (see ➲ Major trauma: treatment principles, p. 330).

IV fluid requirements in major burns depend upon the extent of the burn (use Lund–Browder charts for the appropriate age of the child—see Fig. 8.25) and clinical response (see ➲ Burns: assessment, p. 398).

## Drowning and submersion incidents

Children continue to die from drowning each year despite improved swimming education. Their high surface area to body weight ratio makes them prone to hypothermia. Cardiac arrest after immersion warrants prolonged resuscitation (see ➲ Drowning and near drowning, pp. 268–9). Presume cervical spine injury and immobilize the neck. Prolonged submersion (>8min), no respiratory effort after 40min of CPR, persistent coma, persistent pH <7.0, and persistent $PaO_2$ <8kPa imply a poor prognosis. Hypothermia favours a better prognosis. Of those who survive after hospital CPR, 70% do make a complete recovery.

# Wounds in children

Some children may allow wounds to be explored, cleaned, and sutured under LA, providing they are given an appropriate explanation (sometimes it is worth demonstrating on a teddy first) and a parent is allowed to stay with them. Injection of LA is least painful if a fine needle is employed and the LA is warmed, buffered, and injected slowly. Some children, however, do not tolerate LA. Whilst some superficial wounds may be cleaned and closed (Steri-Strips™ or tissue glue) without anaesthesia, often sedation or GA is needed. Anaesthesia is important in order to allow adequate exploration and cleaning of the wound and to ↓ the risks of infection and tattooing from embedded dirt. Never allow a lack of co-operation to compromise treatment—this is particularly important with facial wounds where wound closure under GA may be needed to provide the best cosmetic result.

## Ketamine

Ketamine can be used in the ED as an alternative to GA and provides excellent analgesia for undertaking minor procedures in children (see Analgesics: Entonox® and ketamine, p. 287) (see also the RCEM guideline 2016, available at: https://www.rcem.ac.uk). Ketamine should only be used by clinicians experienced in its use and capable of managing any airway complications.

Ketamine is *contraindicated* if:

- There is a high risk of laryngospasm (active respiratory infection, active asthma, age <12 months).
- There are severe psychological problems (cognitive or motor delay or severe behavioural problems).
- Cardiovascular disease (congenital heart disease, cardiomyopathy, ↑ BP).
- Significant head injury or neurological disease, porphyria, and hyperthyroidism.

# Paediatric fractures and dislocations

Many paediatric fractures are similar to those in adults and prone to similar complications. Bones in children differ from those in adults in two important respects—they have epiphyses and are softer (hence fractures are more common than significant ligament injuries). Certain types of paediatric fractures reflect these differences:

*Greenstick fracture* An incomplete fracture in which one cortical surface of a bone breaks, whilst the other side bends.

*Torus ('buckle') fracture* Another form of incomplete fracture characterized by buckling of the cortex.

*Plastic deformation ('bowing deformation')* Traumatic bending of the long bone shaft without a visible fracture occasionally occurs in young children.

## Epiphyseal injuries

Injuries to the traction epiphyses are avulsion injuries (eg peroneus brevis insertion into the base of the fifth MT).

Injuries to the pressure epiphyses at the end of long bones adjacent to the articular surface have been classified into five types—the Salter–Harris classification (see Fig. 15.23):

- *Type I*: the epiphysis separates or slips on the metaphysis.
- *Type II*: a small piece of metaphysis separates with the epiphysis (most common type—see Fig. 15.24).
- *Type III*: a vertical fracture through the epiphysis joins that through the epiphyseal plate (see Fig. 15.25).
- *Type IV*: a fracture passes from the articular surface through the epiphyseal plate into the metaphysis (see Fig. 15.26).
- *Type V*: a crush injury to the epiphyseal plate (X-rays may be normal).

Note that Salter–Harris types I and V may not be apparent on the initial X-ray. Undisplaced type I fractures often affect the distal tibia and fibula and may present with circumferential tenderness around the growth plate. Treat with POP and immobilization according to clinical findings.

*Epiphyseal growth plate injury*

A concern specific to any epiphyseal injury is that premature fusion of a growth plate may result, with resultant limb shortening and deformity. The risk correlates to some extent with the mechanism of injury and amount of force involved. The different Salter–Harris fractures carry a different level of risk of long-term growth plate problems. The risk is low for types I and II (particularly if undisplaced), moderate for type III, and highest for types IV and V. Problems are usually averted if Salter–Harris type III and IV injuries are accurately reduced and held (eg by internal fixation). Type V fractures are notoriously difficult to diagnose and often complicated by premature fusion—fortunately, they are relatively rare.

## Dislocations

Dislocated joints are relatively unusual in children. Most commonly involved are the patella (see ➲ Paediatric knee injuries, p. 753) or the radial head ('pulled elbow'—see ➲ Elbow injuries in children, p. 750). Similarly, due to the relative strengths of bone and ligament, injuries to ligaments are much less common in children than in adults.

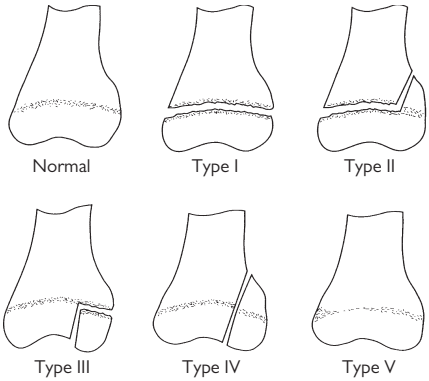



**Fig. 15.23** Salter–Harris classification of epiphyseal injuries.

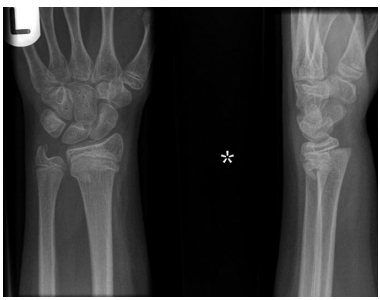


**Fig. 15.24** Salter–Harris II fracture of the distal radius in an 11y old.

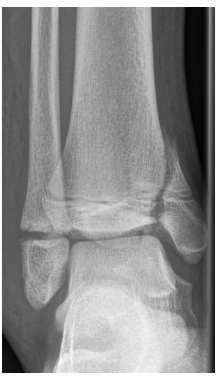


**Fig. 15.26** Salter–Harris IV fracture of the distal tibia in a 15y old.

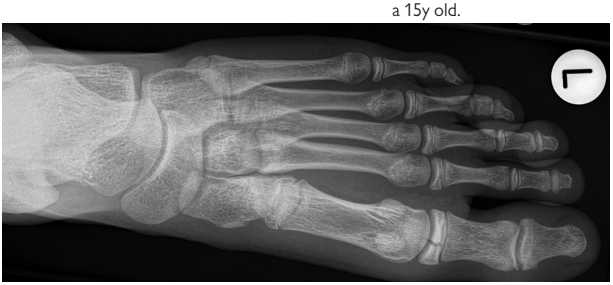


**Fig. 15.25** Salter–Harris III fracture of the proximal phalanx of the big toe in a 14y old.

# Approach to limb injuries in children

Limb injuries are very common in children. Whilst most of the points outlined in the general approach to trauma in adults may be successfully applied, certain modifications may be required.

## History

Carefully elicit the mechanism of injury. The history may be confused or not forthcoming—try to establish a rapport with the child (and parents) nevertheless, in order to gain the child's confidence for the examination.

## Examination

Search for evidence of a fracture (swelling, deformity, bony tenderness) and any associated neurovascular injury. Remember the adage that the most easily missed fracture is the second fracture—examine also for additional injuries to adjacent bones and joints.

## Is an X-ray required?

If in doubt, obtain an X-ray. The ease with which children's bones fracture and the difficulties with history and examination mean that it is sensible to adopt a low threshold for requesting X-rays. Ensure that two views at right angles are taken (eg AP and lateral), including associated joints.

## Interpreting X-rays

Many fractures are subtle and easily missed. To minimize the chance of this occurring, visually trace around the cortex of each bone, looking for any irregularities. Interpretation of paediatric X-rays is complicated by the presence of various ossification centres and accessory ossicles. Both are commonly mistaken for fractures (eg the olecranon epiphysis, the os trigonum, and the bipartite patella). Ossification centres appear and fuse in a relatively predictable fashion, although the rate at which this occurs varies slightly from child to child (see Table 15.14). Knowledge of this process, combined with experience of seeing many paediatric X-rays, greatly assists interpretation. If in doubt about an X-ray, obtain a second opinion (there is no justification for X-raying the uninjured side to see what 'normal' is). As an additional safeguard, most EDs now operate a policy of all X-rays being reported by a radiologist or reporting radiographer within 24hr.

## Treatment

Give prompt, appropriate analgesia (see ➲ Analgesia, p. 735). Follow the treatment suggested for specific fractures (see ➲ p. 747). Many undisplaced fractures will unite satisfactorily with a period of immobilization in POP (eg fractured distal radius), collar and cuff (eg fractured radial head), or broad arm sling (eg fractured clavicle). Minor angulation at the fracture site can be accepted, particularly in young children. Often, however, angulated fractures require MUA.

## Open fractures and dislocations

Give analgesia and IV antibiotics (eg cefuroxime 25mg/kg slow IV bolus), and ensure tetanus cover. Take a digital photograph of the wound and keep it covered to minimize the risk of infection. Apply a dressing, splint the injured limb, and refer the patient to the orthopaedic surgeon.

**Table 15.14** Ossification centres

| Centre | First appears | Fuses |
|---|---|---|
| Humeral head | 0–6 months | 18–21y |
| Capitulum | 3–6 months | 14–16y |
| Medial epicondyle | 4–7y | 18–21y |
| Lateral epicondyle | 9–13y | 14–16y |
| Trochlea | 9–10y | 14–16y |
| Radial head | 4–5y | 14–17y |
| Distal radius | 6–12 months | 17–19y |
| Olecranon | 9–11y | 13–16y |
| Distal ulna | 4–5y | 16–18y |
| Capitate | Birth to 3 months | – |
| Hamate | Birth to 4 months | – |
| Triquetral | 1–3y | – |
| Lunate | 2–4y | – |
| Trapezium | 2–4y | – |
| Trapezoid | 3–5y | – |
| Scaphoid | 3–5y | – |
| Pisiform | 9–12y | – |
| First MC base | 1–3y | 14–17y |
| Femoral head | Birth to 6 months | 15–19y |
| Greater trochanter | 3–4y | 17–19y |
| Lesser trochanter | 11–14y | 15–18y |
| Distal femur | Birth | 17–20y |
| Patella | 2–6y | 4–8y |
| Proximal tibia | Birth | 15–18y |
| Distal tibia | Birth to 6 months | 14–17y |
| Proximal fibula | 2–4y | 16–19y |
| Distal fibula | Birth to 1y | 14–17y |
| Posterior calcaneum | 5–8y | 13–16y |
| Central calcaneum | Birth | 13–16y |
| Talus | Birth | – |
| Navicular | 2–3y | – |
| Cuneiform bones | 1–3y | – |

These dates are subject to individual variation. In general, epiphyses in girls fuse before those in boys.

# Normal X-rays in children

It is useful to have an idea of the normal appearance of X-rays in children. Some examples are shown in Figs. 15.27–15.30.

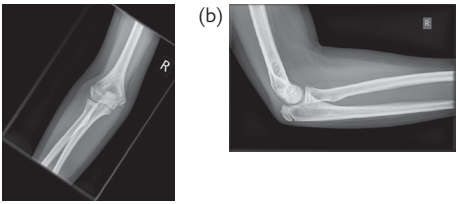



**Fig. 15.27** (a), (b) Normal elbow in a 10y old.

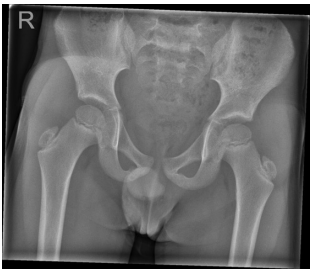



**Fig. 15.28** Normal pelvis X-ray in a 6y old boy.

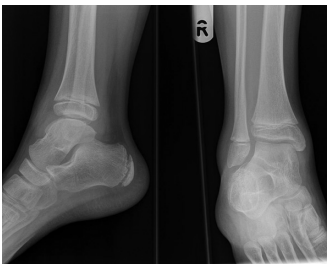



**Fig. 15.29** Normal ankle X-rays in a 10y old.

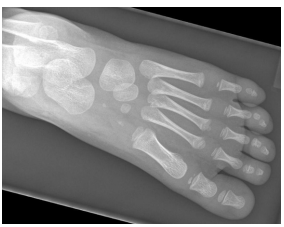


**Fig. 15.30** Normal foot in a 2y old.

# Shoulder and humeral shaft fractures

## Clavicle fracture

This is a common injury in children and adults alike. Most clavicle fractures with no or only minor angulation/displacement heal satisfactorily with conservative management, comprising oral analgesia and rest in a broad arm sling. Follow-up is not usually necessary—give the child/parents an advice leaflet with a contact telephone number to call if there are questions/problems (see ➲ Fracture clinic and alternatives, pp. 436–7). The advice leaflet should include the following advice:

- Use the sling for 3 weeks and painkillers as required. Extra pillows to support the arm may help in the first few days.
- Start exercises of the hand, wrist, and elbow as early as possible.
- Expect a lump (callus) to form at the fracture site.
- Avoid rough play and contact sports for 6 weeks.
- To seek medical attention if the child becomes suddenly short of breath or if there is a problem involving the skin over the fracture.

Sometimes a clavicle fracture is strongly suspected clinically, but not apparent on X-ray—treat as for an undisplaced fracture.

Treat children who have comminuted, very angulated, or displaced fractures (see Fig. 15.31) similarly, but check carefully for neurovascular damage and the X-ray for associated rib fractures and pneumothorax. Arrange fracture clinic follow-up.

## Acromio-clavicular joint injuries

These become more common in older children. Treat with analgesia, rest, sling, and physiotherapy/follow-up as for adults (see ➲ Acromio-clavicular (AC) joint injuries, p. 471).

## Shoulder injuries

Shoulder dislocations are relatively rare in children. Salter–Harris type I and II epiphyseal fractures may occur in the proximal humerus—refer to the orthopaedic team if significant displacement or >20° angulation. Otherwise, give analgesia, collar and cuff, and fracture clinic follow-up.

## Humeral shaft fracture

Check particularly for injury to the radial nerve which runs close to the humeral shaft in the spiral groove. Remember to consider the possibility of NAI, especially if the patient is <3y old or the fracture is spiral. Treat as for adults (see ➲ Shaft of humerus fracture, p. 465) with analgesia, backslab POP, and sling support, with fracture clinic follow-up.

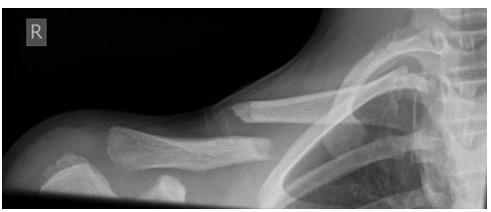



**Fig. 15.31** A 13y old with a displaced clavicle fracture.

# Supracondylar humeral fracture

This follows a fall on an outstretched hand. Swelling may be considerable. Check for associated neurovascular deficit (particularly brachial artery and median and radial nerves). 25% of supracondylar fractures are undisplaced and may not be obvious on X-ray, although a joint effusion will be seen. Most fractures are displaced, angulated, or rotated. The extent of angulation (both in sagittal and coronal planes) is easy to underestimate. Viewed from laterally, the capitulum normally makes an angle of 45° with the humeral shaft (see Fig. 15.32). The anterior humeral line (drawn along the front of the humeral shaft on the lateral view) normally passes through the middle of the ossification centre of the capitulum in the distal humerus. Also, the normal carrying angle (seen in AP view) is 10°. Record the radial pulse frequently, and consider compartment syndrome.

## Treatment

Provide analgesia (eg nasal diamorphine), and refer for manipulation under GA if:

- Neurovascular deficit: operation is urgent if circulation is compromised.
- >50% displacement (see Fig. 15.33).
- >20° angulation of the distal part posteriorly (see Fig. 15.34).
- >10° medial or lateral angulation.

If there is no indication for manipulation under GA, refer for admission and observation if there is much swelling. If no significant angulation, displacement, or swelling, discharge with analgesia, a collar and cuff under a body bandage (elbow at 90°, with confirmed radial pulse present), and fracture clinic follow-up. Consider using a padded backslab POP if significant pain is present.

### *Complications*

Malunion with persistent deformity, stiffness (including myositis ossificans), neurovascular deficit (eg Volkmann's contracture).

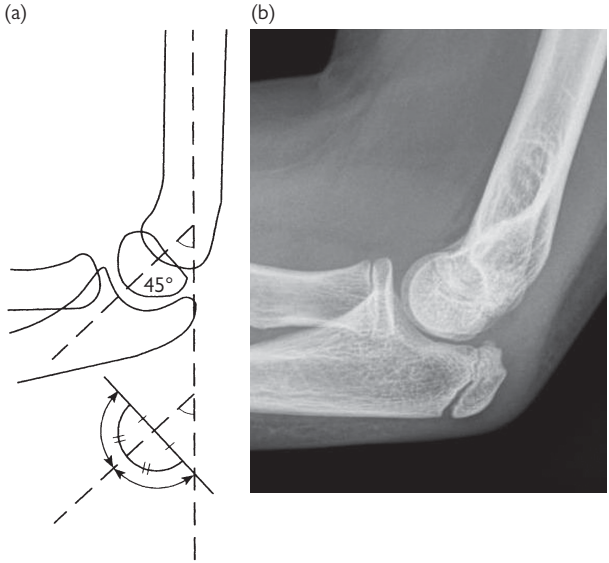



**Fig. 15.32** Normal lateral view—the capitulum makes an angle of 45° with the humeral shaft.

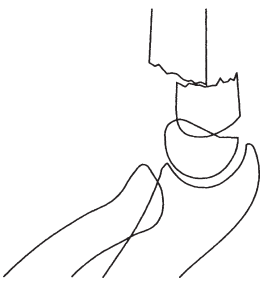


**Fig. 15.33** Supracondylar fracture with >20° angulation and ~50% displacement.

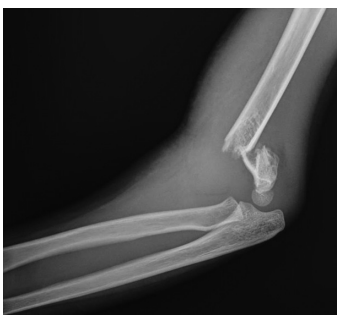


**Fig. 15.34** A 4y old with a supracondylar fracture with >20° angulation and >100% displacement.

# Elbow injuries in children

## Lateral epicondylar epiphyseal injury

Salter–Harris type II injury may follow a fall on an outstretched hand. The elbow is swollen, with ↓ movement and maximum tenderness on the lateral aspect. X-rays demonstrate the fracture, which may be displaced by the pull of the forearm extensors, requiring surgical reduction. Treat undisplaced fractures with a long arm backslab POP, collar and cuff at 90°, analgesia, and fracture clinic follow-up.

## Medial epicondylar epiphyseal injury

Maximal tenderness is apparent on the medial side of the elbow. Check carefully for ulnar nerve damage. Refer immediately if the ulnar nerve is involved or if the fracture is displaced. Treat undisplaced fractures with analgesia, collar and cuff at 90° under clothes (confirm the radial pulse is present), and fracture clinic follow-up.

## Radial head/neck fracture

The radiocapitellar line is drawn down the axis of the proximal radius on the lateral view of the elbow and should bisect the capitellum. Failure to do so suggests an occult radial neck fracture or radial head dislocation. Most of these fractures can be managed satisfactorily with analgesia, collar and cuff, advice leaflet, and no need for follow-up (see ➲ Elbow injuries, pp. 462–3). Refer to the fracture clinic if there is significant angulation.

## Elbow injury without obvious fracture

Treat elbow injuries where there is clinical suspicion of fracture, but none seen on X-ray, along the same lines as for an undisplaced fracture (analgesia, collar and cuff, advice leaflet, no follow-up). This includes children who have ↓ range of movement and whose X-rays show an elbow effusion ('fat pad sign') (see ➲ Elbow injuries, pp. 462–3).

## Subluxation of the radial head ('pulled elbow')

A direct pull on the arm of a child aged 1–5y may result in the radial head being pulled out of the annular ligament ('nursemaid's elbow'). The child then refuses to use the arm. If there is a characteristic history, there is no need to X-ray. The traditional reduction technique involves flexing the elbow to 90°, then supinating the elbow fully. However, manipulating the elbow into full pronation may give a better reduction rate (https://www.bestbets.org.uk). A click is sometimes felt or heard during reduction. If full pronation fails, try full supination and leave for 10min. Allow the child to play and watch—he will usually use the arm again soon. If he does not, obtain X-rays and senior help. Repeat manipulation can be done once, but if that does not lead to a rapid improvement in function, then place the arm in a sling; give analgesia, and arrange review in 1–2 days. The elbow may reduce spontaneously or may need further manipulation. Rarely, repeated manipulation is unsuccessful until sedation is given. After successful manipulation, advise the parents to avoid pulling the arm forcefully. A pulled elbow may recur up to about age 5y if the arm is pulled, but after that the child should have no long-term problems with the elbow.

# Forearm and wrist injuries

## Radius/ulna shaft fractures

Radius and ulna shaft fractures often cause significant displacement or angulation—provide IV analgesia (or nasal diamorphine), immobilize in a broad arm sling, obtain X-rays, and refer for manipulation under GA. Never accept an isolated forearm shaft fracture without X-rays demonstrating the entire radius and ulna; otherwise, a Monteggia or Galeazzi fracture–dislocation may be missed (see ➲ Forearm fractures and related injury, pp. 460–1).

## Distal radial fracture (including Salter–Harris type II injuries)

A common fracture in all ages of children (and adults) after a fall on an outstretched hand. The fracture results in localized tenderness and variable swelling. Check carefully for a second injury (eg involving the thumb or scaphoid). X-rays will demonstrate the nature of the fracture.

### *Salter–Harris type II fractures*

Often have displacement of the distal radial epiphysis (eg see Fig. 15.24), in which case, refer for MUA under GA.

### *Moderate displacement or slight angulation*

May be accepted (particularly in younger children): if in doubt, treat in a backslab POP and arrange fracture clinic follow-up.

### *Minimally displaced or undisplaced greenstick, buckle, or torus fractures*

Commonly occur just proximal to the distal radial epiphysis. Treat with analgesia, elevation, a wrist splint, and written advice, with no follow-up unless problems arise (see ➲ Fracture clinic and alternatives, pp. 436–7). Treat children who present with discrete tenderness over the distal radial growth plate, but no fracture apparent on X-ray, identically to those with a radiologically proven fracture—presume a growth plate injury (sometimes a subperiosteal haematoma can be seen on USS). Beware osteomyelitis, which can cause tenderness over the distal radius and be mistaken for trauma. Parents and children report better functioning and fewer days off school with the use of removable splints, compared with POPs, for minor wrist fractures. Advise that the splint should be retained until pain wears off (usually <3 weeks) and that follow-up is not required if pain settles as expected.

## Scaphoid fracture

Despite being uncommon, particularly in younger children, seek clinical evidence of scaphoid fracture in any child with wrist/forearm injury and obtain scaphoid views if appropriate (see ➲ Scaphoid fracture, pp. 450–1). Treat radiologically evident and suspected fractures as for adults, as described in ➲ Scaphoid fracture, pp. 450–1.

## Metacarpal and phalangeal injuries

Treat these injuries along similar lines to those described for adults (see ➲ Hand fractures and dislocations, pp. 444–5). Remember, however, that children may not tolerate manipulation under LA—anaesthetic help may be required.

# Hip and femoral fractures in children

## Hip fracture

Children rarely sustain neck of femur fractures similar to those seen in adults. In the pre-adolescent child, trauma may precipitate a slipped upper femoral epiphysis (see ➲ The painful hip, pp. 728–9). Younger children who have been subjected to considerable violence may suffer a Salter–Harris type I injury to the proximal femoral epiphysis—carefully exclude other injuries and refer to the orthopaedic surgeon.

## Femoral shaft fracture

May be spiral (the majority) or transverse, depending upon the mechanism of injury (see Figs. 15.35 and 15.36). Considerable energy is required to break a femur—check for other injuries. Resuscitate as necessary with IV fluids and provide nasal diamorphine (see ➲ Nasal diamorphine for analgesia in children, p. 291) or IV opioid analgesia (see ➲ Analgesia in specific situations, pp. 290–1). Perform a femoral nerve block (as described in ➲ Femoral nerve block, p. 313) to provide additional analgesia, using 0.2mL/kg of 0.5% plain bupivacaine (1mg/kg). Allow 20min for this to work, then apply skin traction. Gallows traction may be used on infants and children <2y but is best erected on the ward. A true spiral fracture in a non-ambulatory child suggests child abuse—swelling is often not dramatic.

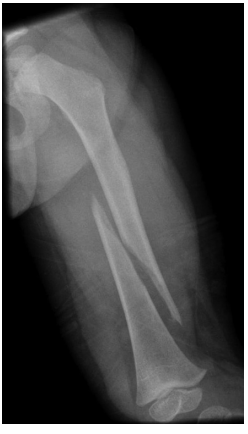


**Fig. 15.35** Femoral shaft fracture in a 3y old.

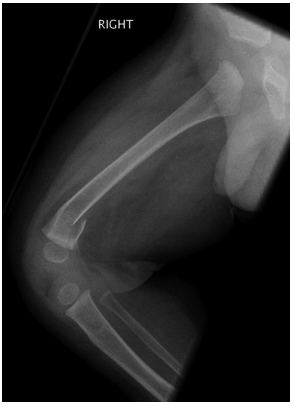



**Fig. 15.36** A 9 month old with a transverse fracture of the distal femur.

# Paediatric knee injuries

## Knee injuries

Knee ligament injuries are rare in children, compared with adults—suspect a fracture or epiphyseal injury instead. This is a reflection of the relative strengths of the ligament and bone in the child. So, for example, an injury which might cause anterior cruciate ligament rupture in the adult will often produce avulsion of its tibial attachment in the child. This tibial plateau fracture will produce a haemarthrosis and will be apparent on the lateral X-ray. Provide analgesia and refer to the orthopaedic surgeon.

## Patella fractures

Do not confuse a congenitally bipartite patella for a fracture. The small bony fragment in a bipartite patella lies superolaterally and has rounded edges.

### *Patellar sleeve fractures*

These are not uncommon in children and adolescents. These osteochondral fractures typically result from high-impact jumping activities or sports. Suspect clinically if there is local pain and tenderness and an inability to actively extend the knee. X-rays can be misleading as only a small bony fragment is avulsed, usually from the inferior pole; however, a large part of the articular surface is removed with it but is impossible to see on plain X-ray. Provide analgesia and splintage, and refer to the orthopaedic team for MRI to confirm the diagnosis ± ORIF.

## Patella dislocation

This is seen relatively frequently in children and is treated in a similar way to that in adults (see ➲ Dislocations of the patella and knee, p. 490). Examine the X-rays carefully, as associated osteochondral 'chip' fractures of the undersurface of the patella occur relatively frequently in children. Refer for fracture clinic follow-up and MRI to establish the extent of the injury.

# Tibial fractures in children

## Tibial shaft fracture

Treat most fractures as for adults—splintage, IV analgesia, and referral for elevation and admission. Compound fractures require IV antibiotics and wound surgery. Displaced or angulated fractures require MUA and POP; undisplaced fractures respond to treatment with above-knee non-weight-bearing POP and subsequent mobilization using crutches.

## Toddler's fracture

Minor trauma in 1–4y olds may result in characteristic spiral undisplaced distal tibial fractures (see Fig. 15.37). These may not be apparent on the initial X-rays—localized warmth and tenderness with a history of trauma may suggest the diagnosis in the otherwise wide differentials of the limping child (see ➲ The limping child, pp. 726–7). If a fracture is visible on initial X-rays, treat by rest in a POP and arrange fracture clinic follow-up. If the diagnosis is made without a visible fracture, treat in POP and review clinically and radiologically at 10 days—further X-rays may then demonstrate a long strip of new periosteal tibial bone formation. Continue to treat according to symptoms.

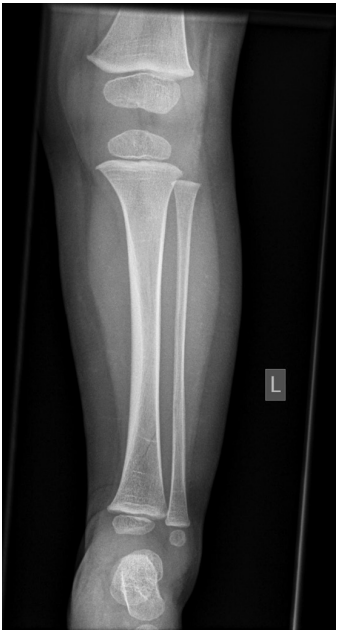



**Fig. 15.37** An 18 month old with an oblique ('toddler's') fracture of the distal tibia which is hard to see. Note the adjacent white horizontal (Harris) growth arrest lines of no relevance to this injury.

# Ankle and foot injuries in children

## Ankle injuries

Ankle ligament injuries are less common in children than in adults, partly reflecting the fact that ligaments are often stronger than the bone to which they attach. The presence of epiphyses can make it difficult to establish whether or not a fracture is present on X-rays. If there is no obvious fracture on X-ray, but there is much tenderness/swelling over the distal tibial or fibular epiphysis, treat it as a growth plate injury (undisplaced Salter–Harris type I fracture) with BKPOP, crutches, elevation, analgesia, and follow-up in the ED or fracture clinic. Some fractures (see, for example, Fig. 15.38) require admission for manipulation under GA.

## Foot injuries

A wide range of foot injuries occur in children. A common difficulty is distinguishing between the normal apophysis at the base of the fifth MT (see Fig. 15.39) and a fracture. The normal apophysis is typically longitudinally parallel to the fifth MT (as opposed to a fracture which is typically transverse or oblique—as shown in Fig. 9.7).

## Calcaneal injuries

(See ➲ Foot fractures and dislocations, pp. 504–5.)

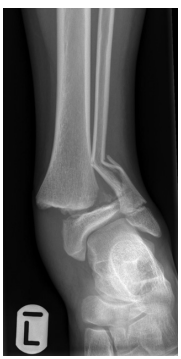


**Fig. 15.38** Ankle fracture in a 12y old.

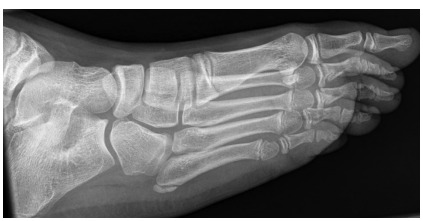


**Fig. 15.39** Normal foot in an 11y old.

# Child abuse

The boundaries of what defines acceptable behaviour and what constitutes child abuse are open to some debate and are certainly affected by historical and cultural factors. For example, corporal punishment, once considered normal and usual, is now unacceptable. The extremes of child abuse, however, are easily defined. There is ↑ evidence that adverse childhood experiences (including abuse) can have negative effects on individuals throughout the rest of their lives.

## Types of child abuse

- Physical abuse/NAI—including bruises, fractures, wounds, burns, poisoning, suffocation, FGM, fabricated or induced illness.
- Neglect.
- Emotional abuse.
- Child sexual abuse.

## Prevalence

It is impossible to be sure how common child abuse is. It is generally agreed that it is much more prevalent than was previously believed. 4% of children are brought to the attention of professional agencies for suspected abuse. It is estimated by the National Society for the Prevention of Cruelty to Children (NSPCC) that over 500,000 children are abused in the UK each year.

## Aetiology

Child abuse affects both boys and girls. The first-born child is more frequently affected. Disabled children are particularly vulnerable to abuse or neglect. Infants and young children are at most risk of serious injury or death, partly reflecting their physical vulnerability. The abuser is often a parent or cohabitant of a parent, more commonly ♂, and may have suffered abuse themselves as a child. Sometimes the child may be targeted because they are unwanted (eg 'she should have been a boy'). Whilst the abuser may be a young parent with unrealistic expectations and living in difficult socio-economic circumstances (unemployment, alcohol/drug abuse, poor living conditions), often they do not conform to this standard description. Child abuse affects all levels of society.

Clear links have been identified between domestic abuse and physical abuse of children. Children whose parents have mental health problems may be more vulnerable to abuse and neglect. The 'toxic trio' of domestic abuse, mental health issues, and substance misuse has been identified as comprising recurrent common factors in families where child abuse has occurred. The term 'developmental trauma' has been used to describe the impact of early, repeated abuse, neglect, separation, and adverse experiences within a child's important relationships (https://www.beaconhouse.org.uk).

### Child behaviour as indicators of child abuse

Consider the possibility of child abuse or neglect (current or past) if a child exhibits any of the following:

- Alcohol and substance misuse (including overdosing).
- Bullying/being bullied.
- Self-harm.
- Developmental delay.
- Eating disorder.
- Escalating or concerning behaviours (violence/aggression, inappropriate or harmful sexual behaviours).
- Features of neglect: failing to attend appointments ('was not brought'), failure to thrive, inappropriate clothing, poor appearance/hygiene.
- Missing episodes and/or exclusion from school.
- Poor or deteriorating parent/peer interactions and/or relationships.

### Role of the junior ED clinician

Managing the child and family where there is suspected child abuse is an extremely delicate skill, requiring considerable tact and experience. The role of the junior clinician is to consider the possibility of child abuse and to involve senior staff at an early stage—see relevant NICE guidance (https://www.nice.org.uk) updated in 2017.

The 2018 publication '*Working together to safeguard children*' outlines how protecting children is everyone's responsibility and should follow a child-centred approach.

### The suspicious history

Certain features should alert to the possibility of child abuse:

- Injuries inconsistent with the history given.
- Injuries inappropriate for developmental age, paying particular consideration to non-mobile babies (eg a baby aged <4 months 'rolled off a bed').
- Changing history of injury or vague history, lacking vivid details.
- Delay in seeking medical attention.
- Concerning parental attitudes (eg apparent lack of concern for child).
- Frequent ED attendances.
- Occasionally, children may verbally disclose abuse. It is paramount to document the voice of the child—capture the child's disclosure in their own words in 'inverted commas'.

### Child criminal exploitation and trafficking

Child criminal exploitation may involve individual groups or gangs manipulating, exploiting, or coercively controlling children into the supply and distribution of drugs from cities to rural locations using mobile phone lines (County lines). Children and young people may present to unscheduled health care settings due to being victims of extreme violence from gang members. County lines can be linked to modern slavery and the sexual exploitation of children.

Children and young people may be trafficked for a variety of reasons, including sexual exploitation, forced marriage, and domestic servitude. If suspected, refer to social care and the police to investigate.

# Presentation of child abuse: bruising

Physical child abuse is commonly referred to as NAI. Children may present with a variety of injuries, which may occur in isolation or in combination.

## Bruising

Children naturally sustain bruises during minor incidents as part of 'growing up'. Bruising over the knees and shins is a normal finding in children, particularly toddlers, who are also prone to sustaining injuries to their foreheads and chins as a result of falls. Older children frequently sustain bruises over the lateral aspect of their elbows and hips, during normal play and sport activities. Bruises in non-mobile babies, however, deserve particular attention and investigation.

As well as considering the possibility of NAI, remember that bruising may occur as part of an unusual pathological disease process (eg Henoch–Schönlein purpura, haemophilia, ITP, leukaemia, and other causes of thrombocytopenia). A Mongolian blue spot is an innocuous congenital finding on the lower back of some young children (especially non-Caucasians), which may be confused with bruising.

The following features warrant prompt consideration of NAI:

- Bruising in unusual sites (eg medial aspect of upper arms or thighs).
- Bruising in non-mobile babies.
- Multiple bruising of different ages (very difficult for the non-expert to judge) at less common sites.
- Uncommon injuries bilaterally.
- Finger 'imprinting' (eg grip complexes around upper limbs or slap marks).
- Imprints or marks from other objects (eg belt, stick).
- Human bite marks (probably adult if canines >3cm apart—ensure photographs next to a ruler are planned after admission).
- Petechiae on the face may reflect smothering and asphyxiation (it has been previously suggested that 2–10% of SIDS may have been smothered), but remember that petechiae also occur with forceful coughing or vomiting.

## Natural progression of bruises

Swelling and tenderness of bruising suggest a relatively recent origin, but this is not very reliable. Accurate assessment of the age of bruising according to its colour is not possible, except that a yellow bruise is almost certainly >18hr old. Oft-quoted natural temporal progression of colour changes of bruising allows only a guess at the age of a bruise—avoid being drawn on this issue, which may have considerable legal implications. Instead, record the findings as accurately as possible—describe the colour, size, and distribution of the bruising. Usually a child suspected of having suffered physical abuse will also be examined by a relevant expert such as a paediatrician and/or a forensic physician (previously called 'police surgeon').

# Child abuse: fractures

Fractures occur in a significant proportion of physically abused children—studies quote figures ranging from 11% to 55%, with ~80% of these fractures occurring in children aged <18 months.

Certain fractures are very common in children. Pay attention to the history of injury and whether or not it appears to be consistent with the fracture(s) sustained. Multiple fractures of different ages (especially if previously undiagnosed and/or not brought to medical attention) should arouse suspicion of NAI.

To help assess the approximate age of a bony injury, see Table 15.15, but bear in mind the fact that the times quoted are approximate and vary according to the age of the child.

**Table 15.15** Natural progression of fractures

| | |
|---|---|
| Presence of soft tissue swelling | 0–10 days |
| Periosteal new bone formation | 10–14 days |
| Loss of definition of the fracture line | 14–21 days |
| Callus formation | 14–42 days |
| Remodelling | ~1y |

## Fractures arousing particular suspicion

The following fracture patterns are particularly suggestive of NAI:

- Multiple fractures of different ages.
- Rib and spinal fractures.
- Fractures in infants who are not independently mobile.
- Long bone fractures in children <3y old.
- Epiphyseal separation and metaphyseal 'chip' fractures of the knee, wrist, elbow, and ankle. These Salter–Harris type I and II injuries are associated with traction, rotation, and shaking.

*Note that a few rare bone diseases may mimic NAI*

- Osteogenesis imperfecta (blue sclerae, dental abnormalities, and brittle bones—autosomal dominant).
- Pathological fractures (through multiple cystic bone lesions).
- Rickets (enlarged, cupped epiphyses, craniotabes, 'bow legs').
- Copper deficiency (eg Menkes' kinky hair syndrome).

# Child abuse: head injuries, wounds and burns

## Head injuries

Most head injuries result from unintentional incidents ('accidents'). In infants, they often result from the parent or carer dropping the child. The skull fractures caused by this tend to be single and linear and involve the parietal bone.

Consider NAI if the following occur:

- Retinal haemorrhages (characteristic, but not diagnostic of shaking—they may also rarely be seen in CO poisoning, for example). In the context of NAI, retinal haemorrhages are often associated with subdural haematomas.
- Occipital skull fracture.
- Multiple, wide, or comminuted fractures.
- Subdural haematoma in an infant or toddler.

## Wounds and burns

Children commonly sustain wounds and burns unintentionally. However, deliberately inflicted burns are found in a significant proportion of physically abused children.

The following suggest the possibility of NAI:

- Torn frenulum of upper lip (can also reflect a 'normal' toddler injury).
- Perineal wounds and burns (see ➲ Sexual abuse, p. 761).
- Small, deep, circular burns with raised edges suggest cigarette burns.
- Hand, lower limb, and buttock burns may follow forced immersion in bath water that is too hot. These burns tend to be of the 'stocking and glove' type, without higher splash burns. Parts of the buttocks may be spared where skin has been in contact with the bath, not the water.

## Fabricated or induced illness

Previously known as 'Munchausen syndrome by proxy', this describes the situation where a parent/carer may invent a history of illness in a child and fabricate physical signs to substantiate it. The history often involves one or more of the following: apnoeic episodes, fits, bowel disturbances, rashes, allergies, or fevers. Classically, the deceiver is the mother. The child may be made ill by administering drugs or poisons. If suspected, do not confront the deceiver, but take blood and urine samples for a toxicology screen and refer to the paediatric team.

Bear in mind that some parents may be naturally very anxious and may exaggerate symptoms, rather than deliberately fabricate them.

## Neglect

The neglected child may be dirty and unkempt, fail to thrive, and/or fall below the third centile for height and weight. Occasionally, nutritional deficiencies may be extreme (eg rickets). Developmental milestones are often delayed (and may even regress).

## Emotional abuse

Ongoing emotional maltreatment of a child is sometimes referred to as psychological abuse. It can cause significant harm to the child development. It can involve deliberately humiliating, isolating, or ignoring a child.

There will likely be an element of emotional abuse as part of other forms of abuse, which may be manifest in various ways: personality/behavioural changes, sleep disturbance, soiling, and nocturnal enuresis.

Note the apparent attitudes of the parents/carers towards their child (eg critical and hostile or remote and unconcerned) and the child's attitude to the parents/carers (if in doubt as to whether this seems appropriate, ask an experienced nurse).

## Sexual abuse

This may affect boys or girls and takes many forms. Child sexual abuse can be contact or non-contact, ranging from exposure to indecent acts, grooming online, through to rape. The abuser is often a ♂ relative or carer who is well known to the child, but women are also capable of committing sexual abuse, as are other children.

The child may present in a variety of ways:

- Injury to the genitalia or anus.
- Perineal pain, discharge, or bleeding.
- Behavioural disturbance, enuresis, and encopresis.
- Inappropriate sexual behaviour.
- The child may allege sexual abuse.
- STI (including anogenital warts).
- Pregnancy.
- Repeated UTIs.

Accurately record statements made by the child 'word for word' using quotation marks. Do not pursue a genital examination, but involve a senior doctor at an early stage. The ED staff will aim to treat injuries that need urgent attention, but to defer examination of the genitalia using a colposcope to the relevant forensic experts. Bear in mind that in the context of an allegation of recent sexual assault, a collection of forensic samples for DNA analysis is likely to be required, so ensure that appropriate advice is given to avoid destroying evidence. Refer to local policies and procedures regarding recent and historical disclosures of child sexual abuse.

# Management of child abuse

## Role of the junior ED doctor and nurse

Junior ED staff need to be vigilant in considering abuse when initially assessing and treating children. See NICE guidance, updated in 2017, on when to suspect maltreatment (https://www.nice.org.uk).

Any suspicion of child abuse should prompt the involvement of an expert senior doctor (paediatrician or ED consultant). In every hospital system, there will be a designated doctor for child protection who is available for advice. He or she will examine the child and arrange hospital admission for further investigations (eg skeletal survey) as necessary. Social care and the police may need to be involved. The child may require examination by a forensic physician, and samples/photographs obtained. Follow local procedures for making a social care multi-agency referral.

The chief consideration is treatment and protection of the child, so do not delay treatment of painful or life-threatening problems, whilst awaiting an 'expert'. Ensure that all documentation is legible and meticulous (use body maps). Remember that siblings may also be at risk.

## UK law: The Children Act 1989 and 2004

These Acts replace previous statutes. Central is the concept that the welfare of the child is paramount. In the short term, the 1989 Children Act may be used to obtain orders to protect children. A variety of orders may be obtained.

### *Police Protection Order*

A police officer has legal powers to take any child into 'police protection' for up to 72hr if deemed necessary for his/her own protection. This order may be used to prevent a child from being taken away from the ED by a parent or guardian against medical advice.

### *Emergency Protection Order*

This has replaced the 'Place of Safety Order'. A court order valid for up to 8 days may be obtained if the child is believed to be at significant risk of harm. Such an order would normally be requested by a social worker.

### *Child Assessment Order*

This court order may be applied by the local authority or NSPCC in order to allow an assessment to be performed of a child who appears to be at risk of injury. This order is valid for up to 7 days.

### *Care Order*

This transfers the care of a child from the parent(s) to the local authority's social care department. If a care order is in force, matters requiring parental consent should be referred to the social worker (not the foster carer). Care orders can last until the child is 18y old. Parental responsibility can be transferred to another person through adoption or special guardianship via a court order. Only the courts are able to lift this order.

### *Residence Order*

This court order defines where a child should live and who has parental responsibility.

### Child Protection Plan

The details of all children who are subject to a Child Protection Plan are maintained by social care. ED staff should be aware of how to access Child Protection Plan information. Refer to local hospital alert systems. Previous hospital case notes are also very useful in this respect. When searching for previous records, remember that many children may be known by several surnames.

### Child protection case conferences

A conference may be called by social care if it is suspected that a child has been abused. Child protection case conferences should be held promptly and aim to define a protection plan for the future protection of the child and family. Unlike the criminal courts, where the onus is on the prosecution to prove abuse 'beyond reasonable doubt', child protection case conferences will determine whether a child is deemed to be at risk of significant harm and whether a protection plan is required. Case conferences consist of a number of individuals, including: an independent chairman (usually a senior member of the social care department), a hospital consultant, a GP, a social worker, the police, a health visitor/school nurse, a teacher, an education welfare officer, and a local authority solicitor. Parents are always invited and older children may also attend.

### Sharing information

Failure to share information is implicated in many serious case reviews—Child Protection Information Sharing is at the heart of protecting children. The General Data Protection Regulation (GDPR) and Data Protection Act 2018 do not prevent the sharing of information for the purpose of keeping children safe. Information can be shared without consent, if requesting consent would place the child at risk (eg suspected fabricated or induced illness). Discuss with a senior clinician. Multi-agency safeguarding hubs may enable effective sharing of information.

See '*Information sharing: advice for practitioners providing safeguarding services to children, young people, parents and carers*', published by HM Government in 2018 (https://www.gov.uk). The seven golden rules to information sharing in this document are summarized as follows:

1. The GDPR, Data Protection Act 2018, and human rights law provide a framework to ensure that personal information about living individuals is shared appropriately.
2. Be open and honest with the individual (and/or family where appropriate) about information sharing, unless this is unsafe or inappropriate.
3. Get advice from other practitioners (or information governance lead) if unsure, without disclosing the individual's identity where possible.
4. Where possible, share information with consent, and where possible, respect the wishes of those who do not consent to having their information shared, unless there are good reasons (eg there is a risk to safety).
5. Base decisions about information sharing on safety and well-being.
6. Ensure information shared is necessary, proportionate, relevant, adequate, timely, and secure.
7. Record what is shared and what is not shared, and the reasons why.

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Note: Tables, figures, and boxes are indicated by an italic *t* , *f* , and *b* following the page number.


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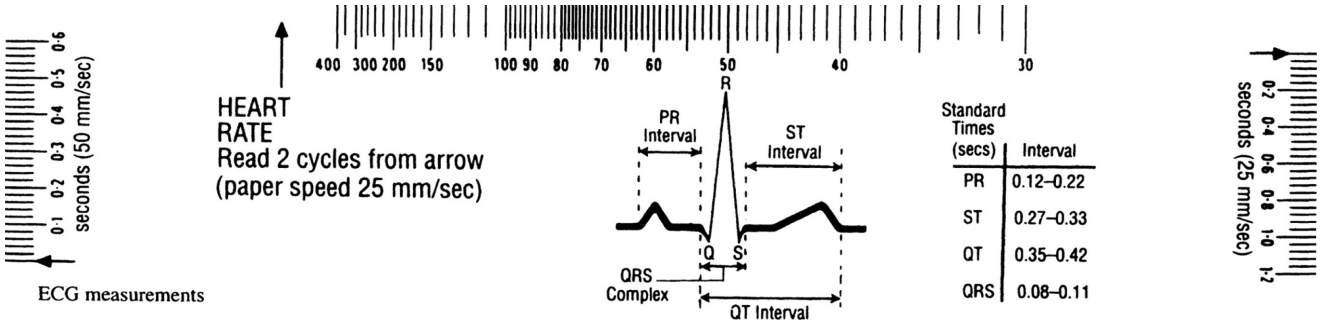


ECG measurements