Adult Emergency Medicine at a Glance

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Preface

Emergency Medicine has undergone a quiet revolution over the past twenty years due to a variety of factors that have changed the way medicine is practiced.

- Increasing demand and expectations of medical care.
- Reduction of junior doctors' hours.
- An ageing population.
- Fragmentation of out of hours care.
- Reduced hospital bed-stay.
- Sub-specialisation of inpatient medical and surgical practice.
- Litigation.

These factors have pushed expert decision-making towards the front door of the hospital so that the correct diagnosis and treatment start as soon as possible in the patient's journey. As other specialties have moved away from the acute assessment and treatment of patients, Emergency Medicine has expanded to fill the vacuum left, and in doing so has increased its realm of practice substantially.

Emergency Medicine is exciting and confronting, intimidating and liberating – it is the chance to exercise and hone your diagnostic and practical skills in a well-supervised environment. Clinical staff who work in the ED have all been through the inevitable feelings of fear, uncertainty and doubt that come with the territory, and want you to experience the enjoyment and satisfaction of working in an area of medicine that is never boring.

When trainees start Emergency Medicine, it is often the first time they have seen patients before any other staff. To use a traditional analogy, they have seen plenty of needles, and may be very good at recognising them, but now they are faced with haystacks, in which may be hidden a variety of sharp shiny objects.

Medical textbooks usually describe topics by *anatomy* or *pathology* (needles), e.g. heart failure, which tends to assume the diagnostic process. In this book we have tried to organise topics by *presentation* (haystacks), e.g. 'short of breath', and have tried to articulate the key features that help us find the needles.

We are both great fans of the '*At a Glance*' series, and have enjoyed the challenge of combining the breadth of practice of adult Emergency Medicine with the concise nature of the '*At a Glance*' format. We hope you enjoy this book and find it useful as you explore this most dynamic area of medicine.

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In addition TH would like to thank: Professor Christopher Bulstrode who has been an inspiration and mentor and without whose support the book would not have happened. My family, and particularly my wife Marina for her support. My work colleagues for continuing to tolerate me most of the time, and Jackie and Tracey, the ED secretaries who keep me in order. Nic Weir, Rob Janas and David Bowden for their help in sourcing images and the final preparation.

JC would like to thank: All the people who have taught me along the way, particularly Trevor Jackson and Steven Pincus. My parents Ron and Christine for making everything possible, and my wife Kerry and sons Jesse and Flynn for making everything worthwhile.

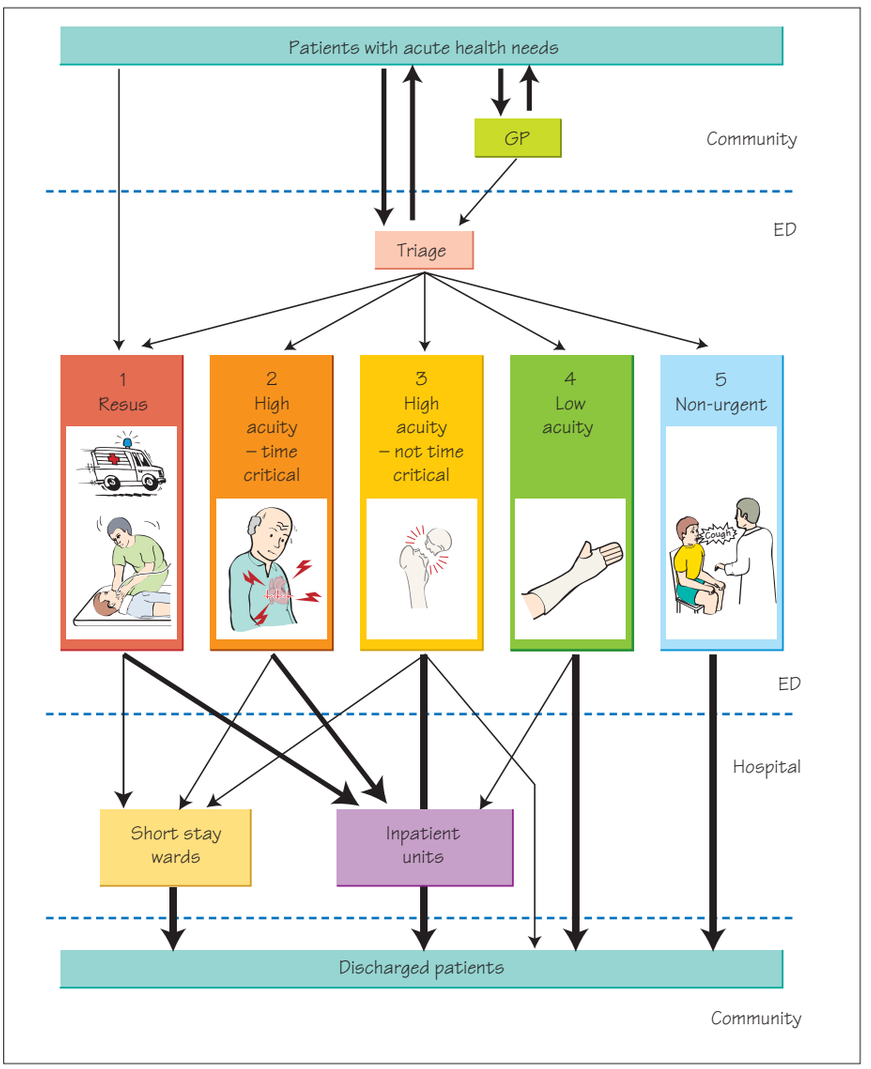
Thomas Hughes
Jaycen Cruickshank

List of abbreviations

AAA	abdominal aortic aneurysm
ABC	airway, breathing, circulation
ABCD²	acronym to assess stroke risk in a patient with TIA
ABCDE	airway, breathing, circulation, disability, exposure
ABG	arterial blood gases
ACE	angiotensin-converting enzyme
ACh	acetylcholine
ACJ	acromioclavicular joint
ACL	anterior cruciate ligament of knee
ACS	acute coronary syndrome
ACTH	adrenocorticotrophic hormone
AD	aortic dissection
AF	atrial fibrillation
AIDS	acquired immunodeficiency syndrome
AMT4	four-point abbreviated mental test score
AP	antero-posterior
APL	abductor pollicis longus
AV	arteriovenous; also atrioventricular
AVN	atrioventricular node
AXR	abdominal X-ray
BDZ	benzodiazepine
BP	blood pressure
bpm	beats per minute
CAGE	acronym for alcohol screening questions
CAP	community-acquired pneumonia
cAMP	cyclic adenonsine monophosphate
CBRNE	chemical, biological, radiological, nuclear, explosive
CK	creatine kinase
CNS	central nervous system
CO	carbon monoxide
COHb	carboxyhaemoglobin
COPD	chronic obstructive pulmonary disease
CPAP	continuous positive airway pressure
CPP	cerebral perfusion pressure
CPR	cardiopulmonary resuscitation
CRAO	central retinal artery occlusion
CRP	C-reactive protein
CRVO	central retinal vein occlusion
CT	computed tomography
CTPA	CT pulmonary angiography
CURB-65	confusion, urea, respiratory rate, blood pressure, age over 65 (acronym for pneumonia severity factors)
CVP	central venous pressure
CXR	chest X-ray; also unit for X-ray dose, 1 CXR ≈ 3 days' background radiation
DIPJ	distal interphalangeal joint
DKA	diabetic ketoacidosis
DM	diabetes mellitus
DSH	deliberate self-harm
DUMBELS	diarrhoea, urination, miosis, bronchorrhoea/bronchospasm, emesis, lacrimation, salivation (acronym for clinical effects of organophosphate poisoning)
DVT	deep vein thrombosis
ED	Emergency Department
EDTA	ethylene diamine tetraacetate
ELISA	enzyme-linked immunosorbent assay
ENT	ear, nose and throat
EPL	extensor pollicis longus
ESR	erythrocyte sedimentation rate
ETT	endotracheal tube
FAST	acronym for focused abdominal sonography in trauma; also face, arm, speech, time to call ambulance
FB	foreign body
FBC/FBE	full blood count/examination
FiO_2	fraction of inspired of oxygen (as %)
FFP	fresh frozen plasma
FOOSH	fall onto an outstretched hand
GA	general anaesthetic
GAβHS	group A β-haemolytic *Streptococcus*
GCS	Glasgow Coma Scale/Score
GI	gastrointestinal
GP	general practitioner
H1N1	swine flu virus
H5N1	avian flu virus
HbA_{1c}	glycated (glycosylated) haemoglobin
HCO_3^-	bicarbonate ion
hCG	human chorionic gonadotrophin
HDU	high dependency unit
HHS	hyperosmolar hyperglycaemic state
HIV	human immunodeficiency virus
HOCM	hypertrophic obstructive cardiomyopathy
HONK	hyperosmolar non-ketotic acidosis
HR	heart rate
HVZ	herpes varicella zoster
IBS	irritable bowel syndrome
ICP	intracranial pressure
ICU	intensive care unit
IgE	immunoglobulin E
IVDU	intravenous drug use
IVF	in vitro fertilisation
IVRA	intravenous regional anaesthesia
IVU	intravenous urogram
JVP	jugular venous pressure
KUB	kidneys, ureters and bladder
LA	local anaesthetic
LCL	lateral collateral ligament of knee
LFT	liver function test
LMP	last menstrual period
LNMP	last normal menstrual period
LOC	loss of consciousness
LP	lumbar puncture
LR	likelihood ratio
LRTI	lower respiratory tract infection
MAOI	monoamine oxidase inhibitor
MAP	mean arterial pressure
MCL	medial collateral ligament of knee
MCPJ	metacarpophalangeal joint
MDI	metered dose inhaler

MI	myocardial infarction
MR	magnetic resonance
N_2O	nitrous oxide
NAC	*N*-acetylcysteine
NICE	National Institute for Health and Clinical Excellence
NIV	non-invasive ventilation
NNT	number needed to treat
NNH	number needed to harm
#NoF	fractured neck of femur
NSAID	non-steroidal anti-inflammatory drug
NSTEMI	non-ST segment elevation myocardial infarction
OD	overdose
OP	organophosphate
OPG	oral pantomogram
ORIF	open reduction and internal fixation
PA	postero-anterior
PCL	posterior cruciate ligament of knee
PE	pulmonary embolism
PEA	pulseless electrical activity
PEF	peak expiratory flow
PEFR	peak expiratory flow rate
PID	pelvic inflammatory disease
PPCI	primary percutaneous coronary intervention
PPI	proton pump inhibitor
PPM	permanent pacemaker
PR	per rectum
PT	prothrombin time
PV	per vaginam
RA	regional anaesthesia
RBBB	right bundle branch block
RoSC	return of spontaneous circulation
SAH	subarachnoid haemorrhage
SAN	sinoatrial node
SARS	severe acute respiratory syndrome
SDH	subdural haematoma
SoB	short(ness) of breath
SOCRATES	acronym for pain history
SOL	space-occupying lesion
SSRI	selective serotonin reuptake inhibitor
STD	sexually transmitted disease
STEMI	ST segment elevation myocardial infarction
STI	sexually transmitted infection
SVT	supraventricular tachycardia
TBSA	total body surface area
TCA	tricyclic antidepressant
TFT	thyroid function test
TIA	transient ischaemic attack
TIMI	thrombolysis in myocardial infarction
TMT	tarsometatarsal
tPA	tissue plasminogen activator
U + E	urea and electrolytes
UA	unstable angina
URTI	upper respiratory tract infection
UTI	urinary tract infection
VBG	venous blood gases
VF	ventricular fibrillation
V/Q	ventilation/perfusion
VT	ventricular tachycardia
VVS	vasovagal syncope
WCC	white cell count

1 Life in the Emergency Department



This chapter describes the way the Emergency Department operates, and some of the unwritten rules. The prevalence of Emergency Department-based drama generates plenty of misconceptions about what occurs in the Emergency Department. For instance, it is generally inadvisable to say 'stat' at the end of one's sentences, and neither of the authors has been mistaken for George Clooney!

What happens when a patient arrives at the Emergency Department?

Alert phone

Also known as the 'red phone' or sometimes 'the Bat-phone', this is the dedicated phone line that the ambulance service uses to pre-warn the Emergency Department of incoming patients likely to need resuscitation.

Triage

The concept of triage comes from military medicine – doing the most good for the most people. This ensures the most effective use of limited resources, and that the most unwell patients are seen first.

Nurses rather than doctors are usually used to perform the triage because doctors tend to start treating patients. Systems of rapid assessment and early treatment by senior medical staff can be effective, but risk diverting attention from the most ill patients.

Reception/registration

The reception staff are essential to the function of the Emergency Department: they register patients on the hospital computer system, source old notes and keep an eye on the waiting room. They have to deal with difficult and demanding patients, and are good at spotting the sick or deteriorating patient in the waiting room.

Waiting room

Adult and paediatric patients should have separate waiting rooms, and some sort of entertainment is a good idea. Aggression and dissatisfaction in waiting patients has been largely eliminated in the UK by the 4-hour standard of care: all patients must be seen and discharged from the Emergency Department within 4 hours.

Treatment areas in the Emergency Department

Resuscitation bays

Resuscitation bays are used for critically ill and unstable patients with potentially life-threatening illness. They have advanced monitoring facilities, and plenty of space around the patient for clinical staff to perform procedures. X-rays can be performed within this area.

High acuity area

This is the area where patients who are unwell or injured, but who do not need a resuscitation bay, are managed. Medical conditions and elderly patients with falls are common presentations in this area.

Low acuity area

The 'walking wounded' – patients with non-life-threatening wounds and limb injuries – are seen here. Patients with minor illness are discouraged from coming to the Emergency Department, but continue to do so for a variety of reasons.

There is a common misconception that patients in this area are similar to general practice or family medicine patients. Numerous studies have found that there is an admission rate of about 5% and an appreciable mortality in low acuity patients, whereas only about 1% of GP consultations result in immediate hospital admission.

Other areas

Imaging

Imaging, such as X-rays and ultrasound, are integral to Emergency Department function. Larger Emergency Departments have their own CT scanner.

Relatives' room

When dealing with the relatives of a critically ill patient and breaking bad news, doctors and relatives need a quiet area where information is communicated and digested. This room needs to be close to the resuscitation area.

Observation/short stay ward

This is a ward area close to the Emergency Department, run by Emergency Department staff. This unit treats patients who would otherwise need hospital admission for a short time, to enable them to be fully stabilised and assessed. The function of these units is described in Chapter 28.

Hospital in the home

Some hospitals run a 'hospital in the home' programme for patients who do not need to be in hospital but who need certain therapy, e.g. intravenous antibiotics, anticoagulation. The Emergency Department is the natural interface between home and hospital.

Culture of the Emergency Department

There is a much flatter (less hierarchical) organisational structure in the Emergency Department than most other areas in the hospital. This occurs because all levels of medical, nursing and other staff work together all the time, and the department cannot function without their cooperation. Ensuring good teamwork requires good leadership, an atmosphere of mutual respect and a bit of patience and understanding.

The resulting atmosphere can be one of the most enjoyable and satisfying places to work in a hospital. A feature of this less hierarchical culture that surprises junior doctors is that nurses will question their decisions; this is a sign of a healthy culture in which errors are less likely to occur, and is actively encouraged.

Emergency Department rules

Being a doctor in the Emergency Department is different from elsewhere in the hospital. There is nowhere to hide and, for the first time in most medical careers, you are responsible for making the decisions. On the positive side, there are plenty of people around to help you, who have all been through the same process.

Some basic advice:

- Write legible, timed, dated notes.
- Show respect for other professional groups and be prepared to learn from them.
- Do not be late for your shifts; do not call in sick less than 6 hours before a shift.
- Patients who re-present are high risk and need senior review.
- Take your breaks. You need them.
- Keep calm.
- If in doubt, ask.
- Do not pick up so many patients that you cannot keep track of them.
- Do not avoid work or avoid seeing difficult patients. We do notice.
- The nurse in charge is usually right.

With so many people working closely together in a stressful atmosphere, it is inevitable that conflicts will occur. Do not let them fester; some ground rules for resolving such conflicts are:

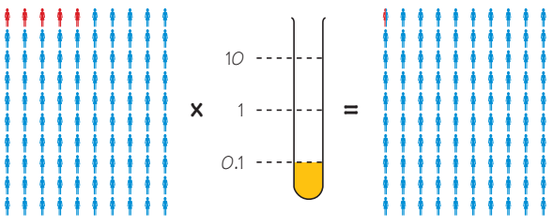
- Resolve it now.
- Do it in private.
- Do it face to face.
- Focus on facts.
- Criticise action, not person.
- Agree why it is important.
- Agree on a remedy.
- Finish on a positive.

2 Diagnosis

A type of d-dimer test has a specificity of 50% and a sensitivity of 95%: this allows you to calculate the likelihood ratios to rule a condition in (+LR) or out (–LR)

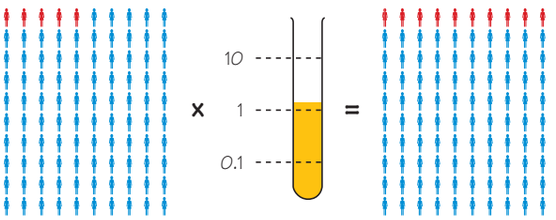
$$+LR = \frac{Sn}{1-Sp} = \frac{0.95}{1-0.5} = 1.9 \qquad -LR = \frac{1-Sn}{Sn} = \frac{1-0.95}{0.5} = 0.1$$

A woman presents with a history suggestive of DVT, and her probability of having a DVT is low; about 5%, according to the Wells score (see Ch. 35).
The d-dimer test comes back negative.
What is the chance she has a DVT?



The result (0.5%) is the risk of missing a DVT in **this patient**. Bearing in mind that no test is perfect, a result below 1% is generally taken as an acceptable level of risk

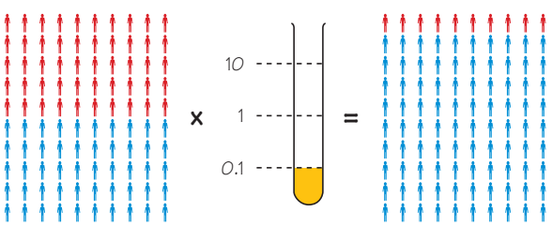
If the d-dimer test had come back positive, what is the chance she has a DVT?



Despite the positive d-dimer, this patient only has a **10%** chance of having a DVT

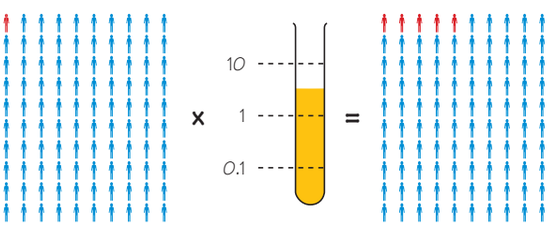
This shows that this d-dimer test is useful for excluding a DVT in a low-risk population, but that a positive test does not mean a DVT is present

A patient who has been immobile following a recent operation for cancer has pain in his lower leg. You assess his risk of DVT as about 50%, but the test comes back negative.
What is the probability he has a DVT?



Despite a negative d-dimer, he still has a 10% chance of having a DVT. D-dimer cannot 'rule out' DVT in a high risk patient

A young man with mild suprapubic pain, whom you estimate has a 1% chance of having a UTI, has a positive urine leucocyte test (+ LR = 5) on dipstick testing. What is the chance he has a UTI?



Despite a negative test with a fairly high + LR he still only has a 5% chance of having a UTI because the condition was so unlikely in the first place

The Emergency Department is a diagnosis machine, taking people with a wide variety of symptoms, labelling them with a diagnosis, treating and then discharging them whenever safe to do so. There are plenty of opportunities for this process to go wrong, and it is important to understand how this can occur.

> 'If you listen carefully to the patient, they will tell you the diagnosis'. W Osler

Despite a couple of thousand years of medical education, we are still not really sure how the diagnostic process works.

- Some people work forward from history, examination and a shortlist of differential diagnoses.
- Others work backwards from a list of likely diagnoses to weight these according to symptoms and examination.
- Experts make their diagnoses by pattern recognition.

It may be that expertise occurs with the development of cognitive flexibility to use multiple diagnostic strategies to integrate and test the result.

Using tests

In the past, the Emergency Department used a few simple tests to inform decision-making. An X-ray of an injured limb has a binary outcome: broken/ not broken. As technology and the scope of Emergency Medicine has increased, the tests have become more numerous and less black and white, and there is a need to rationalise and manage the uncertainty generated.

The most common way of describing a test's performance is to use *sensitivity* and *specificity*.

Confusion matrix

Test result	Actual patient status (truth) Disease present	Disease absent
Positive	True positive (A)	False positive (B)
Negative	False negative (C)	True negative (D)
Total no. patients	With disorder (A + C)	Without disorder (B + D)
	Sensitivity = A/(A + C) and	**Specificity = D/(B + D)**

For example, if a very *specific* test, e.g. Troponin I, is positive, we know that myocardial damage has occurred, because the number of false positives (B) is very low. Similarly, if a very *sensitive* test, e.g. CT scan for abdominal aortic aneurysm (AAA), comes back as negative, we know that it is very unlikely that the patient has an AAA as the number of false negatives (C) will be very low.

SpIN – a very Specific test rules a condition **IN**.
SnOUT – a very Sensitive test rules a condition **OUT**.

However, Emergency Departments use many tests that are not 100% sensitive or specific, and therefore a more powerful, but less intuitive, model is necessary to understand these tests: *likelihood ratios*, which are calculated from the specificity and sensitivity.

Treatment orders

Once the likely diagnosis has been made, a set of treatment orders needs to be decided and documented. A good acronym for this is DAVID, e.g., for an elderly patient with an open fracture of the tibia:

- **D**iet – nil by mouth
- **A**ctivities – elevate limb
- **V**ital signs monitoring – hourly limb observations
- **I**nvestigations – CXR, FBC, U+E
- **D**rugs/treatment – immediate i.v. antibiotics

Bayes' theorem

The chance of something being true or false depends not only on the quality of the test that one is performing, but *importantly*, how likely the event is in the first place.

Thomas Bayes, an eighteenth-century English priest, deduced the principles that underpin the way we use tests in medicine:

pre-test probability × likelihood ratio = post-test probability

To calculate this, we use odds (like horse racing odds) rather than probability, but the two are obviously very closely related.

The likelihood ratio is calculated from the test's sensitivity (Sn) and specificity (Sp) and is a much better measure of a test's clinical usefulness in ruling a disease in (positive LR) or out (negative LR).

$$+LR = \frac{Sn}{1-Sp} \qquad -LR = \frac{1-Sn}{Sp}$$

How do I define pre-test probability?

The triage process uses an expert nurse to assess clinical status and is an assessment of the probability of serious illness. The fact that a patient has arrived at the Emergency Department at all, rather than going to their own doctor, automatically means the probability of significant disease is relatively high.

Clinical decision rules are widespread in Emergency Medicine and help codify knowledge and explicitly define pre-test probability. However, unthinking application of such decision-support tools by clinicians without an appreciation of their flaws and limitations results in bad decisions and/or over-investigation.

A history and examination taken by an experienced clinician remains a very good predictor of pre-test probability of disease. As can be seen opposite, a test applied in an inappropriate population group, i.e. with a pre-test probability that is very high or very low, will give misleading results. Tests are best used in populations with an intermediate probability.

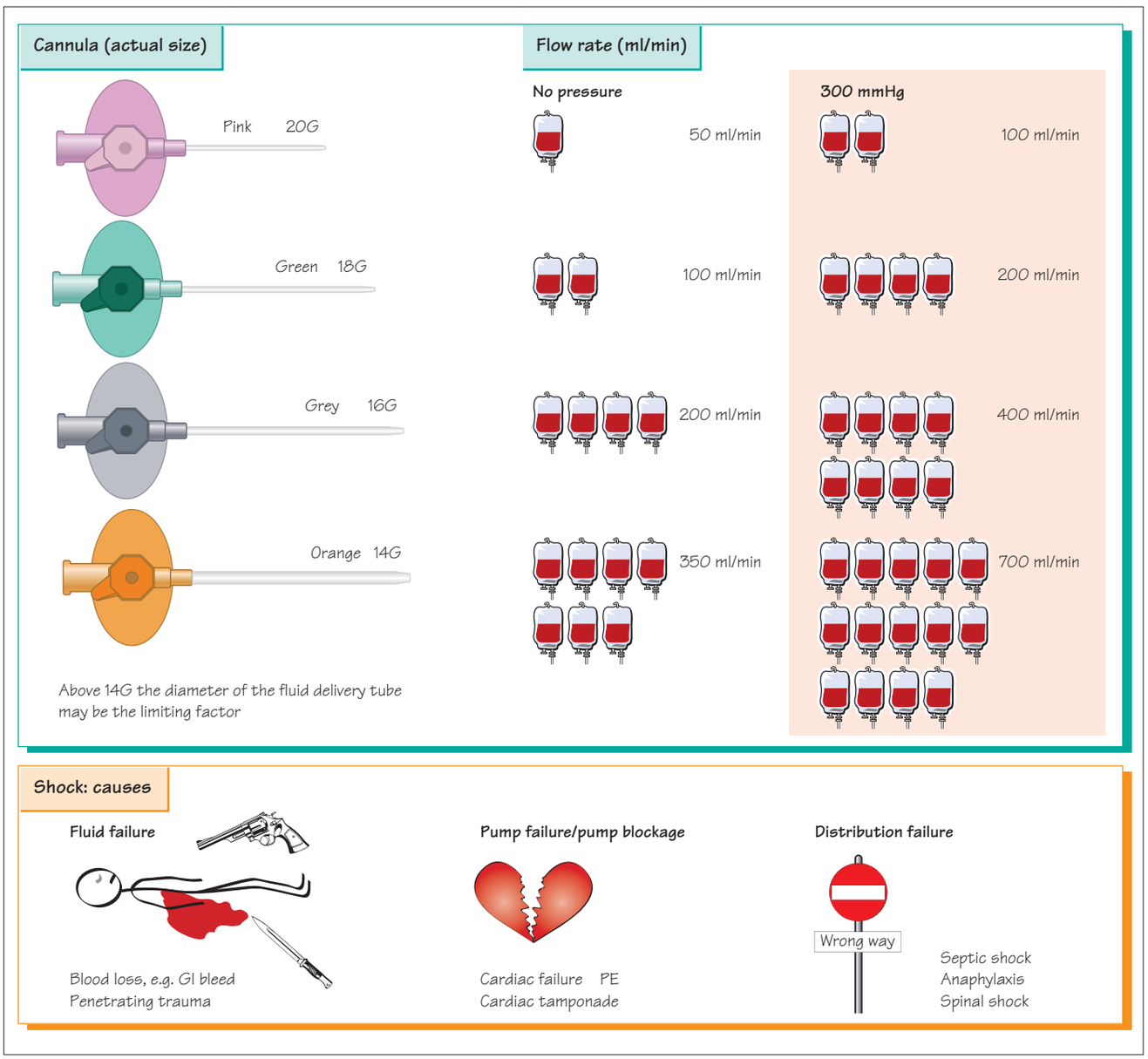
Using likelihood ratios in practice

It helps to think of the likelihood ratio as a multiplier that tells you how much more or less likely a disease is, once you have the result.

The particular advantage of likelihood ratios over other measures of a test's performance is that, as shown opposite, they can easily be applied to individual patients, not just populations.

A good test for ruling in a condition has a positive likelihood ratio of more than 10, meaning that if the patient has a positive test, it is 10 times more likely that they have the disease as a result of the positive test.

3 Shock and intravenous fluids



Intravenous fluid therapy is a common medical treatment, but recently there has been a reassessment of the role of intravenous fluids as some of the hazards have become better understood.

Intravenous access

Poiseulle's Law governing fluid flow through a tube (assuming laminar flow):

$$Flow \propto \frac{Pr^4}{\eta L}$$

where P = pressure difference, r = radius, η = viscosity, L = length.

Therefore the ideal resuscitation fluid should be non-viscous, driven by pressure through a short, wide cannula. For resuscitation, or when giving blood (viscous), a 16 G or larger cannula is preferred. The large veins in the antecubital fossa and the femoral vein are good for resuscitation but are prone to infection and uncomfortable for patients in the long term. A pressure bag inflated to 300 mmHg doubles fluid flow.

Before cannulation the skin should be thoroughly cleaned using chlorhexidine in alcohol. A cannula in the forearm is (relatively) comfortable for the patient and less likely to become infected compared to other sites.

Special cases

• *Central lines* are very useful in very sick patients, patients with poor access or patients whose fluid balance is particularly difficult to regulate. Their length means that they are not ideal for delivering resuscitation fluids. Introducer sheaths offer a large bore central access.

• *Intraosseus needles* have to be drilled into adult bone to give fluid, but can be life-saving. Bone marrow aspirate may be used for blood cross-matching.

• *Venous cut-down* involves cutting the skin to be able to cannulate a vein under direct vision. The long saphenous vein 1 cm above and anterior to the medial malleolus, or the basilic vein in the antecubital fossa, are the most common sites for this.

Types of intravenous fluids

Crystalloids

Normal saline, Hartmann's solution and Ringer's lactate are solutions that match plasma osmolality. All can be used to resuscitate patients, and despite vigorous debate, no one variety has proven superior clinical outcomes.

Dextrose

50% dextrose is used to resuscitate hypoglycaemic patients. 10% dextrose is used to maintain a patient's blood sugar and prevent hypoglycaemia, and 5% is used to give 'free water' to avoid overloading with sodium or chloride.

Colloids

Colloids contain large molecules that help retain fluid within the intravascular space, which improves blood pressure in the short term. Unfortunately these molecules leak out of damaged capillaries, which may cause resistant oedema in the brain and lungs, which increases mortality in head-injured patients. Colloids may be helpful in sepsis, but should only be used by senior doctors.

Blood

A full cross-match takes 30 minutes but type-specific blood should be available within minutes. If blood is needed before the blood type is known, Group O Rhesus negative blood is used.

Whole blood as donated is the best substitute in trauma, but has a short shelf life (days). Separating blood cells into 'packed cells' extends storage time to 3 months, but deterioration may mean that the cells are not fully functional for 24 hours. The citrate used to stabilise blood binds calcium ions, which can cause problems in massive transfusions (>50% blood volume).

Fresh frozen plasma (FFP) or synthetic clotting factors can be used to correct clotting problems. Tranexamic acid, platelets and FFP are given as part of massive transfusion protocol.

Temperature

Evolution has given humans enzymes that function best at 37°C and pH ≈ 7.4. Blood clotting is impaired in a cold acidotic patient, e.g. trauma patient. Temperature <34°C and pH < 7.20 reduce clotting to 1% of normal. Laboratory measurements at 37°C will not accurately reflect the clinical picture. For this reason, clotting factors are given early in trauma resuscitation. Cold fluids (4°C) may be given after cardiac resuscitation as part of an active cooling strategy to preserve brain function.

Shock

Shock is defined as inadequate tissue perfusion, i.e. not meeting the metabolic demands of tissue. Pulse and blood pressure are bedside measures of tissue perfusion, but are insensitive. pH, PCO_2, lactate and mixed venous blood oxygen levels, measured from a central venous pressure (CVP) line, are better indicators.

Types of shock

The body pumps a limited amount of fluid around a series of closed loops. Problems occur when the fluid disappears, the pump fails or the fluid goes to the wrong loops.

Blood failure

Blood loss may be controlled or uncontrolled, internal or external. Severe dehydration may cause similar problems.

Pump failure

The heart may fail due to internal pump problems, e.g. myocardial infarction or heart failure, which impair the ability to pump. Alternatively the pump may fail because there is inflow obstruction (cardiac tamponade, tension pneumothorax) or outflow obstruction (pulmonary embolus, aortic dissection).

Distribution failure

Blood may be distributed to the wrong organs. Inappropriate vasodilation occurs in septic shock, anaphylaxis and spinal shock (due to loss of sympathetic tone below the injury) diverting blood away from vital organs.

Grades of shock

Compensated shock BP → HR ↑

Young adults are able to compensate for loss of blood volume by vasoconstriction and increased cardiac output, maintaining a good BP and perfusion of vital organs.

Decompensated shock BP ↓ HR ↑

The body's compensation mechanisms are overwhelmed, and the blood pressure falls rapidly.

Fluid resuscitation

Traditional teaching: 'Fill 'em up'

• Good blood pressure = good outcome.
• Poor blood pressure = poor outcome.
• Therefore give fluid/blood to achieve good blood pressure.

Unfortunately this is an oversimplification. Short-term poor perfusion is well tolerated and if blood loss has *not* been controlled:

• ↑ blood pressure = ↑ blood loss.

Increased blood loss is due to loss of vasospasm, dilution of clotting factors and dislodgement of clot.

Current teaching: 'minimal volume fluid resuscitation'

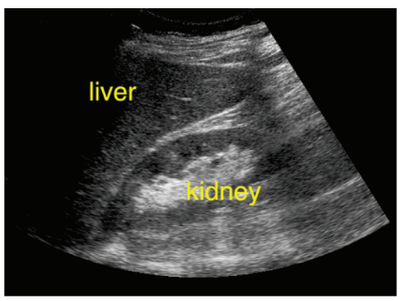
If there is *uncontrolled bleeding* (e.g. penetrating trauma, ruptured AAA), large-bore intravenous access is obtained. The minimum volume of fluid necessary to maintain cerebral perfusion or a systolic BP of 60–80 mmHg is used ('permissive hypotension'). The priority is urgent control of bleeding in the operating theatre.

Exception: if there is brain injury, the need to maintain cerebral perfusion pressure overrides hypotensive resuscitation.

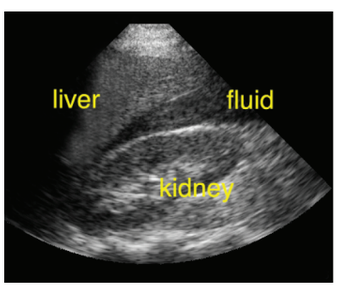
4 Imaging in the Emergency Department

Focussed Assessment using Sonography in Trauma – FAST Scan

Hepato-renal view (Morison's Pouch)



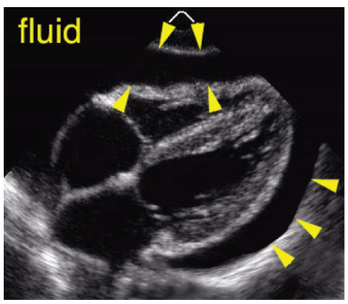
Normal



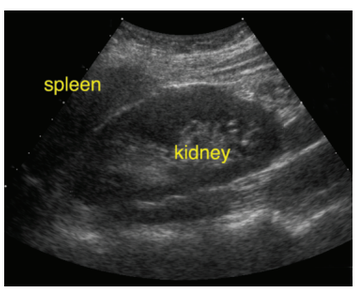
Positive
Shows fluid (blood) between liver and kidney

Cardiac view

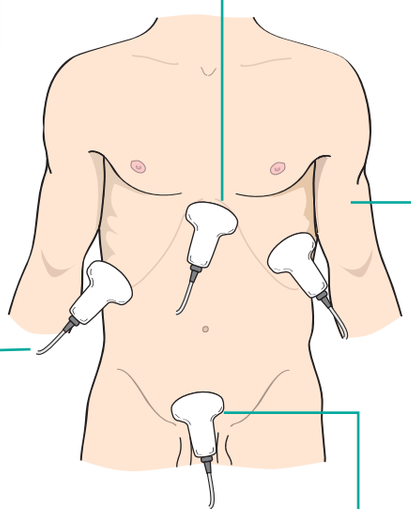
Fluid in pericardial sac ± tamponade



Spleno-renal view

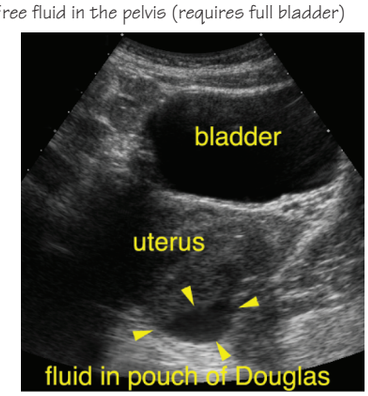


Normal – no fluid between kidney and spleen

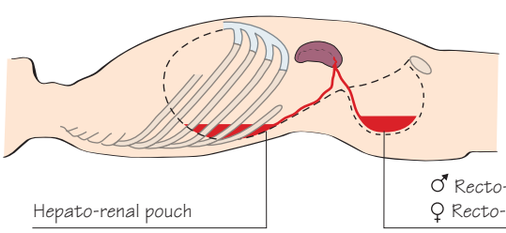


Pelvic view

Free fluid in the pelvis (requires full bladder)



The FAST scan is performed to look for free abdominal fluid – usually blood. The fluid tends to pool in certain areas within the peritoneal cavity



Imaging use in the Emergency Department has increased rapidly over the past few years due to technical advances and increasing pressure to move decision-making earlier in a patient's journey, and to prevent unnecessary hospital admissions. Ultrasound is now a core skill for senior Emergency Department doctors and new hospitals often have a CT scanner in the Emergency Department.

Plain radiography

Plain radiographs interpreted by the treating clinician are used for the majority of Emergency Department imaging. The advent of digital radiography has made real-time reporting by radiologists easier.

X-rays are ionising radiation and cause damage to tissues through which they pass. The energy released is proportional to the density of the tissue. Abdominal or thoracolumbar radiographs should not be performed in young people, especially females, without a very good reason, as the gonads are very radiosensitive. In this book, X-ray doses are expressed in terms of chest radiographs (CXR). One CXR is approximately 3 days of background radiation.

X-rays are not therapeutic. If the result will not change management, radiographs should not be taken. Examples include uncomplicated rib fractures (when not worried about a pneumothorax), coccyx pain and stubbed toes other than the big toe. Soft tissues are poorly shown by plain films, making it an insensitive examination for joints that rely on these for stability, e.g. knee, shoulder.

Reading plain radiographs

1 Check the patient's name and the date of the film, particularly on digital radiography systems, which offer many opportunities for confusion.

2 There should be two good views of limbs: anterior-posterior and lateral.

3 If requesting imaging of more than one area, ask yourself if this is necessary. If not urgent, it may be better to re-examine the patient once they have had some analgesia, or obtain a senior opinion.

4 You will learn more from your radiology department if you engage with them and ask their advice rather than expecting a purely technical service.

5 Many Emergency Departments operate a system whereby the radiographer can flag an abnormality on the radiograph. You should not dismiss something that the radiographer has flagged as abnormal without obtaining a senior opinion.

Clinical ultrasound

Clinical (bedside) ultrasound use has increased exponentially with the availability of cheap robust ultrasound machines, and is now a core skill for Emergency Department doctors. Ultrasound has been described as the 'visual stethoscope' and is revolutionising the assessment and management of patients in the Emergency Department.

Ultrasound was initially used in the Emergency Department in the resuscitation room for:

- Detecting abdominal aortic aneurysms (AAA).
- Focused abdominal scanning in trauma (FAST) scans, searching for blood in the peritoneal cavity.
- Central venous line placement.

However, ultrasound use is now expanding to include:

- Shock assessment: cardiac function, vascular filling, signs of pulmonary embolus, together with the AAA and FAST scans.
- Basic echocardiography.
- Deep vein thrombosis (DVT) scanning.
- Early pregnancy scanning.
- Hepato-biliary scanning.

Disadvantages are that ultrasound is operator dependent, requires training and skill validation, and can divert attention from more important problems.

Computed tomography scan

As resolution and availability have increased and acquisition times have dropped, computed tomography (CT) has become an increasingly useful tool for the Emergency Department. CT is very good for bony injuries, and the trauma CT has proved to be more sensitive and specific than clinical examination in major trauma, but requires a very large radiation dose (1000 CXR).

Neck imaging in high-risk trauma is routinely done by CT (100 CXR), as plain films are insufficiently sensitive at detecting significant injury. Examples of high-risk injuries are a high-speed rollover road traffic collision, and also the elderly patient who falls forward, hitting their face ('fall on outstretched face'), who is at high risk of odontoid peg fracture, and in whom interpretation of plain radiographs is very difficult (see Chapter 11).

Modern CT scanners have enough resolution and speed to be able to resolve cardiac anatomy including the coronary arteries, pulmonary emboli and aortic dissection (400 CXR). CT brain scan (100 CXR) is an essential part of the assessment of stroke or the unconscious patient. CT KUB (kidneys, ureters and bladder; 400 CXRs) is the imaging of choice in renal colic.

Magnetic resonance scan

Magnetic resonance (MR) scanning is rarely used in the Emergency Department apart from possible cauda equina syndrome (acute central disc prolapse pressing on the cauda equina), giving bowel and bladder symptoms. MR scanning can be used to avoid the large radiation dose incurred by CT, e.g. investigating renal colic in young women.

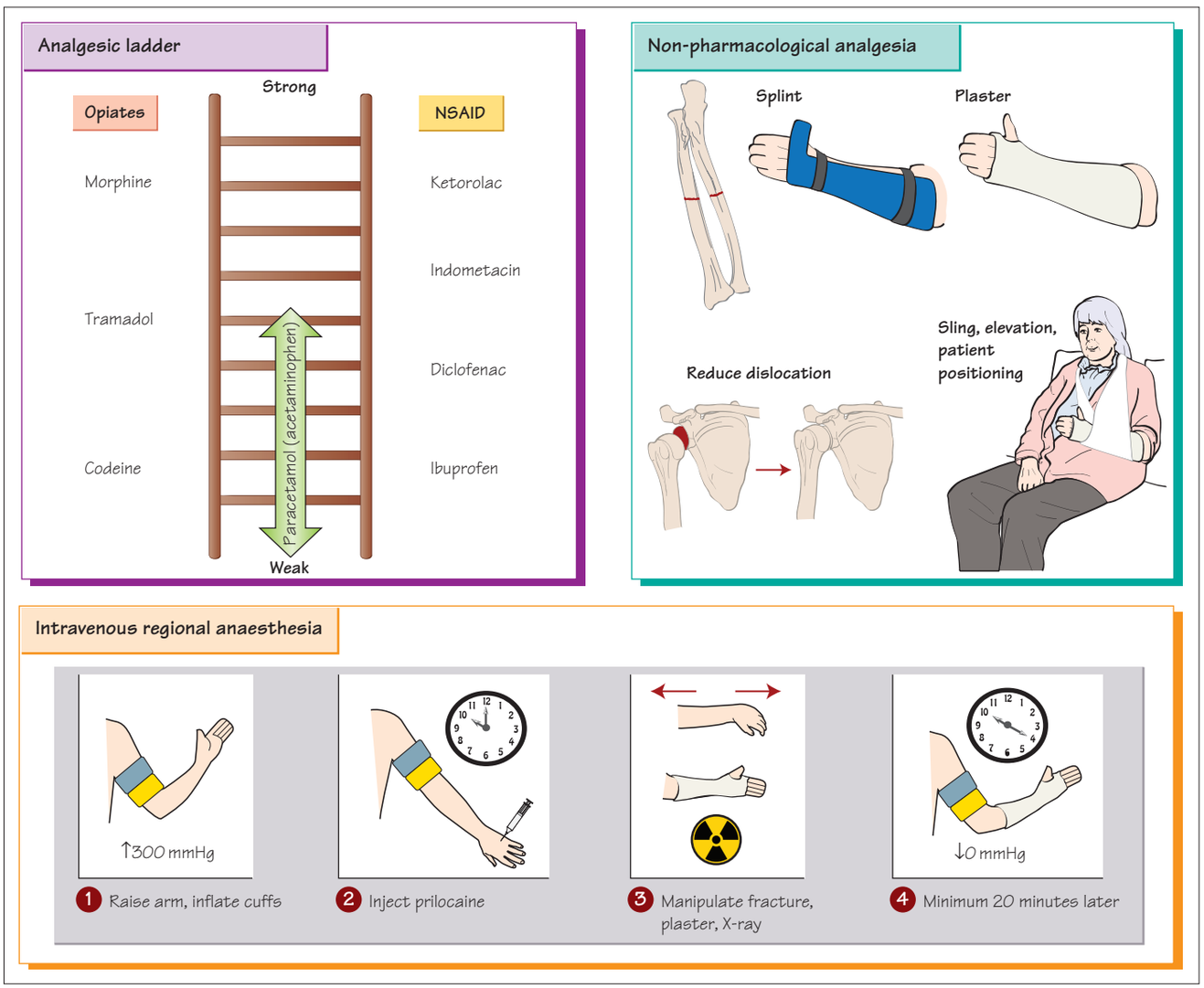
Joints in which stability and function are mainly due to soft tissues, i.e. ligaments and cartilage such as the knee and shoulder, are well imaged by MR scanning, but it is generally difficult to access these directly from the Emergency Department.

Interventional imaging

Interventional imaging has an increasing role for a limited number of severe conditions. Interventional imaging is generally offered in larger hospitals, and together with trauma care, is one of the main drivers for centralisation of acute services into large hospitals.

- Primary percutaneous cardiac intervention with stenting has become the treatment of choice for patients with myocardial infarction.
- Endovascular treatments for patients with AAAs and aortic dissection are increasingly used. Neurosurgical bleeding from aneurysms is treatable by coils, as is otherwise uncontrollable bleeding in the pelvis, e.g. from pelvic fractures.

5 Analgesia



Patients often arrive at the Emergency Department in pain, and painkillers are often used before a definitive diagnosis is made. This is humane, and enables a thorough examination to be performed: there is no reason to withhold analgesia.

Patients are asked to rate the pain out of 10, with 0 being no pain, and 10 being the worst pain they can imagine. This procedure is repeated to gauge the effectiveness of the treatment and ensure the pain is controlled.

In general, a patient's reported pain is taken at face value: 'pain is what the patient feels' and is treated as such. Patients seeking opiates may fake pain, but this is rare.

Non-pharmacological analgesia

Splinting of fractures immobilises the bones, reducing pain. A patient's anxiety and pain makes them tense, which may make pain worse: a calm, supportive atmosphere and excellent nursing care help to keep the patient relaxed.

Nitrous oxide

Nitrous oxide (N_2O) combined with oxygen in a 1:1 mix in cylinders (Entonox®) is often used, particularly out of hospital. It is a short-term analgesic, effective only while the patient is breathing

the gas, as it is rapidly cleared from the body. This 'laughing gas' is generally very safe, but should not be used in patients with a possible pneumothorax.

Paracetamol (acetaminophen) and compound analgesics

Paracetamol (acetaminophen) is effective and safe and can be given orally, rectally or intravenously. Compound analgesics consist of paracetamol combined with another analgesic, usually low-dose codeine. They come in different strengths, the weaker of which are sold without prescription. They are useful analgesics for patients to be able to take home on discharge, but prescribing the constituent drugs separately may allow more flexibility.

Moderate opiates

- *Codeine* is a common component of compound analgesics, and is effective but tends to cause constipation. *Oxycodone* and *dihydrocodeine* are more powerful variants of codeine, but offer little extra benefit, and have high abuse potential.
- *Tramadol* may be more effective than codeine. It has less abuse potential than other drugs of comparable potency but should be used with caution in the elderly.

Major opiates: morphine, fentanyl, pethidine (meperidine)

Opiates induce a feeling of well-being: patients, while still aware of the pain, are not distressed by it. Young patients with major fractures may require large doses of morphine, as will opiate addicts who need analgesia. Intravenous opiates are used because intramuscular absorption is unreliable and the intravenous route enables analgesia to be titrated to response.

- *Intravenous morphine* is the gold standard of Emergency Department analgesia. It is safe, predictable and effective. Morphine is not as lipid soluble as other opiates, so does not give a significant 'high'. Morphine often causes mild histamine release that should not be confused with an allergic reaction. The duration of action of morphine is approximately 3 hours.
- *Fentanyl* is a short-acting synthetic opiate that is particularly useful when performing short procedures, as it is cleared from the body within 30 minutes.
- *Pethidine* (meperidine) is quite lipid soluble and therefore sought after by opiate addicts as it crosses the blood–brain barrier, giving a 'high'. It offers no benefits over morphine and should not be used unless a patient has a definite allergy to morphine and there are no other alternatives.

Non-steroidal anti-inflammatory drugs

Injectable non-steroidal anti-inflammatory drugs (NSAIDs), e.g. *ketorolac*, are very effective in an Emergency Department setting. They are particularly useful in patients with broken bones, colicky pain (e.g. ureteric colic) and abdominal pain, but should be avoided in elderly patients or those with active bleeding. An equally effective alternative is a suppository (e.g. indometacin, diclofenac), which lasts for 16 hours.

Oral NSAIDs are useful as they can also be given to patients on discharge. *Ibuprofen* is the least powerful, but has a relatively benign side-effect profile.

Diclofenac and *indometacin* are more powerful NSAIDs but at a cost of increased risk of side-effects.

Local anaesthesia and nerve blocks

- *Lidocaine* 1% is the local anaesthetic (LA) most often used for wound management and is effective for 20–30 minutes without adrenaline, or for 40–60 minutes with adrenaline.
- *Adrenaline* mixed with lidocaine increases length of action and causes vasoconstriction giving a 'dry' wound that is much easier to assess, clean and close. Fear about using local anaesthetics with adrenaline in digits was related to high concentrations (1 : 10 000); less than 1 : 100 000 adrenaline is safe.
- *Bupivicaine* 0.25% is a long-acting local anaesthetic, lasting for 6–8 hours. Bupivicaine is highly protein bound: adrenaline does not increase duration of action.

A safe maximum dose of lidocaine for wound infiltration is 3 mg/kg, but with adrenaline is 6 mg/kg. For bupivicaine the maximum dose is 2 mg/kg. Local anaesthetic toxicity first causes perioral parasthesia, and then fits and arrhythmias, and is treated by lipid infusion.

Nerve blocks can offer very effective analgesia, e.g. digital and femoral nerve blocks. Bupivicaine and lidocaine can be mixed to provide a combination of rapid onset and long duration of action. Local anaesthetic can also be injected into joints, e.g. for shoulder dislocation.

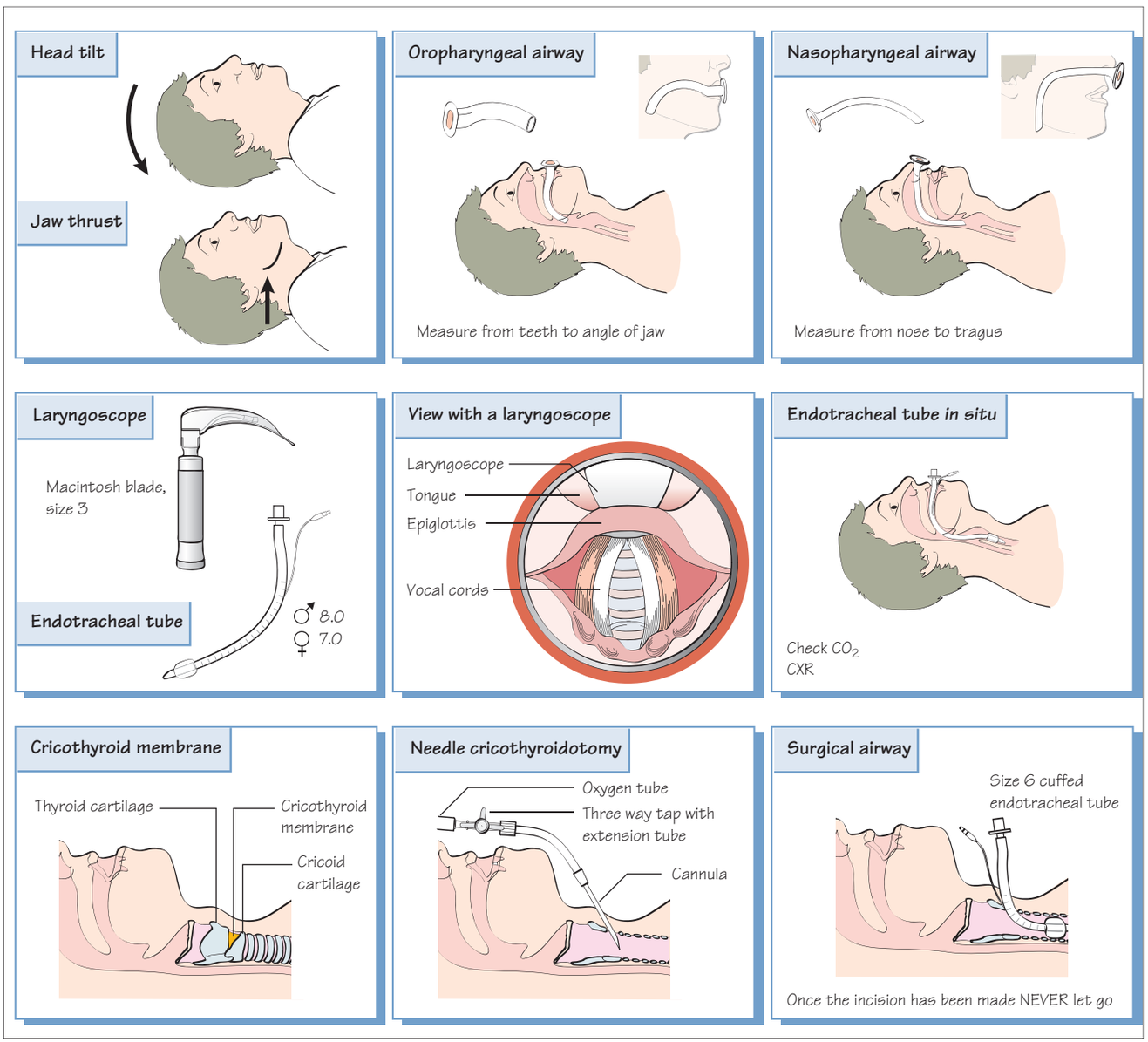
A haematoma block can give good anaesthesia in minor fractures e.g. Colle's fractures (Chapter 15). The skin is carefully cleaned with alcohol and chlorhexidine and then up to 10 mL of local anaesthetic is injected into the fracture haematoma. After about 10 minutes reduction can be performed.

Intravenous regional anaesthesia (Bier's block)

Two intravenous cannulae are sited, one in the affected limb. A double cuff is placed on the affected limb (usually the arm), which is then lifted to exsanguinate it. The cuff is then inflated well above the systolic BP and local anaesthetic, e.g. prilocaine, injected. Bupivicaine should never be used for intravenous regional anaesthesia.

After waiting 5 minutes for the local anaesthetic to have maximal effect, the operation, e.g. fracture reduction, is performed. The cuff must not be deflated until *at least 20 minutes* have elapsed from injection of the local anaesthetic to avoid a bolus of undiluted local anaesthetic perfusing the heart, potentially causing asystole.

6 Airway management and sedation



Airway management in the Emergency Department is more challenging than in the operating room as patients presenting to the Emergency Department must be assumed to be non-fasted, may be physiologically unstable, and may have head, neck or facial injuries.

Oxygenation and ventilation

Oxygenation is ensuring that the body has enough O_2; ventilation is ensuring that there is sufficient airflow to remove CO_2. Oxygen consumption is markedly increased in the acutely unwell patient, and giving high concentrations of oxygen supports the metabolic demands of the body in acute illness. However, high levels of oxygen may paradoxically make some ischaemic injury worse, e.g., brain/ heart due to vasoconstriction. A normal 'Hudson' O_2 mask can give inspired oxygen (FiO_2) concentrations of up to 60%. They should not be used with O_2 <4 L/min to prevent CO_2 build-up. A mask with a reservoir bag or a self-inflating bag-valve-mask can increase FiO_2 to about 90% with high flow (>10 L/min O_2). A

Venturi mask gives accurate low FiO_2 e.g. 28%. Nasal prongs give a variable amount of O_2 approx 25–30% but should only be used with low flow rates (2 L/min O_2).

Ventilatory failure

Under normal circumstances, an increased level of CO_2 is the main driver to breathe. Patients with chronic lung disease, usually COPD, become immune to this drive. For these 'blue bloater' patients, a low blood O_2 level drives breathing: their CO_2 level will be high.

If high FiO_2 is given to these patients, it reduces their respiratory drive, increasing their CO_2 levels further, making them sleepy, which further decreases their drive to breathe, etc. An oxygen saturation target of 91% in these patients balances the need for tissue oxygenation against that for ventilation.

WARNING

- O_2 should be prescribed, with a target saturation
- A patient with O_2 sat >96% probably does not need extra O2 unless high metabolic need, e.g., sepsis, trauma
- If FiO_2 has given a very high CO_2 level, reduce FiO_2 *slowly*
- If in doubt, give O_2 and obtain a senior review

Suction

A Yankauer suction catheter is used to suction blood, vomit or secretions in the oropharynx. To avoid causing the patient to vomit, do not suction the oropharynx if the patient is conscious, and 'only suck where you can see'.

Airway support

The jaw thrust, head tilt, oropharyngeal and nasopharangeal airways are illustrated opposite. The oropharyngeal airway is sized as the distance between the patient's teeth and the angle of the mandible. The nasopharyngeal airway should be the same length as the distance between the tip of the nose and the tragus of the ear.

Laryngeal mask airway

Emergency Department patients are not fasted and the laryngeal mask airway (LMA) does not prevent stomach contents being aspirated, nor can high ventilation pressures be achieved, as might be necessary in asthmatic patients. For these reasons the LMA is not a 'definitive' airway and is not normally used in the Emergency Department.

Endotracheal tube

The most common means to provide a definitive airway, the endotracheal tube (ETT), is a plastic tube that is inserted through the mouth (or rarely the nose) into the trachea. There is a cuff that is inflated to seal against the tracheal mucosa, and a radio-opaque line to indicate position on X-ray. The ETT should be secured, e.g. with tape, and the position checked by CO_2 monitoring and a chest X-ray.

Endotracheal tubes are sized by their internal diameter: 7.0 mm for an adult female, 8.0 mm for a male. There are markings indicating distance from the tip: this is to avoid the tube being pushed too far, e.g. down the right main bronchus, which is larger and straighter than the left.

The decision that the patient needs intubation is the responsibility of the doctor managing the airway. Factors indicating need for intubation include:

- Airway instability: bleeding into airways, airway burns.
- Coma: Glasgow Coma Scale (GCS) < 9, deteriorating level of consciousness, loss of protective laryngeal reflexes.
- Inadequate oxygenation: despite high inspired O_2 (FiO_2).
- Inadequate ventilation: patient tired/drowsy.
- Therapeutic reasons: control seizures, hypothermia.
- Pragmatic reasons: combative patient, need for transport.

A *laryngoscope* is needed to insert the ETT. In some countries, straight (Miller) blades are used; in others, curved (Macintosh) blades. These have a light to enable sight of the larynx.

McGill's forceps have a 'kink' in them to avoid the operator's hands obstructing the field of vision. They are useful for removing loose items in the oropharynx, and manipulating the ETT.

Surgical airway

Rarely, a situation occurs when it is not possible to intubate or ventilate a patient. In this situation, there are two options:

- A *needle cricothyroidotomy* will provide short-term oxygenation, but is not a definitive airway, and CO_2 levels will build up.
- A *surgical airway* through the cricoid membrane using a 6.0 mm cuffed ETT provides a definitive airway.

Procedural sedation

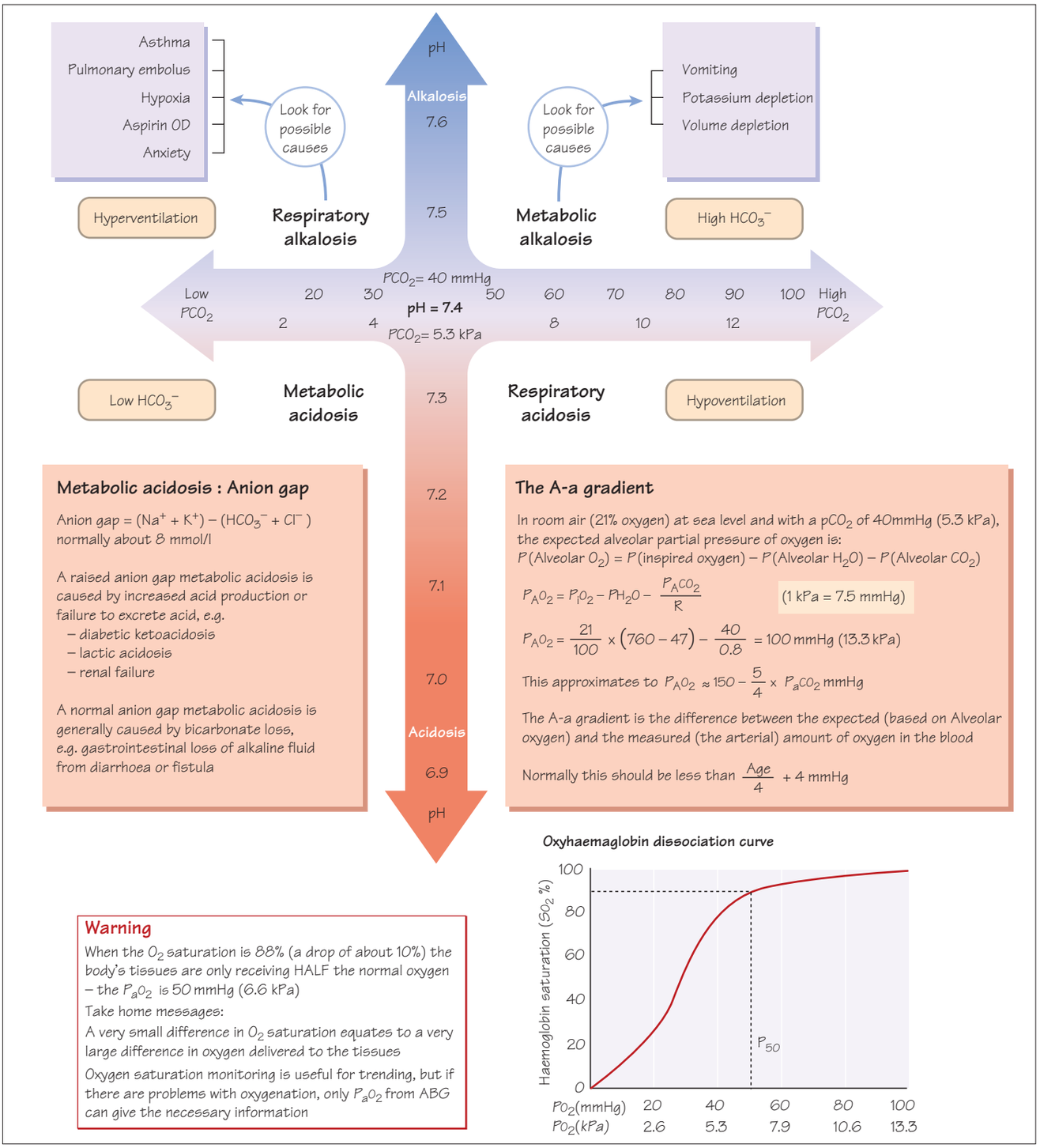
Procedural sedation is often performed in the Emergency Department to allow relocation of dislocations or for short painful procedures. *The person performing the sedation needs appropriate skills and experience to manage any potential situation, including the need for intubation.*

The procedure should be carried out in a resuscitation bay with full monitoring, oxygen and suction equipment. Two doctors should be present at all times to ensure that the doctor administering the sedation has their full attention on the patient's airway. The patient should be fasted for at least 4 hours, should give formal consent, and the doctor should stay with the patient until they are consistently responsive.

After sedation patients should not drive for a day and should be sent home in the care of a responsible adult with instructions to return if unwell.

- *Propofol* is a short-acting anaesthetic induction drug, but is used for sedation by giving as a series of small boluses, titrating for effect. Large doses of propofol abolish protective airway reflexes and may stop the patient breathing. Propofol has no analgesic properties so may need to be given with an analgesic, e.g. fentanyl.
- *Midazolam*, a short-acting benzodiazepine, may be used in combination with an opiate to provide sedation.
- *Ketamine* is a safe and predictable drug that is often used for paediatric sedation. It can be used for sedation and analgesia in adults, and may be combined with a short-acting benzodiazepine to minimise unpleasant emergence phenomena, e.g. hallucinations.

7 Blood gas analysis



Metabolic acidosis : Anion gap

Anion gap = $(Na^+ + K^+) - (HCO_3^- + Cl^-)$
normally about 8 mmol/l

A raised anion gap metabolic acidosis is caused by increased acid production or failure to excrete acid, e.g.
- diabetic ketoacidosis
- lactic acidosis
- renal failure

A normal anion gap metabolic acidosis is generally caused by bicarbonate loss, e.g. gastrointestinal loss of alkaline fluid from diarrhoea or fistula

The A-a gradient

In room air (21% oxygen) at sea level and with a pCO_2 of 40mmHg (5.3 kPa), the expected alveolar partial pressure of oxygen is:
$P(\text{Alveolar } O_2) = P(\text{inspired oxygen}) - P(\text{Alveolar } H_2O) - P(\text{Alveolar } CO_2)$

$$P_AO_2 = P_iO_2 - PH_2O - \frac{P_ACO_2}{R} \qquad (1\ kPa = 7.5\ mmHg)$$

$$P_AO_2 = \frac{21}{100} \times (760 - 47) - \frac{40}{0.8} = 100\ mmHg\ (13.3\ kPa)$$

This approximates to $P_AO_2 \approx 150 - \frac{5}{4} \times P_aCO_2$ mmHg

The A-a gradient is the difference between the expected (based on Alveolar oxygen) and the measured (the arterial) amount of oxygen in the blood

Normally this should be less than $\frac{Age}{4} + 4$ mmHg

Warning

When the O_2 saturation is 88% (a drop of about 10%) the body's tissues are only receiving HALF the normal oxygen – the P_aO_2 is 50 mmHg (6.6 kPa)

Take home messages:

A very small difference in O_2 saturation equates to a very large difference in oxygen delivered to the tissues

Oxygen saturation monitoring is useful for trending, but if there are problems with oxygenation, only P_aO_2 from ABG can give the necessary information

Oxyhaemaglobin dissociation curve

Arterial blood gases

Arterial blood gas analysis provides information about oxygenation (O_2) and ventilation (CO_2), and metabolic disturbance. Some machines also provide electrolytes, lactate and carbon monoxide levels.

Indications for blood gas measurement

1 Diagnostic.
   - Severe shortness of breath.
   - Possible pulmonary embolus.
2 Assessment of severity of illness.
   - Shock, severe sepsis.
   - Diabetic ketoacidosis.
   - Severe vomiting and diarrhoea.
3 Specific situations.
   - Overdose of tricyclic antidepressants or aspirin.

Arterial puncture is painful and should only be performed if the result is going to change management. This is particularly important in young adults with chronic conditions. If just the pH is required, e.g. for a patient with diabetes, this may be obtained from venous blood.

The blood gas machine has three main sensors: pH, PaO_2 and $PaCO_2$. Other values such as bicarbonate and base excess are calculated from these values, not measured directly. Therefore you can deduce the problem using just these three values.

Oxygenation

Hypoxia occurs in two situations:

1 Not enough oxygen reaches the blood.
   - High altitude: not enough oxygen in the air.
   - Hypoventilation: neuromuscular disease, extreme fatigue
   - Obstruction: asthma.
2 Not enough blood reaches the oxygen.
   - Ventilation/perfusion (V/Q) mismatch: the lung tissue is intact but there is no blood passing through it, e.g. pulmonary embolus. If the arterial oxygen level fails to correct with 100% oxygen, this implies 'shunting', i.e. blood is bypassing the lung altogether.
   - Alveolar dysfunction: the apparatus for gas exchange is not working, e.g. interstitial lung disease, pulmonary oedema.

The A-a gradient

If we know the fraction (%) of oxygen the patient is breathing in ($=FiO_2$) we can calculate the A-a gradient. The A-a gradient compares the *expected* amount of oxygen in the blood (= the amount of oxygen in the **A**lveolus), PAO_2, with the *actual* amount of **a**rterial oxygen, PaO_2. Common causes of a large A-a gradient are:

- The blood not reaching the oxygen, e.g. pulmonary embolus.
- A barrier to effective gas exchange, e.g. pulmonary oedema.

Calculating the partial pressure of alveolar oxygen is shown opposite (R is the respiratory quotient and is related to diet).

Pulse oximetery

Pulse oximetry is very useful for monitoring patients, as it is non-invasive. The oxygen saturation is calculated by shining two beams of light through soft tissue, e.g. finger or earlobe, to estimate the fraction of haemoglobin carrying oxygen.

Unfortunately pulse oximetry has a significant flaw that can trip up the unwary. The blood value we want to measure is the PaO_2 = the amount of oxygen carried in arterial blood. Oxygen saturation is only a surrogate measure of the PaO_2: the graph (opposite) shows the relationship between the two.

Under normal circumstances, with an oxygen saturation of 100%, the PaO_2 is 13.3 kPa (100 mmHg).

WARNING

When the PaO_2 is *halved* to 6.6 kPa, the saturation is still 88%. Take-home message: O_2 saturation less than 97% is not good oxygenation.

Acid–base disturbance

Acidosis and alkalosis have a chicken/egg relationship with ventilation, (measured by $PaCO_2$) and respiratory effort: sometimes it is not always clear which came first. To analyse these problems, start with the acid–base disturbance, and then look at the $PaCO_2$.

Acidosis (pH < 7.35)

CO_2 low = metabolic acidosis

If the patient is acidotic and the $PaCO_2$ is low, it is likely the patient is breathing deeply to expel CO_2, to compensate for the metabolic acidosis by hyperventilation. This is often seen in diabetic ketoacidosis and is called Kussmaul breathing.

CO_2 high = respiratory acidosis

If the CO_2 is high, it is likely that this is at least partially responsible for the acidosis, although usually the acidosis is mixed (partly metabolic and partly respiratory).

The normal stimulus to breathe is increased blood CO_2 levels, so a high CO_2 level implies failure of adequate ventilation.

Some patients with lung disease (e.g. COPD) lose their sensitivity to increased blood CO_2 levels and therefore rely on low O_2 levels to drive their breathing. Giving these patients high concentrations of oxygen dangerously reduces their respiratory drive, resulting in a build-up of CO_2. The increase in CO_2 makes the patient sleepy, further reducing respiratory effort.

If faced with a patient who is on home oxygen or is known to have advanced COPD, the safest action is to give enough oxygen to ensure an oxygen saturation of about 91%. Any more than this may abolish the patient's drive to breathe.

Patients with chronically elevated CO_2 levels compensate for this by excreting acid (H^+) renally to rebalance the equation:

$$CO_2 + H_2O \Leftrightarrow HCO_3^- + H^+$$

Therefore these patients will have a chronically raised HCO_3^- (bicarbonate) level.

Alkalosis (pH > 7.45)

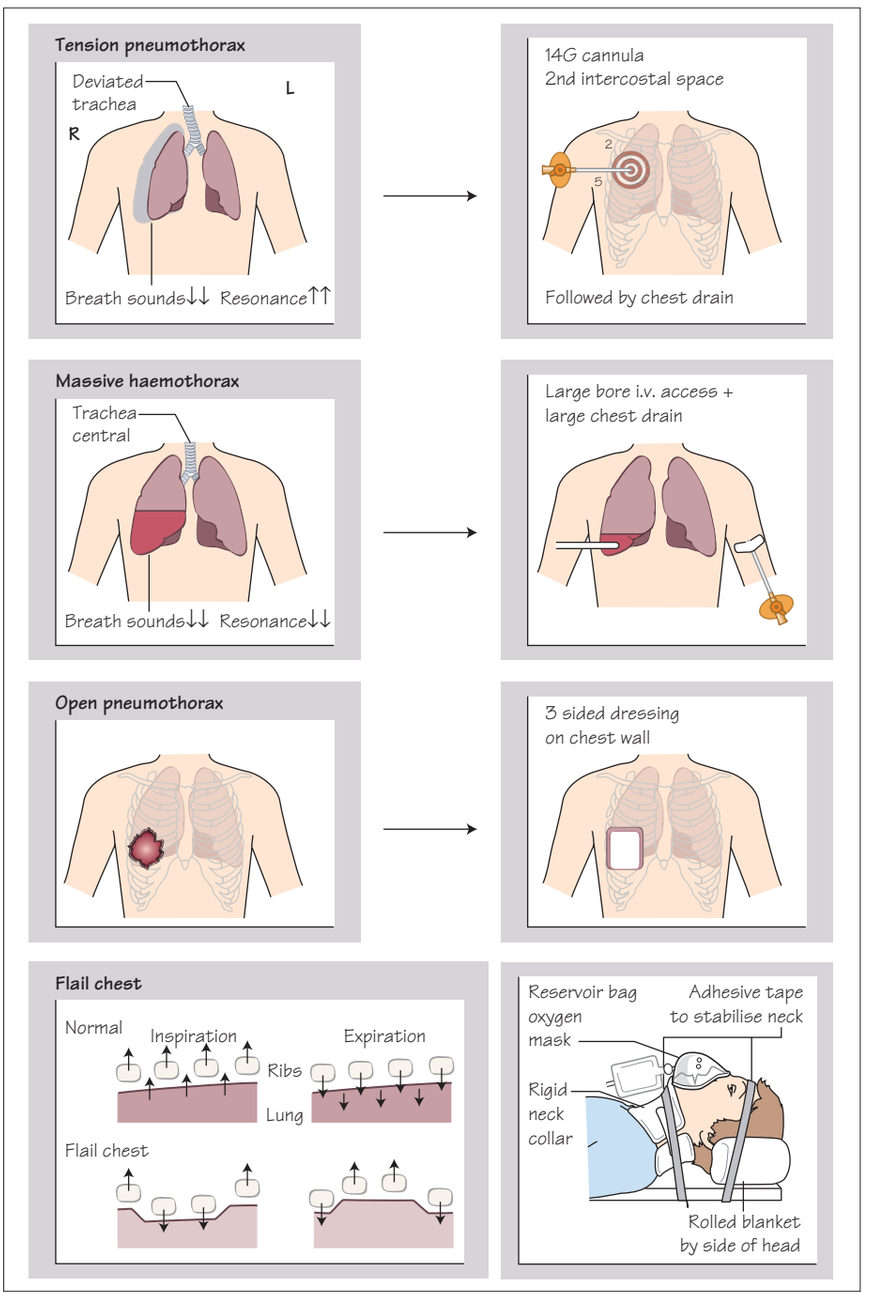
CO_2 low = respiratory alkalosis

Respiratory alkalosis is usually due to anxiety-related hyperventilation although marked hypoxia, e.g. from pulmonary embolus, may also cause this.

CO_2 high = metabolic alkalosis

Metabolic alkalosis is usually caused by loss of acid and/or dehydration, e.g. diarrhoea and vomiting.

8 Trauma: primary survey



Trauma care has been much improved with systematic protocols that enable effective prioritisation of treatment. The first time one sees a trauma patient arriving in the Emergency Department can be confusing and intimidating as there are many things going on simultaneously.

Treatment priority

The *ABC* order of treatment reflects the relative importance of the different things that can go wrong. Under most circumstances, **A**irway problems will kill the patient before **B**reathing problems, before **C**irculation problems.

Problems are treated as they are found. If a problem is found and treated, or the patient deteriorates, one starts again with A and works through B and C.

Cervical spine protection is given highest priority to avoid catastrophic spinal injury.

WARNING

Head injuries are the cause of death in 70% of trauma deaths. It is critical to avoid *hypoxia* and *hypotension*, which cause secondary injury in head-injured patients.

Trauma team

Major trauma is managed by a trauma team of up to five doctors and nurses, led by a senior doctor. Team members perform specific roles, e.g. airway management, procedures. The role of the leader is to stand back and have an overview rather than perform procedures, but in a smaller Emergency Department this is not always possible.

Penetrating vs non-penetrating trauma

Penetrating trauma, caused by knives and guns, is relatively rare in Europe and Australia, where most trauma is 'blunt', e.g. motor vehicle crashes, falls, crush injuries. In penetrating trauma (and ruptured abdominal aortic aneurysm or ectopic pregnancy), blood may be lost faster than it can be replaced: it is *essential* for ongoing bleeding to be controlled immediately. This may require wound compression, tourniquets to stop bleeding, or immediate surgery.

Ambulance transfer and handover

Trauma patients are prepared for transfer by placing them onto a spinal board with their head and neck immobilised. When the ambulance arrives, the trauma team listens carefully to their structured handover: DeMIST.

- Demographics: age, sex, background.
- Mechanism of injury.
- Injuries sustained.
- Signs and symptoms.
- Treatment given.

The key points should be summarised back by the team leader to confirm understanding and prevent errors.

A: Airway and cervical spine protection

If a patient is not talking, check for stridor, or obstruction with blood/teeth/food, and normal chest wall movement with breathing. The tongue can fall back and cause an obstructed airway in a supine, unconscious patient.

Interventions

- Oxygen: 15 L/min using a mask with a reservoir bag.
- Inspect mouth and suction: only suck down side of mouth.
- Does the patient need a definitive airway? See Chapter 6.
- Cervical spine immobilisation (see opposite).

B: Breathing and ventilation

While there are many potential injuries to the chest, there are four breathing problems that are immediately life-threatening.

1 Tension pneumothorax

Tension pneumothorax occurs when a lung injury pumps air into the pleural space, building up pressure. Hypotension and respiratory difficulty are caused by high intrathoracic pressure and kinking of the great vessels. This causes distended neck veins, loss of breath sounds, and a trachea deviated *away from* the pneumothorax. *Tension pneumothorax is a clinical diagnosis, not a radiological one.*

Insert a large (16 or 14 G) intravenous cannula perpendicularly into the anterior chest wall in the second intercostal space in mid-clavicular line. A hiss of escaping air will be heard: leave in place and insert a chest drain as soon as possible.

2 Massive haemothorax

The patient may be in shock, and may have reduced air entry and dull percussion note, although this is often difficult to detect with the patient supine.

Ensure good intravenous access *before* placing a large bore (e.g. 32 Fr) chest drain, as draining the blood may precipitate bleeding, which may require resuscitation and immediate surgery.

3 Open pneumothorax

A large open chest wound gives a collapsed lung, loss of breath sounds and 'surgical emphysema' (air in the subcutaneous tissues that gives a crinkly feel).

Treat by applying an occlusive dressing over the wound that is secured on three sides only, thus acting as a one-way valve.

4 Flail chest

If multiple ribs are broken in more than one place, a segment of chest wall can move paradoxically, i.e. in the opposite direction to the rest of the chest during respirations. This markedly increases the work of breathing.

If a patient is becoming tired, intubation is necessary. If the flail segment is small, and respiratory function is good, analgesia can be achieved by an epidural or nerve blocks, but the patient should be closely monitored.

C: Circulation

Pulse and blood pressure are the key information – shock is described in Chapter 3.

Intravenous access should be a minimum of two 16 G cannulae. Blood should be sent to the laboratory for FBC (full blood count), U+E (urea and electrolytes), clotting, group and save, cross-matching, depending on clinical status of patient.

Stop the bleeding, warm the patient

Obvious bleeding sites should be compressed and dressed. Litres of blood can be lost into the pelvis or into femoral shaft fractures. A pelvic sling should be applied if there is a pelvic injury. Pelvic stability should never be assessed by compressing the pelvis. A traction splint, e.g., Thomas splint, stabilises and reduces pain and bleeding resulting from a femoral shaft fracture.

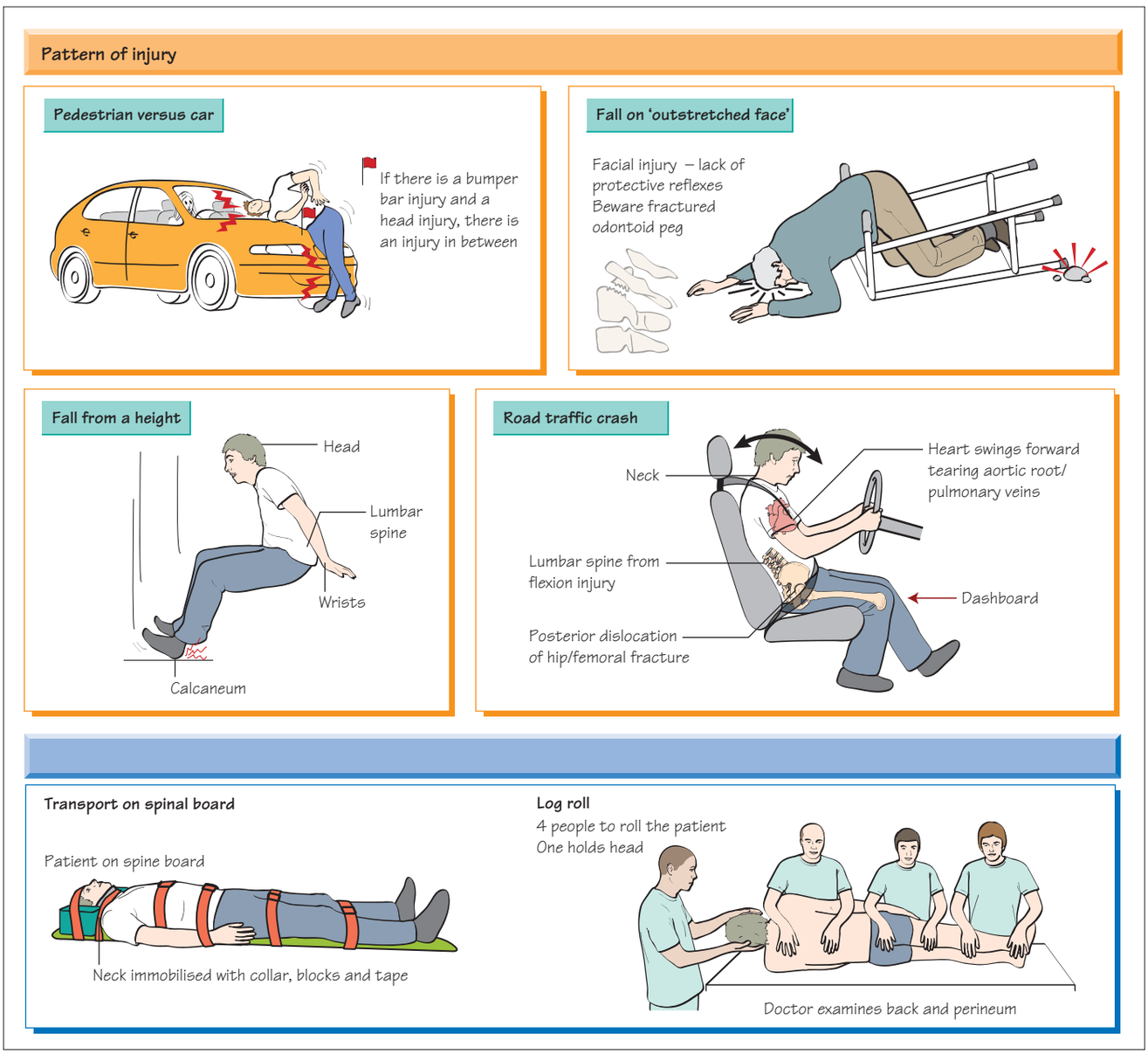
Pericardial tamponade

This produces similar signs to tension pneumothorax, with shock and distended neck veins, but *no tracheal deviation*. Heart sounds and ECG complex size may be reduced. Focused abdominal scanning in trauma (FAST) ultrasound scan should detect tamponade. Treatment depends on the nature of the trauma and clinical status of the patient, but is likely to require urgent thoracotomy.

Disability and neurological status

The Glasgow Coma Scale is described in Chapter 10.

9 Trauma: secondary survey



The secondary survey is a head-to-toe front and back, comprehensive review of the trauma patient to discover all injuries. This allows inpatient units to plan treatment. If an injury is missed at this stage it may not be picked up until it is too late to treat effectively, so thoroughness is essential. *This chapter will not cover limb injuries (Chapters 14–18), or head and neck injuries (Chapter 10).*

While the secondary survey proceeds, a number of other interventions take place:

- Analgesia, usually intravenous morphine, is humane, does not mask injuries and should be given early.
- Tetanus immunisation: give according to local protocol.
- Open fractures should be covered with a saline-soaked dressing and intravenous broad-spectrum antibiotics given immediately.
- A urinary catheter should be inserted if it is unlikely that the patient will be mobile within the next few hours. Urine should be dipstick tested, including βhCG in female patients.
- Arterial blood gases should be taken in severe trauma.

AMPLE

AMPLE is a useful mnemonic for the essential trauma history:
A – Allergies
M – Medication
P – Past medical history
L – Last ate/drank
E – Environment (other potential dangers, e.g. carbon monoxide poisoning, trauma in a burns patient).

Log roll, perineal injury

The log roll is a technique to turn the patient while ensuring that the spine remains immobilised. It is used to examine the patient's back and perineum. A rectal examination is performed to look for blood, lack of tone/sensation or high-riding prostate which indicate bowel injury, spinal cord injury or urethral injury, respectively. Priapism occurs in spinal injury, while blood at the penile meatus implies urethral injury and the need for urological advice before catheterisation.

Patterns of injury

Knowledge about likely patterns of injury is helpful: discovery of one injury should prompt a search for related injuries.

- *Fall from a height*: the calcaneus is often broken, together with the wrists, and the lumbar spine from forced flexion.
- *Deceleration injury*: sudden flexion of the spine, as occurs in a motor vehicle collision, tends to cause injury at the junctions between the flexible parts of the spine (lumbar, cervical) and the more rigid thoracic spine. The vertebrae most often injured are C5/6 and T12/L1. Seatbelt injuries may cause injury to the small bowel or pancreas by squashing these against the vertebrae, particularly with lap-only seatbelts.
- *Abdominal trauma*: the spleen is immobile and sits just below the ribs, where it is vulnerable to damage. Splenic injury may initially be asymptomatic, followed by rupture days later, and so any left upper quadrant tenderness necessitates a CT. Liver lacerations may bleed extensively, as there are large vessels in the liver. The mobility of the bowel generally protects it from blunt injury, while the kidneys are quite well cushioned by soft tissue.
- *Spinal trauma*: if one spinal fracture is identified, there is a high probability that there is another fracture elsewhere, so the whole spinal column should be imaged with CT.
- *Penetrating injury*: for firearm injuries, aside from entry and exit wounds, the damage caused is proportional to the density of the tissue traversed and the energy (mass, velocity) of the projectile. This may be further complicated by cavitation, tumbling, internal deflection and secondary injury from fragments of bone.
- *Knife wounds* can be difficult to assess in the Emergency Department, particularly if the depth exceeds the width. Knowledge of the deep structures is essential to be able to recognise and predict complications, and this should be performed by someone with the necessary experience.

Imaging

Imaging is an integral part of the secondary survey, and occurs in parallel with the clinical examination and treatment.

- Bedside *ultrasound* scanning (Chapters 10 and11) is used to identify causes of shock and patients who need urgent surgery rather than more examination/imaging.
- *Chest X-ray* concerns breathing and is therefore the most important plain X-ray film, and the first to be done.
- *Pelvis X-ray* relates to circulation: fractures of the pelvis may tear sacro-iliac veins, causing catastrophic bleeding. Pressure on the pelvis to 'test stability' may cause this bleeding, so must be avoided; however a pelvic X-ray is 60 CXRs, so should not be performed on patients who have minimal trauma.
- *Computed tomography* of head/neck/chest/abdomen and pelvis is now the 'gold standard' for severe trauma as it minimises the risk of missing injuries, and is quick to perform, at the cost of a substantial amount of radiation (1000 CXRs). The major contraindication to a CT scan is the unstable trauma patient, who needs urgent theatre to control bleeding, not a CT scan.
- *Cervical spine X-ray* is no longer routinely performed early in the assessment, providing the neck is immobilised. Plain X-ray of the cervical spine is not particularly sensitive at identifying injuries, and therefore any patient with a moderate or high chance of neck injury should have CT. This includes all patients requiring a CT scan of the brain, patients with a dangerous mechanism of injury (fall > 5 m, diving injury, rollover road traffic collision) or physiological abnormality (altered neurology).
- *Interventional radiology*: If there is sustained bleeding from poorly accessible sites (e.g. pelvis), embolisation of vessels may be life-saving.

Fluid resuscitation

Patients with significant injuries should have two large-bore intravenous cannulae inserted, but aggressive pursuit of 'normal' measures of pulse and blood pressure may be counterproductive (Chapter 3). For patients with head injury see Chapters 10 and 11.

As described in Chapter 3, hypothermia (<35°C) or severe acidosis (pH < 7.20) will reduce in-vivo clotting function to a fraction of normal, and together they are a lethal combination. An important intervention in the resuscitation stage is to keep the patient warm, using a warm air blanket, warmed intravenous fluids and warmed humidified oxygen.

There is debate over the optimum transfusion strategy, but recent military experience suggests early use of blood and blood products produces better outcomes.

Surgical resuscitation

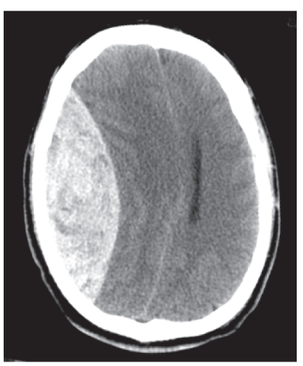
In cases where there is ongoing bleeding that cannot be controlled, the abdomen and/or chest are opened and the bleeding areas packed in 'damage control surgery'. Major fractures are immobilised with external fixators. The patient is then transferred to the intensive care unit (ICU) for stabilisation prior to further surgery at a later time.

10 Major head and neck injury

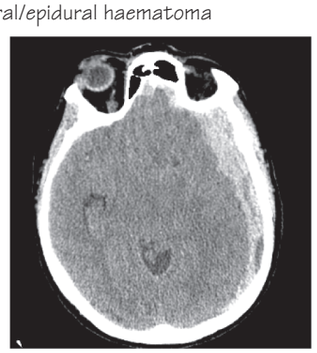
Glasgow Coma Scale

	Motor	Verbal	Eyes open
1	Nil	Nil	Nil
2	Extension	Incomprehensible	To pain
3	Flexion	Inappropriate	To speech
4	Withdraws	Confused	Normal
5	Localises	Normal	—
6	Normal	—	—

CT brain



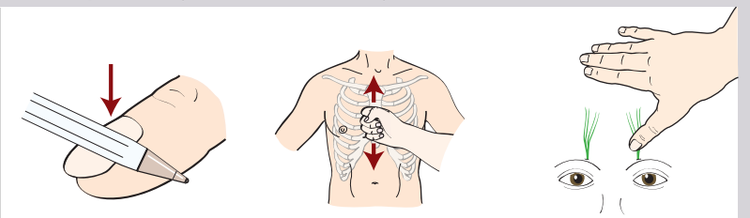
Extradural/epidural haematoma



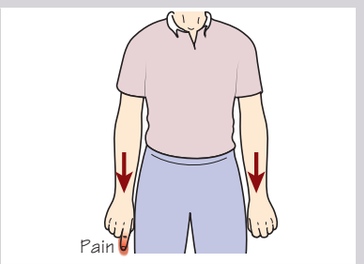
Subdural haematoma

Motor function : The most important component of the GCS

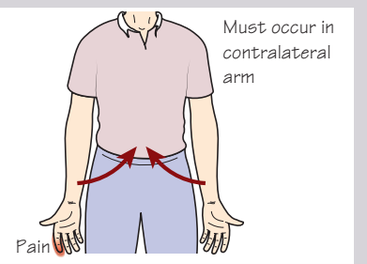
Stimulating motor response in semi-conscious patients



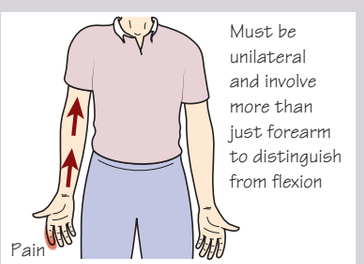
Motor = 2: Extension



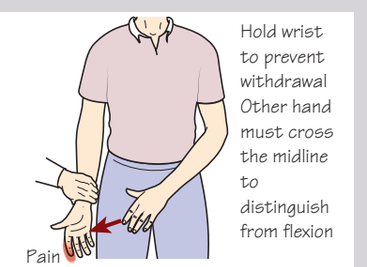
Motor = 3: Flexion



Motor = 4: Withdrawal



Motor = 5: Localises



This chapter covers patients who have suffered a significant head (Glasgow Coma Scale; GCS < 13) and/or neck injury, often as part of multi-system trauma (Chapters 8 and 9). Of trauma-related deaths, 70% are from head injury, and many of these deaths are preventable.

- *Primary brain injury* occurs at the moment of trauma. Prevention is the only way to minimise primary injury, which is why collection of injury data is an integral part of Emergency Medicine. Seatbelts, helmets, car and road design all prevent primary brain injury, as does road safety enforcement (speeding, drink driving).
- *Secondary brain injury* occurs after trauma, and may be preventable by expert medical care. The most common preventable conditions that cause secondary brain injury are *hypoxia* and *hypotension*.
- *Cervical spine injury*: in the context of a major head injury, a cervical spine injury is assumed until proved otherwise. All patients should arrive at the Emergency Department immobilised on a spinal board with a cervical collar and supports.

Aside from an AMPLE history (Chapter 9), information from witnesses may be available from the ambulance crew. Periods of loss of consciousness and amnesia before or after the event are helpful to assess neurological damage.

Airway, breathing and cervical spine

The patient is immobilised on a spinal board, with a rigid cervical collar, together with blocks and tape. Immobilisation is painful after about 20 minutes, and pressure sores can develop in patients with reduced sensation and/or mobility. The patient should be

safely removed from the board as soon as possible, usually as part of the log roll in the secondary survey.

If a patient does need to be intubated and ventilated (Chapter 6), it is very important to establish objective neurological status (see Disability, below) *before intubation*, as it is impossible afterwards due to the muscle paralysis necessary for ventilation.

Circulation

Having established that the blood will be oxygenated, the next challenge is to ensure that enough blood is perfusing the brain. This is dictated by:

> cerebral perfusion pressure (CPP) =
> mean arterial pressure (MAP) – intracranial pressure (ICP)

The brain's normal self-regulation of CPP is impaired in brain injury: it is critical that MAP does not fall below 80 mmHg. CPP can be maintained by increasing MAP or reducing ICP. MAP can be increased by giving intravenous fluids and inotropes (e.g. adrenaline) according to the CVP and MAP. ICP can be reduced by reducing venous pressure: avoid excessive intravenous fluid and elevate the head of the bed by 30°.

Disability

This refers to the brief structured assessment of functional neurological impairment as a result of the head injury.

Glasgow Coma Scale

The Glasgow Coma Scale (GCS) was devised in the 1970s before the advent of CT to predict the need for neurosurgical intervention. The motor component is the most important, but also the most difficult to assess. If the GCS is not assessed using optimal stimulation, poor-quality information will be collected, resulting in poor decisions. Pressing on a fingernail with a pen, and firm sternal pressure, are commonly used; if a spinal injury is possible, pressure on the supraorbital nerve in the supraorbital notch is effective.

Pupil size and reactivity

The pupils' size and reactions give useful information about the patient's neurological status, assuming that no drugs that influence the pupil size (e.g. atropine, adrenaline) have been given.

- If the pupils are of normal diameter (3–5 mm) and reactive, this suggests underlying normal function, and is associated with a good outcome.
- If one pupil is fixed and dilated, this may indicate that the brain on the same side is under increased pressure, stretching the IIIrd nerve.
- If both pupils are small, this suggests either opiate overdose or brainstem injury.
- Having both pupils fixed and dilated is associated with a poor outcome, unless caused by drugs (e.g. atropine, adrenaline) or local eye injury.

Focal limb movement deficit

If limb movement differs from one side to the other (excluding direct reasons e.g. broken arm) consider whether there may be a spinal or brain injury.

Investigations

Any necessary investigations are integrated into the primary and secondary trauma survey as described in Chapters 8 and 9.

Bedside investigations

- Blood glucose monitoring must be early and then regularly repeated in all cases of neurological impairment in the Emergency Department.

Laboratory investigations

- An alcohol level is only useful if negative. If positive it does not rule out the need for imaging. Some countries have mandatory blood testing for all road trauma patients.
- FBC/U + E/clotting profile/blood group and hold.
- ABGs/lactate ensure accurate assessment of oxygenation, ventilation and shock.

Imaging

- CT brain and neck.
- MRI is not indicated in the initial assessment, but may be useful to assess spinal cord injury.

Management

After stabilisation and CT scan, a decision needs to be made about what further care is necessary. The process to achieve this depends on local policy, but there are essentially four groups of patients:

Urgent neurosurgery

This small but important group comprises patients with extradural (epidural), intracerebral or posterior fossa bleeding. Some subdural bleeds, e.g. those resulting in marked midline shift, may also require surgery.

Intensive care

These patients need a period of ventilation in an ICU, which has facilities for ICP monitoring using a bolt drilled through the skull, and that offers ready access to neurosurgery, should this become necessary.

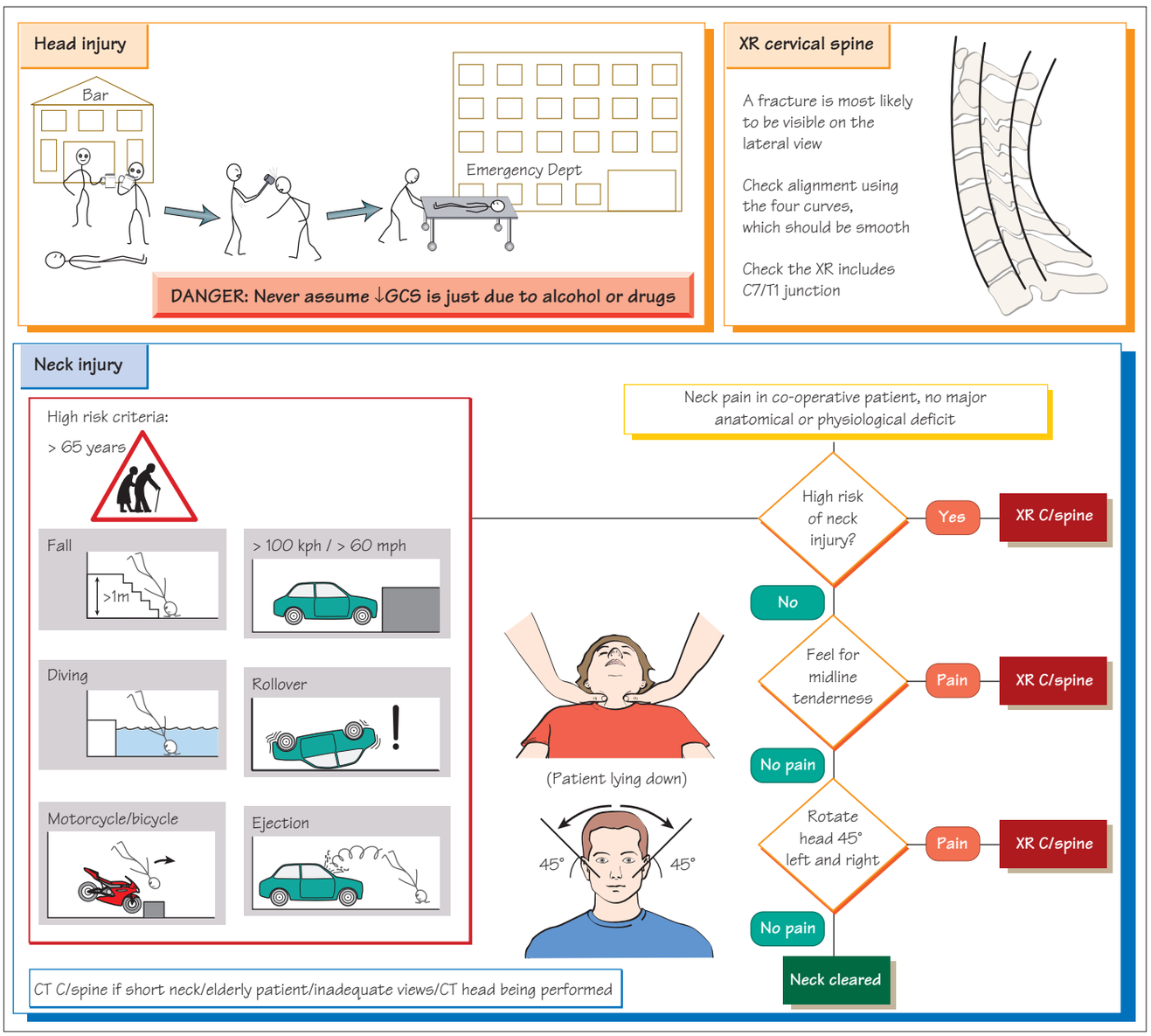
Ward care

Ward care is for patients who need close neurological monitoring on a normal ward with the ability to have an urgent medical review should their condition deteriorate. The Emergency Department observation ward is sometimes used for this group of patients. Post-injury care including follow-up is advisable, as even patients with apparent normal function after head injury can have significant problems (e.g. poor concentration, emotional lability) that are helped by psychological support.

Catastrophic head injury

If the CT shows no chance of survival, this must be explained to the patient's relatives in sympathetic but unambiguous terms. Organ donation should be sought by a member of staff experienced at explaining this, as the opportunity to donate organs is usually very much appreciated in the long term.

11 Minor head and neck injury



Minor head and neck injuries are extremely common reasons to attend the Emergency Department. Within this group of patients there is a very small number who have sustained serious damage: the challenge is to accurately and efficiently identify these. This task is complicated by the fact that alcohol is involved in more than half of these cases. Minor head injury is defined as Glasgow Coma Scale (GCS) 13 or above, and may be associated with a period of loss of consciousness (LOC), and/or amnesia.

Guidelines use clinical features to identify low-risk patients who can safely be discharged and high-risk patients who need further investigation.

Imaging

- Skull radiography (5 CXR) is not helpful as it cannot exclude significant brain injury: only CT brain (100 CXR) can do this.
- Radiography of cervical spine (5 CXR) is appropriate in patients with a low to moderate probability of injury. X-ray

of cervical spine comprises three views: AP, lateral and (odontoid) peg.
• In patients with a high likelihood of neck injury *or* when the X-ray of cervical spine is not of adequate diagnostic quality *or* if a CT brain scan is indicated as well, then a CT cervical spine scan (100 CXR) is preferable.

Head injury: clinical assessment

This should include details about mechanism of injury, previous medical history, loss of consciousness and symptoms since. The key points to establish are:
• Mechanism of injury: pedestrian or cyclist vs vehicle, or ejected from vehicle, or fall >1 metre.
• Age ≥65 years.
• Vomiting >1 episode.
• Pre-traumatic amnesia >30 minutes.
• Seizure.
• Warfarin or coagulopathy.
• GCS < 15 after 2 hours in Emergency Department.
• Suspected skull fracture (open or depressed or skull base).
• Focal neurological deficit.
If any of the above factors is present, it is likely that the patient will need a CT brain scan (100 CXR).

Neck injury: clinical assessment

History: high-risk factors

• GCS <15, unstable physiologically.
• Age >65 years.
• Prior neck problems, neurology.
• Fall >1 metre.
• Axial load to head, e.g. diving, rollover crash.
• Motor vehicle crash involving high speed, ejection from vehicle, bicycle, motorcycle or recreational vehicle.
If a patient has neck pain and any of these features, arrange imaging.

Examination

Look Look for fixed flexion deformity of the neck.
Feel While a clinician stabilises the head, take the collar off and feel for midline tenderness over the spinous processes.

Tenderness over the trapezius muscles is common but does not necessitate imaging. If either Look or Feel is abnormal, arrange imaging, otherwise test movement:

Move Ask the patient to rotate their head 45° left and right.

If this is possible without pain and the above tests have been performed by a doctor with the appropriate training and experience, the neck is 'cleared'.
If any of the findings are abnormal, arrange imaging.

Other investigations

• Investigations indicated as per Chapters 8 and 9.
• Blood glucose.
• Alcohol testing, whether breath or blood, is only useful if it is negative. If positive, it is dangerous to assume that all symptoms are due to the alcohol.

Common diagnoses

Concussion: mild traumatic brain injury

After ruling out significant brain injury, the patient may be discharged to the care of another adult with written head injury instructions. These should express clearly the reasons to return to the Emergency Department, e.g. vomiting or drowsiness.

The patient should be warned about common symptoms following a mild head injury (e.g. poor concentration, labile mood): psychological follow-up may be helpful.

Acute neck sprain

Patients should be warned that pain and stiffness is likely to be worse the following day and that it is important to use sufficient analgesia, e.g. NSAID ± codeine, to keep the neck mobile. The term 'whiplash' is best avoided as it has medicolegal implications. It is interesting that in countries without a compensation culture, acute neck sprains do not cause long-term disability.

Soft foam collars discourage neck movement, preventing recovery and encouraging psychological dependence, so should not be used. Semi-rigid collars (e.g. Philadelphia) are sometimes used for patients with a stable neck injury on expert advice.

Diagnoses not to miss

Reason for fall or injury

Elderly patients who present with a fall may have been on the floor for a prolonged period: look for hypothermia, pressure sores, rhabdomyolysis (Chapter 29). Think about possible causes (e.g. urinary tract infection, postural hypotension or arrhythmia), and keep an open mind about possible elder abuse or domestic violence.

Occult cervical spine fracture

Elderly patients with facial injuries may have fallen so fast they have not been able to protect their face, and therefore are at high risk of cervical spine fractures, especially of the odontoid peg. Have a low threshold for requesting CT, as plain radiographs are usually uninterpretable.

Extradural (epidural) haematoma

A fracture of the temporal bone overlying the middle meningeal artery may cause a large bleed. The classical presentation is of deterioration following a lucid interval; if diagnosis and surgery are rapid, a good outcome is common.

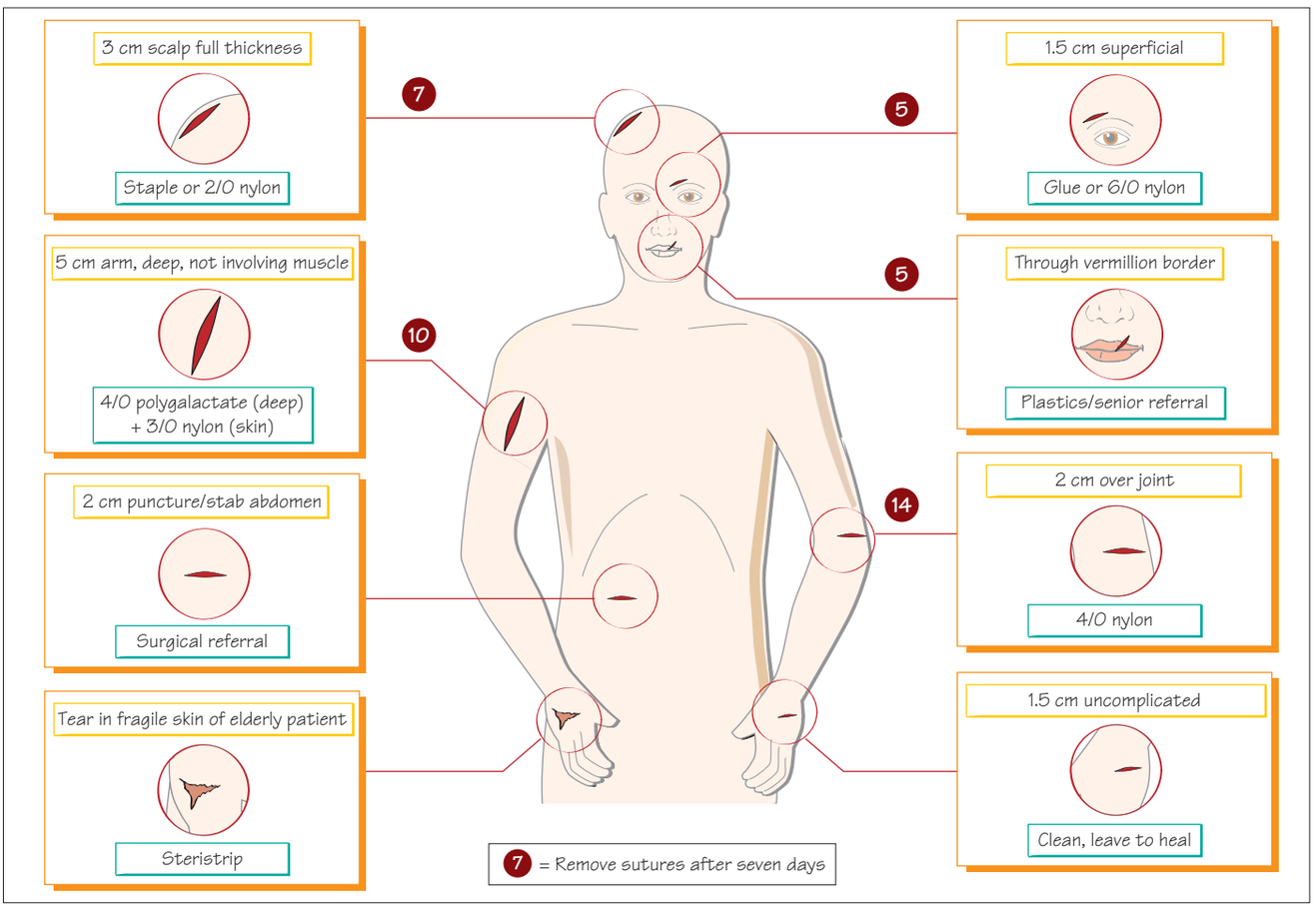
Subdural haematoma

Patients at high risk of subdural haematoma (SDH) include the elderly with recurrent falls, alcoholics and those on anticoagulants. SDH may present following an acute injury, or as a chronic deterioration, and often has a poor prognosis whether surgery is performed or not, due to the underlying conditions.

Cervical spine fracture

C2 and C5/6 injuries are most common. Document and monitor neurological and respiratory function carefully. Insertion of a catheter, pressure area care, and correction of spinal shock using intravenous fluids are essential basic treatments.

12 Wounds



Wounds often involve visible areas, the face and upper limb, where cosmetic as well as functional outcome is important. Wounds are generally *incised* – caused by sharp objects, or *lacerated* – caused by blunt force. An *abrasion* is a wound where the upper layers of the skin are removed, but there is no surface break. A wound where the depth exceeds the width or length is described as a *puncture* wound.

Resuscitation

Bleeding should be stopped using direct pressure or tourniquets: blind clamping should be avoided. Bleeding from scalp wounds can be controlled by full-thickness sutures using 2/0 nylon.

Any wound near a fracture is assumed to communicate with it, and should be covered by a clean saline-soaked dressing and antibiotics administered *immediately*.

Toxic bites (e.g. from snakes, spiders) should be treated according to local protocols. Snake bites can be painless, and venom may cause paralysis or catastrophic anticoagulation. Antivenom derived from animal serum is quite toxic in itself, so should not be given unless toxicity is certain.

History

Medical notes from the Emergency Department are used to write legal reports: avoid words like 'cut' or 'stab wound' unless you are an expert. Unless you witnessed the injury, use 'alleged' and quote the patient's own words wherever possible, e.g. 'Alleged assault – patient says was "hit with bottle outside a nightclub".' Accurate descriptions with *measurements*, diagrams and photographs are very helpful. Occupation/hobbies, hand dominance, allergies and tetanus status should be recorded.

Examination

Look Assess skin loss and viability, contamination, cut muscle or crush injury.

Feel Test motor and sensation (before local anaesthetic infiltration).

Move Test muscle and tendon and muscle function while observing the wound. If the wound is very painful, this is best done after infiltration of local anaesthetic.

Foreign body

Examination cannot reliably exclude foreign bodies (FBs), which are common in motor vehicle accidents, puncture wounds and clenched fist injuries. Imaging is not necessary for most wounds; X-ray if the FB is radio-opaque, i.e. metal or most glass. Ultrasound is useful, but is operator dependent.

Management

Assess the wound

Is the wound complex or dirty?

- Complex: the wound is large, involves crushed tissue, FBs, injection under pressure or extends into deep structures like muscles, tendons or joints. These wounds have a high risk of infection or compartment syndrome (Chapter 15).
- Dirty: if there is obvious contamination or the wounds is >6 hours old. Patients with reduced immune function (e.g. diabetes, steroids) are at increased risk of infection.

Consider the reason for the wound (e.g. fall, domestic violence), and any other potential injuries.

Is it safe to close the wound in the Emergency Department?

All complex or obviously contaminated wounds should be referred for exploration and closure in an operating theatre.

The options for wound closure are:

- Primary closure – close the wound immediately. This gives the neatest scar, but risks infection by trapping bacteria within the wound.
- Delayed primary closure – clean, give antibiotics for 48 hours, then close. This reduces the risk of infection in dirty wounds.
- Secondary healing – allow the wound to heal on its own. It heals more slowly, and there is more risk of scarring.

Analgesia

Local anaesthetic (LA) is injected around wounds to allow thorough cleaning and suturing (Chapter 5). Lidocaine1% ± adrenaline (epinephrine) 1 : 100 000 is the most commonly used LA. Pain on LA injection is reduced by using a small needle, warming the LA and injecting slowly through wound edges. For wounds to be glued, use *topical* lidocaine with adrenaline applied onto a piece of gauze, cover and leave for 20 minutes.

Clean the wound

A tourniquet can be used to ensure a bloodless field. Hair near a wound may need to be cut or shaved, but not eyebrows or eyelashes. Use a syringe and 19 G needle and drinking-quality water to irrigate the wound under pressure: guard against splashback by wearing a mask and eyewear. Remove non-viable tissue and ensure embedded grit is removed to prevent tattooing.

Close the wound

Sutures

Interrupted non-absorbable nylon sutures allow drainage and minimise tissue tension and ischaemia. If there are potential spaces within the wound where a haematoma could form, or there would be tension on the skin sutures, deep absorbable sutures (e.g. polygalactin/polyglycolic acid) are used.

Timing of suture removal is a balance between scarring (shorter time better) and wound strength (longer time better). For facial wounds, 5 days is best; for wounds over extensor surfaces of joints, 14 days.

Glue, adhesive strips, staples, dressings

Tissue glue (similar to domestic Superglue®) or adhesive strips are effective for simple wounds, providing the wound edges are easily opposed without tension. The effectiveness of adhesive strips is increased by pre-coating the skin with Friar's Balsam. Staples are a fast way of closing linear wounds that do not need a perfect cosmetic result, especially scalp, limb or self-harm wounds.

If the wound is dry, a clear vapour permeable dressing allows inspection. If there are exudates, a dressing that is absorbent yet non-adherent is preferable.

Tetanus, antibiotic prophylaxis

Wounds that are dirty or complex are prone to tetanus. If a patient has had full tetanus immunisation, further boosters are not necessary unless the wound is heavily contaminated, e.g. with soil), in which case tetanus immunoglobulin is given.

Antibiotics are not a substitute for adequate wound cleaning. Antibiotics are indicated in wounds at high risk of infection or with established infection: flucloxacillin covers *Staphylococcus* and *Streptococcus*.

Special situations

Bites

Bite wounds from humans or animals are prone to infection due to the combination of crushed tissue and inoculation with saliva. Wounds should be cleaned and 5 days of broad-spectrum antibiotics (e.g. co-amoxiclav) prescribed.

Needlestick

Wounds that risk hepatitis or HIV transmission should be thoroughly cleaned. Blood should be taken and local policies consulted about follow-up.

Pre-tibial lacerations

Elderly patients can tear the thin skin over the anterior tibia. The skin should be stretched to cover as large an area as possible and early plastic surgery review arranged.

Facial wounds

Facial wounds are closed up to 24 hours after injury as cosmesis is important, and the excellent blood supply provides some protection against infection. Antibiotic ointment can be used instead of systemic antibiotics.

13 Burns

Don't miss

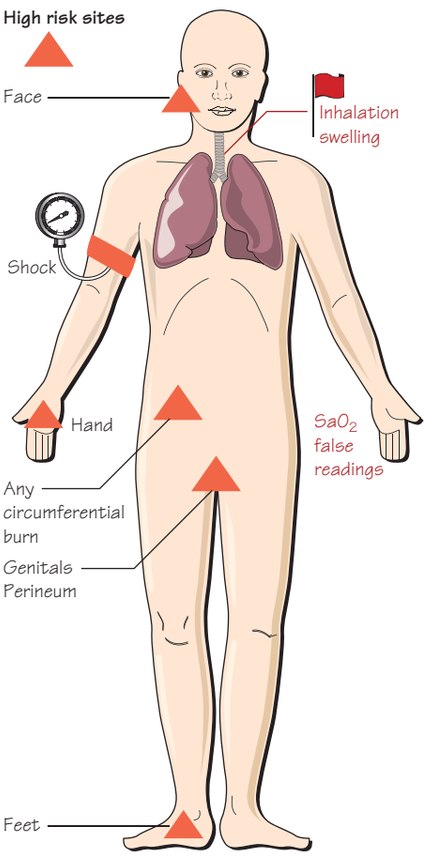
Airway
Airway burns
– Singed eyebrows, eyelashes, nostril hairs, carbon in sputum
– Swelling (delayed) → prophylactic intubation

Breathing
Confined spaces
– Carbon monoxide (CO) – impairs oxygen tranport, headache + drowsy
– Cyanide (CN) – cellular poison, lactic acidosis, shock

Circulation
Shock ΔΔ
- hypovolaemia
- cyanide
- trauma
- blast injury

Disability
Assess GCS, limb function

Exposure
Beware hypothermia – cool the burn, but warm the patient



High risk sites

Compromised ABCDE

Trauma/ blast injuries

Patient co-morbidities
Age > 60
Diabetes
Heart failure
Lung disease
Immunosuppression
Pregnant

Electrical burns
Chemical burns

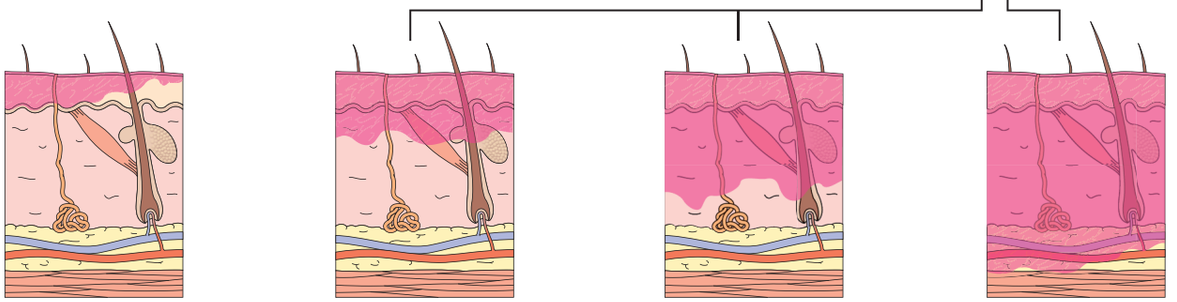
Social reasons
Logistics
Analgesia

CONSULT/TRANSFER TO BURNS UNIT

> 10% Total body surface area (TBSA)

Area of hand including fingers = 1%

Any area



Superficial epidermal
Red and painful, but not blistered

Partial thickness (superficial dermal)
Pale pink and painful with blistering

Partial thickness (deep dermal)
Dry or moist, blotchy and red, and may be painful or painless. There may be blisters

Full thickness
Dry, painless, no blisters, white/brown/black/leathery /waxy

Burns are a common problem, but the vast majority are relatively minor. Serious burns undergo initial assessment and resuscitation as per any trauma (Chapter 8).

History

Eye-witness accounts from ambulance personnel or witnesses are helpful in assessing the risk of associated problems such as trauma (jumping from buildings) or blast injuries. Patients trapped in confined spaces may have inhalation injuries or exposure to toxic gases such as carbon monoxide and cyanide.

AMPLE history (Chapter 9) and tetanus immunisation status are important.

Resuscitation

The patient

- *Airway:* look for signs of inhalation injury: carbon/soot in nostrils or mouth, singed eyebrows/eyelashes/nostril hairs, facial burns, and any orophayryngeal redness or swelling, change in voice or stridor.

Upper airway burns require urgent prophylactic intubation with an armoured (crushproof) endotracheal tube, as the face and oropharynx swell massively within a few hours.

- *Breathing:* oxygen via a reservoir mask for all patients.
- *Circulation:* two large-bore intravenous cannulae, through burned skin if necessary. Take blood for FBC, U+E, LFTs (liver function tests), CK (creatine kinase), group and save, clotting.

The burn

- *Stop the burning:* remove clothes, chemicals, initial cool water.
- *Treat the pain:* large doses of morphine are required for large burns. Pain is caused by air movement over the burn: clingfilm can help analgesia.
- *Secondary survey:* look for signs of other injuries, e.g. jumping from a height, blast injuries. Look for burned areas needing escharotomy (incision of the burn to prevent constriction of underlying tissues), e.g. limbs with circumferential burns, thorax.
- *Measurement of the burn:* size of patient's hand including fingers ≈1% TBSA (total body surface area).
- *Intravenous fluids:* see below.
- *Tetanus:* tetanus immunoglobulin should be given according to local protocol.
- *No antibiotics:* antibiotics should not be given routinely.
- *Investigations:* bedside urinalysis, ECG; blood tests as above, chest X-ray.
- *Transfer:* consider the need to contact a burns unit.
- *Keep warm:* although initial cooling may be helpful, burns patients are at risk of hypothermia. Wrap burned areas in clingfilm and place patient in dry, sterile sheets. Use warm air heating blanket and warmed humidified oxygen to minimise heat loss.

Complicated or non-thermal burns such as those from bitumen, chemicals, and electrical injuries require expert assessment, as the injury may be more extensive than is immediately apparent.

Intravenous fluids

Widespread capillary damage results in massive loss of intravascular fluid and protein. Give warmed crystalloids using the Parkland formula and monitor urine output.

Parkland formula for intravenous fluids in burns

2–4 mL × (% burn) × (bodyweight in kg) over 24 hours; half given in first 8 hours, the rest over 16 hours.

Minor burns

These can be dressed in many ways according to local practice including:

- Dry non-adherent dressings.
- Gel-based hydrocolloid dressings.
- Adhesive woven dressing direct to skin.
- Silver sulphasalazine cream (never on face).

Discharged patients need:

- A clear management plan including analgesia, community support and medical and nursing follow-up.
- In elderly patients consider an Occupational Therapy assessment to identify risks of further injury.

Carbon monoxide and cyanide poisoning

Carbon monoxide poisoning occurs in fires, but is also a mode of suicide; it is most commonly caused by inadequately ventilated heating. Symptoms are often non-specific, e.g. headache, nausea and vomiting that resolves when out of the house.

Carbon monoxide has a much higher (200×) affinity for haemoglobin than oxygen. Oxygen saturation monitors cannot differentiate between carboxyhaemoglobin (COHb) and oxyhaemoglobin, so give falsely normal readings. COHb is measured in blood gas analysers: venous blood may be used. Smokers and city dwellers may have COHb levels of up to 10%.

Treatment of COHb levels above 10% is by 100% oxygen, which reduces the half-life of COHb from 240 to 75 minutes. Hyperbaric oxygen has not been shown to give additional benefit.

Cyanides are formed by furniture burning in enclosed spaces. Diagnosis and treatment is described in Chapter 47.

Electrical burns (including lightning)

Electricity follows the path of least resistance, which tends to be nerves, blood vessels and, to a lesser extent, muscle. Skin burns – entry and exit points – may appear relatively minor, and the full extent of electrical burns may not be apparent immediately.

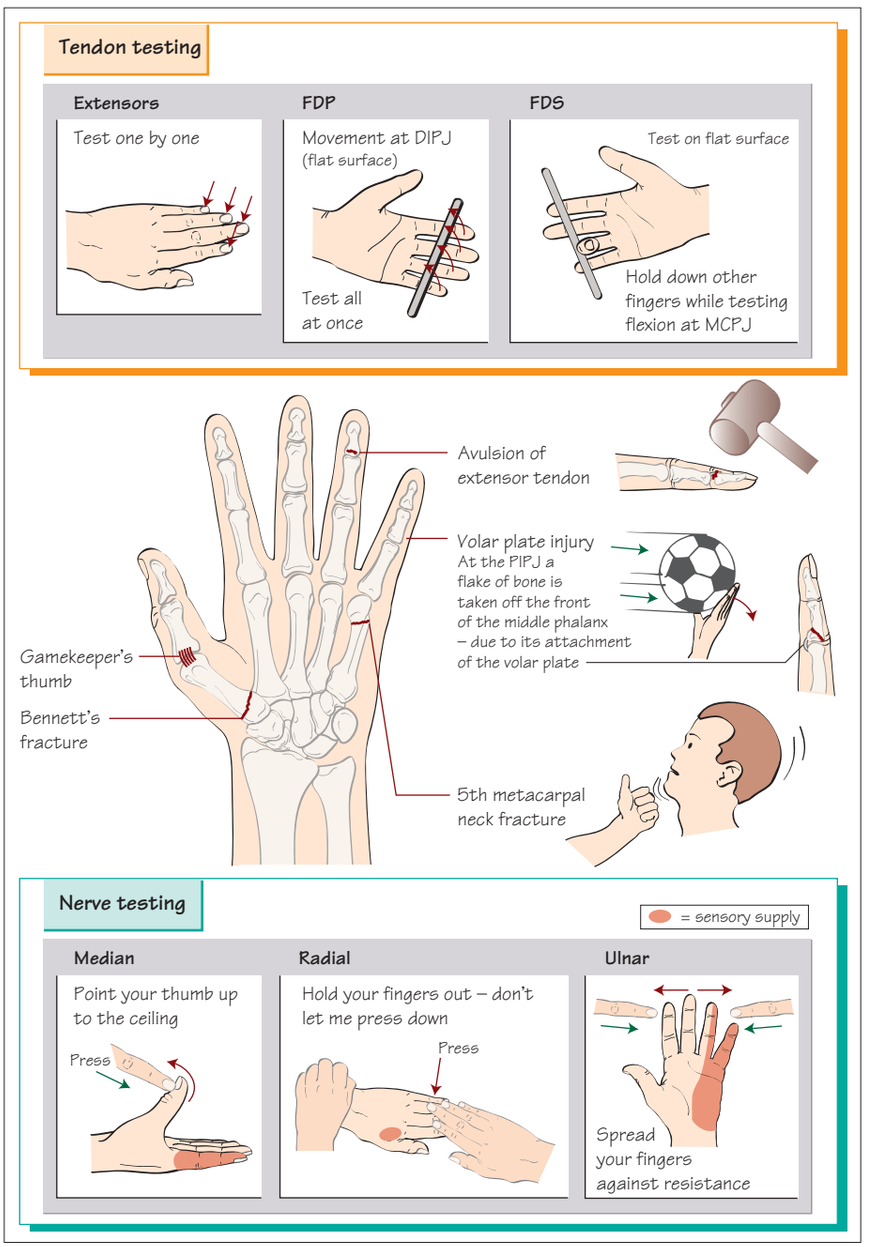
High-voltage (>600 V) injuries produce heat injury in underlying tissues and can cause cardiac damage and compartment syndrome. If a patient hit by lightning makes it to hospital, their chances of survival are good. Admit all patients with high-voltage burns, but patients exposed to domestic electricity (110–220 V AC) may be discharged if they have a normal ECG and no evidence of burns or systemic injury.

Chemical burns

Seek early expert advice with chemical burns. Both alkaline and acid substances can burn the skin: alkalis such as drain cleaner and cement are generally worse as acids tend to precipitate proteins, preventing deep burns. Water is used until pain resolves and pH is neutral.

Hydrofluoric acid burns deeply and painfully, binding calcium ions, causing local and systemic hypocalcaemia. Treatment is by local or systemic calcium gluconate, which can be given as a gel, injection into the wound, intravenously (like IVRA) or intra-arterially.

14 Hand injuries



Hand injuries are a common presentation to the Emergency Department, and the importance of good hand function in day-to-day life requires excellent results. The spectacular range of hand function relies on complex interplay between muscles, tendons, bones and ligaments, all of which may be damaged.

History and examination

Hand dominance, job and hobbies are essential parts of the history. The mechanism of injury may suggest a likely pattern of injury, e.g. a fifth metacarpal fracture following a fight. If there is no history of injury, consider infection (e.g. septic arthritis) or inflammation (e.g. rheumatoid arthritis).

It can be confusing to describe lesions in relation to the anatomical position, so the terms volar or palmar, and dorsal are used, rather than anterior and posterior. Similarly, radial and ulnar are used rather than medial or lateral. Names of digits (thumb/index/middle/ring/little) should be used, rather than numbers.

Look Look for swelling/bruising and compare hands. Check the skin over the knuckles for wounds: human 'bites' need treatment (Chapter 12).

Feel Feel the carpal and metacarpal bones and joints and the 'anatomical snuff box' (Chapter 15).

Move Ask the patient to make a fist: check the fingers are in line, pointing to the scaphoid.
- Check sensory and motor function. Two-point discrimination testing can reveal subtle sensory loss.
- Check the extensor and flexor tendon function.
- Test the thumb ligaments.
- Ask the patient to grip your index and middle fingers 'as tight as they can'.

Management

Immobilise and elevate

Neighbour/buddy strapping involves strapping an injured finger to an adjacent finger, providing protection against hyperextension while still allowing good function.

Volar slab: a strip of plaster on the palmar/volar side of the hand with the wrist in extension and the metacarpophalangeal joints (MCPJs) in flexion provides support and prevents contraction of tendons or muscles.

Multiple layers of elastic or plaster strapping around the thumb is called a thumb 'spica', and provides protection against abduction or hyperextension.

The compartments of the hand have little room to accommodate soft tissue swelling, so elevation in a sling is used to keep the hand above the heart. Rings should be removed.

The majority of hand injuries can be managed as outpatients or by GPs; however, open fractures, or those listed below under 'Do not miss', should be reviewed by the inpatient team.

Common injuries

Metacarpal neck fractures

Little or ring finger metacarpal neck fractures caused by punch injuries are quite stable. Angulation <30° gives a good functional outcome. If more angulated, the fracture may be reduced by flexing the MCPJ to 90° and pushing dorsally.

Fractures and dislocations of the phalanges

Dislocations and fractures with marked deformity should be reduced in the Emergency Department using N_2O/O_2 or a ring block. Mid-shaft or spiral fractures may be unstable due to fracture pattern or muscle action, and require operative fixation, particularly if there is any rotational deformity.

Hammer/mallet finger

Forced flexion of the extended distal phalanx pulls a flake of bone off the distal phalanx. Treat with a mallet splint to ensure the patient does not flex their distal phalanx at all for 6 weeks.

Thenar eminance sprain

The powerful muscles of the thenar eminence can be torn by forced abduction of the thumb – a common injury when falling on a slippery surface, e.g. skiing or skating. More serious injuries, e.g. Bennett's/scaphoid fractures must be excluded.

Nail and fingertip injuries

Injuries to the fingertip are common, and require X-ray to exclude bone injury, but rarely need operative treatment. If the nail is displaced, remove under ring block, trim, and use as a dressing for the nailbed. Nailbed injuries rarely need treatment.

Lacerations

Uncomplicated lacerations (that do not involve underlying structures) on the hand and digits less than 2 cm long do not require suturing, providing the wounds are not at high risk of infection (Chapter 12). Clean, dress and consider topical antibiotic ointment. Above this size, sutures are usually used. Ensure that distal neurovascular function is documented.

Fish-hook injury

Fish-hooks have a barb to prevent fish (or humans) from pulling the hook out. After anaesthetising the area, it may be necessary to advance the hook through the skin to cut off the barb and allow removal.

Diagnoses not to miss

Bennett's fracture

This is a fracture of the base of the thumb or first metacarpal bone, caused by thumb hyperextension. It is unstable and needs operative fixation.

Gamekeeper's thumb

Gamekeeper's thumb is a tear of the ulnar collateral ligament of the thumb at MCPJ level by forced abduction. Complete tears do not heal without surgery.

Tendon injuries

Tendon injuries are easy to miss unless the tendons are individually tested. Tendon lacerations can occur when an extensor, or less commonly, a flexor tendon hits a sharp object, particularly when the is running over a bony prominence. Complete tendon division requires operative repair.

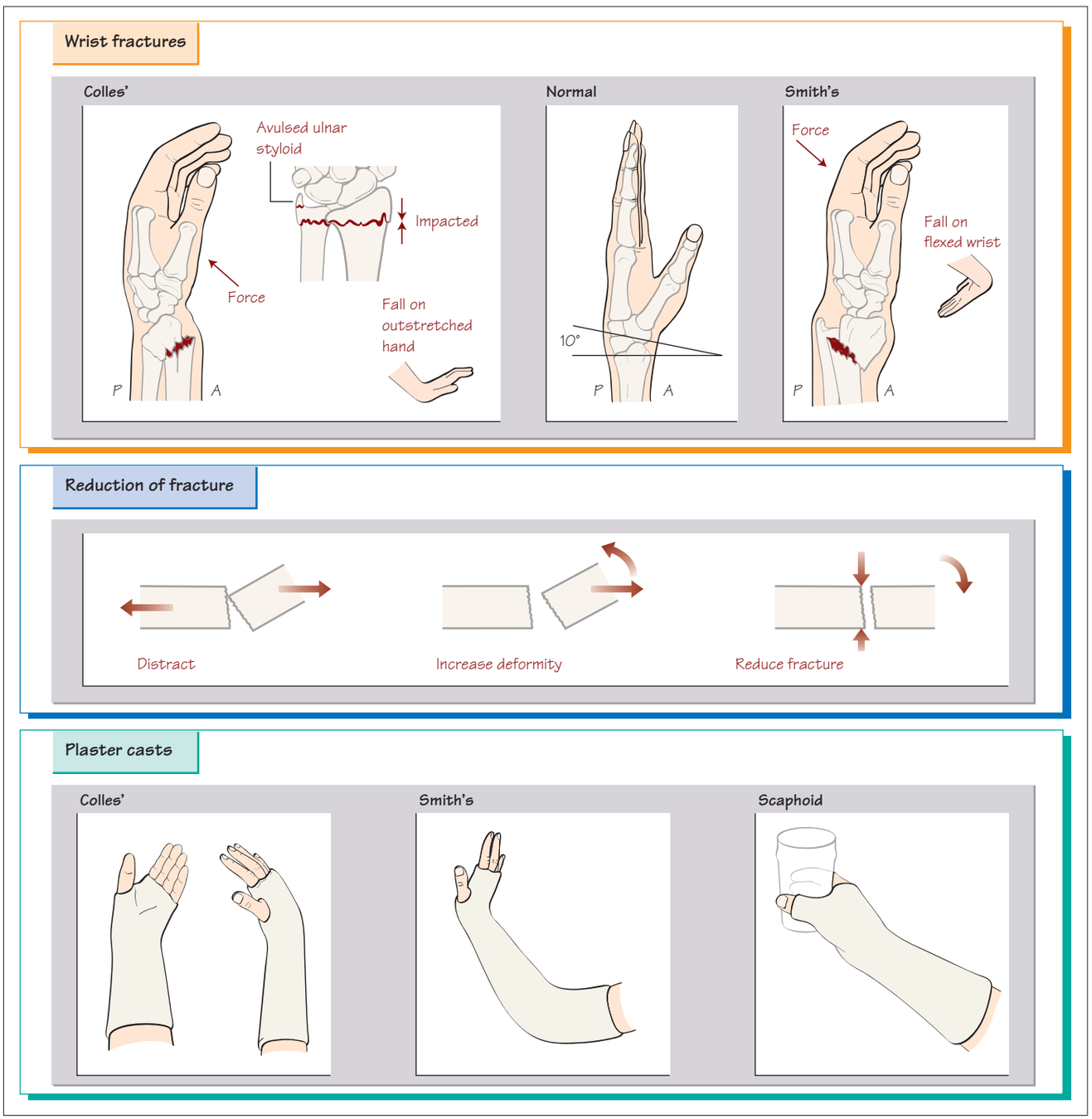
Tendon sheath infection

Tendons run in fibrous sheaths that protect and lubricate the tendon. If infection penetrates the sheath, it may track down the finger and into the hand. Such infections need urgent drainage, washout and antibiotic treatment.

Amputations

All amputations involving bone loss should be referred and reimplantation considered, especially for thumb and index fingers. The amputated part should be wrapped in clean cloth, and then put in a plastic bag inside an ice bath. The amputated part should not touch ice. Successful reimplantation of digits severed distal to the distal interphalangeal joint (DIPJ) is unlikely as the nerves, arteries and veins are too small.

15 Wrist and forearm injuries



Injuries to the wrist and forearm are common, often resulting from a fall onto an outstretched hand (FOOSH). It can be difficult to distinguish subtle fractures from soft tissue injury on clinical history and examination alone, so X-ray is usually necessary.

Fractures of normal bones imply *high-energy* injuries, whereas a fracture occurring as a result of a *low-energy* injury implies poor bone quality – a '*fragility fracture*', and the need to screen for osteoporosis.

In any injury affecting the upper limb, dominance (handedness) and occupation and hobbies must be recorded. If the injury is the result of a fall, consider further investigations (Chapter 30).

Examination: look, feel, move

Compare with opposite side, and look for swelling/bruising. A full range of elbow flexion, pronation and supination makes significant injury unlikely. The radial, median and ulnar nerve function in the hand should be checked (Chapter 14).

Management of fractures: principles

Pain should be controlled by splintage and analgesic drugs before imaging. Elevation of the arm in a sling reduces soft tissue swelling and pain. *If there is any evidence of neurovascular deficit or tenting of the skin by fractures, urgent reduction will be necessary.*

If there is a skin wound over a fracture, this makes it an *open fracture*. Antibiotics ± anti-tetanus treatment must be given *immediately*. The wound should be covered with a saline-soaked dressing, and the patient should go to theatre for debridement as soon as possible.

Plaster of Paris casts are used to hold the fracture in position while it heals. Rings should be removed before plaster is applied, as the digits will swell.

Compartment syndrome results from swelling of muscle within fascia compartments, e.g. of the forearm, leg or foot. If untreated, the muscle dies, resulting in untreatable ischaemic contracture. Patients should be warned about the symptoms: numbness, pain and cold digits. If a patient has pain on passive stretching of a muscle, compartment syndrome is likely; a palpable pulse *does not exclude* compartment syndrome. If elevation does not solve the problem, the plaster *must* be released, and a surgeon *must* review.

Common diagnoses

Colles' fracture

This is a fracture of the distal radius occurring in *osteoporotic* bone resulting from *low-energy* impact, e.g. FOOSH. The fracture may be impacted and the tip of the ulna is often avulsed. Dorsal angulation gives the wrist a 'dinner fork' appearance.

The fracture should be reduced in the Emergency Department using haematoma block and nitrous oxide, or intravenous regional anaesthesia (Chapter 5). Good wrist function depends on restoration of the length of the radius, and avoidance of steps in the articular surface. On the lateral X-ray view, the articular surface of the radius is normally 10° angulated towards the palm. This is often difficult to achieve by reduction, but a neutral (0°) position is satisfactory.

High-energy distal radius fracture

This injury occurs in *normal* bones as a result of *high-energy* impact, e.g. falling off a bicycle. In comparison to a Colles' fracture, there is more likely to be comminution (multiple bone fragments), more soft tissue damage and more pain: intravenous opiates are necessary.

To achieve good function, these fractures need excellent ('anatomical') reduction and may ultimately require operative fixation with plates or wires. A good reduction in the Emergency Department using intravenous regional analgesia or procedural sedation (Chapter 6) minimises soft tissue swelling and may avoid the need for further intervention.

Smith's fracture

A Smith's fracture is sometimes called a reverse Colles' fracture: it is a distal radius fracture, but instead of dorsal angulation, there is volar (palmar) angulation. However, Smith's fractures often occur in *normal* bone, when they are *high-energy* injuries. The structures on the volar (palmar) side of the wrist are at risk of injury, particularly the median nerve.

A Smith's fracture is inherently unstable, and almost always needs open reduction and internal fixation (ORIF) (e.g. with a plate and screws), although a good reduction in the Emergency Department is usually the first step in the management.

Diagnoses not to miss

Scaphoid fractures

The difficulty in diagnosis and the consequences of failure to diagnose make this fracture a frequent source of litigation. The history is usually FOOSH, and clinical signs are pain:

- In the 'anatomical snuffbox' between extensor pollicis longus (EPL) and abductor pollicis longus (APL).
- On axial thumb compression.
- On pressing over the scaphoid tubercle.

A patient with clinical signs of scaphoid injury requires a 'scaphoid view' X-ray. If this demonstrates a fracture, the joint should be immobilised as shown opposite.

Even if the X-ray does *not* show a fracture, the patient should still be immobilised in a splint or plaster cast, and sent home to have a definitive investigation, e.g. repeat X-ray, in 1 week, or CT or MR scan, to prove or refute the diagnosis of scaphoid fracture.

The reason for this approach is that 20% of patients have fractures of the scaphoid that are *not visible* on plain X-ray until at least 1 week after injury. If the scaphoid fracture is missed, avascular necrosis and non-union can result in early osteoarthritis and disabling stiffness of the wrist.

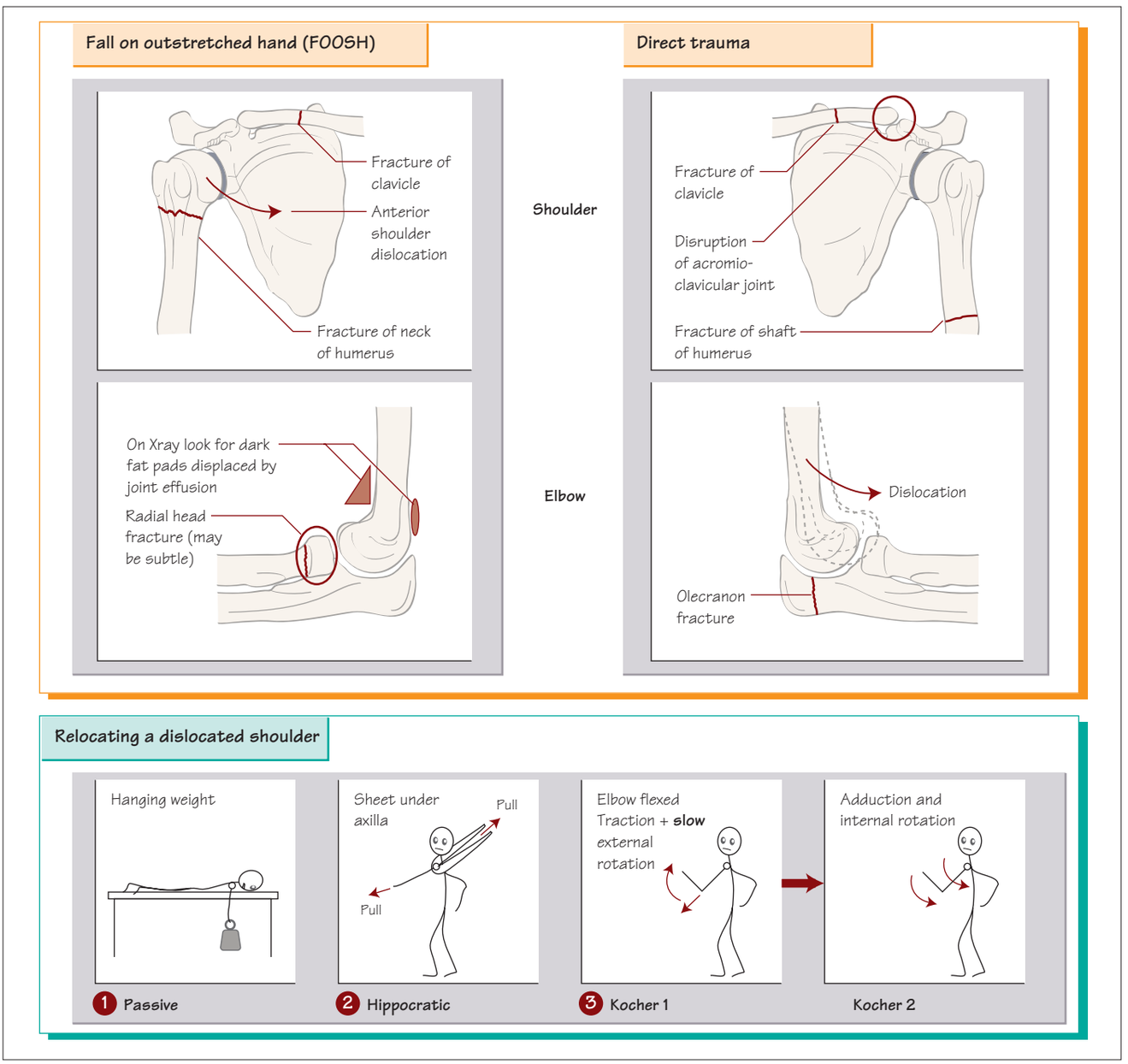
If you perform an X-ray of the scaphoid, it is illogical and therefore medicolegally indefensible not to follow up with a definitive investigation.

Fractures of shaft of radius and ulna

The X-ray must include the joint above and below to ensure that there is no dislocation. These fractures need ORIF.

- *Nightstick fracture*: a mid-shaft transverse fracture of the ulna, usually a 'defence injury' when the forearm is raised to protect the head ('nightstick' is the US term for a police truncheon). Consider possible causes, e.g. domestic violence.
- *Monteggia fracture*: fracture of *proximal* third of ulna and dislocation of the head of the radius at the elbow.
- *Galeazzi fracture*: fracture of *distal* third of radius with associated dislocation of distal radio-ulnar joint; rare.

16 Shoulder and elbow injuries



The extreme mobility of the shoulder joint, which relies on soft tissues – muscles, ligaments and cartilage – for stability, comes at a price. The shoulder is relatively unstable, and prone to stiffness if not used. There is a wide range of injury patterns, which change according to the age of the patient.

History

In any injury affecting the upper limb, dominance (handedness) and occupation and hobbies must be recorded. Shoulder pain can also be referred, e.g. cardiac, diaphragmatic, respiratory.

Injury is usually caused by either a fall onto outstretched hand (FOOSH) or direct trauma.

Examination

Look Compare with the other side.

Feel Start at the medial end of the clavicle and work laterally, feeling for tenderness of clavicle, coracoid process, acromioclavicular (AC) joint, humeral head and greater tuberosity.

Feel the olecranon, epicondyles and radial head. An elbow effusion may be felt below the radial head.

Move Limited or painful shoulder movement warrants an X-ray; very little movement will be possible with a dislocated shoulder or fracture. A full range of elbow extension makes fracture unlikely.

Neurovascular examination

Specific injuries and their corresponding neurovascular deficits are:

- Shoulder dislocation and fracture neck of humerus: test the axillary nerve – loss of sensation over lower deltoid area.
- Humeral shaft fractures – radial nerve.
- Medial epicondyle fracture – ulna nerve injury.
- Elbow dislocations – brachial artery and median nerve.

Imaging

Plain X-rays are indicated in most patients presenting with shoulder pain and reduced range of movement after trauma. Elbow fractures are very unlikely if there is full elbow extension. Fractures are difficult to see and radiographs should be examined carefully for evidence of an effusion: the dark shadows caused by the anterior and posterior fat pads.

Management

Analgesia is achieved by immobilisation (e.g. sling), and oral analgesics before imaging. Patients with severe pain and deformity require intravenous opiates and early assessment. Early active movement of the shoulder is important to avoid stiffness in the elderly.

Ensure urgent orthopaedic referral for:

- Any fracture with neurovascular compromise.
- Open fractures, which require urgent antibiotics.

Common diagnoses

Fractured clavicle

This injury most commonly occurs at the junction between the middle and outer third. Most heal with good function by providing rest in a sling and analgesia.

Acromio-clavicular joint injuries

Acromio-clavicular joint (ACJ) injuries are caused by fall onto tip of shoulder, causing disruption to the ACJ and ligaments. With complete disruption, the clavicle will 'float' above the acromion. ACJ injuries are treated with analgesia, rest in a sling and physiotherapy in the first instance, but the more severe grades of disruption may need fixation later.

Dislocated shoulder

The shoulder usually dislocates anteriorly (95%) from a fall with the arm in the 'hailing a taxi' position – the humerus is externally rotated and abducted. The humeral head may be palpable and the patient will support the arm, holding it by their side.

There are many different reduction techniques, each with their own proponents. It is generally best to start with a passive technique that requires only nitrous oxide/oxygen analgesia and can be conducted by nursing staff. The active techniques require intravenous analgesia ± sedation (Chapter 6).

- **Passive: hanging weight technique.** The patient lies prone on a couch with the arm hanging down with a 2–5 kg weight suspended from their wrist.
- **Active: Hippocratic technique.** Traction of the patient's arm, together with mild rotation. The traditional method of counter-traction involved the doctor's 'stockinged foot' in the patient's axilla. The modern version uses a sheet under the axilla so an assistant at the head of the bed can provide counter-traction.
- **Active: modified Kocher's technique.** This technique must not be rushed and requires good analgesia and sedation.
  1. Flex elbow, continuous gentle traction.
  2. Using the forearm as a lever, the humerus is externally rotated to almost 90° *very slowly* to overcome pectoral spasm.
  3. The arm is brought across the body and the humerus internally rotated to achieve reduction.

Reduction should be confirmed on X-ray, which may show any damage to the humeral head. Patients with a first dislocated shoulder should have the shoulder immobilised for 6 weeks to allow the capsule to heal. Patients with multiple dislocations need surgery to stabilise the shoulder.

Fractured neck of humerus

This injury is common in the elderly, due to FOOSH; underlying causes for falls should be sought (Chapter 30). Early mobilisation with appropriate analgesia is necessary to avoid long-term stiffness ('frozen shoulder') that may be far more disabling than the original injury. Displaced fractures may require reduction ± fixation.

Dislocated elbow

Hyperextension of the elbow forces the humerus anteriorly over the coronoid process of the ulna. Neurovascular status should be checked, and this should be reduced by traction under sedation.

Fractured head of radius

This is the most common elbow fracture, which can be difficult to see on plain X-ray, although the elbow effusion 'fat pad sign' will be visible. Diagnosis can be confirmed by tenderness over the radial head, and reduced pronation/supination. Most fractures make a good recovery with analgesia and early mobilisation.

Fractured shaft of humerus

Twisting injuries produce spiral fractures, bending injuries transverse fractures. Radial nerve injury can occur in fractures of the middle third of the humerus.

Diagnoses not to miss

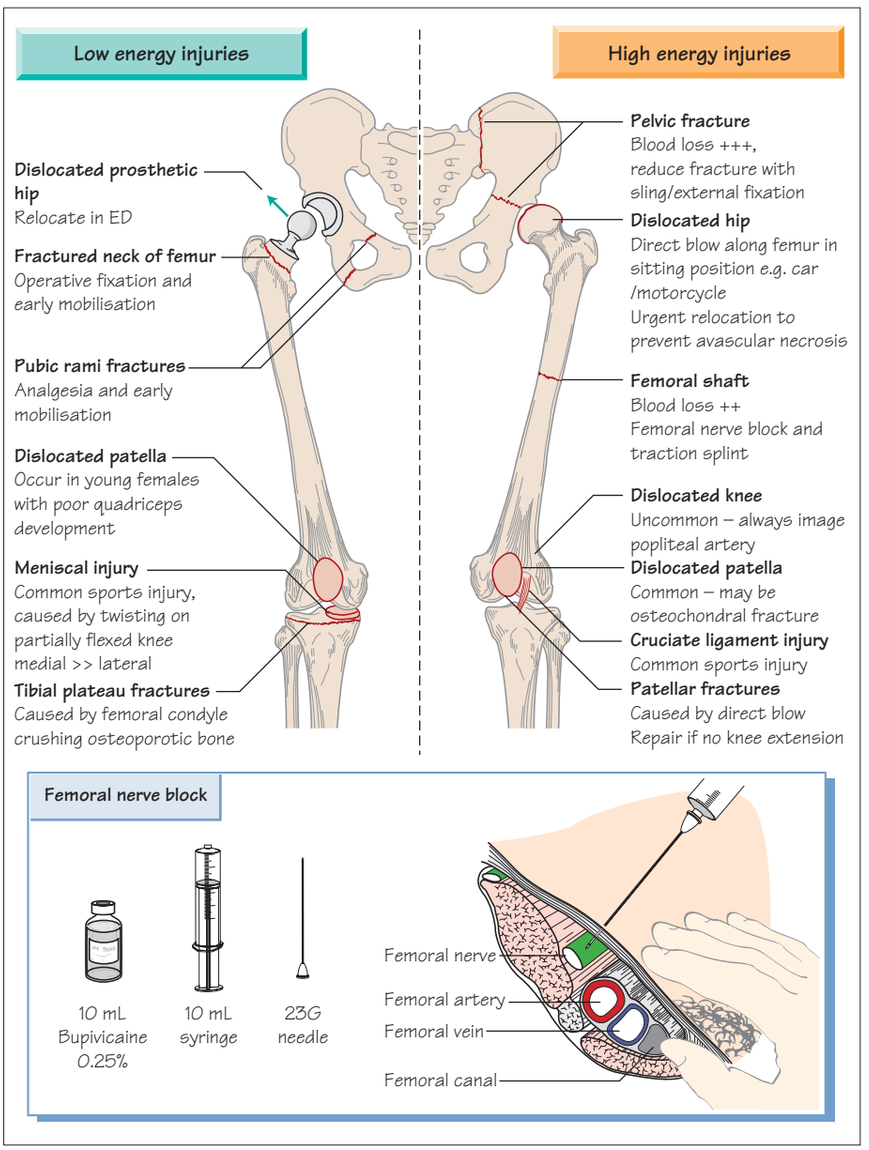
Posterior dislocation of shoulder

This injury is most common after epileptic fits or electrical injury forcing contraction of the strong latissimus dorsi muscles. Posterior dislocation is difficult to spot on X-ray: there is reduced gleno-humeral overlap and the greater tuberosity is not visible, creating the 'lightbulb sign': the humeral head appears symmetrical. If in doubt, ask for an axillary view X-ray.

Scapular fracture

Scapular fractures can be difficult to see on X-ray, but are usually very painful due to distension of the tight capsule and may need admission for analgesia. Significant energy is necessary to fracture a scapula, and other injuries should be sought.

17 Back pain, hip and knee injuries



Back pain

Lumbar back pain is a common presentation to the Emergency Department, and can be very challenging to manage. Patients may arrive at the Emergency Department with an agenda that includes hospital admission for analgesia and rehabilitation. This is not practical or desirable: after exclusion of significant pathology, early mobilisation is the most effective treatment. Back pain may also be caused by hip disease and retroperitoneal organs, e.g. aorta, pancreas.

Red flags

There are four conditions that must not be missed.

1 Abdominal aortic aneurysm (Chapter 19).

2 Malignancy.

3 Epidural abscess or haematoma.

4 Large prolapsed disc causing neurological deficit or cauda equina syndrome.

Therefore any history and examination must document the following.

- Age, back pain history, history of malignancy.
- Pain at rest, pain wakes at night.
- History of trauma, fever, intravenous drug use, anticoagulation.
- Straight leg raising, angle, crossed leg raise.
- Power at each joint (flexion, extension).
- Reflexes: knee, ankle, plantar.

• Incontinence, perineal anaesthesia, reduced anal tone (implies possible cauda equina syndrome).

Crossed straight leg raising: lifting the *unaffected* leg reproduces pain in the other leg. This is a very sensitive indicator of nerve root irritation, e.g. from a prolapsed disc.

If there are no abnormalities and the patient is otherwise well, the diagnosis is likely to be mechanical back pain.

Investigations

Investigations are rarely necessary if no red flag symptoms. MRI is the gold standard for investigating spinal neurological problems. Urgent MRI scanning is indicated if cauda equina is suspected. Lumbar spine X-ray (70 CXR) is only indicated with a history of trauma or if malignancy is suspected.

Management

A positive but firm attitude to encourage mobilisation may be necessary: Emergency Department nursing staff are particularly skilled at this.

The combination of an NSAID (e.g. ketorolac) and paracetamol/codeine-based analgesia is a good starting point. Diazepam acts as a muscle relaxant if there is significant spasm, but should only be given for a couple of days.

Hip and knee injury

The hip is an inherently stable joint, which requires substantial energy to disrupt. The knee's stability depends on muscles, tendons, ligaments and cartilage, all of which are vulnerable to injury. Osteoporotic bone is vulnerable to low-energy injuries, i.e. 'fragility fractures' such as fractured neck of femur (#NoF).

Examination

Look Assess gait and inspect for joint swelling or asymmetry. Look for shortening and external rotation (#NoF) or flexion and internal rotation (dislocation of hip). Swelling of the knee joint may be due to a joint effusion. Acute traumatic effusion occurs as a result of bleeding from bony or ligamentous injury.

Feel Areas of tenderness may indicate fracture, e.g. patella, head of fibula. Knee effusion is detected by pushing the patella down so it makes contact with the anterior surface of the femoral condyle – 'patellar tap'.

Move Assess all hip movements. Internal/external rotation at the hip is a sensitive test for fractures. Assess range of movement of knee, specifically for pain or instability (ligament injury) or locking/unlocking (meniscus tear/loose body).

• Knee ligamentous stability: ACL, PCL, LCL, MCL (anterior and posterior cruciate, lateral and medial collateral ligaments).
• Knee meniscal stability: Apley's test.
• Patellar stability: apprehension test.

Neurovascular examination

Knee dislocation damages the popliteal artery, which *always* needs expert vascular assessment. The common peroneal nerve is at risk in lateral knee injuries: test for dorsiflexion of foot and sensation over dorsum of foot.

Investigations

Bedside investigations

• Blood glucose, urine dipstick, ECG in patients with falls.

Laboratory investigations

• FBC and group and save indicated in all patients with pelvic or femur fractures, as bleeding is often underestimated.

Imaging

• In frail elderly patients, even low amounts of energy can cause fractures. All possible hip fractures should have an X-ray of the pelvis and lateral hip. The pelvis and pubic rami are brittle ring structures, and like a 'Polo' ® mint, they can never be broken in one place only.
• The Ottawa knee rules prevent unnecessary knee X-rays.
• CT is useful for pelvic and tibial plateau fractures.
• MRI is the gold standard for the diagnosis of knee injuries and occult hip fractures.

Treatment

Lower limb fractures are painful. Intravenous opiates are often necessary. A femoral nerve block gives effective analgesia for femoral fractures at/below the trochanter. Femoral shaft fracture requires a traction splint.

Knee

A tense, painful knee haemoarthosis should be aspirated. This also allows examination of cruciate function, reduces intra-articular adhesions, or confirms haemarthrosis vs. blood-stained effusion. By putting the aspirate into a bowl, fat globules floating on the surface will be seen if there is a fracture.

Most patients with isolated knee injuries will be able to go home in a knee brace or a Robert Jones bandage (a wool and crepe bandage built up to support the extended knee) with outpatient clinic follow-up.

Hip and femur

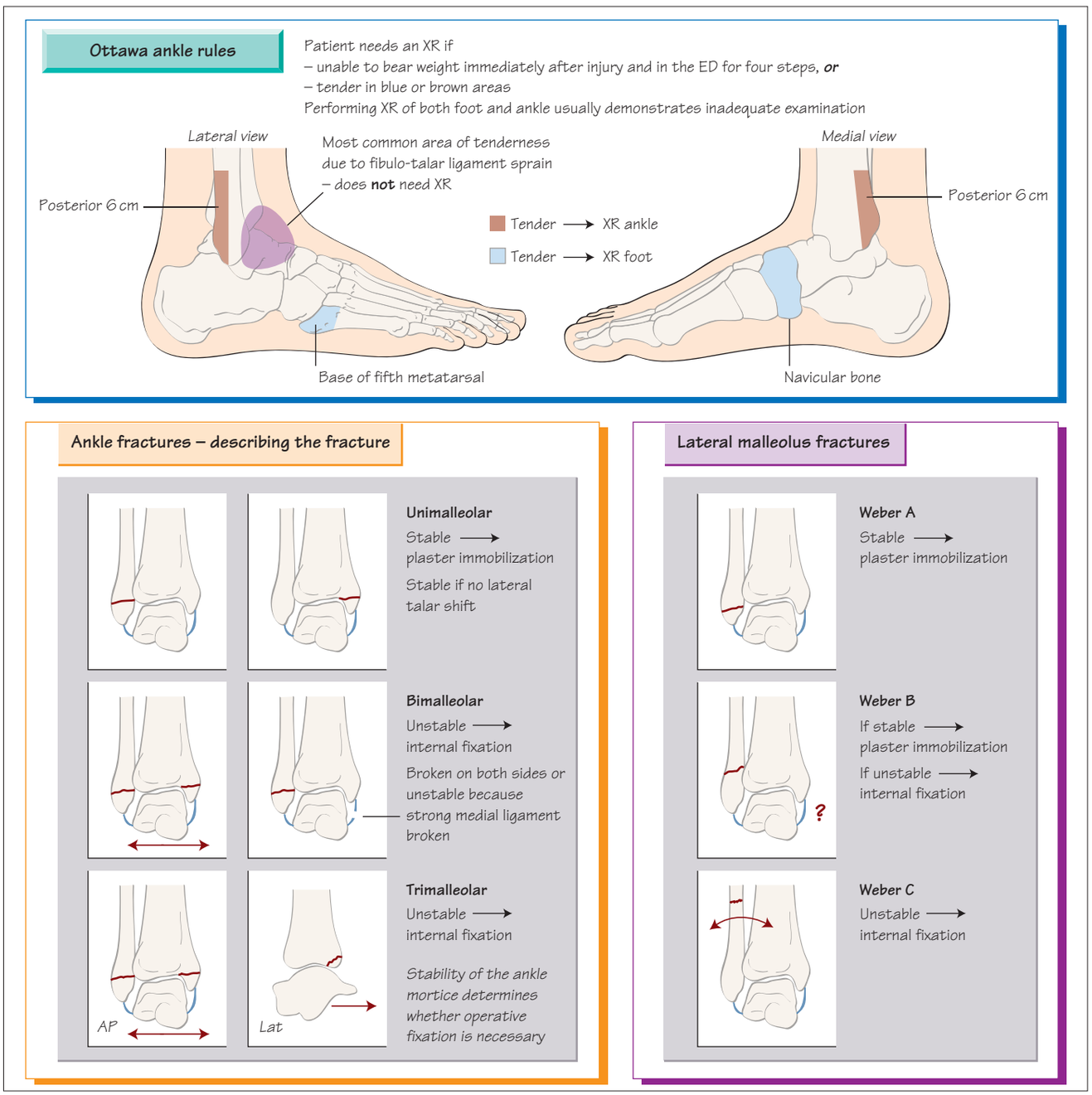
Fractured neck of femur is common in the elderly and requires operative fixation. Consider possible causes for falling (Chapter 29).

A patient who has a clinically suspected fractured neck of femur but normal X-rays needs admission and further investigation. These patients often have fractured pubic rami, or impacted fractures seen on further imaging, e.g. MR.

The Ottawa knee rules

X-ray if:
- >55 years
- Tender at head of fibula
- Tender patella
- Inability to flex to 90°
- Inability to weight bear (4 steps, limping allowed) both immediately and in the Emergency Department.

18 Tibia, ankle and foot injuries



History

Mechanism of injury

High-energy injuries commonly result from axial loading, direct blows or crush injuries, e.g. falls or jumps, motor vehicle accidents. Tibial shaft injuries are severe, and risk neurovascular injury and compartment syndrome.

Low-energy injuries tend to occur from twisting at the ankle joint, particularly ankle inversion. The medial tibiotalar ligament is strong, unlike the weaker fibulotalar and fibulocalcaneal ligaments on the lateral side. Hence most ankle sprains are on the lateral side, caused by inversion of the foot.

Examination

Look A dislocated ankle will look deformed. Bruising and swelling around ankle and foot is not specific for fracture, although bruising over the calcaneus is likely to indicate a fracture.

Feel Widespread mild soft tissue tenderness is common in ankle injuries and needs to be differentiated from specific areas of bony tenderness. Examination must never be rough, but if poor-quality information is collected from the clinical examination, poor-quality decisions will be made. Check neurovascular status.

Move The function of the foot and ankle is critically dependent on the subtalar joint. Support the calcaneus in your non-dominant hand and use your dominant hand to:

- Flex and extend at the ankle.
- Invert and evert at the subtalar joint.
- Twist the forefoot while holding the calcaneum tight.

Imaging

- A dislocated ankle should be reduced before taking X-rays.
- Use the Ottawa rules (opposite).
- Ordering both foot and ankle radiographs together implies inadequate examination.
- CT is useful in injuries to the mid and hindfoot.
- Radiographs of toes other than the big toe are unlikely to change management, and should not generally be performed.

Management

Patients with an open fracture must have intravenous antibiotics urgently; the injury should be photographed and then covered in a saline-soaked dressing pending urgent theatre. Taking a picture of the wound is useful for the surgeon as it will be covered by dressing and plaster.

Analgesia includes splintage and reduction of fractures. Elevation will reduce pain and tissue oedema, which facilitates surgery, and reduces the risk of compartment syndrome. Neurovascular status must be recorded, particularly before and after fracture/dislocation reduction.

Disposal: who can go home?

Admit patients with the following.

- Open fractures.
- Dislocated ankles.
- Failed closed reduction/unstable fracture.
- Fractured talus or calcaneus.
- Tarsometatarsal dislocation: Lisfranc injury.

Patients with stable fracture pattern injuries, who are unlikely to need surgery and who have no evidence of complications, can usually go home. The initial cast should be either a backslab or a split cast; this is because swelling will occur over the first 24 hours after injury. Casts should ensure that the foot is at 90° to the leg to maintain soft tissue length, except for Achilles tendon plasters, which are plantarflexed.

Patients should receive written instructions covering monitoring for signs of neurovascular compromise/compartment syndrome, (Chapter 15) cast care, advice to elevate the limb and follow-up arrangements. If crutches are necessary, patients should be shown how to use them.

Common diagnoses

Ankle sprain

If a fracture is ruled out using a decision rule or radiograph, the likely diagnosis is a 'sprained ankle' – partial rupture of the lateral ligament complex, e.g. fibulo-talar and fibular-calcaneal.

In addition to short-term rest, ice and elevation, patients may benefit from physiotherapy. Compression bandages do not help, but for more severe sprains, immobilisation in a backslab for one week, together with crutches, is advisable.

Ankle fractures

The mortice joint of the ankle is responsible for its structural integrity. If the mortice is intact on one side, the fracture will be stable, and may be managed in plaster. If both sides are unstable, internal fixation will be necessary.

Gastrocnemius tear

The calf muscle may tear in a sudden contraction, common in tennis. Treatment is rest, analgesia and physiotherapy.

Achilles tendon rupture

Occurs when jumping (basketball, racquet sports) – or due to quinolone antibiotics. Squeezing the calf muscles normally causes foot plantarflexion – this does not occur with a complete rupture, which can be confirmed by ultrasound. Complete rupture is repaired, but partial rupture is managed in plaster.

Metatarsal fracture

The second and third metatarsals are most affected by stress fractures. The tendon of peroneus brevis attaches at the base of the fifth metatarsal. Inversion injuries may cause avulsion of this tendon with a flake of bone.

Avulsion fractures

These are easily missed in the 'sprained foot'. Look specifically at the navicular, talus, cuboid and the inferior border of the malleoli for a flake of bone that has been avulsed by a ligament.

Diagnoses not to miss

Talar fracture

Falls on an inverted foot, or forced foot dorsiflexion ('aviator's fracture') may cause a talar fracture. The talus has a central role in both ankle flexion/extension and inversion/eversion. Fractures of the talus often have poor outcomes due to a circuitous blood supply, with high rates of avascular necrosis.

Tarsometatarsal (Lisfranc) dislocation

These injuries are rare, complex and often missed. The history may be of a crush type injury. Typical signs and symptoms include pain, swelling over midfoot (TMT joints) and the inability to bear weight. X-ray findings are subtle and may appear normal. Consider this diagnosis if pain is very high despite no apparent deformity.

Calcaneal fracture

Calcaneal fractures are generally caused by a fall from a height. Fractures are usually comminuted and are associated with fractures of the lumbar spine and wrists, which should be examined carefully.

19 Abdominal pain

Common

Gastritis/ oesophagitis
A burning pain felt in the epigastric region relieved by antacids

Biliary inflammation/sepsis
See opposite

Murphy's sign – the patient stops an inspiratory breath when pressure is applied above the gallbladder. The inflamed gallbladder hits the peritoneum causing pain. A positive Murphy's sign together with fever or ↑WCC is highly predictive of biliary sepsis

Biliary colic
Biliary colic is the pain experienced when the muscular wall of the biliary ducts contracts against a stone. This intermittent pain is often precipitated by a large fatty meal
There is no fever/↑WCC

Renal colic, pyelonephritis
See Urology Chap 20

Bowel obstruction
Vomiiting, distension, and an empty rectum suggest large or small bowel obstruction, usually caused by adhesions or herniation

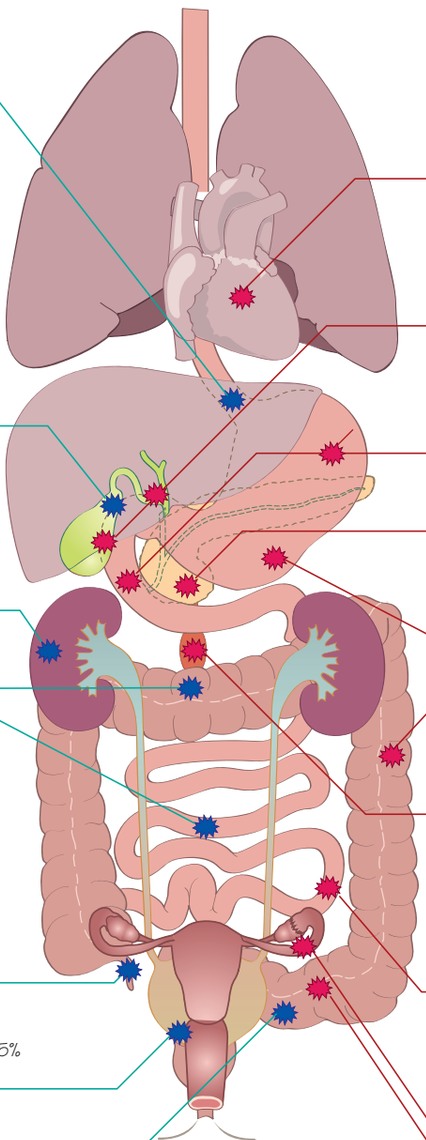
McBurney's point
2/3 of the way from the umbilicus to a point midway along the inguinal ligament

Appendicitis
Starts as a poorly localised mid-abdominal pain, then moves to tenderness at McBurney's point due to peritoneal irritation. Lifetime occurrence 5%

Urinary tract infections
Particularly common in women due to short urethra. Treat according to local policy

Diverticular disease
Caused by chronic constipation, and common in the elderly, diverticulae may rupture, bleed or become infected

Constipation
Constipation is very common, but is a diagnosis that should only be made after having considered and excluded more serious pathologies



Don't miss

These are diagnoses that may be subtle, but that have a high morbidity/ mortality and therefore figure prominently in medico-legal practice

Myocardial infarction
An ECG should be performed in any patient over 40 presenting with abdominal pain.
See Chap 34

Biliary inflammation/sepsis
Likely if there is partial or complete biliary obstruction, creating a reservoir of slow moving bile. Diabetic patients are at increased risk

Gastrointestinal bleed
The commonest cause of lower GI bleeding is upper GI bleeding. See Chap 40

Pancreatitis
Dull central pain, often radiating to the back as the pancreas is retroperitoneal. 30% due to gallstones, 30% due to alcohol – the rest have mixed/unknown causes

Perforation/ peritonitis
Uncommon since H_2 blockers/ PPI – as caused by duodenal or gastric ulceration.
Widespread severe pain, with rigid abdomen, no bowel sounds
Needs resuscitation and urgent surgery

Aortic aneurysm
Suspect in any patient over 50 with abdominal pain. Atherosclerosis and hypertension are major risk factors
Pain may radiate to the back and be mistaken for renal colic
Urgent surgery/stent

Ischaemic bowel
Old age, atherosclerosis and atrial fibrillation are risk factors for this insidious disease that is easy to miss. An elevated lactate can be the only clue

Ruptured ectopic pregnancy
– see gynaecology Chap 23

Sigmoid volvulus
Volvulus is a variant of bowel obstruction due to twisting of the bowel especially sigmoid colon

The abdominal cavity contains the organs that digest food, filter blood and enable reproduction, any of which may give rise to abdominal pain. As with chest pain, patients presenting with a 'textbook' collection of symptoms are the exception rather than the rule.

History

A focused history should be taken, concentrating on the nature and timing of the pain and its associations. Most abdominal space relates to food processing, therefore the relationship of pain to food intake/excretion is important, e.g. pain related to large or fatty meals suggests gallstones. Date of last menstrual period (LMP) is essential information to obtain from any woman of childbearing age.

Nature of the pain

There are three common sorts of abdominal pain.

- *Colicky pain:* pain that comes and goes in spasms, and is usually the result of peristalsis failing to move a solid mass, e.g. the ureter attempting to move a stone to the bladder. The patient may move about, seeking a comfortable position.
- *Peritonism:* the sharp, well-localised pain resulting from inflammation of the parietal (outer) peritoneal surface – peritonitis. The patient lies still to avoid moving the inflamed surfaces.
- *Distension pain* results from an organ or bowel being stretched. The pain is poorly localised and may be felt as central abdominal pain. When the bowel is distended by gas, it may be tympanic: the abdomen sounds 'hollow' when percussed.

Less common types of pain are:

- *Mucosal pain:* burning pain due to inflammation of the mucosa, e.g. reflux of gastric acid into the oesophagus, urinary tract infection (UTI), sexually transmitted infection (STI).
- *Ischaemic pain:* poorly localised gnawing/cramping pain caused by inflammation progressing to ischaemic necrosis, e.g. menstruation, ischaemic bowel.
- *Referred pain:* pain occurring in a different area, e.g. cardiac ischaemia may be perceived as abdominal pain. Conversely, pain from within the abdomen may be perceived elsewhere, e.g. shoulder tip pain from diaphragmatic irritation, penile pain from renal colic, and back pain from retroperitoneal structures.

To make things more complicated, a single pathophysiological process may cause different types of pain simultaneously.

Examination

Inspection

From the end of the bed: is the patient well/ill/critically ill? Immunosuppressed patients may appear deceptively well despite significant disease. Also beware patients with neuropathy, e.g. diabetics who may not experience 'normal' pain. Patients who cannot get comfortable or who are constantly moving are likely to have colicky pain. Patients who lie very still are likely to have peritonitis.

Palpation, percussion and auscultation

Poorly localised general pain is usually felt around the umbilicus, but specific point tenderness suggests peritonitis. Increased bowel sounds are caused by obstruction; absent bowel sounds indicate peritonitis. Rectal examination is an important part of the examination, and stool should be tested for blood.

Investigations

Bedside investigations

- Blood glucose.
- Urine dipstick.
- Urinary βhCG in any woman of childbearing age.
- Ultrasound is used by emergency physicians to rule out abdominal aortic aneurysm or to look for intra-abdominal fluid. If the expertise is available, it may be useful in patients with other diseases, e.g. gallstones.

Laboratory investigations

- FBC, U+E, LFTs and amylase/lipase in all patients.
- Arterial blood gases including lactate in sick patients.
- Group and save/cross-match blood if patient likely to go to theatre.

Imaging

- An erect chest X-ray detects free air from a perforated bowel.
- A supine abdominal X-ray (60 CXR) will demonstrate obstruction but is otherwise unlikely to be helpful.
- Ultrasound is good for biliary, urinary and gynaecological causes of pain.
- CT (300 CXR) is very good at demonstrating most abdominal pathology but is a high dose of radiation.
- MRI is good for imaging abdominal organs, but is not widely available.

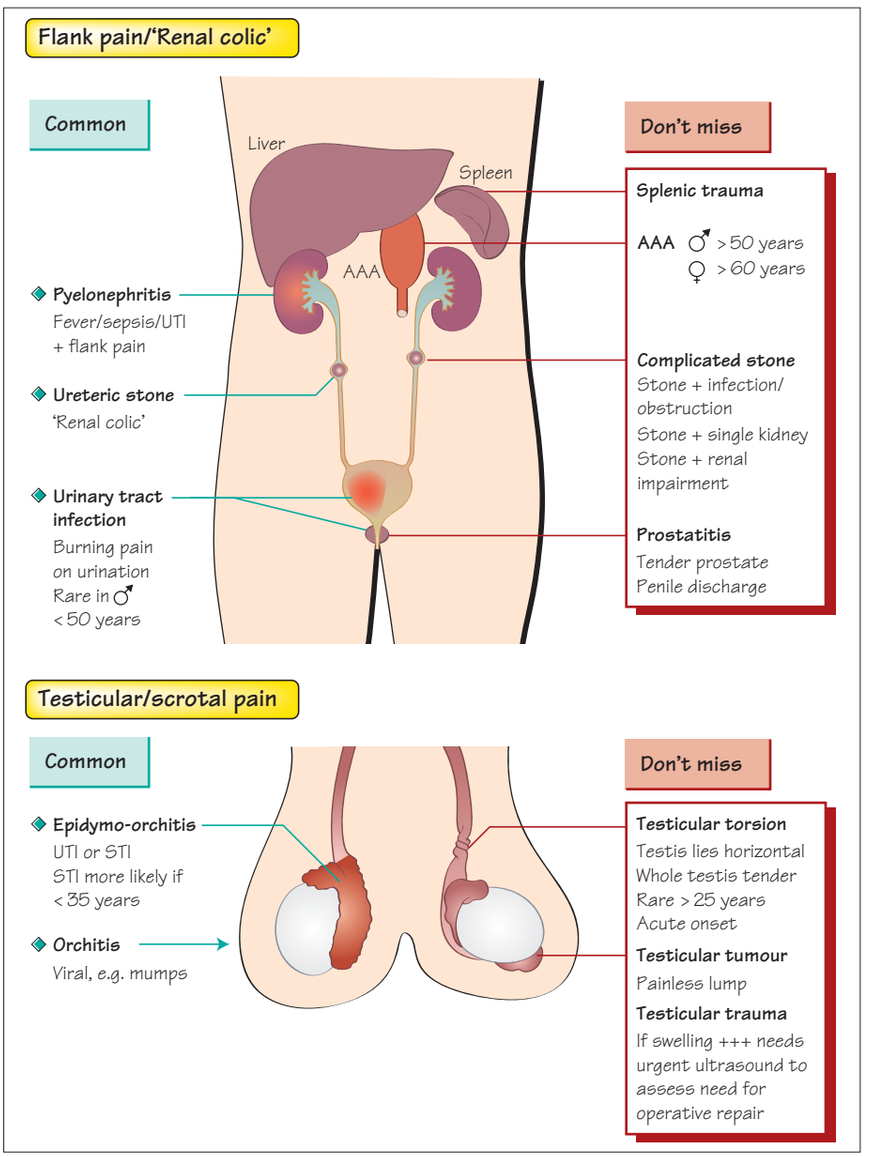
Management

- *Resuscitation* and urgent surgical opinion if clinically unwell. Oxygen for all unwell patients together with observation and monitoring in a suitable clinical area.
- *Intravenous fluids* are an important part of resuscitation, but also replace ongoing fluid losses (Chapter 3). A nasogastric tube keeps the stomach empty, e.g. if there is bowel obstruction.
- *Analgesia:* intravenous morphine with anti-emetic is humane, safe and does not impede diagnosis. Intravenous or rectal NSAID, e.g. ketorolac, is good for peritoneal pain and relaxes smooth muscle so is good for colicky pain, although should be avoided in the elderly.

Disposal: who can go home?

Any patient who has abdominal pain requiring ongoing morphine needs to be admitted. Patients who appear well, in whom serious pathology has been excluded, and whose pain has not recurred after analgesia has worn off, are usually safe to discharge. Other patients should be reviewed by the relevant surgical team.

20 Urology problems



The urinary tract includes the kidneys, ureters, bladder, (prostate), urethra, and external genitalia. Symptoms perceived by patients reflect the embryological origin as well as the current anatomy of these organs.

History

Any previous history of urogenital problems, and a specific focus on:

- *Colicky pain:* intense pain that comes and goes suggests intermittent contraction of a hollow organ, e.g. ureter. Patients with ureteric colic cannot find a comfortable position. Pain may be referred to the genitals.
- *Back pain:* the kidneys are retroperitoneal.
- Ask about urinary frequency, flow, blood or clots.
- *Fever:* with chills and rigors (shaking) suggests sepsis (Chapter 38).
- *Dysuria:* burning pain when passing urine implies urethral inflammation. Abnormal discharge from the genitals suggests STI.
- *Sexual history:* if other symptoms suggest it is likely to be relevant.

Examination

A patient with active renal colic will move around, trying in vain to find a comfortable position; other causes of intra-abdominal pain are usually alleviated by lying still. The abdomen should be palpated for alternative causes of pain (e.g. abdominal aortic aneu-

rysm (AAA), cholecystitis) and the kidneys should be examined for tenderness.

In a patient unable to void urine, a palpable tender bladder that is dull to percussion suggests urinary retention. The external genitalia should be examined if symptomatic, including rectal examination if prostatitis is suspected.

Investigations

Bedside investigations

- Ultrasound: can rule out AAA.
- Blood glucose.
- Swabs from urethra/cervix if appropriate.
- Urine dipstick testing is a rule-out test: if leucocytes, nitrites, blood and protein are all negative, the urine does not need to be sent for culture, unless the patient is immunosuppressed. If nitrites and leucocytes are negative, infection is unlikely (-LR = 0.16), but if the patient's symptoms are very suggestive of UTI, the urine may be sent for culture.
- βhCG
- Urine microscopy – red cell casts imply glomerular bleeding, rather than bleeding elsewhere in the urinary tract.

Laboratory investigations

- FBC, U+E
- LFTs, amylase if abdominal pain.

Imaging

- Ultrasound is operator and body mass index dependent, can detect bladder size, ureteric and renal pelvis dilatation resulting from obstruction but cannot reliably detect stones.
- CT KUB (kidney, ureters, bladder) detects stones and other intra-abdominal pathology, but involves significant radiation (300 CXR). MRI is an alternative that avoids irradiation.
- Contrast radiography: intravenous urogram (IVU; 250 CXR) has been superseded by CT, but X-ray KUB (75 CXR) can track radio-opaque stones.

Common diagnoses

Urinary tract infection/pyelonephritis

Females are more prone to UTIs due to the short urethra. Drinking large quantities of water may help flush out mild infection, but more serious infection needs treatment for 3 days with antibiotics: trimethoprim or nitrofurantoin are common recommendations. Men with UTIs and women with recurrent UTIs need antibiotics for 7 days and should be reviewed in an outpatient clinic.

Pyelonephritis occurs when a UTI ascends to the kidney(s). The patient is systemically unwell with fever, loin/back pain, rigors, headache, nausea and vomiting. The kidney(s) are tender on palpation. Emergency Department treatment includes antibiotics (e.g. gentamicin), analgesia and intravenous fluids. Patients who respond to this may be discharged with oral antibiotics (e.g. co-amoxycillin) and GP follow-up.

Urinary tract stones

Some patients, usually for unknown reasons, form stones in their renal pelvis. If the stones pass into the ureter, they cause intense colicky pain, 'renal colic' and microscopic haematuria (90%).

WARNING

'Renal colic' in a male over 50 years old is AAA until proven otherwise.

NSAIDs, e.g. ketorolac i.v. or diclofenac p.r., are rapidly effective at relaxing ureteric smooth muscle. Morphine is useful for ongoing pain; pethidine (meperidine) should be avoided – opiate-seeking should be suspected if it is requested.

CT confirms the diagnosis, and informs treatment decisions. If there is a stone of less than 5 mm and the pain has resolved, discharge patient on regular NSAID or tamsulosin (an alpha blocking drug that also helps stones pass) with outpatient clinic review.

Patients who are discharged should be warned to return if they develop fever or further significant pain. Otherwise, or if there is evidence of infection, urinary obstruction, renal failure or single kidney, discuss with urology team.

Urinary retention

Urinary retention may occur due to mechanical obstruction or neurological impairment, causing acute or chronic retention that may cause renal damage. Ultrasound can confirm a large residual volume of urine in the bladder after voiding.

Catheterisation should be performed urgently to relieve obstruction and pain. If dipstick testing indicates that the patient's urine is likely to be infected, then catheterisation should be covered by a single shot of gentamicin. If there are blood clots in the bladder, a large irrigation catheter may be needed to flush out the bladder.

If urinary retention occurs in a patient with back pain, consider cauda equina compression (Chapter 17). Constipation, e.g. from opiate analgesics, can cause urinary retention: treating the constipation resolves the retention.

Sexually transmitted disease

Dysuria and/or discharge makes STI more likely than torsion, but if there is any doubt, an ultrasound can confirm normal testicular perfusion. Swabs should be taken and the patient should be followed up in an STI clinic for contact tracing.

Diagnoses not to miss

Testicular torsion

Common in early adulthood, the spermatic cord twists, causing testicular ischaemia. Torsion is diagnosed clinically by a tender, high-riding testis: ultrasound may confirm the diagnosis, but must not delay surgical exploration.

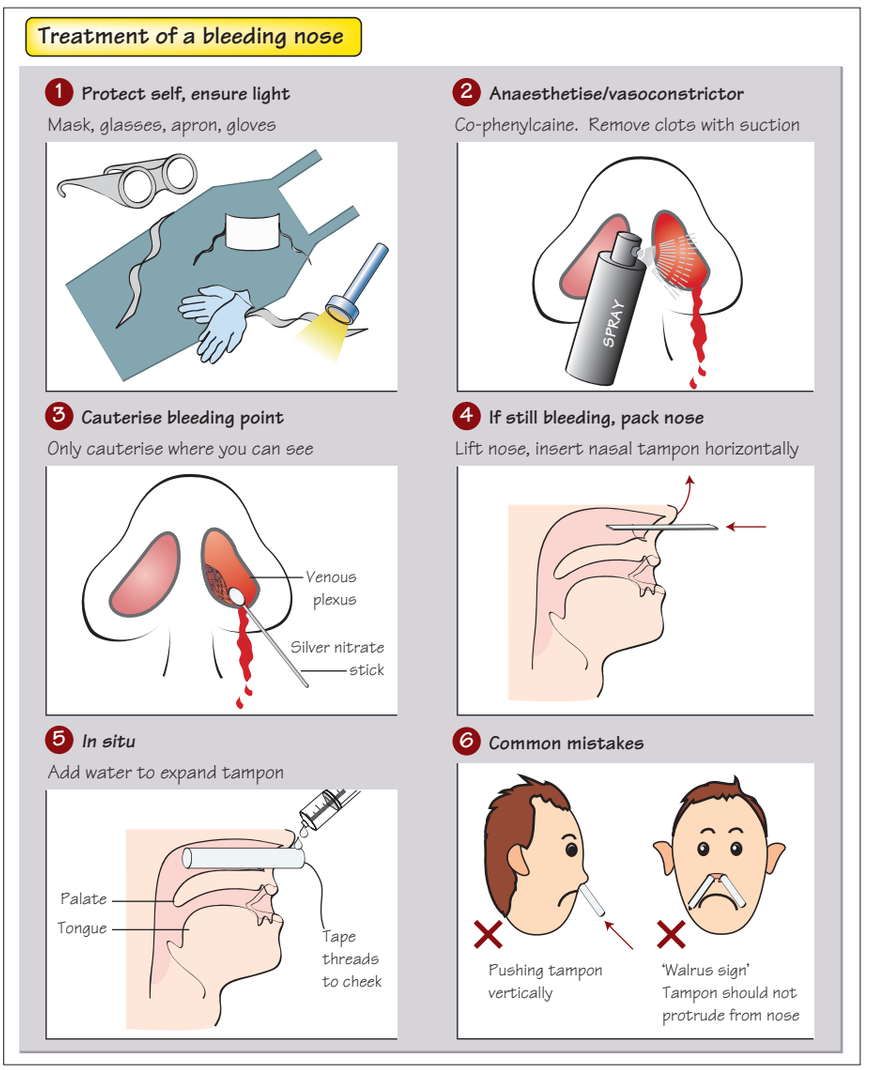
Infected obstructed kidney

The combination of urinary obstruction and infection can rapidly destroy a kidney. Evidence of possible infection should be sought in patients who have obstruction, e.g. stones, in their urinary tract.

Prostatitis

UTIs are uncommon in men, and prostatitis should be considered. The diagnosis is confirmed by a tender prostate on rectal examination, after which urine is taken for culture. Prolonged antibiotic treatment is necessary, e.g. ciprofloxacin for 3 weeks.

21 Ear, nose, throat and dental problems



Ear, nose and throat (ENT) examination needs patience and practice to master. Patients may cough or sneeze, showering you with their body fluids, so protect yourself with gloves, apron, mask and eye protection. Adequate light and topical anaesthesia makes examination easier and your patient more comfortable.

Ear

Common diagnoses

Otitis media/sinusitis

Ear pain is usually caused by infection in the middle ear – otitis media. The eardrum appears dull with prominent blood vessels. Sinusitis presents as headache and a feeling of pressure in the face. These are self-limiting conditions caused by a viral upper respiratory tract infection, blocking drainage from airspaces within the head. Analgesics and decongestant drugs are helpful; antibiotics are not.

Otitis externa

Otitis externa or 'swimmer's ear' is a localised infection of the ear canal, which becomes congested with discharge and debris. Otitis externa is treated with topical antibiotics and steroids, applied using a wick of cotton wool.

Ruptured ear drum

Commonly caused by trauma, barotrauma or infection, a ruptured ear drum normally heals within 2 months. Patients should avoid immersing the ear in water.

Vertigo

Vertigo causes a sensation of *spinning*; it is not just 'feeling faint/light-headed'. Vertigo is caused by conflicting sensory information from ears, eyes and joints. The problem is usually due to peripheral (sensory) problems rather than central (brain) ones.

• A *peripheral* cause is likely if the patient has hearing loss, tinnitus, ear infection, headache, nausea and vomiting.
• A *central* cause is likely if the patient has motor symptoms or cardiovascular risk factors, e.g. atrial fibrillation.

Use the Dix-Hallpike test to differentiate central from peripheral causes. If there are central signs, check blood glucose and ECG – consider transient ischaemic attack (TIA)/stroke or other neurological cause (Chapter 42).

If the patient has no hearing loss, the most common cause of vertigo is vestibular neuronitis, usually caused by a (viral) upper respiratory tract infection. Prochlorperazine (an anti-emetic) +/– intravenous fluids is particularly effective. Antihistamines are structurally similar drugs and can also be used. Vestibular labyrinthitis is similar, but patients may have hearing loss and tinnitus.

Nose and face

Common presentations

Nosebleed

Most patients bleed from venous plexi in the anterior part of the nose – Little's area. Some (usually elderly) patients may have bleeding from the posterior part of their nasal cavity. Ask about warfarin and antiplatelet drugs such as aspirin and clopidogrel. Check FBC/clotting in older patients. Pack the nose and admit according to local protocols.

Facial fractures

Assess stability of upper teeth and mandible, and sensation over the face. If there is a fracture of the orbital floor, examine the eye movements (Chapter 22). Radiographic facial views are necessary, but are difficult to interpret – look for asymmetry. If there is mandibular injury, request XR OPG (oral pantomogram): fractures of the neck of the mandible can be difficult to spot.

Fractured nose

A patient with a painful swollen nose following trauma is likely to have broken their nose. X-rays do not change management. The patient should be discharged and reviewed in 5–7 days in an Ear, Nose and Throat clinic.

Do not miss

Septal haematoma

Septal haematoma – a swelling from the medial side of a fractured nose, usually in a young adult. This requires urgent drainage to prevent avascular necrosis of the cartilage.

Throat

Common presentations

Tonsillitis/pharyngitis

Pharyngitis and tonsilitis can be caused by bacteria or viruses. Viral pharyngitis is more likely if the patient has runny nose/conjunctivitis/diarrhoea. Group A β-haemolytic *Streptococcus* (GAβHS) is responsible for 10% of pharyngitis, and is treated by penicillin/erythromycin if three or more of the following criteria are present.
• Fever.
• Exudate on the tonsils.
• Tender anterior neck lymph nodes.
• Lack of cough.

If two or more criteria are present, rapid antigen tests can be used to identify those patients with GAβHS. Complications of untreated GAβHS are uncommon, and over-treatment with antibiotics is self-reinforcing. Patients who are systemically unwell with extensive bacterial pharyngitis need admission for intravenous penicillin and fluids.

Foreign body in throat/oesophagus

The site of pain suggests location of the foreign body.
• Unilateral pain – foreign body above cricopharyngeus.
• Pain in submandibular region – foreign body in tonsillar fossa.
• Pain around larynx – foreign body in posterior tongue.

If there is pain on every swallow, the foreign body is probably still there; if there is just vague discomfort, the foreign body has probably gone. Radiography is useful for bones, but fishbones, a common cause, are not very radio-opaque.

If there is no danger of the foreign body causing obstruction were it to be pushed into the trachea, it may be removed under direct vision using forceps or suction. Otherwise it is likely the foreign body will need to be removed under general anaesthesia.

Foreign bodies stuck in the oesophagus will often move with a combination of glucagon (which relaxes the lower oesophageal sphincter) and fizzy drink. Failure necessitates endoscopy.

Diagnoses not to miss

Quinsy (peritonsillar abscess)

Quinsy causes a painful, asymmetrically swollen throat with difficulty opening the mouth or swallowing and a 'plummy' voice. Treated by aspiration or drainage in theatre, together with antibiotics.

Epiglottitis, retropharyngeal abscess, Ludwig's angina

These rare but dangerous infections can cause upper airway obstruction, giving stridor, a whistling sound worse on inspiration. Patients are unwell with high fever, sitting forward, with a stiff neck, drooling saliva they are unable to swallow. Treatment is urgent anaesthetic and ENT airway assessment and antibiotics.

Postoperative bleeding

Postoperative bleeding is often a result of infection: these patients should always be reviewed by the Ear, Nose and Throat team.

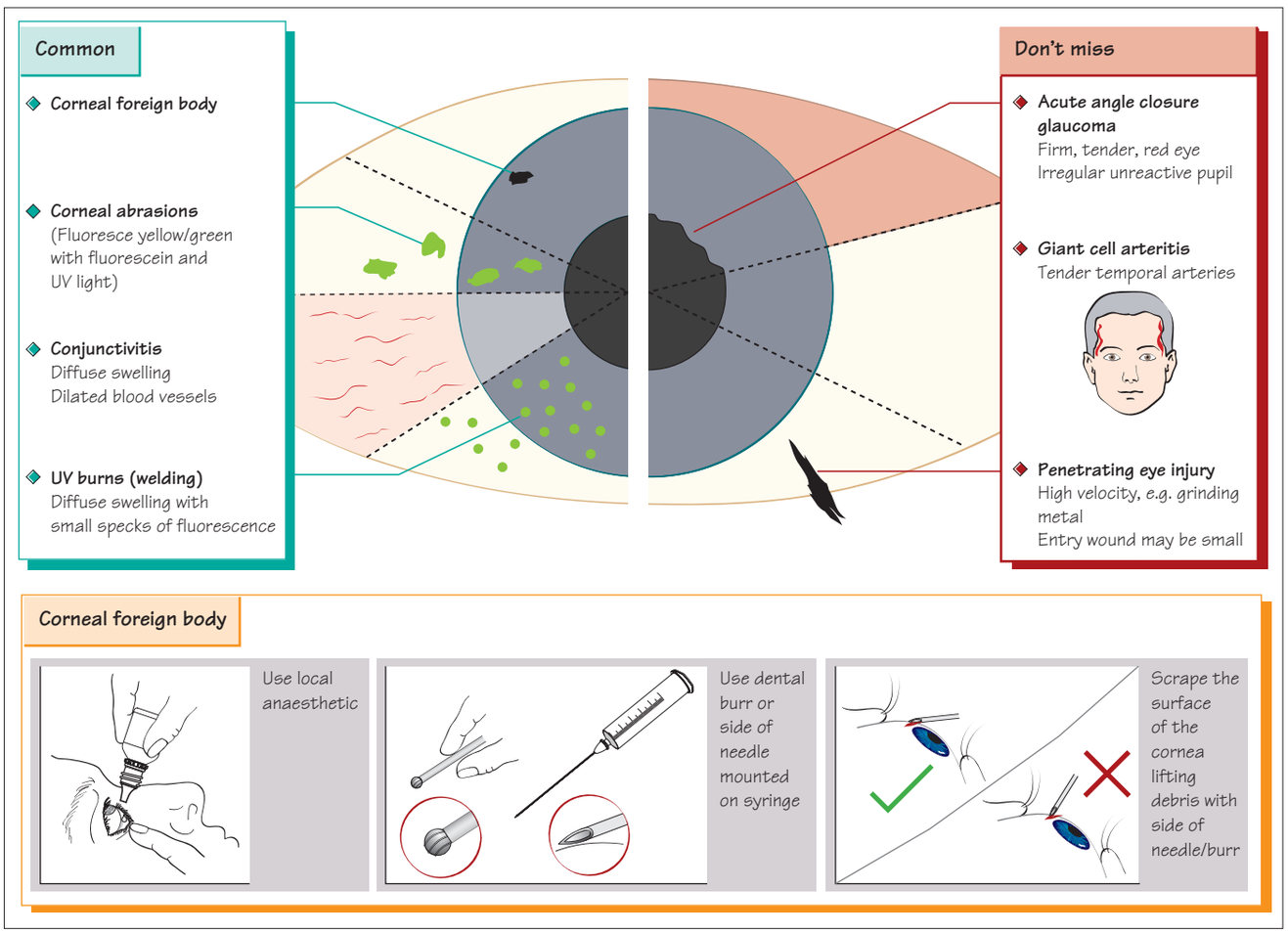
Dental

Dental pain is usually caused by dental caries leading to local infection (pulpitis) and abscesses. Affected teeth are tender to percussion and temperature. Treatment is analgesia and advice to see a dental practitioner. Antibiotics are not normally indicated.

Wounds inside the mouth rarely need treatment as they heal very rapidly, and saliva has a natural antibacterial action. Exceptions are 'through and through' lacerations (through oral mucosa, muscle and facial skin) or lacerations involving the tip of the tongue.

An avulsed tooth should be replaced in the socket immediately if it is to survive. If this is not possible, the patient should carry the tooth between cheek and teeth. A dentist can place a splint to keep the tooth in place.

22 Eye problems



Eye problems seen in the Emergency Department are usually the result of trauma affecting the anterior part of the eye, but can also be manifestations of systemic, CNS or vascular disorders.

A brief general history should include details of diabetes, stroke, hypertension, neurological or cardiac problems and drug treatment. Ask about trauma and the use of hand or power tools prior to the symptoms, as shards of metal or ceramic material are common foreign bodies.

Ask about previous eye problems including treatment, and corrective lenses if worn. If vision is impaired, was the deterioration sudden or gradual?

Examination

Topical anaesthetic drops are necessary if the eye is painful. Check the label carefully as different eye drops are often stored together. Any eye that has had topical anaesthesia must be padded until painful again, to protect the eye while normal protective reflexes are lost. Never give the patient anaesthetic drops to take home.

- *Visual acuity must be recorded for every patient (use glasses if worn, pinhole if glasses not available).*
- *Examine* the skin around the eye and evert the eyelids to check for foreign bodies.
- *Visual fields* are particularly important when retinal or cerebrovascular disease is suspected.
- *Eye movements* should be tested and feelings of double vision sought. Nystagmus and conjugate eye movements are indicative of cerebellar and brainstem function.
- *Pupils* should be examined for size, symmetry and reaction to light.
- *Ophthalmoscopy* is necessary if there is loss of visual acuity.

Slit lamp examination

The slit lamp illuminates and magnifies the cornea and the anterior chamber of the eye. Fluorescein dye makes corneal abnormalities fluoresce yellow-green in ultraviolet light. Intraocular pressure measurement is essential if there is any possibility of glaucoma.

Common diagnoses

Corneal abrasion or foreign body

Foreign bodies embedded in the cornea are usually caused by use of power tools without eye protection. The patient presents with a red, painful, watering eye, and the foreign body is usually easily visible. Use fluorescein to show corneal damage.

Use topical anaesthesia and remove the foreign body and any rust ring scraping with a dental burr or the side of a needle bevel (mount the needle on a syringe barrel to aid manipulation). Treat with antibiotic ointment, which lubricates and protects the healing cornea, and arrange review in 36–48 hours, to assess healing and check for missed foreign body or residual rust.

Welder's arc/flashburn

Electrical arc welding generates intense ultraviolet light. If a dark glass shield is not used, severe bilateral pain and redness develops several hours later. Fluorescein reveals corneal inflammation with tiny dots of fluorescence. Treatment is systemic analgesia and protection/padding of the eyes for the 2–3 days it takes to resolve.

Conjunctivitis

The patient presents with red eyes with watery discharge, usually *bilateral*, and associated with *normal visual acuity.* The cause may be one of the following.

- *Viral:* most common, sometimes after an upper respiratory tract infection, very transmissible; advise the patient to wash hands and avoid sharing towels, but no treatment is necessary.
- *Allergic:* advise the patient to use topical and systemic antihistamines available from pharmacies.
- *Bacterial:* rapid onset, purulent – consider *Gonococcus* or *Chlamydia.*

Conjunctivitis should improve within 10 days; if not, an ophthalmology review is necessary.

Subconjunctival haemorrhage

This dramatic appearance is caused by rupture of a subconjunctival vein and spread of blood below the conjunctiva. No treatment is necessary unless it occurs in the context of head injury, when it indicates a skull base fracture.

Diagnoses not to miss

Globe rupture

If globe rupture is suspected, a ring bandage is placed around the eye to prevent any pressure on the globe. Intravenous antibiotics, analgesics and anti-emetics are given: urgent CT and refer.

Intra-ocular foreign body

Suspect if there is the feeling of a foreign body and the possibility of high-energy material, yet little or no corneal damage. Urgent CT and refer.

Acute angle closure glaucoma

Rare below 60 years of age, this presents with pain, headache, blurred vision with haloes around lights, and nausea. The eye is red, feels firm, and there is a mid-sized irregular unreactive pupil. High intraocular pressure confirms the diagnosis: treatment is intravenous acetazolamide and urgent referral.

Giant cell arteritis

Occurring in the elderly, rapid visual loss is associated with headache, jaw claudication (pain on chewing), tender temporal arteries and a pale and swollen optic disc on fundoscopy. An erythrocyte sedimentation rate (ESR) >50 is likely, but the gold standard for diagnosis is temporal artery biopsy. Commence high-dose steroids immediately and refer.

Dendritic ulcer

These branching ulcers, caused by herpes simplex virus (HSV) infection are best seen with fluorescein, but can be mistaken for abrasions. Treat with topical antivirals and refer.

Orbital floor (blowout) fracture

Patients with a facial fracture should be checked for an upward gaze palsy by holding the patient's head still and moving a finger upwards 50 cm from the face. Diplopia suggests tethering of the inferior rectus muscle/soft tissue, preventing upward gaze, and need for referral.

Central retinal artery/vein occlusion

Central retinal artery occlusion (CRAO) causes *sudden* painless loss of vision, with a pale fundus except for a red macular spot, and is caused by emboli, atherosclerosis or giant cell arteritis. Central retinal vein occlusion (CRVO) is similar, but of slower onset, and is associated with diabetes and hypertension, giving swollen oedematous retinal vessels. Immediate referral is necessary for both.

Transient ischaemic attack

Patients with a transient ischaemic attack (TIA) affecting their visual cortex describe 'a curtain coming down' on their vision – sometimes known as amaurosis fugax (Chapter 42).

Retinal detatchment

Retinal detachment presents with gradual visual deterioration, floaters, flashes or field defects in middle-aged or myopic patients or in patients with diabetes. Opthalmoscopy in the Emergency Department cannot detect all cases of retinal detachment, so consider ultrasound and refer.

Ophthalmic varicella zoster virus

Shingles affecting the trigeminal nerve can manifest with pain or sensory symptoms, which precede the vesicular rash. Treat with oral acyclovir and refer.

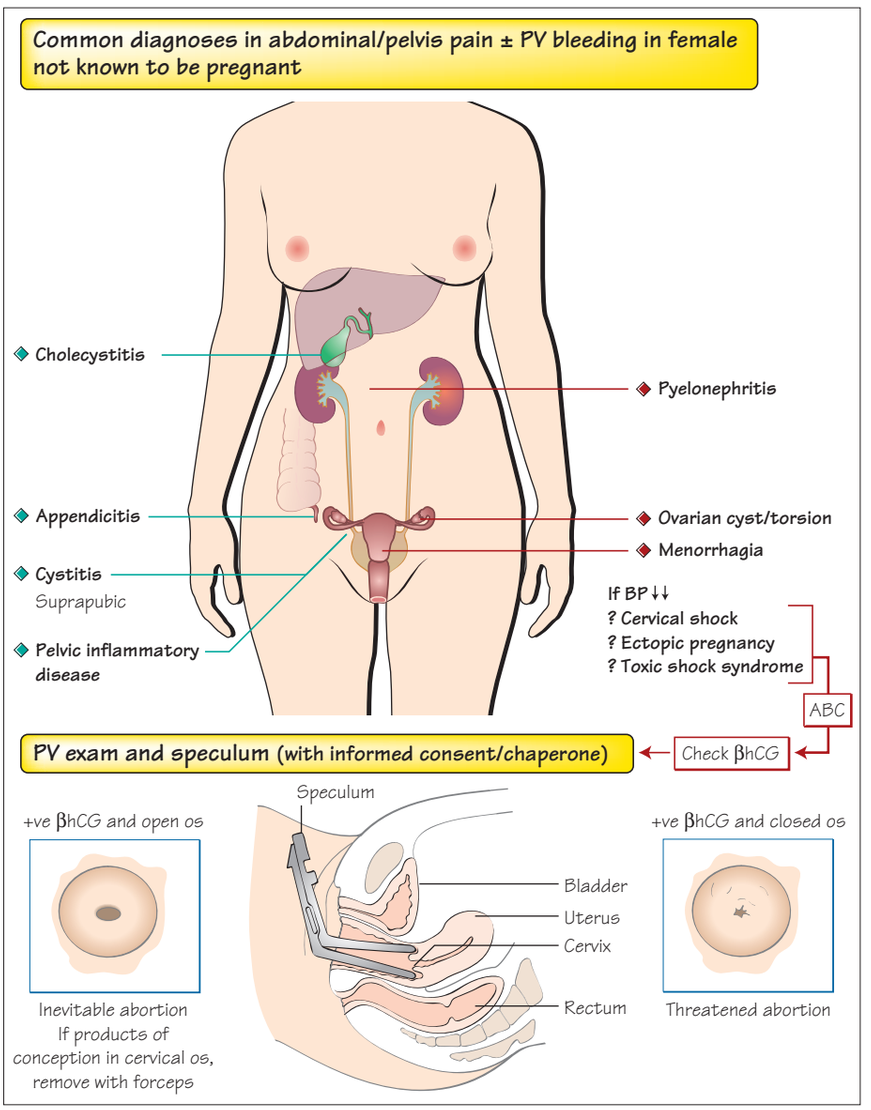
Orbital cellulitis/endophthalmitis

Any suspicion of infection in the orbit needs antibiotics, CT and referral. Pain on eye movement indicates deep infection.

Acute inflammatory eye conditions

A number of conditions can present with painful visual disturbance, and red eyes. These differ from conjunctivitis in that visual acuity is *not* normal, and referral is necessary.

23 Obstetrics and gynaecology problems



Assume that any woman of childbearing age is pregnant until proven otherwise. Pregnancy up to the time of foetal viability (approx 23/40 weeks) is managed by gynaecology, after that by obstetrics.

Resuscitation

PV bleed + abdo pain + shock = ruptured ectopic pregnancy

A ruptured ectopic pregnancy can bleed faster than blood can be replaced. Immediate surgery is necessary: ensure large bore intravenous access, with minimal volume resuscitation (Chapter 3). Speculum examination allows exclusion of possible alternatives: 'cervical shock' (see below) or toxic shock syndrome (Chapter 38).

History

Enquire about:

- Possible pregnancy, menstrual cycle including last normal menstrual period (LNMP);
- Previous pregnancies/miscarriages and Rhesus status, if known;
- Pain – site/nature and associations/radiation – shoulder tip suggests peritoneal irritation/onset – rapid/slow;
- Bleeding/discharge – volume/nature;
- Sexual history including sexually transmitted infections (STIs);
- General symptoms, and those that might indicate other causes for abdominal pain, e.g. fever, bowel and urinary symptoms (Chapters 19 and 20).

Examination

Lower abdominal tenderness suggests a gynaecological cause for pain, but also consider other causes of abdominal pain, particularly appendicitis (Chapter 19) or bowel obstruction. Estimate fundal height in pregnancy.

Speculum and internal examination

A chaperone must always be present when performing internal examination. With speculum examination, take swabs first if necessary. In bleeding in early pregnancy, if the os is open, this indicates an 'inevitable abortion'. If closed, it suggests a 'threatened abortion' or ectopic pregnancy. Manual examination allows assessment of pain on cervical movement – 'cervical excitation', which occurs in pelvic inflammatory disease (PID).

Investigations

Bedside investigations

- Urinalysis, βhCG, STI swabs, ultrasound.

Laboratory investigations

- FBC.
- Blood group if pregnancy possible.
- Quantitative βhCG according to local protocol.

Imaging

- Ultrasound is sensitive for detecting the intrauterine gestational sac and is now an essential component of assessment of problems in early pregnancy. If there is no intrauterine sac and the quantitative βhCG is >1500 units it is assumed that there is an ectopic pregnancy. There is a small risk (↑with IVF) of *heterotopic* pregnancy – simultaneous ectopic *and* intrauterine pregnancy.
- A full bladder is essential for transabodominal ultrasound, providing a 'window' through which the pelvic organs are seen. Transvaginal ultrasound can detect a gestational sac at about 4–5 weeks, 1 week before it would be visible on abdominal ultrasound.

Common diagnoses

Bleeding in early pregnancy

Significant vaginal bleeding occurs in 20% of pregnancies, and of these, half will abort. Ultrasound is essential in the assessment of these patients. Possible diagnoses include the following.

- *Ectopic pregnancy:* Presents with abdominal pain and bleeding. A positive βhCG (quantitative >1500 units) and an empty uterus confirms the diagnosis. While *ruptured* ectopic pregnancy requires resuscitation and immediate surgery as described above, many patients with stable ectopic pregnancy are treated medically, preserving the fallopian tubes.
- *Threatened abortion:* mild pain, bleeding, closed cervical os: if intrauterine gestational sac seen, reassure as most settle.
- *Inevitable abortion:* ongoing pain and bleeding, open cervical os: refer.
- *Missed abortion:* the fetus dies but is not expelled: refer.
- *Incomplete abortion:* ongoing pain/bleeding but no sac: refer.
- *Complete abortion:* closed os, no sac on ultrasound, no ongoing symptoms: no further treatment necessary.

If diagnosis is uncertain and the quantitative βhCG is below 1500 units, the quantitative βhCG should be repeated 48 hours later – in normal pregnancy, levels should double within this time. In all cases, consider anti-D immunoglobulin, provide information about support groups, and arrange outpatient follow-up.

Vomiting in mid pregnancy

Vomiting is common between weeks 6/40 and 16/40. Exclude alternative causes, treat symptomatically with intravenous fluids and metoclopramide, and discharge with follow-up if otherwise well.

Menorrhagia

After ruling out pregnancy, this common symptom is treated with tranexamic acid and mefenamic acid, with GP review.

Pelvic inflammatory disease

Vaginal discharge, fever and pelvic pain, with cervical excitation on examination. Consider STIs – take swabs and arrange follow-up for contact tracing according to local protocol.

Mid-cycle pain

This sharp pain, localised to one side of the lower abdomen, results from rupture of the ovarian follicle during ovulation. After excluding pregnancy and more serious pathology (e.g. appendicitis), reassure and advise simple analgesia: paracetamol/NSAIDs.

Diagnoses not to miss

Cervical shock

Products of conception in the cervical canal can provoke a very intense parasympathetic response, resulting in extreme bradycardia and shock. Removal of the tissue results in rapid resolution of the symptoms.

Ovarian pathology

Many ovarian cysts are incidental findings on ultrasound and cause no symptoms. Ovarian torsion is rare, and may present with non-specific low/mid abdominal pain.

Pre-eclampsia/eclampsia

The triad of hypertension, proteinurea and oedema can occur from week 12/40 to the immediate postpartum period, but is most common in the third trimester. If untreated, this can progress to full eclampsia with seizures.

Magnesium is used to increase the seizure threshold, benzodiazepines being relatively ineffective. The foetus should be monitored, and delivered at the earliest safe opportunity.

Rhesus auto-immunisation

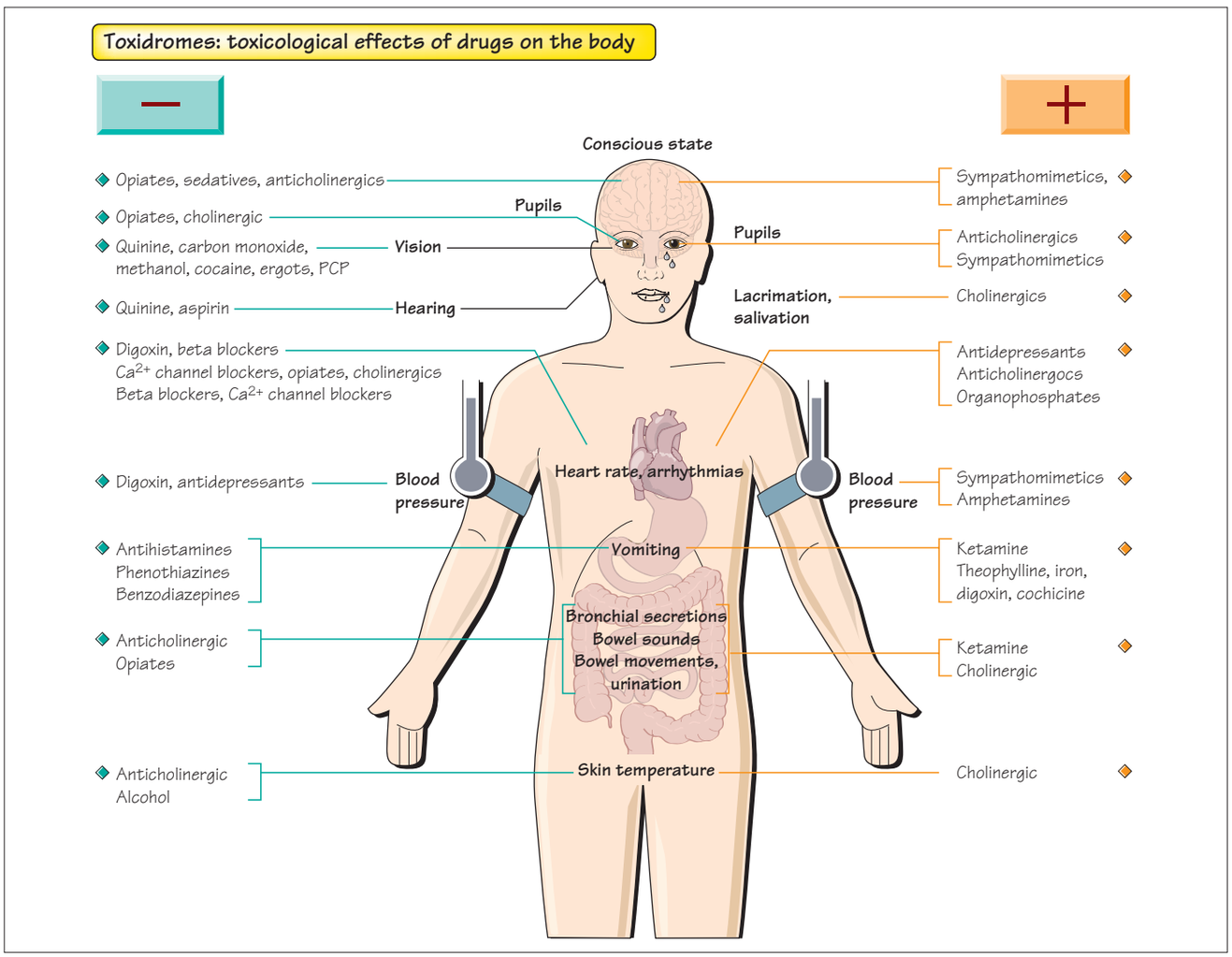
Auto-immunisation occurs if a significant amount of rhesus-positive foetal blood mixes with rhesus-negative maternal blood, causing foetal haemolysis in future pregnancies. Consider in abortion or trauma affecting a pregnant woman. A Kleihauer test detects foetal blood, but may miss small amounts. Anti-D immunoglobulin should be given according to local guidelines.

Bleeding in late pregnancy

Large amounts of blood may be lost, so large-bore cannulae are necessary. Urgent obstetric review is necessary.

- *Placental abruption:* bleeding occurs between the placenta and the uterine wall, jeopardising the foetus.
- *Placenta praevia:* the placenta extends over the cervical os, and bleeding may occur as the uterus enlarges.

24 Toxicology: general principles



Self-poisoning is the most common toxicological problem: serious adverse effects are rare with good basic supportive management. The difficulty of performing human toxicological research means that the evidence base is very limited. Expert advice on the management of poisoning is available through a system of national poisons information centres.

National Poisons Information	
Australia	13 11 26
Canada	911/local Poison Control Centre
Ireland	01 809 2566
New Zealand	0800 764 766
UK	0870 600 6266
USA	1 800 222 1222

History

A straightforward, non-judgemental tone can help establish rapport. The details of the ingestion (e.g. drugs, alcohol, suicide note, social factors and precipitants) should be documented.

Resuscitation: ABCDE

A*irway* Ensure the airway is open and protected, give oxygen if unwell.

B*reathing* Ensure adequate respiratory rate and oxygenation saturation.

C*irculation* Look for signs of shock, obtain intravenous access and bloods.

Shock responds to intravenous fluid in most cases.

Arrhythmias may need antidotes before usual treatments are effective (Chapter 25).

D*isability* Pupils and Glasgow Coma Scale, seizures, agitation.

Exclude hypoxia and hypoglycaemia.

Toxidromes are groups of physical findings suggesting certain drug ingestions (see opposite).

Agitation can be a medical emergency, putting staff and patients at risk. A combination of physical restraint and chemical sedation may be required (Chapter 27).

Many toxic seizures are self-limiting. Benzodiazepines are first-line treatment; barbiturates are second-line. Phenytoin should *not* be used in seizures related to toxic ingestions as its mechanism of action (Na^+ channel blockade) is the same as the cause of many such fits.

E***xposure*** Check temperature, check for trauma or evidence of intravenous drug use (IVDU).

Examination

Any patient who is comatose or has major ABC derangement needs resuscitation first. Poisoning can be difficult to detect clinically: patients may be asymptomatic despite having taken a lethal dose.

It is easy to assume that all signs and symptoms are attributed to poisoning. Impaired consciousness may be due to a head injury.

Management

Supportive care is the mainstay of management: ensuring the patient does not come to harm while the drugs are eliminated. Supportive care is a continuation of the principles of resuscitation above. The patient should be nursed in an environment close to a critical care area where close monitoring is possible. The observation unit of an Emergency Department is ideal.

Risk assessment

Patients should be assessed early for their risk of coming to harm:

- Risk of deterioration: patients who have taken particularly toxic drugs that may have delayed presentation, e.g. tricyclic antidepressants, beta blockers, digoxin. These patients should be observed in a high acuity area until the danger is passed.
- Risk of absconding: patients who try to leave may be intoxicated, confused, attention-seeking, psychotic, seriously depressed and intent on finishing the job, or sometimes just bored. An assessment of their mental capacity is essential (Chapter 26), to allow detention of the patient against their wishes if necessary.

Minimising systemic toxicity

Systemic effects of drugs are minimised by:

- *Decontamination* – minimisation of drug absorption.
- *Elimination* – maximising drug removal.

Specific poisons and their treatments are discussed in Chapter 25.

Decontamination

Activated charcoal

Charcoal is 'activated' by superheating, increasing its surface area to volume ratio to $900 m^2/g$, i.e. the area of a tennis court per gram. Activated charcoal *ad*sorbs drugs, minimising gut *ab*sorption. Charcoal also adsorbs drugs excreted in the bile, some of which would normally be reabsorbed by the body: the enterohepatic circulation.

Charcoal appears helpful if given soon after ingestion, ideally within one hour. It should be given only if:

- the patient is alert, and drinks it voluntarily OR
- the patient is intubated.

Charcoal is not helpful in poisoning due to alcohols (ethanol, methanol, ethylene glycol), hydrocarbons, alkalis, acids or metals.

Gastric washout, Ipecac

Gastric washout is rarely performed, as it is unlikely to offer benefit over charcoal, and carries the risk of aspiration. Washout is appropriate in a patient presenting within a few hours of ingestion of a highly toxic overdose who has already been intubated. Charcoal can be instilled after the washout. Ipecac is a plant extract that causes vomiting; it is rarely used, as it can cause serious GI side effects.

Whole bowel irrigation

Whole bowel irrigation is performed by infusing a clear inert solution (polyethylene glycol) orally until no drug residue is passed. It is useful for slow-release preparations not adsorbed by charcoal, e.g. iron, lithium. It requires close nursing supervision and is messy and so is usually performed in the intensive care unit (ICU).

Elimination

Repeated charcoal

Repeat doses of activated charcoal prevent reabsorption (and therefore enhance elimination) in drugs that undergo enterohepatic recirculation, e.g. theophylline, anti-epileptic drugs and digoxin.

Diuresis

Urinary alkalinisation enhances excretion of weak acids such as aspirin. Forced diuresis with large fluid volumes is dangerous as circulatory overload can occur.

Haemodialysis

Haemodialysis is used in severe poisoning by drugs that cannot be removed by other means, e.g. aspirin, lithium.

Investigations in poisoning

Bedside investigations

Blood glucose
ECG

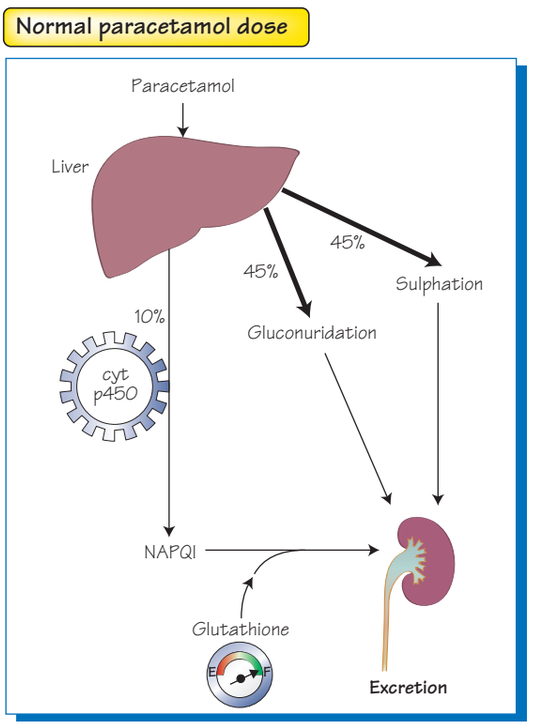
Laboratory investigations

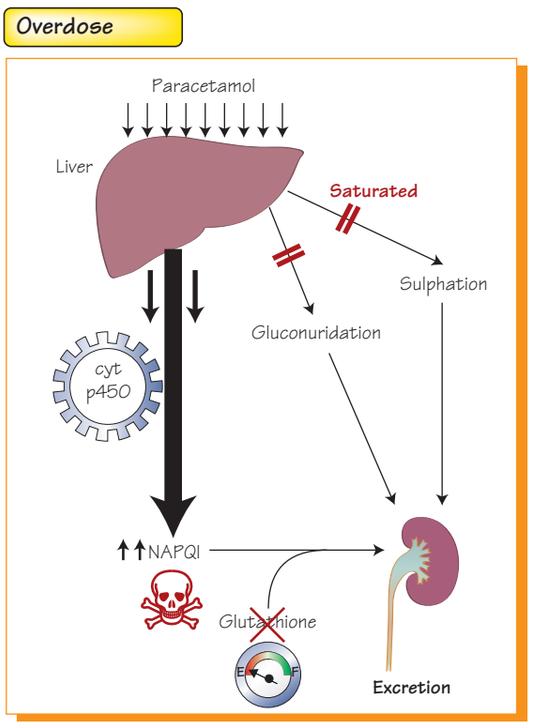
Paracetamol level at least 4 hours after ingestion
Salicylate level if history of aspirin ingestion or acidosis
Other drug levels rarely helpful in the Emergency Department
FBC, U+E
ABG, lactate in sick patients
CK if patient has been unconscious for lengthy period

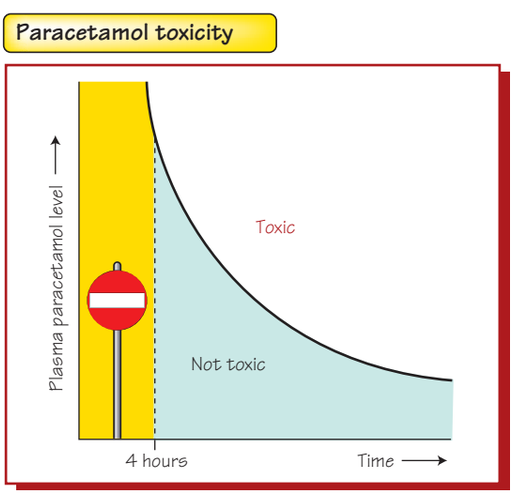
Imaging

Chest X-ray if possible aspiration
Abdominal X-ray rarely necessary, but shows radio-opaque tablets such as iron, lithium
CT of head: the clinical presentation of many poisonings often overlaps with conditions that may cause intracranial bleeding/swelling. An urgent CT scan should be obtained if there is a possibility of traumatic injury or any doubt about the diagnosis

25 Toxicology: specific poisons







Antidotes

Poison	Antidote/treatment
Benzodiazepines	Supportive care
Beta blockers	Glucagon, inotropes, pacemaker
Calcium channel blockers	Calcium, inotropes, glucagon
Carbon monoxide	Oxygen
Digoxin	Digibind (Fab fragments)
Ethylene glycol	Ethanol, fomepizole
Hydrofluric acid	Calcium gluconate
Insulin, hypoglycaemics	Glucose/dextrose >10%, glucogon
Iron	Desferoximine
Methanol	Ethanol, fomepizole
Opiates	Naloxone
Organophosphates	Atropine, pralidoxime
Paracetamol	*n*-acetyl-cysteine, methionine
Tricyclic antidepressants	Sodium bicarbonate
Warfarin	Clotting factors/vitamin K

Paracetamol (acetaminophen)

Paracetamol overdose (OD) is the most common toxicological emergency and the most common cause of liver transplant and death due to poisoning. Patients who start *N*-acetylcysteine (NAC) within 12 hours of ingestion are very likely to survive.

Mechanism of action

In overdose, normal pathways of paracetamol metabolism become saturated, forcing paracetamol down an alternate pathway: the first stage is performed by a cytochrome p450 enzyme, forming a hepatotoxic compound called NAPQI. Under normal circumstances, the NAPQI is quickly cleared from the liver by conjugation with glutathione.

After about 10 hours, the liver's supply of glutathione is exhausted, and NAPQI accumulates, damaging cells. Intravenous *N*-acetylcysteine prevents hepatic damage from NAPQI by substituting for glutathione; methionine is an oral alternative, given over several days.

Patients at increased risk of toxicity

Drugs that induce p450, such as anti-epileptics, and chronic alcohol consumption, increase the rate of formation of NAPQI. Patients with low hepatic glutathione stores, such as those with HIV, anorexia or cystic fibrosis, may also be at increased risk of hepatotoxicity.

Management

Measurement of paracetamol levels before 4 hours after ingestion is unhelpful, except to exclude paracetamol ingestion. The graph opposite predicts the need for NAC in patients above the line. NAC is given as a front-loaded infusion. Patients presenting more than 24 hours after ingestion may still benefit from NAC.

Patients who present more than 8 hours after ingestion and who have ingested >150 mg/kg paracetamol should have paracetamol levels measured and NAC commenced on arrival. Other patients should wait until the paracetamol level is known.

The prothrombin time (PT) is the single best indicator of hepatic function. Patients whose PT is rising 24 hours after ingestion should continue NAC; a PT level >36 sec at 36 hours suggests serious damage. Hepatic and renal function is monitored using PT, LFTs and U+Es.

Antidepressants

Tricyclic anti-depressants (TCAs) such as amitriptyline, imipramine and doxepin are highly toxic in overdose, and are the second most common cause of overdose deaths. By comparison, the SSRI antidepressants are much safer in overdose.

The toxic effects of tricyclic antidepressants are mainly a result of blockade of fast sodium channels, resulting in membrane-stabilising effects on cardiac and neurological cells. They also have an anticholinergic action. CNS effects include seizures, agitation and coma, while cardiotoxicity causes hypotension and ventricular arrhythmias.

Supportive management with close monitoring is essential. Acidosis and progressive lengthening of the QT_c may occur. A QRS >120 msec predicts toxicity; >160 msec indicates imminent seizures and/or ventricular fibrillation (VF).

Sodium bicarbonate is an effective antidote. The alkali reduces the free drug and the large sodium load helps overcome the blockade. Phenytoin should not be used for seizures as it also blocks sodium channels.

Opiates

Opiate overdose causes coma, hypoventilation and small pupils. Patients who are apnoeic or in whom the cause for coma is in doubt, e.g. possible trauma, should be given naloxone, a short-acting antagonist.

In patients who are breathing, it is best to just give oxygen and wait, as there are hazards in giving naloxone.

- Danger to staff from needles around intravenous drug users.
- Naloxone causes acute opiate withdrawal symptoms, which may dissuade drug users from calling for help in the future.
- Naloxone is short-acting, so there is a danger that a patient may wake, run away and collapse when the naloxone wears off. Therefore if naloxone is indicated, it should be a large dose, e.g. 1.6 mg i.m.

In patients who have (iatrogenic) opiate-induced hypoventilation where one does not want to reverse the analgesic effect – just the hypoventilation, this may be reversed by very small doses of i.v. naloxone e.g. 40–80 µg. Naloxone infusion may be necessary with long-acting opiates.

Benzodiazepines

Benzodiazepines (BDZ) are often taken as part of a mixed overdose. Benzodiazepines have a very good safety record in overdose and may protect against seizures e.g. when taken with tricyclic antidepressants.

Flumazenil is a short-acting benzodiazepine antagonist that can precipitate acute benzodiazepine withdrawal and intractable seizures, so should not be used in the Emergency Department.

Alcohol

Ethanol is often taken with overdoses. Paradoxically this may provide a degree of protection from the toxic effects of some overdoses by competing for metabolic pathways.

Toxic alcohols such as methanol and ethylene glycol (antifreeze) are metabolised by alcohol dehydrogenase to toxic compounds. Toxicity can be prevented by either blocking alcohol dehydogenase using fomepizole, or giving ethanol, which is preferentially metabolised. The toxic alcohol can be removed by haemodialysis.

Salicylates

Aspirin overdose, while relatively common, rarely needs treatment. Most patients with significant overdose complain of tinnitus. Direct stimulation of the respiratory centre gives initial hyperventilation and respiratory alkalosis, progressing later to a metabolic acidosis. High levels of salicylates indicate the need for alkaline diuresis (dilute sodium bicarbonate i.v.) or haemodialysis.

Digoxin

Digoxin overdose may be acute or chronic. Chronic digoxin overdosage will give bradycardia, and patients complain of yellow/green vision – xanthopsia. Acute digoxin overdose may cause coma, brady- or tachyarrhythmias. Digoxin has a specific antidote – digoxin antibody fragments.

Iron

Iron overdose is uncommon, but serious. Abdominal X-radiography can identify number and progress of tablets, and serum iron concentrations predict toxicity. Gastrointestinal absorption of iron is normally tightly regulated. In overdose, damage to the gut mucosa allows unregulated iron absorption, exacerbating toxicity. Bowel decontamination with whole bowel irrigation and chelation using intravenous deferoxamine may be necessary.

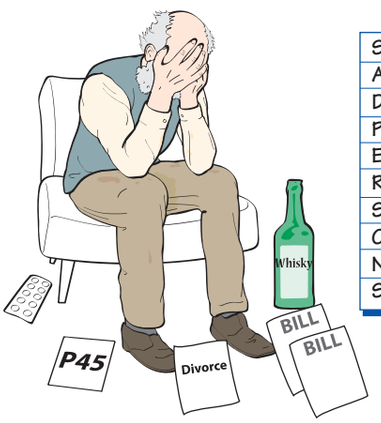
Stuffers and packers

Body *stuffers* are usually street-level drug dealers who are caught and decide to swallow the evidence. Body *packers* are people who seek to smuggle drugs by concealing them within the body. Pyrexia >38°C or pulse >120 indicate significant toxicity – benzodiazepines are useful for agitation.

Stuffers are more likely to suffer toxic effects as the drugs are not packaged to withstand gastrointestinal transit, although the drugs are relatively impure, compared to those ingested by packers. Abdominal radiography and ultrasound can diagnose packers, who need charcoal and whole bowel irrigation.

26 Psychiatry: self-harm and capacity

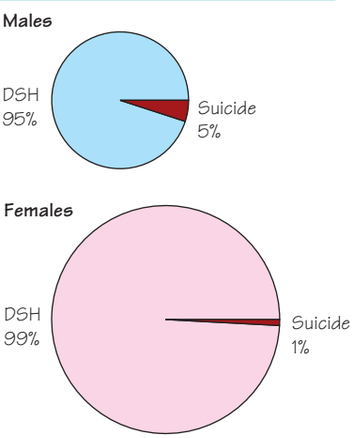
Risk assessment: modified SADPERSONS



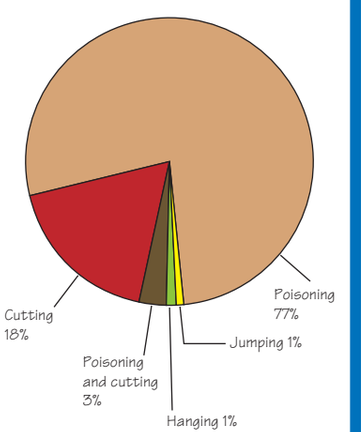
S ex – male	1
A ge >45 or <19	1
D epression/ Hopelessness	2
P sychiatric history	1
E thanol or drug dependence	1
R ational thinking loss – psychotic or organic	2
S ocial isolation	1
O rganised (pre-meditated) suicide attempt	2
N o spouse – single/widowed/divorced	1
S uicidal – ongoing intent	2

A SADPERSONS score of more than 7 indicates at least a moderate risk further harm. Such patients must be prevented from leaving the hospital until a formal psychiatric evaluation has been completed

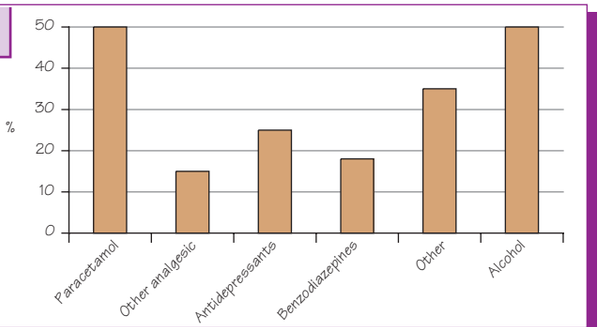
Ratio of self-harm to suicide rates



Method of self-harm (UK)



Poisoning – method



Deliberate self-harm

Most of the patients who self-harm (e.g. overdose or cut themselves) do so as a response to a stress in their life. Common precipitants are problems with relationships or finances.

The majority of deliberate self-harm (DSH) patients seen in the Emergency Department do not have ongoing suicidal intent; of those presenting with an overdose, only a very small fraction go on to commit suicide. Therefore the challenge is to identify patients with a high ongoing risk of suicide.

The modified SADPERSONS scoring system can identify patients at high risk of subsequently committing suicide. The SADPERSONS score should not be viewed in isolation; other indicators that a suicide attempt is associated with a high level of intent are:

- *A violent method*, e.g. hanging, falls, weapons.
- *Avoidance of discovery* where the person has attempted to avoid being found.
- *Premeditation*: most suicide attempts are impulsive, and often related to alcohol consumption. Evidence of having 'put one's affairs in order', e.g. making or changing a will, suggests a high degree of planning. 'Suicide notes' are common, but a carefully considered letter is a more worrying indicator than a scrawled note.

DSH patients need medical treatment if necessary and, if assessed as low risk, may be discharged with appropriate community-based follow-up. If at moderate or high risk, these patients should have a psychiatric assessment before discharge.

Personality disorders

Within the group of DSH patients there are many more patients with personality disorders than with mental illnesses such as depression or schizophrenia. A personality disorder is *not* a mental illness per se, but a pattern of behaviour that is consistently outside social norms. Patients with personality disorders are orientated, do not have hallucinations, delusions or thought disorders; they have normal senses and memory.

Patients with a personality disorder may present in a very similar way to a patient with mental illness. Both may harm themselves, and students are often surprised to find that doctors and nurses appear unconcerned by these patients. This is because patients with personality disorders may be manipulative and attention-seeking, and indifference to their behaviour is less likely to reinforce it.

There are three distinct subgroups of personality disorders:

A Suspicious/odd behaviour
B Impulsive/antisocial/emotionally manipulative behaviour
C Anxious/dependent behaviour.

The patients seen in the Emergency Department with self-harm tend to be from group B, who are more likely to be emotionally labile and form fragile relationships. The label 'borderline personality disorder' stems from the outdated notion that these patients were 'on the border' between psychosis and neurosis. Patients with group B personality disorders may also have drug and alcohol problems and chaotic lives.

Patients with personality disorders are not generally helped by treatments used for serious mental illness: psychiatrists try to avoid admitting them to hospital as this can make the situation worse.

Capacity, consent and ethics

Sometimes a patient may refuse treatment for a potentially life-threatening overdose, or may want to leave the Emergency Department before their treatment is complete. The doctor must then assess whether the patient's autonomy should be overridden to allow treatment.

Ethics

The ethical principles that guide medical treatment are:

- *Beneficence* – doing good.
- *Non-maleficence* – not doing harm.
- *Autonomy* – respecting a patient's decisions.
- *Justice* – fairness.

In some countries, this situation is covered by mental health legislation, in others by legislation covering consent. In England and Wales, the Mental Capacity Act (2005) formalised a framework to assess patients whose mental capacity to consent to treatment is in doubt.

Mental capacity

Assesment of mental capacity is person, time and decision specific: can *this* person make *this* decision at *this* time?

To establish mental capacity to a patient must be able to:

- *Understand* the choices being presented to them
- *Retain* the information about the choices for enough time to be able to
- *Weigh* the relative merits of the choices, then be able to
- *Communicate* the decision to others.

If a patient fails the test for mental capacity (most commonly on the ability to weigh information rationally) then this decision and the reasons for it must be recorded in the notes. Treatment that is necessary to preserve the patient's life may then proceed against the patient's wishes. This may include sedation necessary to safely permit life-saving interventions.

Mental capacity can be difficult to assess in patients with pre-existent disabilities or communication difficulties, and these points may help guide assessment.

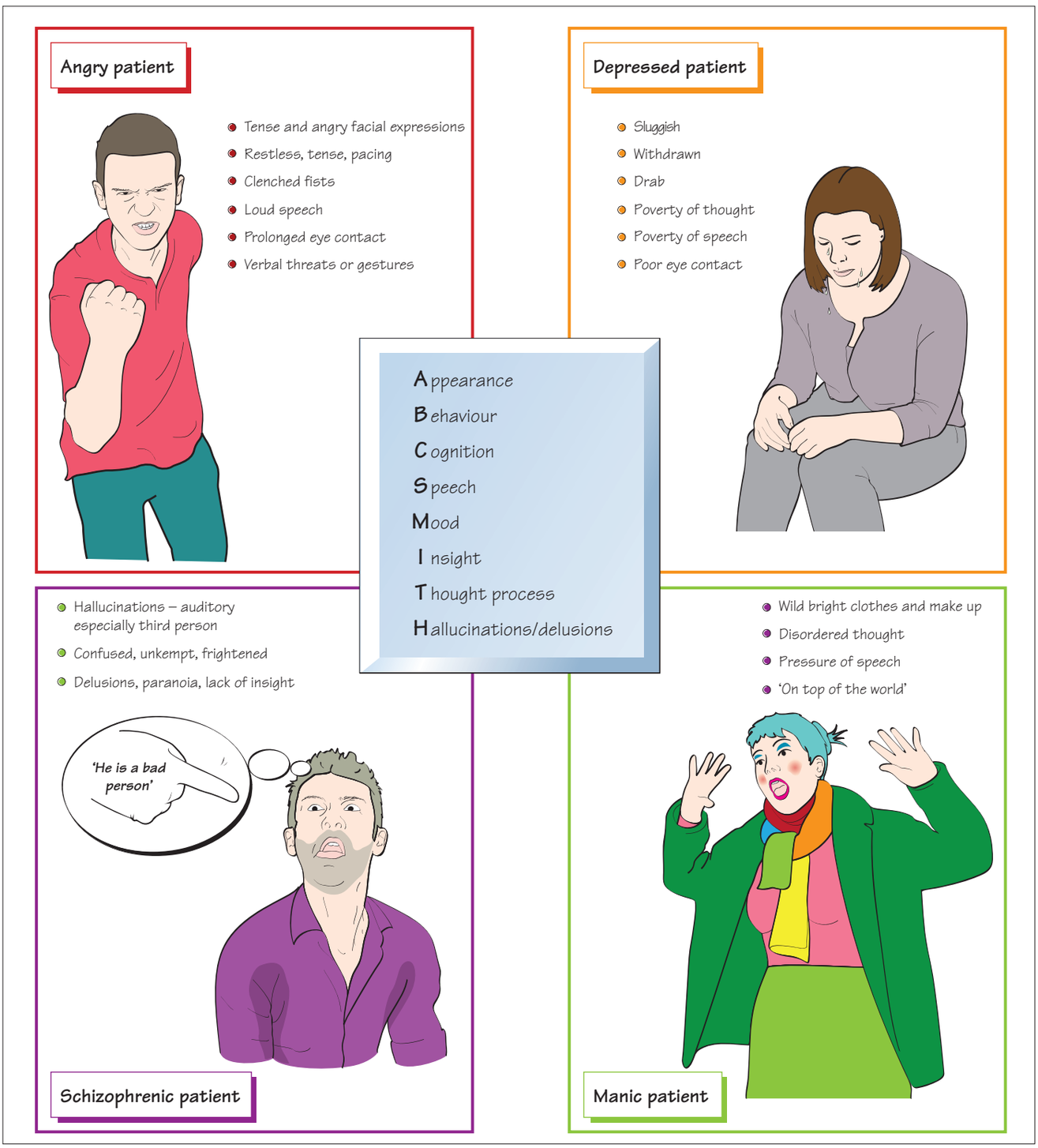
- Everything possible should be done to maximise a patient's capacity.
- An unwise decision by the patient does not automatically prove lack of capacity.
- Capacity should be presumed until evidence to the contrary.
- Decisions should act in the best interests of the patient.
- If a decision has to be made, it should be the least restrictive option that meets the patient's needs.

Advance Directives

Advance Directives, sometimes (confusingly) known as 'living wills', are a legally binding method to specify treatment decisions in the event that a patient does not have capacity to make those judgements. Not all countries have similar legislation, and such documents must be signed and witnessed, preferably by a medical witness who can verify that the patient had capacity to make that decision at that time.

The advance directive should include a statement that the treatment should be withheld *even if the patient's life is put at risk*.

27 Psychiatry: the disturbed patient



Mental state examination: ABCSMITH

The mental state examination is a structured way of collecting and presenting information about patients with psychiatric symptoms.

Appearance grooming/hygiene/dress/eye contact
Behaviour agitation/withdrawn/gestures/co-operation
Cognition inattention/orientation/reasoning
Speech speed/fluency/pressure/volume
Mood sad/happy/angry/flat/labile/apathetic
Insight presence/degree
Thought process content/possession/speed/flow
Hallucinations/delusions presence/organisation/system

The acutely disturbed patient

The majority of incidents of agitation and aggression in the Emergency Department are related to drug and alcohol use. Often it is not possible to immediately identify the underlying cause, e.g. drug or alcohol use or withdrawal, personality disorder, acute mental illness or delirium brought on by an organic disease process. Therefore any treatment system must be robust enough to deal with all these possibilities.

Principles

Prediction and prevention

Patients with a risk or history of violence should be searched by hospital security before being seen by clinical staff. Observation of patients may pick up warning signs. Patients should be interviewed in a quiet room that has outward-opening doors and an alarm system. Adequate numbers of staff should be nearby.

De-escalation and observation

De-escalation is the verbal and non-verbal behaviour that is used to calm a potentially confrontational situation. Seclusion is an option if a suitable room is available, together with a staff member for observation.

Disturbed patients respond positively to honesty and respect and can be presented with options. Limited negotiation may be attempted, e.g. to persuade the patient to take an oral benzodiazepine, but both sides must understand that failure to comply will result in restraint. Such negotiation is more likely to be effective when backed with a credible 'show of force'.

Restraint

If de-escalation has not worked, then restraint is necessary to protect the patient, other patients, the public and members of staff.

If physical restraint is to be used, a minimum of six trained staff are necessary to minimise the risk of injury to staff or the patient. Restraint is initially physical, followed by pharmaceutical sedation. Whenever a patient is restrained or sedated, close clinical and physiological monitoring is essential to ensure patient safety.

Review

When a patient has been restrained or sedated, they should be examined thoroughly for signs of organic disease. Psychiatric wards have limited medical facilities and it is prudent to perform any screening tests in the Emergency Department. This should include bedside tests – urine/glucose, bloods if indicated, e.g. FBC, U+E, LFTs, Ca^{2+}, TFTs (thyroid function tests). Chest X-ray, CT brain and lumbar puncture may be necessary depending on the history, e.g. head trauma.

Sedation

- *Benzodiazepines*, e.g. lorazepam, midazolam, diazepam. These drugs are generally safe and predictable. Routine users of benzodiazepines develop tolerance to these drugs, which will therefore have minimal effect.
- *Neuroleptics*, e.g. haloperidol, chlorpromazine, droperidol. These 'major tranquillisers' offer prolonged sedation and are the first choice for patients with psychotic features.

Benzodiazepines and neuroleptics can be usefully combined for the most agitated patients. Current UK recommendations favour lorazepam and/or haloperidol. If a patient is co-operative, these may be administered orally, otherwise intramuscular injection is effective.

Delerium (organic) or psychiatric symptoms

It can be difficult to distinguish organic from psychiatric disease. Delirium is the cognitive and consciousness impairment that may result from organic disease, e.g. sepsis, drugs, metabolic disorders.

Organic disease is suggested by:
- rapid onset
- fever
- non-sensory neurological abnormalities
- disorientation and confusion
- visual hallucinations.

Psychiatric disease is suggested by:
- chronic symptoms, previous psychiatric problems
- delusional beliefs, paranoia, disorganised thought processes
- auditory hallucinations – especially third person.

Patients with psychiatric illness may also have organic disease. Alcohol and drug use and/or withdrawal may cloud the picture and may need to resolve or be treated before a definitive decision can be made.

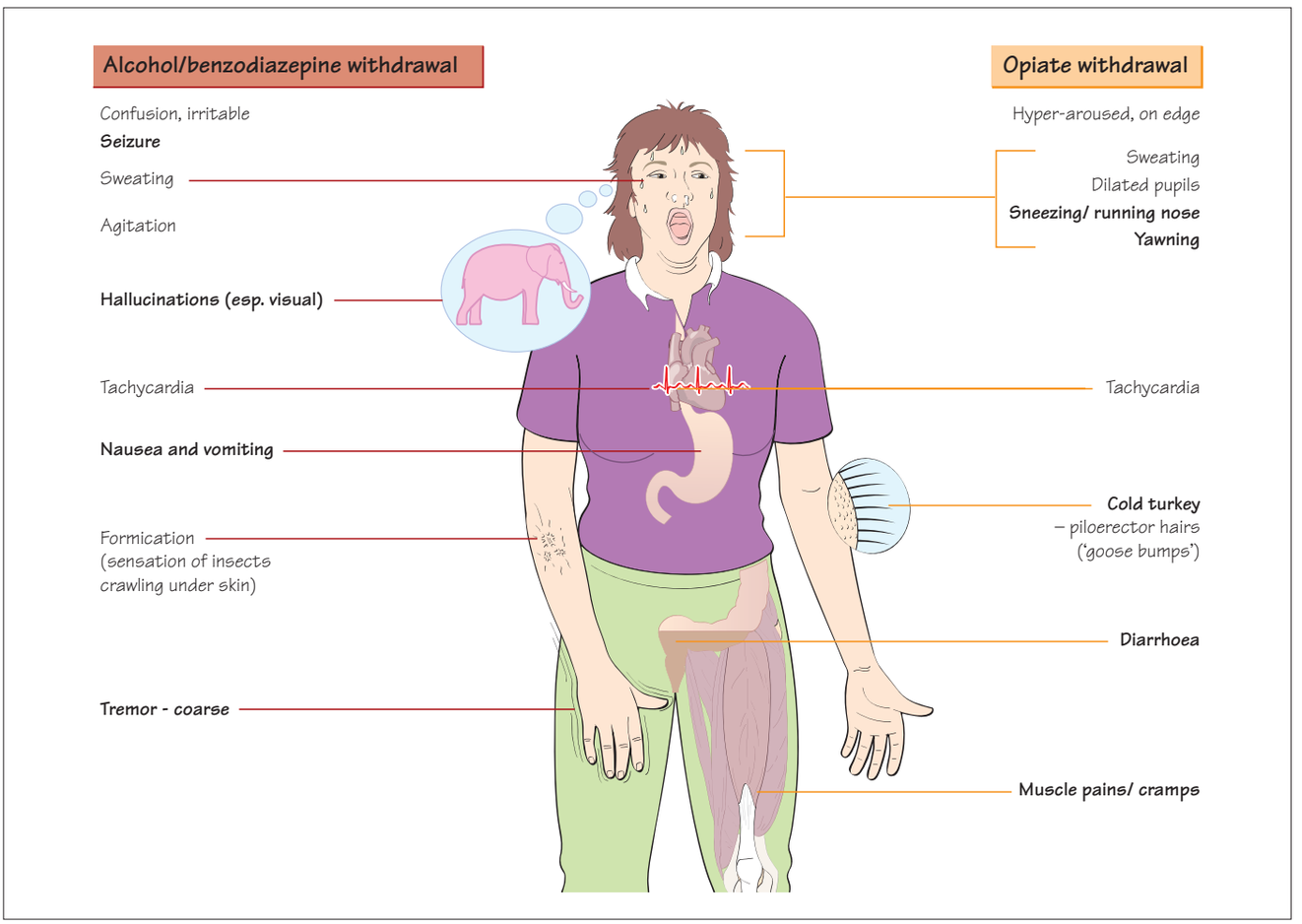
Factitious disorders

The Emergency Department sees a small number of particularly challenging patients with symptoms that have no organic basis: factitious disorders or Munchausen's syndrome. The Internet ensures that such patients are well informed about what symptoms they might have. It is very easy for doctors to become part of the problem, by continuing to search for disease despite absence of objective evidence of any disease process.

The symptoms may be very dramatic, yet the patient may appear unconcerned. The patient may appear to be in great distress, yet their pulse and blood pressure will be normal. Pseudocoma, pseudoseizures, dramatic and non-anatomical patterns of paralysis may occur. True factitious illness should be differentiated from malingering or drug-seeking where there is an obvious secondary gain.

Patients with a history of factitious illness may also develop organic illness. Safe diagnosis of factitious diseases using the minimum investigations necessary can be difficult, and early involvement of a senior doctor is advisable. When challenged, these patients usually leave rapidly and have no interest in engagement with psychiatric services.

28 Observational medicine



Observational medicine units are an expansion of traditional Emergency Department activity. Patients who are likely to be fit for discharge within 24 hours are held in a unit managed by the Emergency Department.

The reason for the rise of observational medicine is the pressure to better use hospital beds in the main part of the hospital, together with a better appreciation of the risks of hospitalisation. Emergency physicians are less inclined to hold onto patients than other specialties and are motivated to ensure rapid discharge wherever possible.

Features that differentiate an observational medicine ward from other wards in the hospital are:

- Frequent consultant-led ward rounds, e.g. three times a day, to ensure rapid progress and decision making.
- A discharge plan is necessary for entry to the unit.
- Ready availability of other health professionals, e.g. physiotherapists, occupational therapists, social workers.

The observation unit should not be used as an alternative to making a decision. There should be rapid turnover of patients: bed occupancy is often 200–300% per day.

Minor injury in elderly patient

A relatively minor injury in an older patient may have a disproportionate effect on their ability to cope safely at home. Common injuries, often the result of a fall, that may incapacitate patients include fracture of the neck of the humerus, Colles' fracture and fractured pubic rami. There are a vast number of potential causes for elderly patients to fall, but common medical causes that can be diagnosed and treated within the Emergency Department include:

- Arrhythmias: ECG.
- Postural hypotension: lying and standing blood pressure.
- Infections: urine dipstick, chest X-ray.
- Medication: polypharmacy increases the risk of drug interactions and adverse effects.

Review in the observation unit by a multidisciplinary team, which includes occupational therapy, physiotherapy and social workers as well as medical and nursing staff, can ensure a rapid, safe discharge from hospital.

Homelessness

The Emergency Department is sometimes the only medical contact that homeless people have. Appropriate emergency medical care is given but it is not the job of the Emergency Department to resolve the multiple chronic and social problems these patients often have. Mental illness and drug and alcohol dependence are common in this group, but there is a limited amount that the Emergency Department can do unless there is ongoing community support.

'Frequent flyers'

There is a small group of patients who come to the Emergency Department very often. This is usually due to a combination of factors, which may include personality disorders, drug and alcohol problems, self-harm, loneliness, homelessness and mental illness. These are difficult patients to manage as they are often very experienced at manipulating healthcare staff. It is best if they are looked after by the most senior staff available, to avoid one group of health professionals being played off against another.

Domestic violence and elder abuse

An Emergency Department visit is an opportunity for intervention: staff need to be alert to the possibility of non-accidental injury in vulnerable people. Drug and alcohol problems often coexist with domestic violence. Emergency Department staff cannot force someone to seek help, but can provide contact details and a quiet area with a telephone. Elder abuse may be particularly difficult to diagnose due to the high rate of natural falls and bruising, together with poor memory.

Drugs and alcohol

Although drug and alcohol dependence are often managed by psychiatrically trained doctors, they are *not* mental illnesses per se. However, drug and alcohol use often coexists with mental illness; this could be viewed as the patient's self-medicating.

Patients sometimes present to the Emergency Department seeking 'a detox'. A detoxification is a (usually temporary) drug-free period. The Emergency Department is not the place for elective management of withdrawal of drugs or alcohol: this can be safely managed in the community.

Patients who are dependent on drugs or alcohol often end up in the Emergency Department as a result of falls, fights, and so on. These patients may experience withdrawal in the Emergency Department, and this must be identified and managed, otherwise they may have seizures or the patient will leave, compromising their medical care. Withdrawal symptoms do not respect class or educational attainment, and one must be alert to symptoms in unexpected patients.

If alcohol problems are suspected, use the CAGE questionnaire (Chapter 29) and, if positive, expect to have to manage withdrawal, and ensure the patient is aware of community support on discharge.

Alcohol and benzodiazepine withdrawal

Withdrawal from alcohol and benzodiazepines is potentially dangerous, as fits may occur. *Thiamine*, usually combined with other B and C vitamins, is given to prevent Wernicke's encephalopathy (characterised by confusion, ataxia, ophthalmoplegia and nystagmus) and Korsakoff's psychosis, the disastrous irreversible consequences of chronic thiamine deficiency common in alcoholics.

Alcohol withdrawal is generally best managed using front-loaded oral *diazepam*, which has active metabolites with a long half-life (2–4 days). Large doses of diazepam, e.g. 20 mg every one to two hours, are given according to symptoms; multiple doses are often required.

Once the patient's symptoms have been controlled, the patient does not have to stay in hospital, and does not need diazepam on discharge, as its pharmacokinetics will ensure a tapering dose of benzodiazepines.

Opiate withdrawal

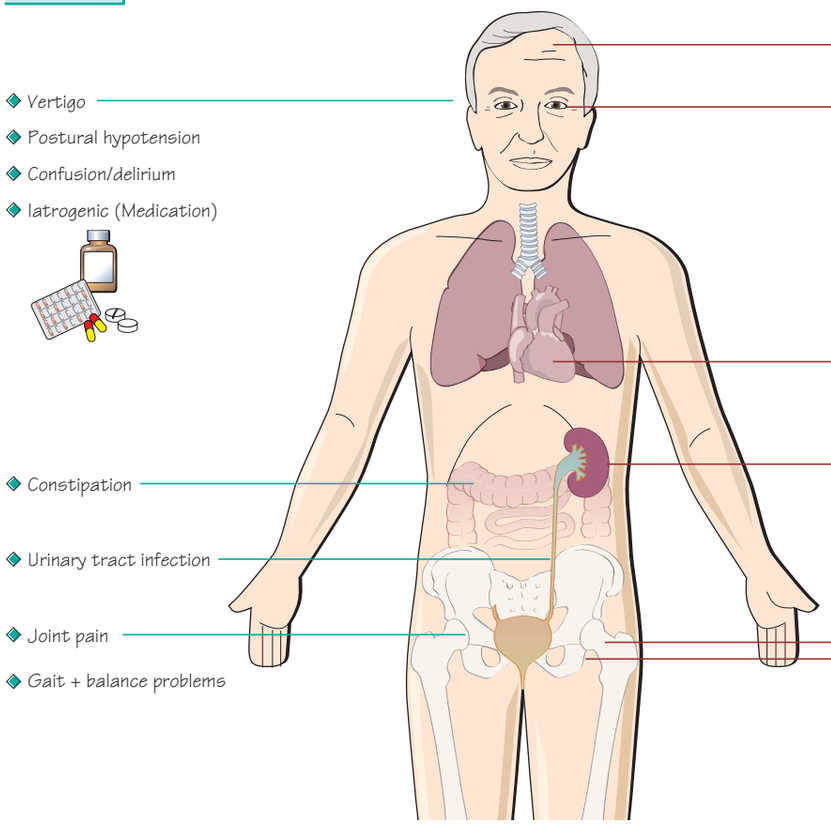
Opiate withdrawal is unpleasant, but not dangerous. Musculoskeletal symptoms respond to NSAIDs; high-dose diazepam helps with agitation and nausea. Clonidine, an alpha antagonist with mild opiate agonist properties, can also be used.

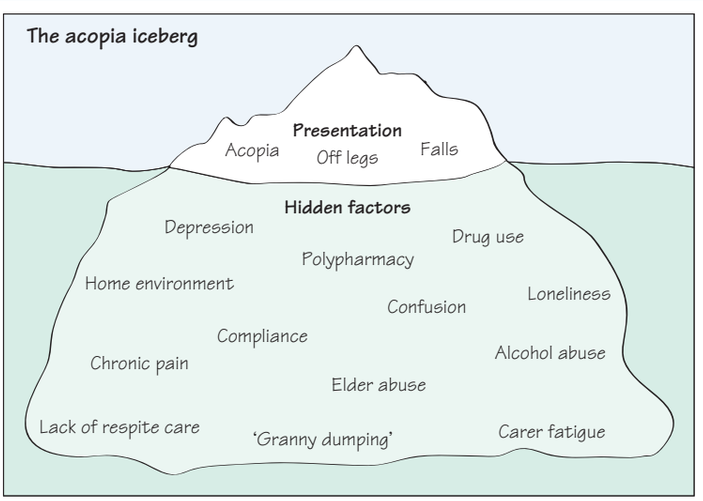
Other patient groups

Other patient groups who often end up in the observation ward include:

- Overdose (Chapters 24 and 25).
- First fit (Chapter 43).
- Minor head injury (Chapter 11).
- Post-sedation (Chapter 6).

29 Loss of function and independence





CAGE screening for alcohol problems

Have you tried to **C**ut down your drinking?
Do you get **A**nnoyed when people criticise your drinking?
Do feel **G**uilty about your drinking?
Do you sometimes have an **E**ye-opener in the morning?
A score of 2 suggests alcohol problems; 3 or 4 is diagnostic [The +LR for a score of 1,2,3,4 are 1.5, 4.5, 13, 100 respectively, and the –LR for a score of 0 is 0.14]

Abbreviated mental test (AMT4)

Age
Year
Date of Birth
Prime Minister/President
A score of less than 4 indicates mental impairment

Caring for patients with multiple long-term conditions, frailty, and functional or cognitive impairment is an increasing challenge for families and health and social services. These patients often present to the Emergency Department with a relatively minor functional decline, but one that renders the patient 'off their legs', 'bedbound' or 'acopic' (unable to cope).

The Emergency Department can offer a rapid, thorough, medical and social assessment, with the aim of making an early decision as to whether the patient should be:

- Discharged home ± increased nursing/social support.
- Transferred to a rehabilitation bed.
- Admitted to a medical bed.

Such decisions require the range of skills of many different health professionals to be integrated, but can provide safe and effective care and avoid unnecessary acute hospital admissions.

History

An early part of the history and examination of these patients should be their Abbreviated Mental Test (AMT4) score. If this suggests impairment (score less than 4), the rest of the history may need cautious interpretation. It is always useful to corroborate the history from at least one other source (carer, relative).

Key questions are:

- Why is the patient here now?
- Is there a (reversible) reason for a loss of function?

Presenting complaints

- *Precipitants*: ask in detail about any recent symptoms, specifically adequacy of oral intake, loss of weight, bowel or urinary symptoms, and symptoms of infection.
- *Falls*: falls are common and a careful history is necessary to work out the aetiology. Ask about the frequency and pattern of falls, possible precipitants such as problems with gait and balance, and syncope/pre-syncope.
- *Medications*: an accurate list of medications and doses, including recent changes, provides insight into the current medical conditions being managed and possible drug interactions and side-effects, both of which are common in the elderly. Assess likely compliance with medication.
- *Cognition*: what is the current level of function, cognitive and mental status, vs the usual level? Ask about alcohol consumption and mood disturbance. Many patients successfully conceal high alcohol consumption: use the CAGE questions.

Ask about family and social supports, and if external services are currently in place to support the patient with their Activities of Daily Living (ADLs) at their home.

Examination

General examination

Look for evidence of infection, chest and urine being the most common. Minor trauma may cause occult hip and pelvic fractures in an osteoporotic patient.

Cardiorespiratory examination

Look for valvular disease and heart failure. Postural hypotension is common and should be excluded in every patient.

Neurological examination

Assess speech, gait and cerebellar function. Look for weakness or changes in reflexes that would indicate stroke. Full sensory examination of limbs is not practical in the Emergency Department, but pinprick and vibration should be performed to search for evidence of peripheral neuropathy (diabetes, vitamin B_{12}/folate/thiamine deficiency).

The rare but treatable condition of normal pressure hydrocephalus occurs in elderly patients and presents as ataxia, incontinence and confusion. CT confirms the diagnosis.

Investigations

Bedside investigations

- Blood glucose.
- ECG.
- Urine dipstick.
- Lying and standing blood pressure.

Laboratory investigations

- FBC and U+E.
- Creatine kinase for any patient 'found on floor'.

Imaging

- Chest X-ray.
- CT head if risk of subdural haematoma, or if stroke is suspected.

Management

Identify medically reversible reasons for a loss of function. This might include treatment of urinary tract infection, constipation, or stopping a recently commenced medication. Beware occult fractures of neck of femur and neck. CT or MRI may be necessary in cases where there is pain but an equivocal plain X-ray examination.

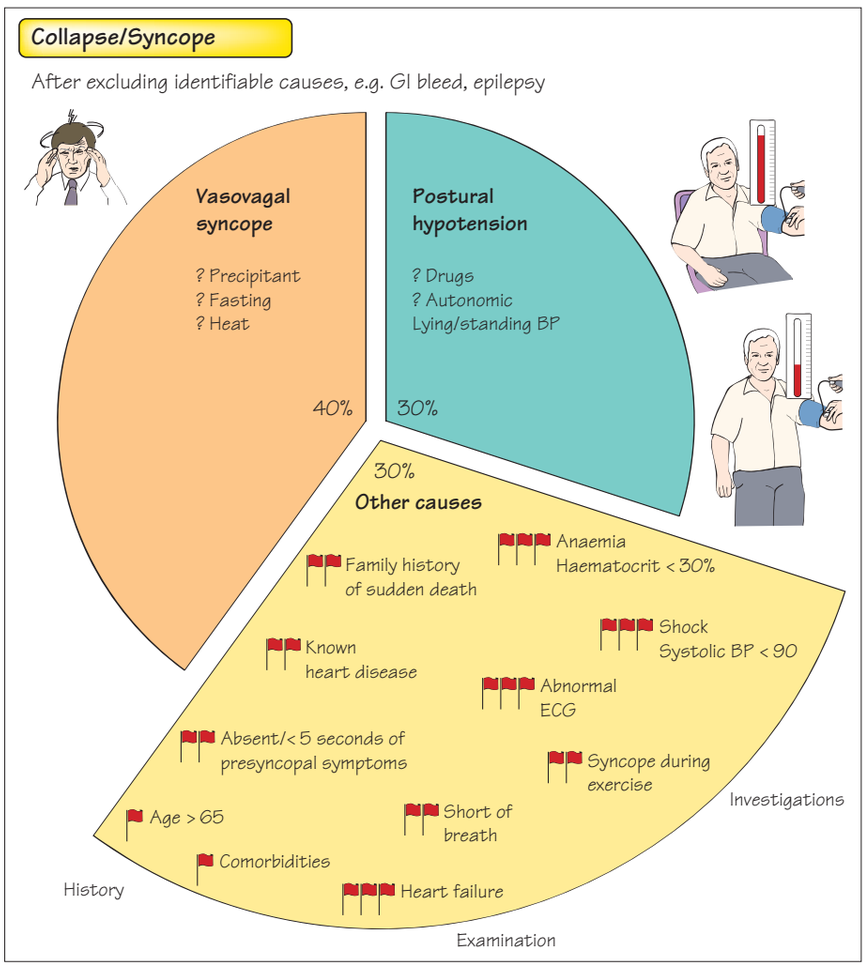
Patients may require new equipment (e.g. a walking aid), a home hazards assessment, or additional home or community services. These may be combined with a rehabilitation programme involving exercise and physical therapy.

Disposal: who can go home?

Patients at risk of falls require a formal falls risk assessment before discharge, and either admission or early referral to a falls clinic. Reversible medical conditions identified and treated within the Emergency Department (e.g. UTI, mild hyponatraemia, polypharmacy) do not necessitate hospital admission.

Discharge patients who appear well, in whom serious pathology has been excluded and who are safe to return to their normal home environment after screening by the multidisciplinary team.

30 Syncope, collapse and falls



Syncope is a brief loss of consciousness that resolves spontaneously and completely. It is distinct from vertigo, seizures, coma and states of altered consciousness. Syncope accounts for 1–2% of Emergency Department visits but 6% of hospital admissions.

History

A detailed history taken as soon as possible after the event offers the best chance to achieve an accurate diagnosis. Patients are by definition asymptomatic by the time they have reached the Emergency Department and may not remember the event. An eyewitness account is therefore particularly valuable in corroborating what happened during the episode, particularly in estimating duration of loss of consciousness and any possible seizure activity.

Single most important question: 'Do you remember falling?'

Nature of the episode: first episode, or recurrent?

Environment

- Lying down/seated, with absent or brief prodromal symptoms (? cardiac).
- Within 2 minutes of standing (? postural hypotension).
- Stressor (pain or emotional upset) with longer prodromes and associated pallor, sweating, nausea or vomiting (? vasovagal/arrhythmia).
- Head rotation or movement (? carotid sinus sensitivity); visit to the toilet (? micturition or defecation syncope); or coughing (? cough syncope).
- Exertion (? cardiac arrhythmia, HOCM), hot/cold?

Prodromal symptoms Prodomal symptoms might be things like 'feeling faint', 'feeling like I was going to pass out', 'feeling the

room spin around me'. Prodromal symptoms that last a few seconds suggest an arrhythmia, those lasting a few minutes suggest vasovagal syncope.

What happened during the syncope? Witnesses may report falls or trauma during the episode. A few seconds of mild tonic-clonic activity may be caused by syncope 'anoxic jerks', and do not necessarily signify epilepsy. Absence seizures mimic syncope.

What happened after the syncope? A long period of confusion occurs after generalised seizures. Neurological deficits, e.g. hemiparesis, may occur after a seizure ('Todd's palsy') or may represent a TIA/stroke (Chapter 42).

Medical and drug history

Polypharmacy, drug interactions and compliance are a particular problem in the elderly. Antihypertensive agents and vasodilators, e.g. for angina or cardiac failure, cause postural hypotension, and many psychoactive drugs prolong the QT interval.

Examination

Examination focuses on the cardiac and neurological systems. Examine specifically for carotid bruits, valvular disease, outflow obstruction or evidence of heart failure. All patients should have postural blood pressure measured.

Abdominal pain may indicate bleeding or abdominal aortic aneurysm (AAA). Rectal examination should be performed, with testing for blood using a faecal occult blood testing kit.

Tongue biting (lateral margins) or incontinence suggests a seizure, but absence does not rule it out. Neurological examination should include a Glasgow Coma Scale (Chapter 10), cranial nerves, limb tone, power and reflexes.

Investigations

Bedside investigations

- Glucose and ECG in all patients. ECG monitoring may capture intermittent (paroxysmal) arrhythmias.
- Urine for UTI and βhCG if pregnancy possible.
- Ultrasound to rule out AAA.

Laboratory investigations

- FBE, U+E, troponin if cardiac disease a possibility.

Imaging

- CT is indicated if there is suspicion of a seizure, or new/focal neurological findings.

Treatment: identify or stratify

Identify

After resuscitation if necessary, patients with an identifiable cause of their syncope should be managed according to the cause.

- Syncope + shortness of breath = shortness of breath (Chapter 36).
- Syncope + chest pain = chest pain (Chapters 34 and 35).
- Syncope + headache = headache (Chapter 41).
- Syncope + gastrointestinal bleed = gastrointestinal bleed (Chapter 40).

Also identifiable from the history and examination will be patients with the two most common causes for syncope:

Vasovagal (neurocardiogenic) syncope

Vasovagal syncope (VVS) causes about 40% of syncopal episodes, and may be precipitated by a stressful event, or lack of food or drink. VVS is usually preceded by pallor, sweating, nausea and dizziness, but in the elderly, these prodromal symptoms may be short or non-existent.

Vagal parasympathetic outflow causes bradycardia; when cardiac output falls, the sympathetic-driven tachycardia and vasoconstriction fails to remedy the situation in time, and there is transient loss of cerebral perfusion. When the patient collapses, venous return increases and cardiac output is restored.

As VVS is a 'benign' diagnosis, there is a significant danger that junior Emergency Department doctors attribute a patient's syncope to VVS too easily: all such patients should be reviewed by a senior doctor.

Postural hypotension

Under normal circumstances, moving from lying or sitting to standing causes a reflex rise in heart rate and blood pressure, to maintain cerebral perfusion. Drugs (vasodilators) and autonomic dysfunction (diabetes, Parkinson's disease) may interfere with this response, which causes about 30% of syncope episodes.

To test for postural hypotension, the BP should be measured repeatedly after moving to vertical, until the BP rises. Treatment is by treating the underlying cause, with appropriate follow-up by GP or outpatient clinic.

Stratify

One is then left with approximately 30% of patients with no clear cause for the syncope, and the aim shifts to risk stratification. Clinical guidelines have not proved robust, but key criteria predicting need for admission are shown opposite. Of these criteria, cardiac failure seems a particularly important predictor of sudden death, probably due to ventricular arrhythmias.

Diagnoses not to miss

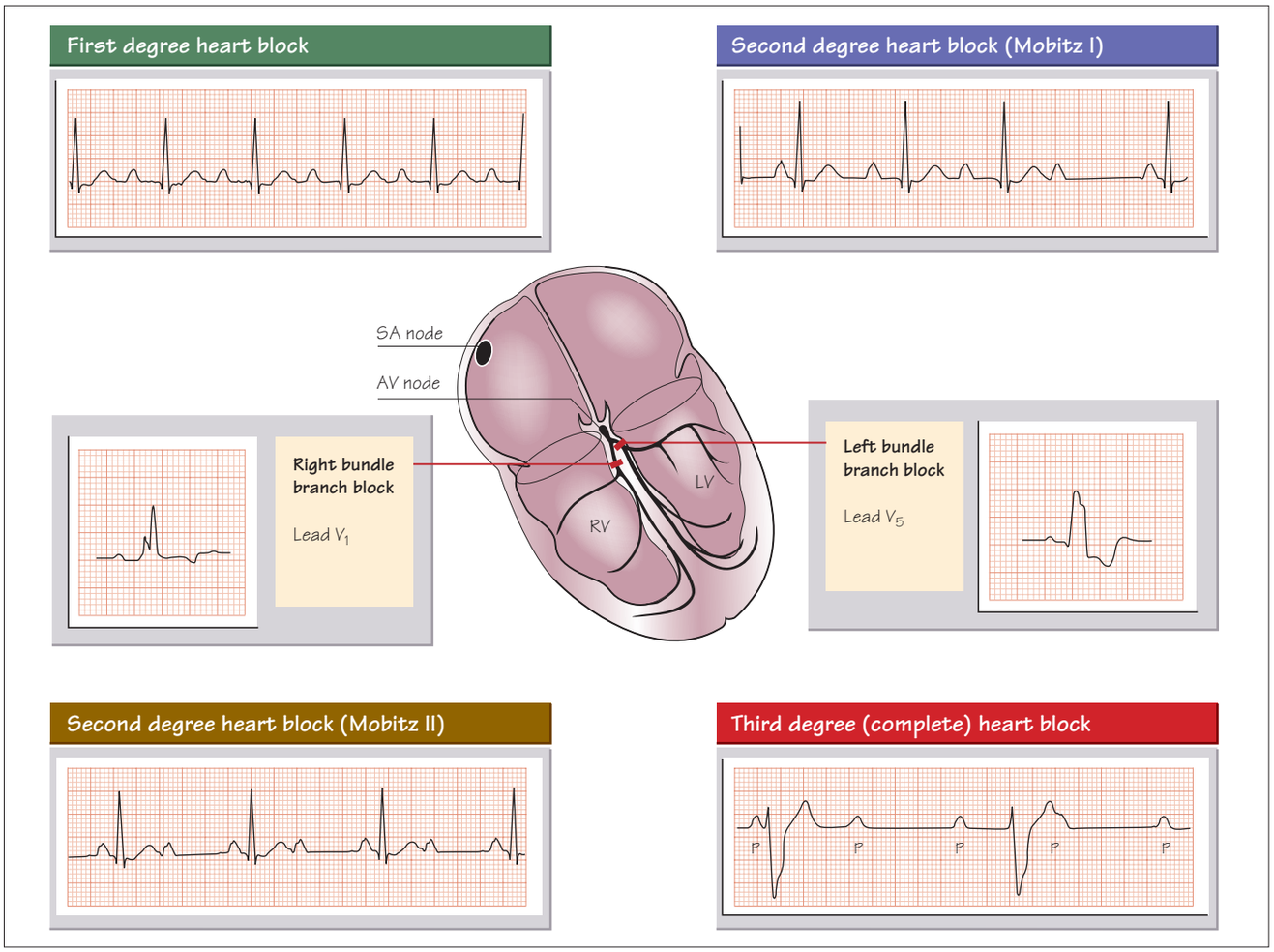
Conditions predisposing to tachyarrhythmia

- *Long QT syndromes (>460 msec)* may be congenital or acquired. Correct electrolyte abnormalities, and avoid medications that prolong QT (e.g. phenothiazines, benzodiazepines, antidepressants).
- *Short PR interval (<120 msec)* together with a slurred upstroke of QRS complex (delta wave) suggest an accessory pathway between the atria and ventricles, e.g. Wolff-Parkinson-White syndrome.
- *Brugada syndrome (RBBB with ST elevation V1–3) and short QT interval (<300 msec)* are congenital conditions that predispose to ventricular arrhythmias.

Conditions predisposing to bradyarrthmia

Sinus node dysfunction, heart block and vagal hypersensitivity – see Chapter 31.

31 Slow heart rate



Bradycardia is a heart rate below 60 beats per minute (bpm). Bradyarrhythmia is rhythm disturbance with a ventricular rate below 60 bpm. Bradyarrhythmias usually result from a defect in the heart's intrinsic conduction system or drugs affecting these areas.

The different parts of the conduction system have different intrinsic rates of depolarisation.

- 60 bpm: sinoatrial node (SAN).
- 50 bpm: atrioventricular node (AVN).
- 40–50 bpm: bundle of His leading to right and left (anterior and posterior) bundles of specialised conductive myocardial cells that conduct from AVN down the interventricular septum.
- 30–40 bpm: ventricular myocardium – the Purkinje fibres that distribute depolarisation around the myocardium.

Therefore, under normal circumstances, the SAN is the pacemaker, as it has the fastest intrinsic rate. If the SAN fails, the AVN takes over as the pacemaker but with a slower rate. If the AVN fails, the ventricles will beat on their own, but at a very slow rate.

Investigations

Bedside investigations

- Blood glucose.
- ECG.
- Cardiac device interrogation. If the patient has an implanted device, e.g. pacemaker, this may have recorded what occurred.

Laboratory investigations

- U+E, LFTs. Ensuring $K^+ > 4$ mmol/L, minimises the risk of arrhythmias.
- Thyroid function tests (TFTs).
- Troponin I.
- Ca^{2+}, Mg^{2+}.

Imaging
- Chest X-ray.
- Echocardiogram.

Common diagnoses

Right bundle branch block
Blockage of the single right bundle does not cause bradycardia, is relatively common and not necessarily pathological: it can be a normal variant. On ECG there is a broad RSR wave (i.e. M-shaped, ≥0.12 sec = 3 small squares wide) in the leads looking at the right side of the heart, V1 and V2.

Left bundle branch block
Blockage of both the left bundles, anterior and posterior, is always pathological, and may be a feature of acute coronary syndrome (ACS). Left bundle branch block (LBBB) implies significant damage to the interventricular septum and, although it does not cause bradycardia, it slows conduction of the wave of depolarisation, impairing contraction of the ventricle. The ECG shows a broad RSR wave (i.e. M-shaped, ≥0.12 sec = 3 small squares wide) in the leads looking at the anterolateral side of the heart II, aV_L and $V_{3–6}$. The abnormal depolarisation and repolarisation of the left ventricle means that ST abnormalities normally associated with ACS cannot be interpreted.

First-degree heart block
In first-degree heart block, *all 'P' (atrial) waves are conducted, but slowly*. The delay occurs in transmission through the AVN, resulting in a P-R interval of more than 0.20 sec = 5 small squares = 1 big square. On its own, first-degree heart block does not need treatment.

Second-degree heart block: Mobitz type I
In second-degree heart block, *some 'P' (atrial) waves are not conducted*. Mobitz type I (Wenkebach) results in a progressive delay in conduction of P waves through the AVN until a P wave is not conducted. This is a relatively benign phenomenon, and can occur in inferior myocardial infarction (MI) as the right coronary usually (70%) supplies the AVN, but rarely needs pacing.

Diagnoses not to miss

Second-degree heart block: Mobitz type II
In second-degree heart block, *some 'P' (atrial) waves are not conducted*. Intermittent conduction of P waves in a fixed ratio, i.e. 1:2, 1:3 is termed Mobitz type II. Mobitz type II heart block implies significantly more damage to the conducting system than Mobitz type I, particularly in the context of an anterior myocardial infarction, when pacing is necessary.

Third-degree (complete) heart block
Third-degree heart block occurs when *no 'P' (atrial) waves are conducted*. The atria beat regularly, as do the ventricles, but there is no association between the two. An artificial pacemaker is usually necessary to preserve cardiac output, and to avoid long pauses which may precede asystolic cardiac arrest.

Extreme bradycardia, pauses
Pauses between ventricular beats of over 2 seconds and heart rates below 50 bpm are likely to be significant, especially if the patient is symptomatic. Possible causes include:
- *Sinus node dysfunction* (sick sinus syndrome, brady/tachy syndrome) may cause both bradycardia and tachycardia at different times, and occurs in older patients due to fibrosis of the sinus node. Inpatient assessment for permanent pacemaker (PPM) ± antiarrhythmics is usually necessary.
- *Vagal hypersensitivity*: e.g. micturition syncope, carotid sinus hypersensitivity – abnormal sensitivity of the heart to normal vagal stimulation can result in bradycardia and pauses. Treatment is by avoidance (sitting down, avoiding pressure on neck) and cardiology review for consideration of further measures (drugs, PPM).
- *Iatrogenic*: Excessive beta blocker, digoxin or rate-controlling calcium channel blocker.
- *Normal finding*: Athletes may have heart rates in the 40s and occasionally in the 30s. This may be accentuated by the normal physiological 'sinus arrhythmia' resulting from changes in venous return due to variation of intrathoracic pressure.

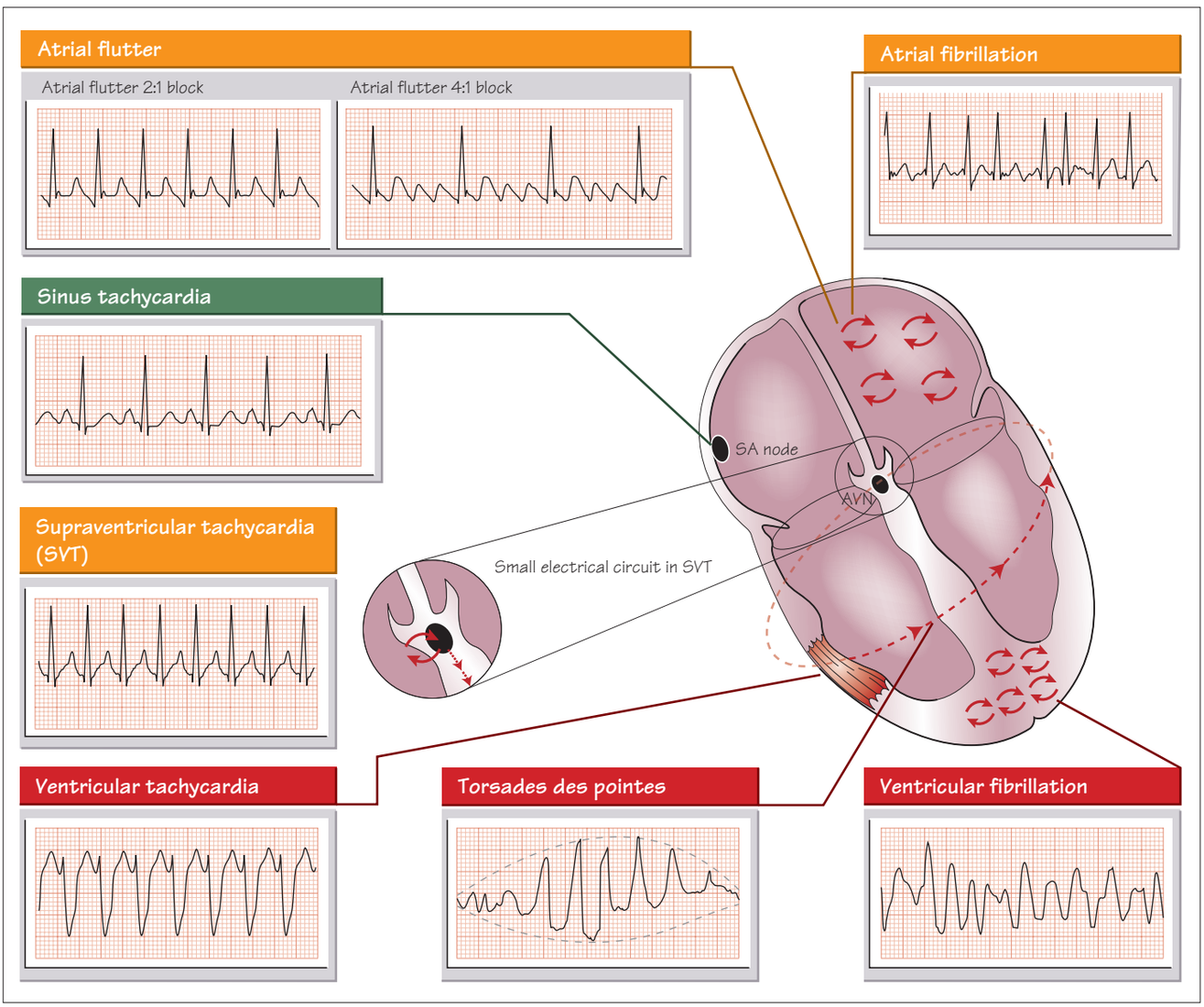
Electrolytes and the patient's thyroid function should be checked, as subclinical hypothyroidism is common in the elderly. Treatment is by treating the underlying cause, but in the acute setting, *atropine* 500 micrograms i.v. blocks the vagal tone, and can be repeated to a maximum of 3 mg.

If further stimulation is required, a low-dose adrenaline infusion or a temporary pacemaker may be necessary. Some defibrillators can perform transthoracic pacing by delivering small electric currents between the pads on the chest to stimulate the heart to beat.

Bifascicular and trifascicular block
The combination of right bundle branch block and left axis deviation, which signifies partial left bundle branch block, is known as bifascicular block. The combination of bifascicular block and first-degree heart block is known as trifascicular block, which is not a good descriptor.

It is particularly important to identify these blocks in patients presenting with falls, or who will need surgery. Trifascicular block may intermittently become complete heart block, particularly during anaesthesia.

32 Fast heart rate



Tachycardia vs tachyarrhythmia

Tachycardia is a heart rate above 100 beats per minute (bpm). Tachyarrhythmia is a rhythm disturbance with a ventricular rate of more than 100 bpm.

Narrow vs broad complex

Tachyarrhythmias usually occur because the heart's intrinsic rate regulation system described in the previous chapter is overridden. The QRS complexes are caused by ventricular depolarisation. If the ventricle's normal (fast) electrical distribution system, the Bundle of His and left and right bundles, are still used by the arrhythmia, then the QRS complexes will be *narrow*, as usual. However, if the impulse bypasses this network, and instead relies on (slow) conduction between normal cardiac muscle cells, the ECG complexes will be *broad*.

Regular vs irregular

Regular complexes imply a degree of stability in the underlying electrical circuits, whereas irregular complexes are caused by chaotic electrical activity.

Paroxysmal

Arrhythmias that are not consistent, i.e. come and go, are termed 'paroxysmal'.

Clinical assessment

A rapid assessment needs to be performed to assess whether the patient is significantly unwell; this is usually fairly clear. It can be more difficult is to establish whether the tachyarrhythmia is the cause of the patient being unwell, or a response to an underlying cause. If it is a simple tachycardia, then often there is an underlying cause. Further assessment and treatment depends on the arrhythmia found.

Investigations

Bedside investigations

- Blood glucose.
- ECG.
- Cardiac device interrogation. If the patient has an implanted device, e.g. pacemaker, this may have recorded what occurred.

Laboratory investigations

- U+E, LFTs. Ensuring $K^+ > 4$ mmol/L, minimises the risk of arrhythmias.
- Thyroid function tests (TFTs).
- Troponin I.
- Ca^{2+}, Mg^{2+}.

Imaging

- Chest X-ray.
- Echocardiogram.

Common diagnoses: atrial

Sinus tachycardia

Sinus tachycardia occurs when the heart is driven faster than usual. Causes include:

- Response to illness, e.g. shock, sepsis.
- Drugs – β_2 agonists such as salbutamol.
- Thyrotoxicosis.
- Anxiety – if other causes excluded.

Treatment is correction of the underlying cause.

Atrial flutter/fibrillation

The chaotic micro-circuits of *atrial fibrillation* (AF) are seen as an *irregular baseline* of the ECG, whereas the larger, more consistent, *atrial flutter* waves are seen as a *sawtooth* pattern at 300 bpm. Of these flutter impulses, only some are conducted through the AVN to the ventricles: a simple ratio governs this, e.g. 2:1 giving 150 bpm, 3:1 giving 100 bpm or 4:1 giving 75 bpm.

In atrial *fibrillation*, conduction is not consistent, resulting in a heartbeat that is irregular in *time* but also in *volume*, as there is no atrial contraction to fill the ventricles consistently.

Treatment depends on patient stability, myocardial status and duration of arrhythmia.

- A patient who is unwell with a low blood pressure due to atrial fibrillation needs urgent electrical cardioversion, with sedation.
- Patients who are stable may be cardioverted with drugs, or, if the arrhythmia is likely to be chronic (older patients), the ventricular rate may be controlled using beta blockers or digoxin.
- If the patient is stable and the arrhythmia has been present for more than 48 hours, they should be fully anticoagulated before cardioversion to prevent embolism of intracardiac blood clots that may have formed.
- Drugs commonly used for cardioversion include flecainide (only if the heart is structurally normal), amiodarone or sotalol.
- Anticoagulation should be considered in patients who have long-term or paroxysmal atrial fibrillation, as this reduces the risk of stroke.
- If the patient has heart failure and atrial fibrillation, both need to be treated.

Supraventricular tachycardia

Commonly causing a ventricular rate of 160–180 bpm, supraventricular tachycardia (SVT) is caused by an abnormal electrical circuit in or near the AVN. Every time an impulse goes round the circuit, it also sends an impulse down the Bundle of His.

Treatment aims to break the electrical circuit by reducing transmission in the AVN. This can be achieved by:

- Increasing the vagal drive to the AVN, e.g. Valsalva manoeuvre (ask patient to blow plunger out of a 10 mL syringe) or carotid sinus massage.
- Pharmacologically blocking the AVN using adenosine, which temporarily upsets the adenosine/cAMP balance, preventing transmission for a few seconds.

If a patient has SVT but has a ventricular conduction problem, e.g. left bundle branch block, this may look like VT. If SVT is treated as VT, no harm will come to the patient, but if VT is treated as SVT, great harm may occur. A simple yet robust rule of thumb is:

> Broad Complex Tachycardia + Ischaemic Heart Disease
> = Ventricular Tachycardia

Diagnoses not to miss: ventricular

See also Chapter 33.

Ventricular tachycardia

Ventricular tachycardia usually results from a single focus of abnormal electrical activity within the ventricles that produces rapid ventricular activation at about 180–220 bpm. Patients who are unstable with VT, e.g. low blood pressure or chest pain, require immediate electrical cardioversion. Patients who are stable may be cardioverted with drugs, e.g. amiodarone.

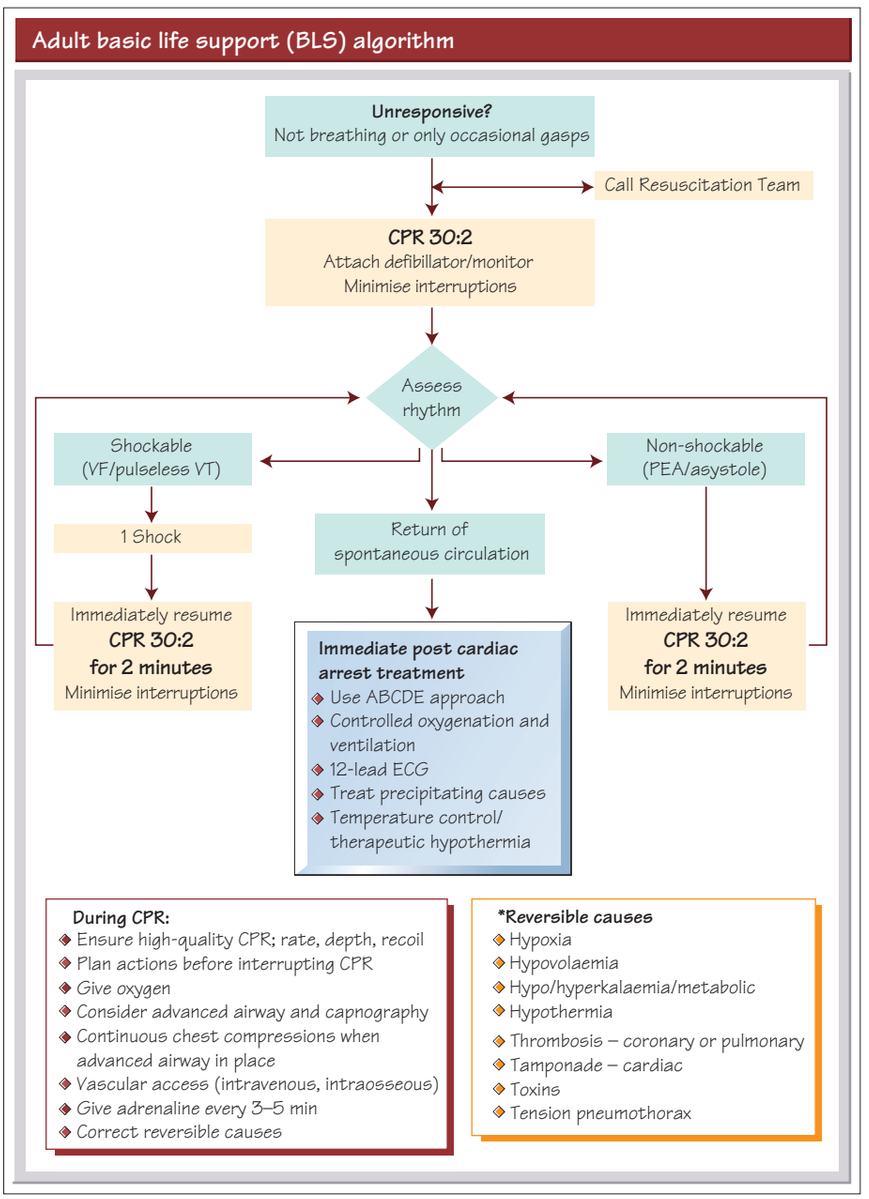
Torsades des pointes

Meaning 'twisting of points', this rare arrhythmia is caused by the focus of VT moving around the myocardium. Because the ECG sees this three-dimensional activity in one plane, the amplitude of the VT appears to vary like a sine wave. Treatment is as for VT, together with intravenous magnesium.

Ventricular fibrillation

Chaotic electrical activity in the ventricles – ventricular fibrillation (VF) – means there are no organised cardiac contractions, and therefore no cardiac output. Without electrical cardioversion to restore normal rhythm, or cardiopulmonary resuscitation (while awaiting cardioversion), there will be no blood perfusing the brain, which is irreversibly damaged within 5 minutes.

33 Cardiac arrest



Ischaemic heart disease is common, and arrhythmias are a common mode of death in otherwise normal 'hearts too good to die'. These arrhythmias are treatable with cardiopulmonary resuscitation (CPR) and defibrillation, which has been one of the success stories of pre-hospital medicine.

Research into causes and treatment of cardiac arrest using human subjects is ethically challenging, which explains the lack of high-level evidence in this area, and why international guidelines can change radically. Ventricular fibrillation and tachycardia are described in Chapter 32.

Asystole

Asystole is the complete absence of electrical activity and is the final pathway for all untreated cardiac arrests, and therefore may indicate that the heart is functionally dead. However there are reversible causes of asystole: see the '4Hs and 4Ts' opposite.

Pulseless electrical activity

Pulseless electrical activity (PEA) occurs when the ECG trace looks normal(ish), i.e. one would expect the rhythm to result in cardiac

output, but there is no pulse. This suggests that the heart is beating, but there is some mechanical reason for its lack of output:

- Tension pneumothorax increases hemithoracic pressure, and also causes mediastinal shift, which kinks the great vessels (aorta and vena cavae).
- Hypovolaemia: there is no blood to pump.
- Tamponade: fluid in the pericardiac sac under pressure prevents venous inflow in diastole.
- Thrombosis: thrombus obstructing right ventricular outflow (pulmonary embolus) or coronary arteries (myocardial infarction).

These and other potentially reversible causes of arrest are listed opposite – the '4 Hs and 4Ts'.

Cardiopulmonary resuscitation

Chest compressions

Chest compressions provide a (reduced) cardiac output by squeezing the heart between the sternum and the spine. Chest compressions need to be of adequate force, depth (one-third of chest wall diameter) and rate – 100/minute to be effective. Even short gaps in compressions significantly reduce the effective coronary perfusion, so must be avoided wherever possible.

Ventilation

The role of ventilation compared to chest compression has been progressively downgraded. Performing ventilation is a barrier to members of the public starting CPR, and chest compressions alone give a similar outcome in most adult situations. This is not the case in children, where hypoxia is the commonest cause of cardiac arrest.

Care must be taken with intubated asthma/ COPD patients: over-ventilation results in hyperexpanded lungs and increased intrathoracic pressure. This in turn reduces venous return to the heart, causing PEA cardiac arrest. Sustained pressure over the anterior chest wall decompresses the chest, restoring venous return.

Drugs

Adrenaline/vasopressin

Adrenaline/epinephrine reduces blood flow to muscle and the gut, preserving blood flow to vital organs: the myocardium, the brain and the kidneys. It is given once every 4 minutes. Anoxic damage to the brain and the kidney predict a poor outcome from an ICU admission. Vasopressin (ADH) is given once only and has a similar action to adrenaline; however, it is more expensive and no survival advantage has been demonstrated.

Amiodarone

Amiodarone is an anti-arrhythmic drug given for persistent ventricular fibrillation or broad complex tachycardia. All anti-arrhythmic drugs reduce heart contractility (negatively inotropic), which is undesirable in cardiac arrest, but amiodarone seems the 'least bad' in this respect.

Defibrillation

Defibrillation is indicated in ventricular arrhythmias (VT/VF) with no pulse. Defibrillators use a DC (monophasic) or AC (biphasic) pulse of electric current to depolarise the entire heart simultaneously. Biphasic defibrillators are smaller, lighter, and as effective as the older monophasic machines.

The success of defibrillation depends on the time to defibrillation. If *no CPR is performed*, this is a linear relationship, with 90% success at 1 minute, to 0% at 10 minutes. If CPR is performed, the rate of deterioration is much slower, and brain and kidney will remain perfused.

When giving defibrillation, every 5 seconds between stopping chest compressions and defibrillation halves the chance of successful defibrillation.

Automatic defibrillators that help the rescuer with a series of voice prompts have been successfully used by non-experts, and *no training is necessary to use them*. They are widely available in public areas such as transport hubs and large entertainment venues.

Return of spontaneous circulation

In the case of return of spontaneous circulation (ROSC), there are a number of things to remember in addition to 'Airway/Breathing/Circulation'.

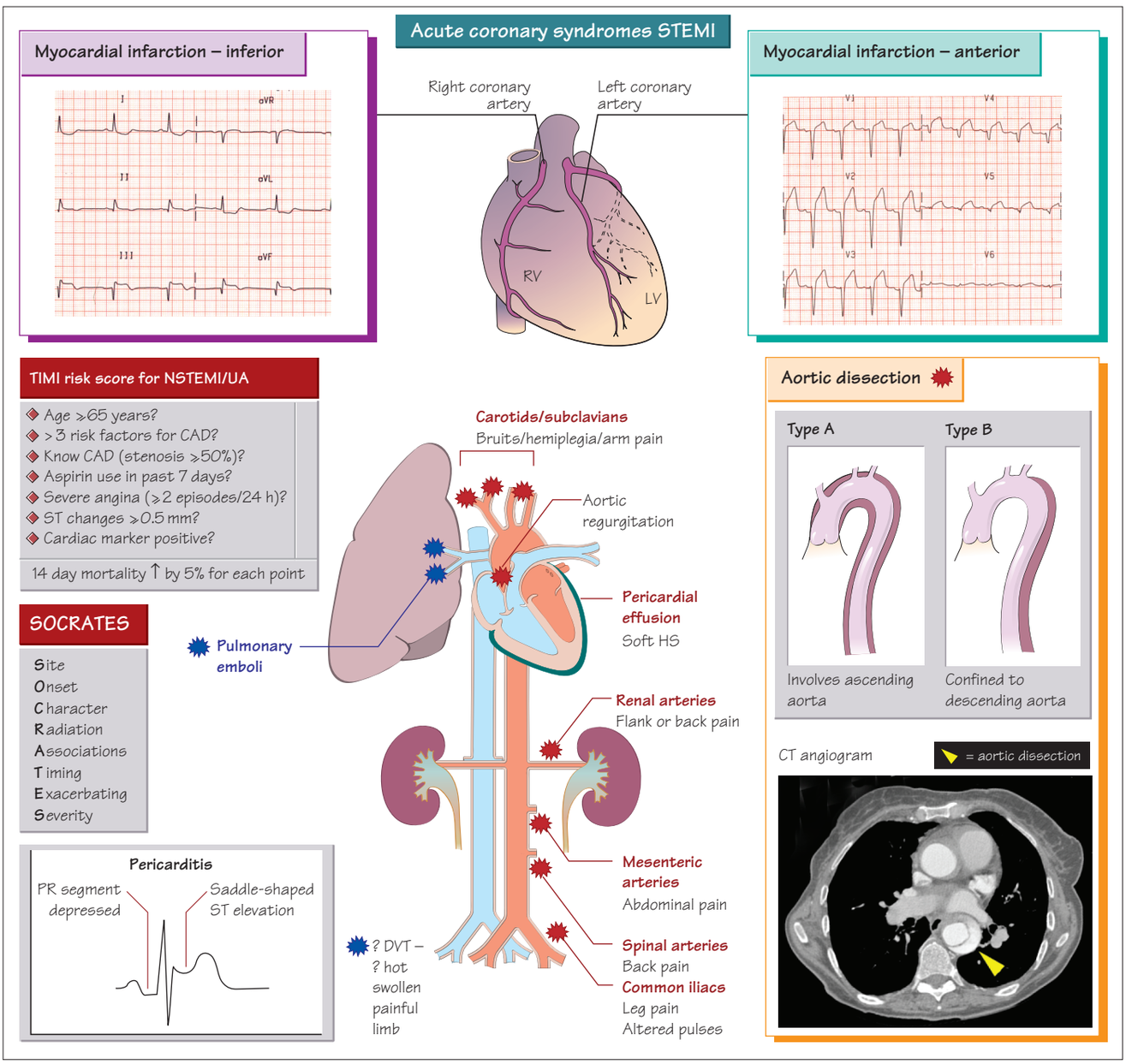
A Arterial blood gas/audit
B Blood pressure
C CXR, cool patient, consider PPCI + stent
D Draw blood for U+E, troponin
E Electrocardiogram (ECG)
F Family – speak to the family
G Gratitude – thank resucitation team
H Handover to inpatient medical team and
I Intensive care

When to stop resuscitation?

This can be a difficult decision and should be made by the most senior person present. Futility of further treatment is judged on the patient's pre-arrest functional state (particularly their exercise tolerance), length of time of resuscitation and response to treatment.

Patients who have potentially treatable causes, e.g. drug overdose, or who have been cooled prior to their arrest may make a full normal recovery after hours of CPR. However, if there is no reversible cause and no ventricular activity 30 minutes after the start of resuscitation, there is little point in continuing.

34 Chest pain: cardiovascular



Chest pain can result from many thoracic and even abdominal organs. This chapter, together with Chapter 35 concentrates on the 'big three' – acute coronary syndrome (ACS), aortic dissection (AD) and pulmonary embolus (PE) and their differential diagnoses. These diagnoses are often difficult to make, and are a common area for medico-legal action.

History

Patients presenting with a 'textbook' collection of symptoms are the exception rather than the rule, and therefore the history must carefully probe the exact natures of the pain; use the acronym SOCRATES (see above).

- *Dull pain*: retrosternal or left-sided pain that is tight, crushing or heavy in nature, and may radiate to the left arm, shoulder or jaw, suggests acute coronary syndrome. Cardiac ischaemia is exacerbated by exercise or stress, relieved by rest, nitrates or oxygen. Autonomic phenomena such as nausea, anxiety or sweating are particularly associated with myocardial ischaemia, as is radiation of pain to both arms or the use of the clenched fist on the chest wall to demonstrate the pain.

• *Sharp pain*: sharp, well-localised pain, exacerbated by breathing and coughing, suggests pleuritic irritation: consider pulmonary embolism and other causes (Chapter 35).
• *Sudden-onset tearing or ripping pain radiating to back/intrascapular region* suggests aortic dissection.
• *Breathlessness* is a common feature of pulmonary embolism and acute coronary syndrome, and may be the only symptom in elderly or diabetic patients. Breathlessness as the main complaint is covered in Chapter 36.

Previous medical history should include risk factors for atherosclerosis – family history, smoking, hypertension, diabetes and hyperlipidaemia. A history of cocaine use should be sought, particularly in young patients with chest pain.

Investigations

Bedside investigations

• ECG: reviewed by doctor within 5 minutes of arrival, interpretation written and initialled.
• Glucose: tight glucose control is important in ACS.

Laboratory investigations

• FBC, U+E.
• Troponin I: baseline and 8–12 hours from onset of pain.
• D-dimer: only if low-risk patient (Chapters 2 and 35).

Imaging

• Erect chest X-ray, preferably PA; AP magnifies the mediastinum.
• CT (300 CXR) with contrast can detect PE, AD and MI. Perfusion (Q) scan (70 CXR) can be used to detect PE in patients with normal chest X-ray.
• Ultrasound confirming DVT supports a diagnosis of PE.

Treatment

Patients should be triaged to an area with full monitoring and nursing care.

MONA is a useful acronym.

Oxygen should be given to keep the saturation above 96%, together with *Aspirin* to chew in all patients who might have ACS.

Oxygen and *Nitrates* may relieve the pain associated with ACS, but if not, intravenous *Morphine* should be given, titrated to pain.

Acute coronary syndromes

ACS is a useful umbrella term reflecting the common symptoms resulting from coronary artery occlusion due to atherosclerotic plaque.

ST elevation myocardial infarction (STEMI)

This term covers the 'traditional' MI: patients with chest pain of more than 20 minutes and *ST elevation >1 mm in two or more contiguous leads*, EXCEPT chest leads V_2 and V_3 where ST elevation must be >1.5 mm (female) or >2 mm (male). Contiguous in this sense means from the same angle e.g. II, III and aV_F or V_4, V_5, aV_L.

Aspirin gives a similar level of benefit to thrombolysis with a fraction of the risk or cost. If nothing else, give aspirin to chew. Other drugs that may be used in ACS include glycoprotein IIb/IIIa blockers, low molecular weight heparin, beta blockers, statins and ACE inhibitors.

Primary percutaneous coronary intervention (PPCI) Opening the coronary artery using a balloon, then holding it open using a stent is the optimum treatment for STEMI treatment if it can be performed within 2 hours of patient presentation.

Thrombolysis Dissolves blood clot using drugs. It is used in situations where PPCI is not immediately available. There is a 1% risk of catastrophic, e.g. cerebral bleeding.

Non-STEMI/unstable angina (UA)

Some patients have myocardial infarction without ST elevation (non-STEMI) or UA, ischaemic chest pain at rest with ST depression. These patients must be distinguished from those who have had a recent MI, as the troponin remains raised for up to 14 days. Immediate PPCI/thrombolysis do not give the same benefit in NSTEMI/UA as in STEMI, therefore treatment is to stabilise and investigate.

The TIMI score (opposite) assists with risk stratification of these patients according to local protocol, e.g.
• Patients with TIMI >2 should be admitted and have an exercise test or other investigation if the troponin 9–12 hours after pain is negative.
• Patients with TIMI ≤2 can be observed in the ED and sent home if the troponin is negative at 6–9 hours with outpatient clinic follow-up.

The GRACE score is a more complicated alternative to TIMI scoring, but does not appear to be significantly superior in the Emergency Department setting.

Pericarditis

Pericarditis is inflammation of the pericardium, usually caused by a viral infection, renal failure or post-MI/cardiac surgery. Pericarditis typically causes a sharp pain over the left chest, worse when lying flat, but relieved when sitting forward.

A high-pitched scratchy 'sandpaper' rub can often be heard on examination. The ECG may show saddle-shaped ST elevation, which must be differentiated from STEMI.

Pericarditis usually responds well to NSAIDs, but in cases where there is doubt, or evidence of complication, e.g. pericardial effusion or high jugular venous pressure (JVP), an echocardiogram should be performed.

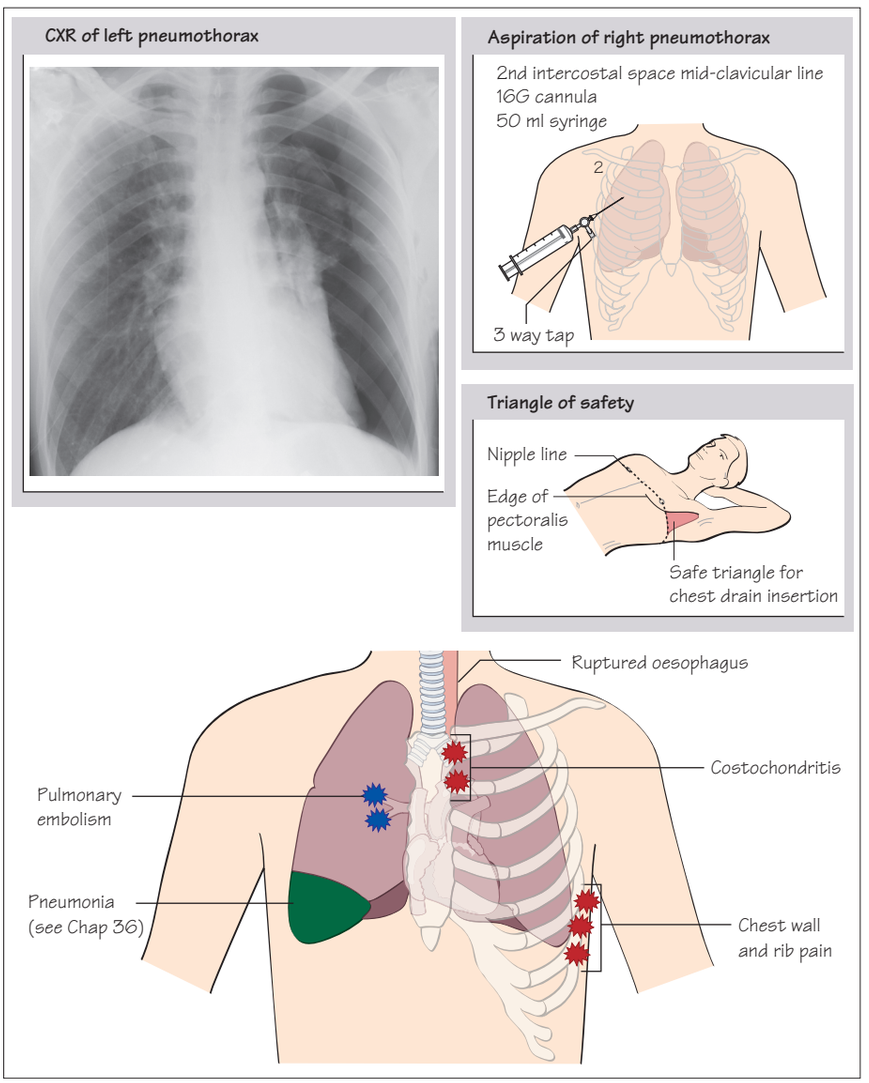
Aortic dissection

A defect in the elastic middle layer of the aortic wall allows blood to flow in, progressively stripping apart the layers. Old age, hypertension and connective tissue disorders such as Marfan's syndrome predispose to dissection. Dissection may involve branches off the aorta, presenting as stroke or MI (usually inferior) and with a diverse range symptoms (see opposite).

Most (90%) patients describe a severe 'tearing' pain radiating to the back between the scapulae. The blood pressure may differ between arms and legs. The chest X-ray may show a wide mediastinum, but a normal chest X-ray does not rule out dissection, nor can a transthoracic echocardiogram. The definitive investigation is CT with contrast.

Careful control of blood pressure prevents extension: labetalol ± vasodilators are used to achieve a systolic BP <120 mmHg. Type A dissection needs surgery or endovascular stenting; type B is usually managed with blood pressure control only.

35 Chest pain: non-cardiovascular



This chapter concentrates on non-cardiovascular causes of chest pain, but there is inevitable overlap with the previous and subsequent chapters, particularly in the history and examination.

History

Non-cardiovascular chest pain is usually caused by inflammation of the soft tissues, which give a sharp or stabbing pain, often described as 'pleuritic' pain. A SOCRATES (Chapter 34) pain history may give pointers to possible causes.

- Pulmonary embolism: shock, syncope, sudden-onset SOB, previous deep vein thrombosis/pulmonary embolism (DVT/PE), malignancy, immobility, haemoptysis.
- Pneumothorax: past history of same or asthma/ chronic obstructive pulmonary disease (COPD), breathlessness.
- Chest infections: cough, sputum, breathlessness and fever (Chapter 36).
- Pericarditis: central sharp pain, recent viral infection, pain worse supine and relieved by sitting or leaning forward (Chapter 34).
- Costochondritis: following viral upper respiratory tract infection.
- Cracked rib: if trauma.
- Vomiting: ruptured oesophagus.

Examination

Tenderness over the chest wall, and reproduction of the *same pain* on palpation, suggests costochondritis or a fractured rib, but does not rule out any of the other, more serious causes of chest pain.

The legs should be checked for calf pain, oedema, swelling and prominent veins that may indicate a deep vein thrombosis. The abdomen should be palpated to look for conditions that could cause pain in the chest, e.g. epigastric pain, liver or gall bladder disease.

Investigations

Bedside investigations

- ECG.

Laboratory investigations

- FBC, U+E.
- D-dimer only if PE suspected *and* low risk.

Imaging

- Chest X-ray (erect, PA).

Common conditions

Pulmonary embolus and deep vein thrombosis

Pulmonary embolism is a notoriously difficult diagnosis due to a wide spectrum of presentations, from the moribund patient with a large 'saddle' embolus occluding their pulmonary arteries to the COPD patient with mild pain and breathlessness. The same is true with DVT: physical signs are inconsistent and overlap with normal findings.

Investigations for PE/DVT are similarly ambiguous; there is no simple, cheap test capable of accurately ruling out PE or DVT in *any* patient. The use of a d-dimer test is discussed in Chapter 2, but *should only be used in low-probability patients.* To complicate things further, there are different d-dimer tests, ELISA (enzyme-linked immunosorbent assay) being the most sensitive.

Wells score for PE	
Clinical signs of DVT	3
PE most likely diagnosis	3
Heart rate >100 bpm	1.5
Immobilisation or surgery <4/52	1.5
Previous definite PE/DVT	1.5
Haemoptysis	1
Active cancer	1
Score <2 = 3% chance of PE (low)	
Score 2–6 = 20% chance of PE (mod)	
Score >6 = 50% chance of PE (high)	
Alternative cutoff	
Score ≤4 = PE unlikely	
Score >4 = PE likely	

Wells score for DVT	
Active cancer	1
Paralysis, paresis or plaster	1
Immobilisation or surgery <4/52	1
Tenderness over deep veins	1
Entire leg swollen	1
Calf swelling >3 cm difference	1
Pitting oedema	1
Collateral superficial veins	1
DVT not most likely diagnosis	–2
Score ≤0 = 3% chance of DVT (low)	
Score 1–2 = 25% chance of DVT (mod)	
Score >2 = 75% chance of DVT (high)	

In the case of a patient with a moderate to high pre-test probability, definitive imaging needs to be performed to prove or disprove the diagnosis. In the case of PE this is CTPA (300 CXR) or Q (perfusion) scan (70 CXR) ± ventilation scan (70 CXR). For DVT an ultrasound is the usual choice. The main value of the CXR is in excluding other causes of chest pain/SOB.

Features that are positive in PE	%
Respiratory rate >16 breaths/minute	92
↑ A-a gradient (Chapter 7)	90
Breathlessness	85
Tachycardia >100/minute	80
Sharp chest pain	75
Fever >37.8°C	45
Haemoptysis	30
Dull chest pain	15
$S_I Q_{III} T_{III}$	10

Treatment of DVT/PE is by anticoagulation; thrombolysis is sometimes used in severe cases but is controversial. If there is high probability of PE/DVT or significant delay before investigation, anticoagulation should be started before investigation. Many patients with uncomplicated DVT/PE are now investigated and treated as outpatients.

Pneumothorax (spontaneous)

Pneumothorax is air in the pleural space, between the lung and the chest wall. Primary pneumothorax occurs in individuals without other chest pathology: risk factors are smoking, being thin and male. Secondary pneumothorax may occur due to trauma (e.g. rib fractures) or lung pathology (e.g. bullae, COPD). Breathlessness and chest pain are common symptoms.

Treatment is guided by the type, size and symptoms of the pneumothorax seen on chest X-ray. Patients with uncomplicated pneumothorax <2 cm (≈50% hemithorax volume) can be discharged with outpatient review, and instructions to return if ↑SOB. Patients with a larger pneumothorax should have a maximum of two attempts at aspiration through the second intercostal space in the mid clavicular line.

In secondary pneumothorax, symptomatic patients, patients >50 years and those with a complete pneumothorax are likely to need a chest drain. Small-diameter chest drains (10–14 Fr) are used for pneumothorax, as they are less traumatic, using Seldinger technique.

Costochondritis, chest wall pain and fractured rib

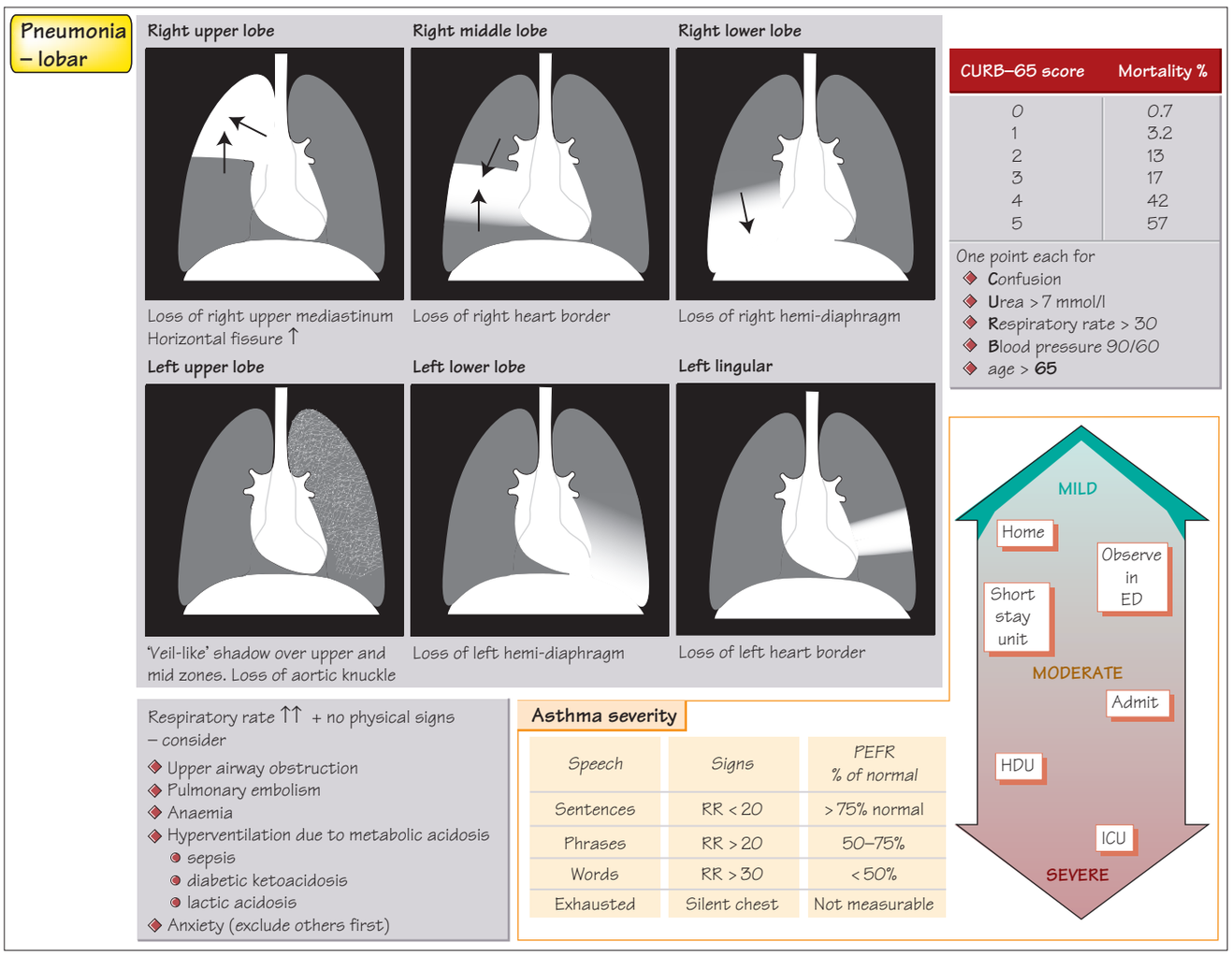
Inflammation of the costochondral cartilages at the edge of the sternum produces sharp pain when the ribs move with breathing. This inflammation often occurs after a viral upper respiratory tract infection. A similar sharp pain may be caused by a rib fracture. If there is no evidence of any more significant problem, treat with NSAIDs.

Diagnosis not to miss

Ruptured oesophagus

This is a rare consequence of severe vomiting or straining. Pain is a consistent finding and there may be dysphagia, haematemesis, subcutaneous emphysema and pneumothorax. Diagnosis is by a combination of chest X-ray, contrast swallow and CT. Management includes resuscitation, antibiotics and urgent surgical review.

36 Shortness of breath



History

Feeling 'short of breath' (SoB) generally indicates a problem with the lungs; however, it can also be a feature of cardiac disease, metabolic disturbance, anaemia and anxiety. Exacerbating or relieving factors such as exercise/position or timing (at night/early morning) are helpful.

Abrupt onset of SoB associated with sharp chest pain suggests pneumothorax, or pulmonary embolism (PE; Chapter 35). Dull chest pain and SoB implies a cardiac cause (Chapter 34).

Examination

Assess 'work of breathing', and vital signs: temperature, BP, heart rate, respiratory rate and accessory muscle use. Inability to talk, exhaustion, cyanosis and bradycardia are preterminal signs. Specific findings that should be sought include:

• *Wheeze*: a musical noise usually heard *in expiration*, due to partial obstruction of small airways. This may be caused by bronchospasm (asthma), loss of lung elasticity causing collapse (chronic obstructive pulmonary disease; COPD) or obstruction by mucus or fluid. Fluid in the terminal bronchioles (pulmonary oedema) may cause 'cardiac asthma', due to left-sided heart failure. Wheeze audible only over one part of the lung suggests bronchial obstruction, e.g. foreign body or compression – malignancy.

• *Crackles*: crackles are caused by airway opening, so are generally *inspiratory*. Coarse crackles ⇒ fluid (pulmonary oedema), or infection in small airways. Fine crackles are heard in chronic lung diseases.

• *Stridor*: a whistling noise worse *on inspiration*, due to partial upper airway obstruction.

• *Sputum*: increased production of sputum and green colour suggest infectious illness. Pink, frothy sputum occurs in pulmo-

nary oedema. Blood-stained sputum (haemoptysis) is usually due to infections (80%), but may also occur in lung cancer or PE.
- *Reduced air entry* and chest wall movement with:
  - percussion note dull, ↑ breath sounds ⇒ consolidation;
  - percussion note dull, no breath sounds ⇒ pleural effusion;
  - percussion note hyperresonant, ↓ breath sounds ⇒ pneumothorax.

Investigations

Investigations are determined by symptoms – mild asthma exacerbations do not require investigations other than peak expiratory flow (PEF). Arterial blood gases (ABGs) are painful and should be limited to patients in whom they are likely to influence management, e.g. O_2 saturation <92%.

Bedside investigations

- Peak expiratory flow meter for all patients with asthma/COPD (pre- and post-bronchodilator).
- Urine for *Legionella* or streptococcal urinary antigen.
- Blood glucose, ECG.

Laboratory investigations

- FBC, U+E.
- ABGs (Chapter 7).
- Blood cultures and atypical pneumonia serology indicated only in patients with severe pneumonia.

Imaging

- Chest X-ray – rarely needed in straightforward asthma.
- CT is very good for imaging lungs and CT pulmonary angiography (CTPA) (300 CXR) is the imaging of choice for possible PE, although a Q (perfusion) scan (70 CXR) can be used in patients without lung pathology who have a normal chest X-ray.

Management

Check airway, give oxygen. If the patient has type II respiratory failure (e.g. from COPD) and is on long-term oxygen, give oxygen to achieve a saturation of approximately 91%.

Pneumonia

A patient with fever, cough and pleuritic chest pain has pneumonia until proven otherwise. In community-acquired pneumonia (CAP), the clinical or X-ray features do not accurately predict the organism responsible.

The CURB-65 scoring tool is helpful to predict the need for admission/intensive treatment, but must be moderated by the patient's social circumstances and general condition. For a stable patient with CAP and a CURB-65 score of 0 who does not need to be admitted, amoxicillin is usually the first-choice antibiotic. For more serious or complex infections such hospital-acquired pneumonia, immunosuppression, cancer, HIV, and high-risk groups (travellers, intravenous drug users), consult local antibiotic guidelines that reflect local patterns of disease and organism sensitivity.

Asthma and chronic obstructive pulmonary disease

Asthma can be unpredictable: ensure early senior assessment in ill patients. The main danger for both asthma and COPD patients is *tiredness*, which results in a vicious cycle of raised PCO_2, exhaustion and hypoventilation. In addition a few asthmatics may have extreme *tightness*, where tight bronchoconstriction makes it very difficult to move any air in and out of the alveoli – the 'silent chest'.

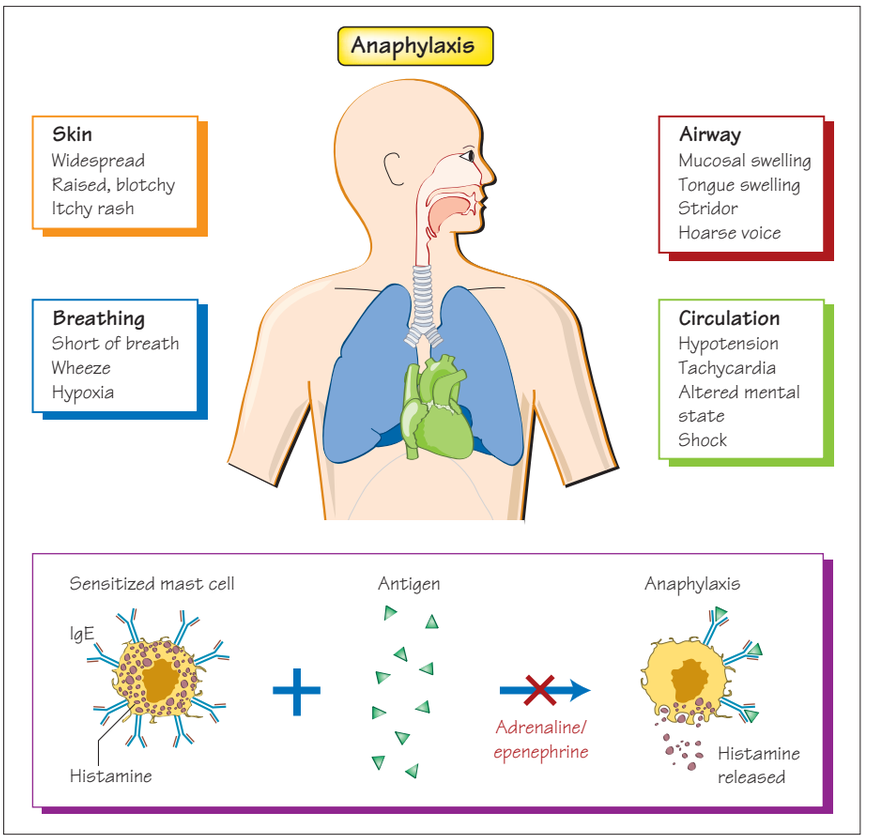
Treatment is guided by regular PEF measurements: the mainstay is *oxygen* and *β_2 agonist bronchodilators* (e.g. salbutamol, 5 mg) delivered by oxygen-driven nebuliser or metered dose inhaler (MDI) and spacer. In severe cases, the nebulisers are given continuously. Intravenous β_2 agonists are rarely necessary as adequate drug delivery is usually possible with nebulisers.
- *Steroids* are given to reduce inflammation in the small airways. Intravenous hydrocortisone 100 mg should be used in unwell patients, but milder cases may have oral prednisolone 50 mg. Antibiotics are not indicated unless there is evidence of infection.
- *Anticholinergics* (e.g. nebulised ipratropium bromide) are used in more severe cases of asthma and all cases of COPD, but only need to be given once every 4 hours.
- *Magnesium sulphate* and *aminophylline* are second-line drugs used for severe asthma, but use is controversial.
- *Non-invasive ventilation* (NIV) is used in both COPD and asthma to reduce the work of breathing, and to try to avoid intubation. *Intubation and ventilation* is sometimes necessary if the patient is tired and their PCO_2 is rising.

Most COPD patients who arrive at the Emergency Department will require admission to hospital. Asthmatics who revert to a PEF >75% normal after having three nebulisers over an hour and a dose of steroids over 1 hour, can usually be discharged. Patients being discharged from the Emergency Department should have:
- A written asthma plan.
- 3–5 days of oral prednisolone 30–50 mg.
- An agreed review, e.g. GP or outpatient clinic.
- Inhaler technique check.

Acute pulmonary oedema

These patients often present in the early morning with extreme SoB, crackles and sometimes wheeze – 'cardiac asthma'. The underlying problem is acute left ventricular failure, usually due to myocardial ischaemia/infarction which pushes the patient to the 'wrong' side of the Frank-Starling curve. Treatment is oxygen, aspirin and reducing cardiac preload using nitrates, continuous positive airway pressure (CPAP) and angiotensin-converting enzyme (ACE) inhibitors in the short term and loop diuretics, e.g. furosemide, in the medium term.

37 Anaphylaxis



Anaphylaxis is a severe, life-threatening systemic IgE-mediated hypersensitivity reaction. Anaphylaxis causes compromise of airway, breathing and circulation, together with a characteristic itchy skin rash. Deaths occur because of late treatment or under-treatment. Adrenaline as an intramuscular injection is very safe as the localised vasoconstriction it provokes effectively makes it slow release.

The short time course of anaphylaxis means the stimulus is usually known. Ask about a history of atopy, and specific triggers (e.g. stings, nuts, food) and medications (e.g. antibiotics, anaesthetic agents, NSAIDs and angiotensin-converting enzyme (ACE) inhibitors) and contrast media.

WARNING

Don't be afraid of adrenaline/epinephrine

Airway

Visible swelling of upper airway (tongue and throat), stridor and hoarseness are very worrying signs.

In upper airway obstruction, oxygen saturations are meaningless – do not be reassured by a normal saturation.

Give adrenaline 500 micrograms i.m. immediately and get senior help urgently.

Give high-flow oxygen via a reservoir mask.

Breathing

Shortness of breath, wheeze, respiratory rate, use of accessory muscles of breathing may progresses to fatigue, hypoxia/cyanosis and respiratory arrest.

Give adrenaline 500 micrograms i.m. immediately (if not already done) and get senior help.

Circulation

Massive vasodilatation leads to anaphylactic shock and circulatory collapse.

Give adrenaline 500 micrograms i.m. immediately (if not already done) and get senior help.

Disability
Shock may result in altered conscious state.

Exposure
Remove the antigen if possible, e.g. wasp sting.

There is usually a widespread red rash, which is itchy (urticaria), and there may be angioedema (deep tissue swelling).

Why give adrenaline?
Anaphylaxis is caused by mast cells releasing histamine in response to IgE-mediated stimulus. Adrenaline stabilises the mast cells, preventing further histamine release. It also has other beneficial effects:
- Beta-2 agonist in lungs, counteracting bronchospasm.
- Counteracts vasodilation.

By giving the injection intramuscularly, the local vasoconstriction it provokes means the adrenaline is released slowly, avoiding tachycardia and hypertension. If ongoing symptoms persist after 5 minutes, repeat the dose of intramuscular adrenaline.

If symptoms persist despite the repeat dose of adrenaline, or the patient is very unwell with incipient airway obstruction, a senior doctor may consider using bolus doses of 50–100 µg of adrenaline (= 0.5–1 mL of the 1 : 10 000 premixed resuscitation syringes) with a flush. This may make the patient feel unwell and have a tachycardia, but usually rapidly resolves the symptoms.

Secondary drugs for anaphylaxis
Corticosteroids
Systemic corticosteroids take at least 30 minutes to have an effect, even when given intravenously, but they may be helpful in preventing recurrence of reaction – a biphasic reaction.
- Hydrocortisone, 100 mg i.v.
- Prednisolone, 50 mg oral.

Antihistamines (H_1 antagonists)
These are second-line agents and *should not be used before adrenaline* to treat anaphylaxis, but may be suitable for treating mild allergy, particularly the itchy urticarial rash. The sedating antihistamines seem more effective in the acute phase:
- Diphenhydramine, 25–50 mg i.v.
- Chlorphenamine, 10–20 mg i.v.

The role of H_2 antagonists, such as ranitidine, is controversial; they may have a role as an additional treatment to H_1 antagonists in severe anaphylaxis.

Anaphylaxis mimics
ACE inhibitors are responsible for anaphylactoid facial swelling, which occurs without any obvious precipitant, but is differentiated from anaphylaxis by absence of rash. It is probably caused by bradykinin build-up as swapping to an angiotensin II blocker (-sartan) prevents further episodes.

Scrombotoxin poisoning is seen when people eat fish, particularly tuna, that is past its prime. The scrombotoxin has a histamine-like effect, giving a red itchy rash, but no systemic upset. Notify public health officials. Treatment is supportive.

Morphine causes direct histamine release but without systemic upset, and this does not represent allergy.

Other diagnoses that can be confused with anaphylaxis include vasovagal syncope, syndromes associated with flushing (e.g. carcinoid), panic attacks, angioedema and other causes of shock.

Investigations
Investigations should not delay the resuscitation.

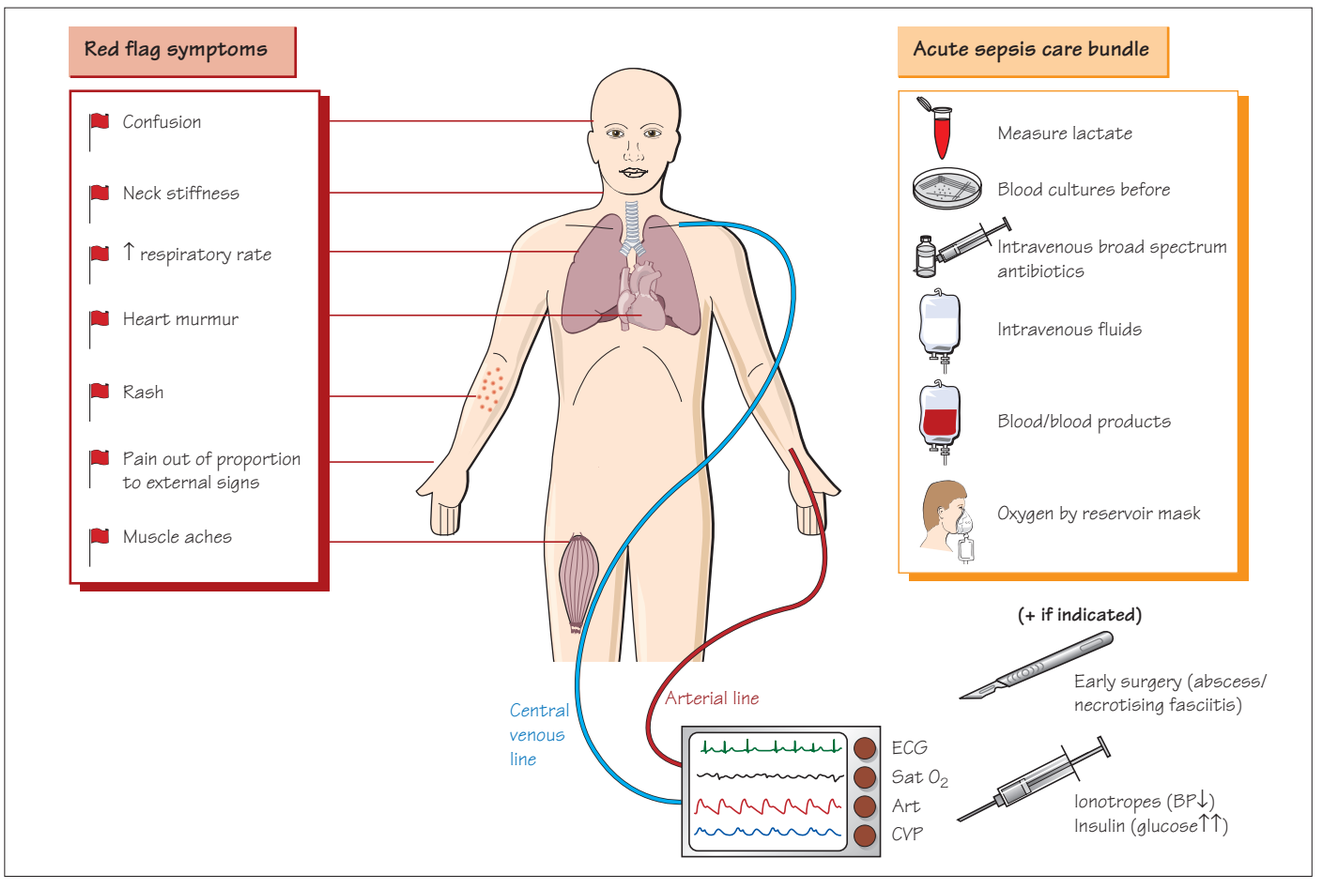
Bedside investigations
- Blood glucose, ECG

Laboratory invstigations
- FBC, U+E, ABGs if indicated
- Mast cell tryptase levels
  - useful if the diagnosis is in doubt;
  - one initial sample (minimum) with optional samples at 2 and 24 hours.

Management
Patients with an anaphylactic reaction should be observed for 4–6 hours as there is a risk of recurrence of symptoms – 'rebound' phenomenon. Review by a senior clinician determines need for further treatment or a longer period of observation, e.g.
- Patients with a previous history of biphasic reactions or reactions with the possibility of continuing absorption of allergen;
- Severe reactions with no apparent cause;
- Reactions in patients with severe asthma;
- Patients presenting in the evening or at night, or those who may not be able to respond to any deterioration.

Disposal: who can go home?
Discharge a patient who has anaphylactic reaction only once the patient is stable and:
- has been reviewed by a senior clinician;
- has been given education and clear written instructions to avoid allergen, and return to hospital if symptoms return;
- has been given 3 days of antihistamines and oral steroids
- has been considered for an adrenaline auto-injector (Epi-pen®);
- has a plan for follow-up, including referral to allergy specialist, and contact with the patient's GP;
- alert has been noted in patient's medical record (written/electronic).

38 Sepsis



Patients with fever and infections are a very common problem in the Emergency Department. Amongst the many straightforward cases of viral or bacterial infection are small numbers of patients with life-threatening sepsis: this chapter concentrates on the more severe end of the spectrum.

There is very good evidence of the importance of early diagnosis and aggressive resuscitation of patients with severe sepsis. Intensive resuscitation with invasive monitoring and vasoactive drug support must start in the Emergency Department if the patient is to have the best chance of survival.

Uncomplicated sepsis
Infection with no evidence of systemic effects

Severe sepsis (mortality 30%)
Infection + sepsis-induced hypoperfusion/organ failure

Septic shock (mortality 50%)
Infection + systolic blood pressure <90 mmHg despite adequate fluid resuscitation

History

There are many non-specific features such as fever, vomiting, muscle aches and pains, headache and malaise that are present in both viral infections and systemic sepsis.

Specific features are localising symptoms that suggest a focus for the infection, e.g. dysuria, cough and production of green sputum. Rigors (like shivering) and high fever >38°C make bacteraemia more likely.

Ask about immunisation, comorbidities, e.g. diabetes, heart/liver/renal failure, medications esp. immunosuppressants, drug use and contact with sick people or travel in the last 6 months.

Examination

From the end of the bed:

- Is the patient well/ill/critically ill?
- Is the patient in an appropriately monitored area?
- Does the patient have oxygen?

Localising signs

- Upper respiratory tract infection (URTI): pharyngitis, tonsillitis, otitis media, sinusitis (Chapter 21).

• Lower respiratory tract infection (LRTI/pneumonia): the commonest cause of septic shock (Chapter 36).
• UTI/pyelonephritis: Gram-negative septicaemia a common cause of septic shock (Chapter 20).
• Gastroenteritis (Chapter 40).
• Meningitis: the classic signs are reduced state of consciousness, neck stiffness, headache, vomiting, photophobia together with a high fever. The non-blanching, re/dark purple vasculitic rash is only caused by meningococcal sepsis/meningitis. Neck stiffness may be absent in 30% of cases.

Investigations

Bedside investigations

• Blood glucose, urine test, βhCG.

Laboratory investigations

• FBC, U+E, LFTs, arterial blood gas (ABG).
• Lactate level: lactate results from anaerobic metabolism, and is a useful marker of the severity of tissue hypoperfusion.
• C-reactive protein (CRP), according to local protocol.
• Clotting screen/Group and Save.
• Blood cultures if severe sepsis/septic shock/neutropenic/immunosuppressed or suspected endocarditis.
• Urine culture, pneumococcal and *Legionella* antigen.
• Lumbar puncture/meningococcus PCR if indicated.

Imaging

• Chest X-ray.
• CT head: if meningitis suspected, treat first.

Management

Oxygen, broad-spectrum antibiotics and intravenous fluids are the mainstays of treatment. Time to antibiotics is crucial – they should never be delayed, e.g. waiting for CT or LP. The notion of a 'bundle' of care is often used, which is a set of goals to achieve within that point of the patient pathway (opposite). For severe or transmissible infectious diseases such as meningitis, the diagnosing doctor has a duty to notify the health authorities to trace contacts and ensure prevention of further cases.

Disposal: who can go home?

Patients who appear well, in whom serious infection has been excluded, and who can tolerate oral fluids and medication are usually safe to discharge. Patients need a letter to their GP, with specific mention of tests that need follow-up, e.g. cultures.

Common diagnoses

Cellulitis

Cellulitis is a bacterial infection (usually *Staphylococcus* and/or *Streptococcus*) of the skin and subcutaneous tissues resulting in pain, erythema, swelling and warmth. Patients with underlying diseases such as venous stasis, peripheral vascular disease or diabetes are particularly vulnerable. Minor skin wounds or intravenous cannula sites allow organisms to enter the skin.

Mild cellulitis is treated with oral flucloxacillin or a first-generation cephalosporin. Monitoring is helped by drawing a line around the red area, so it is obvious if the cellulitis is increasing. Admission and intravenous treatment is necessary for more severe cases and those with underlying diseases.

Diagnoses not to miss

Severe sepsis and meningitis

In uncomplicated sepsis and meningitis, there is narrow window of opportunity to catch the disease *before* the patient becomes critically unwell with severe sepsis. Rapid disease progression means that antibiotics (e.g. ceftriaxone 2 g i.v.) must be given as soon as the disease is suspected, together with the other parts of the 'care bundle'.

> rash + unwell = meningococcal septicaemia
> meningococcal septicaemia + meningitis = meningococcal meningitis

If there is meningitis without rash, give dexamethasone with the first dose of antibiotics and also consider encephalitis.

Public health officials should be notified.

Immunocompromised patients

Sepsis occurring in an immunocompromised patient is life-threatening. Neutropenia (<1/mm^3) usually occurs approximately 10 days after chemotherapy. Other immunocompromised patients include: haematological malignancy, HIV/AIDS, and those on immunosuppressive medication, e.g. tacrolimus, methotrexate, ciclosporin, prednisolone.

These patients need urgent high-dose broad-spectrum antibiotics and admission. Patients on steroids cannot produce extra endogenous steroids as part of the stress response; therefore extra exogenous steroids must be given.

Travellers

Consider malaria, typhoid fever, dengue fever, hepatitis and sexually transmitted diseases such as HIV or gonorrhoea. Patterns of disease change faster than books are written, so consult an infectious disease physician.

Toxic shock syndrome

A widespread red rash together with features of septic shock, caused by an exotoxin from staphylococcal/streptococcal infection in a (forgotten) vaginal tampon.

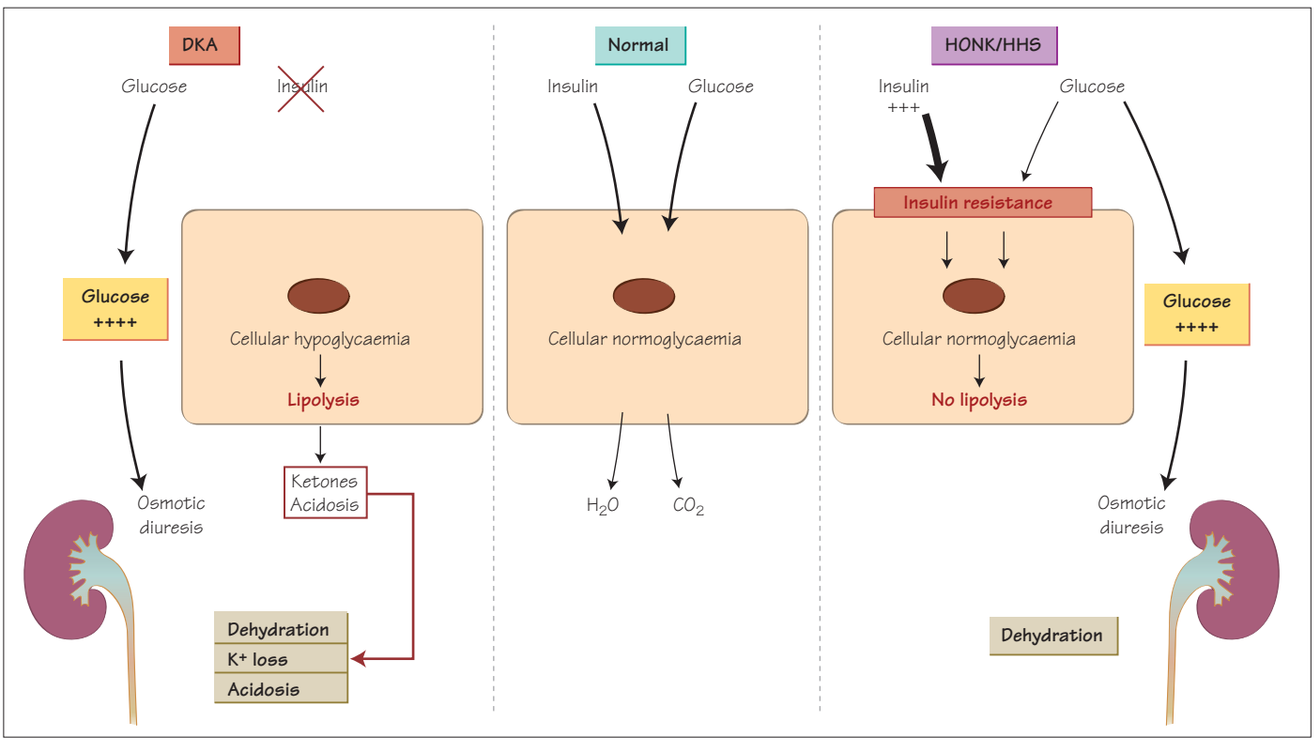
Infective endocarditis

Presenting with an insidious combination of fever, malaise, night sweats, heart murmur and embolic phenomena, there is usually a history of valvular heart disease or intravenous drug use.

Necrotising fasciitis and gas gangrene

These severe soft tissue infections should be considered if there is fever together with pain ± gas in soft tissues that is out of all proportion to the clinical appearance. Early antibiotics, together with rapid aggressive surgical debridement, are necessary.

39 Endocrine emergencies



Diabetes

Diabetic emergencies result from either lack of (diabetes mellitus (DM) type 1) or resistance to (DM type 2) insulin and the treatments for these conditions. Young adults with diabetes often go through a period of suboptimal control: treatment of acute problems of this difficult chronic disease should be sympathetic and collaborative.

What sort of diabetes/control does this patient have?

As patients with DM(2) are sometimes treated with insulin, confusion can occur; these rules of thumb may help.

- If the patient has a high body mass index, they probably have DM(2).
- A patient with DM(1) and good diabetic control will have four or more injections of insulin per day, will know their HbA_{1c} (which will be low), and will be prone to hypoglycaemia.
- A patient with DM(1) and poor diabetic control will have one or two injections of insulin per day, may not know their HbA_{1c} and will be prone to diabetic ketoacidosis (DKA).

History

The history should cover the type of diabetes, its history and complications, i.e. atherosclerotic (MI/CVA), neurological (peripheral neuropathy/retinopathy), renal.

Why has the patient attended now? What are the stressors/factors that have caused the disease to decompensate? For example, infections/illness/psychological.

Examination

Look for diseases that may have triggered a loss of diabetic control, e.g. UTI, pneumonia.

In people with long-standing diabetes, absence of pain does not mean absence of disease: neuropathy can mask significant disease. Myocardial infarction or abdominal conditions such as infection or pancreatitis may be painless. The feet are particularly vulnerable – look at the soles for evidence of neuropathy, ulceration or infection, and consider osteomyelitis.

Investigations

Bedside investigations

- Blood glucose: treat hypoglycaemia as below. Hyperglycaemia readings above 20 mmol/L are inaccurate, and not useful in guiding treatment.
- ECG: myocardial infarction (MI) may occur in patients less than 30 years old with poorly controlled diabetes.
- Urine or blood should be checked for ketones.
- pH measurement: a venous pH is adequate in all but the most sick patients and avoids painful arterial puncture.

Laboratory investigations

- FBC, U+E, BG/VBG, clotting.
- Pseudo-hyponatraemia may occur in patients with very high levels of glucose, due to the method of measurement.
- Serum osmolality if hyperosmolar non-ketotic acidosis/ hyperosmolar hyperglycaemic state (HONK/HHS) suspected.

Imaging

- Chest X-ray in all HONK/HHS or severe DKA.

Common diagnoses

Hypoglycaemia (glucose < 3 mmol/L)

Hypoglycaemia occurs as a result of too much insulin, too much exercise, too little carbohydrate or a combination of these. Most patients recognise the symptoms of hypoglycaemia, and self-treat with oral glucose, followed by complex carbohydrates. Untreated, they become confused and eventually comatose. A bedside blood glucose check is essential for all such patients, whether they are known to have diabetes or not.

Treatment

Treatment of hypoglycaemia in the Emergency Department is 50 mL of glucose 50% if i.v. access is available, 1 mg glucagon i.m. if not. If there is recurrent hypoglycaemia, an infusion of glucose 10% is necessary. Patients with hypoglycaemia due to long-acting preparations of insulin or oral hypoglycaemics should be admitted.

Diabetic ketoacidosis

The majority of DKA episodes occurs in patients with known diabetes, but new diagnoses are made on the history of polyuria, polydipsia, weight loss and dehydration. The rapid sighing deep 'Kussmaul' breathing serves to remove CO_2 – respiratory compensation for the metabolic acidosis.

To diagnose DKA requires diabetes, ketones and acidosis.

- Blood glucose >11 mmol/L
- Ketones in urine or blood
- pH <7.30 (venous blood)

The pH correlates the severity of DKA – the most severe being pH <7.10. In addition to diagnosing DKA, it is important to search for a cause, which is often omission of insulin or illness.

Treatment principles

DKA occurs because glucose cannot enter cells without insulin. Without glucose, cells switch to metabolising fat, producing organic acids. High blood glucose forces osmotic diuresis, resulting in electrolyte loss and dehydration. Treatment aims to replace the massive fluid and electrolyte loss, and allow the body to normalise the pH. Treatment protocols vary but common features are:

- *Resuscitation phase*: 30 mL/kg, i.e. about 2 litres of i.v. normal saline, given over 1 hour. Soluble insulin is started at 6 units/hour using an infusion pump.
- *Rehydration phase*: gradual rehydration avoids rapid intracellular osmotic/sodium shifts that may cause (fatal) CNS oedema or demyelination. As the insulin takes effect, potassium enters the cells with insulin and glucose, and serum potassium drops sharply. Normal saline is continued at a reduced rate, e.g. 250 mL/hour, but with potassium, e.g. 40 mmol/L initially. Regular U+E measurement should take place to monitor the potassium.

Insulin infusion (0.1 units/kg/hour) is continued at a fixed rate until blood ketones are cleared, using an infusion of 10% glucose to prevent hypoglycaemia. If blood ketone measurement is not available, insulin may be given on a sliding scale according to the glucose level. Education about the importance of regular insulin may help prevent further episodes.

Rare diagnoses

Hyperosmolar non-ketotic or hyperosmolar hyperglycaemic state

HONK/HHS occurs in DM(2) patients: insulin resistance allows small amounts of glucose to enter cells. This is enough to prevent lipolysis, so hyperglycaemia occurs, but acidosis does not. Hyperglycaemia results in osmotic diuresis and a hyperosmolar state that develops more slowly than DKA – over days rather than hours, which eventually results in coma.

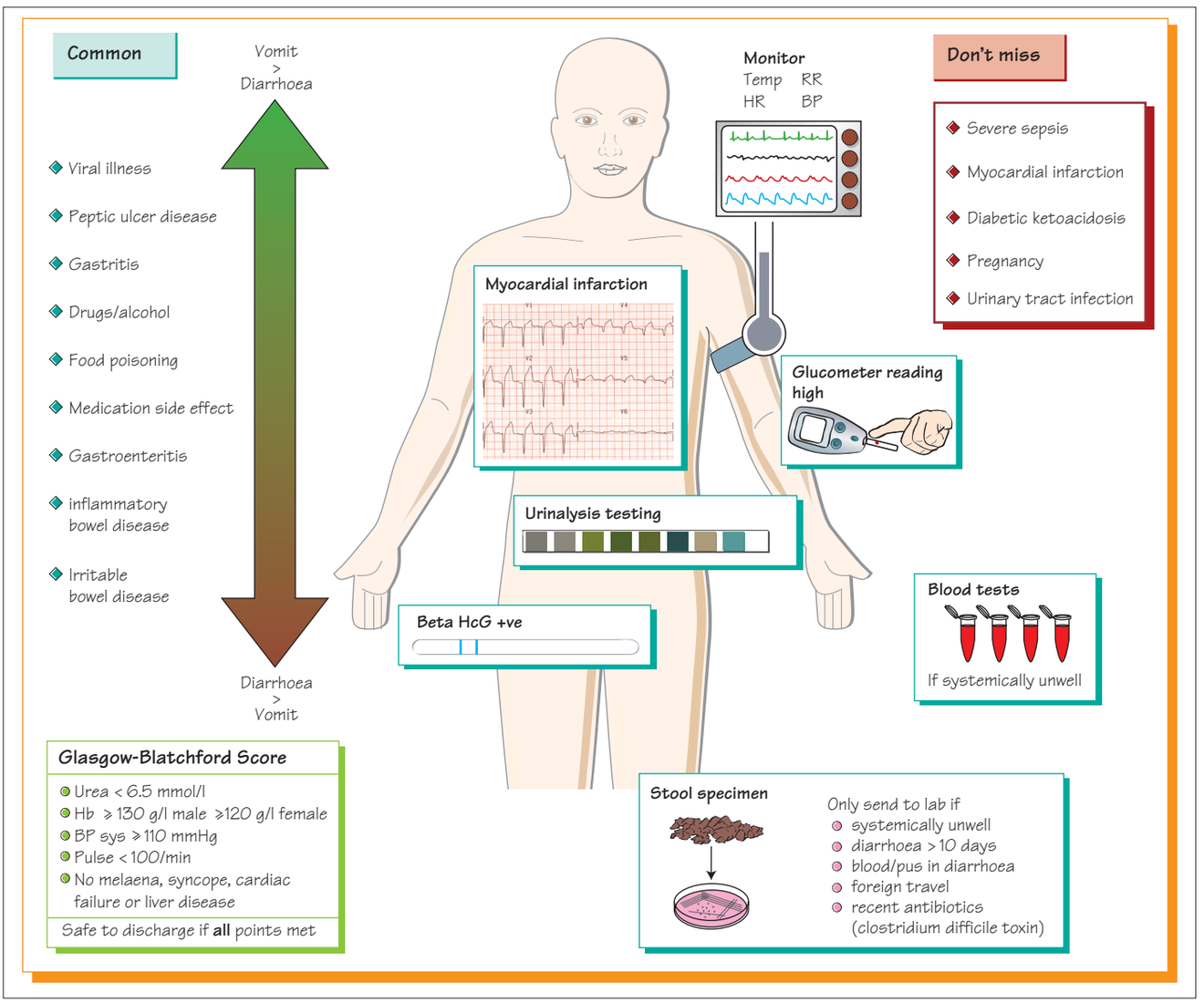
A patient with HONK/HHS is therefore markedly hyperosmolar (>320 mOsm/kg) and hyperglycaemic (>40 mmol/L), but not acidotic. Rehydration to correct these deficits should be slow, spread over several days, to avoid (fatal) CNS oedema or demyelination. Anticoagulation is necessary to prevent the hyperviscous blood forming clots, and underlying causes (e.g. UTI, pneumonia) should be sought. ICU/HDU care should be considered as mortality is high.

Hypoadrenalism

The most common cause of adrenal suppression is exogenous – pharmacological steroids given for more than 5 days suppress endogenous steroid production. Extra stress (e.g. infection, trauma) will require extra steroid (e.g. hydrocortisone) for the body to respond effectively.

Endogenous hypoadrenalism – Addison's disease – is fortunately very rare, as the features of the classic presentation of slim, hyperpigmented female with hypotension, hyponatraemia, hyperkalaemia and abdominal pain are very inconsistent and easy to overlook in the Emergency Department. Diagnosis is confirmed by the lack of endogenous steroid production when synthetic adrenocorticotrophic hormone (ACTH) is given (short synacthen test).

40 Gastroenterology



History

Vomiting

The amount, frequency and nature of the vomit should be recorded. Heavily bile-stained or faecal vomit implies obstruction. A few streaks of blood in the vomit after repeated vomiting suggests a small ('Mallory-Weiss') tear at the gastro-oesophageal junction, but large amounts of fresh blood in the vomit may indicate bleeding from the oesophagus or stomach. Dark-brown changed blood ('coffee grounds') vomit occurs with stomach or duodenal pathology, e.g. ulcer or gastritis.

Diarrhoea

The amount, frequency and nature of diarrhoea are important. Bleeding from the distal large bowel and anorectal area will be bright red. More proximal bleeding makes the stools black and shiny with a distinctive smell – melaena, but iron therapy also causes black stools.

Lower gastrointestinal tract bleeding can be caused by some infections, but should always raise suspicion of inflammatory bowel disease or malignancy. Upper gastrointestinal bleeding is a common cause of lower gastrointestinal bleeding.

Other history

Food, travel and medication (including recent antibiotic use) history should be taken. A history of rigors, muscle aches and headache suggests sepsis (Chapter 38). A history of contact with viral gastroenteritis is helpful but does not rule out other causes. Alcohol history is important, particularly when liver disease is

suspected – use the CAGE score (Chapter 29). Abdominal pain may be a feature of infectious diarrhoea and inflammatory bowel disease, but if pain is the predominant symptom see Chapter 19.

Examination

Inspection

Look for shock: tachycardia, hypotension, cool peripheries – assess level of dehydration. Document presence of jaundice, anaemia (pale conjunctivae) or oedema (low protein state or heart failure)

Palpation and percussion

Look for surgical causes such as peritonitis, appendicitis (Chapter 19). A rectal examination is an essential part of the assessment – stools should be checked for blood using faecal occult blood testing kit. Constipation ± overflow diarrhoea is a common problem in the elderly.

Investigations

Bedside investigations

- Digital rectal examination with testing for faecal blood.
- Blood glucose, urinalysis and ECG.

Laboratory investigations

- FBC, U+E, Group and Save.
- LFTs, amylase.

Imaging

- Abdominal X-ray (60 CXR) is rarely helpful unless bowel obstruction is suspected.
- Chest X-ray (erect) will demonstrate gas under the diaphragm if there is a perforation of the bowel.

Management

Resuscitation/oxygen if unwell, observation and monitoring. If bleeding and shock, obtain large-bore intravenous access, notify blood bank and the gastroenterology/surgical team.

Vomiting is treated by treating underlying causes. Metoclopramide or prochlorperazine are first-line treatments. Persistent vomiting may require $5HT_3$ antagonist such as ondansetron.

Treat dehydration with intravenous crystalloid solutions. Potassium replacement is necessary in persistent vomiting, as the kidneys lose K^+ to retain H^+ ions, causing hypokalaemia.

Common diagnoses

Gastritis/oesophagitis/peptic ulcers

Symptoms are 'heartburn', or burning epigastric pain. Risk factors include smoking, alcohol, aspirin and NSAIDs. Treated by antacids/H_2 blockers/proton pump inhibitors (PPI). Follow-up should be arranged to detect *H. pylori*, which can be treated with antibiotics. For ruptured peptic ulcer see Chapter 19.

Mallory-Weiss tear

A small tear at the gastro-oesophageal junction resulting from vomiting, producing blood streaks in the vomit. If the Glasgow-Blatchford Score is 0, the patient may be discharged with no follow-up, otherwise admit and observe.

Gastroenteritis

The majority of gastroenteritis is viral in origin, and treatment is supportive only. If the patient is unwell, consider toxin-mediated (e.g. staphylococcal) or invasive disease due to food poisoning (e.g. *E. coli*, *Campylobacter*), travel-related illness (e.g. *Salmonella*, *Shigella*) or inflammatory bowel disease. *Clostridium difficile* infection occurs after broad-spectrum antibiotic use, and produces a toxin detectable in stools.

Patients who are not dehydrated, in whom serious pathology has been excluded, are usually safe to discharge. Educate patients about oral rehydration using small sips of diluted fruit juice or sugar-containing fruit drinks diluted 1:5 with water to make oral rehydration fluid. The elderly and those with heart failure may need admission, but most patients can be safely discharged.

Rectal bleeding (haematochezia)

Localised bleeding in or near the anal canal is commonly caused by anal fissures or haemorrhoids. If the patient is well, they can be reassured in the ED, but patients should have follow-up to exclude more serious causes. Inflammatory bowel disease, diverticulitis and lower GI carcinoma should be considered in patients who are unwell.

Anorectal abscess

More common in patients with inflammatory bowel disease and diabetes, these large abscesses cause poorly localised pain. Keep nil by mouth and refer for surgical drainage.

Constipation, irritable bowel syndrome

Patients who are well but have intermittent abdominal pain, bloating and diarrhoea, often have a diagnosis of irritable bowel syndrome (IBS) or constipation with overflow, but these can only be safely diagnosed once serious pathology has been excluded.

Diagnoses not to miss

Inflammatory bowel disease

Exacerbation of inflammatory bowel disease (ulcerative colitis, Crohn's disease) commonly presents with intermittent bloody diarrhoea and abdominal pain. Weight loss and systemic manifestations such as mouth ulcers, rashes, joint and eye problems may be present. The gastroenterology team must be informed about any patient with inflammatory bowel disease presenting with gastrointestinal symptoms, even if the patient needs surgical care.

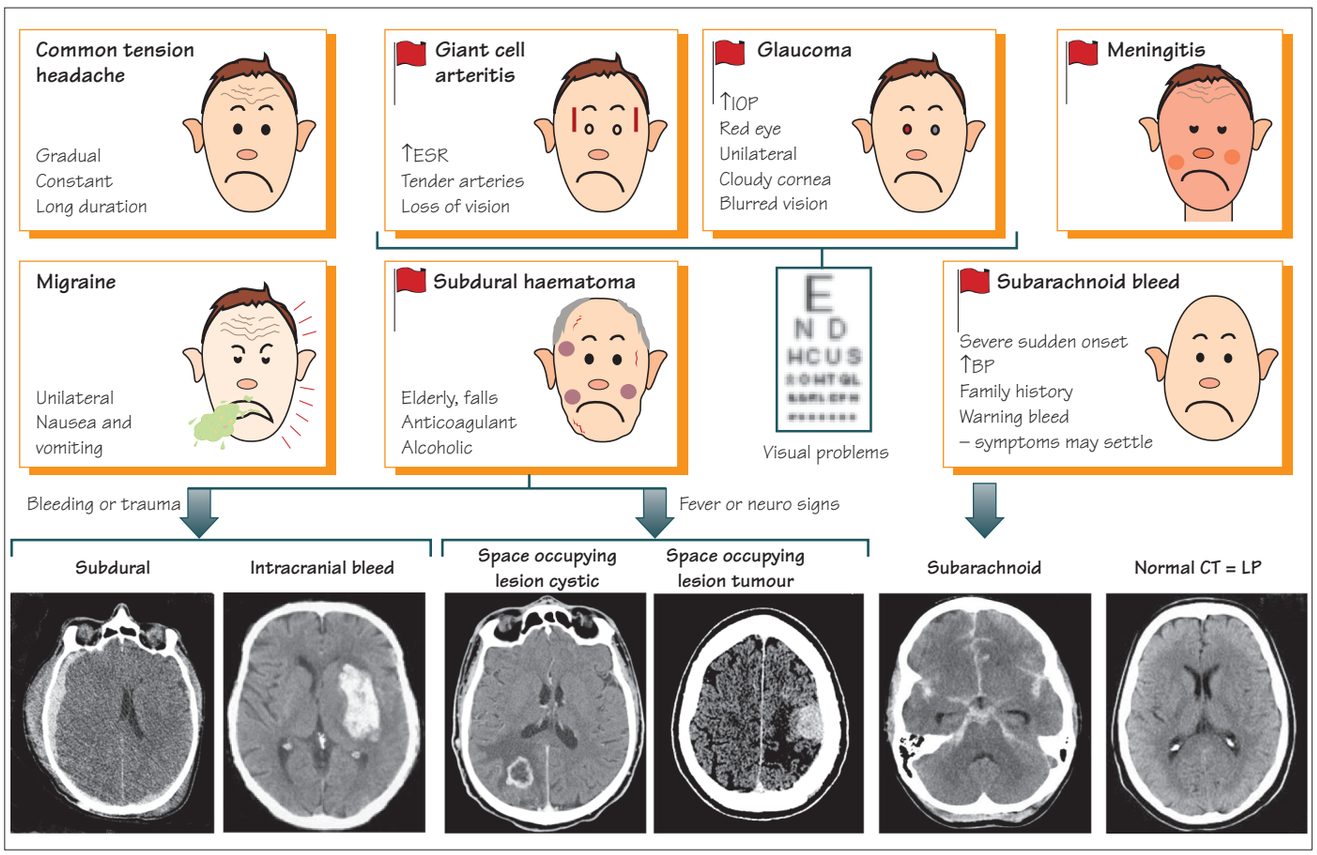
Oesophageal varices

Oesophageal varices are caused by portal hypertension, usually secondary to cirrhotic liver disease. Catastrophic bleeding can result, exacerbated by coagulopathy due to lack of (hepatically manufactured) clotting factors and platelet dysfunction. Treatment is by resuscitation and urgent endoscopy.

Lower gastrointestinal tract cancer

Cancer should always be considered in lower gastrointestinal bleeding. While the patient may not need admission, it is important to make sure that these symptoms are followed up.

41 Headache



History

Many conditions cause headache – local, systemic and non-organic. As there are usually no clinical findings, the history is everything.

- Is this the first episode, or a recurrent headache? Longstanding headaches are unlikely to be life-threatening.

A unilateral headache is typical of migraine, cluster headaches, or giant cell arteritis. Descriptions such as pressure, tightness, throbbing, are not specific for particular diagnoses. Other associated symptoms that should be sought:

- Focal neurological symptom (migraines, space-occupying lesion).
- Nausea and vomiting (migraine, infections, ↑intracranial pressure; ICP).
- Alcoholism, anticoagulant medication (subdural haematoma).
- History of recent trauma (concussion, subdural haematoma).
- Worse on waking, or on straining/bending over/coughing (↑ICP).
- Fever, photophobia, neck stiffness (meningitis).
- Visual disturbance, (migraine, glaucoma, giant cell arteritis).
- Pain/tenderness on the side of head, jaw claudication (pain on chewing), visual disturbance (giant cell arteritis).

Reduced consciousness, confusion, seizures or focal neurological symptoms suggests a significant problem.

WARNING

Sudden-onset headache = subarachnoid haemorrhage until proven otherwise

The headache of subarachnoid haemorrhage may be described as 'like being hit over the back of the head' and may occur with peaks of blood pressure, e.g. exercise or sexual intercourse.

Examination

Inspection

From the end of the bed – is there evidence of recent trauma, excessive bruising, rash, photophobia, altered state of consciousness or irritability? Assess consciousness state using the Glasgow coma scale (Chapter 10).

Neurological examination

There may be no neurological signs in subarachnoid haemorrhage (SAH). Intracranial space-occupying lesions may result in focal signs; look for upper motor neuron signs – focal weakness with increased tone, reflexes, upgoing plantar reflex.

General examination findings on the basis of the history:

- Fever, look for focus of infection, e.g. ENT.
- Cranial nerve examination.
- Meningism implies inflammation of the meningeal layers covering the brain and spinal cord. Movement of the neck or straight leg raising may cause pain.
- In patients over 50 years, check temporal artery tenderness and intraocular pressure.

Investigations

Bedside investigation

- Blood glucose.

Laboratory investigations

- FBC/CRP/blood cultures if infectious cause.
- INR/clotting if on warfarin or suspected coagulopathy.
- ESR if suspected giant cell arteritis.

Lumbar puncture

Lumbar puncture (LP) must not be performed in drowsy or unconscious patients, due to risk of death from coning (↑ICP forces the brain through the foramen magnum, compressing the brainstem). LP must not delay treatment if there is suspicion of meningitis. In possible SAH, LP should be performed at least 12 hours after the onset of headache to allow xanthochromia time to develop. Xanthochromia is the yellow coloration of CSF that occurs when bilirubin leaks from red blood cells, and should be measured quantitatively in the laboratory.

Imaging

- High resolution CT (100 CXR) is sensitive at detecting SAH within 12 hours of onset of pain. MR is good for detecting late-presenting SAH.

Management

Treatment of patients with specific symptoms is discussed below. NSAIDs should not be given to patients with possible intracranial bleeding. Mild dehydration often exacerbates headache: a combination of the following treatments is usually effective.

- *Fluids*: two litres of normal saline.
- *Analgesia*: paracetamol, NSAID, codeine.
- *Anti-emetic*: metoclopramide, prochlorperazine, chlorpromazine.

The combination of chlorpromazine and i.v. fluids seems particularly effective for migraine. Opiate analgesia should be avoided, as there is a high risk of dependence, especially with pethidine (meperidine).

Disposal: who can go home?

Any patient who has severe headache not responding to standard treatments requires admission (10% of patients). Patients who have responded well to treatment, in whom serious pathology has been excluded, are safe to discharge.

Common diagnoses

Tension headache

Very common, multiple possible triggers including psychosocial stressors. Gradual onset of a bilateral/generalised headache, with relatively constant nature over time.

Migraine

Migraines are common, female > male, often with a family history. Many patients will recognise their typical symptoms: unilateral, preceded by nausea and/or visual disturbance ± temporary unilateral numbness. Migrainous neuralgia/cluster headaches are rare, and present with brief (<30 minutes) episodes of eye, facial or head pain or autonomic symptoms clustered in time.

Diagnoses not to miss

Subarachnoid haemorrhage

Subarachnoid haemorrhage (SAH) may be caused by cerebral aneurysms, arteriovenous (AV) malformations, hypertension or head trauma. SAH may occur at any age but is most common between 40 and 60 years. A large SAH typically presents with acute headache, progressing to coma, and is not difficult to diagnose, but the outlook is often poor.

A small bleed with subtle symptoms is difficult to diagnose, as the history may be the only guide that this may be a warning bleed. Identification and intervention in this group of patients may prevent a catastrophic bleed. If the history is good for a SAH and CT is normal, LP is necessary: MRI or CT angiography can resolve equivocal results.

Meningitis and encephalitis

It can be difficult to distinguish between a viral infection from meningitis or encephalitis, as they may present with a similar picture of fever, headache and neck stiffness (Chapter 38). Immediate empirical antibiotic ± antiviral treatment is vital, followed by lumbar puncture if appropriate.

Subdural haematoma

Elderly and/or alcoholic patients who tend to fall are at risk of chronic subdural haematoma (SDH), especially if there is an increased bleeding risk, e.g. warfarin. Physical examination cannot exclude a subdural haematoma: low threshold for CT is required.

Space-occupying lesion

Patients presenting with a headache and new neurological symptoms need a CT to exclude intracranial pathology, e.g. bleeding, space-occupying lesions (SOL).

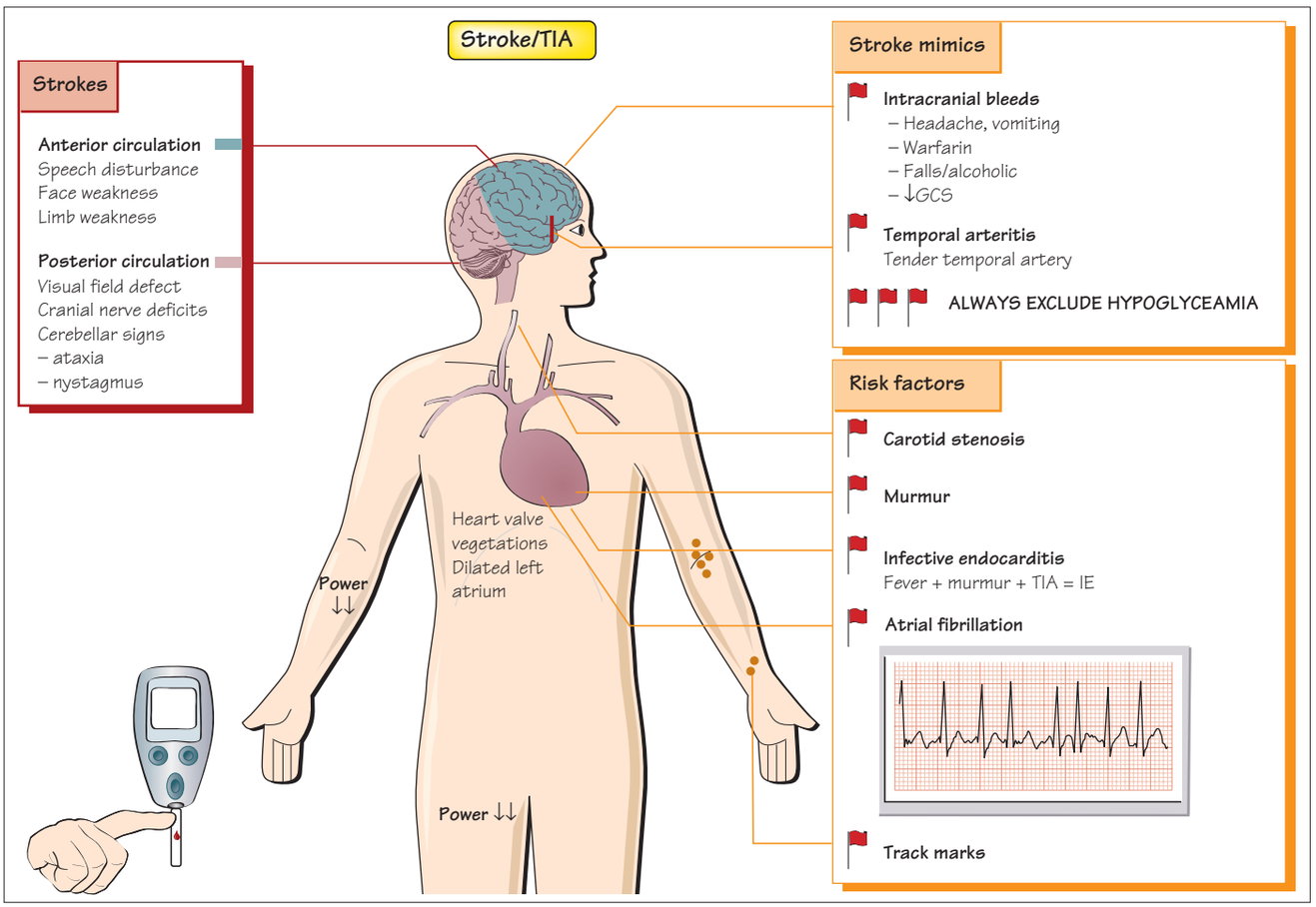
Giant cell arteritis

Must be considered in patients >50 years with unilateral headache ± visual disturbance, jaw claudication, tenderness over temporal artery. If ESR↑, commence on steroids and refer for temporal artery biopsy.

Glaucoma

Acute glaucoma can present with a unilateral headache, and visual disturbance (Chapter 22).

42 Stroke and transient ischaemic attack



Stroke is a major cause of mortality and chronic disability. The advent of thrombolysis for stroke has prompted campaigns to increase public awareness of stroke symptoms e.g. 'FAST – face, arm, speech, time to call ambulance'. Stroke thrombolysis in carefully selected patients reduces disability in survivors. Strokes can be prevented by identification, investigation and treatment of transient ischaemic attack (TIA) patients at high risk of stroke.

Clinical assessment

The time of onset, type and duration of symptoms is critical to decision-making. Many patients have an indistinct onset of stroke symptoms, e.g. have woken with symptoms, and are therefore ineligible for thrombolysis. History and examination should cover:

- Time of onset.
- Risk factors for atherosclerosis (diabetes, smoking, hypertension, ↑cholesterol, family history).
- Risk factors for embolism, e.g. atrial fibrillation (AF), valvular disease, coagulopathy.
- Contraindications to thrombolysis.
- A brief but thorough neurological examination.
- Consider stroke mimics (see below).

Investigations

Bedside investigations

- Blood glucose.
- ECG.

Laboratory investigations

- FBC, U + E, clotting.

Imaging

- Immediate CT/MR to exclude haemorrhage if inside thrombolysis window. Unconscious patients may need intubation to perform CT safely.
- Chest X-ray.

Stroke

A stroke is defined as a 'focal neurological deficit of cerebrovascular cause that persists beyond 24 hours'.

Stroke subtypes: clinical syndromes

- *Anterior circulation infarction* (40%): limb weakness combined with visual field loss and/or cortical dysfunction (e.g. aphasia/apraxia) suggests a partial (20%) or complete (20%) cortical infarc-

tion. If the dominant hemisphere is affected, aphasia and apraxia are common, whereas involvement of the non-dominant hemisphere gives contralateral hemi-neglect.

- *Lacunar infarction* (30%) of the internal capsule results in motor/sensory loss affecting two or more of face, arm and leg *without* visual or speech disturbance.
- *Posterior circulation infarction* (15%): visual problems and/or cerebellar symptoms (ataxia, nausea/vomiting) and cranial nerve deficits are the hallmark of a posterior infarction, although weakness and sensory deficits may also occur.
- *Haemorrhage* (15%) intracerebral (10%), subarachnoid (5%). Warfarin therapy, early vomiting, severe headache, drowsiness and hypertension are common in these patients.

Stroke mimics

- *Hypoglycaemia* must be excluded on arrival. Hypoglycaemia can give a strikingly similar clinical picture to stroke, is easy to diagnose and treat, and if untreated will result in significant neurological damage.
- *Seizures* may give a transient hemiplegia, 'Todd's palsy'.
- Recent trauma or falls suggest *subdural* haematoma.
- Fever suggests *infection*, e.g. sepsis, meningitis, encephalitis, brain abscess, septic emboli.
- A history of cancer warrants exclusion of brain *metastases* using CT with contrast.
- *Migraine* can give a transient hemiparesis, visual, speech or sensory symptoms.
- *Giant cell arteritis*: suspect if visual disturbance in patients over 50 years old. Check for tender temporal artery, raised ESR (Chapter 22).
- Isolated lower motor neurone facial weakness could be due to *Bell's palsy*. Symmetrical normal forehead movement ('raise your eyebrows') implies an upper rather than a lower motor neurone deficit. Inflammation affecting the facial nerve gives unilateral facial muscle weakness. Although most cases will resolve over a few weeks, prednisolone for 10 days improves recovery if started within 3 days of onset. Inability to close the eye needs ophthalmology review.
- *Functional* (psychological/psychiatric) disorders may present as stroke, e.g. somatisation.

ROSIER score

This simple scoring system is better at *excluding* stroke than diagnosing it (+LR = 6.5; –LR=0.09) using a cutoff of 1, i.e. ROSIER ≥1 = stroke more likely, ROSIER <1 = stroke less likely.

ROSIER score for stroke

Clinical sign	Score
LOC or syncope	−1
Seizure	−1
Asymmetric face weakness	+1
Asymmetric arm weakness	+1
Asymmetric leg weakness	+1
Speech disturbance	+1
Visual field defect	+1

If total ≥1 stroke more likely (+LR = 6.5)
If total <1 stroke unlikely (−LR = 0.09)

Treatment

Ischaemic stroke

Patients with ischaemic stroke, confirmed by clinical symptoms and absence of bleeding on CT, *and* who are less than 3 hours from symptom onset are eligible for thrombolysis. After establishing absence of contraindications and obtaining informed consent, thrombolysis is performed with tissue plasminogen activator (tPA).

While some patients benefit greatly from thrombolysis, at a population level the picture is more complicated. Within 3 hours of onset, NNT ≈10, i.e. ten patients have to be treated to save one life or prevent one dependent patient, at the cost of intracranial bleeding (often fatal) in about 5% of patients (NNH ≈20). Beyond 3 hours, the risk/benefit ratio is less favourable.

If the patient has an ischaemic stroke but is not eligible for thrombolysis, aspirin should be started immediately. To prevent aspiration, patients should have a swallowing assessment before being given food or drink. Pressure area care and adequate hydration are essential in immobile patients.

Haemorrhagic stroke

Intracranial bleeding in patients on anticoagulants has a poor prognosis, and must be rapidly halted using fresh frozen plasma or prothrombin complex concentrate (expensive) and vitamin K. Surgical treatment should be considered.

Transient ischaemic attack

A transient ischaemic attack is a focal CNS disturbance caused by transient brain ischaemia from emboli or thrombosis with *complete resolution* within 24 hours. This can make differentiation from stroke difficult; patients with a neurological deficit that is resolving spontaneously should not receive thrombolysis.

The ABCD^2 score estimates the short-term risk of stroke. Patients with high scores must be investigated and treated urgently to prevent a stroke, e.g.

- Carotid stenosis: endarterectomy
- Atrial fibrillation, cardiac failure: anticoagulation

Antiplatelet therapy, e.g. aspirin, should be started immediately if TIA suspected and no contraindications.

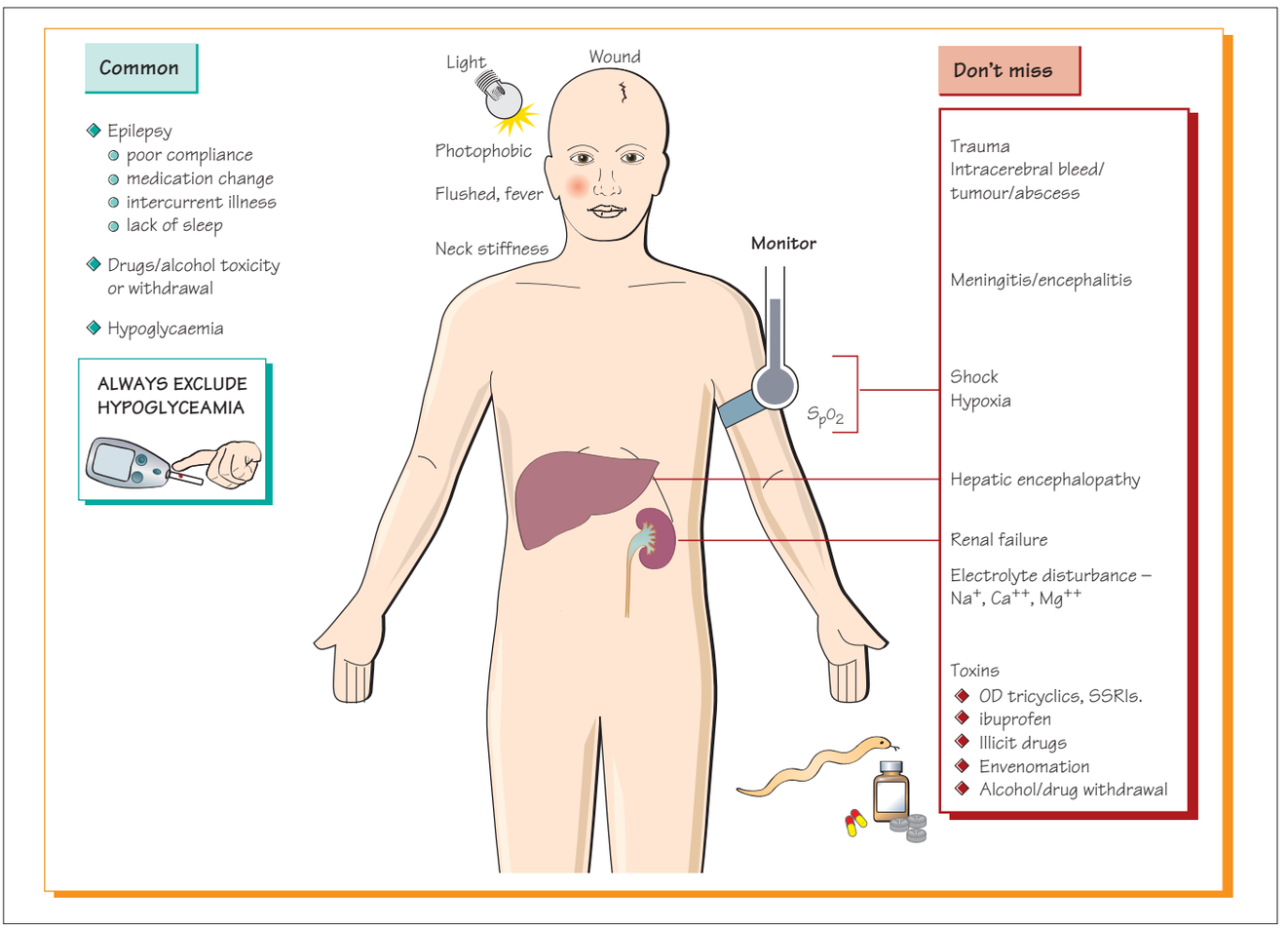
Antiplatelet therapy, e.g. aspirin, should be started immediately if TIA suspected and no contraindications.

ABCD^2 score for TIA

Clinical detail	Score
Age >60 years	1
Blood pressure > 140/90	1
Clinical features:	
Unilateral weakness	2
Speech disturbance without weakness	1
Duration	
>60 mins	2
10–60 mins	1
Diabetes	1

Total >4 = high risk – need urgent investigation
Total >5 = 8% risk of stroke in next 48 hours

43 Seizures



Seizures are the result of electrical 'storms' in the brain. Most seizures occur in people with known epilepsy: these patients often do not come to hospital unless the seizure is different from their normal pattern, or occurs in public. Most seizures last less than 5 minutes, so will have finished by the time the patient reaches the Emergency Department.

In a *generalised* or *tonic-clonic* seizure, the patient loses consciousness, their body tenses (*tonic* phase) and then undergoes a series of rhythmic contractions affecting all their muscles (*clonic* phase). *Partial* seizures exhibit a wide spectrum of patterns of motor activity and sensory disturbance according to the area of brain affected.

Primary seizures occur in patients with no underlying pathological cause. Secondary seizures occur as a result of some pathophysiological process.

Stop the seizure

The ABC rules apply, but with caveats. Never try to splint or force the mouth open. Patients may be cyanosed during the active phases of seizures, but if there is upper airway obstruction try a nasopharyngeal airway. Key points:

- Ensure the patient will not hurt themselves while fitting.
- Give oxygen 100% by reservoir mask.
- Check blood sugar. If below 4 mmol/L, give 50 mL of 50% dextrose i.v. or 1 mg i.m. glucagon.

First line: benzodiazepines

If intravenous access is available, use the doses as listed under 'Second line' below, otherwise give:

- midazolam 10 mg buccal *or*
- diazepam 10 mg rectal.

Second line: benzodiazepines

If the first dose of benzodiazepine has not terminated the seizure after 10 minutes, give a further dose of benzodiazepine:

- lorazepam 4 mg i.v. *or*
- diazepam 10 mg i.v.

If the patient is an alcoholic, give high-dose intravenous thiamine to prevent long-term brain damage.

Third line: anticonvulsant

Phenytoin (or the pro-drug fosphenytoin) is given as a loading dose over 30 minutes and then (if necessary) as a continuous infusion. Patients already taking phenytoin do not need a loading dose. Phenytoin is a sodium channel blocker with cardiac effects, so ECG and blood pressure must be monitored during use, and contraindications include cardiac conduction problems or significant heart failure.

Fourth line: sedation and intubation

A generalised seizure lasting over 30 minutes, or recurrent seizures within 30 minutes without return of consciousness, is described as status epilepticus. If such convulsions continue for more than 60 minutes despite treatment this is 'refractory status epilepticus', and the patient should be sedated and intubated to control the seizure, and transferred to the ICU.

After the seizure: the post-ictal period

After a generalised seizure has finished, it is normal for the patient to be unconscious for a few minutes, longer with high doses of benzodiazepines. When consciousness returns, it is common for the patient to be transiently confused, agitated and sometimes aggressive. Partial seizures may have little or no post-ictal period.

Search for a cause

Was it a seizure?

As with syncope, a good witness history taken as soon as possible after the event provides the most valuable evidence. Other likely differential diagnoses are:

- Syncope/collapse (Chapter 30).
- Hypoxia, metabolic causes – hyponatraemia, hypocalcaemia.
- Toxic causes – see opposite (Chapter 25).

Seizure vs pseudo-seizure

Pseudo-seizures ('non-epileptic attacks') can be very difficult to differentiate from generalised seizures: about 25% of patients intubated for 'seizures' are having a pseudo-seizure. Pseudo-seizures are more likely if there are asymmetric movements, pelvic thrusting, head rolling, resistance to eye opening or no post-ictal period. In pseudo-seizures, incontinence is unusual and tongue biting, if it occurs, involves the tip rather than the sides of the tongue that one would expect in a generalised seizure. The reasons for pseudo-seizures are usually complex and the labelling has significant risks, so should only be confirmed by a senior doctor.

Causes, triggers and auras

In a person with known epilepsy, common causes are non-compliance, changed dose of anticonvulsant, or drug interactions. Alcohol or benzodiazepine withdrawal causes seizures. *Triggers* are factors that may cause fits in people who are not normally prone to fits, e.g. lack of sleep, infections. An *aura* is the feeling that a patient with epilepsy has that warns them that a seizure is imminent.

Examination

Look for head trauma, injuries occurring as a result of the seizure, signs of sepsis/systemic illness or toxidromes (Chapter 24). Any suspicion of a rash or neck stiffness should prompt immediate antibiotics for meningitis (Chapters 38 and 41).

Cardiovascular examination is particularly important because cardiac causes are common differential diagnoses in the young (tachyarrhythmias, hypertrophic obstructive cardiomyopathy: HOCM) and the elderly (bradyarrhythmias, postural hypotension, valvular disease). Collapse due to cardiac causes may be accompanied by a few seconds of jerking limbs due to transient inadequate brain perfusion –'anoxic jerks', which can result in mis-labelling as 'seizures'.

Neurological examination after the post-ictal period is often normal, but if there are focal neurological signs, this should prompt further investigation, e.g. CT. Immediately after a generalised seizure, the plantar reflexes may be upgoing, and there may be ankle clonus. Todd's palsy is a transient unilateral weakness following a seizure that resolves over a few hours, but can be difficult to differentiate from a TIA/stroke.

Investigations

Bedside investigations

- Blood glucose, ECG.

Laboratory investigations

- FBC, U + E, calcium.
- Anticonvulsant levels – not always helpful but should be performed if toxicity suspected (e.g. patient is ataxic).
- Prolactin rises after a seizure: the sample should be taken between 10 and 20 minutes after the seizure, but should only be ordered if the diagnosis is unclear.

Imaging

- CT indicated for first seizure or abnormal neurology.

Disposal: who can go home?

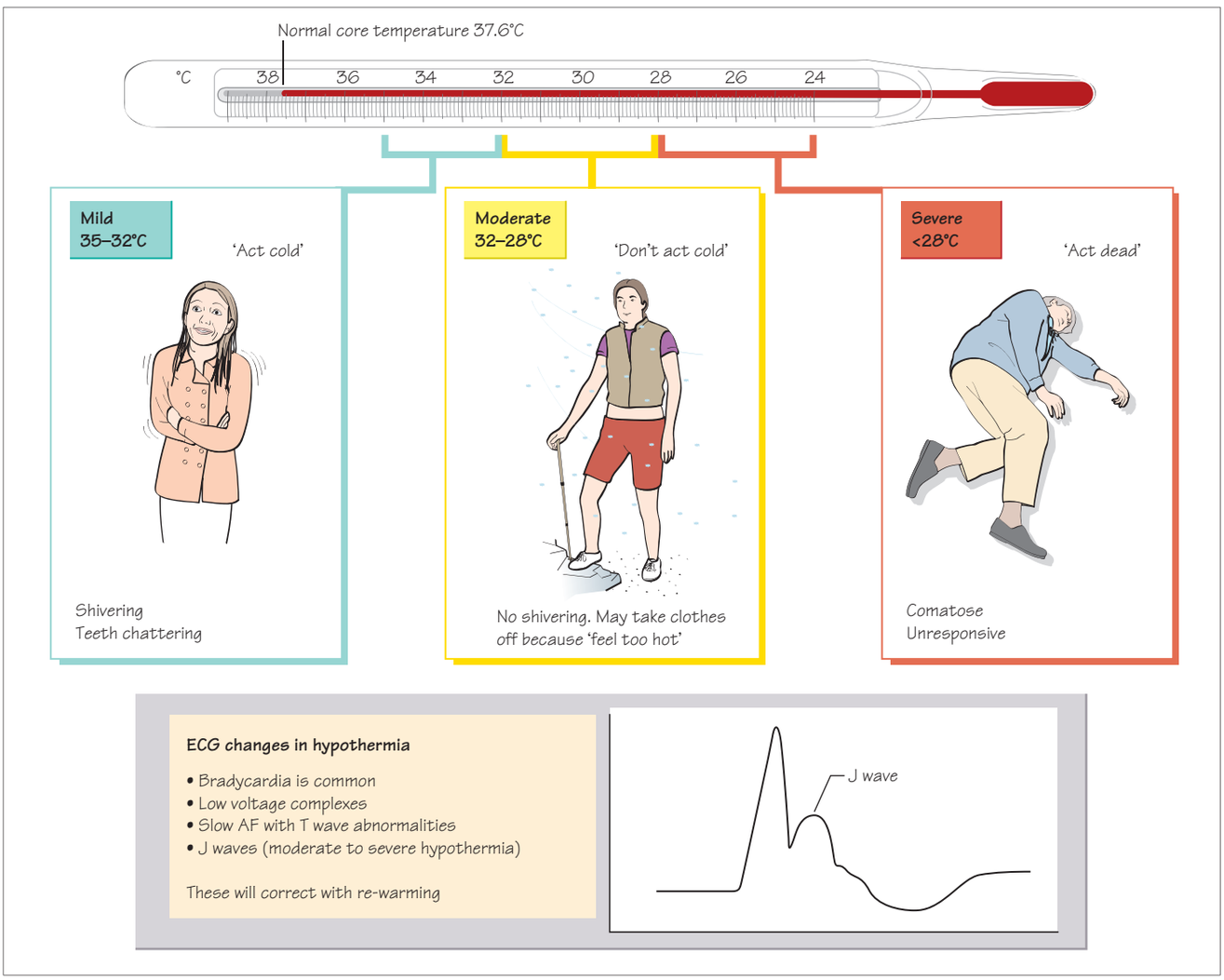
Known epilepsy

Often a person with known epilepsy is brought to the Emergency Department just because they have had a (normal for the patient) seizure in public. If the seizure is within their normal pattern, they have a full recovery, and will be with a responsible adult, discharge is likely to be safe.

'First fit'

Patients not previously known to be epileptic should be observed for at least 4 hours. If no serious underlying cause is found and there are no complications, they may be discharged. On discharge they must be advised, *and this must be recorded in the notes*, to avoid any activity in which a further fit would be dangerous e.g. *driving*, operating machinery, climbing ladders, unsupervised swimming, until they have been reviewed by a specialist. Outpatient clinic follow-up should be organised following EEG and CT.

44 Hypothermia and hyperthermia



Hypothermia

Hypothermia is common in patients 'found on floor' in winter months. The elderly and alcoholics are at risk because of falls. Phenothiazines reduce hypothalamic sensitivity to temperature, and alcohol causes peripheral vasodilatation.

Conduction is the fastest way of losing heat, followed by convection and radiation. Under normal circumstances the hypothalamus responds to cold by directing the body to preserve core temperature by progressively shutting down blood supply to the outside shell – the skin, then the limbs. Muscle contractions are used to generate heat by shivering.

Diagnosing hypothermia

Core temperature is best measured with a nasopharyngeal or oesophageal temperature probe.

Primary assessment

- Remove wet clothing, nurse in a warm, well-monitored environment.
- Standard ABC assessment priorities apply.
- Avoid moving the patient in severe hypothermia, as this may precipitate ventricular fibrillation (VF).
- Consider rhabdomyolysis in patients 'found on floor' – measure creatine kinase (CK).

Rewarming

Deciding whether and how to rewarm is not always straightforward. Factors to consider are:

- Level of hypothermia.
- Rate of cooling.

- Age of the patient.
- Cardiac arrest.

Non-invasive rewarming

Patients with *mild hypothermia* need no special measures as they will generate their own heat – warm blankets are sufficient. Aluminium foil sheets are not effective.

Most patients with *moderate hypothermia*, and *stable* patients with *severe hypothermia*, can be effectively rewarmed at 1–2°C per hour using the combination of:

- Warmed humidified oxygen.
- Warm air blanket.

Warmed humidified oxygen uses the lungs as a heat exchanger and prevents the normal heat loss by evaporation from the lungs. The warm air blanket blows air at 43°C over the body.

Invasive rewarming

Patients with *severe hyperthermia* who are *unstable* may require invasive rewarming, either by filling a body cavity with warmed fluids, or directly heating the blood.

Infusing fluid warmed to 40°C into either the left hemithorax or peritoneum, draining and repeating can rewarm at up to 4°C per hour and is relatively simple to achieve using equipment available in any Emergency Department. The left hemithorax is preferred as it directly heats the heart and great vessels. Unfortunately the less invasive options, bladder or stomach washout, are not effective. The most rapid (1–2°C per 5 minutes) way of rewarming a patient is to use cardiac bypass, but this requires anticoagulation, and is not available in many centres.

Intravenous fluids Warmed fluids prevent further heat loss but are ineffective at rewarming. Practical experimentation with bathwater will demonstrate that adding 1 litre of water at 43°C does not significantly change the temperature of 70 litres of water at 28°C.

Shock may develop as rewarming occurs: intravenous fluids should be warmed to 43°C and given cautiously. In elderly patients, fluid overload may cause cardiac failure as the heart and kidneys may be unable to cope with the increased volume load.

Cardiac arrest

As the heart cools, charactistic 'J' waves are seen on ECG; atrial fibrillation, bradycardia and ventricular fibrillation may occur with progressive cooling, ending with asystole. Cardiac arrest is difficult to diagnose, as a very weak and slow pulse is difficult to detect: ECG and ultrasound are helpful to confirm.

The traditional maxim '*You're not dead until you're warm and dead*' holds true. Full normal recovery has been recorded in patients with prolonged periods of asystole. However, a common feature of patients who survive hypothermic cardiac arrest appears to be that they are not hypoxic while they are cooled.

Normal resuscitation protocols are followed, but defibrillation is often ineffective below 30°C, and is therefore not repeated until the temperature has been raised. Inability to provide invasive rewarming may make resuscitation futile. The decision to stop resuscitation is difficult, but some indicators are:

- A potassium level of more than 10 mmol/L.
- Patients with pre-existent organ failure that would preclude successful discharge from the ICU. Old age alone does not preclude survival.

Hyperthermia

Hyperthermia may be a result of environmental conditions, toxic effects of drugs or, very rarely, disease.

Heat stroke

This is when environmental conditions overwhelm the body's ability to lose heat, sometimes exacerbated by exercise in the heat. Heat exhaustion is a mild form of heat stroke.

The patient will have a temperature above 40°C, confusion/neurological abnormalities and (usually) absent sweating. Rhabdomyolysis and hepatic inflammation may be present, together with a raised white cell count (WCC).

Treatment is by cooling with cold intravenous fluids, tepid sponging and ice packs. Benzodiazepines and intubation may be necessary.

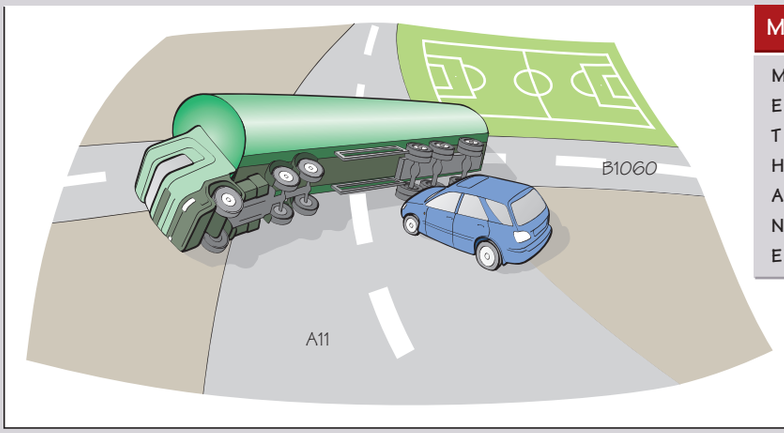
Drugs

Neuroleptic malignant syndrome is a rare side-effect of phenothiazine drugs (antipsychotics), which can present with rigidity, confusion and hyperthermia, usually soon after starting/increasing the drug.

Serotonin syndrome may present with a similar picture of rigidity, confusion and hyperthermia. This may occur in when a selective serotonin reuptake inhibitor (SSRI) drug interacts with another drug, often a monoamine oxidase inhibitor (MAOI). Users of amphetamines and related compounds such as MDMA ('ecstasy') also sometimes develop hyperthermia.

The treatment for all these conditions is benzodiazepines to reduce muscular rigidity, and cooling with cold fluids, tepid sponging and ice packs.

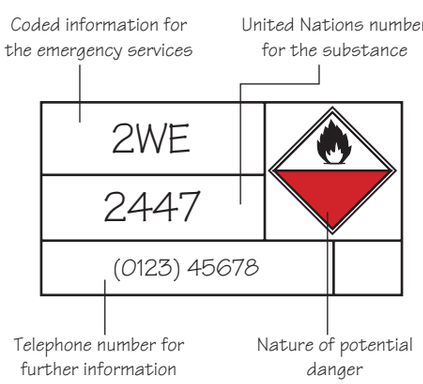
45 Pre-hospital medicine

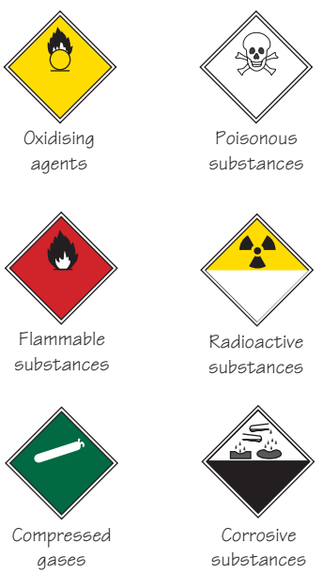


METHANE

- **M** – Me – who you are
- **E** – Exact location
- **T** – Type of incident
- **H** – Hazards present
- **A** – Access – how can emergency services access the site?
- **N** – Number and type of casualties
- **E** – Emergency services present/ needed

"I am XYZ and am ringing you from the intersection of the A11 and B1060 roads notifying you of a two-vehicle crash involving an oil tanker and a SUV. The oil tanker is on its side and appears to be leaking oil and has a hazchem sign stating 2WE 2447 and a flammable symbol, but there is no fire at present. Access is by road and there is a sports field nearby. There are two injured people in the SUV, one of whom is trapped. There are currently no emergency services, and Police, Fire and Ambulance are required."





Hazchem code

1	Jet	3	Foam
2	Fog	4	Dry agent

- **E** Evacuate
- **P** Full protection, may be explosive. Dilute – safe to wash to drain
- **R** Full protection. Dilute – safe to wash to drain
- **S** BA required, may be explosive. Dilute – safe to wash to drain
- **T** BA required. Dilute – safe to wash to drain
- **W** Full protection, may be explosive. Contain, do not allow to enter drains
- **X** Full protection. Contain, do not allow to enter drains
- **Y** BA required, may be explosive. Contain, do not allow to enter drains
- **Z** BA required. Contain, do not allow to enter drains

BA = Breathing apparatus

Pre-hospital and retrieval medicine are subspecialty areas within Emergency Medicine, and demand for these skills is increasing. Doctors with critical care skills in rapid-response vehicles or helicopters attend the scene of an incident or transfer patients from one healthcare facility to another that is better able to meet their needs.

Practising medicine out of a hospital environment is particularly challenging for a variety of reasons.

- Patients may be very distressed, which can make them more difficult to assess and treat.
- Technical reasons: monitoring equipment may be sensitive to vibration. Noise makes stethoscopes very difficult to use.
- Practical reasons: poor visibility, limited amount of equipment, cramped space or limited access.
- Personal reasons: out of 'comfort zone', motion sickness, extremes of heat and cold.

Despite this, doctors who do pre-hospital work tend to be particularly enthusiastic about it. When out of the normal hospital environment, it is particularly important to be appropriately dressed and equipped. Without a basic level of safety equipment, the Incident Officer will not allow you onto a potentially hazardous site.

Dress

- Strong footwear: boots with good soles and toecaps.
- High-visibility jacket/trousers with designation, e.g. 'doctor'.
- Safety helmet ± torch.
- Tabard/hat identifying role.
- Warm clothes.

Equipment

- Stethoscope.
- Trauma scissors.
- Pens and paper.
- Marker pen.
- Identity card.
- Mobile telephone.

If you are taking medical equipment, ensure you know what you have, and how it is packed. Ambulance staff keep a limited range of drugs.

At the scene

If possible, go past the crash and park on the same side. Leave plenty of space for emergency vehicles, leave hazard lights on and lock the car. Leave the keys with the police, if present.

Call the emergency services if this has not already been done. Put on any protective clothing you have.

Safety is the first priority – make sure all naked lights are extinguished and danger from other traffic is minimised. Do not put yourself in danger.

Who is in charge?

If Ambulance Service personnel are present at a incident, they will usually be responsible for medical resources, and you should report to the Ambulance Incident Officer. If it is a large incident there will be Police and Fire Services present, each of which will have their own Incident Officer and control structure. Fire Service takes control if hazardous substances are present; police if firearms may be involved.

The Incident Officer controls the scene by nominating people to perform tasks. Nothing should take place without their knowledge and, at the end of every task, staff should report back to the Incident Officer.

Aircraft

Helicopters are particularly useful in rural and remote areas with difficult vehicle access. In countries with well-developed trauma systems, helicopters allow rapid transfer of patients to major trauma centres, bypassing smaller centres that do not have the range of facilities to care for complex multi-trauma. The optimum range for helicopters is 50–200 kilometres (30–130 miles).

A helicopter weighing 3000 kg generates 3000 kg of downthrust: make sure there is no loose debris on the landing site, which should be 30 × 100 m. Keep well away from the landing site and wear eye protection. Do not shine lights on the landing surface or at the helicopter unless specifically requested to do so.

Do not approach a helicopter unless the rotors have stopped or you have been specifically bidden to approach by the pilot. The helicopter team will usually come to you. Only approach the helicopter from the front – the tail rotor spins at head height.

Fixed-wing aircraft are useful over longer distances, >200 km (120 miles), and have more space, but take longer to prepare.

Radio procedure

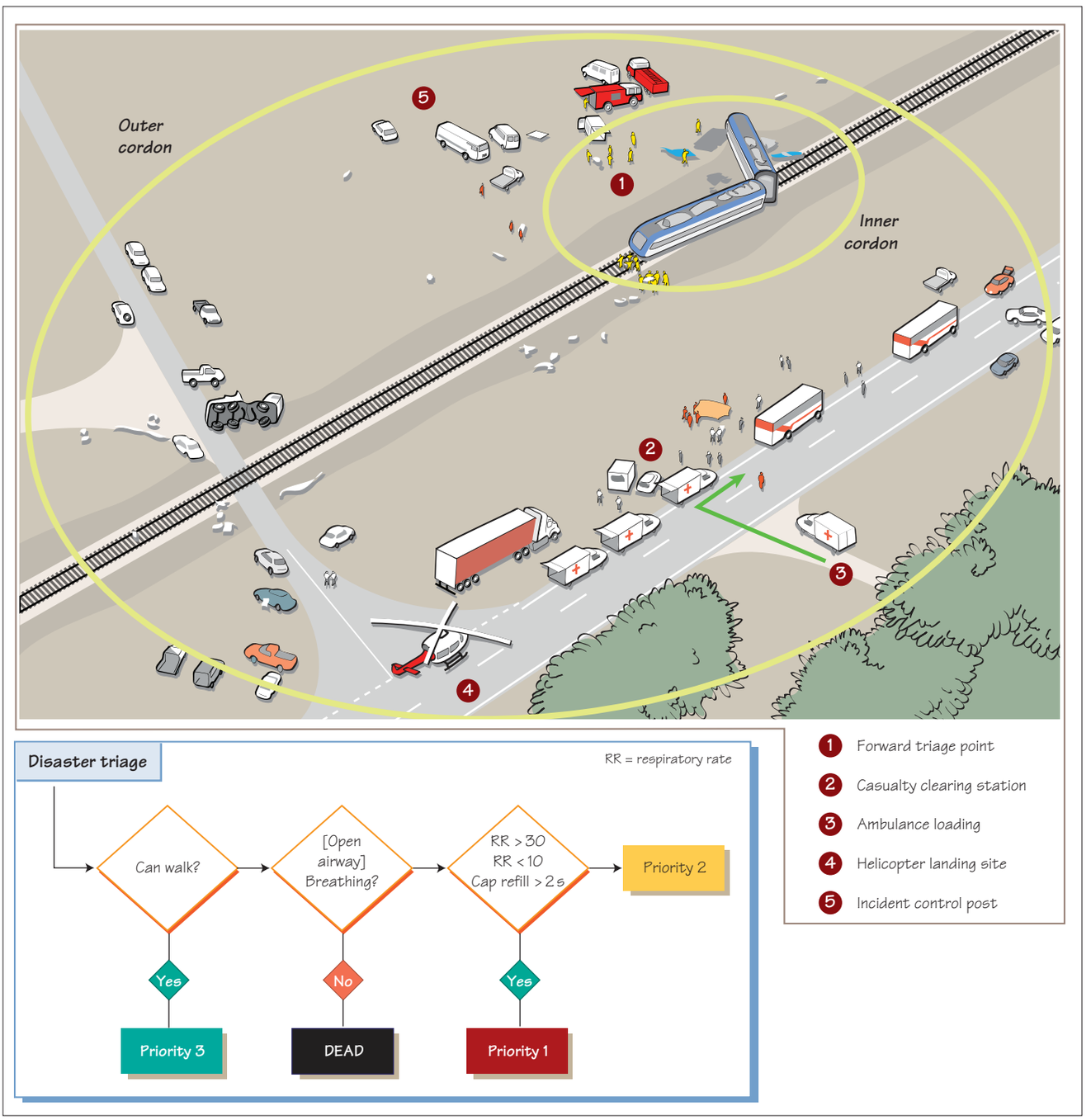
Radios are generally used to communicate at larger incidents. If you have to use one, you will be issued with a call sign. When you talk:

- remember to press the button;
- state who you are;
- end with 'over' to let the other party know you have finished speaking. ('Over and out' is only for movies!)

Phonetic alphabet

A	Alpha	**M**	Mike	**Y**	Yankee
B	Bravo	**N**	November	**Z**	Zulu
C	Charlie	**O**	Oscar	**0**	zero
D	Delta	**P**	Papa	**1**	wun
E	Echo	**Q**	Quebec	**2**	too
F	Foxtrot	**R**	Romeo	**3**	thu'ree
G	Golf	**S**	Sierra	**4**	fa'wer
H	Hotel	**T**	Tango	**5**	fy'vah
I	India	**U**	Uniform	**6**	six
J	Juliet	**V**	Victor	**7**	sev' en
K	Kilo	**W**	Whisky	**8**	ate
L	Lima	**X**	X-ray	**9**	niner

46 Major incident



In medicine, it is normally assumed that there are sufficient resources and personnel to meet patients' needs. In a major incident this may not be the case, and there is a need for a system to ensure that limited resources are used in the most effective way.

Military-style command structures are used to ensure safety and clear lines of responsibility; orders from the scene controller must be obeyed by all on site.

Notification

METHANE is the mnemonic used for notifying a major incident. It is a standardised structure that tells the listener all the key information efficiently (Chapter 45).

Incident management

As the emergency services arrive, the police will take charge and cordon off the incident. The inner cordon contains the immediate area around the incident, and the outer cordon contains all the emergency services attending the incident. Each of the emergency services at an incident has their own separate control officer – a Bronze level controller. Only very large incidents would need high-level (Silver/Gold) controllers.

When the Fire Service has checked that the area is safe, healthcare personnel are allowed access to casualties in the inner cordon. There may be significant ongoing hazards, e.g. unsafe buildings, and in terrorist incidents secondary devices are commonly used to target rescuers.

The Ambulance Incident Officer (AIO) will request medical team(s) from hospitals if the incident is large or serious. Medical teams need to have safety clothing, identifying tabards, equipment and supplies relevant to the incident (Chapter 45). Several medical teams of one doctor and one nurse working together may be needed, together with a Medical Incident Officer to co-ordinate treatment and evacuation.

Incident management priorities

1. Command
2. Safety
3. Communication
4. Assessment
5. Triage
6. Treatment
7. Transport

Major incident triage

Triage is not about *treating* patients, but just *sorting* them so that subsequent medical care can be effectively targeted, and for this reason is best performed by nursing staff. Labels ensure that treatment teams can easily find the high-priority patients, and do not waste time reassessing dead patients. The categories of patient are P (= Priority) 1–3.

Triage in the context of a disaster is very different from normal hospital triage. It is sometimes described as reverse triage as the least ill patients, the walking wounded (P3), are separated first. These are patients who may have significant but not life-threatening injuries.

In a large incident, a forward triage point within the inner cordon allows stabilisation of patients before they are transported to the main casualty clearing station. Once patients arrive at the casualty clearing station, they are re-triaged and evacuated to hospitals according to their injuries and the specialised facilities that different hospitals can offer, e.g. trauma, burns, intensive care, neurosurgery.

The casualty clearing station is the nearest area to the inner cordon that has good transport access. Buildings offer shelter, power and light, which are useful if casualties will need to be held for any length of time.

At the hospital

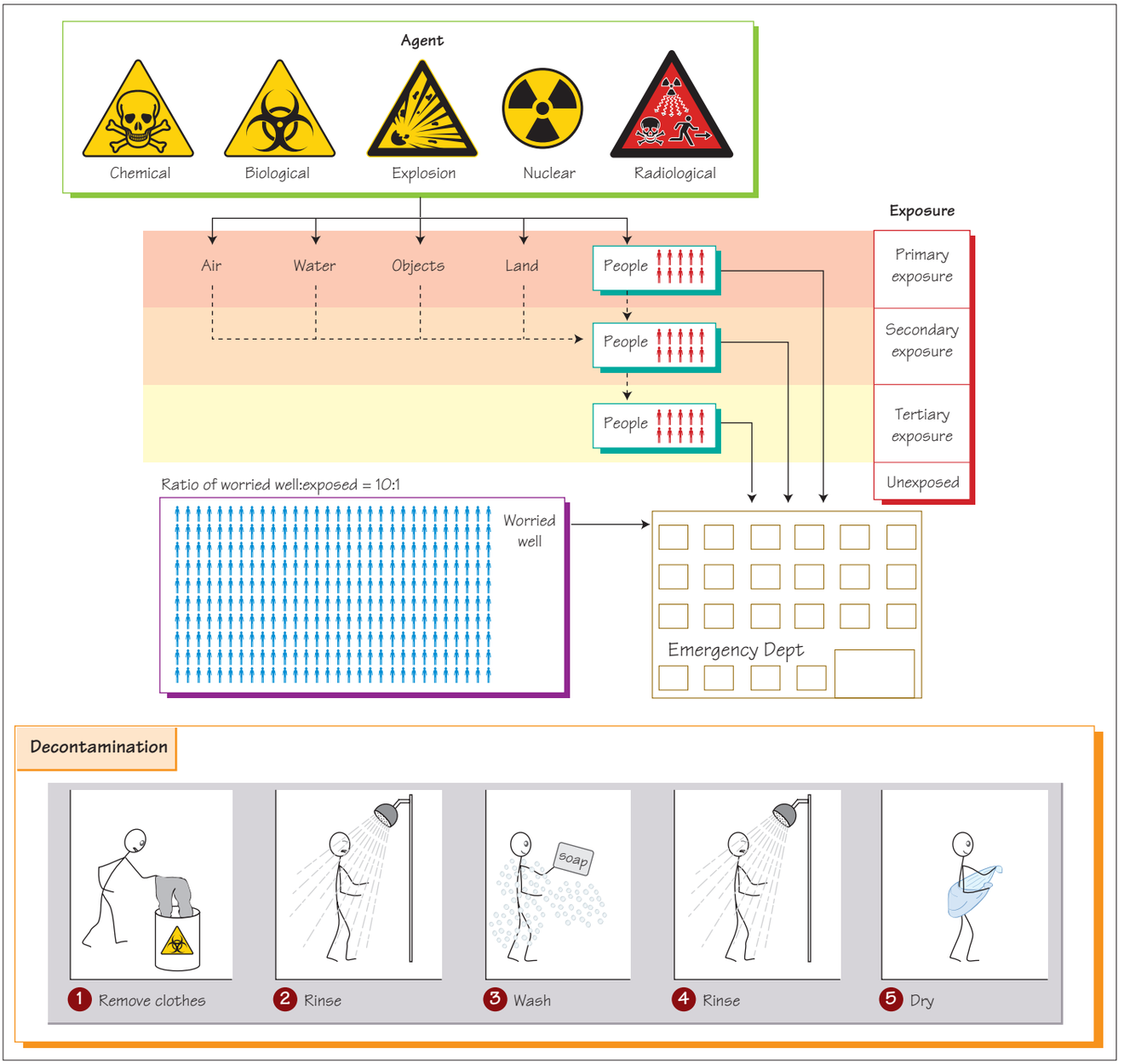
Preparations start as soon as notification is received from the Ambulance Service of a major incident. Medical, nursing, support and administrative staff are notified by the hospital switchboard using a cascade system.

Security within the Emergency Department must be kept very tight ('lockdown') to ensure patient and staff safety, and a robust system of staff identification is essential. Preparations should be made to handle media and relatives away from the Emergency Department. The Emergency Department is cleared of all non-life-threatening emergencies. Inpatient teams conduct ward rounds to discharge all possible patients, and non-urgent operations are cancelled.

Patients are re-triaged on arrival at the Emergency Department and teams of doctors and nurses are allocated to deal with casualties (P1 and P2) in the Emergency Department. An area away from the main Emergency Department is used for 'walking wounded' (P3) patients. X-ray facilities and treatment rooms are necessary, so outpatient clinic areas are often used.

Emergency Department staff need physical and emotional support to cope with a disaster. Food, drink and rest are important for a sustained response, and measures should be taken to ensure that emotional support is available. A 'hot debrief' happens immediately after such an incident, followed up by a full debriefing meeting a few weeks later, so that all involved may discuss and learn from the experience, and the hospital's plans can be updated with this information.

47 Chemical, biological, radiation, nuclear and explosive incidents



Hazardous materials are used in industrial and research facilities, and there is an ongoing risk of terrorist attacks in large urban areas. Health systems must be able to treat both the public and members of emergency services who may have been exposed to hazardous agents. This must be done without harming health-care workers or other patients, as some agents have the potential for secondary harm. Rapid identification and treatment of these agents and use of standard life support protocols are effective.

Several factors determine the impact of hazards on health services.

- Toxicity: ability to kill or incapacitate.
- Latency of action: harmful effect may be delayed, e.g. phosgene gas which produces pulmonary oedema 24 hours after exposure.
- Persistence: resistance to degradation or decontamination (e.g. VX, a nerve agent, 'sticks' to people and objects).
- Transmissibility.

The major incident plan (Chapter 46) is a framework for dealing with a CBRNE incident, as even a small number of patients will impair an Emergency Department's ability to function normally. Toxicological advice is available from national centres listed in Chapter 24.

General approach

Security

Once a CBRNE incident is declared, all patients are considered 'dirty' (potentially contaminated); after screening ± decontamination, they become 'clean'. Clean and dirty areas must be clearly marked and physically separated. Hospital staff working in dirty areas must be protected with Personal Protective Equipment. This may range from gown, gloves, mask, glasses to full 'space suit' type of protection.

The hospital should have a 'lockdown' policy that can be rapidly implemented, limiting entrances – one for staff, the Emergency Department for patients. This minimises the risk of dirty patients putting other patients and staff at risk in clean areas.

Decontamination

Disaster planning often assumes a high degree of compliance – that all dirty patients are identified at the scene, obey officials, do not panic, wait patiently to be decontaminated at the scene and only then are transported to the hospital for medical care.

In reality, even assuming full co-operation by all casualties, there is a time lag between the incident and recognition that an incident may involve hazardous materials. In urban areas, patients may arrive at the Emergency Department before the hospital has been notified of the incident. Therefore all hospitals need facilities to decontaminate patients arriving at a hospital, and these should allow basic treatment of patients while being decontaminated.

Decontamination is managed by the Fire Service, with medical supervision being provided by hospital staff. Inflatable structures are used to provide shelter and a degree of privacy for those undergoing decontamination.

Decontamination involves patients:

1 Removing their clothes, which are bagged and treated as contaminated waste.
2 Rinsing to remove surface contamination.
3 Washing all over with soap and sponges.
4 Rinsing again.
5 Drying and putting on clean paper suit.

Failure to decontaminate patients before they enter the hospital results in contamination of other patients, staff and facilities. This is dangerous and necessitates shutting parts of the hospital for decontamination.

Chemical

Organophosphates

Organophosphates (OP) are used as pesticides and 'nerve agent' chemical weapons: exposure occurs due to ingestion or skin contact. OPs inhibit cholinesterase, resulting in rapid build-up of actetylcholine (ACh). The ubiquity of ACh as a neurotransmitter result in widespread nerve dysfunction.

- Muscarinic effects – DUMBELS (diarrhoea, urination, miosis – small pupil, bronchorrhoea and bronchospasm, emesis, lacrimation, salivation).
- Nicotinic effects – weakness, tremor, fasciculation and paralysis.
- CNS effects – agitation, seizures, coma.

Large doses of atropine are used to reverse the muscarinic effects, followed by pralidoxime to prevent cholinesterase irreversibly binding with the OP.

Cyanide

Cyanide binds to mitochondrial cytochromes to prevent aerobic respiration. Poisoning causes a profound lactic acidosis despite apparent good oxygenation. Without treatment there is rapidly progressive circulatory collapse and death.

Treatment depends on certainty of diagnosis and level of toxicity, but options are:

- chelation using cobalt EDTA;
- high-dose hydroxycobalamin to give cyanocobalamin (vitamin B_{12});
- sodium thiosulfate to produce thiocyanates.

Biological

Although bacterial contamination (e.g. anthrax) is always a possibility, the significant challenges in the immediate future are likely to be viral. The SARS (severe acute respiratory syndrome) infections in 2003 served as a warning about the rapid global spread of infectious disease in the modern age. The swine flu (H1N1) pandemic has proved very benign compared to predictions based on the avian flu (H5N1) influenza, but influenza viruses can combine and mutate.

Radiation

There are two groups of casualties to consider:

- Exposed to radiation;
- Contaminated with radioactive material.

The first group pose no risk to staff, whereas the latter needs careful decontamination, as outlined above. Decontamination should be monitored with a hand-held Geiger counter to ensure complete removal of all radioactive material, and staff should wear radiation dosimeters.

Radiation exposure can be fatal due to bone marrow toxicity and gastrointestinal failure. The time to develop vomiting is proportional to the patient's exposure, and therefore outcome – shorter times indicate higher exposure.

The 'worried well'

In real-life CBRNE incidents, large numbers of asymptomatic people present to Emergency Departments, concerned that they may have been exposed to a hazardous substance. The number of these people is usually much higher than those actually affected by the hazard. However, the former group may become inadvertently contaminated by those who have been exposed (secondary and tertiary exposure).

Depending on the material involved, the Emergency Department must rapidly develop and monitor a way of triaging the 'worried well' so that they can be reassured and swiftly discharged.

Case studies: questions

Case 1: Shortness of breath

A 40-year-old patient known to be asthmatic presents to the Emergency Department with increasing shortness of breath. For the last 2 days, this has been gradually increasing, and she is now using her salbutamol inhaler every 2 hours. She has a fever and productive cough with green sputum. Her chest hurts on the left when she coughs. On examination, she is alert, speaking a few words at a time. On listening to her chest, there is generalised wheeze, together with crackles heard over the left base. She has a peak expiratory flow rate (PEFR) of 190 mL/min, a respiratory rate of 40 breaths/minute, BP 130/80 mmHg, heart rate 110 bpm. A chest X-ray reveals left lower lobe pneumonia.

ABG on room air	Normal values
pH 7.30	pH = 7.35–7.45
Po_2 8.1 kPa (61 mmHg)	$Po_2 \approx$ 11.3–13.3 kPa (≈85–100 mmHg)
Pco_2 4.9 kPa (37 mmHg)	$Pco_2 \approx$ 4.8–5.9 kPa (≈36–44 mmHg)

1 *How severe is this patient's asthma?*
2 *How severe is this patient's pneumonia?*
3 *Comment on this patient's ABG result.*
4 *How should the asthma and pneumonia be treated?*
5 *What will you do for this patient when she is eventually discharged from hospital?*

Case 2: Acutely painful red eye

A 60-year-old female presents with a painful eye, blurred vision, headache and vomiting.

1 *Before you see the patient, list your differential diagnosis, with a brief comment to justify each diagnosis.*
2 *What other clinical findings would you expect if the diagnosis was glaucoma?*
3 *Describe the Emergency Department management of glaucoma.*
4 *If the patient had a painless loss of vision, what other conditions would be likely?*

Case 3: Arterial blood gas interpretation

An 18-year-old patient presents with vomiting due to 'possible gastroenteritis'. The presenting symptoms developed quickly, with fever and non-specific abdominal pain. On examination the patient appears unwell, slightly short of breath, heart rate 120 bpm, BP 90/60 mmHg, respiratory rate 32 breaths/minute, temperature 38.5°C, and no specific findings apart from diffusely tender abdomen.

1 *What does the blood gas on room air indicate?*

pH 7.17; Po_2 11.7 kPa (88 mmHg); Pco_2 2.7 kPa (20 mmHg).

Na^+ 135 mmol/L; K^+ 4.2 mmol/L;

Cl^- 105 mmol/L; HCO_3^- 6 mmol/L.

2 *What is the anion gap?*
3 *What are the differential diagnoses?*

The next patient is a 59-year-old smoker who has previously been treated for anxiety and depression, but has recently been bedbound following a knee operation. He presents to the Emergency Department feeling short of breath over the last day or so. On examination he has a respiratory rate of 32 breaths/minute, heart rate 100 bpm, BP 130/80 mmHg.

4 *(a) If this patient had the following ABG results, what acid–base disturbance would this be?*

pH 7.5; Po_2 10.6 kPa (79 mmHg); Pco_2 2.7 kPa (21 mmHg).

(b) If this patient has the following ABG results, what acid–base disturbance would this be?

pH 7.25; Po_2 7.2 kPa (54 mmHg); Pco_2 5.8 kPa (44 mmHg).

5 *Using the ABG results in 4(a), when this blood test was performed, the patient was breathing 35% oxygen by mask. What should the Po_2 be in a healthy patient with this inspired oxygen concentration, and what does the difference imply?*
6 *How should the patient with the ABG results in 4(b) above be managed?*

Case 4: Abdominal pain

A 57-year-old man presents to triage with abdominal pain. He is usually fairly well but overweight, and has high blood pressure. He had a sudden onset of right flank pain, which is constant, 9/10 severity. He is pacing around the room, and cannot sit still. He has a BP of 147/87 mmHg, heart rate 96 bpm.

1 *What are the differential diagnoses?*
2 *What bedside tests are helpful in differentiating between these diagnoses?*
3 *What imaging would you request?*
4 *Describe your treatment.*
5 *Your investigations show a 4 mm stone in the ureter. How will you decide if the patient is admitted or discharged?*

Case 5: Chest pain

A 40-year-old accountant presents to the Emergency Department with chest pain. He has known hypertension and has smoked 20 cigarettes per day for 20 years. He says the pain is tight across his chest, started three hours ago, and describes it as a 'pressure or tight feeling'. The pain radiates to his neck, and he feels nauseated and short of breath.

1 *List the differential diagnoses for this patient.*
2 *What initial investigations and treatment are appropriate?*
3 *What are the key treatments if the ECG confirms STEMI?*

A 24-year-old IT worker presents with left-sided chest pain, which came on suddenly earlier in the day. He is usually well, and is currently on an adventure holiday. His pain is worse on breathing and coughing. He is slightly short of breath, and has a recent cough and runny nose.

4 *What are the differential diagnoses for this patient?*
5 *A chest X-ray does not reveal a cause of his pain. What further test(s) is appropriate?*

Case 6: Bleed in early pregnancy

You have two patients in the Emergency Department with vaginal bleeding in early pregnancy. The first patient thinks she is having

a miscarriage. Her period is 10 days late, and a urinary pregnancy test was positive a few days ago. However, today she started bleeding and she has lower abdominal pain, worse on the right.

She feels faint and her blood pressure is 80/50 mmHg.

1 *What are the differential diagnoses?*
2 *What immediate actions would you take?*
3 *What further management is needed?*

The second patient is 7 weeks pregnant. She has two children and has had a miscarriage in the past. She has had bleeding all day, with some dark discharge.

She has no abdominal pain. Her GP confirmed her pregnancy with a blood test and ultrasound 1 week previously, which showed an intrauterine gestational sac. An ultrasound is performed in the Emergency Department, demonstrating an empty uterus.

4 *How should this patient be managed?*

Case 7: Head injury

As a member of the trauma team you are helping looking after a patient with a head injury. Your patient is a 23-year-old man who intervened in an argument between an intoxicated man and his girlfriend. He was punched in the face and knocked to the ground, hitting his head on the ground. The man is brought to the Emergency Department semi-conscious, responding only to painful stimuli. Pupils are 4 mm wide, equal and reactive to light, and the patient smells of alcohol. Observations: BP 110/80 mmHg, heart rate 100 bpm, respiratory rate 24 breaths/minute and oxygen saturation 91%.

1 *Is the reduced consciousness state due to alcohol/drugs or head injury?*
2 *What other injuries should be considered?*
3 *What are the priorities in the first phase of stabilisation?*
4 *What features should be sought on further examination?*
5 *The patient has confused speech, opens eyes only to painful stimuli and when you press on the patient's left fingernail, the patient's right hand crosses the midline to try to remove the stimulus. What is the patient's score on Glasgow Coma Scale (GCS)?*

You review him 20 minutes later. He is now making incomprehensible sounds, eyes closed, withdraws from painful stimuli. He is snoring and his oxygen saturation is 94% on 15 L/minute by reservoir mask.

6 *What is the GCS now?*
7 *What should happen next?*

Case 8: Burns

A 33-year-old man is brought in by ambulance with burns. His neighbour heard a loud noise and found the patient unconscious at the bottom of the stairs. The patient has 20% partial thickness burns, including burns to the face. The neighbour is well despite dragging the patient out of the burning house and the thick smoke.

1 *What are the important things the ambulance crew may be able to tell you during handover of this patient?*
2 *Outline the initial assessment and resuscitation of this patient.*
3 *What things must not be overlooked in the resuscitation?*
4 *What analgesia will this patient require?*
5 *Describe the intravenous fluids that this patient is likely to require.*
6 *Should the patient be transferred to a burns centre?*

Case 9: Unconscious

An unconscious 19-year-old man is brought in by his university friends. They think that he has taken a lot of white tablets. One friend says he took an overdose because he had chronic pain and nothing was helping; another thought he was taking recreational pills. Another friend arrives saying he has just found five empty packets of paracetamol (acetaminophen) hidden under his bed, and one empty packet of antidepressants. On examination he has a patent airway, is breathing spontaneously, respiratory rate 12 breaths/minute, BP 90/50 mmHg and warm dry skin, heart rate 120 bpm. His GCS is 13, and he has large pupils, which are symmetrical and reactive to light. Respiratory examination is unremarkable. Abdominal examination reveals no bowel sounds, and a distended bladder.

1 *Describe the immediate management.*
2 *This patient's findings are consistent with which toxidrome pattern?*
3 *What drug is likely to have caused the toxidrome, and how should this be managed?*
4 *What other lethal overdose may be asymptomatic at this stage?*
5 *A colleague suggests giving this patient activated charcoal. Is this a good idea?*

Case 10: Vomiting

You are in the Emergency Department one afternoon when three patients present together with vomiting. The previous evening they all went out to a restaurant to celebrate their football team *'The Magpies'* winning. The restaurant's popularity is based on cheap food and alcohol rather than scrupulous hygiene.

- Patient A is a 20-year-old male, who has diarrhoea, vomiting, a high fever, headache and muscle aches. Has vomited about ten times since 0400. He looks unwell and pale, with cool limbs and has mild non-localising abdominal pain: temp 38.5°C, BP 100/50 mmHg, heart rate 110 bpm.
- Patient B is a 23-year-old female. She has vomiting, high fever, and general aches and pains. She noticed a small amount of bright blood in the vomitus, after the third vomit, and it has continued as she has vomited often since 0400. She looks unwell and pale, with cool limbs: temp 38.7°C, BP 90/50 mmHg, heart rate 115 bpm.
- Patient C is a 22-year-old female. She has vomiting and no diarrhoea. The vomiting has been present for 5 days. She suggests that perhaps her housemates caught a bug from her, otherwise it must have been the food (or alcohol) from last night. Her vomiting is worse this morning. She looks well, and is apyrexial with BP 110/60 mmHg, heart rate 90 bpm.

1 *What possible diagnoses should be considered in these patients?*
2 *What tests are appropriate for patient A?*
3 *Outline your management of patient A.*
4 *What is the likely cause of the bleeding in patient B? What further information would you seek?*
5 *Patient C has had vomiting for longer than her housemates. What other diagnoses should be considered?*
6 *Patient A has been on oral prednisolone 50 mg for 2 weeks for asthma. How will this affect your management?*

Case 11: Weakness

A 57-year-old male patient presents to the Emergency Department after developing weakness of his right hand that lasted about 5 minutes. He now feels completely normal and thinks he is probably wasting your time coming in for a check-up. He has had no palpitations, chest pain or shortness of breath. He has a past history of high blood pressure and cholesterol, but says he is

otherwise fit and well and has no previous medical history. He smokes 10 cigarettes per day and has a 40 pack-year history, takes no medication, and has no known allergies. On examination, BP is 150/95 mmHg, heart rate is 80 bpm and regular. There were no carotid bruits, murmurs or signs of cardiac failure. Neurological examination was unremarkable.

1 *What investigations will you perform?*
2 *Does this patient require admission to hospital?*
3 *What can be done to prevent stroke?*

The patient re-presents 3 days later with a recurrence of the same symptoms, but this time it lasts for 2 hours. You call the stroke team, arrange a CT head which is normal, and then the symptoms resolve, just as the stroke team arrive.

4 *What is your further management?*
5 *How might this be different if the symptoms are persistent?*
6 *What conditions can mimic ischaemic stroke, and what are the key clinical features of each? What investigations are useful in excluding stroke mimics?*

Case 12: Collapse

A 22-year-old man presents to the Emergency Department after an episode of collapse. The collapse did not happen during exercise and the patient does not remember collapsing. Similar episodes have happened before, and the patient now feels completely normal.

1 *What features are suggestive of seizures?*
2 *What features are suggestive of a cardiac cause?*
3 *Describe the patient's ECG (see below).*
4 *The patient is now asymptomatic. What further action would you take?*
5 *If this had been a 60-year-old man presenting with a collapse (with a normal ECG), what factors would influence you to admit the patient for observation and cardiac monitoring?*

Case 13: Headache

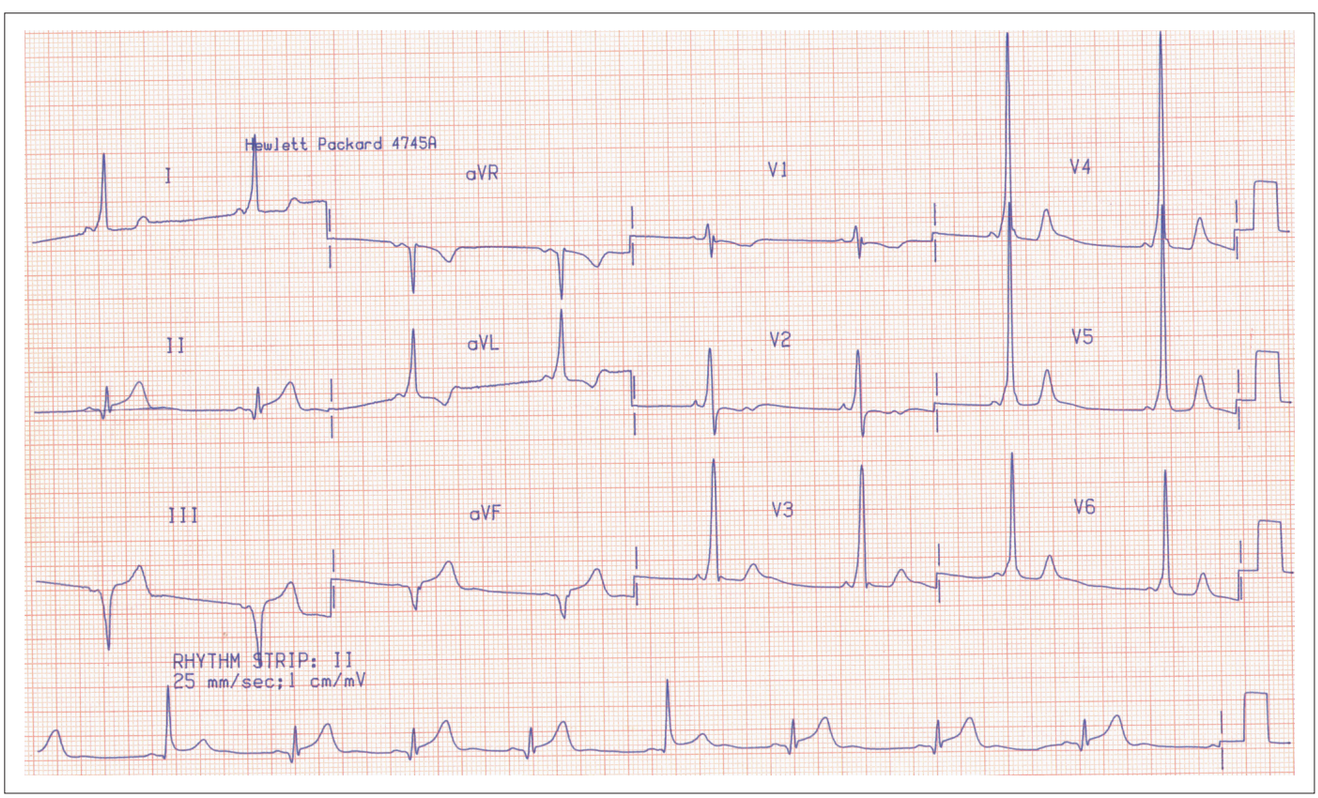
Two patients present to the Emergency Department with headache.

The first patient is a 27-year-old who was having sexual intercourse and had a sudden onset of severe occipital headache. It was the worst headache he has ever had. The headache has now gone and the man is reluctantly here with his girlfriend who wanted him 'checked up'.

1 *What is the most likely diagnosis?*
2 *What investigations will you perform?*

The second patient is a 51-year-old woman who has a long history of headaches. Today the headache is typical of her usual headache (unilateral, with visual symptoms, the light hurts her eyes), and she has nausea and vomiting. The left side of her face feels numb. Her GP is unavailable; he usually gives her an injection of pethidine (meperedine) and then she goes home to sleep it off.

3 *What is the most likely diagnosis?*
4 *What investigations will you perform?*
5 *What treatment is necessary?*



Case studies: answers

Case 1: Shortness of breath

1 The patient has severe asthma because they can only speak a few words at a time, and has increased heart and respiratory rates and hypoxia.

2 CURB-65 uses indicators of organ failure to predict mortality related to the pneumonia: one point each for:

- Confusion (neurological).
- Urea >7 mmol/L (renal).
- Respiratory rate >30 breaths/minute (lungs).
- Blood pressure <90 mmHg systolic or <60 mmHg diastolic (cardiovascular).
- Age 65 years or older.

While there are alternative, similar, predictive tools that could be used, as complexity increases, such tools become less reliable in 'real-life' practice. The CURB-65 has the advantage of being simple and memorable. The CURB score in this case is 1 (one point for respiratory rate), which puts her in the mild/low-risk group.

3 The patient has a mild acidosis: with hyperventilation one would expect the PCO_2 to be low, so the PCO_2 in the normal range is worrying. The symptoms have been present for 2 days, so the (relatively high) PCO_2 is likely to be a sign of relative hypoventilation due to tiredness.

4 A CURB-65 score of 1, on its own, suggests oral antibiotics without hospital admission; however, the severe asthma mandates admission. Therefore the treatment plan would be as follows:

(a) Asthma needs oxygen, continuous nebulisers of salbutamol (or in less severe cases, 12 puffs via spacer); intravenous corticosteroids (hydrocortisone 100 mg). Nebulised ipratropium bromide every 4 hours can be given, but the mainstays of treatment of asthma are β_2 agonists and steroids.

(b) Monitor with regular ABGs over the first few hours. If the patient deteriorates ($PO_2\downarrow$ and/or $PCO_2\uparrow$), magnesium, aminophylline and non-invasive ventilation may be used, depending on local protocols: early senior advice is necessary. Intubation is necessary for patients who present in extremis or who deteriorate despite other measures.

(c) Admission to hospital with appropriate monitoring, e.g. to high dependency unit (HDU).

(d) In view of the patient's level of illness, the first 24 hours of antibiotics should be intravenous.

5 When the patient is discharged, asthma education and an asthma plan are important, together with a letter to the GP, and a reducing course of steroids with enough medication to last until the next appointment. Inhaler technique should be checked, and the importance and benefit of use of spacers for aerosol puffers explained.

Learning points

- In a hyperventilating patient, expect a low PCO_2; if it is in the normal range (and is a true arterial rather than venous sample), this is a red flag warning of tiredness and impending deterioration.
- Scoring tools are helpful, but should not be used in isolation. It is important to consider the whole patient, together with their social circumstances before discharge is considered.

See Chapters 7 and 36.

Case 2: Acutely painful red eye

1 The important differential diagnoses are:

(a) Acute angle-closure glaucoma (correct age, eye symptoms and headache).

(b) Migraine headache (all common except the eye pain).

(c) Acute iritis/scleritis/episcleritis (unilateral red, painful eye, with loss of vision).

(d) Foreign body in eye (unilateral red eye with blurred vision, history, e.g. metal grinding without goggles).

(e) Retinal detachment (floaters and visual loss).

(f) Giant cell (temporal) arteritis in a patient over 50 years old.

2 The findings would include altered (reduced) visual acuity, a red eye, a cloudy cornea, dilated or mid-sized pupil that is unreactive or less reactive. Measurement of the intraocular pressure would reveal raised intraocular pressure.

3 Management of acute angle-closure glaucoma includes immediate ophthalmological referral and commencement of intravenous acetazolamide. Analgesia and antiemetics may also be necessary.

4 If the loss of vision is painless, central retinal artery occlusion or central retinal vein occlusion is likely.

Learning point

- Exclude the important threats to vision first: in this patient, glaucoma and giant cell arteritis.

See Chapter 22.

Case 3: Arterial blood gas interpretation

1 Metabolic acidosis. The pH is low, which means the patient is acidotic. The PCO_2 is low, because the patient is hyperventilating to try to compensate for the acidosis (excess H^+ ions) by moving the equilibrium of this equation to the left by expelling CO_2 from the body.

$$H_2O + CO_2 \Leftrightarrow H^+ + HCO_3^-$$

This patient has a metabolic acidosis with partial respiratory compensation: partial because the patient is still acidotic despite the low PCO_2. Patients with exhaustion, coma or respiratory problems may lose the ability to compensate in this way.

2 The anion gap measures the gap between positively and negatively charged ions in plasma.

$$\text{Anion gap} = (Na^+ + K^+) - (Cl^- + HCO_3^-)$$
$$(135 + 4) - (105 + 6) = 33 \text{ mmol/L}$$

The normal value is approximately 8 mmol/L.

3 Given that this is a raised anion gap metabolic acidosis, the main differential diagnoses are diabetic ketoacidosis, lactic acidosis, and renal failure. Given that the patient is febrile, lactic acidosis may be caused by dehydration/shock/sepsis. This clinical presentation is also consistent with diabetic ketoacidosis. An urgent bedside glucose test is essential. The level of acidosis here means the patient is significantly unwell and needs urgent senior review. In this case the diagnosis is not yet clear; the glucose and lactate should be available as soon as possible.

4 **(a)** Respiratory alkalosis. The pH is high, which means the patient has an alkalosis. The PCO_2 is low, which means the patient has a

respiratory alkalosis. This is most likely due to hyperventilation due to anxiety. However, patients may hyperventilate to maintain oxygenation. The respiratory assessment should focus on excluding physical causes.

(b) Respiratory acidosis. The pH is low, which means the patient has an acidosis. The PCO_2 is high, which means the patient has a respiratory acidosis. It is likely the patient has respiratory failure.

5 The PO_2 is too low for someone on 35% oxygen (approximately double the normal concentration) unless there is some lung pathology that may be causing a ventilation/perfusion mismatch or poor gas diffusion in the alveoli.

The A-a gradient

In room air (21% oxygen) at sea level and with a PCO_2 of 40 mmHg (5.3 kPa),[1] the expected alveolar partial pressure of oxygen is:

$$P(\text{Alveolar } O_2) = P(\text{inspired oxygen}) - P(\text{Alveolar } H_2O) - P(\text{Alveolar } CO_2)$$

$$PAO_2 = PiO_2 - PH_2O - \frac{PaCO_2}{R} \quad [\text{R = respiratory quotient – approx 0.8}]$$

$$PAO_2 = \frac{21}{100} \times (760 - 47) - \frac{40}{0.8}$$

$$PAO_2 \approx 100 mmHg (13.3 kPa)$$

The A-a gradient is the difference between this and the measured value.

The upper limit for the A-a gradient should be

$$= \frac{Age}{4} + 4 \text{ (in mmHg)}$$

[1](1 kPa = 7.5 mmHg)

$$\text{Predicted } PAO_2 \text{ (alveolar oxygen)} = (35/100) \times (760 - 47) - (21/0.8)$$
$$= 0.35 \times 713 - 26$$
$$= 250 - 26 = 224 \text{ mmHg}$$

This gives an A-a gradient of 224–79 = 145 mmHg.
Normal A-a gradient should be Age/4 + 4 = 56/4 + 4 = 19 + 4 = 23 mmHg (3.0 kPa)
This means that there is a significant ventilation/perfusion mismatch or oxygen diffusion problem in the lung. Bearing in mind his recent immobility, pulmonary embolus or pneumonia should be suspected.

6 Inspired oxygen concentration should be increased to a target oxygen saturation of 91% and a senior doctor should review the patient. Reversible causes of respiratory failure such as asthma or pneumonia should be treated; there should be close monitoring in a high dependency unit with repeat ABGs.
See Chapter 7.

Case 4: Abdominal pain

1 Renal colic vs leaking abdominal aortic aneurysm (AAA). 'Renal colic' type flank pain in a patient over the age of 50 is a leaking AAA until proven otherwise. Right upper quadrant (biliary) pain or pleuritic pain involving the right lower chest can also present as flank pain.

2 In this context, the critical intervention is to rule in or rule out AAA as:

AAA + pain = ruptured AAA.

- Hypotension or a pulsatile/expansile mass implies AAA.
- Blood in urine makes renal colic more likely but does not rule out AAA.
- Bedside ultrasound performed by a trained Emergency Department doctor gives an instant definitive diagnosis as to the presence or absence of an AAA, but cannot confirm or deny leakage.

3 Bedside ultrasound is quick, and does not interrupt resuscitation and is the 'gold standard' of care. In stable patients, CT with intravenous contrast provides greater anatomic detail and can demonstrate AAA location, leakage and potential complications, e.g. involvement of renal or spinal arteries. This is helpful in planning surgery/stent placement, and also demonstrates other abdominal pathology that may be present.

4 If leaking AAA, minimal fluid volume resuscitation should be used. Site 2 × large-bore (14 G or 16 G) cannulae, but do not give fluid if the patient is verbally responsive. Notify surgeon or radiologist (if stenting) and anaesthetist. Discuss with blood bank to arrange 6 units of cross-matched/type-specific blood, and give analgesia. Perform baseline blood tests: FBC, U+E, LFTs.

5 A 4 mm stone will most likely pass without intervention, provided:

- the pain has resolved;
- there are no complications such as obstruction, infection or renal failure;
- there are no social reasons requiring inpatient admission.

The patient should be appropriate for discharge if pain-free, or pain is easily managed with oral analgesics and outpatient urology review. Discharge with medication such as NSAIDs or alpha blockers (e.g. tamsulosin) to reduce the number of episodes of ureteric spasm and improve patient comfort.

Learning points

- 'Renal colic' in a patient over 50 is AAA until proven otherwise.
- CT is the diagnostic investigation of choice for 'renal colic'.

See Chapters 3, 19 and 20.

Case 5: Chest pain

1 Differential diagnoses include:

(a) Acute coronary syndrome
  (i) Myocardial infarction, STEMI vs non-STEMI
  (ii) Unstable angina.
(b) Aortic dissection.
(c) Pulmonary embolism.
(d) Upper gastrointestinal tract causes.
(e) Musculoskeletal pain.

2 Start with supportive treatment first:

(a) MONA: morphine, oxygen, nitrate (GTN), aspirin ± intravenous morphine.

(b) ECG within 5 minutes, to detect STEMI or other condition requiring immediate treatment.
(c) Work up to consider if reperfusion therapy is appropriate.

3 Mortality is reduced by:
(a) Antiplatelet therapy. Aspirin alone gives a similar benefit to thrombolysis with minimal risk; ensure aspirin has been given unless there is a real contraindication. Previous mild gastrointestinal upset is not a reason to withhold aspirin.
(b) Reperfusion options include primary percutaneous coronary angioplasty or thrombolysis. Primary angioplasty is the gold standard, but in situations where this is not possible for more than 90 minutes, thrombolysis with clot-dissolving drugs should be considered.

4 Differential diagnoses include:
(a) Pneumothorax.
(b) Pneumonia.
(c) Pulmonary embolism.
(d) Musculoskeletal.

5 A chest X-ray would show a pneumothorax or pneumonia, so a normal chest X-ray effectively excludes these conditions. As this person has a low pre-test probability of pulmonary embolism, a negative d-dimer test could rule out thromboembolism. However, this may not be necessary, and a senior medical review may confirm that this person is most likely to have musculoskeletal pain, probably a result of a cracked rib. X-ray examinations of the ribs are not necessary, as seeing a fracture will not change management.

Learning points

• ECG is the key diagnostic triage tool for patients with chest pain.
• A patient who has presented with chest pain should never be discharged without a senior review. These patients are a frequent cause of litigation.
See Chapters 34 and 35.

Case 6: Bleed in early pregnancy

1 Ruptured ectopic pregnancy or cervical shock. Ruptured ectopic pregnancy is rare, but can bleed faster than blood can be replaced. Simple miscarriage (inevitable abortion) does not cause hypotension, unless complicated by clot in the cervix, 'cervical shock', causing profound vagal drive resulting in bradycardia and hypotension.

2 Notify senior Emergency Department staff and the gynaecology service. Large-bore intravenous access should be obtained, e.g. 2 × 16G cannulae. Intravenous fluid administration should be guided by patient status, not blood pressure. Fluid should not be given while the patient is responsive (e.g. is talking), but if necessary small bolus of crystalloid (e.g. 500mL) should be given up to a maximum of 2 litres when blood (cross-matched, type-matched or group O negative) should be used. An urgent speculum examination will determine whether the cervical os is open or not. Removal of any products of conception or clot from the cervical os using sponge forceps will resolve cervical shock.

3 Send bloods for FBC, clotting and cross-match at least two units of blood. Arrange immediate transfer to the operating theatre and notify anaesthetic staff. Arrange a urinary catheter. Check the patient's blood group: if rhesus-negative the patient will require anti-D immunoglobulin.

4 Given there was a previous normal ultrasound and the uterus is now empty, miscarriage (inevitable abortion) is likely. However, the Emergency Department ultrasound and the results reported by the patient must be verified. Perform a speculum examination to check the state of the cervical os. Verify the previous ultrasound and βhCG results, arrange a formal ultrasound and refer to the gynaecology team. If the results from these tests are equivocal, the gynaecology team may use serial quantitative βhCGs on an outpatient basis to clarify the situation. In a normal pregnancy, βhCG levels double every 48 hours: falling or stagnant results means failed pregnancy. The patient's blood group should be checked: if rhesus negative, anti-D is not always necessary in complete abortion before 12 weeks. Outpatient review and counselling should be offered.

Learning points

• Ruptured ectopic pregnancy is a life-threatening emergency.
• Do not forget to check for Rhesus status in obstetric or gynaecological emergencies.
See Chapter 23.

Case 7: Head injury

1 From the history, head injury is highly likely to be the cause of this clinical picture. To hit one's head on the ground implies loss of normal protective reflexes, i.e. unconscious while falling and/or a high degree of force. A history of alcohol or drug consumption should not change the management as it is dangerous to assume that the reduced consciousness state is due to alcohol. Therefore the patient must be assumed to have a significant head injury.

2 Cervical spine injury: any patient with a head injury should be assumed to have a cervical spine injury. Other traumatic injuries are also possible, e.g. fractured ribs, facial trauma, dental, nose, etc., but the cervical spine must be protected to prevent damage to the cord by movement of an unstable vertebral column.

3 Call the trauma team to ensure adequate numbers and skills of staff. The main treatment priorities are:
(a) Airway: ensure patent and protected.
(b) Cervical spine immobilisation: rigid cervical collar.
(c) Breathing: avoid hypoxia in head-injured patients.
(d) Circulation: avoid hypotension in head-injured patients.
(e) Check glucose.
(f) Imaging: chest X-ray, pelvis X-ray, ultrasound.

4 After the primary survey, continue to assess the patient:
(a) Disability: assess Glasgow Coma Scale (GCS) score and pupils, carry out neurological examination, looking for focal deficits.
(b) Exposure: remove clothing and log roll the patient.
(c) Full secondary survey.
(d) Document injuries.

5 The patient's score on the GCS is 11.
(a) Confused speech = 4
(b) Eyes open to pain = 2
(c) Localising pain = 5

6 The patient's score on the GCS is 7.
(a) Incomprehensible sounds = 2
(b) Eyes closed = 1
(c) Withdraws from pain = 4

7 A falling GCS >2 points or absolute value <9 is a neurosurgical emergency. This patient needs urgent intubation while cervical

spine protection is maintained, and urgent head CT to detect bleeding or raised intracranial pressure.

Learning points

• It is essential to avoid hypoxia or hypoventilation in head-injured patients.
• Make sure you can calculate the GCS quickly and correctly.
See Chapters 8–11.

Case 8: Burns

1 DeMIST:
- Demographics: age, gender.
- Mechanism of injury: fire, fall, smoke inhalation, possible blast injury.
- Injuries sustained: from initial assessment.
- Symptoms and signs, including vital signs.
- Treatment given.

A structured handover is important to ensure transmission of all relevant information. Performing handover in a standardised way minimises communication errors, which are common in stressful situations. The team leader should repeat the key points of the handover back to the ambulance crew so that all present are aware of these, and to check understanding. In addition, details from the AMPLE history may also be available.
- Allergies.
- Medications.
- Previous medical problems.
- Last ate or drank.
- Environment: any hazards, e.g. chemical/heat/cold.

2 Call the trauma team to ensure adequate numbers and skills of staff.

(a) Airway: look for evidence of inhalational burns, carbon/soot in nostrils, facial burns and singed eyebrows/eyelashes/nostril hairs, oropharyngeal redness, swelling, or stridor. Facial and airway burns swell massively in the hours following a burn, and risk occluding the airway. Expert assessment and early prophylactic intubation are necessary to avoid this. This patient is unconscious (i.e. GCS 3), therefore unable to protect his own airway from aspiration of vomit, therefore would need intubation anyway.

(b) Cervical spine protection: this patient has probably had a fall and is unconscious so cannot protect their cervical spine, so a collar is necessary.

(c) Breathing: give 100% oxygen, look for chest trauma.

(d) Circulation: look for shock. While burns alone may cause shock, this patient has other potential causes of shock due to the trauma, so other injuries must be sought.

(e) Disability: assess consciousness state and GCS.

(f) Exposure. initially, cool the burn, then warm the patient.

(g) Analgesia, usually morphine (see below), may require large doses.

(h) Secondary survey to follow, and an estimation of percentage of BSA of the burn, which is needed to calculate fluid resuscitation requirements.

3 Things that can easily be overlooked in a resuscitation situation are:

(a) Airway burns.

(b) Major trauma, electrical injury, inhalational exposure to CO or cyanide.

(c) Forensic issues: good documentation is important, as the police will often require a report.

(d) Deliberate self-harm: if possible, consider paracetamol levels/toxicological testing.

4 Early analgesia includes cooling the burn (but not the patient), dressings (e.g. cling film) and intravenous opiate analgesia.

5 Early intravenous fluids to treat shock:

(a) 20 mL/kg of crystalloid as a bolus if shocked. For an adult this will mean 1–2 litres immediately. Large-bore intravenous access is necessary, and blood/blood products often necessary.

(b) Using the Parkland Hospital formula: 2–4 mL/kg crystalloid × % burn × bodyweight in kg over 24 hours, half given in first 8 hours. This is *in addition* to normal maintenance fluids. Example: 3 mL × 70 kg × 20% burns = 4200 mL per 24 hours, which means 2100 mL over 8 hours then the rest over 16 hours. However, resuscitation should be titrated to adequate circulation/perfusion and urine output, and central venous monitoring may be necessary.

6 Yes. Factors for admission/transfer to a specialist burns centre will depend on local arrangements and policies, but usual reasons to transfer a patient include:
- Full thickness burns.
- Partial thickness burns >10% [burned area, not simple erythema]; or
- Burns involving areas that are difficult to nurse: face/neck, hands, feet, perineum/genitalia/axilla; or
- Circumferential burns: may require incision (escharotomy) to prevent limb ischaemia or allow respiratory movements if chest wall burns.
- Inhalational burns (intubate before transport).
- Electrical or chemical injury.

Learning point

• Severe burns are thankfully rare, but require meticulous assessment and treatment to avoid complications. Pay particular attention to those points that are 'easy to overlook' (question 3 above).
See Chapters 8, 9 and 13.

Case 9: Unconscious

1 Resuscitation: the unconscious patient should be assessed and managed using the standard 'airway, breathing, circulation' protocol. Never forget glucose: hypoglycaemia is an easily treatable cause of unconsciousness that can cause great damage if untreated. Head injury or intracranial pathology should be considered.

2 Toxidromes are collections of symptoms or signs associated with certain toxic drugs/toxins. This patient has an *anticholinergic toxidrome*: tachycardia, altered state of consciousness, dry skin, dilated pupils, urinary retention and reduced bowel sounds are features of this syndrome.

3 A tricyclic antidepressant (TCA) overdose (e.g. amitryptiline), the second most common cause of death due to poisoning in the UK. It is important to recognise the toxidrome because these patients often deteriorate quickly. Recreational drugs such as amphetamines can cause some of these symptoms (wide pupil and tachycardia), but not usually the dry skin, dry mouth and urinary retention. TCA poisoning causes sodium channel blockade giving cardiac and neurological effects. The patient is at risk of ventricular arrhythmias, fitting and coma. If there are signs of serious

toxicity and acidosis, sodium bicarbonate should be given through a large peripheral or central vein.

4 Paracetamol (acetaminophen) is the commonest cause of death from poisoning, and is usually asymptomatic until the patient goes into liver failure 3–5 days after the overdose. *N*-acetylcysteine should be given now, as with five empty packets, it is likely the patient has taken a toxic dose (>150 mg/kg), and there is no clear time of ingestion from which to plot a paracetamol level on the graph.

5 Activated charcoal appears helpful if given soon after ingestion (within 1–2 hours). The patient needs a protected airway (i.e. is alert and drinks it voluntarily, or is intubated). This patient has a reduced GCS, increasing the risk of aspiration and therefore activated charcoal should not be given.

Learning points

- A clear understanding and chronology of events surrounding presentation are often not apparent when a patient first attends the Emergency Department. Assessment and treatment must 'cover all the bases', and so overdose must be considered in this situation.
- Always test for paracetamol, as by the time overdose is symptomatic, it is too late.

See Chapters 24 and 25.

Case 10: Vomiting

1 Differential diagnoses:

- Infectious gastroenteritis: *Salmonella*, *E. coli*, *Shigella*, *Campylobacter* (usually >48 hours).
- Toxin-mediated food poisoning: *Clostridium*, *Staphylococcus aureus*, *Bacillus cereus*
- Infectious gastroenteritis: viral.
- Alcoholic gastritis.
- Inflammatory bowel disease (unlikely in two patients at same time).

2 Bedside test: blood glucose.

Laboratory tests:

- FBC, U+E and amylase, to detect an elevated white cell count, assess dehydration and rule out acute pancreatitis.
- Blood cultures if clinically septic.
- Stool sample microbiology and culture if suspected food poisoning, as these are generally notifiable diseases due to public health and safety issues.

3 The patient appears to have toxin-mediated food poisoning. Management is supportive with antiemetics such as metoclopramide or prochlorperazine, and intravenous saline to treat dehydration. Patients can usually be discharged home safely when tolerating oral fluids and oral antiemetics.

4 Patient B probably has a Mallory-Weiss tear as there is a history of vomiting followed by subsequent blood-streaked vomits. Blood in the first vomit is much less likely to be Mallory-Weiss tear. Check for symptoms of peptic ulcer disease or reflux oesophagitis, such as indigestion or burning epigastric pain.

Use the Glasgow-Blatchford score to identify patients at low risk of further problems. A patient who fulfils the following criteria can be discharged safely, providing the patient has the resources to re-attend hospital if required.

- Haemoglobin level >12.9 g/dL (men) or >11.9 g/dL (women).
- Systolic blood pressure >109 mmHg.
- Pulse <100 bpm.
- Blood urea level <6.5 mmol/L (blood urea nitrogen level <18.2 mg/dL).
- No melaena or syncope.
- No past or present liver disease or heart failure.

5 Patient C has had symptoms for 5 days. This means that food poisoning from the previous night is unlikely to be the cause. The absence of diarrhoea despite this duration of illness means that other causes should be considered, including pregnancy (morning sickness), urinary tract infection, Addison's disease and medication side-effect.

6 Normal hypothalamic/adrenal function is suppressed by more than 5 days of exogenous steroids. Steroids provide immunosuppression that makes a patient more vulnerable to disease, and also suppress the body's normal inflammatory response to disease, potentially masking more serious presentations, e.g. sepsis. If steroids cannot be absorbed, e.g. due to vomiting, sudden withdrawal occurs, which may precipitate an Addisonian crisis. As this presentation may include a combination of dehydration, vomiting and low blood pressure, it can easily be missed. A short synacthen test is easy to perform in the Emergency Department, and then intravenous saline and steroids should be given.

Learning point

- Although it is tempting to reassure and discharge such patients, one must be alert to other, more serious, diseases that may also present with such common symptoms. That said, one must also avoid over-investigating patients with minor illnesses presenting to the Emergency Department.

See Chapters 38 and 40.

Case 11: Weakness

1 The following investigations should be performed:

- Bedside: ECG, looking specifically for atrial fibrillation or ischaemic heart disease; and a fingerprick blood glucose test to exclude hypoglycaemia.
- Blood tests include FBC, U+E and clotting.
- Chest X-ray to detect cardiac or respiratory disease.
- CT head, carotid Doppler and echocardiogram need to be performed within 1 week, based on the clinical information given. The use of early CT for transient ischaemic attack (TIA) is used mainly to detect stroke mimics, most of which do not have full resolution of symptoms.

2 The $ABCD^2$ score accurately predicts the risk of short-term stroke. Patients with $ABCD^2$ score >4 or crescendo (increasing) symptoms are at high risk (5%) of further ischaemic events within the next week. These patients need urgent investigation to identify and correct reversible factors. This patient has $ABCD^2$ score of 3:

- Age >60 years = 0 points
- BP >140/90 mmHg = 1 point
- Clinical features unilateral weakness = 2 points
- Duration of symptoms <10 minutes = 0 points
- No diabetes = 0 points

A patient with an $ABCD^2$ score of 3 should be reviewed by a specialist within 1 week.

3 Stroke can be prevented by:

(a) Aspirin or other antiplatelet agents (give to all patients unless contraindicated).

(b) Suppression of atrial fibrillation.

(c) Anticoagulation if there is persistent or paroxysmal atrial fibrillation or cardiac failure.

(d) Carotid enderarterctemy if carotid stenosis >70%.

4 The $ABCD^2$ score is now 5, by adding 2 points for duration of symptoms longer than 60 minutes. The recurrence of symptoms so soon after the initial episode suggests crescendo symptoms. Both of these mean that the patient should be admitted for treatment and urgent investigation including carotid Doppler and echocardiogram.

5 If the patient's symptoms persist, the likely diagnosis is ischaemic stroke. The patient is potentially eligible for thrombolytic therapy if stroke is confirmed by clinical symptoms and absence of bleeding on CT, and if less than 3 hours from symptom onset. After establishing absence of contraindications and obtaining informed consent, thrombolysis is performed with an infusion of tissue plasminogen activator (tPA).

6 A number of conditions can mimic stroke:

(a) *Hypoglycaemia*, which *must* be excluded in all patients on arrival.

(b) Space-occupying lesions or intracranial pathology:

(i) Intracerebral abscess

(ii) Brain tumours (primary or secondary)

(iii) Intracerebral haemorrhage, especially if coagulopathy/anticoagulation medication

(iv) Subdural haematoma (especially falls in elderly/alcoholic patients).

(c) Temporal arteritis: tender temporal artery with visual disturbance.

(d) Infective endocarditis and septic emboli: any patient with fever, murmur and TIA. Intravenous drug users at particularly high risk. Look for splinter haemorrhages.

(e) Post-ictal states: transient hemiplegia ('Todd's palsy') is reasonably common. Patients need a CT scan if they have a focal neurological deficit, new-onset seizures, or seizures that are not typical for them.

Learning point

• Stroke is a major cause of disability, and early aggressive investigation and treatment can effectively prevent strokes.

See Chapter 42.

Case 12: Collapse

1 A previous diagnosis of seizures makes seizure more likely, especially if there has been medication change, poor compliance or poor sleep, all of which may precipitate a seizure. A witness description of loss of consciousness, with a history describing the tonic and then clonic phase of a seizure, followed by altered state of consciousness, and gradual return to normal is classical for seizures. Biting of the tongue and incontinence are common.

2 A past history of cardiac disease or cardiac medication should be sought. A family history of cardiac problems or sudden death is important. A prodrome that is brief or absent suggests an arrhythmia. Collapse occurring on exertion can suggest a cardiac cause such as hypertrophic obstructive cardiomyopathy (HOCM) or arrhythmia.

3 There is a short PR interval (<120 ms), and slurred upstroke of the QRS delta (from the shape of the Greek letter Δ) wave. This ECG appearance is caused by an abnormal electrical connection between the atria and ventricles. Premature activation of the ventricles (the delta wave) occurs because the conduction is not subject to the normal electrical delay that occurs in the atrioventricular (AV) node, hence the short P-R interval.

4 This is an important ECG to recognise: although the patient is now asymptomatic, it is likely that he is having episodic tachyarrhythmias. Treatment includes drug therapy and referral for ablation to a cardiologist specialising in electrophysiology.

5 High-risk features in patients presenting with collapse suggest need for admission for cardiac monitoring and further investigation. These include:

(a) History of heart failure (an important predictor) or ischaemic cardiac disease.

(b) Syncope on exertion, breathlessness.

(c) Abnormal ECG: long QT intervals, short QT interval.

(d) Hypotension.

(e) Age > 65 years, comorbidities, lack of social support.

(f) Family history of sudden death.

(g) Haematocrit <30%.

Learning points

• Collapse with loss of consciousness is always serious.

• Collapse occurring in patients with evidence of cardiac disease, especially heart failure, is associated with much higher mortality.

See Chapter 30.

Case 13: Headache

1 Subarachnoid haemorrhage (SAH): this presentation is consistent with a herald/sentinel/warning bleed. Although SAH is rare in young people, diagnosis at this stage, before a catastrophic rupture, can be life-saving.

2 If SAH is suspected, a CT brain is mandatory. High-resolution (64-slice) CT scanners are nearly 100% sensitive for SAH in the first 12 hours after onset of symptoms. If CT is normal, lumbar puncture should be performed at least 12 hours after onset of symptoms to detect xanthochromia. Xanthochromia is caused by bilirubin, a blood breakdown product, in the CSF. If these results are equivocal, MR scanning may be performed, which may pick up evidence of bleeding up to 2 weeks after symptoms.

3 Migraine headache. Other possibilities include tension headache, glaucoma or giant cell arteritis. Other serious illnesses such as meningitis and intracranial space-occupying lesions should be considered, but are unlikely if the headache is in the same pattern as previous migrainous headaches.

4 CT brain is not necessary if the pattern of symptoms is the same as has occurred previously in this patient.

5 Pethidine (meperidine) is a very poor choice of analgesic drug for this situation. It is a lipid-soluble opiate with a short duration of action and pethidine has a high risk of causing dependence/addiction. Better choices for treatment of severe acute migraine would be metoclopramide, prochlorperazine or chlorpromazine together with generous amounts (2 litres) of intravenous fluids. Triptans, e.g. sumatriptan, block 5HT 1B/1D receptors and can be helpful, but are expensive and have higher rates of rebound headache than the other treatments described. The doctor treating this patient should advise their senior doctor in the Emergency Department, who can contact the patient's GP to help ensure that the patient does not become habituated to opiates.

See Chapter 41.

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Note: Q following a page reference indicates the case study questions.