Resuscitation xxx (2016) xxx–xxx

Contents lists available at ScienceDirect

Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation

Review article

Impact brain apnoea – A forgotten cause of cardiovascular collapse in trauma

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ARTICLE INFO

Article history:
Received 20 December 2015
Received in revised form 20 February 2016
Accepted 5 May 2016

Keywords:
Impact brain apnoea
Traumatic brain injury
Resuscitation

ABSTRACT

Objective: Early death following cranial trauma is often considered unsurvivable traumatic brain injury (TBI). However, Impact Brain Apnoea (IBA), the phenomenon of apnoea following TBI, may be a significant and preventable contributor to death attributed to primary injury. This paper reviews the history of IBA, cites case examples and reports a survey of emergency responder experience.
Methods: Literature and narrative review and focused survey of pre-hospital physicians.
Results: IBA was first reported in the medical literature in 1705 but has been demonstrated in multiple animal studies and is frequently anecdotally witnessed in the pre-hospital arena following human TBI. It is characterised by the cessation of spontaneous breathing following a TBI and is commonly accompanied by a catecholamine surge witnessed as hypertension followed by cardiovascular collapse. This contradicts the belief that isolated traumatic brain injury cannot be the cause of shock, raising the possibility that brain injury may be misinterpreted and therefore mismanaged in patients with isolated brain injury. Current trauma management techniques (e.g. rolling patients supine, compression only cardiopulmonary resuscitation) could theoretically compound hypoxia and worsen the effects of IBA. Anecdotal examples from clinicians attending head injured patients within a few minutes of injury are described. Proposals for the study and intervention for IBA using advances in remote technology are discussed.
Conclusion: IBA is a potential cause of early death in some head injured patients. The precise mechanisms in humans are poorly understood but it is likely that early, simple interventions to prevent apnoea could improve clinical outcomes.

Introduction

Trauma remains the commonest cause of death in under 45's[1] with traumatic brain injury (TBI) the commonest mode of this death.[2,3] TBI also results in considerable morbidity to many patients and families. In the US alone, 5.3 million people live with TBI related disability.[4] This societal and economic burden is such, that even small measures to improve outcome could have a great effect.

A number of deaths appear to occur within 10 min, the so-called critical phase of head injury[5] some of which, at post mortem, are found to have diffuse swelling with little parenchymal injury. These patients who die of "TBI" with minimal parenchymal injury may have had hypoxia as a contributor to their brain injury.

Impact Brain Apnoea is the cessation of breathing after traumatic brain injury. It is frequently witnessed in animal TBI models with apnoea duration increasing with energy of brain injury. It is thought to be exacerbated by alcohol and early artificial ventilation through the period of apnoea is thought to be an effective treatment.

Impact Brain Apnoea (IBA) is a phenomena that may occur during the critical phase and is characterised as the patient appearing lifeless or dead; pale with no respiratory effort.

Clearly if this period of apnoea does not self-terminate or no artificial ventilation is instigated the patient or animal has a hypoxic cardiac arrest.

In recent years IBA has most commonly been described in two contexts – motorcycle racing (because the mechanism and first few

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http://dx.doi.org/10.1016/j.resuscitation.2016.05.007
0300-9572/

minutes are often witnessed by medics) and blast injury (again often witnessed in the military field).

Animal studies imply it occurs in many other mechanisms of injury, though the immediate few minutes after injury are rarely witnessed.

Impact Brain Apnoea is distinct from airway obstruction that commonly occurs with brain injuries resulting in a reduced level of consciousness. Whilst basic manoeuvres to open an airway (e.g. jaw thrust) will relieve airway obstruction, they will not necessarily result in return of ventilation if apnoea is present. Nonetheless, basic life support airway interventions are the first component of assessment and treatment in trauma patients with reduced consciousness.

The increased mortality associated with hypoxic events following TBI is significant and has been demonstrated with multiple studies.[6,7] Airway management techniques (such as intubation) that can be provided by advanced pre-hospital services have a role in preventing aspiration and minimising hypoxic episodes but this is not the focus of this paper.[8,9]

The aim of this paper is to describe the history and phenomena of impact brain apnoea as a clinical entity. In addition, we describe the clinical experience of those who have encountered this condition while working in pre-hospital care.

Method

We conducted a literature review of the PubMed database from 1969 using MESH terms [Brain Injury] and combinations of [Pre-Hospital], [Apno(e)a], [catecholamine], and [resuscitation]. We reviewed references from papers found on the PubMed searches and abstracts from recent international scientific meetings relating to trauma and brain injury. We sought historical articles through cross-referencing.

In addition, we contacted a group of clinicians involved in pre-hospital and motorsport medicine to whether the phenomena was still witnessed within modern trauma systems. These papers and the anecdotal experiences of PHEM clinicians informed this review of IBA and the implications for practice and research.

Historical accounts of Impact Brain Apnoea in humans

In 1705, Alexis Littré reported the death of a condemned criminal who, anticipating his executioner, head banged until dead without breaking his skull. Sabatier, Boyn and Mourier also reported similar incidents of alleged fatal concussion though this concept of death from head injury without intracranial lesions was questioned by Miles.[10] Some believed the brain was thrown into a state of vibration which temporarily suspends brain functions.[11] Others argued it related to capillary microhaemorrhages that were sometimes seen.[12] Although brainstem haemorrhages classically are attributed to Duret, it was Richard Bright (1831) who first described them but even he specifically stated that he did not think they would account for the symptoms of concussion or coma.[13] Duret (of a similar opinion) "sought as carefully as possible at the autopsies" and found only "small lesions quite insufficient to explain so grave phenomena".[14] Duret performed many experiments and came to the conclusion that the gravest action following a blow to the head was "about the collet of the medulla and particularly at the level of the floor of the ventricle and on the restiform bodies". It is damage (not necessarily visible damage) to this area that he and others shortly after him believed resulted in concussion and convulsions.[14,15]

Whilst we rarely consider the respiratory changes of concussion, they form part of Koch and Filehne's[16] early description of the condition: Concussion is a state of more or less disturbed consciousness with lost or practically lost reflectivity. The appearance is that of sleep or apparent death, there is occasional vomiting. The respiration is slow, shallow and regular, the pulse is weak, slow and generally regular, the pupils are dilated and react sluggishly, the temperature is subnormal.

Some eminent physicians argued that lesions could not be the cause of unconsciousness as unconsciousness was transient yet the lesions would take time to resolve. Moreover, some patients with more severe lesions were, in comparison, relatively well.[10] Because of this, a vascular theory of reduced consciousness – an "anaemia of the brain" was put forward by Hutchinson and experiments were performed by Miles to investigate this.[10]

In 1958, Newcastle neurosurgeon Ian Maciver and colleagues wrote in the Lancet on "The role of respiratory insufficiency in the mortality of severe head injuries".[17] They claimed that anoxia causes much of the death following TBI and that if this could be prevented, many would survive their brain injury. His group believed that direct brain stem injury causing cessation of respiration (by respiratory centre damage) and aspiration obstructing the airway (through vagal nuclei injury akin to bulbar poliomyelitis) were the primary hypoxic events. Regarding aspiration they questioned lying patients supine and advocated physician led ambulance team dispatch to such patients to relieve airway obstruction. Maintaining brainstem oxygenation they claimed would radically alter outcome. Over 50 years later virtually all pre-hospital systems still transport patients supine,[18] in contrast to the lateral trauma position used in Norway which may offer less respiratory compromise [19]).

"The combination of coma, hyperpyrexia, and decerebrate rigidity, which precedes death in these cases of severe injury, is similar to that following asphyxia without any associated trauma, and we believe that cerebral oedema is essentially caused by anoxia secondary to respiratory difficulties. The ultimate success or degree of permanent damage, in our view, depends on the degree and duration of the associated hypoxia." [17]

Post mortem findings in acute brain injury

The authors are aware of many cases of death where the cause of death at post mortem is "head injury" despite an absence of any specific lesion beyond generalised brain oedema. Fischer described this in 1870: "you will ask" he said "what sort of queer disease picture is this that to-day threatens the patient with the severest of symptoms, all of which tomorrow will have passed completely away".[13,20] In two cases, he had found congestion of the surface vessels and dural sinuses but nil else – hence the term "cerebral anaemia". Possibilities of cerebral vasospasm causing restriction in CBF were argued with those supporting a neurogenic cause.[13] In the UK Jefferson argued against a vascular theory of concussion.[13]

Jefferson says the following of "Trauma as a disease process" – The discovery of cortical injury (though much less than the surgeon can inflict with impunity), the finding of subarachnoid haemorrhage (though no more in amount than many with leaking aneurysms survive), the presence of relatively small quantities of subdural blood, are not enough to account for death, though I should be the last to deny that all of these things can cause death when they are severe enough. But it must be strongly asserted that the findings in human beings are often disappointingly small – disappointing, that is, to the mind which is prepared only for anatomical findings. Yet, it is not these things, nor the petechial haemorrhages of classical times, which tell us most: it is rather the variability or their absence.

In 1977, Yates demonstrated from a 5-year study of trauma necropsies, that in patients dying in hospital up to 72 h post injury, those with obstruction of the airway had less severe injuries than those in whom no such obstruction could be found.[21]

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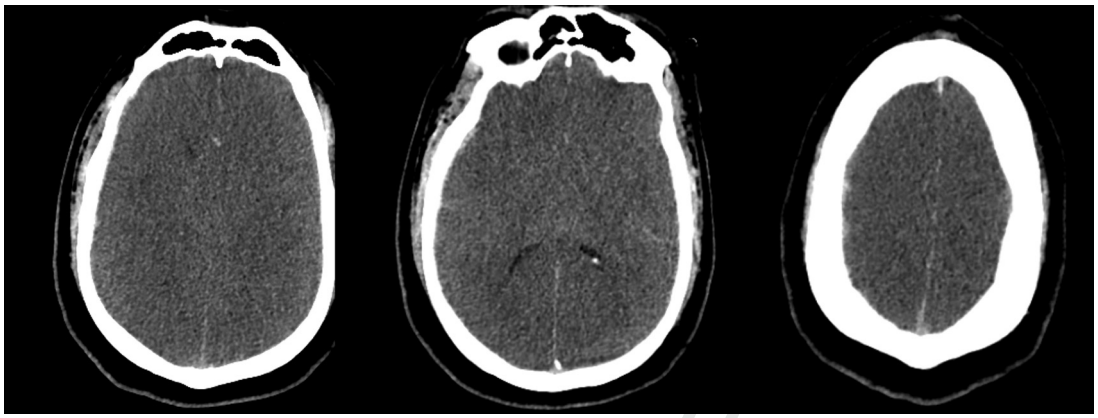

Fig. 1. CT image of patient who had come off their motorbike at high speed. Emergency services found him to be in cardiac arrest. Note, while there is clear hypoxic brain injury and swelling effacing the ventricles, there is little or no obvious parenchymal injury. Sometimes these CT scans are reported as DAI, but hypoxia may be of greater significance.

The association of apnoea and head injury is recognised as a post-mortem phenomena in more recent forensic literature.[22] More recently, the role of hypoxia in shaken baby syndrome has been studied. Geddes and Whitwell[23] challenged the accepted belief that the substrate of severe encephalopathy seen in post-mortem following "shaken baby syndrome" was diffuse axonal injury. They proposed that it was hypoxia and suggested that many conditions other than trauma could result in hypoxia.

Radiological manifestations of IBA

The authors experience in treating neurotrauma patients within contemporaneous trauma systems is similar. Patients with minimal injury on CT are sometimes labelled as having diffuse axonal injury (DAI), but careful inspection of the mechanism of injury and pre-hospital events might suggest otherwise (Fig. 1). Similarly, MRI sequences demonstrating microhaemorrhages are classically interpreted as DAI, however hypoxia can cause both cerebral and retinal haemorrhages,[24] again emphasising the need for correlation with the injury mechanism and subsequent clinical history.

Additional considerations in the aetiology and expression of Impact Brain Apnoea in humans

Alcohol

Alcohol may prolong the period of apnoea, a factor considered relevant in homicide cases although the importance outwith the legal assessment of death is limited.[22] Case reports in humans suggest that alcohol potentiates apnoea in neurotrauma.[25] Similarly, animal studies demonstrate impairment of respiratory control exacerbated by alcohol after TBI.[26]

Catecholamine surge

The dramatic rise in blood pressure occurring immediately after TBI has been purported to be a manifestation of Cushing's response to a rise in intracranial pressure (which Harvey Cushing[27] actually extended from Spence and Horsley's observations 9 years earlier [28]). In contrast Rosner et al. demonstrated that adrenaline levels increased 500 fold and noradrenaline levels 100 fold following TBI, a potential mechanism for hypertension which can persist until hypoxia results in complete cardiac collapse and shortly after, cardiac arrest. In survivors, catecholamine levels can remain elevated for several days.[29] This stress response is thought to account for hyperglycaemia, gastric mucosal ulceration, myocardial injury and neurogenic pulmonary oedema commonly seen after TBI.[5]

Miller (Edinburgh Professor of Surgery in 1842)[11] wrote in his "Practice of Surgery" that a man stunned by a blow or a fall is often "bled on the instant". This compounds the cardiovascular collapse that occurs after severe TBI. We remain concerned that 150 years on, we may still misinterpret respiratory and cardiovascular collapse post TBI as haemorrhagic shock rather than the real cause which is acute brain injury.

Hypotension following isolated TBI is a relatively common phenomenon, making up approximately 13% of hypotensive trauma patients.[30] However, standards of care such as the widely taught Advanced Trauma Life Support (ATLS) course state that "shock does not result from isolated brain injuries".[31] Such teaching may miss a treatable cause and/or delay meaningful interventions for IBA patients who may have a favourable prognosis.

Animal models of Impact Brain Apnoea

The phenomenon of apnoea following brain injury has been demonstrated consistently in multiple animals with multiple models of injury (Table 1).

Table 1 Animal models of head injury demonstrating apnoea following head injury. The phenomenon has been demonstrated across a range of mammals and injury mechanisms. In 1874, Koch and Filehne[16] demonstrated that repeated blows to a board applied to a dog's cranium resulted in apnoea with no apparent brain injury at post mortem. Four year later Duret[14] demonstrated the same with fluid injection into the cranium. Miles (1892), observed the physiological effects of striking the heads of multiple animals (rabbits, pigs, cocks) with a force "insufficient to cause death".[10,32] He noted irregularities and slowing of the heart and a reduced respiratory rate. He believed reflex vasospasm led to tetanus, paralysis and cardio-respiratory collapse (an "anaemia of the brain"), postulating the shock wave passing through CSF causing damage at the point of narrowing – the aqueduct and floor of the 4th ventricle.[10] Whilst there is little evidence for the mechanism he described, the phenomenon of apnoea following trauma is clear. Kramer gave the first clearly recorded example of impact brain apnea and concurrent hypertensive surge in a dog model of brain injury in 1896[33] (Fig. 2). Kramer states 'The so-called syncopic death after severe concussion is produced by a paralysis of the respiratory centres, the cardiac centres remaining intact.' And most importantly states that 'This fatal result may in many cases be prevented by the prompt institution of artificial respiration.' Signifying a potential mechanism in mammals to avert an otherwise inevitable death.

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Table 1
Animal models of head injury demonstrating apnoea following head injury. The phenomenon has been demonstrated across a range of mammals and injury mechanisms.

Author	Year	Animal	Injury mechanism	Summary
Koch and Filehne[12]	1874	Dog	Hammering on a board applied to cranium	Repeated blows to head → apnoea with no apparent brain injury
Duret[10]	1878	Dog	Sudden forceful injection of fluid into cranium	Causes slowing of respiration and intense bradycardia.
Miles[6]	1892	Rabbits, pigs, cock	Multiple experiments	All animals respond similarly
Polis[18]	1894	Cat, Dog, Rabbit	Concussive	Concussive injury → apnoea and blood pressure surge
Kramer[19]	1896	Dogs	Bullets against brass plate on skull and and weights	Concussion → apnoea that with artificial respiration is survivable. Blood pressure surge also noted.
Miller [20]	1927	Cats and dogs	Multiple experiments	Similar findings to Polis and that duration of respiratory cessation corresponded to intensity of blow.
Denny-Brown and Russell[21]	1940	Cats and Monkeys	Acceleration/ Deceleration	Demonstrated that injuries of low energy → respitaory gasp. Increasing emergency → increasing apnoea duration to a point of death. Occurs in decerbrate animals hence they attributed it to a brain stem phenomenon.
Walker et al. [22]	1944	Cats (151) Dogs (15) Monkeys (21)	Hammer Weight Drop Projectile	Respiratory response is energy dependent – low energy → gasp Higher energy → prolonged apnoea. Appears "brainstem" mediated.
Sullivan et al. [23]	1976	Cat (n = 36)	Fluid percussion	Between 1.5 to 2.2 atmosphere, apnoea occurred with no visible brain injury animals subjected to fluid percussion injuries exceeding the lethal threshold for apnea recovered normal function with respiratory support.
Carey et al. [24]	1989	Cats	Gun shot	Respiratory arrest is a constant feature, duration proportional to energy transfer. Early respiratory support saves life.
Kim et al.[25] and Levasseur et al. [26]	1989	Rats	Fluid Percussion	Ethanol increases duration of apnoea.
Adelson	1996	Rats	Weight drop	Ventilatory support peri-trauma eliminated mortality
Atkinson et al	1998	Rats	Fluid percussion	Respiratory apnoea duration is proportional to energy transfer
Rafaels[27]	2011	Rabbit	Blast Injury	Greater blast intensity was associated with longer post-blast apnoea
Lusardi et al. [28]	2011	Rat	Lateral Fluid Percussion injury	Intraperitoneal caffeine prevents impact brain apnoea
Rafaels[29]	2012	Ferret (mammal with gyrencephalic brain)	Blast injury	First mammal with gyrencephalic brain demonstration of blast causing apnoea of increasing duration with increasing blast.

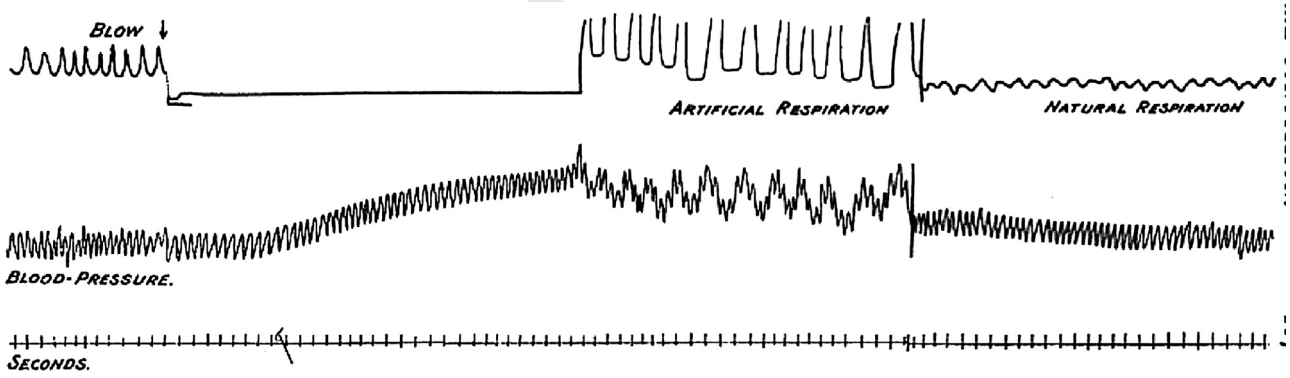

Fig. 2. Respiratory and blood pressure response following the blow of a 22-calibre shot striking a brass plate attached to the side of a dog's head. 55 s of apnoea was broken by artificial respiration (15 breaths) before slow (irregular) spontaneous ventilation took over. The animal survived, but was then sacrificed to reveal a skull fracture and small subdural with venous engorgement of pia mater, but the brain was essentially normal.[33]

In 1940, Denny-Brown and Russell described a correlation between impact force and the duration of apnoea. They also identified that concussion is more easily induced if the head is free to move when struck. They asserted that rotational shearing forces rather than "vibrations" were specifically damaging, dividing concussion into acceleration or compression concussion. Acceleration concussion, (occurring with rotationary head movement) produced a transient paralysis occurring beyond a change in head velocity between 0 to 28 ft s^{-1}. They considered this to be related to brainstem malfunction. Interestingly neither force nor energy transmitted were key factors in this rotationary form of concussion, which may also be a mechanism of the "knock-out-punch" in boxing. With acceleration concussion they noted an initial rise in blood pressure (with stimulation of the vasomotor centre) followed by a bradycardia and vagally induced fall in blood pressure "comparable to acute surgical shock".[34] They attribute death to failure of the veno-pressor system. No macroscopic or microscopic lesions within brain parenchyma were found. Basic brain stem functions, e.g. swallowing and corneal reflexes are abolished for 1–2 min post impact. Of note, boxing gloves appear to reduce linear acceleration,

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but have less effect on rotational acceleration.[35] In contrast, they believed compressive concussion had a more selective incidence on the respiratory centre.

Duret described a tetanic stage of concussion when an animal is struck on the head with a blow of sufficient force to cause immediate unconsciousness.[36] With this, altered respiratory patterns or apnoea are common in a manner suggestive of a more modern description of IBA.

In 1944 Walker[37] reported IBA in a study of 151 cats, 15 dogs and 21 monkeys concluding that impact resulted in a sudden supratentorial electrical discharge that then extinguished over 20 s. The EEG returned to normal at about 3 min. Walker noted respiratory cessation and a sudden surge in blood pressure (if the cord was intact) and a reflex bradycardia (if the vagus was intact). These phenomena occurred within one cardiac cycle of the blow and hence Walker believed these were direct cerebral cortex and brainstem effects.

Studies using more modern mechanisms of injury have continued to demonstrate the phenomenon of impact brain apnoea. These include mechanisms of weight drop in rats where increasing weights result in increasing apnoea duration (when over 2 min this results in death).[38] The authors suggested that this model specifically reflects what happens in childhood TBI. Approximately, 44% of childhood TBI is associated with diffuse cerebral swelling (loss of CSF spaces) and may well reflect hypoxic injury rather than direct parenchymal injury. Blast injury models have demonstrated in the ferret (a mammal with a gyrencephalic brain) that increased blast intensity increases apnoea duration[39,40,41]. Similarly, the fluid percussion injury model demonstrates apnoea duration correlation with magnitude of energy delivered to the brain.[42] Lusardi et al. additionally demonstrated that intraperitoneal injection of 25 mg kg^{-1} caffeine within 1 min of injury completely reversed impact brain apnoea and reduced epilepticorm activity 4 weeks post-injury.[43] Whether this is a practical treatment in comparison to supporting and mechanically ventilating a patient is questionable.

Case examples of Impact Brain Apnoea in the modern era

Historical and animal models clearly describe cardiorespiratory effects of TBI in the minutes following head injury. However, contemporaneous experience by clinicians working in PHEM also describes the phenomena within modern trauma care. The following examples from the authors describe the mechanisms and clinical findings within the critical phase of head injury.

Case 1: An urban example

A 70 year old, physically active lady was witnessed to be hit by a bus with her head striking the windscreen.

Management: An experienced physician witnessed the event, was on scene immediately and noted that she was not breathing. She performed mouth-to-mouth resuscitation for 3 min following which breathing started. Following this, a jaw thrust was required to maintain her airway. On arrival of the first paramedic crew her breathing was described as erratic and required assistance with bag-valve-mask ventilation. On arrival of the air ambulance physician, she had a normal breathing pattern, was swallowing her own secretions, had a wound to the occipital region but was unresponsive to painful stimuli. She was moved to the ambulance with oxygen therapy after no other injuries were found. In the ambulance en route to hospital she started to move non-purposefully, and 15 min later was able to obey commands.

Outcome: Head Computerised Tomography (CT) demonstrated a small frontal contusion. 2 h later she was fully orientated and communicating normally in the emergency department and was discharged from hospital the next day.

Case 2: A motor racing example

At the start of a superbike (1000cc) race, Rider A stalled. Rider B did not see Rider A was stationary and collided from behind at full race speed (100mph). The race doctor witnessed the incident and found rider B to be supine, apnoeic and appeared lifeless (Fig. 2). A rapid and controlled helmet removal allowed access to the airway and ventilation was commenced using a bag-valve-mask. The presence of a strong pulse was confirmed. The patient's pupils were dilated and sluggishly reactive. No significant injury was identified other than a presumed closed head injury (Fig. 3.)

Management Bag-valve-mask ventilation was continued whilst preparations were made for Rapid Sequence Induction (RSI) and packaging. However, 6 min after time of wounding, Rider B began to make spontaneous respiratory effort. By 8 min Rider B began cerebrating; which rapidly progressed to him becoming combative with a best GCS of 12/15 (E3, V4, M5). Pupils were, by this stage, mid-sized and reactive.

His clinical state mandated rapid sequence induction and intubation for transport, and Rider B was subsequently packaged and delivered directly to a Major Trauma Centre.

Outcome Initial CT PanScan revealed minor cerebral petechial haemorrhages with no other significant injuries of note. Rider B was ventilated overnight in the Critical Care Unit. Repeat CT Head the following day was unchanged, and he was successfully weaned from sedation and extubated. Rider B made excellent progress and was discharged from hospital later that week. He has successfully returned to work and, indeed, motorcycle racing.

These cases demonstrate IBA as a clinical entity that appears to indicate life threatening injury, but which resolves with simple and relatively non-invasive respiratory intervention, thus supporting Kramer's assertion that the phenomena is reversible in humans as well as in animals.[33]

Review of clinical experience

The incidence of IBA is difficult to establish, as it is relatively rare for a pre-hospital care providers to attend a patient within the first couple of minutes of injury. Recently, Menzies et al. presented nine cases of IBA at motorcycle events in Ireland (London Trauma Conference, 2015). We established an online survey to gauge if others (in addition to the authors) had witnessed IBA and to establish the mechanisms of injury with which it was associated. The results of this can be found in the appendix.

Outcome from IBA

The prognostic factors associated with a positive outcome from Impact Brain Apnoea are unknown although the period of apnoea associated with degree of force is likely to be significant.[42] Ultimately for those patients with prolonged apnoea and no intervention death will ensue.

Prevention of IBA associated mortality/morbidity

Animal models and anecdotal experience demonstrates that early ventilation through the period of apnoea results in survival. This was demonstrated by Kramer (1876), suggested clinically by Levine[44] and is manifest in the clinical cases described in this paper.

The logistical problem is delivering ventilator support in the minutes after injury and in most cases prior to the arrival of the emergency services. Increased awareness of the phenomenon as part of first aid training, emphasising airway and rescue breathing may encourage bystanders to recognise and manage IBA in the first few minutes prior to ambulance arrival. The use of new technologies that through Smartphone Apps alerts medically trained

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Fig. 3. Motorcyclist lying apnoeic following a collision (with permission).

personnel within a few hundred metres may also enable earlier intervention and prevention of hypoxia.[45]

We are moving into a world where the detection, altering and intervention for life threatening injury or illness through portable, even wearable technology is a real possibility. Automated sensors in phones and cars to detect sudden decelerations and altered physiology allow for the automated alerting of emergency services, bystanders and first aiders with supporting technology to guide meaningful hyper-early interventions. Since this is the period in which traumatic death commonly occurs, such technologies may provide a significant shift in preventable trauma death.

Future research

Research in traumatic brain injury has previously focused on hospital-based care. However, no pharmacological trials have demonstrated any survival benefit once patients reach ITU. We must ensure that research funding and efforts are focused on all phases of the patient's journey including the critical early phase of brain injury that takes place minutes after trauma. New and emerging technologies may finally give us access into those precious few minutes. It is up to the resuscitation community to find ways to penetrate and intervene in this critical and life changing time frame.

Conclusions

Impact Brain Apnoea is a phenomenon seen in animals and reported in humans following traumatic brain injury. The duration of the apnoea reflects the energy transfer in the injury. Prolonged apnoea can cause death however, ventilatory intervention can carry those injured through the period of apnoea. Concurrent with the apnoea is a catecholamine surge that can result in cardiovascular collapse that can be misinterpreted as hypovolaemia. Greater awareness of the phenomenon of IBA and the ability to empower bystanders and those in the immediate vicinity to maintain airway and provide rescue breaths as appropriate may help reduce this form of secondary brain injury.

Conflicts of Interest

MW is an unpaid director of a social enterprise (GoodSAM) alerting trained first aiders to nearby life threatening emergencies

No other conflicts declared.

Appendix A.

As part of this review we sought to investigate the experience of IBA amongst the pre-hospital care community. Through social media, we asked PHEM clinicians, paramedics and the pre-hospital community in general to complete an online survey. There were 104 responses (including 55 paramedics, 21 Pre-hospital/Emergency doctors, 21 First Aiders). Of this group 28 said they had never seen IBA, 23 said they had seen it once, 20 had seen it twice and 29 had seen it more than twice. The commonest mechanism of injury it was seen in was road traffic collision (motorcyclist (17), pedestrians (15), cyclists (8)) followed by falls (23) and assaults (4) being relatively infrequent. This survey is open to considerable selection bias (it could be assumed that those who had seen IBA would be more likely to respond), however it does demonstrate that the phenomenon has been witnessed multiple times and is probably under-reported.

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