Review

Improving outcome in severe trauma: trauma systems and initial management—intubation, ventilation and resuscitation

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ABSTRACT
Severe trauma is an increasing global problem mainly affecting fit and healthy younger adults. Improvements in the entire pathway of trauma care have led to improvements in outcome. Development of a regional trauma system based around a trauma centre is associated with a 15–50% reduction in mortality. Trauma teams led by senior doctors provide better care. Although intuitively advantageous, the involvement of doctors in the pre-hospital care of trauma patients currently lacks clear evidence of benefit. Poor airway management is consistently identified as a cause of avoidable morbidity and mortality. Rapid sequence induction/intubation is frequently indicated but the ideal drugs have yet to be identified. The benefits of cricoid pressure are not clear cut. Dogmas in the management of pneumothoraces have been challenged: chest x-ray has a role in the diagnosis of tension pneumothoraces, needle aspiration may be ineffective, and small pneumothoraces can be managed conservatively. Identification of significant haemorrhage is not easily definable. A hypotensive approach may limit further bleeding but could worsen significant brain injury. The ideal initial resuscitation fluid remains controversial. Appropriately selected patients early aggressive blood product resuscitation is beneficial. Hypothermia can exacerbate bleeding and the benefit in traumatic brain injury is not adequately studied for firm recommendations.

INTRODUCTION
Severe trauma is arbitrarily defined as trauma scoring >15 on the injury severity score (ISS)—an anatomically based scoring system derived from the most significant injury sustained by the three most seriously affected body regions. It is applied retrospectively and so is little use in the resuscitation room. Every day 300 000 people are severely injured and 10 000 die,1 making trauma an increasing global problem second only to HIV/AIDS in the 5–45-year-old age group.4–7 As the victims are mainly fit and healthy younger adults the disease places a significant socioeconomic burden on society.

In the UK there are around 17 0004 traumatic deaths annually, but severe trauma makes up <0.01% of the overall emergency department (ED) workload. It requires considerable resource together with decision making and practical procedures in areas in which many doctors, especially trainees, lack experience.

The way we treat trauma patients continues to evolve, but keeping abreast of the relevant multidisciplinary literature is difficult. We aim to summarise recent advances in the initial phase of trauma care, highlighting areas where new knowledge challenges previous ED management. We will also discuss trauma systems. The article is accompanied by a case to illustrate key learning points. We suggest that the article is read and then the case worked through.

ORGANISATION OF REGIONAL TRAUMA CARE SYSTEMS
The rarity of severe trauma means that the average district general hospital in the UK seeing 60 000 patients/year can expect to see less than 60 cases/year. Developing and maintaining expertise in the management of a rare condition is challenging. Furthermore, a multiply injured person may require input from a range of specialties often including those located in tertiary centres (eg, neurosurgery, cardiothoracic). Specialist care has been demonstrated to improve outcome: patients with severe traumatic brain injury cared for by non-neurosurgical centres have a 26% greater mortality.5

A regional trauma system aims to match the patient’s needs to the receiving hospital. Within such a system ambulances will, when the patient’s clinical condition permits, bypass smaller hospitals and deliver the most severely injured patients to larger major trauma centres (MTC). An MTC “has all the surgical specialties and support services to provide care for major trauma regardless of their pattern of injury.”5 There is a clear association between increased volume and improved outcome, so MTCs should see at least 400 major trauma patients/year.6 It is important to recognise that such a system involves much more than just the individual trauma centre but incorporates public health, injury prevention, emergency medical services, all trauma receiving hospitals, rehabilitation services, research, education, and clinical governance. Implementation of a trauma system is associated with a 15–50% reduction in mortality7–9 and a reduction in preventable deaths to close to zero.6 Mortality of patients transported to an appropriate centre is reduced from 19% to 12%.10 Such regionalised systems are, however, not widely distributed.11

PRE-HOSPITAL CARE
Beyond simply delivering patients to the appropriate facility, the care provided before reaching hospital has the potential to alter outcome. Involvement of doctors in the provision of prehospital care of trauma is an evolving field. There is
not yet clear evidence of benefit and further research is needed. The main advantages of involving a doctor in some cases of (severe) trauma are their expanded procedural skill and therapeutic base combined with a greater disease specific experience. Examples include trapped patients requiring procedural sedation for extrication, patients requiring limb manipulation, and patients in cardiac arrest from cardiac wounds who may make a good (neurologically intact) recovery if on-scene thoracotomy is performed. Conversely, there is no clear evidence of benefit from pre-hospital intubation by paramedics.\textsuperscript{12–17}

**RECEPTION OF THE SEVERELY INJURED PATIENT: THE ROLE OF THE TRAUMA TEAM**

The complexity and time dependent injuries of severe trauma patients mandate a team approach to care. Trauma teams may be run on a single or two-phased model, with an in-house ED team for less severe trauma and a multidisciplinary team for major cases. The team should meet before the patient’s arrival, introduce themselves, pre-order blood and investigations as required, and ideally debrief cases together. Hospitals with a dedicated trauma team/system and high turnover may have an improved outcome, especially in the more severely injured patients, with an estimated 40% of deaths being preventable if delivered to expert care.\textsuperscript{18} Trauma teams led by senior staff are more likely to provide appropriate care.\textsuperscript{19}

**AIRWAY: EARLY MANAGEMENT IN SEVERE TRAUMA**

Poor airway management both pre-hospital and on arrival continues to be identified as an avoidable cause of morbidity and mortality.\textsuperscript{19, 20} A review of 741 cases in the UK identified inadequate airway management in 7%.\textsuperscript{19} Adequate oxygenation is vital for the prevention of secondary brain injury. Maintenance of a patent airway is critical. The use of nasal airways has been discouraged due to potential intracerebral placement, but the evidence for this exists only from case reports. They have been used at London HEMS (helicopter emergency medical service) for 20 years without incident.\textsuperscript{21}

The airway must be protected as well as patent. The best predictor of aspiration/hypventilation is reduced level of consciousness, usually measured using the Glasgow Coma Score (GCS). Expert opinion is that a GCS <9 suggests a high risk, but there is little trial evidence for this. Intubation may be necessary at a higher GCS to facilitate investigation. Cerebrally agitated trauma patients with a GCS of 13 or 14 have a 12.5% incidence of abnormal CTs requiring neurosurgical intervention.\textsuperscript{22} Intubation of patients with a GCS >9 may be necessary to manage the agitated patient, provide adequate ventilation (especially in chest trauma), allow procedures to be performed, and ensure a humane approach to care. Once a decision to intubate is made careful consideration must be given to the drugs used for rapid sequence induction (RSI) and the technique employed. In-line cervical immobilisation should be employed at all times.

**Drugs for rapid sequence intubation: induction agents**

Cardiovascular stability is a major concern during RSI in trauma patients. Consequently etomidate is still commonly used for induction of anaesthesia, despite suppressing cortisol synthesis. Its use both in patients with severe sepsis/septic shock and as a sedative infusion on intensive care is associated with excess mortality; however, the morbidity/mortality from a single dose for induction of anaesthesia in trauma patients is not known.\textsuperscript{23–26} Doubt about etomidate has helped stimulate increasing interest in ketamine. Widely used pre-hospital as an analgesic, use as an induction agent in trauma has been previously discouraged because of concerns about increasing intracranial pressure. There is, however, little evidence for this occurring in the acute trauma setting; indeed it may offer neuroprotective benefits.\textsuperscript{27} Thiopentone and propofol both decrease cerebral metabolic effect but frequently cause hypotension, which is associated with poor outcome in severe head injury. These agents should only be used by practitioners very familiar with their pharmacodynamics.\textsuperscript{28, 29} Whichever agent is chosen, careful dosing and judicious monitoring are essential safety measures.

**Drugs for rapid sequence intubation: muscle relaxants**

Provision of good intubating conditions in a short period of time is a fundamental determinant of the choice of muscle relaxant for RSI in trauma. Suxamethonium achieves these conditions in 45–60 s but can increase oxygen consumption, raise gastric and intraocular pressure, and lead to hyperkalaemia and malignant hyperthermia.\textsuperscript{30} Rocuronium (Esmoner, Organon) at doses of 1–1.2 mg/kg has a similarly rapid onset\textsuperscript{31} and has a better side effect profile. Practitioners have remained cautious of rocuronium due to its longer duration of action when compared to suxamethonium. However, it is questionable whether poly-trauma patients, with their frequent thoracic injuries and high metabolic rate, would resume spontaneous respiration in time to prevent hypoxic injury in the event of a failure to intubate and oxygenate within the 6–8 min of paralysis following a dose of suxamethonium. A novel reversal agent for rocuronium, sugammadex (Org 25969, Organon), which is capable of reversing paralysis in 3 min, is available.\textsuperscript{32} This may result in rocuronium becoming a more favoured choice.

**Cricoid pressure: risks and benefits**

Cricoid pressure (CP) was adopted as an integral part of RSI because of the high incidence of aspiration pneumonitis in obstetric anaesthetic practice.\textsuperscript{33} Its use has never been studied in a randomised trial, it may offer incomplete protection from aspiration, and it has a number of adverse effects such as potentially impairing bag mask ventilation (should this be required) and lowering the barrier pressure at the gastro-oesophageal junction.\textsuperscript{34} CP has a variable effect on the quality of the laryngeal view obtained—neutral, improving or worsening it.\textsuperscript{34} Unpublished observational data from London HEMS on pre-hospital trauma airways suggests removal of CP is associated with an improved laryngeal view in 50% of cases. If the larynx is not well visualised CP should be removed to facilitate intubation rather than risking hypoxia/hypercarbia by repeated intubation attempts. Further improvement in laryngeal view may be facilitated by manipulation of the larynx under direct vision (bimanual laryngoscopy) or backwards-upwards-rightwards pressure (BURP) applied to the thyroid cartilage.\textsuperscript{35}

**Adjuvant agents in rapid sequence intubation**

Laryngoscopy causes an increased intracranial pressure through sympathetic stimulation. In traumatic brain injury this may be harmful. A number of agents have been suggested to attenuate the rise in intracranial pressure. These include fentanyl 3 μg/kg and lignocaine 1.5 mg/kg delivered intravenously over 1 min 3 min before administration of the induction agent.\textsuperscript{36} Suxamethonium induced fasciculation may also increase intracranial pressure, although this is debatable with one small trial performed on ventilated patients on an intensive care unit showing no change in intracranial pressure post-suxamethonium.\textsuperscript{37} There is some evidence that using a small dose (one
tenth of the intubating dose) of a non-depolarising muscle relaxant, such as vecuronium or atracurium, attenuates this. However, there is no robust evidence to support any of these techniques.

**BREATHING: PNEUMOTHORACES AND VENTILATORY STRATEGY**

**Pneumothoraces: modifications of received wisdom**

Pneumothoraces are frequent and may expand to produce tension physiology: impaired ventilation from reduced air entry and hypotension from vena caval compression. This is traditionally treated by insertion of a large bore cannula into the second intercostal space in the midclavicular line. It is now recognised that this technique may fail due to inadequate cannula length (in around one third of patients), cannula blockage, displacement or kinking.

Cannula decompression is not without risk and is carried out far more commonly than the incidence of tension pneumothorax. Unless there are clear signs of tension physiology requiring emergent decompression, such as hypoxia (<92%) or hypotension (<90 mm Hg), a chest x-ray should be obtained before any intervention. If immediate intervention is required and a chest drain is not immediately available then either a thoracostomy may be cut (in ventilated patients) and the chest drain inserted later or a cannula placed as above. Medistinal shift is common in the absence of tension physiology and so cannot be relied on to make the diagnosis of tension pneumothorax.

Small and occult pneumothoraces (visible on chest CT, not chest radiograph) may not require treatment. Pneumothoraces behave very differently in spontaneously ventilating patients compared with mechanically ventilated patients. Rapid development of tension physiology is much more likely in the ventilated patient.

**Ventilation: lung protective strategies**

Recently there has been an increase in the understanding of the potential harm from ventilation. Large tidal volumes, inadequate positive end expiratory pressure (PEEP), and non-decelerating wave forms are now believed to give rise to alveolar damage—termed atelectrauma and volutrauma. Not only can this cause pulmonary damage but also cytokine release, which may contribute to the systemic inflammatory cascade and organ failure. A ventilatory strategy should be adopted with tidal volumes around 5–6 ml/kg and plateau airway pressures below 30 cm water. A minimum PEEP of 5 cm water should be applied and adjusted upwards to ensure adequate oxygenation, but this may precipitate hypotension in hypovolaemic patients. Any persistent hypoxia should prompt immediate search for a cause, such as interruption of the oxygen supply, incorrect position or blockage of the tracheal tube, pneumothorax, hypovolaemia or inadequate ventilation.

**CIRCULATION: RECOGNITION, ASSESSMENT AND RESUSCITATION**

Haemorrhage remains a leading cause of death following trauma. The first phase of resuscitation is to prevent further blood loss. Every effort should be made to minimise patient movement as this may disrupt clot formation. An early log roll or pelvic stress examination often add little useful clinical information but may promote haemorrhage. The risks and benefits of each action need careful consideration. Long bone fractures should be splinted, and if there is suspicion of significant bony pelvic injury splintage should be applied here too.

Military experience has revived the use of the tourniquet for exsanguinating limb haemorrhage that cannot be controlled by direct pressure. In such circumstances a decision of life versus limb may need to be made. Any points of external haemorrhage should be controlled by pressure.

Recognition of when a patient is bleeding can be difficult. The use of pulse, respiratory rate and blood pressure are neither sensitive nor specific for haemorrhagic shock and young adults may lose 30% of their circulating volume with little change in their vital signs. The use of 90 mm Hg as the determinant of shock is arbitrary; indeed patients with a systolic blood pressure (SBP) of 91–109 mm Hg have an increased mortality.

Other measures of tissue hypoperfusion should be sought, including altered mental status, reduced urine output, increased respiratory rate, metabolic acidosis, base deficit, and raised lactate. Early identification of patients with occult shock (also termed cryptogenic or compensated shock) may improve outcome.

Most cases of shock in trauma will be hypovolaemic; however, other aetologies such as obstructive (pericardial or pleural tension physiology), myocardial (usually blunt myocardial injury) or distributive (usually neurological) should be sought and treated. A normal ECG and troponin rule out significant myocardial contusion, while ultrasonography can rapidly assess for pneumothoraces or pericardial effusion.

**Resuscitation goals**

Fluid resuscitation was previously administered vigorously, aiming to replace estimated fluid losses and restore organ perfusion so minimising subsequent organ failure. However, this approach is associated with a transient rise in blood pressure that may remove clot and cause dilutional coagulopathy, dilutional anaemia, and cooling. In 1992 Bickell et al demonstrated a reduction in mortality in patients with truncal penetrating disease whose fluid resuscitation was delayed until definitive haemostasis had been achieved. The benefit applied to patients with vascular and cardiac injuries taken quickly to the operating room. This approach is widely advocated and has been applied to blunt trauma with little evidence of benefit in one (arguably underpowered) trial.

Resuscitation targets are controversial. Pre-hospital fluid resuscitation is no longer aimed at achieving ‘normal’ blood pressures. In blunt trauma 250 ml boluses of fluid are given to keep the radial pulse palpable; in penetrating disease central pulses are sufficient. Once in hospital for penetrating disease it is reasonable to aim at a systolic blood pressure associated with evidence of cerebration or 70–90 mm Hg, until haemostasis is achieved. It may be reasonable to aim for SBP around 90 mm Hg in blunt trauma with no associated traumatic brain injury for short periods if the patient is to be swiftly transferred to theatre or angiography for haemostasis. The trauma team should use judicious fluid resuscitation to prevent lower blood pressures than suggested, which risks subsequent organ failure and death.

The benefits of hypovolaemic resuscitation must be balanced against the risks of organ hypoperfusion. Resuscitation targets are adjusted to achieve organ perfusion if there is no rapidly identifiable surgically or angiographically correctable bleeding point. Initially this is to achieve a mean arterial pressure of over 65 mm Hg (adjusted to the patients age and comorbidities) and then to maximise cardiac output to match oxygen supply with demands.

For blunt trauma with head injury and ongoing bleeding requiring urgent intervention, controversy exists. The injured brain lacks autoregulation and thus relies on a higher perfusion pressure than under normal physiological conditions for...
adequate oxygenation. Currently a cerebral perfusion pressure (the difference between mean arterial pressure and intracerebral pressure) of over 65 mm Hg is advocated. A case-by-case decision should be made, balancing the requirements of cerebral oxygenation against the risk of worsening haemorrhage. In isolated head injury hypotension must not be tolerated: a mean arterial pressure of over 85–90 mm Hg or SBP of 100–110 mm Hg is a reasonable target in the ED before invasive monitoring is applied.

Once haemostasis has been achieved, resuscitation aims to restore tissue perfusion, of which there is no ideal measure. After pulse and blood pressure have been normalised, evidence of end organ hypoperfusion persists 40–80% of the time. This may be suggested by clinical assessment, low urine output, low cardiac output, central venous oxygen saturation <70%, raised lactate, and a persistently increased base deficit.

Resuscitation to supraphysiological goals is associated with harm, while tailored goal directed therapy (resuscitation to a cardiac output or stroke volume rather than blood pressure and pulse) commenced before organ failure is associated with improved mortality. A recent trauma resuscitation trial compared normalisation of pulse, blood pressure and central venous pressure to resuscitation until a maximum stroke volume was obtained (measured by oesophageal Doppler). The oesophageal Doppler group demonstrated a more rapid lactate clearance, fewer infectious complications, and a shorter hospital stay.

Resuscitation fluid

The initial resuscitation fluid of choice remains controversial. Hartmann’s solution avoids the acidosis of 0.9% saline but is slightly hypotonic, which may contribute to cerebral oedema and fluid loading. The SAFE trial suggested an advantage for saline over albumin for traumatic head injury resuscitation in subgroup analysis. Hypertonic saline has been advocated for rapid restoration of cerebral perfusion pressure and reduction of intracranial pressure, but a randomised trial (vs 0.9% saline) did not show benefit. An earlier trial of hypertonic saline in dextran had suggested a survival advantage (in patients with traumatic hypotension), but a recent pre-hospital trial was stopped early due to futility and early excess mortality. A pragmatic approach is to avoid colloids, as these are associated with coagulopathy, and use Hartmann’s for most trauma cases and 0.9% saline for patients with severe head injury.

In contrast to Bickell’s work, there is some evidence from the treatment of trauma victims in the Iraq war suggesting a benefit for ‘damage control’ resuscitation. This philosophy applies in the small subgroup with haemorrhagic shock and involves the use of packed red cells and fresh frozen plasma in near equivalent volumes as the initial resuscitation fluid.

TEMPERATURE: A ROLE FOR HYPOTHERMIA?

Traditional teaching favours early aggressive warming for trauma patients as hypothermia contributes to acidosis and coagulopathy and worsens microvascular blood flow. Normothermia should always be the aim in haemodynamically unstable trauma patients. While therapeutic hypothermia improves outcome post (medical) cardiac (ventricular fibrillation) arrest, its use in traumatic brain injury is controversial, with many trials not showing benefit and potential harm in patients over 45 years old. However, there is limited evidence that not aggressively warming haemodynamically stable head injured trauma patients with mild (32–34°C) hypothermia on arrival in the ED may be advantageous and that cooling may be of benefit in a subgroup of severely head injured patients with high intracranial pressure. The risks and benefits of cooling in traumatic brain injury are closely matched. Induced hypothermia may one day have a role in penetrating injury induced cardiac arrest.

SUMMARY

Optimal management of major trauma requires close attention to detail at all levels of organisation—from the structure of the healthcare system in large geographical regions to the functioning of individual members of the trauma team as they manage an individual patient. Major trauma centres, where patients from a wide area are received by a team led by senior clinicians, improve care but are not yet widely adopted. More worryingly there is evidence that fundamental aspects of care, such as airway management, are still too often inadequate. Within the resuscitation room the traditional ABC approach still rules, but some long accepted dogmas have been challenged. Numerous aspects of initial management and resuscitation, both

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**Main messages**

- Globally trauma morbidity/mortality is increasing and is second only to HIV-AIDS as a cause of death in the 5–45 year age group.
- Regional designated trauma centres and dedicated hospital trauma teams with trauma expertise improve outcome.
- Preventable deaths are still common in UK trauma care, even in basic resuscitation areas such as airway management.
- Circulating volume should be preserved by minimal handling and judicious fracture splintage.
- Vigorous early fluid resuscitation may be harmful. Blood pressure resuscitation targets are controversial. If a strategy of relative hypotension is to be successful, rapid and aggressive surgical and/or radiological haemostasis should be followed by restoration of adequate tissue perfusion.

**Current research questions**

- What procedures have benefit in the pre-hospital arena? Does early intubation by experienced practitioners improve outcome in severe trauma?
- Is etomidate associated with a mortality or morbidity increase in trauma patients? Does the choice of induction agent effect morbidity/mortality in severe trauma?
- What are the best physiological goals against which to resuscitate trauma patients in the emergency department and later in the intensive care unit/operating theatre? Should we apply the principles of early hypotensive resuscitation to blunt trauma?
- In which patients should we use blood product based resuscitation? What blood products should we use? In patients not requiring blood products, what resuscitation fluid is best?
- In patients with isolated severe traumatic brain injury, is the use of early mild therapeutic hypothermia of benefit?
Clinical scenario

A young man is being transported to your emergency department. His car has hit a tree at high speed. The paramedic calls ahead with the following information:
- Airway initially obstructed but relieved with the placement of a Guedel airway
- Observations: respiratory rate 25, oxygen saturations 95% on 15 litres oxygen via a non-rebreathing mask, systolic blood pressure 105 mm Hg, heart rate 100 beats/min (bpm)
- External evidence of head injury with a Glasgow Coma Score of 6 (withdrawing all limbs to painful stimuli, but no verbal or eye response)
- Clinical suspicion of pelvic and left femoral fractures
- Two large bore cannulae in situ and femoral/pelvic splints in place

Pre-hospital and on arrival, require further research, but best practice continues to evolve.

QUESTION 1
What preparations would you like to make following the ‘blue call’ and before the arrival of the patient?

ANSWER
The trauma team is activated, led by the duty ED consultant. The duty trauma surgeon, anaesthetic co-coordinator and theatre staff are notified. Each member of the team has a pre-defined role. It includes critical care trained nurses, an operating department practitioner, and a radiographer. Equipment including cannulae, fluids, rapid fluid infuser, an ultrasound machine, warming equipment, blood slips and x-ray requests are prepared. The resuscitation room supply of O negative/O positive blood is checked. Space is created in a dedicated trauma resuscitation bay.

CLINICAL COURSE
The patient arrives and a structured handover is given to the trauma team. There has been no significant change from the information already provided.

QUESTION 2
How would you manage this case once he has arrived in the ED? What are the initial treatment priorities and how will you achieve them?

ANSWER
This patient requires intubation for his head injury, partially obstructed airway, his expected clinical course, and to facilitate humane care. This is performed by rapid sequence (induction) intubation with an assistant to immobilise the cervical spine. There is no ideal agent for this, save one with which the intu- bator is confident and experienced. Paralysis is induced with suxamethonium or rocuronium. If the laryngeal view is poor it may be improved by removal of cricoid pressure and use of laryngeal manipulation. Hypoxia and hypotension are to be avoided as they worsen outcome. Fentanyl (3 µg/kg delivered slowly 3 min before intubation) and/or lignocaine may be used to blunt the increase in intracranial pressure from laryngoscopy, but there is no robust evidence for this.

QUESTION 3
How will you ventilate this patient?

ANSWER
Your patient is ventilated at 6 ml/kg (lean body weight) with 5 cm water PEEP on 100% oxygen. The ventilation rate is adjusted to an end tidal carbon dioxide of 4–4.5 kilopascals (kPa) (and more precisely an arterial partial pressure of carbon dioxide of 4.5 kPa). Sedation is maintained, preferably with short acting agents such as propofol, and analgesia provided using agents unlikely to induce hypotension such as fentanyl.

CLINICAL COURSE
Post-intubation the blood pressure is 96/55 mm Hg, pulse is 98 beats/min (bpm), and oxygen saturation is 96%. A bedside extended focused assessment with sonography for trauma (eFAST) is performed at the end of the primary survey and shows left sided haemopneumothorax. There is no evidence of intrapericardial or intraperitoneal fluid.

QUESTION 4
What action would you like to take following this information?

ANSWER
The eFAST demonstrated a haemopneumothorax, which was suggested by low saturations post-intubation while ventilating with 100% oxygen. A team member is tasked to gown, glove and prep the chest wall. At any sign of further deterioration an immediate thoracostomy would be performed. A chest film should immediately be obtained to confirm the diagnosis while the team members prepare themselves and their equipment.

The chest film may be performed during the primary survey. The team are protected against radiation.

CLINICAL COURSE
The chest film confirms a left sided pneumothorax.

A chest drain is successfully inserted with full aseptic technique and antibiotic cover into the fifth intercostal space, just anterior to the mid-axillary line.

The vital signs are now: oxygen saturation 99%, end tidal carbon dioxide 4.2 kPa, blood pressure 102/50 mm Hg, pulse 94 bpm.

QUESTION 5
What investigations will you request now?

ANSWER
An arterial blood gas, post procedure chest x-ray, and pelvic x-ray. (Arguably if the chest drain is working well, there is no physiological deterioration, the patient is ventilating at normal pressures—tidal volume, and the CT is within the department, then an immediate CT may be a better use of time)

CLINICAL COURSE
The patient remains ventilated on 100% oxygen with a tidal volume of 6 ml/kg and a PEEP of 5 cm water, to maintain an arterial partial pressure of carbon dioxide of 4.5 kPa. Sedation is
continued with propofol and fentanyl. An arterial gas shows pH 7.31, a partial pressure oxygen of 29 kPa, partial pressure of carbon dioxide of 4.6 kPa, bicarbonate of 17 mmol/l, base excess of −5, and a lactate 3.3 mmol/l. The chest drain is functioning well and in good position on repeat chest x-ray with 280 ml blood loss in 15 min. The blood pressure varies between 96–108/66–72 mm Hg and the pulse varies between 90–106 bpm. A pelvic x-ray shows fractured pubic rami with no vertical or horizontal shift.

**QUESTION 6**

While the trauma series is being completed what else would you like to do to facilitate excellent patient care? What do you think of the physiological evidence before you? What further investigations are required?

**ANSWER**

You check the pelvic splint and ensure it is in a good position over the greater trochanters. The pelvic splint is left in situ and the pelvis is not clinically assessed for instability at this early stage, as doing so may open any fractures and disturb blood clot leading to further bleeding. A femoral splint, which does not splint against the pelvis, is applied with minimal patient movement and a femoral nerve block applied. No log roll is performed as it is thought this will add little to the further evaluation of the patient while it risks fracture movement and clot disruption.

The team have noted several sources of blood loss, tachycardia, intermittent hypotension (for a trauma patient) and biochemical evidence of tissue hypoperfusion. Judicious fluid resuscitation with crystalloid bolus are used to achieve a SBP around 110 mm Hg and mean arterial pressure of 85–90 mm Hg, given the severe brain injury and no immediate requirement for surgery or angiography. A total of 500 ml is administered.

This patient will require further immediate imaging with a contrast CT of the head, neck, chest, abdomen and pelvis to define the injury complex more completely. No lateral cervical film is taken in view of definitive imaging of the cervical spine with CT. The team should accompany the patient to CT to ensure adequate available resources and maintain effective lines of communication.

The team leader ensures that each team member records his or her contribution to patient care in the patient’s trauma medical record.

The patient is warmed, given the clear clinical evidence of shock. Further resuscitation is dictated by the findings on imaging and clinical course.

**Competing interests** None.

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