

OPERATIONAL CRITICAL CARE. INTENSIVE CARE AND TRAUMA

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Abstract

Trauma management involves good prehospital, emergency, surgical, anaesthetic and intensive care decision-making. Optimal outcome depends on keeping abreast of the latest thinking in an ever-changing and increasingly technology-rich environment. The intensive care unit needs to be represented as early as possible in the damage-control resuscitation phase.

Improved trauma system care has resulted in an increasing number of multiply injured military patients surviving their initial trauma. These patients require intensive care and are at risk from sepsis and multiple organ failure. Attention to detail is important, preservation of organ function, infection control and nutrition to maintain muscle strength allowing normal metabolic function to return.

Multiply injured patients often require lengthy periods of mechanical ventilation and a variety of therapeutic interventions may have to be considered during management of the disease process.

As we are now seeing more survivors in the military trauma system the focus now needs to be morbidity reduction in order for these survivors to be best prepared for their rehabilitation phase of care.

Introduction

Military patients requiring intensive care for severe trauma often pose particular challenges. The treatment of these patients can be protracted (commonly referred to as the prolonged-care phase) and the initial injury may become of secondary importance to the effects of systemic inflammatory response syndrome, acute lung injury (ALI), nosocomial infection and inter-current multi-organ dysfunction syndrome (MODS). Multiply injured patients often require lengthy periods of mechanical ventilation and a variety of therapeutic interventions may have to be considered as the disease process progresses. In practical terms trauma patients requiring intensive care fall into two groups: firstly there are those requiring this level of support close to their time of admission to hospital. These include thoracic injuries, major head injuries and patients with circulatory shock as well as those who require an extended recovery period following resuscitative surgery. A second group is made up of those patients who are suffering from the later complications of trauma, systemic inflammatory response syndrome (SIRS), sepsis, ALI and MODS. The first group are dealt with in theatre and if patients fall into the second group they should be having their care delivered at a Role 4 facility in the UK [1].

Despite significant advances in management over the last 20 years, trauma remains the chief cause of death and disability in individuals under 40 years of age. Trauma is a global disease with both military and civilian victims with immense public health implications and numerous challenges; including the need for an increased emphasis on accident prevention and rehabilitation, particularly in the developing world [2]. The Essential Trauma Care Project has defined and promoted core essential trauma care services that every injured person in the world should be able to receive. This project is a collaborative effort of the World Health Organization and the International Society of Surgery [3]. Box 1 outlines some of the issues raised in the intensive care management of trauma patients.

- 1). Consideration of the injury mechanisms and potential structures at risk in blunt and penetrating trauma.
- 2). Pre-hospital management and the time line to definitive care.
- 3). Pre-operative management of the trauma patient including indications for intubation and approaches to fluid management.
- 4). Clearance of the cervical spine in a sedated and ventilated trauma patient.
- 5). Timing of fracture fixation and damage control surgery.
- 6). Ventilation strategies and analgesia, in patients with multiple injuries.
- 7). Use of inotropic support for maintenance of blood pressure.
- 8). Timing of Aeromedical evacuation to level 4 care.
- 9). Multiple organ failure and sepsis.
- 10). Rehabilitation from injury.

Box 1. Issues raised in the intensive care management of trauma patients.



Figure 1 Field ICU bed with associated equipment.

Case history

An 35-year-old male driver of a military vehicle was caught in a roadside IED. He lost control at speed and the vehicle rolled at approximately 50mph. He was given buddy aid at the scene and attended by a MERT team. He was hypoxic and combative with a Glasgow Coma Score of 13 and oxygen saturation of 90% on pulse oximetry (SpO₂). He had obvious blunt chest injuries bilaterally there was clinical evidence of an open femoral fracture on the right and a partially amputated left lower limb.

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He was intubated *en route* to the field hospital. A femoral traction splint was applied on the right and a tourniquet *in-situ* on the left leg. He had bilateral decompressions of the thoracic cavity. On admission to the field hospital to he was noted to have equal pupillary reactions with an obvious scalp injury. He had severe bruising over his chest wall anteriorly on both sides and a centrally mobile flail segment. He had mild inspiratory noise on auscultation on the right and an intercostal drain was sited through the thoracostomy incision. Intercostal drains were sited bilaterally. His abdomen was soft and he had a normal FAST scan (Focused Abdominal Ultra-Sound Scan for Trauma) in the emergency department. There was no evidence of pelvic injury. He had received 700mls of crystalloid pre-hospital and his mean arterial pressure on admission was 80mmHg. He had impaired gas exchange, the fractional inspired oxygen concentration (FiO₂) was 0.5 with a pH 7.29 PaO₂ 9.6 kPa PaCO₂ 5.6 kPa BE -5.1 SaO₂ 94%.

Initial cervical spine, and pelvic radiographs were unremarkable. His chest x-ray revealed fractured ribs 3-7 anteriorly on the left and 5/6 on the right and an area of contusion in the right mid-zone. He was taken for a CT scan of the head and neck (including sagittal reconstruction views of the cervical spine); which were reported as normal. In view of the impairment of gas exchange and evidence of contusion on CXR, helical CT scanning of the chest and abdomen was performed at this time. He was noted to have a contained rupture of the spleen and contusions in both frontal lobes on his CT head. After discussion with the surgical team he was transferred to the operating theatre and had external fixation of his femoral fracture by a consultant orthopaedic surgeon and completion of his left lower limb amputation. He was subsequently transferred to the intensive care unit. (FiO₂ 0.6, pH 7.30 PaO₂ 9.4 PaCO₂ 6.8 BE - 6.0 Temp 34.5°C). He was actively warmed and his acidosis corrected. His sedation was reduced and he responded appropriately to command. However, his condition subsequently deteriorated and his oxygen requirements increased. He required inotropic support for maintenance of blood pressure and vital organ perfusion. He was transferred by a CCAST team to a role 4 facility in the UK at 48 hours. Intra-cranial pressure monitoring was instituted and he was found to have normal measurements. On day 5 of intensive care he required an emergency laparotomy due to obvious bleeding and a splenectomy was performed. Subsequent recovery was hampered by the development of ventilator-associated pneumonia (VAP). Protective ventilation strategies were employed over the next few days but adult respiratory distress syndrome (ARDS) developed. He responded to intermittent prone-ventilation. He had a percutaneous tracheostomy performed at day ten and was eventually weaned from ventilation at day eighteen. He was transferred to the high-dependency unit (HDU) on day twenty-two after admission.

Initial management

In the absence of catastrophic external haemorrhage trauma management begins with airway, breathing and circulation; ideally the Intensive Care Unit should be represented within the trauma team. This is usually an anaesthetist whose role goes beyond assessment of the airway. If the patient is awake somebody needs to take a full history, including when they last ate or drank, allergies, medications and any other comorbidities.

If the patient requires urgent intubation and ventilation, cervical spine immobilisation must be maintained during intubation in all trauma patients at risk of cervical spine injury. The cervical hard collar may need removal to allow adequate mouth opening but a suitable assistant must apply manual in-line immobilisation. The ICU doctor needs to co-ordinate patient transfer in conjunction with the trauma team leader, whether to

the CT scanner, operating theatre or intensive care unit.

A nominated scribe must be advised of all therapeutic interventions undertaken and encouraged to document vital signs, blood results and other relevant information.

Discussion of the need for intensive or high dependency care after resuscitation must be done earlier rather than later and the necessary communication made with both the trauma team leader, operating theatre team and ICU staff.

Acute fluid resuscitation

Volume replacement is the accepted method for treating hypovolaemia but it can also cause significant compromise of other organ systems. Continuous infusions of even blood or normotonic fluids cause significant peripheral tissue oedema, contribute to increases in extra-vascular lung water with impairment of both gas exchange and cardiac function. Traditionally multi-trauma patients have been given aggressive fluid resuscitation and blood pressure support to maintain vital organ perfusion. There is now a clear view that fluid resuscitation should be limited until haemostasis is achieved, delays in the emergency department when surgical intervention to stop bleeding is required are a cause of increased mortality [4]. Most experimental studies have looked at models of pure vascular bleeding and reliable clinical studies are few; the only prospective randomised ones have been done in penetrating trauma. In the case of head injuries however, a low systemic arterial blood pressure (SABP) relates to a poor outcome; the patient with an altered mental status with multiple injuries (that are bleeding) presents a dilemma. Aggressive fluid administration exacerbates bleeding from an occult injury or inadequately splinted femur but blood pressure support in terms of maintaining cerebral perfusion would be considered to be a key therapeutic intervention [5]. The current consensus view in prehospital care is that fluid should not be administered to the trauma victims prior to haemorrhage control if a radial pulse can be felt. Boluses of 250ml should be titrated for other patients. If the radial pulse returns, fluid resuscitation can be suspended and the situation monitored. In penetrating injury, the presence of a central pulse should be considered adequate [6]. It is recognised that head-injured patients are a special group and may require higher blood pressure to maintain cerebral perfusion. (Systolic BP > 100mmHg) The continued application of this protocol in the hospital setting is still not established [7]. The use of artificial colloids is not without risk, due to the pathophysiological processes (capillary leak and inflammation) involved in the response to tissue trauma [8]. The use of lower-volumes of resuscitation fluid would seem sensible in the case of chest trauma in particular and the use of hypertonic saline may have some place; although it has certainly not gained universal acceptance [9]. It should be remembered that the presence of non-responsive shock in blunt trauma, may be due to direct myocardial injury. Transoesophageal echocardiography (TOE), if available is an effective tool in the assessment of cardiac trauma in conjunction with ECG and cardiac enzyme measurements. It can also be used in the early assessment of aortic injuries although high index of suspicion must be retained, even in the face of normal TOE studies [10]. In the absence of a TOE but with a high index of suspicion (mediastinal widening on plain x-ray remains the most reliable sign), it is recommended that the patient should have a contrast-enhanced spiral CT of the thorax as part of the initial management [11].

Regardless of the treatment or monitoring methods used, the goal of resuscitation is to fully restore homeostasis and prevent end organ damage. Current resuscitation approaches, of which there are a number, support the theory that hypoperfusion results in metabolic acidosis. The initial resuscitation and management priorities in the emergency department are different from those in

the operating theatre (or ICU) but the ultimate endpoints of resuscitation are the same. Fluid requirements, or their absence serve as an imprecise endpoint of resuscitation. Invasive haemodynamic monitoring provides the clinician with systemic or global values but may not currently the ability to detail regional or microcirculatory flow. While bolus infusions of crystalloid or colloid solutions are still recommended for patients *in extremis* and for patients with isolated extremity injuries, patients who have a strong potential for ongoing internal haemorrhage should now be managed with a deliberate hypotensive approach until definitive haemostasis has been achieved [12]. The most appropriate fluid for resuscitation remains the subject of debate, but trials of hypertonic saline, colloid, and blood substitute solutions have all been recently conducted or are under way. Topical and systemic haemostatic agents and new types of tourniquets all have the potential to improve outcomes and simultaneous 1:1 ratios of red blood cell and plasma infusions may also enhance survival rates [13].

Cervical spine clearance

Cervical spine injury occurs in 5-10% of blunt polytrauma patients. Despite several published clinical guidelines, this is a controversial topic. The application of these guidelines to the obtunded trauma patient is limited. The presence of a severe head injury increases the relative risk of a cervical spine injury by as much as 8.5 times and a focal neurological deficit by 58 times. When the patient is unlikely to be evaluated fully within 24hrs, prolonged immobilisation shifts the risk-benefit analysis from waiting for an opportunity to do a full clinical evaluation, to a non-clinical clearance, given that the vast majority (95%) will not have a cervical injury. Some units combine cervical plain films (lateral, antero posterior and odontoid views) with sagittal reconstructions of the entire cervical spine CT [14].

Use of a protocol using CT scanning alone for blunt trauma patients who were obtunded has shown the risk of missing a cervical spine injury is 0.04% [15].

Current practice at the Royal London Hospital is whole cervical spine helical CT scanning currently represents for assessment of cervical spine injury in the unconscious trauma patient. CT scanning had a sensitivity of 98.1%, a specificity of 98.8%, and a negative predictive value of 99.7%. In contrast, an adequate lateral cervical spine film had a sensitivity of 53.3%, and unstable injuries will be missed. It must be remembered though that no imaging modality will have 100% sensitivity, and there will always be injuries missed by any given protocol [16].

Timing of surgery (Damage Control)

The two main surgical goals for the critically injured trauma patient undergoing a damage control operation are: haemorrhage and source (contamination) control. It is increasingly clear that the coagulopathy stems from both low temperature and acidosis working together -the 'triad of death'. Traumatic coagulopathy is a complex multifactorial process and there appear to be six primary mechanisms involved in its development: tissue trauma, shock, haemodilution, hypothermia, acidosis, and inflammation. Shock is the main driver of early coagulopathy, but requires tissue injury as an initiator. As shock progresses and intravenous therapy is initiated, haemodilution exacerbates the established haemostatic derangements. Where bleeding is untreated, severe hypothermia and acidosis aggravate the established coagulopathy. The clinical importance of the inflammatory reaction is only now being recognized [17].

The concept of damage-control surgery is now well established and has become the accepted method of management of unstable trauma patients over the last decade [18]. This refers to a three-phase surgical approach taken on the basis of patient physiology, the response to fluid loading and level of blood loss. Its

application has been shown to be of benefit both in penetrating abdominal injury and severe chest trauma [19]. Damage control surgery (DCS) is a well-established concept now on deployments and consists of an initial time-limited operation to save life (theoretically less than 1 hour, in practice it may be longer), a period of resuscitation in the intensive care unit, then further definitive surgery. Surgery does not follow resuscitation, it is part of resuscitation and DCS is a component of Damage Control Resuscitation (DCR) [20].

The optimal timing of fracture fixation in the multiply injured is still widely debated. There is no doubt that early fixation of fractures reduces inflammation at the site of injury and decreases pain and opiate requirements. There is also evidence that this approach reduces overall pulmonary complications and promotes early mobilisation [21].

Larger studies tend to indicate that the early stabilisation of femoral fractures with definitive intramedullary nailing appears to be the treatment of choice, even for patients with combined head and chest injuries [22]. It must be remembered that the initial fixation can only take place if the multi-trauma patient is adequately resuscitated. Also, the impact of fatigue on outcome, if surgery is performed in the middle of the night must not be dismissed [23]. For all the reasons noted above, it must not be assumed that definitive fracture fixation will necessarily be carried out prior to ICU admission. Indeed it is recognized now that damage control is not just a surgical concept but really applies to the whole of the patient management and particularly whenever the patient is admitted (or re-admitted after surgery) to the ICU [24].

Intensive Care Admission

A secondary survey should have been completed in the emergency department but if the patient has been expedited to the operating theatre then this may not have been completed prior to ICU admission. For all patients, even if a secondary survey has been completed then a tertiary survey must be performed. When the patient first arrives in the ICU, continued, resuscitation and further evaluation of possible injuries must proceed simultaneously. As in the emergency department, ongoing resuscitation takes precedence over fine detail. Nonetheless, the patient needs to be fully evaluated as quickly as possible so that all injuries and concurrent medical conditions are recognised. This complete reevaluation of the trauma patient that typically occurs in the ICU has been called the tertiary survey (TS) [25]. This should be performed in all trauma patients, regardless of whether or not they require critical care management. The goal is to identify all injuries. Missed injuries can have a significant impact on morbidity, as well as on mortality, from trauma. The more severely injured patients, particularly those with traumatic brain injury (TBI), are at the greatest risk of having occult lesions.

A common pitfall is to focus only on the immediately life threatening injuries, while inadvertently ignoring less obvious but potentially debilitating injuries. The specific components of the tertiary survey include a thorough review of all the patient's injuries and care to date and completion of all abbreviated or deferred elements of the primary and secondary surveys.

Items to be specifically included are an acquisition of a complete and accurate history, performing a thorough physical examination, review of all imaging studies, and laboratory data, and ordering of additional tests as indicated.

Repeated limb compartment checks and continued presence of distal pulses must be recorded in all with limb injuries. Clinical vigilance and identification of a rising serum creatinine kinase levels at this stage can prevent limb loss later. A thorough examination of the eyes and ears is also indicated; this is an often

overlooked aspect of the trauma examination.

Ventilation and respiratory support

Mechanical ventilation is often necessary in severe trauma and particularly blunt chest trauma. Chest CT is known to be superior to chest radiography in assessing the extent of lung contusions. In patients sustaining multi-trauma the development of acute lung injury (ALI) and the acute respiratory distress syndrome (ARDS) is not uncommon. The use of mechanical ventilation is often inevitable in these circumstances and in fact their development is more likely in patients with prolonged ventilation. Current clinical care has moved to the benefits of low tidal volume ventilation at high positive end-expiratory pressures (PEEP), following the publication of the ARDS-net study showing significant mortality benefits in patients with ARDS [26]. Established ARDS characteristically has refractory hypoxaemia, intrapulmonary shunting and a respiratory and metabolic acidosis. Mark inflammatory infiltrates and capillary leak, along with fibrosis and atelectasis (resulting from loss of surfactant) leads to a loss of compliance. The use of the ARDS criteria in trauma patients has been questioned on the basis that some patients seem to behave very differently to others. Some patients have rapid resolution of their symptoms and early weaning from mechanical ventilation, whereas others go on to have ARDS-related pulmonary fibrosis and prolonged weaning. High minute ventilation is characteristic of the long-term ARDS patients, indicating increased dead-space, along with higher peak inspiratory pressures and poorer lung compliance, when initially ventilated. It is accepted however that the lower tidal volume ventilation strategies described earlier should still be applied to both subsets of patients [27]. The mechanisms behind longer-term lung injury may be explained by alveoli behaving differently in different patients; some patients lungs potentially more affected by direct trauma and the stretch effect of mechanical ventilation [28].

Prone ventilation is known to improve oxygenation in severely hypoxic patients; by redistributing blood flow away from previously dependent lung regions and a reduction in ventilation – perfusion (V:Q) mismatch. Improvements in outcome however remain unproven. The problems associated with prone ventilation include dislodgement of tracheal tubes and intravascular catheters, increased intra-abdominal pressure, facial oedema and eye damage. Whether to use pressure-controlled or volume controlled ventilation is also unproven. No other ventilation strategy; apart from lowering tidal volume when ventilating patients with ARDS has been shown to affect the outcome [29]. Novalung GmbH (Hechingen, Germany) has developed a new system using an arteriovenous shunt and a membrane lung that is characterised by an extremely low flow resistance. The system allows the avoidance of a pump by using the patient's arterio-venous pressure gradient as the driving force. The system uses a membrane gas exchange system (lung assist device [LAD]) based on heparin-coated hollow fibre technology with optimised blood flow by reduction of resistance. The LAD is connected to the patient via arterial and venous cannulae inserted by seldinger technique. The system runs with a low-dose heparin infusion that does not exceed normal antithrombotic anticoagulation. Recent studies have suggested a survival benefit as this device allows less aggressive lung ventilation to achieve CO₂ clearance and maintain blood pH [30]. It has been used successfully to facilitate transfer of trauma patients with early acute lung injury over long distances for specialised care [31] and is gaining acceptance in UK intensive practice at the time of writing.

High frequency oscillatory ventilation (HFO) has been widely used to ventilate paediatric patients for more than 2 decades. Only recently, HFO has become available for support of adults weighing more than 35 kg with acute lung injury or acute

respiratory distress syndrome (ARDS). HFO uses a relatively high mean airway pressure. This may sustain lung recruitment more effectively than levels of positive end-expiratory pressure that are typically used during conventional ventilation. HFO also uses tidal volumes that are smaller than those used with conventional ventilation. These features may protect against ventilator-induced lung injury (VILI) more effectively than lung-protective strategies with conventional modes of mechanical ventilation. HFO may be of particular benefit in the young fit trauma patient who goes on to develop severe ARDS, as they have the rehabilitation potential to justify a period of prolonged ventilation, which may be required if HFO is employed [32].

Early (before day 8) tracheostomy has also been shown to be beneficial in the multiple trauma patient, reduce the incidence of pneumonia and decrease the time to weaning from mechanical ventilation [33]. The UK TrachMan Study (early versus late tracheostomy) however is yet to report. It has been noted previously that percutaneous tracheostomy and the use of non-invasive ventilation in intensive care may be appropriate skills for field ICUs to deliver [34].

The Advanced Trauma Life Support (ATLS) algorithm of intubating hypoxic patients with isolated chest contusions has been challenged. It has been shown that patients with significant pulmonary contusion (PaO₂ / FiO₂ ratio < 300) can be treated safely with non-invasive ventilation alone and in fact patients that required intubation were not for reasons of respiratory failure [35]. In patients with unilateral chest trauma ventilator-associated lung injury is a distinct possibility in the healthy lung, as this more compliant lung may be preferentially ventilated leading to excessive stretching and shearing.

Haemodynamic support

The optimal management of fluid balance in severe trauma, involving mechanically ventilated patients has not got universal consensus. Questions revolve around whether patients should be kept 'dry' to help prevent pulmonary oedema; or whether the development of oedema is inevitable due to the underlying condition and therefore fluids should be used to optimise the circulation with the aim of improving cardiac output, oxygen delivery and vital organ perfusion. In all cases the objective is to restore oxygen delivery to the tissues while correcting the underlying cause (for example, surgical intervention to arrest haemorrhage or eradicate infection). Speed is essential. Delays in making the diagnosis and initiating treatment, as well as sub-optimal resuscitation, contribute to the development of peripheral vascular failure and irreversible defects in oxygen use which can culminate in vital organ dysfunction [36]. The first priority is to secure the airway and, if necessary, provide mechanical ventilation. Because mechanical ventilation abolishes or minimises the work of breathing, reduces oxygen consumption, and improves oxygenation, early respiratory support benefits patients with severe shock. Tissue blood flow must be restored by achieving and maintaining an adequate cardiac output and by ensuring that systemic blood pressure is sufficient to maintain perfusion of vital organs. Traditionally, a mean arterial pressure of 60 mm Hg (or systolic blood pressure of 80 mm Hg) has been considered sufficient, but some evidence suggests that a mean pressure of 80 mmHg may be more appropriate. It may be contended that the patient's normal blood pressure should be targeted. Blood transfusions are not without risk and this must be borne in mind when considering the relative benefits of transfusion in terms of oxygen carriage [37]. The use of goal-orientated resuscitation in trauma (as has been advocated in severe septic shock) is gaining support, using the oxygen delivery index. (DO₂I) but the results from studies are not by any means conclusive. Measurement of central venous saturation (ScvO₂) as an indicator of cardiac output (ScvO₂>70% being optimal) and directing the use of fluid

resuscitation and inotropic support has a place in the trauma population. Measurements of base deficit and lactate are also of use [38]. The circulating volume must be replaced within minutes since rapid restoration of cardiac output and tissue perfusion pressure reduces the chances of serious organ damage, particularly acute renal failure. There is no longer any place for the use of dopamine or diuretic infusions to maintain urine output in renal impairment; as these manoeuvres have been shown to do nothing to prevent subsequent renal failure. Efforts should be concentrated on maintaining perfusion via adequate fluid input and use of vasopressors as necessary, to prevent sustained hypotension [39]. It has been recognised for some time however that early depression of cardiac function is associated with poor outcome in patients with trauma [40]. With the decline in the use of the pulmonary artery flotation catheters (PAC), less invasive forms of cardiac monitoring are now being used (such as oesophageal doppler), with the potential to get haemodynamic variables measured, much earlier. There has been an on-going debate about crystalloid versus colloid and the outcome on patients. It has been reported in a large prospective multi-centre trial that the use of albumin or normal saline in the intensive care unit shows similar outcomes at 28 days [41]. Although resuscitation has conventionally aimed at achieving normal haemodynamic values, survival of many critically ill patients is associated with raised values for cardiac output, oxygen delivery, and oxygen consumption. In reality a balanced approach is the norm; fluids are managed with close monitoring of numerous parameters, including peripheral perfusion, pulse rate, CVP response to fluid challenges and stroke volume response to fluids, oesophageal Doppler probe or lithium-dilution continuous cardiac output monitoring. (LiDCCO) Adequate volume resuscitation and inotropic support improves survival in systemic inflammatory response syndrome (SIRS) whilst achieving the same level of cardiac support with inotropes alone does not [42].

Traumatic Brain Injury (TBI)

Primary brain injury is caused directly by the initial impact and this damage is generally referred to the pathology existing immediately following the trauma; secondary injury refers to the destructive changes that evolve over time (hours to days) following the primary event. Direct injury can occur to the brain parenchyma, as well as to the skull, meninges, dura, and blood vessels, which result in space-occupying lesions or haematomas. Intensive care treatment is aimed at lessening the impact of secondary injury by controlling intra-cranial pressure (ICP) and modification of cerebral perfusion pressure (CPP) Maintenance of a cerebral perfusion pressure greater than 70 mm Hg is now widely recognized as a vital component of management of traumatic brain injury.

A systolic blood pressure of less than 90 mmHg occurring between the time of TBI and completion of resuscitation is associated with a 33% increase in mortality. The highest blood sugar occurring in the first 24 hours of ICU care is linearly correlated with mortality. Fever should be prevented in TBI patients as it will merely increase the cerebral metabolic requirement for oxygen. (CMRO₂).

For the patient with TBI, a cerebral perfusion pressure (CPP) of 60–70 mmHg is generally sufficient to maintain cerebral oxygenation. Excessive hyperventilation of patients with TBI will cause ischemia if CO₂ reactivity is preserved. The interpretation of a given ICP measurement must be made in the light of the underlying pathology and the speed of its evolution. Current evidence suggests that 20–25 mmHg is the upper threshold above which treatment to lower ICP should be started. The management of hypotension must include not only fluid replacement but also identification of the cause. There is minimal evidence for the routine use of anticonvulsants to prevent seizures.

A standardised protocol for the management of ICP appears to provide more consistent control. Protocols need to be agreed

jointly between the neurosurgical and intensive care medical team. There are a number of protocols for the prevention of secondary brain injury following major trauma. They are generally directed at the maintenance of cerebral perfusion pressure (CPP) to ensure adequate cerebral blood flow. The Brain Trauma Foundation (www.braintrauma.org) found that mortality increased as the average CPP fell below 70 mm Hg, and that aggressive therapy was required to control intracranial pressure (ICP) and systemic arterial pressure. A number of historical trials have suggested that the ICU and ward mortality for patients with head injuries on a neurosurgical ICU is reduced by the implementation of a target CPP-guided protocol but there are no prospective randomised trials comparing goal-directed therapy with previous conventional head injury management. The evidence suggests that patients with head injuries in the following groups should undergo ICP monitoring:

- i. Those with a Glasgow Coma Score less than 11.
- ii. Those requiring prolonged non-neurosurgical surgery.
- iii. Those with head injury and abnormal CT brain scan [43].

Invasive Intracranial pressure (ICP) monitors have been used on neuro-intensive care units in head injured patients for many years. They have become a standard of care (many would argue a RCT would now be ethically impossible to perform). Recently a report has shown that morbidity is improved if a The morbidity of Head injured patients is improved if treated on a neuro intensive care unit (whether they need surgical intervention or not) Early indications suggest it may be the maintenance of cerebral perfusion pressure. As there is data to show that the incidence of adverse events from inserting intra-cranial pressure monitoring equipment is minimal, it should be safe to use in the field, However it is clear that it is not the monitor itself which makes the difference, and it has been pointed out that therefore all military intensivists and ITU nurses will need significant exposure to these patients pre-deployment for all of the clinical governance issues to be covered [34].

It is becoming increasingly clear that the management of patients with multiple trauma with severe TBI is a very complex task. Diagnosis and therapy need to be primarily directed at preventing multiorgan failure and minimizing secondary brain injury. Thus, early control of haemorrhage, stabilization of circulation and tissue oxygenation, optimization of cerebral perfusion pressure and cerebral blood flow, and adequate treatment of increased intracranial pressure are mandatory [44].

Thoracic trauma

The leading cause of significant blunt chest trauma is road traffic accidents (RTAs). RTAs account for 70–80% of such injuries. Falls and acts of violence are other causative mechanisms. Blast injuries can also result in significant blunt thoracic trauma [45]. Blunt trauma commonly results in chest wall injuries. The pain associated with these injuries may compromise ventilation. Pulmonary contusions, are frequently associated with major chest trauma and may impair ventilation by a similar mechanism. Shunting and dead space ventilation produced by these injuries can also impair oxygenation. Blunt trauma that causes significant cardiac injuries (e.g., rupture of a chamber) or severe great vessel injuries (e.g., thoracic aortic disruption) frequently results in death before adequate treatment can be instituted. The clinical presentation of patients with blunt chest trauma depends on the mechanism of injury and other organ systems involved [46].

The management of the pain associated with chest injuries has also come under scrutiny and is worth considering; especially if the patient is managed with non-invasive ventilation (compared with those who are mechanically ventilated and more likely to have intravenous sedation and analgesia). The pain from rib fractures is known to decrease pulmonary function. Comparisons

of intravenous patient controlled analgesia with morphine and epidural infusions of bupivacaine plus fentanyl have shown superior analgesia via the epidural route [47]. Moreover epidural analgesia has demonstrated improvements in pulmonary function and modifications of the immune response (as measured by lower levels of interleukin IL-8), as compared with PCA [48]. It has been shown to reduce infection rates and shorten mechanical ventilation in patients with more than three rib fractures [49]. The use of epidurals can be difficult however, especially in patients with coagulopathy, non-radiologically cleared thoracic spines and those that are already mechanically ventilated. As such there is limited use of epidural analgesia in the emergency trauma patient and in field conditions.

Penetrating chest trauma comprises a broad spectrum of injuries and severity. The clinical consequences depend on the mechanism of the injury, the location of the injury, associated injuries, and underlying illnesses. Organs at risk, in addition to the intrathoracic contents, include the intraperitoneal viscera, the retroperitoneal space, and the neck. A patient with combined intrathoracic and intra-abdominal wounds has a markedly greater chance of dying. These patients present a particular challenge for the surgical team [50].

Extremity trauma

Extremity injuries pose a complex problem to the multiply injured trauma patient. Additionally, special consideration should be taken for those patients that have injuries isolated to the extremity and require extensive resuscitation and/or operative intervention. Consultation with orthopaedic, surgical and anaesthetic teams is essential to the expeditious care of patients and improving outcomes. In this setting of polytrauma with severe mangled extremities intubation, fluid resuscitation, and proximal vascular control are required during initial resuscitation. Once stabilised, treatment the injuries to the extremities are staged. In muscle compartments, the muscle damage may result in myoglobinuria and acute renal failure in the acute phase; the long-term effects are of muscle damage and contractures (Volkman's contracture). The prevention of the syndrome depends on recognizing conditions that may lead to compartment syndrome and measuring intra-compartmental pressure. A similar syndrome can occur in muscle compartments through any form of injury or trauma that causes muscle swelling. The rise in compartment pressure initially causes extrinsic compression of the venous circulation, and reduces trans-capillary flow. The renewed use of tourniquets in the initial treatment of severe peripheral injuries has lead to fears that limbs may be lost due to muscle compartment ischaemia as a result of this treatment [51].

Abdominal compartment syndrome

Theoretically, compartment syndrome can develop in any compartment, where swelling within the compartment results in a rise in intra-compartmental pressure. This may be within the abdomen, where trauma, ileus, retroperitoneal bleeding, mesenteric oedema and intra-abdominal fluid can all contribute to a rise in abdominal pressure. Once the pressure rises above arterial pressure, ischaemia occurs in the compartment. In the abdomen, this may manifest as acute renal failure or bowel ischaemia, giving rise to a progressive metabolic acidosis. Abdominal compartment syndrome (ACS) often occurs in severely injured patients, especially those who undergo laparotomy with abdominal packing. Early recognition with decompressive laparotomy, leaving an open abdomen with a temporary covering, (such as the Bogota bag) provides immediate improvement in organ function and physiologic status.

ACS may be recognized by the presence of a tensely distended abdomen, elevated peak airway pressures, inadequate ventilation and decreased urine output. However, these findings are relatively

nonspecific. Intra-abdominal pressure can be monitored by instilling 100 ml of saline into the catheterized bladder, which at that volume remains a passive reservoir. The intra-abdominal portion of the bladder can then serve as a transducer to record abdominal pressure, without any contribution from its own musculature. The tubing is held parallel to the patient at the level of the pubis until urine forms a meniscus distal to the sampling port. A clamp is placed distal to the port and a needle is inserted through the port and connected to a water manometer or electronic pressure transducer. A pressure greater than 30 mm Hg is associated with oliguria due to a decreased renal blood flow associated with increased renal venous pressure and a calculated increase in renal vascular resistance. ACS is also associated with an intra abdominal pressure of 25 mm Hg can also cause elevation in intracranial pressure, presumably by increasing central venous pressure, resulting in significant decreases in cerebral perfusion [52].

Pelvic trauma

The management of unstable pelvic fractures has evolved over the last two decades. Radiographic signs of pelvic instability include more than 5 mm of displacement of the posterior SI complex, the presence of a posterior fracture gap (as opposed to impaction), and avulsion fractures of the posterior iliac spine, sacrum, ischial tuberosity, or transverse process of the fifth lumbar vertebra. The first major goal in controlling pelvic bleeding is stabilization of the unstable pelvic injury.

This can be achieved immediately through the use of a pelvic binder applied around the pelvis often (but not always) followed by application of an external fixation device, and subsequent delayed surgical repair once haemorrhage control is satisfactory. Pelvic trauma patients should have intravenous access placed via tributaries that drain into the superior vena cava. Evolving protocols for the haemodynamically unstable patient suggests that initial management should be aimed at the detection of intra-abdominal bleeding using FAST or diagnostic peritoneal lavage within 30 minutes of arrival. Patients with intra-abdominal bleeding should undergo laparotomy immediately, with concomitant pelvic stabilization, to control pelvic venous bleeding. In patients with no intra-abdominal bleeding, or those in whom pelvic arterial bleeding persists after laparotomy, the patient it is recommended that they receive pelvic angiography no more than 45 min after arrival. Optimum bleeding control can usually be achieved with coil embolisation [53]. The risk of deep vein thrombosis is as high as 60% in trauma patients with pelvic fractures and there may be a subsequent need for insertion of an inferior vena cava filter for the prevention of pulmonary embolus. These interventions may not be currently achievable in UK Role 3 field facilities.

Other considerations

As with all ICU patients, general supportive measures however are essential and trauma patients are no exception. Sepsis is the main cause of death in this group and efforts should be made to prevent and treat infections. Infection control and hand-washing in particular have a major role to play, in this respect (Figure 2). Early enteral feeding and thrombo-embolic prophylaxis are also important. Range of motion of all joints should be performed at least daily on patients who are unconscious or unable to conduct their own exercises in conjunction with the normal daily respiratory



Figure 2. Field sink in ICU.

• Trauma patients	Traumatic brain injury guidelines followed? Tertiary survey completed? Surgical plan robust?
• Infection control	Strict adherence to hand hygiene? Field-placed venous access lines changed? Assess need for current central venous access? Appropriate antibiotics?
• Ventilated patients	Head-of-bed elevation? ARDSNet protocol indicated/employed? Oral care protocol? Systematic ventilator liberation efforts? Sedation and analgesia protocol? Is paralysis justified? Pressure area protection optimised?
• Deep vein thrombosis prophylaxis optimised?	
• Stress ulcer prophylaxis required?	
• Glycaemic control best and safest for circumstance?	
• Pain management	Pain well controlled? Candidate for regional anesthesia?
• Nutrition optimization?	
• Candidate for evacuation? Safe for transport?	CCAST arrangements? Records ready?

Box 2. Checklist for the austere intensive care unit [55].

physiotherapy. Box 2 provides a checklist for intensive care provision in an austere environment.

Military trauma patients tend to be young. Successful outcome is often measured in terms of mortality but from the patients perspective a return to a functionally useful life and full employment is usually what they want. Intensive care only forms one link in the chain of survival and as such the goal should be viewed as enabling the patient to benefit from the rehabilitation phase, which commences in and follows discharge from ICU [54].

Previously it had been considered that trauma patients without intracranial haemorrhage or focal neurologic deficits are typically considered low risk for lasting neuropsychological and emotional deficits. However it has been demonstrated that the majority of trauma survivors without intracranial haemorrhage display persistent cognitive impairment, which is nearly twice as likely in those with skull fractures or concussions. This cognitive impairment was associated with functional defects, poor quality of life, and an inability to return to work [56]. The increased use of follow-up clinics for survivors of intensive care will hopefully help with the identification of patients requiring ongoing help and support than has been the case in the past [57].

Sepsis

The impact of sepsis on intensive care patients cannot be understated. Non-compliance with the 6-hour surviving sepsis campaign bundle was associated with a more than two fold increase in hospital mortality. Non-compliance with the 24-hour sepsis bundle resulted in a 76% increase in risk for hospital death. All medical staff should practise relatively simple, easy and cheap bundle-based interventions within a strict time frame to improve survival rates in patients with severe sepsis and septic

shock [58]. The role of the ICU in sepsis is discussed in detail elsewhere in this edition [59]

Evacuation to role 4

The issue of evacuation from Role 3 to UK based Role 4 facilities



Figure 3. CCAST preparing patients in ICU.

will be dealt with in a separate article. Suffice to say this is a major logistic undertaking and requires communication with all those in the evacuation chain (Figure 3).

Summary

From planning through execution, providing critical care services in the field is demanding work. However, experience has shown us that austerity is no barrier in preventing success and high standards of care. Our current knowledge highlights the importance of paying close attention to logistic details, preparing for a wide diversity of patients and illnesses and employing all available resources, including aeromedical evacuation assets.

The expectation should be that care in the field will parallel accepted standards, including the adherence to simple preventive strategies such as sepsis bundles and other quality improvement measures. Rather than associate the equipment and technology with critical care, intensive care should imply the vigilant attention of a skilled multidisciplinary team [55].

Medical lessons learned from Vietnam and previous military conflicts led to the development of civilian trauma systems in the United States. Operations in Iraq and Afghanistan have allowed further development of military trauma systems. Improved military trauma systems care have resulted in an increasing number of multiply injured patients surviving their initial trauma. Patients requiring intensive care for trauma are at risk from sepsis and multiple organ failure [60].

Optimal outcome depends on keeping abreast of the latest thinking in an ever-changing and increasingly technology-rich environment. Only with the continued audit of the service delivery to trauma patients in intensive care can standards be maintained and morbidity and mortality improve in the future [61].

References

1. Roberts MJ, Salmon JB, Sadler PJ. The Provision of Intensive Care and High Dependency Care in the Field *J R Army Med Corps* 2000; **146**: 99-103.
2. O'Neill B, Mohan D. Reducing motor vehicle crash deaths and injuries in newly motorising countries. *BMJ* 2002; **324**: 1142-1145.
3. Mock C, Kobusingye O, Joshipura M, Nguyen S, Arreola-Risae C.. Strengthening trauma and critical care globally. *Current Opinion in Critical Care* 2005; **11**: 568-575.
4. Clarke JR, Trooskin SZ, Doshi PJ, Greenwald L, Mode, CJ. Time to laparotomy for intra-abdominal bleeding from trauma does affect survival for delays up to 90 minutes. *J Trauma* 2002; **52**: 420-425.
5. Fowler R, Pepe PE. Fluid resuscitation of the patient with major trauma. *Current Opinion in Anaesthesiology* 2002; **15**: 173-178.

6. Revell MP, Porter KM, Greaves I. Fluid resuscitation in pre-hospital trauma care: a consensus view. *Trauma* 2002; **4**: 21-28.
7. Dutton RP, Mackenzie CF, Scalea TM. Hypotensive resuscitation during active hemorrhage: impact on in-hospital mortality. *J Trauma* 2002; **52**: 1141-1146.
8. Parkhouse D. 2005. Sugar solutions used in resuscitation. *J R Army Med Corps* 2005; **151**: 5-10.
9. Myers C. Fluid resuscitation. *European Journal of Emergency Medicine* 1997 **4**: 224-232.
10. Porembka DT. 1997. Transesophageal echocardiography in the trauma patient. *Current Opinion in Anaesthesiology* 1997; **10**: 130-141.
11. O'Connor CE. Diagnosing traumatic rupture of the thoracic aorta in the emergency department. *Emerg Med J* 2004; **21**: 414-419.
12. Pepe PE, Dutton RP, Fowler RL. Preoperative resuscitation of the trauma patient. *Current Opinion in Anaesthesiology* 2008; **21**: 216-221.
13. Moore EE, Johnson JL, Cheng AM, Masuno T, Banerjee A. Insights from studies of blood substitutes in trauma. *Shock* 2005; **24**: 197-205.
14. Morris CGT, McCoy E. Clearing the cervical spine in unconscious polytrauma victims. *Anaesthesia* 2004; **59**: 464-482.
15. Sanchez B, Waxman K, Jones T, Conner S, Chung R, Becerra S. Cervical spine clearance in blunt trauma: evaluation of a computed tomography-based protocol *J Trauma* 2005; **59**: 179-183.
16. Brohi K, Healy M, Fotheringham T *et al.* Helical computed tomographic scanning for the evaluation of the cervical spine in the unconscious, intubated trauma patient. *J Trauma* 2005; **58**: 897-901.
17. Hess JR, Brohi K, Dutton RP *et al.* The coagulopathy of trauma: A review of mechanisms. *J Trauma*. 2008; **65**: 748-754.
18. Loveland JA, Boffard KD. Damage control surgery in the abdomen and beyond. *Br J Surg* 2004; **91**: 1095-1101.
19. Gillham MJ, Parr MJA. Resuscitation for major trauma. *Current Opinion in Anaesthesiology* 2002; **15**: 167-172.
20. Hodgetts TJ, Mahoney PF, Kirkman E. Damage control resuscitation *JR Army Med Corps* 2007; **153**: 299-300.
21. Roberts CS, Pape H-C, Jones AL, Malkani AL, Rodriguez JL, Giannoudis PV. 2005. Damage control orthopaedics: evolving concepts in the treatment of patients who have sustained orthopaedic trauma. *J Bone Joint Surg Am* 2005; **87**: 434-449.
22. Nau T, Aldrian S, Koenig F, Veceseli V. Fixation of femoral fractures in multiple-injury patients with combined chest and head injuries. *ANZ J Surg* 2003; **73**: 1018-1021.
23. Revell MP, Porter KM, Greaves I. When is the best time to fix fractures? *Trauma* 2003; **4**: 159-67.
24. Sagraves SG, Toschlog EA, Rotondo MF. Damage control surgery-the intensivist's role. *J Intensive Care Med* 2006; **21**: 5-16.
25. Enderson BL, Reath DB, Meadors J, *et al.* The tertiary trauma survey: A prospective study of missed injury. *J Trauma* 1990; **30**: 666-669.
26. Acute Respiratory Distress Syndrome Network. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. *N Engl J Med* 2000; **342**: 1301-1308.
27. Dicker RA, Morabito DJ, Pittet J-F, Campbell AR, Mackersie RC. Acute respiratory distress syndrome criteria in trauma patients: why the definitions do not work. *J Trauma* 2004; **57**: 522-528.
28. Schiller HJ, McCann UG, Carney DE, Gatto, LA, Steinberg JM, Nieman GF. Altered alveolar mechanics in the acutely injured lung. *Crit Care Med* 2001; **29**: 1049-1055.
29. Moloney ED, Griffiths MJD. Protective ventilation of patients with acute respiratory distress syndrome. *Br J Anaes* 2004; **92**: 261-70.
30. Bein T, Weber F, Philipp A *et al.* A new pump-less extracorporeal interventional lung assist in critical hypoxemia/hypercapnia. *Crit Care Med* 2006; **34**: 1372-1377.
31. Zimmermann M, Philipp A, Schmid, F-X, Dorlac W, Arlt M, Bein T. From Baghdad to Germany: Use of a new pumpless extracorporeal lung assist system in two severely injured US soldiers. *ASAIO Journal* 2007; **53**: e4-e6.
32. Fessler HE, Derdak S, Ferguson ND *et al.* A protocol for high-frequency oscillatory ventilation in adults: results from a roundtable discussion. *Crit Care Med* 2007; **35**: 1649-1654.
33. Dimopoulou I, Anthi A, Lignos M *et al.* Prediction of prolonged ventilatory support in blunt thoracic trauma patients. *Intensive Care Med* 2003; **29**: 1101-1105.
34. Henning JD, Mellor A, Hoffman A, Mahoney PF. Military intensive care. Part 3: future directions. *JR Army Med Corps* 2007; **153**: 288-290.
35. Vidhani K, Kause J, Parr M. 2002. Should we follow ATLS guidelines for the management of traumatic pulmonary contusion: the role of non-invasive ventilatory support. *Resuscitation* 2002; **52**: 265-268.
36. Tisherman SA *et al.* 2003. Clinical practice guidelines: endpoints of resuscitation. Eastern Association for the Surgery of Trauma. (www.east.org)
37. McIntyre L, Hebert PC. To transfuse or not in trauma patients: a presentation of the evidence and rationale. *Current Opinion in Anaesthesiology* 2002; **15**: 179-185.
38. McKinley BA, Valdiva A, Moore FA. Goal-orientated shock resuscitation for major torso trauma: what are we learning? *Current Opinion in Critical Care* 2003; **9**: 292-299.
39. Durham RM, Moran JJ, Mazuski JE, Shapiro MJ, Baue AE, Flint LM. Multiple organ failure in trauma patients. *J Trauma* 2003; **55**: 608-616.
40. Rady MY, Edwards JD, Nightingale P. Early cardiorespiratory findings after severe blunt thoracic trauma and their relation to outcome. *Br J Surg* 1992; **79**: 65-8.
41. SAFE Study Investigators. A comparison of albumin and saline for fluid resuscitation in the intensive care unit. *N Engl J Med* 2004; **350**: 2247-56.
42. Tomlinson L, Bellingan GJ. Trauma and acute lung injury. *Trauma* 2002; **4**: 147-157.
43. Fakhry SM, Trask AL, Waller MA, Watts DD. IRTC Neurotrauma Task Force. Management of brain-injured patients by an evidence-based medicine protocol improves outcomes and decreases hospital charges. *J Trauma* 2004; **56**: 492-499.
44. Sumann G, Kampfl A, Wenzel V, Schobersberger W. Early intensive care unit intervention for trauma care: what alters the outcome? *Curr Opin Crit Care* 2002; **8**: 587-592.
45. Lavery GG, Lowry KG. Management of blast injuries and shock lung. *Current Opinion in Anaesthesiology* 2004; **17**: 151-157.
46. Sayman AG, Findlay GP. The management of blunt thoracic trauma. *Br J Anaes / CEPD Review* 2003; **6**: 171-174.
47. Wu CL, Perkins FM, Barquist E. 1999. Thoracic epidural analgesia versus intravenous patient-controlled analgesia for the treatment of rib fracture pain after motor vehicle crash. *J Trauma* 47: 564-567.
48. Moon MR, Luchette FA, Gibson SW *et al.* Prospective randomised comparison of epidural versus parenteral opioid analgesia in thoracic trauma. *Ann Surg*; **229**: 684-691.
49. Bulger EM, Edwards T, Klotz P, Jurkovich GJ. Epidural analgesia improves outcome after multiple rib fractures. *Surgery* 2004; **136**: 426-430.
50. Asensio JA, Arroyo H, Veloz W *et al.* Penetrating thoraco-abdominal injuries: ongoing dilemma - which cavity and when? *World J Surg* 2002; **26**: 539-543.
51. Parker PJ, Clasper J. The Military Tourniquet. *JR Army Med Corps* 2007; **153**: 10-15.
52. Gentilello LM, Pierson DJ. Trauma critical care. *Am J Respir Crit Care Med* 2001; **163**: 604-607.
53. Fangio P, Asehnoune K, Edouard A, Smail N, Benbamou D. Early embolization and vasopressor administration for management of life-threatening hemorrhage from pelvic fracture. *J Trauma* 2005; **58**: 978-984.
54. Moecke H, von Knobelsdorff G. The anesthesiologist in prehospital and hospital emergency medicine. *Current Opinion in Anesthesiology* 2008; **21**: 228-232.
55. Venticinque SG, Grathwohl KW. Critical care in the austere environment: Providing exceptional care in unusual places. *Crit Care Med* 2008; **36**: S284-S292.
56. Jackson JC, Obrebsky WB, Bauer R *et al.* Long-term cognitive, emotional, and functional outcomes in trauma intensive care unit survivors without intracranial hemorrhage. *J Trauma* 2007; **62**: 80-88.
57. Griffiths JA, Barber VS, Cuthbertson BH, Young, JD. A national survey of intensive care follow-up clinics. *Anaesthesia*. 2006; **61**: 950-955.
58. Gao F, Melody T, Daniels DE, Giles S, Fox S. The impact of compliance with 6-hour and 24-hour sepsis bundles on hospital mortality in patients with severe sepsis: a prospective observational study. *Critical Care* 2005; **9**: R764-R770.
59. Johnston AMcD. Sepsis and Intensive Care. *J R Army Med Corps* 2009; **155(2)**: 142-145.
60. Eastridge BJ, Jenkins D, Flaherty S, Schiller H, Holcomb JB. Trauma system development in a theater of war: experiences from operation Iraqi freedom and operation enduring freedom. *J Trauma*. 2006; **61**: 1366-1373.
61. Brooks AJ, Sperry D, Riley B, Girling KJ. Improving performance in the management of severely injured patients in critical care. *Injury* 2005; **36**: 310-316.