MINI-SYMPOSIUM: BULLET AND BLAST INJURIES

(i) An overview of the pathophysiology of gunshot and blast injury with resuscitation guidelines

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Summary
Because of the blurring of the ‘front line’ the knowledge and skills once only the province of the military surgeon is now required by the civilian trauma team. The mechanisms of injury and basic resuscitative principles are set out.

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Introduction
Recent years have seen an increase in gunshot wounding and blast injury in the UK associated with criminal and terrorist activities. Injury patterns previously confined to the military environment are now seen in civilian practice. Thus any doctor treating trauma victims should have a basic understanding of the mechanisms involved in such injuries. This overview intends to provide a short explanation of the biophysics behind these injury mechanisms, the pathophysiology of the main organ systems affected and early resuscitation guidelines.

Gunshot injuries

Basic ballistics

Ballistics is the study of the motion of an object when it has been launched, shot, hurled, thrown, etc. or by other means projected such as bullets. The wounding effects of projectiles are determined by:

- the physical properties of the missile (mass, shape, composition),
- the flight characteristics (velocity, stability and yaw or tumbling characteristics).

In addition the reaction of the differing types of tissue the missile encounters affect wounding outcome.

Modern cartridges (Fig. 1) have in one container the components necessary to propel a bullet from a gun. As the cartridge is fired the burning powder produces a gas. As the volume of this gas increases, the internal pressure within the barrel of the gun rises, forcing the bullet down the barrel. As it does so, it is engaged by the spiral grooving or rifling of the barrel causing it to spin. This stabilises the flight of the projectile, maintaining its trajectory, range and accuracy.

Velocity is the single most important factor in creating a wound usually classified as either low-velocity (<600 m/s or <2000 ft/s) or high-velocity (>600 m/s or >2000 ft/s)
injuries. The initial velocity is described as the velocity of the projectile at 5 m (15 ft) from the muzzle.

Low-velocity wounds are more common in civilian practice and are usually less severe, whereas high-velocity wounds are more severe in nature, causing widespread tissue damage, and are more commonly seen in the military setting.

The terms high and low velocity however can be misleading. For instance, a shotgun injury is technically a low-velocity injury but is frequently responsible for major soft tissue, nerve, vascular and joint injuries. More useful is perhaps the use of low-energy and high-energy wounds, which is indicative of the amount of tissue damage sustained suggesting the concept that energy transfer from the missile to the tissue is responsible for the severity of the wound.\(^1\)

This relationship is where the kinetic energy of the bullet or other projectile:

\[
\text{kinetic energy (KE)} = 0.5 \times \text{mass} \times (\text{velocity})^2.
\]

Thus as the mass of the bullet increases, there is a linear increase of its kinetic energy, but if the velocity is increased, KE and wounding potential increase exponentially.

The velocity is dependent upon shape, weight and calibre (calibre is equivalent to the diameter of the bore of the weapon) known as the ballistic properties of the bullet and weapon. Differences in ballistic properties affect flight through the air, e.g. some slow more rapidly than others. The ballistic coefficient represents the ease with which the missile penetrates the atmosphere. Controlled missile flight is a factor of its ballistic coefficient and the spin imparted to it by the rifling of the barrel. The flight of the missile through the air is also affected by yaw. Yaw is produced by the inherent asymmetry of any bullet as it deviates from its longitudinal axis during flight. This is identified mostly during the initial and final phases of flight producing phases of instability and thus deceleration of the round. The rifling of the barrel reduces yaw to a minimum.

The design and composition of a bullet varies. Usually they have a lead core to increase mass, which may or may not be jacketed (enveloped) with copper. A bullet is described as having a nose or point, a body and a base or heel.

The nose may be jacketed or not and can be rounded, pointed, flat, hollow, semi-hollow or full. A rounded nose makes for non-expansion on impact; a pointed nose, most a military type, tends to penetrate tissues more deeply, whereas a flat nosed round tends to expand on impact creating a larger superficial wound.

The body contains the core, usually lead, providing the necessary concentricity and balance for flight. The base or heel may be flat or boat tailed, the latter to reduce air drag to improve velocity and range. The combination of these properties further affect tissue damage.

The energy transfer from the bullet to the tissues depends on six factors:\(^2\):

1. The residual KE at impact. This is dependant on range, i.e. the further the target is away from the weapon the lower the KE.

2. The stability and entrance profile of the round. i.e. if the bullet has already started to tumble then the wound on impact will be ragged and more superficial. If flight has remained stable then impact wounding will be smaller and more circumscribed.

3. The calibre, construction and design of the bullet.

4. The penetration of the bullet, i.e. the distance it travels within the tissues. If it is penetrating, it is retained within the wound and thus its whole KE is delivered to the surrounding tissues. If it perforates, i.e. passes through the tissues and exits, the KE transfer is significantly less (Fig. 2).

5. The biological characteristics of the tissues the round impacts, i.e. tissue elasticity, cohesiveness and density.

6. The mechanism of tissue disruption, i.e. stretching, tearing, crushing.

The impact velocity necessary to penetrate skin is approximately 40–50 m/s (150–170 ft/s).\(^3\) Most entry wounds are small, round to oval in appearance with clean well-defined edges. Tattooing surrounding the entry wound, caused from the powder within the cartridge, suggests that a close range injury has occurred. However, the wound may be insignificant and be easily missed or be surrounded by a ring of damaged skin with the appearance of an abrasion or bruise. A flat nosed bullet may cause a large superficial entry wound and appear to have caused far greater tissue damage.

As the bullet traverses the tissues, a temporary cavity forms for approximately 10–30 ms (Fig. 3) due to stretching of the tissues as the bullet passes through them forming a cavity with a relative vacuum behind it. The volume of the cavity formed is proportional to the KE of the bullet and its
size. The maximum size of 10–40 times the diameter of the round is reached within 1–4 ms of impact. Internal pressures within the cavity of between 100 and 200 atm (10–20 MPa) have been measured.\(^2\) In high-velocity injuries this may create damage of an almost explosive nature. The subsequent vacuum sucks foreign material such as pieces of clothing, dirt, etc. into the wound causing further contamination. The temporary cavity collapses and reforms repeatedly with diminishing amplitude, eventually leaving a permanent small cavity. The more the elastic nature of the surrounding tissues has been exceeded the larger the size of the permanent cavity. Surrounding this cavity therefore is a zone of contused soft tissue. In low-energy wounds, such as an airgun rifle wound, this may be only a few cells in depth. However, in high-energy transfer wounds such as those with a velocity of > 600 m/s (2000 ft/s) this volume of devitalised

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**Figure 2** Rounds within tissues. Pictures (a) and (b) show AP and lateral views of the cervical spine with an AK47 rifle round remaining within the soft tissue. The nose of the round remains pointed for deep penetration, although the body of the round has already been destorted by ricochet. Fortunately this round was at the end of its range and the patient survived. Pictures (c), (d), (e) and (f) show a round on the shoulder X-ray, the round after being removed and the patient from whom it was removed. The round had entered on the left side of the neck, passed through the soft tissue and exited on the right side only to re-enter the patient to be stopped by the distal clavicle. This demonstrates a typical rounded nose, fully jacketed with copper, flat-based round frequently used in the military setting. The initial entry and exit wounds are the same size.

**Figure 3** Demonstrates the passage of a round as it penetrates a gelotine block with the formation of a cavity.
tissue becomes increasingly significant and may extend several centimetres away from the track of the projectile. Fascial planes can serve as channels along which energy dissipates leading to remote tissue damage. Therefore disruption of muscular capillary beds, fractures and rupture of gas containing viscera can occur remotely from the impact without direct injury. The densities of the tissues within the path of the projectile are important on the overall wound- ing. The skin and lung, with relatively low density but large elasticity can be virtually spared from significant damage. Bone however, with a higher density but low elasticity, can be completely shattered. The liver, spleen and muscle are widely disrupted when the KE transferred exceeds the elasticity of the tissue. Larger arteries and nerve trunks are remarkably resistant to injury, and while neurapraxias do occur, nerves are rarely completely disrupted unless in the direct path of the missile.

Exit wounds occur when the round has sufficient KE to perforate and travel through the body. Frequently, but not always, the exit wound is larger than the entry wound. As the round loses energy it begins to tumble within the tissue area thus creating a larger more irregular, less well-defined wound. In high-velocity injuries cavity formation may occur at the exit wound additionally sucking in substantial quantities of contaminated material (Fig. 4).

**Bone**

A velocity of approximately 60 m/s (195–200 ft/s) is required to breach the cortex of bone. Fracture patterns are variable and may be complete or incomplete, and obviously fractures have concomitant soft tissue injury. Low-velocity projectiles tend to cause relatively minor, stable fracture configurations such as unicortical involvement, passing completely through the bone, or a chip to the bone. High-velocity projectiles are more likely to cause unstable fracture configurations with butterfly fragments and large amounts of comminution. Bones are fractured either due to direct contact with the bullet or by a secondary energy transfer via the temporary cavitation. Bone fragments may themselves become secondary missiles, causing further damage to more distant structures, although retracting to the original site on dissipation of the KE.

In the immature skeleton, physeal injuries are usually as a result of direct injury from the projectile passing close to the growth plate. This can be easily identified on initial X-rays. However, physeal arrest has been associated with remote injury.

**Wound assessment and initial resuscitation**

As with any injury, assessment following ATLS guidelines should be undertaken at presentation with, if possible, an immediate full history and examination. Tetanus prophylaxis, an initial dose of broad spectrum IV antibiotics should be administered. Full assessment of the patient should include head to foot examination including a log roll and rectal examination. Identification of entrance and exit wounds should be made and documented before being initially cleaned and dressed with a betadine soaked gauze dressing.

In the civilian environment, patients are generally clean and present with low-energy transfer wounds (i.e. wounds from a handgun) and can often report the type of weapon used. This has lead to a conservative approach being adopted in the management of these wounds. Examples of low-energy wounds include airgun rifle injuries where the entrance wound can be as small as 0.5 cm in diameter and the round retained within the soft tissues, with minimal injury to the skin, subcutaneous tissue and muscle. Fractures, if present are of a stable configuration, can be treated conservatively not requiring operative intervention.

Military-type wounds are the antithesis and must be managed more aggressively with early surgery. The presence of massive soft tissue damage with gross contamination, unstable fracture configurations with or without joint involvement, neurological deficit, vascular injury, gastrointestinal tract involvement and late presentation (wounds >8 h post-injury) require immediate surgical intervention with washout and formal wound excision. Other indications for surgery are tendon injury, superficial fragments in the palm of the hand or sole of the foot, certain cases with spinal involvement and injuries to the bony pelvis. A retained metallic fragment within a joint cavity is an absolute indication for intervention (as most rounds contain lead). Further management considerations are discussed later within this mini-symposium.

**Blast injury**

**Blast physics**

An explosive is a material capable of producing an explosion by its own energy. Explosives produce heat and gas. An explosion or blast follows a sudden release of energy from a chemical, gaseous, mechanical or even nuclear means dissipated by a blast wave, propelling fragments and surrounding material, and causing heat formation. The gas is the primary mechanism by which the explosive produces its effects. Thus following detonation of an explosive in air, a shock front (a wave travelling in excess of the speed of sound in that medium) travels away from the centre of the charge. The initial shock wave following an explosion is a special form of high-pressure stress wave, with an instantaneous wave front. The surrounding atmosphere is heated by the passage of this shock wave and then forced outwards by the expansion of gases formed within the explosion. After a short distance of travel of the shock front a further shock front is formed in the air (Fig. 5). This has a lower peak pressure and initial velocity than the detonation shock wave and has a zone of rarefied air immediately behind the high-pressure area. This second shock front is called the blast wave. Within this blast wave are the products of the explosion, i.e. the gas and fragments of debris. The blast wave travels supersonically before decaying into an acoustic wave as it loses velocity and magnitude. The blast wave travels further than the detonation shock wave, exerting its effects further from the explosion centre. If the explosion is in the open, without being confined by buildings, etc. or is underwater, then a simple blast wave is produced (Fig. 6).
Figure 4  Entry and exit wounds. These pictures demonstrate the entry and exit wounds with the associated bony injury observed. In both cases the entrance wounds are small and the exit wound significantly greater in size. The top patient unfortunately had a complete disruption of the brachial plexus and axillary artery demonstrating the high-energy nature of the firearm that sustained this injury. This resulted in amputation. The lower patient was neurovascu larly intact.
This simple wave form has an almost instantaneous rise to peak overpressure, which then declines exponentially through ambient pressure to sub-atmospheric pressure, corresponding to the rarefied zone behind the blast front. The overpressure lasts for approximately 10 ms, with the sub-atmospheric pressure zone lasting for considerably longer. Confinement of the explosion within a building or underwater produces a complex blast wave pattern containing multiple overpressure peaks, due to reflections of the blast wave. The biological effects of the blast wave depend on the peak overpressure and its duration.

Classification of blast injuries

Blast injuries fall into four main categories:

(i) Primary blast injury relates to the interaction of the initial shock wave with the body. Gas containing structures such as the ear, lungs, and gastrointestinal tract are at particular risk. Solid organs including the skin are more resistant to the blast wave. Thus a patient with pure primary blast injury may display little external evidence of trauma.

(ii) Secondary blast injury occurs as a result of the blast wave or wind and is caused by bomb fragments and other ‘secondary’ projectiles energised by the explosion causing penetrating or non-penetrating wounds. Any part of the body may be affected.

(iii) Tertiary blast injury occurs as the result of gross body displacement, i.e. the body being thrown through the air as a result of the blast wind. This leads to crush injuries. The combination of primary blast injury from the shock wave and tertiary injury due to body displacement leads to limb avulsion injuries. This occurs as the initial shock wave causes long bone fractures and the body displacement flailing of the limb with subsequent avulsion.

(iv) Quaternary blast injury is a miscellaneous collection of all other mechanisms. These include thermal injury to exposed skin caused by the radiant and convective heat of the explosion, methaemoglobinemia due to poisoning by dinitrobenzene or potassium perchlorate (from the explosive), and acute septicaemic meliodosis due to inhalation of soil particles contaminated with Pseudomonas pseudomallei. A high incidence of psychological sequelae in injured and uninjured survivors is also seen.

Primary blast injuries result from the interaction of the shock wave with the body and are therefore a type of non-penetrating injury. The interaction of the blast wave with the body wall generates two types of waves. These are known as stress waves and shear waves.

Stress waves are longitudinal pressure waves with similar properties to sound waves. They travel at approximately the speed of sound, but differ from sound waves because of their high amplitude and velocity. The initial shock wave following an explosion is a special form of high-pressure stress wave, with an effectively instantaneous wave front, which travels through a medium at speeds greater than the speed of sound in that medium. The properties of this wave form explain the effects produced on tissues. Effects include high local forces produced with small rapid distortions, thus producing microvascular disruption, without gross lacerations. Organs with differing acoustic impedance are affected i.e. gas containing organs more readily. Tissue interfaces reflect and reinforce stress waves causing enlargement of wave pressures far from the site of body impact.

The coupling of stress waves through the abdominal and thoracic walls is responsible for the primary blast injury to the gastro-intestinal tract. This occurs due to:

(a) The development of pressure differentials across the delicate structures such as alveolar septa causing disruption.

(b) As the stress wave passes from a solid into a gas filled tissue interface, a component of the compressive stress wave is reflected back as a tension wave. Most materials are weaker in tension than in compression and thus disruption and therefore damage at the tissue interface occurs.

(c) When the stress wave compresses a gas containing structure such as an alveolus or bowel segment, the subsequent expansion causes damage to the wall of the structure.

Shear waves are pressure waves transmitted perpendicular to stress waves and are of long duration and low
velocity. These result from the deformation of the body wall and compression of the visceral structures. The tearing of structures from their attachments and shearing of solid organs is caused by the asynchronous movements of tissues with differing inertia. Shear waves are thus responsible for the primary blast injury of solid abdominal viscera, mesenteries and large bowel. The musculoskeletal system being solid is relatively resistant to the pressure waves although it has been demonstrated that initial shock waves of sufficient intensity can cause long bone fracture.6

Pathophysiology of primary blast lung injury

Human post-mortem data of fatal primary blast injury are rare. Gross findings are of heavily consolidated haemorrhagic lungs. (Fig. 7) Haemorrhages may be multiple, subpleural lesions or those coalescing to involve the entire thickness of a lobe. These are often bilateral and multilobar. Animal experiments of primary blast injury demonstrate that the most consistent lesion was bilateral traumatic haemorrhage. Tension pneumothorax resulting from the rupture of subpleural cyst formed following alveolar septal tearing may occur and air embolism may occur.

Haemorrhage into the alveoli and the resultant pulmonary oedema cause a ventilation perfusion mismatch with increased intrapulmonary shunt, reduced lung compliance, resulting in hypoxia and increased work of breathing. This response is similar to that seen in other non-penetrating lung injury.

Initial clinical observations of the physiological responses to blast injury vary considerably, largely due to the varying times at which these observations are made post-injury and the secondary associated injuries sustained. Observations of victims dying immediately following blast exposure with little external evidence of injury led to the theory that the blast wave causes an acute cardiovascular and respiratory response. Experimental work on animals subjected to a blast wave causes an acute cardiovascular and respiratory response is similar to that seen in other non-penetrating injury.

Initial symptoms of blast lung include dyspnoea, cough (which may be dry or productive), haemoptysis, chest pain or discomfort (characteristically retrosternal). Signs include tachypnoea, cyanosis, reduced breath sounds with dullness to percussion, coarse crepitations and rhonchi are well described. Surgical emphysema and retinal artery emboli may also be evident.

Pneumothorax/haemopneumothorax presenting with sudden shortness of breath, pain and deviated trachea require immediate attention.

Pathophysiology of intestinal primary blast injury

Because of its many tissue/gaseous interfaces the intestine is highly susceptible to primary blast injury. Secondary and tertiary penetrating injuries must be managed in a conventional manner. The primary characteristic of intestinal primary blast injury is the intramural haematoma, although extreme overpressure shock waves will cause immediate gut laceration. Intramural haematomas may be minor, mucosal or submucosal haemorrhage only with oedema, through to complete disruption of the muscular layers and serosa, causing perforation. Individuals sustaining blast injury are observed to sustain injury mainly in the ileocaecal region and colon which are more likely to be gas filled. Pre-laparotomy detection of these injuries is difficult. The natural history is uncertain and later perforation up to 14 days post-injury is reported. Thus Cripps et al. have proposed a histological classification for small and large bowel intramural haemorrhage which identifies contusions at high, intermediate and low risk of perforation. However, this triad is not shown in animals undergoing abdominal blast exposure. Therefore it is likely, that such cardiorespiratory responses are multifactorial, and that the additional conventional injuries associated with haemorrhage are likely to alter the physiological picture, but this has yet to be shown.

Clinical features of blast lung

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Clinical features of intestinal primary blast injury

Abdominal pain, vomiting, haematemesis, distension, rectal pain with tenesmus and the presence of loose stools with fresh blood, or melena are indicative of such injury. Abdominal guarding with rebound tenderness and absent bowel sounds are indicative of intra-abdominal injury. Plain X-ray, ultrasonography and CT scanning may assist in the diagnosis of immediate perforation and fluid within the abdomen. Diagnostic peritoneal lavage has undisputed value in the diagnosis of intraperitoneal bleeding, but it is
insensitive to the retroperitoneal and mesenteric haematoma, likely to occur in intramural haemorrhage of blast injury. In some patients the indications for laparotomy are obvious, in others primary blast injury represents a diagnostic challenge, injury remaining clinically silent until complications are manifest.

Pathophysiology of auditory injuries

The ear is particularly susceptible to rapid pressure changes, but because these injuries are not life threatening, they can be easily overlooked and frequently under recorded. A small explosive causing an overpressure wave in a confined area can cause significant auditory damage. Injuries are categorised anatomically. The external ear is more likely to sustain injury from secondary or tertiary blast effects and should be managed accordingly. The shock wave on reaching the tympanic membrane (TM) causes displacement medially, creating a range of injury from intra-tympanic haemorrhage to radiation laceration. Perforation may disintegrate the TM completely or leave inverted or everted drum flaps. Small fragments of keratinising squamous epithelium may be distributed throughout the middle ear and mastoid system. If not removed they may subsequently form cholesteatoma. Disruption of the ossicular chain may be associated with TM rupture. The frequencies of such injuries are variable in the reported literature.

Many blast survivors experience a profound, short-lived sensory neural hearing loss with tinnitus. The duration varies from a few hours to a permanent deficit. A few individuals experience vertiginous problems after blast injury. This may be caused by post-concussional states rather than labyrinthise damage. Otolaryngological examination and follow up is recommended in all blast injury patients suspected of auditory injury.

Pathophysiology of central nervous system primary blast injury

Serious head injury is the most common cause of death in terrorist bombings. The enclosure of the brain within the skull makes survivable primary blast injury unusual. Brain and spinal cord injuries are usually caused by the secondary and tertiary effects of blast. Experimental studies on rabbits sustaining a large energy blast have demonstrated haemorrhage into the ventricles from the choroid plexus and haemorrhage into the pia, but none into the brain substance itself. Extradural spinal cord haemorrhages were also observed in these animals. Other animal studies in rats using histological techniques have demonstrated hypertrophy of microglial cells, a characteristic feature of neural degeneration, evident in the superficial zones of the cerebral and cerebellar cortices between day 1 and day 14 post-injury. These have reverted to normal levels by day 28.

While clinical and experimental evidence exists for directly induced damage to the nervous system by the shock wave, more is known about the effects of air emboli in cerebral vessels. This is probably a more significant mechanism of injury. Air gains access to the circulation via alveolar-venous fistulae secondary to pulmonary blast injury and has been considered to be one of the principal causes of immediate and early death due to primary blast injury. Thus symptoms and signs of air emboli should be sought early in patient assessment. These include headache, vertigo, ataxia, convulsions, altered levels of consciousness, weakness or sensory loss, facial or tongue blanching and retinal artery air emboli. The immediate action for this is the administration of oxygen. Definitive treatment requires hyperbaric oxygen, although this is not universally available. It reduces the volume of gas bubbles and improves blood flow to hypoperfused tissues.

Pathophysiology of orthopaedic blast injuries

Most skeletal injuries, as already suggested, occur due to the effects of secondary and tertiary blast injury. Traumatic amputation occurs as the result of a combined primary and tertiary injury. In survivors of blast injury only 1.5% have traumatic amputations of limbs, with the exception of those injured by anti-personnel mines, discussed elsewhere in this symposium. However, traumatic amputation is relatively common in those patients dying early, reported as 19% by Mellor et al. in an analysis of servicemen killed or injured by blast in Northern Ireland between 1970 and 1984. It was considered that the blast wind caused rapid displacement as being the primary mechanism for this injury. However, it is now considered not to be the case. If traumatic avulsion were to occur through flailing alone, then amputation would occur through or close to joints as observed in fast jet pilots ejecting from aircraft and being exposed to slipstream wind speeds of 1100 km/h, which approximates to blast wind speeds. However, post-mortem data demonstrate that traumatic amputations following blast occur through the shafts of long bones and are not associated with joints (Fig. 8). Experiments undertaken on goats by Hull et al. have shown that the coupling of the initial shock waves into long bones generates stress waves which cause fracture of the long bone shaft. These occur most frequently in the upper third of the tibia and upper or lower third of the femur. The displacement of the body by the blast wind causes separation of the limb from the body through the fracture site. Large external, foreign fragments may also

Figure 8 Traumatic amputation through the lower tibia.
cause traumatic amputation of limbs, but this is not the most usual mechanism.

**Fragment wounds**

The current favoured weapon of the insurgent and terrorist bomber is the improvised explosive device (IED). These often crude devices are capable of inflicting massive injury to those in the immediate locality of the explosion. The casing of the bomb disintegrates into injurious fragments with nails and ball bearings sometimes added to maximise wounding. In a military environment the limbs are most frequently injured with relative sparing of the chest and abdomen due to the widespread use of personal body armour (Fig. 9). In those individuals closest to the blast epicentre the wounds sustained may be as a result of a combination of all four categories of blast injury (Fig. 10). In the case illustrated, it is clear that surgical wound excision is mandatory to remove all non-viable and foreign material. In situations where fragments have not penetrated vital structures and where the limb wounds are small a conservative approach can be taken. Subsequent infection rates are low. This has been the experience of the two authors during tours of duty in Iraq.

**Initial resuscitation of blast injury**

Isolated primary blast injury is almost never seen, unless under experimental conditions. More frequently and therefore of more relevance to this review, is the combined primary blast injury associated with secondary and tertiary

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**Figure 9** Orthopaedic blast injuries. These pictures show the results of explosive devices which have gone off very close to the vehicles in which these patients were travelling. They are the result of two separate incidents. The X-rays demonstrate multiple pieces of retained fragments but also the severity of the bony injuries. Neither of these patients were propelled through the air following the explosion but were retained within their vehicles. Patient (a) was wearing body amour at the time of the explosion and did not develop any signs of primary blast injury to the lungs or intestine within the first 48 h. There is, however, no experimental evidence to suggest that body amour reduces transmission of the initial shock wave, but it could be considered that the bony injury results from a combination of primary blast injury and secondary fragment injury. This patient was neurovascularly intact on presentation. Patient (b) had a complete disruption of the tibial artery trifurcation and an asensate foot on presentation despite the soft tissue injury not appearing to be as significant on initial examination. This patient also sustained intra-abdominal pathology and multiple burns to his exposed skin.
blast injury leading to combined penetrating and non-penetrating injuries. First aid and resuscitation should be commenced immediately. The airway should be checked and cleared, high flow oxygen should be utilised and intravenous fluid resuscitation commenced. Currently, the regimen chosen for fluid resuscitation in this type of casualty remains a subject of some debate. Most recently, the NICE guidelines of 2004\textsuperscript{20} have reported that hypotensive resuscitation should replace normotensive fluid resuscitation in trauma casualties. However, evidence supporting this is limited to those victims with penetrating injuries having short delays to definitive surgical management. Recent randomised trials in animal models investigating the physiology of resuscitation after blast, combined a primary blast injury with an associated controlled haemorrhage of 30% blood volume.\textsuperscript{21} Normotensive resuscitation was compared with hypotensive resuscitation for prolonged periods of up to 8 h post-injury. Normotensive resuscitation follows ATLS guidelines providing the equivalent of 2 l of 0.9% NaCl to a 70 kg man in order to regain a systolic blood pressure of 110 mmHg. Further bolus doses of 0.9% NaCl are given in order to maintain this level of systolic blood pressure. Hypotensive resuscitation follows BATLS guidelines and provides 3 ml/kg/min of 0.9% NaCl until a systolic pressure of 80 mmHg is obtained or a palpable radial pulse and maintained at this level with bolus doses of 0.9% NaCl. These studies demonstrate a considerable survival advantage in those animals receiving normotensive resuscitation and that prolonged hypotensive resuscitation was not compatible with survival after primary blast injury. Also identified within these experiments was that the blast injury exacerbated the fall in arterial blood pH when treated with hypotensive resuscitation which contributed to the more rapid demise of the blast injured model.

The risk of re-bleeding associated with normotensive resuscitation remains high on the list of complications in such cases. However, each individual case must be considered separately. In the civilian setting fortunately in the UK such injuries remain relatively uncommon and time to definitive surgical intervention is short. However, this has more relevance within the military setting where delay to evacuation and definitive surgery can be significantly prolonged.

References