

Received 17 December 2012

Received in revised form 3 April 2013

Accepted 7 April 2013

Available online 8 December 2013

Blast-related Fracture Patterns: A Forensic Biomechanical Approach

Arul Ramasamy^{*, a, b}, Adam M Hill^{a, b}, Spyros Masouros^a, Iain Gibb^c, Anthony M J Bull^a, Jon C Clasper^{a, b}

^a Imperial Blast Biomechanics and Biophysics Group, Rm 4.28 Royal School of Mines, Imperial College London, South Kensington, London SW7 2AZ, UK

^b Academic Department of Military Surgery and Trauma, Royal Centre for Defence Medicine, Birmingham Research Park, Vincent Drive, Birmingham B15 2SQ, UK.

^c DCentre for Defence Imaging, Horton Block, Fort Blockhouse, Gosport, Hants, UK.

* Corresponding Author: Arul Ramasamy. E-mail: Email: a.ramasamy09@imperial.ac.uk.

Abstract Improved protective measures and medical care has increased the survivability from battlefield injuries. In an attempt to reduce the debilitating consequences of blast injury, understanding and mitigating the effects of explosion on the extremities is key. In this study, forensic biomechanical analyses have been applied to determine mechanisms of injury after the traumatic event. The aims of this study were (i) to determine which effects of the explosion are responsible for combat casualty extremity bone injury in two distinct environments, namely open, free-field (open group), and in vehicle or in cover (enclosed group), and (ii) to determine whether patterns of combat casualty bone injury differed between environments. Medical records of casualties admitted to a military hospital in Afghanistan were reviewed over a six-month period. Explosive injuries have been sub-divided traditionally into primary, secondary and tertiary effects. All radiographs were independently reviewed by a military radiologist, a team of military orthopaedic surgeons and a team of academic biomechanists, in order to determine 'zones of injury' (ZoIs), and their related mechanisms. Sixty-two combat casualties with 115 ZoIs were identified. Thirty-four casualties in the open group sustained 56 ZoIs; 28 casualties in the enclosed group sustained 59 ZoIs. There was no statistical difference in mean ZoIs per casualty between groups ($p = 0.54$). There was a higher proportion of lower limb injuries in the enclosed group compared with the open group ($p < 0.05$). Of the casualties in the open group, 1 ZoI was owing to the primary effects of blast, 10 owing to a combination of primary and secondary blast effects, 23 owing to secondary blast effects and 24 owing to tertiary blast effects. In contrast, tertiary blast effects predominated in the enclosed group, accounting for 96 per cent of ZoIs. These data clearly demonstrate two distinct injury groups based upon the casualties' environment. The enclosed environment appears to attenuate the primary and secondary effects of the explosion. However, tertiary blast effects were the predominant mechanism of injury, with severe axial loading to the lower extremity being a characteristic of the fractures seen. The development of future mitigation strategies must focus on reducing all explosion-related injury mechanisms. Integral to this process is an urgent requirement to better understand the behaviour of bone in this unique environment.

Keywords: Forensic science, Explosions, Fractures, Biomechanics, Forensics, Blast injury, Landmines.

1 Introduction

Since World War I, explosive weapons and fragmentation devices have accounted for over 70 per cent of all deaths and injuries to combatants in conflict [1-4]. Survivability from battlefield injuries has increased from 69.7 per cent in World War II to 88.6 per cent most recently in Iraq [5]. This has been attributed to a number of factors including improved torso protection, enhanced pre-hospital care, and rapid aeromedical

evacuation to medical facilities capable of providing optimized damage control, resuscitation and surgery. Consequently, there has been an increased incidence of severely injured casualties surviving with multiple extremity injuries. In Iraq and Afghanistan, extremity injuries comprise 54-68% of combat wounds, of which approximately one third are fractures [6,7]. This has been echoed in literature on civilian blast victims; Frykberg & Tepas [8] reported that 85 per cent of terrorist bombing victims

requiring surgery have soft tissue extremity injuries, with or without fractures.

Our understanding of extremity musculoskeletal injuries is limited by the paucity of blast injury research. Despite musculoskeletal trauma being the most common injury in military conflict and civilian terrorist activity, the study of the blast injury patho-physiology has focused almost exclusively on primary blast injuries of the pulmonary or central nervous systems [9-12], resulting in the

development of improved protective measures and medical interventions. As a consequence, there exists a dearth of scientific investigation into blast pathophysiology of the musculoskeletal system, appropriate injury profiling and subsequent predictive modelling, all fundamentally distinct from that described in blunt trauma research^[13,14]. It is therefore incumbent upon clinicians, scientists and engineers to have a better understanding of underlying injury mechanisms of extremity trauma in order to drive the development of novel treatment and mitigation processes.

Forensic injury biomechanics can be considered to be the scientific field focused on if and how mechanical forces cause disruption to anatomic regions of a body^[15]. Using a forensic injury biomechanical approach to blast injuries, it is possible to deconstruct the complex explosive injury process into its component injurious parts, and determine how they interact and disrupt physiological systems.

Despite the large number of studies reporting the injury profile from explosive events, there have been no attempts to describe the effects of explosion based on the pattern of skeletal injury found. Therefore the aims of this paper are to firstly describe the mechanical and physical processes that result in bone fracture in an explosive environment, secondly to determine which effects of explosion are responsible for bone injury and finally to determine the effects of the casualties' environment on the pattern of bone injury. This will then serve as a template for future research and the development of mitigation through environmental change, or protective clothing.

2 The Physics of Blast and its Effect on Bone

Detonation of an explosive initiates a shock wave process whereby the wave propagates through the explosive, causing an instantaneous (less than 1 ms) chemical reaction. Behind the detonation wave, the explosive has been converted to hot, high pressure gas: the detonation products. Local pressures are typically in the region of 25×10^5 atm while temperatures are from 2,000 to 6,000 °C^[16]. The hot gas expands forcing out the volume it occupies. As a consequence, a layer of compressed air (blast wave) forms in front of this gas volume containing most of the energy released by the explosion.

There is an instantaneous sharp rise in pressure within the air surrounding the explosion, rapidly attaining its peak overpressure. As the blast wave moves through the air, the pressure wave disperses in inverse proportion to the third power of the spherical explosive's radius^[17]. Overexpansion of the detonation products results in the development of a sub-atmospheric pressure phase. In this phase, a partial vacuum is created and air is sucked in. This is also accompanied by high suction winds that carry debris for long

distances away from the explosion source. The classical waveform (Friedlander wave) describes pressure changes from a fixed location relative to the explosive event (figure 1)^[18]. It is idealized because the effects of structures and the ground have been omitted, as they produce multiple reflective waves that distort the waveform.

The physical properties of the blast wave in respect to physiological dysfunction are essentially the peak pressure, the impulse (the time integral of pressure), and the duration of the positive phase overpressure^[19]. Wakeley^[20] commented that a 'high peak overpressure is of little use if not sustained sufficiently long to distort the structure beyond its power of elastic recovery, and a large impulse is of little value if the pressure is less than the structure is able to withstand'. It has also been proposed that the dynamic overpressure of the detonation products (blast wind) and thermal energy released in the explosion contribute to blast injury^[21,22]. By convention, blast injuries are classified according to the mechanism by which they are produced and their effect on the skeletal system is summarized below.

2.1. Primary orthopaedic blast

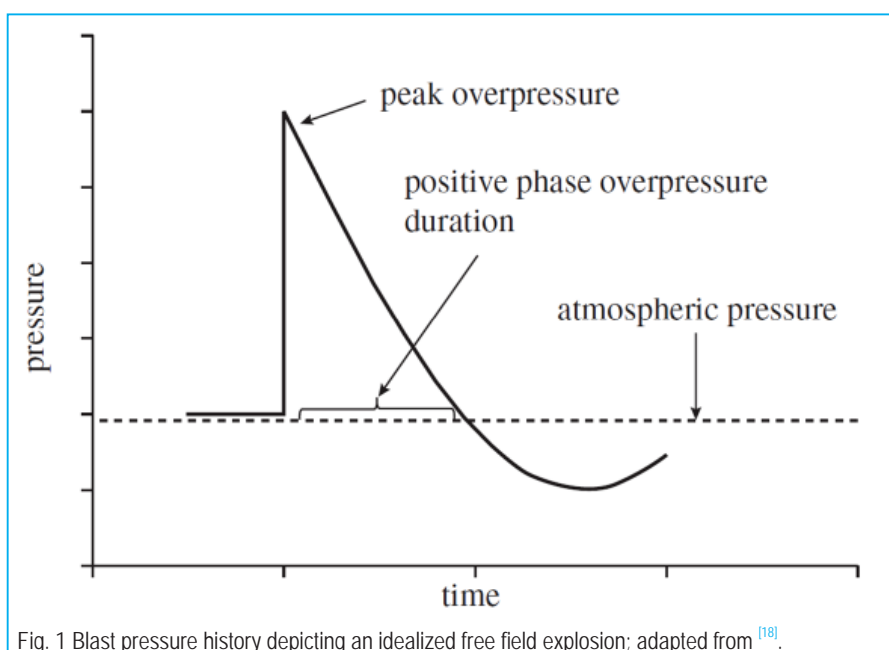


Fig. 1 Blast pressure history depicting an idealized free field explosion; adapted from^[18].

injury

Primary orthopaedic blast injury is related to the effect of the blast wave on skeletal structures. Blast waves, interacting with the body, will transfer energy at interfaces between tissues of differing acoustic impedance. This leads to cellular disruption, soft tissue destruction and bone micro-fractures. Hull^[23] demonstrated that a goat limb, shielded from the effects of the detonation products and fragments, could be fractured by the blast wave alone, when placed in close proximity (less than 50 cm) to the seat of the explosion. Based on finite element analysis, Hull predicted that the stress wave will have been propagated from the explosive to the limb prior to any displacement of the limb. If the blast wave entered the tibia laterally (bomb detonation to the side of the victim), the bending forces exerted by the blast wave, combined with the geometry of the tibia and the differential movement afforded by the knee and ankle joints result in the peak stresses being situated within the upper third. The resulting shear and axial stresses

exceed the tensile failure stress of bone causing fracture. From clinical experience, the proximal third of the tibia and femur are the most common sites for traumatic amputation in these circumstances (figure 2)^[24].

Once the bone is fractured by the blast wave, the detonation products expose the bone to significant bending stresses. It is suggested that these stresses, occurring at the site of blast-wave induced bone fracture, are the probable mechanism of traumatic amputation^[25,26].

Clinically, this manifests as a traumatic amputation, with the proximal stump containing a short oblique or transverse fracture morphology (figure 2b).

2.2. Secondary orthopaedic blast injury

Secondary blast injury is marked by penetrating trauma from bomb casing fragments, from materials implanted within the explosive (e.g. nails, screws), or from local materials energized by proximity to the explosion. These fragments can cause fracture either directly or indirectly. Direct impact of a high

energy fragment into bone typically results in a highly comminuted fracture^[27] (figure 3a). Experimental evidence has shown that these injuries result in multiple bone fragments with no periosteal attachment and thus no blood supply. In addition, these direct high transfer wounds produce significant contamination of the fracture site and into the medullary canal, thereby increasing the risk of developing long-term infective complications (osteomyelitis). In cadaveric studies, Huelke^[27] demonstrated that direct fractures only occurred when steel projectiles weighing 1.05 g were travelling at velocities greater than 185 ms⁻¹, and that the degree of comminution and size of injury increased with velocity.

As a projectile passes through tissue it imparts radial velocity to the surrounding medium, thereby causing a large temporary cavity^[28]. The projectile, after penetrating one bone cortex, encounters the marrow-filled cancellous bone and propels the marrow radially at high velocity, fracturing the thin trabeculae. When the projectile penetrates the second

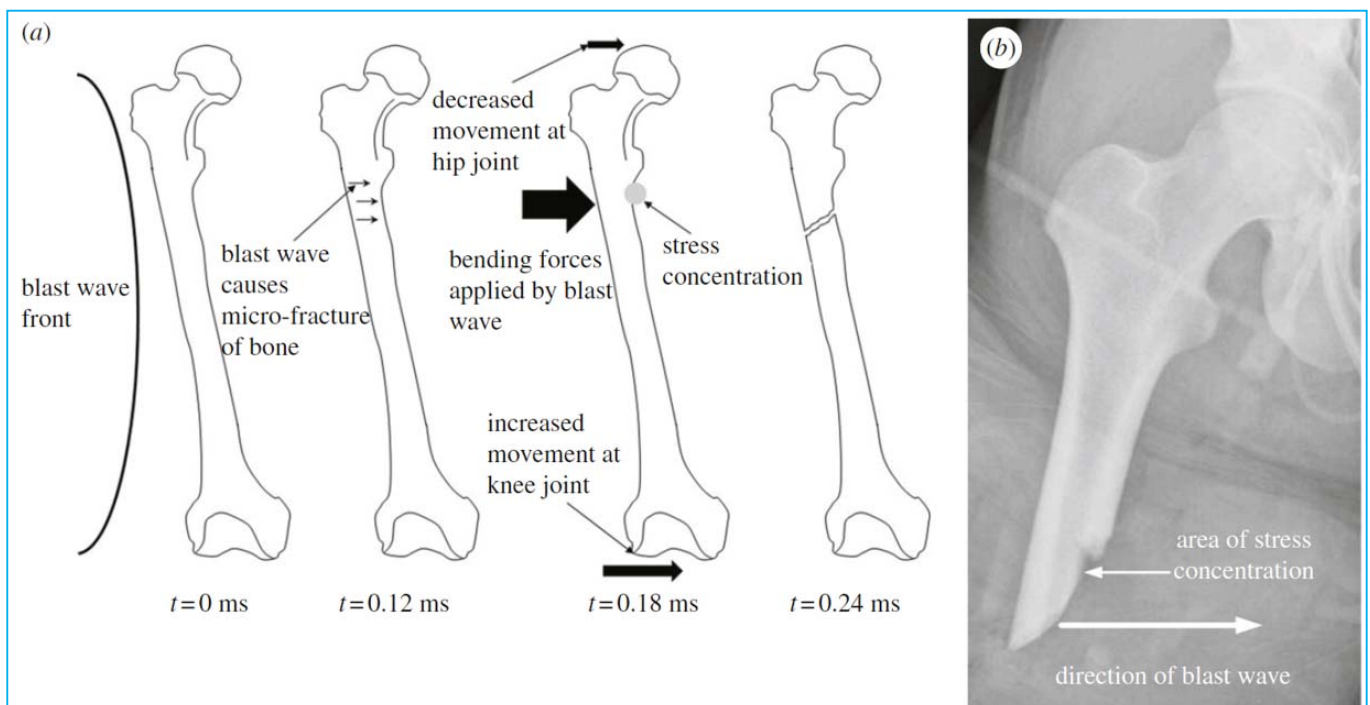


Fig. 2 (a) The blast wave interacts with the femur causing micro-fracture within the bone. Due to the bone geometry and the differential movement allowed by the knee and hip joints, the bending forces exerted on the femur by the blast wave result in an area of stress concentration. The peak hoop and axial stresses within this area exceed the tensile failure stress of bone, resulting in fracture; adapted from^[23]. (b) A traumatic amputation of the femur. Note the absence of significant soft tissue disruption or fragments and the short oblique fracture pattern of the stump.

bone cortex, the exit hole is enlarged by the cavitation in the cancellous bone. Due to the relatively inelastic nature of bone compared with soft tissues, the cavity formed in the cancellous bone does not collapse and a permanent cavity is formed. With higher velocity impacts (more than 500 ms^{-1}) the cavitation phenomenon produces widespread destruction of cancellous bone with increased fragmentation of the cortical bone on the exit hole. A similar effect has been noted with increasing projectile diameter^[27,29]. Hence the size of the cavitation cavity and the relative size of the cortical defects can provide forensic indication as to the size, velocity and direction of the projectile.

If the fragment is travelling at a slower velocity, full penetration of the bone does not occur and only a single cortex is breached. In these cases, the classical 'drillhole' fracture is produced (figure 3b). Clinically, these injuries have a good prognosis and do not require surgical reconstruction. Rose et al.^[30] reported 12 cases of drill-hole fractures of the femur treated conservatively with no complications.

Indirect fractures can be caused by a high energy fragment passing in close proximity to bone^[28]. Such injuries are caused by the high pressures exerted on the bone surface by the leading edge of the rapidly expanding temporary cavity^[31]. The fractures show no bone loss and the fragments retain periosteal attachments and are therefore likely to remain viable. The fracture configuration in these injuries is usually simple (i.e. transverse or oblique) with little comminution. This is analogous to primary blast injuries.

2.3. Mixed primary and secondary orthopaedic blast injury

If the casualty is located at the seat of the explosion, the effects of the shock wave and the

detonation products occur almost instantaneously. This classically occurs upon detonation of an anti-personnel mine. The antipersonnel mine is designed to release a large amount of explosive energy at a short

range, aiming to maim rather than kill. Upon detonation, the blast wave is transmitted directly into the limb causing a brisance (shattering) effect on bone (figure 4). This occurs within 200 ms of mine detonation. One or



Fig. 3 Radiographs depicting the features of secondary orthopaedic blast injury: (a) highly comminuted fracture from direct impact with a high energy fragment; (b) incomplete 'drill-hole' fracture from impact with a low energy fragment.

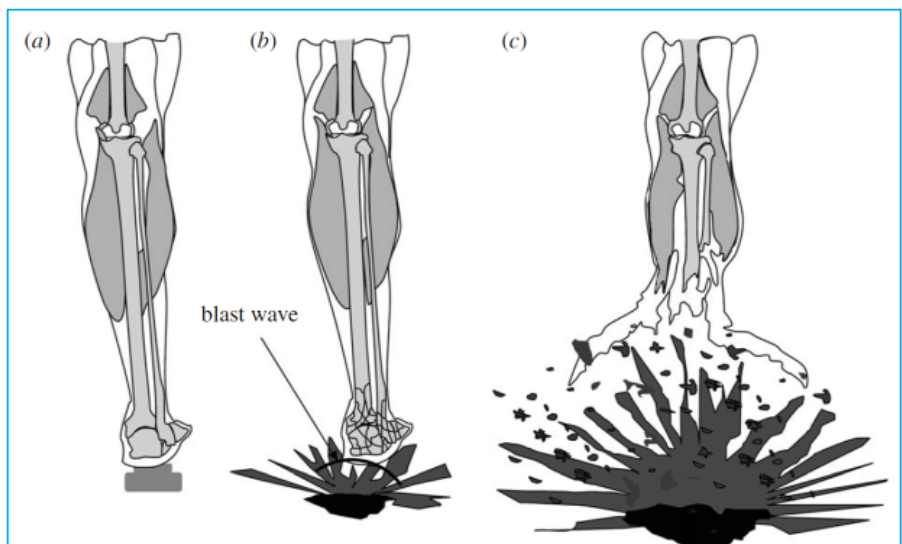


Fig. 4 Upon detonation of an anti-personnel mine (a), a blast wave is transmitted to the limb causing a brisance effect on the bones (b). Some 1–2 ms after detonation, the detonation products reach the limb and place huge stresses on the already damaged bone resulting in multiple fractures and potentially traumatic amputation of the affected limb (c).

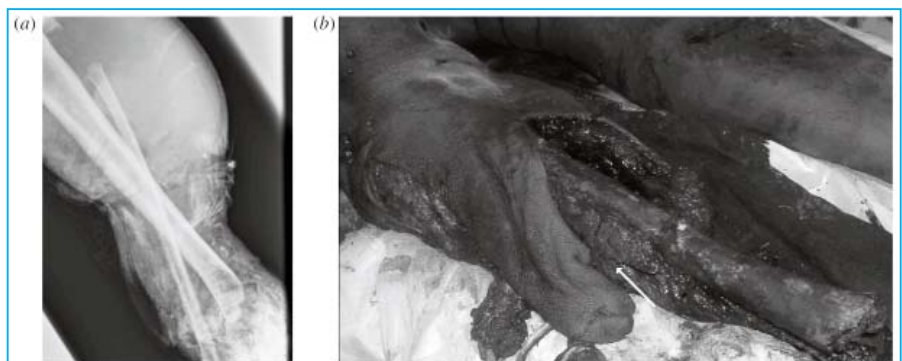


Fig. 5 (a) Radiograph of a combined primary and secondary blast injury to the lower limb following an anti-personnel mine detonation. The close proximity to the seat of the explosion results in the significant amounts of foreign debris seen on the radiograph. (b) The clinical appearance underlines how the detonation products are driven through the fascial planes resulting in the classical umbrella effect of the remaining soft tissues (white arrow).

two milliseconds post-detonation, the detonation products and casing/environmental fragments contact the limb (figure 4) causing destruction of traumatized soft tissue and applying maximal stresses on bone previously damaged by the blast wave [32,33].

The net result is either a total or sub-total amputation of the limb, with the zone of soft tissue injury (including significant amounts of foreign debris and fragments) extending more proximally to the damaged bone (figure 5).

2.4. Tertiary orthopaedic blast injury

Tertiary orthopaedic blast injuries occur as a result of bodily displacement of the casualty or impact against solid structures [34]. As such the injuries witnessed bear similar characteristics to those seen in civilian blunt trauma. When bone is subjected to external loads, local instabilities arise from osseous imperfections. This results in the nucleation, multiplication and growth of micro-cracks, their localization in certain areas, and finally the formation of a

macroscopic fissure (fracture) owing to the coalescence of localized micro-cracks in the most densely damaged area [35]. The pattern of the resulting fracture is a function of the direction and intensity of the load applied, the geometry of the bone injured, and the subject- and location-specific material properties.

Kress et al. [36] reported the results of 588 long bone fractures induced by impacting whole limbs and dissected bones using a pneumatically driven impactor travelling at velocities of 3.5–7.5 ms⁻¹. They reported that with loads applied perpendicular to the axis of the bone, the most common fracture reported was a tension wedge (figure 6a) and that this did not change with the direction of the impact. Tensile wedge fractures originate at a location directly opposite the point of impact and the wedge segment radiates back through the bone initially forming a 90° vertex angle. This suggests failure owing to direct stress, i.e. axial loading of the bone in tension at the far cortex [37]. They also noted that the level of comminution at the fracture

site was related to increasing speed of impact. Spiral fractures only appeared when the bones were subjected to additional torsional loads and these fractures occurred 100 per cent of the time when a pure torsional load was applied (figure 6b). This implies failure owing to shear stress [37] and is directly equivalent to similar fractures seen in metacarpals and phalanges [38].

Severe axial loading of the lower limbs from underground explosions, or casualties landing on their feet after being thrown can also be expected, with comminuted calcaneal (heel) fractures being a prominent feature (figure 7); a pattern of injury similar to parasuicide injuries sustained by falling from significant heights [39,40]. In cadaveric biomechanical testing, Yoganandan et al. [41] demonstrated that axial loads greater than 6.2 kN (approx. 8 times body weight) were sufficient to cause intra-articular calcaneal fractures in 50 per cent of cases.

2.5. The effect of environment on blast injury

The location of the explosion can have a significant effect on both the severity and spectrum of injuries seen following an explosion. Leibovici [42] compared the effects of explosions occurring in open spaces with those in confined spaces. He found that explosions in confined spaces were associated with a higher incidence of primary blast lung injury, increased injury severity and increased severity of burns compared with explosions in open air. Kosashvili [43] reported that explosions occurring in confined environments (e.g. restaurants or transportation) caused the highest number of severe injuries and casualties required the largest number of surgical interventions: open space explosions caused the largest number of casualties but with the smallest percentage of severe injuries or death.

Despite the large number of studies reporting the injury profile

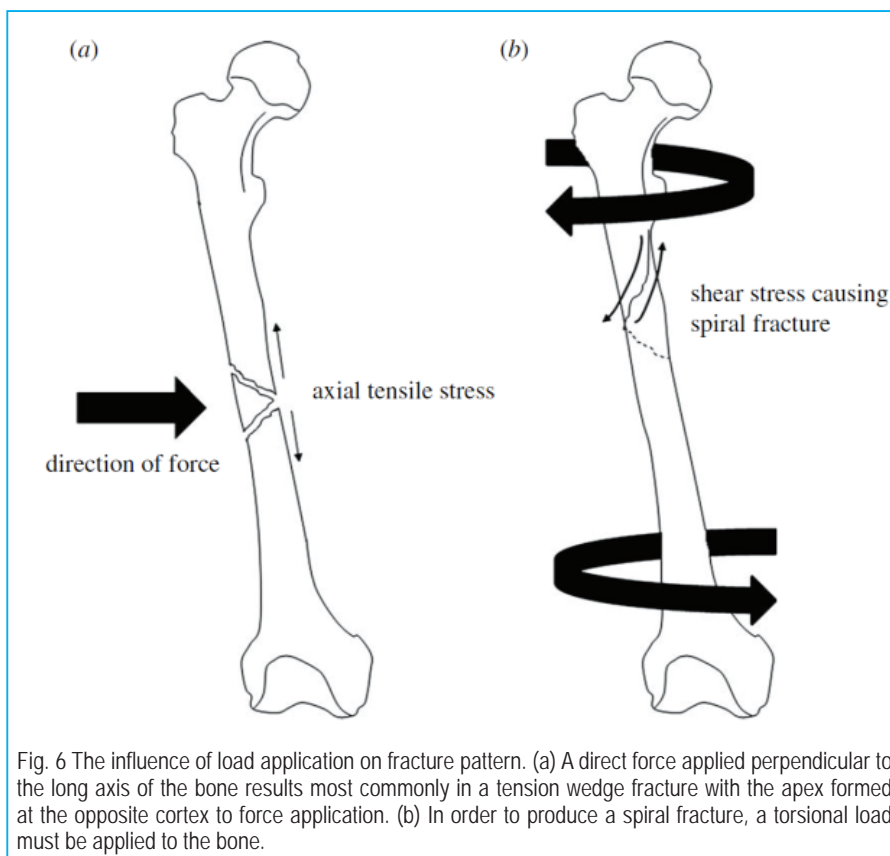


Fig. 6 The influence of load application on fracture pattern. (a) A direct force applied perpendicular to the long axis of the bone results most commonly in a tension wedge fracture with the apex formed at the opposite cortex to force application. (b) In order to produce a spiral fracture, a torsional load must be applied to the bone.

from explosive events, there have been no attempts to describe the effects of explosion in non-fatal casualties based on the pattern of skeletal injury found. Considering that the fracture configurations caused by the individual blast components are very different, our hypothesis is that the pattern of fractures seen in victims of explosions would be dependent upon the incident environment.

Therefore, the specific aims of this study are to firstly determine which effects of the explosion are responsible for combat casualty extremity bone injury in two distinct environments: (i) in the open (open group) and (ii) enclosed space (either in vehicle or in cover, enclosed

group), and secondly to determine whether patterns of combat casualty bone injury differed between environments.

3 Method

We reviewed all Emergency Department records, medical documentation and radiographs of surviving casualties injured by an explosive blast mechanism, presenting to the medical treatment facility situated at Camp Bastion, Helmand Province, southern Afghanistan between April and September 2008. From this review, we identified all casualties (both civilian and security forces) who had sustained

an extremity fracture. Paediatric patients (less than 18 years of age) were excluded from the analysis. Once identified, the radiographs were reviewed independently by a military radiologist, a team of military orthopaedic surgeons and academic biomechanists. Based on the environment of the incident, patients were categorized into two groups: open, free-field, known as the open group; and in vehicle, or in cover, known as the enclosed group.

Due to the complex nature of the fractures recorded, it was evident that individual reporting of single bones would be inappropriate and may significantly skew the results. As such we developed the term 'zone of injury' (ZoI) to describe an anatomical region injured by a particular blast mechanism (figure 8).



Fig. 7 Tertiary blast injury following an improvised explosive device. The comminuted fractured calcaneus (heel, white arrow) is a result of severe axial loading.



Fig. 8 A complex lower limb injury involving 29 bones. In this case the foot and ankle complex was designated a single zone of injury (ZoI) and determined to have been caused by a mixed primary and secondary blast mechanism.

For each ZoI, the anatomical location of injury and blast mechanism (primary, secondary, combined primary and secondary, and tertiary) was determined based upon the fracture characteristics (table 1). Additionally, the presence of an open fracture (i.e. fracture associated with a break in the skin) was also recorded.

The data were analysed using SPSS v. 17.0 (SPSS, USA) statistical software. For categorical data, χ^2 -test was applied. For non-parametric continuous data, the Mann–Whitney test was used and a p-value less than 0.05 was considered statistically significant.

4 Results

We identified 62 casualties who were eligible for inclusion in this study during the study period. In total they sustained 115 ZoIs (1.82 ± 0.98 ; mean number of ZoIs per casualty \pm s.d.). Thirty-four casualties were in the open group and sustained 56 ZoIs (mean 1.65 ZoIs per casualty \pm 1.16); 28 casualties in the enclosed group sustained 59 ZoIs (mean 2.1 ZoIs per casualty \pm 0.81). There was no statistical difference in the mean ZoIs in the open versus enclosed group (Mann–Whitney test, $p = 0.54$). However, there were more open fractures within the open group (48/59) compared with the enclosed group (20/49) (χ^2 -test, $p < 0.001$).

The effect of the environment on the mechanism of injury is presented in figure 9; 27 (43.6%) casualties sustained more than 1 ZoI (range 1–6). Of those, 11 had more than one mechanism of injury attributed to their fractures. Casualties in the open group sustained 1 ZoI from primary blast effect, 10 ZoIs from a combination of primary and secondary effects, 23 ZoIs owing to secondary effects and 24 ZoIs from tertiary effects. Fragmentation was a component in injury causation in 33

(58.9%) injuries. In contrast, tertiary blast effects predominated in the enclosed group, accounting for 57 (96%) ZoIs. In addition, there was no primary or combined primary and secondary blast effect ZoIs in this group, with only two secondary blast ZoIs recorded.

Twenty-seven (43.6%) casualties sustained more than 1 ZoI (range 1–6). Of those 11 had more than one mechanism of injury attributed to their fractures.

The anatomical pattern of injury is represented in figure 10. There was a higher proportion of lower leg injuries in the enclosed group (54/59) compared with the open group (40/58; χ^2 -test, $p < 0.05$).

A sub-group analysis of the mechanism of injury in the lower limb (figure 11a) demonstrates that in the enclosed group tertiary blast effects predominated in 47/48 (96%) ZoIs. In contrast, the open group showed a far more even distribution of injuries resulting from combined primary and secondary, secondary, and tertiary effects. In the upper limb, secondary blast effects predominated overall, affecting 14/23 (61%) ZoIs, with this effect being more pronounced in the open group (figure 11b).

5 Discussion

Our data clearly demonstrate a distinct correlation between the pattern of injury and environment. The enclosed environment afforded by structures appears to mitigate the primary and secondary effects of the explosion. When a detonation occurs close to, but outside a structure the resulting blast wave diffracts around, reflects off, and, to a much lesser extent, transmits into the interior^[44]. Because only a small portion of the blast wave is transferred internally, the risk of blast wave related injuries is substantially reduced. Test data reported by Champion et al.^[13]

illustrated the effect of a 17 kg bare charge of C-4 explosive detonated 3 m away from an armoured vehicle. The peak incident overpressure outside the vehicle was 28 times that inside the vehicle and the impulse was 3 times that inside the vehicle. Although these structures can reduce the effects of primary and secondary blast injury, the momentum imparted by the blast to the structure (vehicle) can cause acceleration and displacement of the occupants, and may in part explain the high proportion of tertiary blast injuries noted in this group.

In contrast, secondary fragments from the explosion were more likely to result in fractures of casualties caught in the open. The development of combat body armour, designed to protect the torso, has been shown to significantly reduce the severity of injury and improve survivability from ballistic trauma^[45]. As a consequence, there has been an increase in survivors with severe penetrating trauma to the extremities without central involvement and this effect may be reflected in the large number of casualties in the open group surviving with extremity fractures caused by high energy fragments.

In this study, data were collected on survivors of explosion. It is conceivable that the injury patterns in fatalities maybe considerably different to those seen in survivors with a higher proportion of primary blast injuries. Data on fatalities during the study period were not available in this study. Mellor^[46] reviewed the post-mortem data of 216 servicemen killed by explosion during the Northern Ireland civil conflict. Due to the availability of accurate incident data, he was able to correlate blast loading to death from explosions. Fifty-one per cent of the fatalities were subjected to peak overpressures greater than 550 kPa and of the 43 fatalities who sustained a traumatic limb amputation, 32 were subjected

to this level of blast loading. Indeed, the presence of a primary blast limb injury in an enclosed environment may be indicative of a breach in the structure and therefore act as a surrogate marker of the intensity of blast loading to which the occupant was subjected. We believe that future evaluation of post-mortem data of blast fatalities, using the methods demonstrated in this study, would help provide further forensic evidence in the evaluation of actual explosive incidents, and the effectiveness of protective measures.

In this study, 43.6 per cent of casualties had more than 1 ZoI, further demonstrating the devastating effects of explosion on the human body. A significant number of those with multiple ZoIs had more than one blast mechanism to account for their injuries. This suggests that the development of future mitigation strategies must be focused on reducing all the different mechanisms of injury caused by an explosion.

Anatomical analysis of the data revealed that the lower limb was more frequently affected in the enclosed group compared with the open group and nearly all of the lower limbs injured in the enclosed group sustained tertiary blast injuries. This may be attributed to the momentum effects of the explosion causing casualties to be thrown long distances before landing on their feet, or secondary to vertical acceleration and local floor-pan deformation from under-vehicle mine detonation^[47]. As this was the predominant mechanism and location of injury in this study, further research is currently being undertaken to fully investigate the biomechanics of lower limb injury in explosions^[14].

The classification system used in this study was not developed to provide a prognostic indicator of overall clinical outcome. In a review of ballistic classification systems,

Rosell & Clasper^[48] commented that a main indicator of outcome will require some form of quantitative assessment of soft tissue injury and concluded that ballistic injuries should be treated on an individual basis, considering the soft tissue, anatomical location of the injury and the involvement of any joints. The use of plain radiographs prohibits the evaluation of soft tissue injury fully, but we believe that this forensic approach can aid the development of future mitigation strategies by identifying the root cause of the injury mechanism. Integral to this process is an urgent requirement to better understand the response of bone in this unique environment. This can only be achieved via a collaborative approach between clinicians, natural scientists and engineers, combining physical and numerical modelling tools with clinical data from explosive incidents.

Reference

- [1] Boyd, N. A. 1975 A military surgical team in Belfast. *Ann. R. Coll. Surg. Engl.* 56, 15–25.
- [2] Melsom, M. A., Farrar, M. D. & Volkers, R. C. 1975 Battle casualties. *Ann. R. Coll. Surg. Engl.* 56, 289–303.
- [3] Palinkas, L. A. & Cohen, P. 1985 Combat casualties among US Marine Corps personnel in Vietnam: 1964–1972. San Diego, CA: Naval Health Research Centre.
- [4] Ramasamy, A., Harrisson, S. E., Stewart, M. P. & Midwinter, M. J. 2009 Penetrating missile injuries during the Iraqi insurgency. *Ann. R. Coll. Surg. Engl.* 91, 551–558. (doi:10.1308/003588409X464720)
- [5] Mazurek, M. T. & Ficke, J. R. 2006 The scope of wounds encountered in casualties from the Global War on Terrorism: from the battlefield to the tertiary treatment facility. *J. Am. Acad. Orthop. Surg.* 14, S18–S23.
- [6] Owens, B. D., Kragh Jr, J. F., Macaitis, J., Svoboda, S. J. & Wenke, J. C. 2007 Characterization of extremity

wounds in Operation Iraqi Freedom and Operation Enduring Freedom. *J. Orthop. Trauma* 21, 254–257. (doi:10.1097/BOT.0b013e31802f78fb)

[7] Ramasamy, A., Harrisson, S., Lasrado, I. & Stewart, M. P. 2009 A review of casualties during the Iraqi insurgency 2006—a British field hospital experience. *Injury* 40, 493–497. (doi:10.1016/j.injury.2008.03.028)

[8] Frykberg, E. R. & Tepas III, J. J. 1988 Terrorist bombings. Lessons learned from Belfast to Beirut. *Ann. Surg.* 208, 569–576. (doi:10.1097/00000658-198811000-00005)

[9] Bowen, I. G., Fletcher, E. R. & Richmond, D. R. 1968 Estimate of man's tolerance to the direct effects of air blast. Washington, DC: Headquarters Defence Atomic Support Agency.

[10] Cooper, G. J., Townend, D. J., Cater, S. R. & Pearce, B. P. 1991 The role of stress waves in thoracic visceral injury from blast loading: modification of stress transmission by foams and high-density materials. *J. Biomech.* 24, 273–285. (doi:10.1016/0021-9290(91)90346-O)

[11] Hayda, R., Harris, R. M. & Bass, C. D. 2004 Blast injury research. *Clin. Orthop. Rel. Res.* 422, 97–108. (doi:10.1097/01.blo.0000128295.28666.ee)

[12] Zuckerman, S. 1952 Vulnerability of human targets to fragmenting and blast weapons. *Textbook of air armament*, pp. 1–243. London, UK: HMSO.

[13] Champion, H. R., Holcomb, J. B. & Young, L. A. 2009 Injuries from explosions: physics, biophysics, pathology and required research focus. *J. Trauma* 66, 1468–1477. (doi:10.1097/TA.0b013e3181a27e7f)

[14] Ramasamy, A., Masouros, S. D., Newell, N., Hill, A. M., Proud, W. G., Brown, K. A., Bull, A. M. J. & Clasper, J. C. In press. In-vehicle extremity injuries from improvised explosive devices: current and future foci. *Phil. Trans. R. Soc. B* 366. (doi:10.1098/rstb.2010.0219)

[15] Hayes, W. C., Erickson, M. S. & Power, E. D. 2007 Forensic injury biomechanics. *Annu. Rev. Biomed. Eng.* 9, 55–86. (doi:10.1146/annurev.

- bioeng.9.060906.151946)
- [16] Baker, W. 1983 Explosion hazards and evaluation. Amsterdam, The Netherlands: Elsevier.
- [17] Dewey, J. M. 1964 The air velocity in blast waves from T.N.T. explosions. *Proc. R. Soc. Lond. A* 279, 366–385. (doi:10.1098/rspa.1964.0110)
- [18] Friedlander, F. G. 1946 The diffraction of sound pulses. I. Diffraction by a semi-infinite plane. *Proc. R. Soc. Lond. A* 186, 322–344. (doi:10.1098/rspa.1946.0046)
- [19] Clemedson, J. 1956 Blast injury. *Physiol. Rev.* 36, 336–354.
- [20] Wakeley, C. P. G. 1945 Effect of underwater explosion on the human body. *Lancet* 245, 715–718. (doi:10.1016/S0140-6736(45)90478-8)
- [21] Cullis, I. G. 2001 Blast waves and how they interact with structures. *J. R. Army Med. Corps* 147, 16–26.
- [22] Horrocks, C. L. 2001 Blast injuries: biophysics, pathophysiology and management principles. *J. R. Army Med. Corps* 147, 28–40.
- [23] Hull, J. B. 1995 An investigation into the mechanism of traumatic amputation by explosive blast. Doctor of medicine thesis, University of Birmingham.
- [24] Hull, J. B. & Cooper, G. J. 1996 Pattern and mechanism of traumatic amputation by explosive blast. *J. Trauma* 40, S198–S205. (doi:10.1097/00005373-199603001-00044)
- [25] Hull, J. B. 1992 Traumatic amputation by explosive blast: pattern of injury in survivors. *Br. J. Surg.* 79, 1303–1306. (doi:10.1002/bjs.1800791220)
- [26] Hull, J. B., Bowyer, G. W., Cooper, G. J. & Crane, J. 1994 Pattern of injury in those dying from traumatic amputation caused by bomb blast. *Br. J. Surg.* 81, 1132–1135. (doi:10.1002/bjs.1800810815)
- [27] Huelke, D. F., Harger, J. H., Buege, L. J., Dingman, H. G. & Harger, D. R. 1968 An experimental study in bio-ballistics: femoral fractures produced by projectiles. *J. Biomech.* 1, 97–105. (doi:10.1016/0021-9290(68)90012-2)
- [28] Callender, G. R. & French, R. W. 1935 Wound ballistics: studies in the mechanism of wound production by rifle bullets. *Milit. Surg.* 77, 177–201.
- [29] Huelke, D. F., Buege, L. J. & Harger, J. H. 1967 Bone fractures produced by high velocity impacts. *Am. J. Anat.* 120, 123–132. (doi:10.1002/aja.1001200110)
- [30] Rose, S. C., Fujisaki, C. K. & Moore, E. E. 1988 Incomplete fractures associated with penetrating trauma: etiology, appearance and natural history. *J. Trauma* 28, 106–109. (doi:10.1097/00005373-198801000-00016)
- [31] McMillen, J. H. 1945 Shock wave pressures in water produced by impact of small spheres. *Phys. Rev.* 68, 198–209. (doi:10.1103/PhysRev.68.198)
- [32] Nechaev, E. A., Gritsanov, A. I., Fomin, N. F. & Minnullin, I. P. 1984 Mine blast trauma. St Petersburg, Russia: Russian Federation Ministry of Public Health and Medical Industry.
- [33] Trimble, K. & Clasper, J. C. 2001 Anti-personnel mine injury; mechanism and medical management. *J. R. Army Med. Corps* 147, 73–79.
- [34] Bowen, I. G., Albright, R. W., Fletcher, E. R. & White, C. S. 1961 CEX-58.9. A model designed to predict the motion of objects translated by classical blast waves. Oak Ridge, TN: Department of Commerce.
- [35] Zioupos, P., Hansen, U. & Currey, J. D. 2008 Microcracking damage and the fracture process in relation to strain rate in human cortical bone tensile failure. *J. Biomech.* 41, 2932–2939. (doi:10.1016/j.jbiomech.2008.07.025)
- [36] Kress, T. A., Porta, D. J., Snider, J. N., Fuller, P. M., Psihogios, J. P., Heck, W. L., Frick, S. J. & Wasserman, J. F. 1995 Fracture patterns of human cadaver long bones. IRCOBI. Brunnen, Switzerland: IRCOBI.
- [37] Benham, P. P. & Crawford, R. J. 1987 Mechanics of engineering materials. Harlow, UK: Longman Scientific & Technical.
- [38] Kozin, S. H., Thoder, J. J. & Lieberman, G. 2000 Operative treatment of metacarpal and phalangeal shaft fractures. *J. Am. Acad. Orthop. Surg.* 8, 111–121.
- [39] Galloway, A. & Zephro, L. 2007 Skeletal trauma analysis of the lower extremity. In *Forensic medicine of the lower extremity* (eds J. Rich, D. E. Dean & R. H. Powers). New York, NY: Humana Press.
- [40] Scalea, T., Goldstein, A., Phillips, T., Sclafani, S. J. A., Panetta, T., McAuley, J. & Shaftan, G. 1986 An analysis of 161 falls from a height: the ‘jumper syndrome’. *J. Trauma* 26, 706–712. (doi:10.1097/00005373-198608000-00005)
- [41] Yoganandan, N., Pintar, F. A., Kumaresan, S. & Boynton, M. 1997 Axial impact biomechanics of the human foot–ankle complex. *J. Biomech. Eng.* 119, 433–437. (doi:10.1115/1.2798290)
- [42] Leibovici, D. 1996 Blast injuries: bus versus open-air bombings—a comparative study of injuries in survivors of open-air versus confined-space explosions. *J. Trauma* 41, 1030–1035. (doi:10.1097/00005373-199612000-00015)
- [43] Kosashvili, Y., Loebenberg, M. I., Lin, G., Peleg, K., Zvi, F., Kluger, Y. & Blumenfeld, A. 2009 Medical consequences of suicide bombing mass casualty incidents: the impact of explosion setting on injury patterns. *Injury* 40, 698–702. (doi:10.1016/j.injury.2008.06.037)
- [44] Ngo, T., Mendis, P., Gupta, A. & Ramsay, J. 2007 Blast loading and blast effects on structures—an overview. *ESME* 7, 76–91.
- [45] Peleg, K., Rivkind, A. & Aharonson-Daniel, L. 2006 Does body armor protect from firearm injuries? *J. Am. Coll. Surg.* 202, 643–648. (doi:10.1016/j.jamcollsurg.2005.12.019)
- [46] Mellor, S. G. 1992 The relationship of blast loading to death and injury from explosion. *World J. Surg.* 16, 893–898. (doi:10.1007/BF02066988)
- [47] Ramasamy, A., Hill, A. M., Hepper, A. E., Bull, A. M. J. & Clasper, J. C. 2009 Blast mines: a background for clinicians on physics, injury mechanisms and vehicle protection. *J. R. Army Med. Corps* 155, 258–264.
- [48] Rosell, P. A. E. & Clasper, J. C. 2005 Ballistic fractures—the limited value of existing classifications. *Injury* 36, 369–372. (doi:10.1016/j.injury.2004.10.023) ■