(i) An overview of the pathophysiology of blast injury with management guidelines

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Abstract

Explosive weapons remain the leading cause of death, injury, and disability to combatants in battle. Recent conflicts in Iraq and Afghanistan have seen considerable advances in the surgical knowledge and skills needed to save life and limb of multiply injured casualties. Global terrorism has seen explosive weapons move from battlefield to urban centres, often with devastating effects.

Orthopaedic training prepares for the management of general civilian trauma scenarios, but blast injury pathophysiology and management is rarely considered. It is important that future trauma surgeons have a working knowledge of blast injury and how it affects the musculoskeletal system so that they can manage such patients.

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Introduction

Explosive devices account for the majority of deaths and injuries in combat.[1] The conflicts in Iraq and Afghanistan have seen significant medical and logistical advances, including improvements in vehicles, body armour, point of wounding care and evacuation. The consequences of these changes include an unprecedented number of survivors, with multiple and often complex extremity injuries.

Recent world events have highlighted that civilian surgeons in densely populated urban areas, can be expected to manage these complex injuries in multiple casualty situations.

What is blast?

An explosive is a material capable of producing an explosion using its own energy. Upon detonation the explosive is converted into a hot (up to 6000 °C), high pressure (35×10^6 pounds per square inch) gas called the detonation products[1] and a shock wave is propagated. As the detonation products rapidly expand, air is compressed (the blast wave) in front of the expanding gas volume, which contains the majority of the explosive's energy.

The blast wave energy dissipates in an inverse proportion to the third power of the distance from the detonation point.[2] The detonation products over-expand, leading to a sub-atmospheric pressure phase after which air is drawn back in. The resultant turbulence energizes debris into projectiles. The classical waveform (Friedlander wave) describes pressure changes at a fixed location relative to the explosive event in free field conditions (Figure 1).[3]

The components of the blast wave that are responsible for the pathophysiological effects on biological tissue are the amplitude of the peak pressure, the impulse (the time integral of pressure), and the duration of the positive phase overpressure. 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.[4,5]

Blast injuries can classified according to the mechanism by which they are produced and these are summarized below[6] (Table 1).

Primary orthopaedic blast effects

Blast waves, interacting with the body, transfer energy at interfaces between tissues of differing acoustic impedance. Hull (1992)[8] demonstrated that a goat limb, shielded from the effects of the detonation products, could be fractured by the blast wave alone when placed in close proximity to the point of detonation (seat) of the explosion. Using finite element modelling techniques, he predicted that a blast wave will reach the limb prior to any effect caused by the detonation products. If a blast wave penetrates a tibia from a lateral trajectory, the bending forces exerted, interacting with the geometry of the tibia, result in peak stresses being situated within the proximal third of the bone leading to fracture (Figure 2). This echoes clinical experience, where the most common site for traumatic amputation in these circumstances is the proximal tibia.[9]

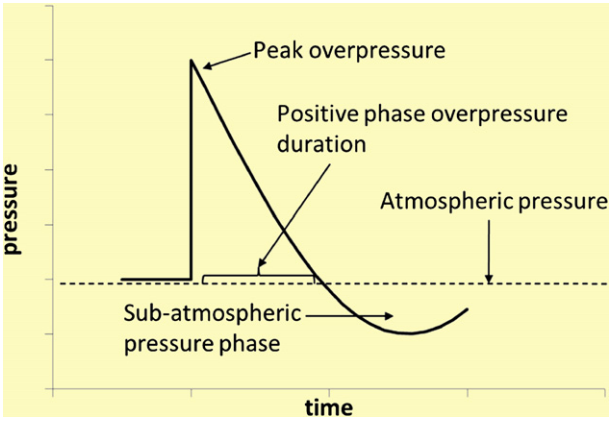

Figure 1 Blast overpressure plot.

Secondary orthopaedic blast effects

Secondary blast injury is caused by penetrating trauma from materials within the explosive, fragments from its casing or debris energized by the explosion. These projectiles can cause fracture either directly or indirectly. High-energy fragments colliding with bone typically result in a highly comminuted fracture with extensive periosteal stripping. In addition, these high-energy transfer wounds are associated with significant contamination which, in conjunction with avascular bone, increases the risk of long-term infective complications. This has been reflected in military studies, for example Brown et al. (2010)[10] reported a 24% infection rate in a review of long bone fractures.

Indirect fractures can be caused by a high-energy fragment passing in close proximity to bone.[11] Such injuries occur due to the leading edge of a rapidly expanding temporary cavity (see below) causing high pressure on the bone surface.[12] These fractures show little or no bone loss or periosteal stripping and are therefore likely to remain viable. The fracture configurations in these injuries are usually simple (i.e. transverse or oblique) with little comminution.

Secondary soft tissue blast effects

A propelled fragment colliding with the body directly damages soft tissue in its path and, if sufficiently energized, generates a radial high pressure compression wave in the tissues just as an explosion does in the atmosphere, as described above. The wave creates a temporary cavity of sub-atmospheric pressure as the fragment traverses, which pulls in external debris, increasing the risk of wound contamination.

The sizes of the temporary and permanent cavities are determined by the kinetic energy of the causative fragment and the nature of the tissue through which it passes.[13] Areas of devitalized tissue can extend several centimetres and the zone of injury is often much greater than the remaining wound tract (Figure 3). Additionally, the irregular morphology of shrapnel in comparison to a uniform bullet, increases the transfer of kinetic energy to surrounding tissues, thereby inducing greater damage.[14] As a consequence, simple surgical debridement of the wound track may not be sufficient to remove all non-viable tissue.

Mixed primary and secondary orthopaedic blast effects

If the victim is situated close to the seat of the explosion, the effects of the blast wave and detonation products occur almost instantaneously. Once bone has fractured due to the blast wave impact, the detonation products expose surrounding tissue to continuing destructive forces.[15,16] Hull (1992)[17] suggested that these forces are the likely mechanism of traumatic amputation. The net result is either a total or sub-total amputation, with the zone of injury (including foreign debris and fragments) extending proximal to the fracture site.

Mixed primary and secondary soft tissue effects

Nechaev et al. (1994),[16] described three major zones of injury following a mine blast, based on histological studies of combat casualties during the Soviet occupation of Afghanistan and on animal models (Figure 4).

Zone I, closest to the seat of the explosion, is characterized by traumatic amputation with widespread destruction of all tissues. There is significant contamination and, based on the degree of soft tissue injury, surgical amputations performed through Zone I are considered non-viable.

Within Zone II, arteriograms performed in animal studies demonstrate that there is persistent impairment of blood flow. Of note, focal areas of damage are seen, localized near neurovascular bundles and osteofascial planes, suggesting that the transmission of the blast wave is through these structures.

Blast injury classification and clinical manifestations in the musculoskeletal system. From Ramasamy et al. (2010)[7]

Blast injury	Mechanism of injury	Clinical manifestations
Primary blast effects	Blast wave	Primary blast lung, gastrointestinal injury, soft tissue deformation and traumatic amputation
Secondary blast effects	Fragments from explosive device and energized debris	Penetrating wounds to the torso and extremities resulting in significant soft tissue injuries and fractures
Combined primary and secondary blast effects	Combination of primary and secondary injuries when victim is near the seat of the explosion	Massive soft tissue injury and sub-total/traumatic amputation of the limb
Tertiary blast effects	Acceleration and deceleration injuries to spine and extremities. Crush phenomena	Fractures from impact with solid objects. Soft tissue crush injuries leading to compartment syndrome and nerve injury
Quaternary blast effects	Thermal injuries and others	Burns

Table 1