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Review Article

Primary blast lung injury - a review

T.E. Scott[1], E. Kirkman[2], M. Haque[3], I. E. Gibb[4], P. Mahoney[5] and J.G. Hardman[6]

[1]Academic Department of Military Anaesthesia and Critical Care, Royal Centre for Defence Medicine, ICT Centre, Birmingham, B15 2SQ, UK, [2]Defence Science and Technology Laboratories, Porton Down, Salisbury, SP4 0JQ, UK, [3]Anaesthesia & Critical Care Research Group, Division of Clinical Neuroscience, Nottingham University Hospital, Queens Medical Centre, Nottingham, NG7 2UH, UK, [4]Room MBF4, Main Building, Institute of Naval Medicine, Crescent Road, Alverstoke, PO12 2DL, UK, [5]Academic Department of Military Anaesthesia and Critical Care, Royal Centre for Defence Medicine, ICT Centre, Birmingham, B15 2SQ, UK and [6]Anaesthesia & Critical Care Research Group, Division of Clinical Neuroscience, Nottingham University Hospital, Queens Medical Centre, Nottingham, NG7 2UH, UK

Abstract

Bomb or explosion-blast injuries are likely to be increasingly encountered as terrorist activity increases and pre-hospital medical care improves. We therefore reviewed the epidemiology, pathophysiology and treatment of primary blast lung injury.

In addition to contemporary military publications and expert recommendation, an EMBASE and MEDLINE search of English speaking journals was undertaken using the medical subject headings (MeSHs) 'blast injury' and 'lung injury'. Review articles, retrospective case series, and controlled animal modelling studies published since 2000 were evaluated.

6-11% of military casualties in recent conflicts have suffered primary blast lung injury but the incidence increases to more than 90% in terrorist attacks occurring in enclosed spaces such as trains. The majority of victims require mechanical ventilation and intensive care management. Specific therapies do not exist and treatment is supportive utilizing current best practice.

Understanding the consequences and supportive therapies available to treat primary blast lung injury are important for anaesthetists.

Key words: blast injuries; explosions; lung injury

Once the preserve of the military physician, primary blast injury will increasingly be seen by civilian medical practitioners as industrialisation of developing economies progresses, and also as terrorist activity continues to increase significantly.[1] Primary blast injury syndrome is a potentially life threatening, multi-system disease, of which primary blast lung injury (PBLI) is a fundamental component. It results from exposure to blast over-pressure after an explosion. Whilst responsible for significant numbers of immediate fatalities, improving personal protection in the military context means that more survivors of blast exposure are presenting to the medical services with the condition.[2] Though a vast amount of blast injury animal research has been undertaken since the Second World War, this has predominantly been concerned with delineating either safe or lethal exposure limits. Despite this research, the mechanism of primary blast injury at the cellular level remains mainly speculative, though gross cardiovascular and lung injury patterns are well recognized. Most casualties with PBLI require management in a critical care environment but the sporadic nature of the disease means that it is unlikely that a controlled evidence base will ever guide this care. Crude non-animal models exist, none of these however replicate the complex and multifaceted biological response to blast exposure, and none are based on human data.

Editor's Key Points

- Aftermaths of terrorist activity are likely to impact many health professionals.
- Explosions produce high energy pressure waves and extreme heat
- Bomb-blast injuries are often lethal but in survivors there is often haemorrhagic shock and multi-organ failure.
- High-pressure injury to the lung results in acute lung injury.

Explosives and blast waves

An explosion occurs when "energy is released over a sufficiently small time and in a sufficiently small volume so as to generate a pressure wave of finite amplitude travelling away from the source".[3] An explosive is any substance that can undergo exothermic oxidation turning from a solid or liquid into a gas very quickly using its own energy. All explosives are a mixture of an oxidizing agent, a fuel and an initiator.[4] In "low order" explosives such as gunpowder, these are mixed as separate powders and burns at a rate below the speed of sound in a process known as deflagration. They produce gaseous products that will only explode if confined (e.g. pipe or pressure cooker bomb). "High-order" explosives are chemical substances which contain the fuel and oxidiser within the same chemical compound. They do not burn but detonate when a shock wave passes through the material producing large pressure and temperature gradients. The detonation wave travelling through such explosive material will propagate at some 8,000 m s^{-1}, reach pressures of up to 250,000 atmospheres and temperatures of up to 7,000 °C.[5] Such a detonation generates a shock wave of very high pressure in the surrounding air that radiates away from the source supersonically as the explosive shock wave. Blast waves are described as being either simple or complicated in nature.[6] A simple (or 'Friedlander') blast wave occurs when a spherical high-explosive detonates whilst suspended in an open-air environment. It is characterised by an instantaneous increase in pressure to a peak value followed by an exponential decrease in pressure to below atmospheric levels, creating a negative pressure phase (Fig. 1). The energy from such an explosion is dissipated in three dimensions and thus deteriorates as the inverse cube as a function of distance from the centre. As such this "stand-off" distance is a fundamental factor in determining the likelihood of suffering primary blast injury. Depending on the explosive power, circumferential zones of injury pattern will be created. In order of proximity this will be; non-survivability, risk of primary blast injury (and other injury patterns) and then other injury patterns only.

Complex waves are generated when the detonation occurs in the presence of reflecting surfaces such as within buildings[1] or vehicles (Fig. 2). They are the product of reflected waves enhancing each other and the original shock wave in a directly additive effect. This then results in a significantly greater positive pressure phase (up to a 20-fold increase) and so greater injury. The chaotic nature of complex waves in confined spaces means that the stand-off distance becomes less important. The shock wave is then followed by the blast wind, a high-speed body of gas moving away from the explosion at up to 2,000 km h^{-1}. This may be followed by a release wave. This is a flow of gas back towards the epicentre as the vacuum created by the blast wave is filled with now non-energised gas particles. The combination of the shock wave and blast wind is known as the blast wave.

Most explosions are short duration with a positive pressure phase of 10 milliseconds or less.[7] Injury resulting from such explosions is related to the blast impulse, a function of pressure multiplied by time. Examples of long duration blast waves would be nuclear or volcanic explosions or explosions caused by specialised blast (thermobaric) weaponry.

Primary blast lung injury

PBLI is defined as "radiological and clinical evidence of acute lung injury occurring within 12 h of exposure and not due to secondary or tertiary injury".[8] The nomenclature of other blast injury patterns after an explosion is outlined in Table 1. This classification does not recognise the significant psychological injury suffered by witnesses and first responders to explosive events, which should perhaps be regarded as a quinternary injury pattern. In reality, the different blast injury mechanisms will rarely exist in isolation and a single casualty will suffer a spectrum of blast injury mechanisms. Secondary blast injury (fragmentation) predominates in most recent conflicts.[9]

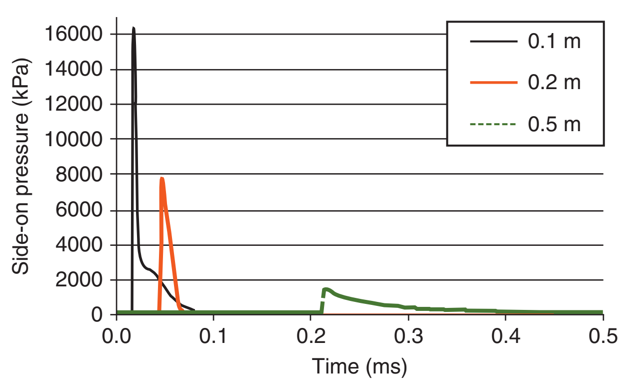

Figure 1 Simple wave. Blast over-pressure at different distances from a free field explosion generated by a 160g spherical mass of TNT. A negative pressure phase is not demonstrated in this example. Courtesy of Professor Ian Horsfall, The Defence Academy, UK.