

Primary blast injury

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Primary blast injuries result from the overpressure and are, as such, unique to blast. The effect of blast overpressure is most marked at the interface between air and tissue or liquid. Tympanic membrane (TM) rupture is the most common primary blast injury. Patients may be asymptomatic or have a degree of transient hearing loss and otorrhoea. Previously, the presence of TM rupture was used as a marker for other occult blast injuries. This has been challenged recently by findings that show TM injury is not ubiquitous in the presence of more severe primary blast injury. The blast environment and orientation of the ear canal to the shockwave are likely to determine the chance of injury. researched blast phenomenon. The exact mechanism of blast lung remains contested but it depends on the propagation of energy from the shockwave into the lung tissue, where it causes disruption. Proposed mechanisms of injury include spalling (disruption of tissues at air-liquid interfaces), implosion (compression and re-expansion of air-filled structures) and rapid acceleration of tissues of different densities. Large animal models have demonstrated that the level of injury is related to the rate of chest wall displacement, rather than the maximal depth of deflection. The severity of injury is dependent on the strength of the blast, the range from detonation and the surrounding environment. Those working closely with explosives may wear personal armour that assists in decoupling the effect of the primary blast. Current examples of such armour tend to be cumbersome. More common varieties of torso body armour provide protection against penetrating injury but probably do little to mitigate the effects of primary blast. The pathophysiology of blast lung includes both immediate and delayed responses. There is an immediate bradycardia and apnoea of variable length, which is likely to be a vagally mediated reflex. The lung injury itself is typified by alveolar capillary rupture with subsequent intrapulmonary bleeding and oedema. The extent of this injury is proportional to the blast exposure and may range from microscopic petechial injury to areas of frank haemorrhage. While rarely seen in isolation, primary blast may leave little external evidence of injury since the skin itself is rarely affected. Clinical features of blast lung include progressive hypoxia, which may not be apparent at the time of injury and is related to the inflammatory response to intrapulmonary haemorrhage and worsening oedema. Other structural lung injuries are associated with primary blast, although the prediction of these injuries by blast conditions is not consistent and is likely to be complicated by tertiary impact. Pneumothoraces may occur as a result of pleural rupture in the absence of penetrating injury. Injury to the larger vessels may lead to haemothoraces and the formation of alveolar-venous or bronchovenous fistulae. Air embolism due to such fistulae may cause acute hypoxia with cardiovascular collapse and is a leading cause of death in those who do not survive until treatment. The diagnosis of blast lung is clinical with findings of hypoxia following blast exposure. Typical 'bat-wing' pulmonary infiltrates are seen on the chest radiograph and computed tomography may discriminate these injuries from the more peripheral contusions seen in blunt trauma. Imaging may be useful in detecting associated structural lung injuries. Treatment of blast lung is largely supportive. Mechanical ventilation may be required but consideration should be given to the possibility of air embolism and pneumothorax that may be exacerbated. Some

centres advocate the use of prophylactic bilateral pleural decompression, although there is little evidence to suggest an effect on outcomes. Patients with significant blast lung injury are highly likely to have sustained other blast-related injuries and any management plan should consider their overall condition.

TABLE 34.3 Classification of blast injuries.

Classification	Injury type	Examples
Primary blast	Overpressure	Tympanic membrane injury, blast lung, intestinal blast injury
Secondary blast	Penetrating/ All penetrating injuries	fragmentation
Tertiary blast	Blunt	Blunt and crush injuries, traumatic amputation
Quaternary blast	Miscellaneous	Burns, inhalation injury
Effect of device	Radiation	sickness, additions infection

incidence of abdominal injury due to air blast has not been extensively examined, although a recent review of multiple incidents, including a variety of blast conditions, showed that abdominal injury is not common – seen in around 3% of incidents. Damage is dependent on coupling of the blast overpressure and the shockwave to a stress wave that travels through the abdomen. The effect of shockwave dispersal is most marked at tissue–air interfaces. As such, the hollow organs are those most commonly injured. The caecum is probably most sensitive to intestinal blast injury. Conversely, the small bowel and its extensive mesentery may be more susceptible to large shear waves causing mesenteric tearing. The presentation of primary blast injury to the bowel may be delayed relative to the acute onset of blast lung. Abdominal symptoms may be absent initially with progression to pain and frank peritonitis, should perforation and contamination occur. Given the lack of external injury, indications for operative intervention are largely clinical, as with conventional blunt abdominal trauma. The patient should be assessed anaesthetically with particular consideration to the effect of anaesthesia and ventilation on any concomitant blast lung injury. The most common operative finding of intestinal blast is subserosal haemorrhage. Tearing of the mucosal surface with bleeding into the lumen of the tract may occur following repeated exposure to relatively lower blast overpressures. As in blunt injury, there is a propensity for mural haematomas to progress to perforation as a result of tissue necrosis. Full-thickness injuries to the bowel with immediate perforation can occur with a greater exposure to blast overpressure. The surgical management of blast bowel injury is that of any penetrating and blunt trauma, with primary repair or resection as indicated. Surgical judgement is essential regarding the findings of non-perforated but contused bowel. A damage control approach to such injuries may allow for repeat assessment later, but in a physiologically well patient, in whom a relook would not be justified, the surgeon must decide to resect a segment or risk future progression of these lesions to perforation. The solid organs are more resistant to primary blast. Parenchymal disruption due to the shockwave has been described at very high levels of overpressure, although the experimental data for these injuries are sparse. Injury, with subsequent bleeding, may result from rapid distractions of organ attachments and mesenteries. These blast conditions are more likely to be encountered in enclosed conditions and differentiation of primary from tertiary injuries is difficult. Primary blast injury

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