

# Blast injuries



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Health-care providers are increasingly faced with the possibility of needing to care for people injured in explosions, but can often, however, feel undertrained for the unique aspects of the patient's presentation and management. Although most blast-related injuries (eg, fragmentation injuries from improvised explosive devices and standard military explosives) can be managed in a similar manner to typical penetrating or blunt traumatic injuries, injuries caused by the blast pressure wave itself cannot. The blast pressure wave exerts forces mainly at air-tissue interfaces within the body, and the pulmonary, gastrointestinal, and auditory systems are at greatest risk. Arterial air emboli arising from severe pulmonary injury can cause ischaemic complications—especially in the brain, heart, and intestinal tract. Attributable, in part, to the scene chaos that undoubtedly exists, poor triage and missed diagnosis of blast injuries are substantial concerns because injuries can be subtle or their presentation can be delayed. Management of these injuries can be a challenge, compounded by potentially conflicting treatment goals. This Seminar aims to provide a thorough overview of these unique primary blast injuries and their management.

## Introduction

Blast injuries are physically and psychologically devastating. Although explosions can result from industrial or recreational accidents, terrorist acts that cause injury in military and civilian settings are taking place at an increasing rate. Conservative estimates show that these events have risen four-fold from 1999, to 2006, worldwide, and injuries related to these acts have increased eight-fold (figure 1).<sup>1</sup> Historically, civilian care providers and the health-care systems in which they work have been largely spared from managing patients injured by explosions because most blast-related injuries happen in combat settings. However, nowadays this is not the case. Special-interest, militant, and extremist groups have realised the profound effect explosions can have in civilian settings. Nightclubs, trains, subways, planes, and other popular sites have been targeted in recent years by these groups and caused substantial civilian casualties.<sup>2-6</sup> Health-care systems must be able to provide care for the people and communities that are affected. Thus, every physician involved with emergency care needs to understand the unique injury patterns and management of people injured by an explosion. This Seminar provides an overview of explosion physics, types of explosives, and mechanisms of blast injury, and discusses primary blast injuries and their management.

## Explosion physics

Explosions result from the almost instantaneous conversion of a solid or liquid into gas after detonation of an explosive material.<sup>7</sup> Gas rapidly expands outwards from the point of detonation and displaces the surrounding medium—usually air or water. This expansion of gas causes an immediate rise in pressure, creating a blast wave that subsequently dissipates over distance and time.<sup>8-11</sup> As the blast wave displaces the surrounding air, it generates winds of substantial velocity (several hundred km/h depending on the size of the explosion). Blast winds can immediately propel objects or people, thus causing injury. After the initial energy of the explosion has dissipated, a period of relative low

pressure develops (negative-pressure phase) as a result of the void created by the displaced air, before pressures normalise. These pressure changes can be measured over time and then plotted graphically to produce a Friedlander curve for a simple open-space explosion (figure 2A).<sup>7</sup> The graph shows the positive-pressure and negative-pressure phases of the explosion. The maximum pressure is called the peak or blast overpressure. The blast overpressure and the positive-pressure phase are responsible for causing the range of blast injuries.

Several factors affect the magnitude of the blast overpressure, the propagation speed of the blast wave, and subsequently the likelihood of blast injury.<sup>7</sup> The first factor is the medium in which the explosion takes place. Water is non-compressible and thus, a blast wave in water propagates rapidly with a slow rate of dissipation<sup>7,9</sup> and has a greater potential for injury than does an explosion in air.<sup>10</sup>

Second, the distance a person is from an explosion determines the magnitude of their exposure to peak overpressure—the closer one is to an explosion, the greater the blast overpressure experienced. As the blast wave expands, the energy of the blast dissipates and the overpressure rapidly decreases in a manner inversely proportional to the cube of the distance from the explosion.<sup>9</sup> Thus, if the distance from an explosion is doubled, the peak overpressure will decrease to one-eighth of the original value. For example, a 1 kg explosive might generate an immediate blast overpressure of more than

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### Search strategy and selection criteria

We searched Medline using the search terms: "blast injury", "blast overpressure", "body armor", "explosive injury", "explosions", "explosive agents", "improvised explosive device", "land mines", "primary blast injury", "terrorism". Article titles and abstracts were reviewed for relevance and selected articles were used in this Seminar. Preference was given to clinical articles published between 2003, and January, 2008, but commonly referenced and highly regarded older publications were also included. Additionally, the bibliographies of selected articles were reviewed for other relevant articles. Pertinent review articles and book chapters published within the past 20 years were used when appropriate.

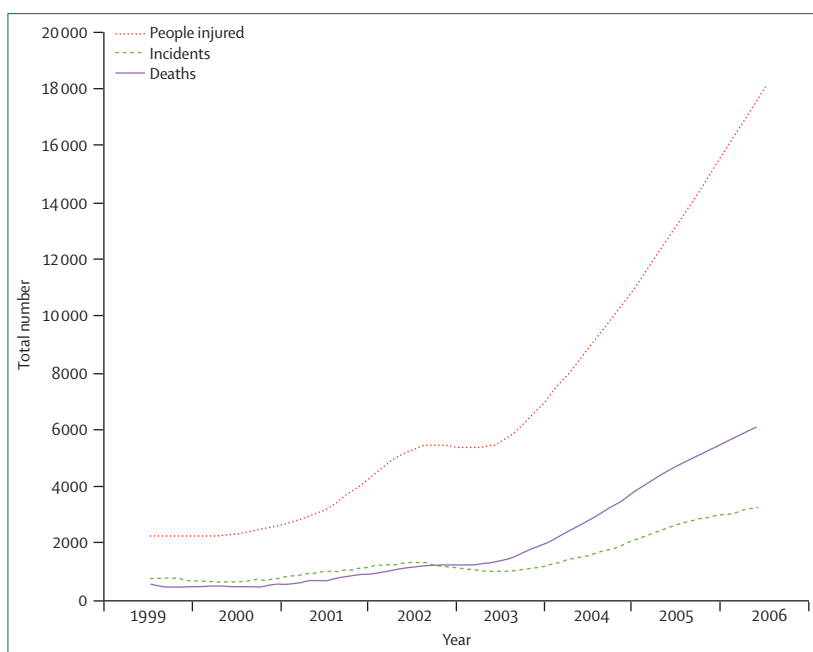


Figure 1: Worldwide trends in terrorist explosive events from 1999 to 2006  
Data obtained from RAND®-MIPT Terrorism Incident Database.<sup>3</sup>

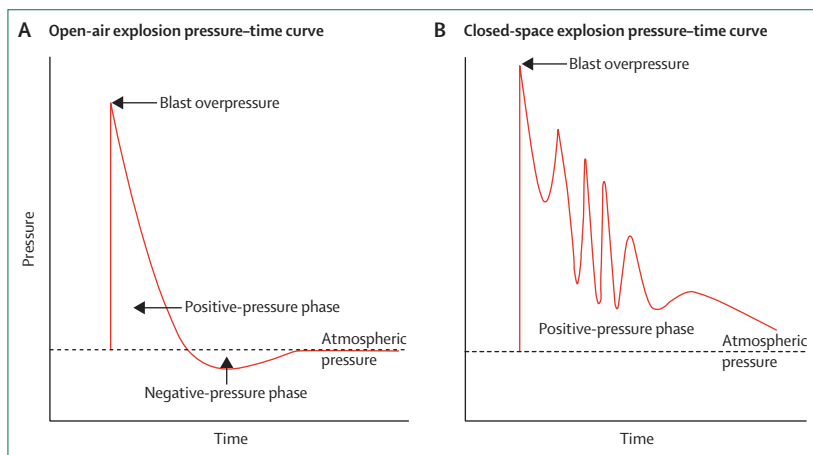


Figure 2: Simple (A) and complex (B) explosion pressure-time curves

500 kPa at the epicentre of detonation, which is a lethal force. However, 3 m from that point the blast overpressure could be as little as 20 kPa, potentially causing no injury.

The third factor is that the effective blast overpressure is amplified as pressure waves reflect back from solid surfaces and increase its force. For example, people in close proximity to a wall will be subject to enhanced blast overpressure and be at a raised risk of blast injury. The final factor is the explosion site's effect on the peak overpressure.<sup>12-15</sup> In an open space, a blast wave spreads circumferentially from its origin and quickly dissipates. However, in a confined or enclosed space (eg, a bus, room, or building) the maximum pressure is markedly amplified in magnitude because the explosive forces are contained. This confinement raises the peak overpressure

and the duration of the positive-pressure phase. The resultant complex explosive pressure curve has multiple peaks attributable to reflected pressure waves and can have a brief static hyperpressurised phase (figure 2B).<sup>7</sup> Results of a comparative analysis of four explosive events showed that closed-space explosions cause an increase in mean injury severity scores (18 vs 4), primary blast injury in admitted patients (77.5% vs 34.3%), and overall mortality (46.0% vs 7.8%).<sup>14</sup>

### Types of explosives

Chemical explosives are classified in several ways. The most common scheme categorises them as either low-order or high-order-explosives on the basis of the speed of detonation.<sup>16</sup> Low-order explosives burn rapidly (deflagrate) with a velocity of less than 1000 m/s and produce large volumes of gas that only explode if confined (eg, a pipe bomb). High-order explosives do not burn, but instead detonate when a shock wave passes through the material with a velocity usually greater than 4500 m/s, generating a substantial blast overpressure, even if unconfined.

Common examples of low-order explosives include black powder and smokeless powder, which are used as propellants for bullets and artillery shells. Black powder, thought to be invented as early as the 3rd century BCE, is a mixture of 75% potassium nitrate (saltpetre), 15% charcoal, and 10% sulphur.<sup>17</sup> Smokeless powder, developed in 1864, is made from a dried mixture of nitrocellulose, ether, and alcohol.<sup>16</sup> High-order explosives include a wide range of chemically pure compounds (eg, nitroglycerin, trinitrotoluene, acetone peroxide, cyclonite, pentaerythritol tetranitrate), and compositions (dynamite, ammonium nitrate-fuel oil, and plastic explosives [C4 and Semtex]).<sup>17</sup>

Nitroglycerin, discovered in 1846, was the first high-order explosive identified.<sup>16</sup> It is inherently unstable and dangerous to handle. However, in 1867, Alfred Nobel found that nitroglycerin could be made more predictable by combining it with inert materials, which gave rise to dynamite. Nowadays, dynamite is widely used for demolition, mining, and by the construction industry. However, it was used in terrorist attacks such as the 2004 train bombing in Madrid, Spain.<sup>6</sup>

In the 20th century, several other high-order explosives were developed, including trinitrotoluene, cyclonite, and pentaerythritol tetranitrate. Plastic explosives are made by combining a high-order explosive such as nitroglycerine or cyclonite with plasticisers, which make these explosives easily mouldable. This unique clay-like quality is a reason for their extensive use in explosive demolition and by military forces.<sup>18</sup> Furthermore, plastic explosives have historically been difficult to detect by security authorities, and, hence, are favoured by terrorists. For example, they were used in the 1988 downing of Pan Am Flight 103 in the UK, the 2000 attack on the USS Cole warship in Yemen, and the 2002 Mumbai train bombing in India.<sup>3,5</sup>

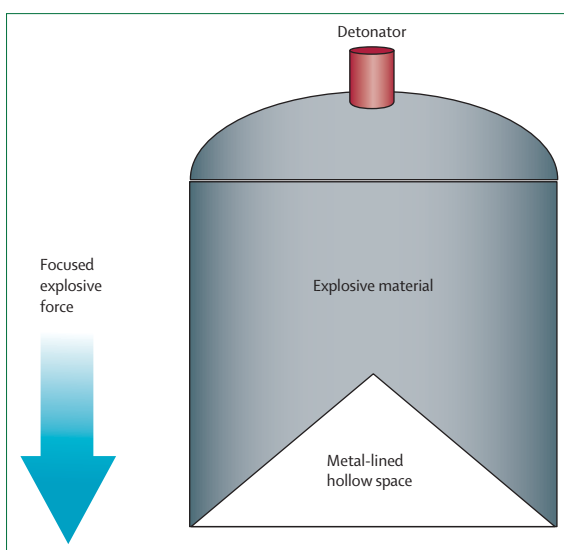
Triacetone triperoxide and hexamethylketone triperoxide diamine are examples of high-order liquid explosives.<sup>2,19</sup> These compounds can be manufactured in the home in small amounts with relative ease and are dangerously unstable. Peroxide-based explosives were identified as the explosives used in the 2005 London subway bombing.<sup>2</sup>

Another high-order explosive, ammonium nitrate–fuel oil in a 94:6 ratio, is frequently used by the mining industry and in civil construction. The use of this explosive by terrorists generates special concern because of the relative ease of obtaining the ammonium nitrate (fertiliser) and fuel oil ingredients. Car bombs of ammonium nitrate–fuel oil have been used in terrorist attacks in Northern Ireland and the Middle East, and in the 1995 Oklahoma City bombing in the USA.<sup>4</sup>

High-order explosives are further subdivided into primary explosives (those that can be detonated by mechanical shock, friction, or heat) and secondary explosives (those that usually need an initiating explosion to detonate). Primary explosives include lead azide, mercury fulminate, and lead stiphnate.<sup>16</sup> These materials are often used to initiate the detonation of secondary explosives, such as dynamite, ammonium nitrate–fuel oil, and cyclonite.

Shaped charges and explosively formed penetrators have been used to directionally amplify blast overpressures.<sup>20,21</sup> These charges have a lined, hollow space in their base which focuses much of the explosive's energy in a given direction (figure 3). This directional focus improves control and raises the likelihood of damage,<sup>20</sup> and, hence, makes them appealing to terrorist groups.<sup>20–22</sup> In 2006, the first use of a dense inert metal explosive (DIME) was publicly reported.<sup>23</sup> Composed of a homogeneous, high-order explosive (eg, cyclonite) and small particles of chemically inert materials such as tungsten, these explosives are designed to have a small but very effective explosive radius. They are deemed weapons of low collateral damage because the mass of the inert metal causes the pressure wave to drop off quickly. On exploding, the DIME casing disintegrates into very small particles. This microshrapnel and the explosive force are lethal at close range, and people who survive the blast often have limb amputations and retained heavy metal tungsten alloys, which is of public concern because these alloys have been associated with animal carcinogenesis.<sup>23</sup>

The term improvised explosive device broadly describes any makeshift incendiary device constructed to injure, incapacitate, harass, or distract.<sup>24,25</sup> The explosive component of many of these are conventional military weapons such as large artillery shells, whereas others contain plastic explosives such as C4, trinitrotoluene, other high-order explosives, or qualify as shaped charges or DIMEs.<sup>21,23,24</sup> These explosives are then delivered to their targets by non-conventional means;<sup>24</sup> for example, they are often placed along transport routes and triggered to detonate under vehicles. Various devices are used to trigger improvised explosive devices, including electronic



**Figure 3: Shaped-charge schematic**

Shaped charges are used to focus blast energy in a given direction to increase the likelihood of blast injuries.

transmitters, tilt switches, thermal switches, motion detectors, pressure-sensitive bars, and trip wires. Although trigger mechanisms can be sophisticated, the rudimentary triggers (eg, pressure devices made from calculator buttons, washing machine timers, and undetectable trip wires) are easy to conceal and difficult to counter. Anti-handling devices to prevent tampering or deactivation are occasionally linked to improvised explosive devices.<sup>25</sup>

Vehicle-borne improvised explosive devices are vehicles laden with explosives and detonated at high-value targets or official checkpoints. These vehicles can contain many hundreds of kilograms of explosives and generate a large quantity of shrapnel. The aircrafts used in the 2001 World Trade Center attacks in New York City, USA, were regarded as vehicle-borne improvised explosive devices. House-borne improvised explosive devices are large explosives placed within a house and detonated when targeted people are in range. These vehicle-borne and house-borne explosive devices deliver large, direct blast overpressures to people not protected by a vehicle or barrier.

To augment the effect of improvised explosive devices, and, hence, increase blast injuries, people that make these bombs might use shaped charges, or include fragmenting items such as ballbearings, rocks, or scrap metal. Additionally, soiled material, such as faeces, might be added, which results in contaminated penetrating fragments. Furthermore, the explosive device can be detonated in combination with chemical weapons—such as a chlorine tank—with the intent to release toxic chlorine gas.

For decades, improvised explosive devices have been used in military conflicts, including conflicts in Northern Ireland, Vietnam, Chechnya, and the Middle East.<sup>25</sup> Their use in Iraq against coalition military forces and civilians caused two-thirds of the military deaths and 38–40% of

all injuries sustained during the ongoing conflict.<sup>21,26,27</sup> Furthermore, these devices were used in the terrorist bombings in Madrid, London, and Mumbai.<sup>6,28,29</sup>

### Mechanisms of blast injury

Historically, the patterns of injury caused by explosions were divided into primary, secondary, and tertiary injuries.<sup>30,31</sup> The term quaternary injuries was then used to describe miscellaneous injuries, and recently a quinary pattern has been proposed.<sup>8,32,33</sup> People wounded in explosions usually have multiple injuries, which blur the distinction between these injury patterns and creates what has been defined as a multidimensional injury.<sup>32,34</sup> However, the simplicity of this taxonomy provides a valuable theoretical framework for understanding blast injuries.

#### Primary blast injuries

Primary blast injuries take place when the blast overpressure reaches the person and transmitted forces exert their effect on the body, causing direct tissue damage. In 1950, Scharadin<sup>35</sup> described three explosive forces that can cause injury: spallation, implosion, and inertia. These forces have a concentrated effect in regions of air–tissue interface, and are substantially greater in closed-space than in open-space explosions, resulting in a raised incidence of injury.<sup>7,36–38</sup> However, the exact degree to which these forces actually cause damage is not entirely known.

Spallation takes place when a pressure wave passes from a dense medium to a less dense medium, resulting in displacement and fragmentation of the dense medium into the less dense medium.<sup>7,35,37,38</sup> For example, an explosive detonated under water will cause the dense water to spall into the less dense air, causing fragmentation represented by an upward splash.

Implosion forces take place when gaseous contents within tissues are suddenly compressed by the blast overpressure.<sup>35</sup> As the positive-pressure phase passes, the gas re-expands and releases a large amount of kinetic energy. Ho<sup>39</sup> describes a simple example of spalling and implosion forces causing injury in the lungs, and he outlines how a blast wave travels through the relatively incompressible blood in the capillary. Spalling forces disrupt the endothelium of the capillary wall as the wave enters the alveolus and the compressed gas in the alveolus re-expands, forcing air emboli into the capillary.<sup>39</sup>

Inertial, or shearing, forces are similar in their pathophysiological effect to deceleration forces as seen in non-blast trauma injuries, such as from motor vehicle collisions.<sup>35,40,41</sup> In response to peak overpressure, tissues of varying densities move at different speeds, and thus as the overpressure passes through an organ, structural components of different densities can be tethered and damaged by these shearing forces.

#### Secondary blast injuries

Secondary blast injuries are created by debris that is physically displaced by the blast overpressure or blast

winds.<sup>8,20,35</sup> This debris can cause a combination of penetrating and blunt trauma injuries similar to wounds seen in civilian trauma (eg, stab wounds, assaults, ocular injuries). People wounded by terrorist bombs often have a substantial number of secondary blast injuries because these munitions usually contain fragmenting items such as ballbearings, nails, rocks, or scrap metal to maximise the lethality of the device.<sup>6,32,42,43</sup> Commonly, small puncture wounds that represent secondary-blast injuries hide these fragments and severe underlying injuries.

The distance over which fragments travel and can cause injury is much greater than the distance over which a blast overpressure travels. Thus, fragments can cause secondary-blast injury hundreds to thousands of metres from the explosion's epicentre, whereas primary-blast injury usually happens within tens of metres.<sup>44</sup> Hence, secondary blast injuries are more common than are primary blast injuries.<sup>45–49</sup>

#### Tertiary blast injuries

Tertiary blast injuries are caused when a person is physically displaced by the force of the peak overpressure and blast winds and sustains blunt trauma injury such as closed head injuries, blunt abdominal trauma, tissue contusions, or fractures. Additionally, collapse of buildings or surrounding structures confers a raised risk of severe tertiary blast injury (ie, head trauma, traumatic asphyxia, and crush injuries) and mortality compared to explosions that are in a closed space that does not collapse.<sup>12,13,15,50,51</sup>

#### Quaternary and quinary blast injuries

Quaternary blast injuries—occasionally termed miscellaneous blast injuries—are caused directly by the explosion but are not classified as primary, secondary, or tertiary injury.<sup>8,52,53</sup> They include but are not limited to burns, toxic substance exposures (eg, radiation, carbon monoxide poisoning, cyanide poisoning), asphyxia, and psychological trauma.

Lastly is the quinary pattern of injury. This classification was suggested on the basis of a case series in which a hyperinflammatory state was seen in patients after a bombing in Israel.<sup>8,32,33</sup> These patients manifested hyperpyrexia, diaphoresis, low central venous pressure, and a positive fluid balance. The future study of injuries sustained from bombs will enable improved characterisation of this proposed blast injury pattern.

### Primary blast injury patterns and management

Primary blast injuries happen with greatest frequency at air–tissue interfaces.<sup>54</sup> Thus, organ systems with high air content, such as the pulmonary, gastrointestinal, and auditory systems, are most susceptible. Auditory injury happens at the lowest blast overpressure (35 kPa) and most frequently, whereas pulmonary and intestinal injury tend to arise at increased pressures (75–100 kPa) and are less common.<sup>13,55</sup> Other body systems affected by blast

overpressure include the central nervous, musculoskeletal, and less commonly the visual and cardiovascular systems.<sup>38</sup>

Besides injuries specific to organ systems, a global response involving shock-like physiology, uncompensated by vasoconstriction, can follow a thoracic blast overpressure. This response results in transient bradypnoea, bradycardia, and systemic hypotension and is mediated through pulmonary C fibres in the vagus nerve. It usually resolves minutes to hours after the explosion exposure.<sup>56–58</sup>

### Pulmonary system

The pulmonary system is at a raised risk for primary blast injuries because of its substantial air–tissue surface area.<sup>14,59</sup> Explosive forces can give rise to pulmonary haemorrhage and contusions, direct barotrauma, and arterial air embolism,<sup>11,47,59–61</sup> causing substantial injury—immediate and delayed.<sup>36,62</sup> 17–47% of people who die from explosions have evidence of pulmonary primary blast injury,<sup>48,63–65</sup> and up to 44% of people hospitalised and 71% of those who are critically ill and hospitalised have pulmonary injury.<sup>13,36,42,59,66</sup> In settings that generate a high blast overpressure or an increased positive-pressure phase (closed space explosions), the incidence of pulmonary primary blast injury increases three-fold.<sup>13,14,67</sup> However, with early diagnosis and aggressive treatment, in-hospital mortality rates for these patients range from 3.4% to 25.0%.<sup>36,62</sup> Furthermore, people who are discharged have a very good prognosis at 1-year follow-up.<sup>36,62,68</sup>

Pulmonary contusions—a manifestation of blast lung injury—are caused by spalling and implosion forces that disrupt the alveolar structure, capillary walls, and the intra-alveolar space of the lung parenchyma, allowing for perivascular pooling of blood.<sup>11,38,47,61</sup> The blast overpressure simultaneously exerts compressive forces on the extravascular fluid, driving it into the alveolar space and causing pulmonary oedema and alveolar haemorrhage.<sup>69</sup> Progressive vascular leak and inflammatory changes from this lung injury develop over 12–24 h, possibly contributing to some delayed presentations,<sup>69–72</sup> although, most blast lung injuries develop immediately.<sup>36,62</sup> Tissue injury results in mucosal petechial haemorrhages with varying degrees of confluence and can be seen on examination of the upper airways.<sup>38,72</sup> Furthermore, these microbleeds are evident under the visceral pleura. The ribs protect the lung parenchyma from the full force of the blast overpressure, which results in stripes of haemorrhagic congestion that correspond to the intercostal spaces.<sup>38</sup> Traumatic interstitial emphysema can develop from implosion forces driving air into the interstitial space.<sup>11,60</sup>

The clinical picture of dyspnoea, cough, and hypoxia that accompanies these injuries is referred to as blast lung syndrome<sup>9,11,60</sup> and represents impaired gas exchange and vascular shunting with ventilation mismatching.<sup>47</sup> Pulmonary barotrauma can cause pleural tears or lacerations, which give rise to pneumothoraces, haemothoraces,

pneumomediastinum, or subcutaneous air.<sup>9,11,38,47,60</sup> Theoretically, altitude changes associated with air evacuations or transfers might exacerbate blast-related pulmonary barotraumas,<sup>54</sup> although this was not seen in one military conflict.<sup>60</sup>

When blast lung injury or pulmonary barotrauma is present, shearing forces can disrupt the bronchovascular tree, creating bronchopulmonary fistulas.<sup>61</sup> Arterial air emboli can subsequently develop immediately after the explosion or can arise later, especially if the patient needs positive-pressure ventilation. Additionally, haemorrhagic shock can entrain air into the arterial system, leading to arterial air embolism.<sup>73</sup> When massive, these emboli can cause stroke, myocardial infarction, spinal cord infarction, intestinal ischaemia, or death.<sup>60,61</sup> Microscopic arterial air embolisms can result in confusion, mental status changes, vision disturbances, or vague complaints of pain and weakness. Air in the retinal arteries, tongue blanching, or livedo reticularis (lacy, mottled skin discolouration) can be subtle indicators of arterial air embolisms and should be investigated.<sup>9,60,73</sup>

When blast lung injury is suspected, a chest radiograph should be undertaken on any person with pulmonary complaints, evidence of other primary blast injuries, or suspected exposure to a high blast overpressure. Bilateral pulmonary infiltrates in a butterfly pattern are commonly seen in patients with this type of injury (figure 4).<sup>36,62</sup> However, the development of lung injury can be delayed and, hence, patients with pulmonary signs or symptoms and a normal chest radiograph should be observed

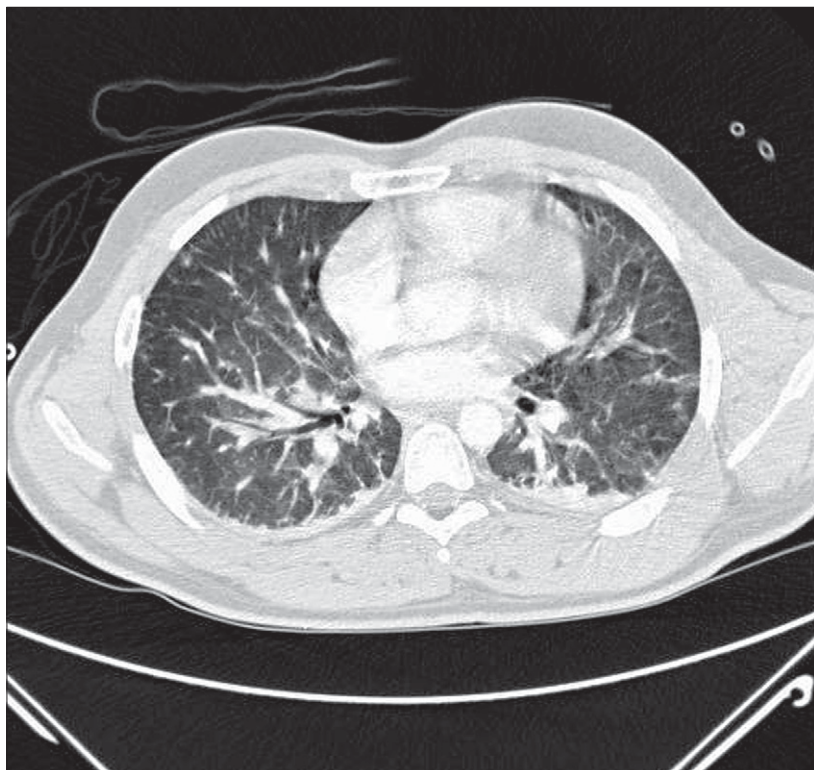


**Figure 4: Chest radiograph of a patient with blast injury**

This radiograph shows classic bilateral pulmonary infiltrates seen in blast lung injury.

for 6–8 h before discharge.<sup>8</sup> If symptoms are substantial or persistent, a chest CT should be done because blast lung injury and pulmonary complications can be missed on chest radiograph (figure 5).<sup>56,74</sup> One study showed that, in addition to chest radiograph findings, PaO<sub>2</sub>:F<sub>i</sub>O<sub>2</sub> ratio and the presence or absence of bronchopleural fistulas can be used to identify the severity of the lung injury and help predict respiratory management and outcomes (table).<sup>62</sup>

Management of pulmonary primary blast injury can be a challenge because the therapies for the different injuries often conflict. For example, haemodynamic instability dictates volume resuscitation, whereas excessive crystalloid can lead to pulmonary oedema in patients with pulmonary contusions.<sup>36</sup> Therefore, fluid resuscitation needs to be carefully monitored in moderate to severe blast lung injuries, and invasive monitoring with a pulmonary artery catheter should be considered.<sup>8,60,75–77</sup> Additionally, when treating pulmonary primary blast injury and blast-induced pulmonary contusions, high priority should be given to optimising the patient's physiological respiratory status with non-invasive ventilation techniques and adequate pain management, because positive-pressure ventilation can worsen pulmonary barotrauma and increase the patient's risk of arterial air embolism.<sup>9,60,62,73,78</sup> Prompt chest drainage of the pneumothoraces or haemothoraces can help minimise the need for positive-pressure ventilation.



**Figure 5: Chest CT of a patient with blast injury**

This CT shows the pulmonary consolidations seen in blast lung injury that can potentially be missed on chest radiograph.

Finally, the use of prophylactic chest tube thoracostomy should be considered in severe blast lung injuries that need positive-pressure ventilation or for patients who need air transportation.<sup>56</sup>

Blast lung injury induces poor lung compliance.<sup>62,79</sup> When positive-pressure ventilation is needed, lung-protective techniques should be used. These strategies include maintaining acceptably low oxygen saturations (90%) and low tidal volumes (5–7 mL/kg), pressure-controlled ventilation, positive end-expiratory pressures (PEEP), and permissive hypercapnia.<sup>2,36,62,79–83</sup> By contrast, strategies to minimise the sequelae from arterial air emboli include maximising spontaneous ventilation, low PEEP, and using 100% F<sub>i</sub>O<sub>2</sub> to encourage quick absorption of emboli.<sup>73,84</sup> Although no prospective studies describe the best management of an arterial air embolism, most researchers support placing the patient in a recumbent, left-lateral decubitus position to decrease the risk of systemic embolisation.<sup>11,59,73,75</sup> However, some investigators postulate that by placing the most likely affected lung in the dependent position, alveolar pressures become lower than vascular pressures, which makes further entrainment of air into the vascular system less probable.<sup>54,60</sup> Despite little clinical data, many experts endorse hyperbaric oxygen therapy as the preferred treatment for arterial air embolism.<sup>85–87</sup> For best outcomes, this therapy should be used as soon as the patient's clinical condition allows it, although treatment benefits after delays of up to 60 h have been shown.<sup>85–89</sup>

### Gastrointestinal system

The gastrointestinal system is at a similar increased risk of primary blast injury because of its air content.<sup>61,77</sup> The incidence of abdominal injury might be as high as 14% to 24%;<sup>66,74</sup> however, composite data suggest that the figure is lower.<sup>12,13</sup> Abdominal injury occurs more commonly after underwater or closed-space explosions, owing to the heightened blast overpressure and extended positive pressure phase.<sup>9,14,61,74,90</sup> In these settings, a two-fold to four-fold increase in gastrointestinal injuries has been reported.<sup>13,14</sup>

The colon and ileocaecal region are the visceral structures at greatest risk of intestinal perforation, which is caused by implosion forces rupturing the bowel wall.<sup>57,61,66,91</sup> The intestinal wall contuses when the implosion and shearing forces cause the wall's structural layers to separate.<sup>91</sup> The resultant intramural oedema and haemorrhage with microthromboses compromise intestinal perfusion and put the intestine at risk of delayed perforation.<sup>66,91,92</sup> Furthermore, interruption of the mesenteric blood supply by shearing forces or arterial air embolism leads to intestinal ischaemia.<sup>73</sup> Although abdominal solid organ injury can arise as a primary blast injury, it is more likely to be a result of a secondary or tertiary blast injury unless the person was in close proximity to the explosion or experienced substantial blast overpressure.<sup>30,74,90</sup>

Patients with haemorrhagic shock from intestinal injury should undergo volume resuscitation until emergency surgery can be done.<sup>93,94</sup> However, overly aggressive resuscitation can potentially worsen pulmonary primary blast injury.<sup>9,60</sup> Permissive hypotension (ie, systolic pressures between 80 mm Hg and 90 mm Hg) might improve outcomes in traumatic resuscitation,<sup>95</sup> although people with concomitant blast lung injury could benefit from this treatment, those with traumatic brain injury should have their blood pressure normalised to minimise cerebral hypoperfusion.<sup>95,96</sup>

Liberal use of a focused abdominal sonography for trauma examination to identify intraperitoneal fluid can assist in operative decision-making when assessing abdominal complaints for life-threatening injuries.<sup>45,96–98</sup>

However, a negative examination does not necessarily exclude abdominal primary blast injury.<sup>96</sup> People who are haemodynamically stable with abdominal pain or penetrating injuries should have an abdominal CT.<sup>96</sup> Although this scan is specific for solid organ injury and perforation,<sup>99,100</sup> it lacks the sensitivity to exclude intestinal contusions and mesenteric injury definitively.<sup>101,102</sup> Hence, symptomatic patients must be observed for 6–8 hours and re-examined.<sup>60</sup> Additional investigations by use of diagnostic peritoneal lavage might be helpful.<sup>60</sup> Patients with injuries that need operative management should be assessed for blast lung injury with a chest radiograph because positive-pressure ventilation will be needed during surgery.

### Auditory system

The auditory system is the system most commonly affected by blast overpressure.<sup>12,13,103</sup> Depending on the explosion's setting, between 2% and 32% of all people injured,<sup>13,104</sup> and up to 94% of those with primary blast injuries will have a ruptured tympanic membranes.<sup>36,66,105</sup> Thus, some experts postulate that an intact tympanic membrane suggests little exposure to blast overpressure, alleviating the need for further assessment for primary blast injuries. However, results of studies have shown that a substantial proportion of survivors with and without tympanic membrane rupture have blast lung injury.<sup>6,105</sup> Asymptomatic patients with intact tympanic membranes have a very low likelihood of occult pulmonary or intestinal primary blast injury.<sup>8</sup> Patients with pulmonary or abdominal symptoms, or tympanic-membrane rupture should be assessed for additional primary blast injuries, either by radiographic imaging or observation.<sup>8</sup>

Primary blast injuries can include other audio-vestibular injuries. A temporary threshold shift for audible noises can result in a transient sensorineuronal deafness or tinnitus, which often resolves over several hours to days.<sup>103</sup> Ossicle disruption or damage to the sensory structures might cause permanent conductive hearing deficits that need surgical intervention.<sup>103,106–108</sup> Furthermore, perilymph

	Mild blast lung injury	Moderate blast lung injury	Severe blast lung injury
<b>Clinical findings</b>			
PaO <sub>2</sub> /F <sub>2</sub> O <sub>2</sub> ratio	>200	60–200	<60
Chest radiograph infiltrates	Localised	Bilateral or unilateral	Massive
Bronchopleural fistulas	–	+/-	+
<b>Respiratory management</b>			
Positive pressure ventilation	+/-	+	+
Unconventional therapies*	–	–	+
Positive end-expiratory pressure (cm H <sub>2</sub> O)	≤5	5–10	>10
<b>Outcomes</b>			
Adult respiratory distress syndrome	0%	33%	75%
Mortality due to blast lung injury	0%	0%	75%

\*Unconventional therapies included extracorporeal membrane oxygenation, independent lung ventilation, high-frequency jet ventilation, and nitric oxide. Data from Pizov and co-workers.<sup>52</sup>

**Table: Blast lung injury severity and associated management and outcomes**

fistulas can cause vertigo or dizziness, which must be differentiated from pathology of the central nervous system.

Small tympanic-membrane ruptures can often spontaneously heal by means of conservative management.<sup>105,109–111</sup> Ruptures that involve more than 5% of the membrane surface will probably need surgical intervention (17–89% of patients), which is proportional to the percentage perforated.<sup>104</sup> Delays to assessment and treatment are associated with poor hearing outcomes, and appropriate early referrals should be made if symptoms persist.<sup>109,110</sup>

### Central nervous system

Secondary and tertiary blast-related brain injuries that involve intracranial haemorrhage, direct parenchymal damage, and cerebral contusion represent most central nervous system injuries, although researchers now believe that brain injury can also be caused by primary blast forces.<sup>112–118</sup> After explosions, cerebral concussive syndromes are common and associated with post-traumatic-stress disorder,<sup>113,118,119</sup> and affected people experience substantial memory dysfunction and cognitive deficits.<sup>120</sup> Shellshock, an old term used to describe the psychoemotional state of a person after exposure to an explosion, is now thought to be a combination of post-traumatic-stress disorder and cerebral concussive syndromes resulting from the blast overpressure.<sup>116,117,121,122</sup> Unfortunately, methodical clinical investigations of primary blast-related brain injury are scarce and the results of diagnostic testing (eg, neuroimaging, neuropsychological tests, and serum biomarkers) are typically inconclusive and difficult to interpret.<sup>119</sup> Results of recent studies suggest, however, that tympanic perforation might be a predictor for concussive brain injury although not necessarily for other primary blast injuries.<sup>123,124</sup> Research is underway to improve knowledge of the characteristics, prognosis, and treatment of blast-related brain injury.<sup>112</sup>



Figure 6: Medical fasciotomy for compartment syndrome after blast injury

### Musculoskeletal system

Extremity and musculoskeletal injuries can be caused by primary, secondary, or tertiary blast forces. In combat settings, these injuries represent up to 54% of combat wounds.<sup>27</sup> This is largely attributable to the use of improvised explosive devices, which cause devastating injuries to vascular and orthopaedic structures.<sup>125–128</sup> Compartment syndromes and traumatic amputations are two extremity injuries that warrant discussion in relation to primary blast injuries.

Compartment syndromes are common after exposure to an explosion. Ritenour and colleagues<sup>128</sup> reported that 86% of fasciotomies in combat casualties were carried out after explosion-related trauma. Fractures, direct tissue damage, and burns can elevate extremity compartmental pressures, which lead to tissue injury, ischaemia, and necrosis.<sup>129</sup> Although almost all researchers classify this syndrome as a tertiary or quaternary blast injury,<sup>8,129</sup> compartment syndrome can occur in apparently uninjured blast-exposed extremities, thus raising the possibility that primary blast forces might contribute.<sup>128</sup> Delayed compartment syndrome has been reported in people after explosions and is postulated to be associated with several factors, including the presence of severe diffuse injury, pelvic fractures, and burns covering a large body surface-area that raise the need for large-volume or extended resuscitation.<sup>128,130–132</sup> Secondary extremity compartment syndrome after over-resuscitation has, however, been described in patients with injuries not sustained from explosives.<sup>133,134</sup> Thus, the contribution of other proposed factors to this syndrome,<sup>113,128</sup> such as blast overpressure, blast-induced release of inflammatory mediators, and the need for air evacuation, remains unclear. Liberal use of early

fasciotomy to avoid or treat compartment syndrome is recommended in these high-risk patients (figure 6).<sup>96,115</sup> Further research is needed to define the contribution of primary blast exposure to the development of compartment syndrome.

1–7% of people injured by explosions have traumatic amputations<sup>12,63,67,135</sup>—this rate has remained relatively constant throughout recent military conflicts.<sup>135</sup> These injuries result from high blast-overpressure forces causing bony fractures while concomitant strong blast winds rupture soft tissue structure,<sup>129,136,137</sup> leading to partial or complete extremity amputations. The exact overpressure needed to cause these injuries is not known. Traumatic amputations are mostly regarded as primary blast injuries,<sup>47,56,129,136–138</sup> however, some researchers classify them as tertiary blast injuries.<sup>8,11</sup> Results of large studies show that they are not only associated with immediate mortality rates between 10% and 85%,<sup>63,67</sup> but they also act as a marker for poor prognosis and are seen in 10% of delayed deaths.<sup>63</sup> Hence, traumatic amputations warrant aggressive intervention and mandate a high suspicion for additional primary blast injury.

### Other systems

Primary blast forces occasionally affect other body systems. For example, up to 10% of people injured by explosions have ocular trauma, although most of these injuries are attributable to secondary blast injury forces.<sup>115</sup> However, ruptured globes, hyphemas, conjunctival haemorrhage, serous retinitis, and orbital fractures have been reported as primary blast injuries of the visual system.<sup>811,61,115</sup> Additionally, cardiac contusions, myocardial-wall haemorrhage, and atrial rupture have been described in the cardiovascular system.<sup>30,40,115,139</sup>

Blast-induced burns are present in up to 27% of people injured by an explosion<sup>12,12,56,67,140</sup> and are associated with immediate mortality and a high rate of coexisting primary blast injury.<sup>36,67</sup> Although technically quaternary blast injuries, these burns affect the integumentary, pulmonary, and cardiovascular systems, and complicate management of primary blast injuries. The direct thermal energy of the explosion and the secondary burning of structures, vehicles, clothes, or equipment contribute to blast-induced burns. The hands and face are the most commonly affected areas from the initial explosion. Trunk and lower-extremity burns are often attributable to fire from smouldering vehicles or buildings and affect large surface areas.<sup>140</sup>

These burns can be complicated by crush injuries, making debridement and management difficult and infection rates high. Furthermore, the combination of blast lung injury and the systemic inflammation of large surface burns can lead to adult respiratory distress syndrome and hypoxia. Aggressive crystalloid resuscitation is crucial in thermal burns, although abdominal compartment syndrome and extremity



myonecrosis can occur with excessive resuscitation.<sup>141</sup> Use of intravenous albumin, vasopressors, and pulmonary catheters could be needed.<sup>141</sup> Meticulous records of treatments, vital signs, urinary output, and infusates are important.<sup>141</sup> Patients with blast-induced burns should be cared for in a burn centre because of the elevated rates of infection and multiorgan dysfunction.

## Conclusion

Worldwide, a paradigm shift has taken place in modern-day conflict. The rise in urban warfare tactics by terrorist and paramilitary groups means that local health systems must be prepared to manage devastating explosion-related injuries. Acute care providers (ie, physicians, nurses, and ancillary staff of emergency medicine, surgery, orthopaedics, and anaesthesiology), who are most likely to be the first receivers of injured people, have an urgent responsibility to know and understand the diagnostic and management issues unique to blast injuries.

### Conflicts of interest

We declare that we have no conflicts of interest.

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