

# Applied Pathophysiology of Blast Injuries

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### Introduction

Explosive devices are common terrorist tools, with the objectives of producing multiple victims and maximizing the numbers of deceased (*Slater, 1997*). The 2017 Manchester suicide, for example, injured more than 500 people and killed 23. This device utilized Triacetone Triperoxide (TATP), the same explosive utilized in the 2015 Paris and 2016 Brussels attacks (*CTVNews, 2017*).

An analysis of a global terrorism database found that bombings were responsible for 38.5% of fatalities inflicted by terrorism on civilian targets, the highest of any attack type (*Magnus, 2018*). Explosive devices have been utilized in major terrorist attacks in London, Manchester, Paris, Madrid, Oslo, Bali and Oklahoma. Explosive devices can cause significant property damage. Terrorist explosions also generate intense media coverage, a further terrorist objective.

In 2014, in the United States, there were a total of 912 explosive incidents and 642 bombings, including five churches and 15 schools (*United States Bomb Data Center Explosives Incident Report (2014)*). There were 41 deaths. In addition, there were 19 first responders injured.

In a military context, improvised explosive devices are a common method utilised in asymmetric warfare, a psychological weapon against the will of the stronger party (*McKenzie, 2001*).

### Explosives and Explosions

Explosives are highly energetic substances with fast reaction rates. The explosive reaction releases the potential energy stored in their chemical bonds. This released energy is transformed into a combination of thermal and kinetic energy.

The instantaneous power generation of HMX (High Melting Explosive), a powerful high explosive, is far more than the capacity of all the power generators in the United States combined (*Venugopalan, 2015*).

### The Physics of Explosions

Explosives are broadly classified as low and high explosives (*Thurman, 2006*). Low explosives include black powder (mixture of potassium nitrate, charcoal and sulphur) and smokeless powder (nitrocellulose based) (*Laska, 2016*). Their explosive reaction, or deflagration, occurs at subsonic speeds, and does not generate a shock wave. The reaction proceeds on a flame front and can be considered analogous to peeling an onion – one layer at a time. Commercially, low explosives are used as propellants, pyrotechnics or as an initiator for other explosives.

High explosives include dynamite, C4, Semtex, Pentaerythritol tetranitrate (PETN), TATP and ANFO (ammonium nitrate fuel oil). Their explosive reaction, detonation, is characterised by a supersonic over-pressurization shock wave (*CDC, n.d.*). This fast-moving shock

Deflagration	Detonation
Surface phenomenon (propagation is by layer-to-layer burning)	Shockwave phenomenon (high speed shock wave travelling through the explosive medium propagates detonation)
The rate of deflagration is lower than the speed of sound in the medium	The rate of detonation is higher than the speed of sound in the medium
The products of deflagration go away from (opposite to) the direction of propagation of deflagration.	The products of detonation travel in the same direction as that of the propagation of detonation

**Table 1:** (Venugopalan, 2015)

wave occurs within the explosive (initiating further reactions) and outside (causing a blast wave and heat). Commercially, high explosives are utilised for their shattering effect, such as building demolitions and rock blasting (Thurman, 2006).

### The Chemistry of Explosive Devices

Almost all chemical explosives contain oxygen, nitrogen and oxidisable fuel elements, such as carbon and hydrogen. The oxygen and nitrogen are often bonded to each other to form chemical side chains, including nitrate (NO<sub>3</sub>), nitrite (NO<sub>2</sub>) and nitroxy (NO). The bound oxygen facilitates the rapid oxygenation of the fuel, compared to conventional fuels, e.g. coal, which rely on atmospheric oxygen to oxidise and release energy.

Low explosives contain a fuel and a source of oxidation, but these are generally in different compounds, mixed together. Gunpowder is a combination of potassium nitrate, charcoal and sulphur, which can be made to decompose with the following reaction:  $2KNO_3 + 3C + S > K_2S + N_2 + CO_2$

The large amount of gas released enables its use as a propellant (firearms) and as a blasting agent. The heat generated

Some substances, such as smokeless powder function as low explosives if ignited, but, if 'detonated', can behave as a high explosive (Heramb, 2002).

enables the reaction's self-propagation. High explosives are described as metastable compounds. The compound is stable until a certain, usually small, amount of energy is added, creating instability. This energy can be a thermal, mechanical or electrical stimulus. Once triggered, a chemical reaction ensues, utilising the oxygen within the molecule as an oxidising agent, releasing large amounts of energy.

### Classification of Blast Injury

Zuckerman's 1940 classification of blast injuries is still relevant today (Zuckerman, 1940).

Injuries are organized by the mechanism through which they are sustained, and are classified as primary, secondary, tertiary and quaternary (Table 2), each with a unique pattern of injury. The blast injury categories can occur in isolation or in combination.

Category	Cause	Typical Injuries
<b>Primary</b>	Blast wave transmitted to gas-containing structures – direct effect of blast pressure on tissues	Blast lung, tympanic membrane rupture, intestinal rupture
<b>Secondary</b>	Impact of airborne debris & shrapnel	Penetrating traumatic injuries
<b>Tertiary</b>	Transposition of the entire body due to blast wind or structural collapse	Blunt traumatic injuries
<b>Quaternary</b>	All other forces	Thermal & chemical burns, toxin/radiation exposure, asphyxiation, infection, PTSD

**Table 2:** Categories of Blast injury

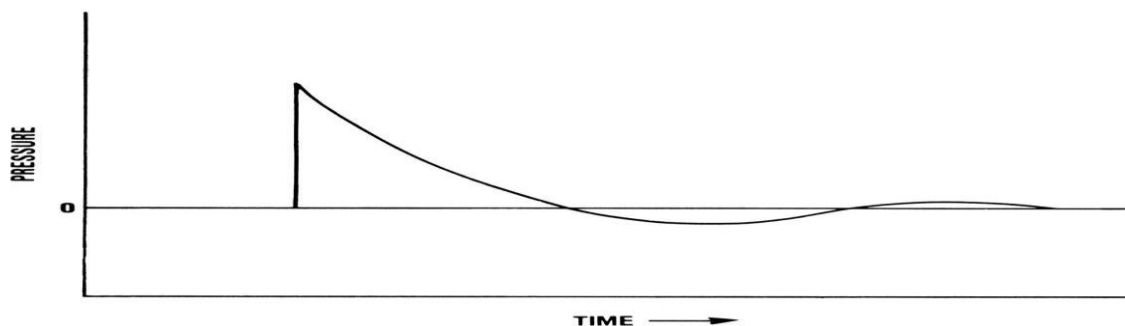
### Primary Blast Injury Pathophysiology

Primary blast injury (PBI) is organ and tissue damage caused by the blast wave associated with high explosives. PBI accounts for the majority of explosion-related deaths but a smaller proportion of the total morbidity from explosions.

On detonation of the explosive device, a blast wave is generated by the rapid

transformation of the explosive material from a solid (or liquid) to a gas (Kizer, 2003) (Harrison, Kirkman, & Mahoney, 2007). Almost instantaneously, the explosive increases in volume by up to 100,000 times. This results in an expanding shock wave of extremely high pressure.

When the blast front (the leading edge of a blast wave) reaches a victim it causes a massive, almost instantaneous rise in ambient pressure (Figure 1).



**Figure 1:** Blast injuries. Idealized graph of a blast pressure wave over time (Bowen & Bellamy, 1988)

Explosive gases continue to expand away from their point of origin, and a relative vacuum follows the peak positive overpressure, forming a longer-duration negative underpressure. Positive overpressure and negative underpressure

waves are both capable of causing significant PBI.

When a blast wave interacts with a body, two types of pressure waves are generated (Horrocks, 2001). Stress waves are

longitudinal forces (in the direction of travel) moving at supersonic speeds. They create spalling effects at tissue-gas interfaces (Horrocks, 2001), (Shuker, 1995), (Bowen & Bellamy, 1988) (Glenshaw, Vernick, Li, Sorock, Brown, & Mallonee, 2009), and occur when a shock wave travelling through a tissue reaches an interface with a tissue of lower density (such as gas), with a force that exceeds the tissue's tensile strength, disrupting it (Mackenzie, 2013). Fragmentation on the surface of the higher density tissue follows, resulting in microvascular damage. Stress waves can also cause implosion of gas-containing structures (i.e. alveoli or bowel) due to the higher-pressure tissues surrounding them. Gas-containing organs, especially the lungs, bowel, and middle ear, are therefore those most susceptible to PBI.

Shear waves are longer duration and lower velocity transverse pressure waves (at right angles to direction of travel) that result in tearing of tissues and organs (brisance) (Horrocks, 2001). They occur because blast waves lead to differences in the rates of acceleration and deceleration of tissues with different densities, creating a 'shear force'.

## Secondary Blast Injury Pathophysiology

Secondary blast injury (SBI) is caused by flying objects striking individuals. In many explosions this mechanism is responsible for the majority of casualties. The air around the blast is displaced by the wave of overpressure, resulting in high-velocity blast winds. Loose objects are similarly displaced, and these form projectiles with the potential to cause both blunt and penetrating injuries. The velocity of these objects dissipates more slowly than the

primary blast wave, so casualties well outside of the "lung contusion and bowel rupture" radius can still end up being severely damaged with penetrating injuries. Penetrating thoracic trauma, including lacerations of the heart and great vessels, is the most common cause of death in the setting of secondary blast injury (Glenshaw, Vernick, Li, Sorock, Brown, & Mallonee, 2009).

Deliberate placement of projectiles, in the context of explosive design, also occurs. For example, military explosive casings (e.g. hand grenades) are specifically designed to fragment and to maximise damage from flying debris (or shrapnel). Similarly, civilian terrorist bombers often deliberately place screws or other small metal objects around their weapons to increase secondary blast injuries. Alice Hawthorne died after the Atlanta Centennial Park explosion after being struck in the head by a nail (Gomes, 2011). In other instances, the target structure provides raw material for the projected objects, e.g. shattered glass from a blasted-out window or the glass facade of a building (Keyes, 2005). After the 1998 US embassy bombing in Nairobi, flying glass wounded victims up to 2km away (Wightman J. , 2001).

These projectiles may produce minimal external signs and follow an unpredictable path through the body. They can also cause significant wound contamination.

## Tertiary Blast Injury

Tertiary blast injuries are caused by individuals flying through the air and striking other objects. Unless the explosion is of extremely high energy or focused in some way (e.g. through a door or hatch), a person with tertiary blast injury usually is very close to the explosion source.

## Quaternary Blast Injury

Miscellaneous blast-related injuries include burns (chemical or thermal), injury from falling objects, crush injuries from collapsed structures and displaced heavy objects; and toxic dust, gas, or radiation exposure. Other injuries within this category include post-traumatic stress disorder (PTSD) and infection.

## Epidemiology of Blast Injuries

The incidence of explosions and resultant blast injuries have increased throughout the twentieth century. Although some of these events have been attributed to increasing industrialisation, the majority are the result of attacks using explosive weapons (Horrocks, 2001)

The scientific literature regarding the incidence, prevalence, and injury profile of explosive injuries is heterogenous. This is due to the diverse causation methods behind explosion-related injuries, the variety of attack environments and methods adopted in terrorist bombings and the bomb preparation. For example, 6-11% of military casualties in recent conflicts have suffered primary blast lung injury, but this increases to over 90% in enclosed space terrorist attacks e.g. trains (Scott et al, 2017).

Attack environment is an important factor in the resulting injury profile, with the highest contrast seen between the most enclosed environments (trains, buses) and open space. with significantly fewer patients suffering targets in open space were less likely to suffer blast lung, TM perforation, or intra-abdominal rupture. injuries induced by the blast wave or fireball, This is attributed to the rapid decay

of the blast wave in open-space, as opposed to the multiple reflections that prolong the loading duration in enclosed spaces (Peters, 2011). An earlier study (Defence Atomic Support Agency, 1968) suggests that positioning a victim near a reflecting surface, perpendicular to the blast wind, halves the overpressure needed to cause similar injury. These factors have been exploited by terrorists in recent history (e.g. Madrid 2004, London 2005) and render enclosed environments attractive targets (O'Neill, Robinson, & Ingleton, 2012). Casualties in open space were more susceptible to penetrating trauma, which may be a result of the uninhibited trajectory of fragments.

Demonstrating the heterogeneity of injury patterns, Israeli bus bombing data years shows the prevalence of penetrating trauma (Almogly, Luria, Richter, Pizov, Bdolah-Abram, & Mintz, 2005). Investigators found that intra-abdominal trauma was predominantly caused by fragment impact rather than the blast wave, in contrast to earlier incidents. This may be related to changes in IED design, including a shift towards higher mass shrapnel, and also increases in detonation of IEDs at chest height rather than under a seat. This may explain the observed higher incidence of penetrating head, neck and face trauma (*Wightman & Gladish, 2001*), (*Arnold, 2003*).

Primary blast injuries are uncommon. Secondary and tertiary injuries predominate following an explosion (*Wightman & Gladish, 2001*), (*Arnold, 2003*). Table 3 demonstrates factors relating to the event that would convey a greater likelihood of PBI, in addition to these more immediately obvious injuries.

Factor		Explanation
<b>Explosive Device</b>	Size	Larger explosive devices generate higher and more sustained overpressures
	Suicide Bomb	Usually detonated in close proximity to groups of people, increasing the risk of primary blast injury
	Enhanced blast munitions	Includes fuel-air explosives. Designed to result in increased primary blast injury
<b>Geography</b>	Enclosed space	Blast wave reflect off and reverberate within fixed structures resulting in complex and multiple waves of overpressure, augmenting blast effects
	Underwater	Sustained propagation of blast wave results in primary blast injury even at distance from the detonation
<b>Victim</b>	Proximity	The closer to the detonated device, the higher the overpressure to which a victim is exposed
	Personal protective equipment	Does not protect from PBI. The protection from secondary and some tertiary blast injury results in increased risk of primary blast in survivors
	Burns	Flash burns occur in patients who were in close proximity to the detonation, also at higher risk of primary blasts injuries

**Table 3:** Explosion and patient factors increasing risk of primary blast injury. Adapted from text (Horrocks, 2001), (Kizer, 2003), (Ciraulo & Frykberg, 2006).

### Confined Spaces

Leibovici examined injury patterns after four explosions in Israel, two in the open air and two in the enclosed space of a bus (Leibovici, 1996). The devices in each event were similar. The mortality after the open-air attack was 8%, compared to 49% after the bus bombings. The Injury Severity Score (ISS) of survivors was higher in the bus bombing, with a mean ISS of 18, compared to a mean of 4 after open air bombings. In addition, a greater proportion of the casualties from the bus had a primary blast injury (77% vs 34%).

### Organ Systems Affected by Blast Injury

Air and fluid-filled organs are the most vulnerable to the effects of the blast wave. The blast overpressure is responsible for disruption of both air-fluid interfaces ('spallation') and tearing at fixed points of attachment ('brisance'), causing middle ear, lung and bowel injuries (Peters, 2011). At pressures above 200kPa, rupture of the tympanic membrane is an expected consequence, while the lung and bowel can remain intact up to pressures of 400kPa (Peters, 2011). Although these injuries predominate, there is evidence that the primary blast wave can also cause cerebral oedema, endothelial injury, liver and splenic lacerations, testicular rupture, retroperitoneal bleeding, and muscular compartment syndromes (Scott, 2017). Although tympanic membrane rupture is not predictive of a significant lung injury

(missing up to 50% of those with a significant blast lung injury) (Peters, 2011)

it may be a predictor of traumatic brain injury (Wolf, 2009)

<u>Overpressure (kPa)</u>	<u>Blast Loading</u>
35	Minimum pressure at which tympanic membranes will rupture
104 – 121	Pressure at which half of tympanic membranes will rupture, minimum threshold for blast lung injury
202	All tympanic membranes should have ruptured, minimum pressure for fatal blast injuries
290 – 390	50% fatality rate from pulmonary blast injuries
400 – 550	95 – 100% fatality from pulmonary blast injury

**Table 4.** Sequence of organ injury is dependent on the maximum pressure exposure (Peters, 2011).

### Auditory Injury

The auditory system is especially vulnerable to the effects of primary blast injury. The vestibular system, which controls balance and equilibrium, is also frequently injured leading to persistent dizziness and vertigo. Most dizziness resolves within 4-30 days of injury; persistent dizziness after six months is a poor prognostic indicator (Akin, 2017).

The most common injury is tympanic membrane rupture, due to overpressure, occurring in 20-44% of the blast-exposed (Engles, 2017). Overpressure causes a frequency dependent reduction in stiffness of the tympanic membrane, creating a conductive hearing loss, and is often associated with disruption of the ossicular chain (Van Haesendonck, 2018). The common symptoms of a tympanic membrane injury are middle and high frequency conductive hearing loss and hyperacusis (Mizutari, 2019). The conductive hearing loss is usually temporary, with a spontaneous healing rate of 38-74% in a traumatic rupture (Mizutari, 2019). If one ear is facing toward the blast wave, “head shadow” may protect the contralateral ear from injury (Van Haesendonck, 2018).

The blast overpressure can also damage the inner ear and cause a sensorineural hearing loss and tinnitus. Permanent hearing loss after blast injury is more likely to be a sensorineural loss than conductive (Aşık, 2018). A third of those with instant sensorineural hearing loss will recover spontaneously within minutes (Mizutari, 2019). 80% of those injured in the Boston Bombings had a sensorineural hearing impairment and 38% of soldiers in the Iraq war suffered from tinnitus on return to their home countries (Mizutari, 2019). Permanent tinnitus and sensorineural hearing impairment are associated with cognitive and depressive disorders and can have a major impact on quality of life after blast trauma (Mizutari, 2019)

### Cardiothoracic Injury

Pulmonary barotrauma, the most common fatal PBI, may include:

- 1.) Pulmonary contusion.
- 2.) Systemic air embolism.
- 3.) Haemopneumothoraces, pneumomediastinum or subcutaneous emphysema
- 4.) Acute respiratory distress (ARDS) occurs as a direct result of lung injury, or from shock due to other organ injuries.

5.) Pulmonary oedema, with increased pulmonary capillary permeability (or “blast lung”)

Non-fatal PBLI can impair pulmonary function for many days. The heart, lungs, and great vessels are protected by the thoracic cage but are vulnerable to blast overpressure and penetrating injury.

Primary blast lung injury (PBLI) is defined as the “radiological and clinical evidence of acute lung injury, occurring within twelve hours of exposure, and not due to secondary or tertiary injury” (Scott, 2017).

*In available military data, approximately 6-11% of casualties who survive to reach hospital have a PBLI, as do 80% of non-survivors (Scott, 2017). In civilian casualties, the incidence is much higher, owing to the increased association with an enclosed-space explosion. In the Madrid train bombings, 94% of the serious casualties were diagnosed with PBLI (Scott, 2017). The risk of a significant lung injury is both related to the proximity to the blast, but also the positioning of the victims relative to their surroundings.*

<b>Diagnostic criteria applied within 12 hours from the time of the blast</b>
Exclusion of secondary or tertiary lung injuries: <ul style="list-style-type: none"> <li>▪ Penetrating thoracic injuries</li> <li>▪ Rib fractures, clavicles, scapulae</li> </ul>
Objective evidence of lung injury based on the presence of both radiological abnormalities and internationally agreed oxygenation criteria for acute lung injury

**Table 4.** Stringent criteria for the detection of primary blast injury of the lung (Mackenzie, 2013)

Distinguishing primary blast lung injury from those who have a combination of primary, secondary and tertiary blast injuries does not appear to have any impact on prognosis and is of academic interest only (Mackenzie, 2013). In reality, most will have a combination of lung injuries, including thoracic fractures (ribs, clavicles, scapulae, vertebrae, sternum), pulmonary contusions and lacerations, impalement, crush injuries, asphyxiation, inhalational and aspiration injuries.

Blast wave injury to the lung manifests as alveolar capillary rupture with widespread striped pulmonary haemorrhages, corresponding to the intercostal spaces which are least protected from the blast

overpressure (Wolf, 2009) (Mackenzie, 2013).

Parenchymal tears may create small or large localised air pockets (‘blast emphysema’ or ‘pneumatoceles’) or may result in a pneumothorax (Mackenzie, 2013).

The natural history of blast-related lung injury can be classified into three phases: Primary, Early, and Late, summarized in Table 5.



Blast-related Lung Injury	Time from Blast to Injury	Mechanism	Time from Blast to Manifestation	Lung Pathology	Clinical Manifestations
<i>Primary</i>	Milliseconds – seconds	Blast wave	Mins – hours	Scattered foci of alveolar haemorrhage Alveolar disruption ('blast emphysema') Parenchymal laceration Pneumothorax	Apnoea followed by tachypnoea Low saturations Haemoptysis
<i>Early</i>	Seconds – minutes	Blast ejectate Blast wind Building collapse Crush injury Airway obstruction Smoke inhalation Aspiration Bone fractures	3 – 18 hours	Contusion Laceration Haemothorax Negative pressure pulmonary oedema Inhalation/aspiration Pneumonitis Fat embolism	Tachycardia Hypotension Hypovolaemia Pulmonary oedema Hypoxia CO-Hb Tension pneumothorax Chest pain
<i>Late</i>	Hours to days	Over-resuscitation LV dysfunction Massive transfusion Injurious mechanical ventilation Pulmonary inflammation	8 – 36 hours	Fluid overload Coronary air/fat emboli TRALI VALI ARDS	Poor lung compliance V/Q mismatch Pulmonary infiltrates Hypercapnia Hypoxia Cough Acidosis Leucocytosis LV/RV dysfunction on echocardiography Fever

**Table 5.** The natural history of blast-related lung injury. (Mackenzie, 2013).

In 17% of those deceased at the scene of the blast, a PBLI is the only significant finding at autopsy (Scott, 2017). The mode of death in this population is not fully understood, but study of animal models suggest it may be related to air embolism, or to the immediate autonomic nervous system response to a PBLI (Scott, 2017).

Injury to the pulmonary vessels allows air to enter the circulation. This has been demonstrated on carotid Doppler within 30 minutes following a controlled blast injury to a beagle (Mackenzie, 2013). Air entrained into the pulmonary veins can lead to systemic arterial air embolism, which have been located in cerebral, coronary and renal circulations (Mackenzie, 2013). The signs and symptoms of these are similar to other sources of systemic emboli. Post-

mortem evidence of significant air embolism has been shown in several animal studies with isolated PBLI, and demonstrated in a small number of humans, however due to delays in performing autopsies and failure to recognise it in survivors mean that the prevalence is largely unknown (Mackenzie, 2013).

Blast injury to the thorax can cause a vagal reflex arc, resulting in apnoea, bradycardia, and hypotension. It can be thought of as a "massive, sudden Valsalva manoeuvre". With a moderate injury the apnoea is transient, followed by a period of rapid, shallow breathing. However, with severe blast injury, the apnoea may be prolonged and be the primary mode of death (Scott, 2017). The treatment of blast lung injury is

mostly supportive, comprising pleural decompression, avoiding mechanical ventilation if possible, utilising a lung-protective strategy if ventilation is necessary, and hyperbaric oxygen therapy for arterial air embolism (Wolf, 2009) (Mackenzie, 2013). Minimising volume resuscitation may reduce the burden of pulmonary oedema but may conflict with treating the haemodynamic instability associated with blast multitrauma (Wolf, 2009).

Thoracic PBI can also result in pericardial tamponade without penetrating trauma (i.e. secondary blast injury) (Ciraulo & Frykberg, 2006). (O'Neill, Robinson, & Ingleton, 2012)

### Central Nervous System Injury

Blast traumatic brain injury is referred to as the "invisible wound" (Shively, 2016). The proportion of soldiers returning from Afghanistan and Iraq with mild traumatic brain injury ranges from 4.4% - 22.8%, and a large proportion of these are blast-related (Campos-Pires, 2018).

Symptoms range from headache, to depression, anxiety, post-traumatic stress disorder, sleep disturbance, poor concentration and memory impairment (Shively, 2016). There are rarely any structural changes observed with conventional imaging techniques (MRI) (Shively, 2016).

In a human post-mortem case-series, the blast wave caused injury to grey-white matter interfaces, areas lining the ventricles/adjacent to the cerebrospinal fluid and around blood vessels (Shively, 2016). These changes were seen in both acute blast-injured post-mortem brains, and also post-mortem brains of those who had a repeated history of blast trauma. The postulated mechanism is either via direct

injury from the blast overpressure disrupting air-fluid interfaces or, via energy transferred to the torso which may propagate pressure waves via blood vessels or cerebrospinal fluid (Boutillier, 2019).

### Abdominal and Pelvic Injury

The air-filled structures in the abdomen are most vulnerable to the blast overpressure, and the incidence of injury increases in enclosed spaces or underwater (Wolf, 2009). Primary rupture tends to occur in the colon and ileo-caecal regions due to their fixed points of attachment. Contusions can occur anywhere along the bowel wall, and may separate the intramural layers with micro-haemorrhages and oedema, leading to delayed perforation (Wolf, 2009), with potential for peritonitis or collections. The incidence of injury to the gastrointestinal tract and solid organs during blast trauma is approximately 14-24%, the majority being penetrating injuries caused by shrapnel (Wolf, 2009), (Yazgan, 2016). The mesentery may be injured directly, leading to bowel ischemia. The bowel is also vulnerable to arterial air embolism (Wolf, 2009), (Scott, 2017). Vascular injury within the abdomen is non-compressible; sources may be within the mesentery, associated with a pelvic fracture, or in the retroperitoneal space. Non-compressible torso bleeding is associated with poor outcomes (Pearce, 2017), however there are trials ongoing in the field with resuscitative endoluminal balloon occlusion of the aorta (REBOA) to prolong survival to hospital to allow for definitive management. Whether this will be suitable in the immediate aftermath of an explosion is unclear.

Extended focused abdominal sonography in trauma (eFAST) is a useful screening test; but contrast computed tomography is

the modality of choice for detecting intra-abdominal injuries and is useful in locating fragments of shrapnel. It will not detect all mesenteric injuries or contusions, and in those with clinical signs of intra-abdominal injury, serial abdominal examinations over 24 hours or more are advised (*Wolf, 2009*).

### Extremity Injury

Extremity wounds are associated with a higher ISS, mortality and longer hospital stay (*Ashkenazi, 2018*). Up to 15% of bombing victims will have an extremity injury, and a quarter of these will present with haemorrhagic shock. Up to 7% of those injured by an explosion have traumatic amputations, associated with high immediate mortality rates of between 10 and 85% (*Wolf, 2009*). The overpressure can cause fractures and the blast winds can rupture soft tissues resulting in a partial or complete limb amputation (*Wolf, 2009*).

Those with extremity injury benefit from early haemorrhage control, which can be achieved via external combat tourniquet application or surgical cut-down and clamping of proximal vasculature by skilled personnel in the field, whilst awaiting a definitive surgical management. Preventative wound care involves early scrub-down, low-pressure irrigation, and antibiotic administration. Immobilising fractures will control pain, reduce bleeding, and prevent secondary infection (*Ignatiadis, 2019*), (*Ashkenazi, 2018*). Compartment syndrome is an early complication of the fractures, crush injury and burns associated with blast trauma and account for 86% of fasciotomies performed in combat casualties (*Wolf, 2009*).

Debridement of extremity wounds should be performed within 24-48 hours, and

definitive surgical repair within a week. Blast injuries are usually a contraindication to reimplantation. Often limbs can be salvaged initially but may require many revision surgeries.

### Clinical Implications in the Tactical Environment

Responding into a tactical environment risks exposure to explosive devices. There are multiple potential mechanisms of injury. Injuries in a specific scenario are predictably influenced by environmental factors, e.g. enclosed spaces or building collapse.

Controlled detonations in enclosed spaces, or adjacent to solid objects, increases consequences of blast wave due to reflections.

### Pitfalls

The clinical consequences of exposure to a blast wave may not be obvious for a period of time.

The terminal ballistics of shrapnel is unpredictable. Injuries may occur some distance from the expected path of the projectile.

### Conclusions

Despite the disruptive efforts of law enforcement and intelligence organizations, explosive material and expertise is still accessible to criminal and terrorist organisations. Safe and effective response is enhanced by an understanding of the physics and applied pathophysiology of these events.

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**Addendum**  
**Further Reading**

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