Ministry of Defence

Synopsis of Causation

Blast Injury to the Thorax and Abdomen

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Disclaimer

This synopsis has been completed by medical practitioners. It is based on a literature search at the standard of a textbook of medicine and generalist review articles. It is not intended to be a meta-analysis of the literature on the condition specified.

Every effort has been taken to ensure that the information contained in the synopsis is accurate and consistent with current knowledge and practice and to do this the synopsis has been subject to an external validation process by consultants in a relevant specialty nominated by the Royal Society of Medicine.

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1. Definition[1]

1.1 Blast injury describes any injury caused by the effects on a living organism of a blast wave generated by an explosive detonation.

1.2 An explosion results from the instantaneous release of a large amount of energy from the detonation of an unstable source which produces the blast. The blast wave comprises two parts – a high pressure shock wave followed by a 'blast wind' due to movement of displaced air. Heat generated by the explosive reaction is disseminated in the blast wind which can displace objects and cause incineration of inflammable material.

1.3 There are three sources of explosive detonation:

- **Chemical reaction** - such as gelignite, trinitrotoluene (TNT), nitroglycerin, Semtex and gunpowder, where detonation produces a rapid and violent chemical reaction, which results in the sudden release of gas and heat which in turn generates a high central pressure.

- **Nuclear reaction** - due to fusion or fission of atomic nuclear particles in unstable elements such as uranium or plutonium producing a vast amount of instantaneous energy.

- **Physical reaction** – due to an excessive build up of pressure within a container which ruptures when the internal pressure overcomes the restraining force of the structure, for example when a gas bottle becomes overheated and the container explodes.

1.4 Chemical explosives are grouped as High-order or Low-order explosives. High-order explosives such as Trinitrotoluene (TNT), Semtex, nitroglycerine and dynamite produce a supersonic wave of overpressure in air. Low-order explosives such as gunpowder or petroleum-based devices produce a subsonic blast and, although they may produce secondary missiles and cause burns, they are unlikely to produce primary blast injury.[2]

1.5 Conventional bombs using high-order explosives produce a blast wave which starts centrally at the source and spreads outwards radially. Some types of modern military ordnance utilise enhanced-blast devices which generate an initial blast which activates a secondary explosion resulting in a much wider source from which the blast wave emanates.[3]

1.6 Explosive detonation can be initiated by a heat source, an electrical charge or a chemical reaction. Detonations which occur within a confined space or which are confined by a restrictive container tend to result in enhanced energy release when the container ruptures under the pressure of the explosive reaction. An explosion can thus produce a variety of physical effects which cause injury such as rapid change in atmospheric pressure, production of heat and emission of light.

1.7 Nuclear explosions produce extreme levels of energy with high pressure blast, extreme heat and light and have the added consequences of the emission of ionising radiation

from both the source of the explosion and from fallout of radioactive debris.

1.8 Explosions occur in many circumstances and have become increasingly common over the past 80 years through increasing industrialisation, the development of increasingly potent and exploitable explosives, the proliferation of advanced weapons systems, the scope and nature of recent conflicts and the increase in indiscriminate terrorist use of explosive devices.

2. Clinical Features[1,4]

2.1 The effects of blast are traditionally divided into four categories of injury:

- **Primary** blast injury is due to the effect of the atmospheric overpressure of the blast wave that emanates from a detonation and primarily affects the air-containing organs of the body such as the lungs, intestine and middle ear. Shearing forces caused by the rapid change in pressure may also cause injury to other organs.

- **Secondary** blast injury is due the effect of projectiles activated and set in motion by the blast being dispersed at speed with high kinetic energy causing penetrating wounds or blunt contusion injury. Projectiles can be derived from the container of the explosive, or from parts of a vehicle or other items in the vicinity of the explosion. In military ordnance the casing of an explosive weapon is designed and constructed in a way which produces fragmentation on detonation (shrapnel). Many devices constructed by terrorists are packed with metal objects such as bolts or nails to produce multiple projectiles intended to maximise casualties on detonation.

- **Tertiary** injury is caused by the individual being thrown through the air by the force of the blast. Injury is caused by contact with solid obstacles with resulting contusion, limb and spinal fractures or additional penetrating wounds. It includes limbs being torn from the body by the force of the explosion.

- **Quaternary or miscellaneous.** Quaternary injuries cover a miscellaneous group of effects including burns from ignited clothing, flash burns from the effect of hot air in the blast and inhalation of hot air or toxic residue from the explosive reaction. Associated effects such as infection or radiation toxicity are also included in this category.

2.2 The blast wave can cause a wide range of injuries to multiple parts of the body and incidents can produce a varying number of casualties depending on the location and situation of the detonation. Severity of injury generally depends on the proximity of the individual to the centre of the explosion. Mortality is high, with those within close vicinity being most likely to die before medical attention can be given. In a series of 828 cases of service personnel who were victims of explosions in Northern Ireland, 216 (26%) were killed, most of whom died before treatment could be initiated.[5]

2.3 Injuries can include traumatic amputations, fractures, penetrating wounds, burns, eye injury, perforation of the eardrum and primary blast injury to the air-containing organs of the thorax and abdomen. Haemorrhage, hypovolaemic shock and respiratory distress complicate the picture and management of victims of blast poses a significant challenge to those providing immediate care at the location and at emergency treatment centres.

2.4 **Injuries to thorax and abdomen.** Air is much more easily compressed than water, and organs which contain air are more susceptible to extreme changes in pressure. The lungs and intestine are therefore particularly prone to damage from the effects of overpressure, although penetrating wounds, contusive effects and the effect of associated fractures in the chest or abdomen can add to the pattern of injury.

2.5 **Blast Lung.** The syndrome of blast lung forms a clinical entity and is due to disruption of the tissue structure of the lung by the rapid compression and expansion of the air within the air passages and alveoli. This is complicated by biochemical changes in the lung and the circulating blood due to breakdown of haemoglobin and fat by oxidation.

2.5.1 Blast lung is diagnosed from clinical findings with a related history of exposure to explosive detonation and should be actively considered in all casualties within close proximity to the explosion. Observation of the patient for the effects of blast lung should be implemented in all who have been in the near vicinity of an explosion with monitoring of blood oxygen levels to identify early signs of blast lung. All patients with a perforated tympanic membrane should have a chest x-ray as soon as possible. The absence of tympanic membrane rupture was considered to be a negative indicator of potential lung damage as the pressure at which the lung ruptures is higher than that for the eardrum. However a recent case study of a series of 640 civilian casualties of terrorist bombings in Israel found that 18 patients with blast lung had no evidence of tympanic membrane rupture, throwing doubt on a correlation between tympanic membrane injury and blast lung.[6]

2.5.2 The typical features of blast lung are dyspnoea, cough which may be dry or sometimes productive of frothy sputum, chest pain which is usually retrosternal and haemoptysis. Physical signs may show tachycardia, cyanosis, dullness to percussion and signs of pneumothorax. Auscultation may reveal coarse added sounds, both crepitation (crackles) and rhonchi (wheezes), due to pulmonary contusion and oedema. The condition can usually be confirmed by chest x-ray.

2.5.3 Radiographic changes comprise diffuse pulmonary opacities sometimes with the characteristic appearance of 'butterfly lung'. These features develop within 2 hours and progress over 24-48 hours. Serial radiographs may be needed to identify and monitor progress of these opacities which represent areas of exudates formed from exudation of fluid into interstitial tissue and alveoli with congestion of pulmonary veins and arteries. Radiographic signs of pneumothorax may also be seen, as may structural injuries such as rib fractures or foreign bodies which have penetrated the chest wall. Computed tomography (CT) scanning may identify the early changes in some cases where the condition is not immediately apparent. [2,7]

2.5.4 Blood gas analysis shows hypoxia and hypocapnia. Blood analysis may show an altered acid/base balance or the effects of carbon monoxide inhalation.

2.6 **Heart and circulation.** The heart may be directly injured by blast with resulting haemorrhage and haematoma formation below the epicardium or endocardium, laceration of cardiac muscle or damage to the valve mechanism. Endocardial haematoma may occur around the papillary muscles and epicardial haematoma is more common on the diaphragmatic surfaces. Disruption of the structure of the coronary arteries by tearing of the vessel wall or subintimal haemorrhage may lead to myocardial infarction. Air embolism of the coronary arteries may also cause myocardial injury and is a significant cause of death soon after the injury. Cardiac arrhythmia may result from either myocardial damage or through dysfunction of the autonomic nervous system from cerebral embolism or direct damage to nerves. Damage to blood vessels may cause arteriovenous fistula, and blockage of the vessel or rupture of the vessel wall may

complicate the picture. Disseminated intravascular coagulation may also occur. Hypotension due to hypovolaemic shock is common.[1]

2.7 **Abdomen and pelvis.** The gas-containing intestines are vulnerable to the effects of overpressure. Complete rupture of the gut wall with escape of intestinal contents into the peritoneal cavity and haemorrhage due to ruptured blood vessels may present with clinical signs of peritonitis or concealed haemorrhage. Shearing injuries to the omentum, the spleen, liver, bladder, uterus, testes or kidneys may also present signs of acute abdominal injury. Secondary effects from penetrating projectiles or tertiary effects of blunt trauma may also need to be excluded. Abdominal injuries may not be immediately apparent as symptoms may develop insidiously. Careful observation is needed to ensure these effects are not overlooked.

2.8 **Musculoskeletal.** Traumatic contusions, penetrating wounds, limb and spinal fractures, and amputations may occur. The associated effects of crush injury or severe burns may result in compartment syndrome with rhabdomyolysis, hyperkalaemia and renal failure.[4]

3. Aetiology[1,8]

3.1 **Explosive overpressure.** The pressure wave generated by a high-order explosive detonation results from the sudden rapid production and expansion of gaseous products from the chemical reaction of the explosion. Heating and subsequent displacement of the surrounding atmosphere by the heat generated in the reaction adds to the effect.

3.1.1 The primary pressure wave-form shows an immediate rise in pressure reaching a peak level of overpressure within milliseconds, followed by an exponential fall in pressure over the succeeding milliseconds and a negative trough of under-pressure of considerably less amplitude than the initial peak. The pressure wave spreads outwards in a radial fashion with a fall-off in amplitude of the peak overpressure in proportion to the distance it travels from the epicentre.[1] The process spreads at supersonic speeds of up to 5000 m/sec. Lung rupture is likely at an overpressure of more than 100 kPa (15 psi) above atmospheric pressure. Higher levels may be needed to rupture the gas-containing organs of the abdomen. The tympanic membrane is likely to rupture at lower levels of overpressure at around 40 kPa (6psi).[4] The likely survival rates from blast injury are related to the maximum incident overpressure and its duration, which falls exponentially with time. Exposure to initial blast pressures of 1000 psi carries a survival rate of only 1%.[9]

3.1.2 The magnitude of a blast wave varies according to the nature and quantity of the explosive involved, with the range of effect depending on the degree of confinement within solid surroundings. The intervention of large obstacles also affects the rate of decay of the wave energy.

3.1.3 In air, the force of the pressure wave declines by the cubed root of the distance travelled, so that an individual 3 m from the explosion would experience nine times greater overpressure than someone 6 m away.[4] Because of the rapid rate of decay, the radius of effect of the blast wave is significantly less that that of the projectiles energised by the explosion. The blast wave in its idealised form tends to occur only within close proximity to the epicentre of the explosion as surrounding rigid structures (such as buildings or walls) reflect the wave, resulting in a complex wave-form. Oscillatory features can develop briefly making it difficult to predict the likely extent of overpressure at any given point.

3.1.4 Thus, an explosion in open countryside is more likely to produce the typical primary wave-form of overpressure, but primary blast injuries are likely to be confined to those who are in close proximity of the detonation. With greater distances from the epicentre, casualties are more likely to suffer secondary injury from projectiles. Depending on the magnitude of the explosion, tertiary injury is more likely in individuals who are close to the epicentre, where the pressure wave is intense enough to hurl the individual through the air. A study of 297 victims of four bombing events compared the injuries of those who had been in open air with those who had been in an enclosed space. Similar explosive devices had been used in all four incidents. Of the 204 victims involved in open-air bombings, 15 (7.8%) died. In contrast, 93 victims were involved in bus bombings and, of these, 46 (49%) died. Primary blast injuries in those who were admitted to hospital occurred in 25 (12%) in the open air and 31 (33%) in the bus bombings, adding weight to the anecdotal view that explosions

in confined spaces cause a higher incidence of primary blast injury and a higher mortality rate.[10]

3.1.5 The pattern of explosion underwater is different. As well as a pressure shock wave transmitting through the water, gas produced by the explosion forms a large bubble, which expands as its rises. On reaching the surface, the shock wave reflects downwards and increases the rate of decay of the wave-form. As the gas bubble erupts from the surface, it produces a negative pressure at the surface which has the overall effect of sucking up a dome of water droplets. The resultant pressure gradient is such that at the surface the overpressure is significantly less than at depth. Individuals who are in the water at the time of an explosion are more likely to suffer blast injury when deeper in the water and, if they are treading water, the lower parts of the body are more likely to be affected by blast. Air-containing organs are more easily compressed than solid tissue, and primary blast injury is likely to predominate as the density of water restricts the movement of projectiles, so there may be no other sign of injury in immersion explosion casualties.[1]

3.1.6 In circumstances other than those where exposure to the blast occurs while immersed in water, conventional explosions are unlikely to produce solitary primary blast injury. Because of the rapid decay of the blast wave most victims who are close enough to suffer the effect of overpressure are close enough to be subjected to the displacement of projectiles, and secondary and tertiary injuries are almost inevitable. In enhanced-blast detonations the pressure wave is generated over a much wider area, with a prolonged phase of overpressurisation causing a higher proportion of primary blast injuries.[3]

3.2 The initial effect of a blast wave as it strikes the surface of the body is to compress and displace the surface tissues. This may occur with sufficient force to cause surface contusion. Pressure is then transmitted through the body in the form of both stress waves and shear waves.[1,11]

3.2.1 Stress waves are similar to sound waves but have high amplitude and velocity which generate high local forces, with rapid microscopic distortions of the tissue. Reflection of the wave at interfaces with more solid structures enhances tissue distortion. Rapid compression and rapid rebound over-expansion of spaces in gas-containing organs results in tearing of the wall or lining of the structure. In the lung this results in damage to the delicate walls of the alveoli, and in the abdomen causes contusion and tearing of the wall of the intestine with possible rupture.

3.2.2 Shear waves are longer duration, low velocity waves formed by the compression of the body wall and larger organs. These produce tearing between tissues of different density and can result in larger internal lacerations and contusion.

3.3 **Pathological changes in the lung** Animal studies of the effect of blast overpressure have shown typical changes of rupture of alveolar septa, pulmonary haemorrhage and oedema, with subsequent alveolar flooding and respiratory insufficiency. In humans this would result in adult respiratory distress syndrome (ARDS).

3.3.1 A study in 2003 looked at the post-mortem effects of blast injury to the lung in a series of eight cases in which there was no evidence of associated disruption of the thoracic cavity or penetrating injury to the chest. The findings were compared with a control group, matched for age, who had not been exposed to blast.

3.3.2 Macroscopic examination of the blast victims' bodies showed multiple superficial lacerations and contusions, surface impaction of foreign materials, abrasions and flash burns. Mutilation and fractures of the upper extremities and rupture of the spleen were seen in several cases.

3.3.3 Microscopic findings in the lungs showed a predominance of alveolar rupture, thinning of the alveolar walls, and enlargement of the alveolar spaces. Electron microscopy showed thinning and micro-perforation of the alveolar walls as well as more gross changes. Sub-pleural and perivascular haemorrhage and haemorrhage into the alveoli were also seen. Congestion and vascular engorgement of the pulmonary arteries, arterioles, veins, venules and alveolar capillaries were also present. Tissue oedema around the alveoli was also a feature in those who survived for longer.

3.3.4 Venous air embolism was seen in four cases. As none of this group had survived long enough for artificial ventilation to be carried out, this was probably a primary effect of blast. Pulmonary fat embolism was also seen in three cases and in one of these it was extensive. Survival time in this subgroup was less than twenty minutes. Pneumothorax with collapsed lung was present in some.

3.3.5 These changes produce gross disruption of lung function with abnormalities in both ventilation and perfusion of the lung, resulting in marked changes in blood gas chemistry.[12]

3.3.6 In addition to the structural pathological changes that occur, it has become apparent that haemorrhage can produce effects from the oxidative breakdown of haemoglobin to methaemoglobin and other oxidants which further disrupt lung function. These biochemical changes appear to have more widespread systemic effects through 'oxidative stress', which is associated with breakdown of further haemoglobin and loading of the circulation with products which impair cardiac and renal function.[13,14] A more recent study of this effect in animals has shown that overloading with antioxidants before exposure to blast reduces the effects of oxidative stress with restored haemoglobin oxygenation, supporting the view that this factor increases the effect of blast and has possible implications for future development in the management of the condition.[15]

3.4 The heart may also be involved in the effects of blast injury. Direct injuries such as endocardial contusions and lacerations and haematoma of the epicardium may result from shearing forces. Direct myocardial injury is infrequent, although myocardial infarction due to damage to the anterior descending coronary artery may occur. Myocardial injury is more likely to be secondary to air embolism in the coronary arterial system. Tearing of vessel walls can directly injure blood vessels or blockage can occur from air embolism, fat embolism or thrombosis.[1] Acute anteroseptal myocardial infarction, presumably due to subintimal haemorrhage from a tear, has been noted in a

subject exposed to blast with no other obvious injury and no evidence of existing atherosclerosis.[16]

3.4.1 It has been apparent that other cardiac effects from blast can occur without any apparent direct damage to the heart. These effects have been investigated in animal studies, with a group of ten rats being exposed to blast, half to the thorax and half to the abdomen. Those exposed to thoracic blast showed changes of transient apnoea, bradycardia and hypotension. None of those exposed to abdominal blast showed any significant cardiopulmonary responses. This effect appears to be a reflex reaction, possibly mediated through the vagus nerve and supports anecdotal observations of collapse of victims who have no other significant blast injury.[17]

3.5 Abdominal and pelvic organs are also at significant risk from blast injury, particularly in immersion blast exposure. High overpressures can cause localised rupture of the wall of the gut, with associated bleeding and release of intestinal contents into the peritoneal cavity. Haemorrhages are common and vary from minor petechiae to more substantial haematomas of the mucosal lining of the gut, particularly at lower-pressure blast exposure. Extensive bleeding from the wall of the gut may result, and secondary necrosis of the gut wall with delayed perforation may follow haemorrhage. Tearing of the mesentery due to shearing forces may also be found. Rupture of abdominal organs such as the spleen and liver and retroperitoneal haematoma have been described in connection with primary blast injury but the picture may also be complicated by similar damage from secondary or tertiary injury. The effects of primary abdominal blast injury may not be immediately obvious until signs of concealed haemorrhage, peritoneal irritation or intestinal obstruction become manifest.[1,4]

4. Prognosis[1,3]

4.1 Management of patients with thoracic and abdominal blast injuries represents a significant challenge at initial triage and subsequent stages of care. The patient usually has multiple injuries to numerous parts of the body, some obvious, some concealed, some superficial and some life-threatening. Early identification of blast lung, cardiac damage and hidden abdominal injury is crucial to patient survival.

4.2 The severity of tissue damage from blast depends on the nature and location of the explosion and the proximity of the individual to the source of detonation. Victims close to the source of the explosion are likely to suffer severe multiple injuries as well as blast injury. The distance involved tends to be proportional to the magnitude and type of explosion. Fatal injury is more likely to be higher for those close to the source of the blast.

4.2.1 These effects are well illustrated in a study of a bombing incident in a public house in Birmingham. There were eleven fatalities out of more than 100 people in or near to the premises at the time. Seven of those who died could not be accurately placed within the area at the time of the blast but were probably within a range of three metres. However, two of the fatalities were placed within two metres of the bomb when it went off. Of those who sustained very severe injuries, most were within three to four metres of the source. However, one person who died was seated at a corner table three alcoves away and was surrounded by at least 15 people who sustained only superficial injuries or no injury. Twelve passers-by who had been outside the public house at the time of the blast were identified. Of these, seven suffered injuries that needed treatment in Casualty, four had no injury and one (who was adjacent to the source of the blast) had what were classed as serious injuries. Therefore although there was some disparity between incidence of fatality and the proximity to the explosion, the most severe injuries were sustained by those who were within three metres of the blast.[9]

4.3 Initial management must be aimed at establishing basic life support, with attention to maintaining the airway, sustaining respiration and supporting circulation. This includes control of haemorrhage, management of fractures and traumatic amputation and prevention of further injury.

4.4 Supported respiratory ventilation with oxygenation is needed in many cases. Some victims may be apnoeic immediately following the blast, and simple bag and mask ventilation may be necessary at the site of the incident. Endotracheal intubation should be instituted as soon as practicable to maintain more prolonged artificial ventilation. The risk of air embolism from positive pressure ventilation has led to the trial of a variety of methods of ventilation (such as limited peak inspiratory pressure with permissive hypercapnia, intermittent mechanical ventilation and high frequency jet ventilation) which have had variable degrees of success.[18]

4.5 For those who respond to treatment, respiratory function starts to recover within two weeks. Although the effects of blast injury have been studied extensively, there appears to be little information on the longer-term effects of the injuries. One study reports the results of follow-up over one year of a series of 11 victims of two terrorist bombs who had been treated for blast lung. Most of these had multiple injuries as well as blast lung.

All had sustained eardrum perforation and ten required assisted ventilation. There were six men and five women, with the average age of the group being 28 years +/- 9.8 years. They were followed-up at respiratory clinics at regular intervals. At twelve months following the injuries, none had any lung-related complaints and none had any residual clinical signs in the chest. Apart from two cases, pulmonary function was within the expected normal range. One patient showed an obstructive pattern of impairment associated with probable pre-existing chronic obstructive pulmonary disease from smoking. One patient showed some loss of lung volume as well as an obstructive element. Exercise capacity was almost fully recovered in all. This group all initially had substantial bilateral chest x-ray changes indicative of lung contusion and exudates. These changes started to resolve within one week and had returned to normal within five weeks. At twelve months the chest x-rays remained completely normal. For this young and relatively healthy group, recovery from blast lung injury was effectively complete within one year.[19]

4.6 Little information on the long term effects of cardiac blast injury has been found. Standard methods of circulatory support and medication are needed for the immediate treatment of cardiac arrhythmia and myocardial infarction. Residual cardiac damage depends on the extent and location of direct cardiac injury or myocardial infarction and subsequent development of complications such as heart block.

4.7 Follow-up studies of the effects of abdominal blast injury are also sparse. Exploratory laparotomy with subsequent surgical debridement and repair is needed in many cases of suspected blast-induced haemorrhage or perforation. Penetrative injuries from secondary blast effects also require surgical intervention. In some cases where the abdominal effect appears to be limited to minor petechial haemorrhage, expectant conservative treatment may suffice. Intraperitoneal fibrosis has been reported as a late complication of primary abdominal blast injury.[1] Intestinal obstruction from small bowel adhesions arising 50-60 years after abdominal blast injury has also been described.[20]

5. Summary

5.1 The gas-containing organs of the thorax and abdomen are susceptible to the primary effects of blast-induced atmospheric overpressure. Injury results both from the effects of stress waves, which cause rapid compression and rebound expansion of air spaces, and shear waves, which cause differential shearing and tearing of tissues of differing density within the chest and abdomen.

5.2 Secondary and tertiary effects of blast may also cause injury to the chest and abdomen by penetration and contusion.

5.3 Because of the rapid decay of the blast pressure wave in air, primary blast injury tends to affect victims who are close to the source of detonation and is usually associated with multiple injuries from secondary and tertiary effects. Primary blast injury occurs as a sole injury mainly in those exposed to enhanced-blast detonations or those immersed in water.

5.4 Primary blast lung must be considered in all casualties who have been subjected to blast, but particularly in those who have perforated eardrums, although the absence of eardrum perforation does not exclude the presence of blast lung injury.

5.5 Primary abdominal blast injury tends to occur at higher blast pressures or from immersion events.

5.6 Treatment of blast injury encompasses substantial life support measures, management of respiratory distress, support for cardiovascular effects and dealing with intra-abdominal haemorrhage or perforation of the bowel.

5.7 For those who respond to initial treatment of blast lung the long term prognosis appears to be good, with a high chance of regaining full respiratory function.

5.8 Effects of cardiac damage may persist in the longer term, and there is some evidence that intraperitoneal fibrosis or intestinal obstruction may present at some time following blast injury to the abdomen.

6. Related synopses

Blast Injury of the Ear

Compartment Syndrome

7. Glossary

air embolism	A bubble of air drawn into a damaged blood vessel which can travel through the circulation and cause an effective obstruction to blood supply to a vital organ, usually the lung or the brain.
alveoli	Pl. of 'alveolus'. The tiny thin walled air sacs within the lung at the end of the bronchial system through which oxygen enters and carbon dioxide leaves the blood.
apnoeic	Pertaining to apnoea – cessation of breathing.
arrhythmia	An irregularity or abnormality of heartbeat often associated with dysfunction of the heart or the mechanism controlling heart rate.
arteriovenous fistula	A condition where an artery becomes directly joined to a vein, causing the blood supply from the artery to be short-circuited and fail to reach the tissue it supplies.
autonomic nervous system	The part of the nervous system that controls functions such as breathing and heart rate that are not under voluntary control.
bradycardia	Slowing of the heart rate.
butterfly lung	An appearance on chest x-ray where opacities in the lung spread outwards bilaterally from the midline into the lung tissue giving the appearance of wings.
crepitation	A fine crackling sound heard through a stethoscope, usually indicating fluid within the air sacs of the lung.
cyanosis	A condition where the skin and mucous membranes develop a blue discoloration, indicating lack of oxygen in the tissues.
debridement	Surgical removal of foreign material and dead tissue from a wound to prevent infection and promote healing.
dullness to percussion	A physical sign elicited by tapping over a part of the body. When applied over air-containing organs, a dull note indicates consolidation and loss of air from the tissue.
dyspnoea	Breathlessness.
endocardial	Pertaining to the membrane lining the cavities of the heart.

embolus	Matter, usually a blood clot, which travels through a blood vessel and which can result in blockage of supply to a vital organ. Embolism – the resultant effect of an embolus.
epicardium	The inner layer of the fibrous sac (pericardium) that encloses the heart.
epicentre	The very centre or focal point of an explosive detonation.
exponential	A mathematical function or curve that changes by a logarithmic factor. A rapidly accelerating or decelerating change in rate.
haematoma	A collection of blood which may be partially clotted and usually confined within tissue by membranes or partitions, in this context caused by bleeding from damaged blood vessels.
haemoglobin	A protein in red blood corpuscles which carries oxygen in the blood stream.
haemoptysis	Spitting-up of blood, or sputum which contains blood.
hyperkalaemia	The state of having a raised level of circulating potassium in the blood serum.
hypocapnia/ hypercapnia	Hypocapnia - the state of a lowered level of carbon dioxide in the blood. Hypercapnia – the state of an elevated level of carbon dioxide in the blood.
hypotension	Low blood pressure.
hypovolaemic shock	A condition caused by loss of blood or body fluid, resulting in progressive failure of circulation.
hypoxia	The state of having too low an oxygen level in the blood.
intraperitoneal fibrosis	Fibrous tissue which develops at the site of injury or inflammation and which produces scarring and adhesions within the peritoneal cavity.
kPa	Kilopascal - Unit of gas pressure equivalent to 1000 Pascals. 101.3 kPa = 1 Atmosphere – the unit of air pressure equivalent to 14.7 psi (pounds per square inch).
laparotomy	Surgical exploration of the abdomen through a skin incision.
mesentery	The double-layered membrane attached to the back wall of the abdominal cavity that supports the intestine

necrosis	Death of tissue cells or of an organ caused by injury or disease.
oedema	An abnormal build-up of fluid between tissue cells which causes diffuse swelling of tissues.
omentum	An extension of the membranous lining of the intestine and surrounding organs forming an ‘apron’ over the coils of intestine.
perivascular	Pertaining to the zone of tissue surrounding a blood vessel.
petechiae	Pl. of petechia. Tiny purplish spots on the skin or in a membranous lining of a body organ caused by minute haemorrhages.
pneumothorax	A condition caused by air getting between the lung and the chest wall which results in collapse of the lung.
psi	Pounds per square inch - superseded unit of pressure.
retrosternal	Situated or occurring within the chest behind the breast bone.
rhabdomyolysis	A potentially life-threatening condition resulting from breakdown of muscle tissue and the escape of muscle contents into the blood. Often associated with crush injuries.
rhonchi	The wheezing sounds heard from the chest when breathing is impaired by obstruction of the air passages.
septa	Pl. of ‘septum’. Partitions or membranes separating two or more body cavities.
subintimal haemorrhage	Bleeding occurring between the vessel wall and the inner lining of an artery.
sub-pleural	Occurring between the membranous lining of the lungs and the underlying tissue.
tachycardia	A rapid heartbeat which can be of several forms, occurring naturally after exercise but also associated with abnormality of the heart or circulation.
triage	The process of prioritising sick and injured people for treatment according to the potential seriousness of the injury.
tympanic membrane	The membrane between the outer and middle ear which acts as an amplifier of sound – the ‘eardrum’.

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