Editorial

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Blast injury and the neurosurgeon

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Blast injuries are caused by explosions, which result from activation of explosives. A mind-boggling array of explosives exists which can inflict severe, lethal and sublethal injuries to humans, often in the form of mass casualties. Till recent times, these injuries were seen exclusively by military surgeons and physicians working in warzone scenario. This is no longer the case, with rising ethnic strife, terrorist bombings, easy availability of firearms and explosives to terrorist groups operating in civil areas. A new pattern of conflict is emerging, where the state is pitted against a determined clandestine group, who would have no qualms in killing hundred or even thousands of innocent people (even children, as seen in Beslan, Russia in 2005) so long such acts provide them with visibility and force the government of the day into negotiations in its own terms. Some of the prominent terrorist strikes against civilian populations have been:

(a) Serial Mumbai blasts in 1993
(b) Oklohama city bombing in 1995
(c) Simultaneous car bombings in US embassies in African countries in 1998
(d) World trade center bombing on Sep 11, 2001
(e) Bali bombings in Oct 2002
(f) Madrid in Mar 2004
(g) London transit system on Jul 7, 2005
(h) Delhi blasts in Oct 2005 & other blasts that rock the country at frequent intervals

TERRORISM

Terrorism is defined by the United Nations and terrorism expert AP Schmid as an, "anxiety-inspiring method of repeated violent action, employed by (semi-) individual, group or state actors for idiosyncratic, criminal or political reasons, whereby, in contrast to assassination, the direct targets of violence are not the main targets. The immediate human victims of violence are generally chosen randomly (targets of opportunity) or selectively (representative or symbolic targets) from a target population, and serve as message generators. Threat- and violence-based communication processes between terrorist (organization), imperiled victims, and main targets are used to manipulate the main target (audience), turning it into a target of terror, a target of demands, or a target of attention, depending on whether intimidation, coercion or propaganda is primarily sought". Terrorism occurs from ideological, racial or international conflicts and is likely to remain a problem for many nations in the foreseeable future.

Terrorism and blast injuries go hand-in-hand. Suicide bombings are the most dangerous yet effective forms destruction of the intended target, and are the most common form of violence in the middle-east. Former Prime Minister of India, Rajiv Gandhi was killed with scores of others by a suicide bomber of the LTTE in 1991. Trucks and car bombs are also used because these vehicles can carry explosives in large amounts without attracting attention. One compact sedan can hold up to 500 pounds of explosives, have a lethal blast range of 100 feet, and flying shrapnel/glass hazard of 1250 feet.

EXPLOSIONS AND EXPLOSIVE DEVICES

A bomb is basically a container filled with explosive material whose explosion is triggered by a timer or by impact. Terrorist bombs, also known as improvised explosive devices (IEDs), are usually custom made and may use any number of designs. These are of two types: conventional, which are filled with chemical explosives or dispersives, which are filled with chemicals and/or other projectiles such as nails, steel pellets, screws and nuts designed to disperse. Nuclear devices, which rely on nuclear fission or fusion, have not been seen to date in terrorist attacks. Bombs are relatively inexpensive to make with instructions readily available on the internet. Explosions are caused by a rapid chemical conversion

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of a solid or liquid into a gas with resultant energy release. Explosives are of high or low order.

High order explosives such as trinitrotoluene (TNT), Semtex, nitroglycerine, ammonium nitrate fuel oil, etc. are designed to detonate quickly, generate heat and loud noise, fill space with high pressure gases in 1/1000th second, and produce a blast wave. This blast wave is a supersonic hyper pressurisation shock wave that expands from the point of detonation outward in a pressure pulse, moving in all directions, exerting pressure of upto 100 tons. These waves have a shattering effect, as the displaced air then compresses and forms a vacuum returning to the point of detonation (negative wave)[1].

Low-order explosives produce a subsonic explosion without the blast wave. Energy is released relatively slowly and burns by a process of deflagration. Low order explosives include gunpowder, Molotov cocktails and pure petroleum based bombs. A list of substances considered to be explosive is available at www.atf.gov.

Effects of explosives: Explosives exert their lethal and damaging effects by several mechanisms. The blast wave has been described above. In addition, they damage and kill by fragmentation effect, blast wind, incendiary thermal effect, secondary blast pressure effects, ground and water shocks for subterranean or underwater explosions.

Fragmentation: Fragmentation effect occurs from projectiles included in the container, projectiles produced from the destruction of the container, and from objects surrounding the detonator and target. These projectiles can travel up to 2700 feet per second.

Blast wind: Blast wind is created by the motion of air molecules responding to pressure differentials generated by the blast. These winds may be as high as those seen in hurricanes but are not sustained.

Incendiary thermal effect: This factor is different for high- and low-order explosives. High-order explosives produce higher temperatures for shorter periods of time, usually resulting in a fireball at the time of detonation. Low-order explosives have a longer thermal effect and can cause secondary fires.

Secondary blast pressure effects: These are caused by the blast wave's reflection off surfaces prolonging and magnifying the effect, particularly in closed spaces. Greater energy transfer to the body occurs. Underground and underwater explosions propagate the shock waves further and with more force than air.

TYPES OF BLAST INJURIES

(a) *Primary Blast injuries:* These are direct result of the over-pressurisation wave's impact on the body. These injuries occur mainly to the gas-filled organs – the auditory, pulmonary and gastrointestinal systems. Extreme pressure differentials, implosion and spalling are responsible for the injury. Spalling occurs when the shock waves travel from one denser medium to rarer medium, leading to microscopic and macroscopic tears at the interface of the two mediums.

(b) *Secondary blast injuries*: These result from flying debris and bomb fragments – the fragmentation effect – leading to penetrating ballistic or blunt force injuries.

(c) *Tertiary blast injuries:* These occur as a result of individuals being thrown by the blast wind. Victims may tumble along the ground or be thrown through the air and strike other objects with resultant blunt or penetrating trauma.

(d) *Quaternary blast injuries:* These are defined as any explosion-related injury or illness not due to any of the above such burns and inhalational injuries. Temperatures from explosive gases can reach 3000°Celsius. Victims close to detonation can sustain third degree burns that can be fatal. Quaternary injuries can also include exacerbation of underlying chronic conditions[2,3].

(e) *Quinary blast injuries*: This has been proposed and is felt that absorption of toxic materials can result in hemodynamic problems[4].

Patterns of injury: Explosions in closed spaces or that result in structural collapse have higher mortality rates. Arnold et al, in an epidemiological review of terrorist bombings that produced 30 or more casualties, found that 25% victims died immediately in structural collapse, one in 12 died in confined space bombings, and one in 25 died in open air bombings[5]. A triphasic distribution was also noted in mortality: a high immediate mortality, a low emergency attendance and a late (in-hospital) mortality. The deaths were due to:

- complete dismemberment in 14%
- multiple injuries in 39%

- head and chest injuries in 21%
- head injuries in 12%
- chest injuries in 11%

Hidden injuries such as air emboli and cardiac dysrrhythmias may account for fatalities in which no other cause is found[2].

Structural collapse victims sustained more inhalational and crush injuries (secondary, quaternary injuries) and fewer primary blast injuries. Confined space bombings resulted in more primary and quaternary blast injuries, while open-air bombings led to higher rates of soft tissue injuries or more secondary blast injuries[5]. Terrorist bombings result in casualties with high injury severity scores, increased immediate mortality, more frequent need for surgical intervention – particularly orthopedic, greater requirement of intensive care, longer hospital stay, and most pertinently, a young target age group. Victims of bombings have higher hospital resource utilization than victims of other trauma.

NEUROLOGIC INJURIES

Severe head injury is a major cause of immediate and early mortality in bomb blast and missile injuries. Head injuries accounted for 29% of injuries in the Madrid bombings and 80 of the victims of the Oklahoma City bombing. Eight had severe brain injuries and 72 had mild to moderate head injuries, with 46% having concussions and 35% with closed head injuries[2,6,7].

Pathophysiology: Pathophysiology of blast injuries of the brain remains a complicated and poorly understood aspect of trauma, although some deductions arrived at from head injury management can be applied to it. There is a paucity of literature – both experimental and clinical – concerning the blast injury to the CNS. Most of the available literature describes effect of blast on CNS in laboratory animals, and it is still not clear whether the observations can be extrapolated to humans exposed to these injuries. In a study on rats subjected to non-penetrative blast, widespread response of microglial cells was seen in animals sacrificed between 1 and 14 days, and surface antigens like CR3 were upregulated. The animals sacrificed after 28 days did not show these changes[8]. There is upsurge of large number of macro (CR3) receptors, major histocompatiblity complex class I and class II antigens and monocyte/macrophage antigens.

Ultrastructural studies confirmed a wider occurrence of perivascular macrophages/microglia after the blast and the cells were laden with massive amounts of phagosomes resembling degenerating pinealocyte processes. It appears that seemingly quiescent macrophages/microglia present normally in pineal gland are activated by blast injury. The induced changes including the increase in cell numbers and endocytosis however were reversible in long-surviving animals[9]. In another study, choroids plexus infiltration with monocytes/lymphocytes together with widening of intercellular spaces between the epithelial cells, massive eruption and possible extrusion of the cytoplasm into the ventricular lumen was seen[10]. Choroid plexus is extremely sensitive to blast wave as manifested by its structural alteration and the vigorous expression of CR3 receptors and MHC antigens by the epiplexus cells. Possibly immune response might have been triggered in the CSF and ventricular system following the blast. Aminoguanidine has been shown to be neuroprotective against blast injury in rats[11].

Penetrating missile injuries: Missile wounding can be understood in terms of physical interactions between the missile and the tissues through it passes. Besides the crushing action of the missile, two types of pressure waves are generated:

1. Juxta-missile pressure: Extremely high pressures (thousands of atmospheres) are generated immediately infront of and at right angles to the moving missile. This can lead to instantaneous death, internal brain herniations.
2. Pressure waves from kinetic energy transfer: A large, subatmospheric temporary cavity is formed by the missile passing through the brain. The kinetic energy is transferred to the brain, which is propelled radially, creating a large subatmospheric cavity. When the elastic limit of the outwardly displaced tissue is reached, it falls inward whence it was displaced. This cycle may be repeated several times before deranged and lacerated tissues come to rest around the permanent cavity. This oscillatory, outward and inward rush of tissues creates a long-lasting (in milliseconds) lower amplitude (20-30 atmospheres) pressure wave, which propagates throughout the medium. These lower amplitude pressure waves cause damage to tissues at a distance from the site of actual missile injury.

Neurologic manifestations: Severe cases are "brought

in dead". Shock after blast injury is vagally mediated and can be blocked with atropine in experimental animals[12]. Blast waves can cause concussion or mild traumatic brain injury. Neurologic impairment from blast injury was initially attributed to air emboli in the cerebral circulation. However, studies in animals suggest that the overpressure wave is transferred to the central nervous system, causing diffuse axonal injury. High levels of blast overpressure can cause skull fractures or *coup-contre-coup* injuries[3,13]. Subarachnoid and subdural haemorrhages can occur. Victims may complain of headache, fatigue, poor concentration, lethargy, depression and insomnia. Other symptoms include retrograde amnesia, apathy, psychomotor agitation, indicating posttraumatic stress disorder (PTSD). In a recent study employing angiography on serving US personnel in Iraq, vasospasm has been found to be an important component of blast injury to the brain, and is associated with poor outcome[14].

In a study from the former Yugoslavia, soldiers with blast injury showed elevation in blood thrombaxane A2 (TxA2), prostacyclin (PGI2) and disulfidopeptide leukotrienes compared to non-blast injury patients. Eicosanoid changes after blast injury suggest that blast injury causes major physiologic stress[15]. A variety of effects on the CNS suggests that blast injury could be responsible for some aspects of PTSD[16]. Memory deficits may be induced by release of nitric oxide in the mesiodiencephalic reticular formation and the hippocampus in response to blast injury[17]. Management of these patients follows the established guidelines for patients with traumatic brain injury. Bell et al found good results of early, aggressive endovascular (microballoon angioplasty)[14].

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