Mild traumatic brain injury and bomb blast: stress, injury or both?

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TRAUMATIC BRAIN INJURY (TBI) caused by bomb blast has been described as the "signature wound of the war on terror".[1] The use of improved body armour, tourniquets, advances in resuscitation, trauma systems, early damage control surgery and specialised surgery have led to increased rates of survival for those injured at proximity to point sources of explosions that previously might have caused death.[2,3] These survivors frequently suffer a TBI of variable severity, ranging from mild concussion to severe "blast brain" with cerebral swelling, with or without penetrating injury and burns. This raises a number of questions, such as whether the brain is susceptible to injury from explosions in the absence of penetrating injury, what the mechanisms of such injury might be, what the current acute markers of brain injury that are adequate for assessment are, and what the longer-term outcome might be. These questions are less problematic when there has been moderate to severe injury with multiple affected organ systems.[2] However, the longer-term impact of mild TBI is not clear. Here, we focus on psychological and cognitive deficits resulting from mild TBI due to blast injury, consider the degree to which these overlap with post-traumatic stress disorder (PTSD), and discuss the treatment options in the rehabilitation phase.

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Abstract

- The mechanisms of traumatic brain injury (TBI) due to bomb blast are different and more complex than non-blast TBI and therefore civilian brain injury data may not always be relevant.
- Concussion occurring in United States soldiers deployed in Iraq is strongly associated with post-traumatic stress disorder (PTSD) and physical health problems 3–4 months after they return home. Multiple bomb blast exposures may be cumulative in their post-concussional effects.
- Compulsory focused cognitive and psychological screening of all personnel pre- and post-deployment into operational zones is an important strategy for identification of personnel at risk of long-term sequelae, including PTSD, particularly in those who have been concussed or exposed to bomb blast.
- Education and early-treatment programs are effective in reducing post-concussional syndrome and depression and in treating PTSD.
- There are likely to be increasing numbers of Australian personnel returning from the Middle East and Afghanistan who have had exposure to blast injury, and Joint Health professionals need to consider and document any TBI component when assessing other aspects of their mental status.

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Scope of the problem

There are limited published epidemiological data for the current wars in Iraq and Afghanistan. The studies that have been published have defined TBI as any disturbance of consciousness, including being dazed or seeing stars, or not remembering the injury.[4,5] Population estimates of total TBI in these and other studies are between 5% and 10% of deployed soldiers, noting that 4% of non-deployed soldiers also experience TBI during the same time period.[6] A criticism of these studies could be that patients with more significant head injury may not have been included, as they are likely to not return home with their unit and to spend longer in hospital.[4,6] The Rand Corporation study from April 2007 to January 2008 estimated that nearly 20% (320 000) of United States military service members returning from Iraq and Afghanistan reported symptoms of PTSD or major depression, yet only slightly greater than half of these sought treatment. Nineteen per cent of returning service members

reported that they experienced a possible TBI while deployed, and 7% reported both a TBI and current PTSD or major depression.[5,7]

Other studies of deployed personnel link health status,[4,6] post-concussive symptoms,[8] and neuropsychological outcome[6] to psychiatric disorders such as PTSD and major depression. At the time these surveys were completed, participants were either living at home, on duty, or both.[4,6]

Although the Rand study estimated that about 320 000 service members may have experienced a TBI during deployment, ranging from mild concussion to severe penetrating TBI, only 43% reported ever being evaluated by a physician.[7] It is clearly not possible to verify whether the blast injury exposure occurred, nor how severe it may have been, but this gives some estimate of the prevalence of exposure. The extent of impairments caused by exposure to blast injury, with or without loss of consciousness, and whether they require treatment, is largely unknown.[7]

Of 433 patients treated at the Defense and Veterans Brain Injury Center at Walter Reed Army Medical Center between January 2003 and April 2005, 68% received their injury from blast, 89% had a closed TBI, and 43% had a post-traumatic amnesia of less than 24 hours. Limb amputations had occurred in 19%. Post-concussive symptoms were almost universal.[9] A further 183 brain-injured patients were admitted to four polytrauma rehabilitation centres (PRCs) between September 2001 and January 2006, of whom about 20% had closed head injury due to blast — a slightly smaller proportion than those with closed brain injury from other causes (most likely vehicle trauma).[2] The longer-term outcome of these individuals is unknown, although the PRC experience is that better baseline functioning and a shorter time from injury to reception at the PRC predicted a better outcome.[2]

Studies of acute morbidity demonstrate that head injury is common in terrorist bombings, and common among those dying in such incidents, although these studies are largely of civilians not wearing helmets.[10,11] Fractures of the base of skull and subdural haematoma were seen in survivors of the Madrid train bombings.[12]

Mechanisms of blast brain injury

The mechanisms of blast brain injury are different and more complex than non-blast TBI and therefore civilian brain injury data may not always be relevant. Bomb blast causes injury by the effect of the blast pressure wave, penetrating trauma from fragments, the effects of collapsing structures and of being thrown by the blast wind, and injuries due to burns, asphyxia and exposure to toxins.[13-15] The blast pressure wave or primary blast injury is unique to explosions.[16]

Direct effects of the blast pressure wave

Although the intensity of the blast wave declines as a function of distance (to the third power) from its source,[11] enhanced explosive devices, with secondary ignition of disseminated explosive, spread the point source from which the explosion radiates, thereby causing more damage from the primary blast effect.[13] Possible mechanisms of brain injury from the primary blast are the primary pressure wave transiting the skull and body,[11,17,18] air embolism,[19] and acceleration–deceleration of the head.[20]

Where the pressure wave meets tissues of different density, there will be further turbulence — the greater the density differential, the greater the turbulence, creating the potential for shear injuries. The brain has areas of different density: at the white–grey matter interfaces, the blood–brain interfaces, and the ventricular system. Experimental animal research has demonstrated transcranial transmission of pressure waves,[21] resulting in skull fractures and structural and ultrastructural damage to the brain.[22,23] Transmission of an intravascular pressure wave up the neck may also contribute.[14,17] Protective devices provide limited protection.[18]

Air embolism

In the lung and gut, the air–gas interface creates the phenomenon of spalling, where air enters the pulmonary circulation by blasting through the alveoli, resulting in blast lung. The possibility of air entering the arterial circulation and causing embolism has been difficult to demonstrate in autopsy on humans, but is evident in animal studies from examination of the retina and autopsy soon after the explosion and before the emboli have a chance to dissolve.[19] Stroke, myocardial infarction, blindness and acute abdomen have been described as occurring from air embolism after blast injury.[16] Air embolism is unlikely to be a factor in mild TBI following blast exposure, unless there is severe pulmonary blast injury.

Acceleration–deceleration of the head

A solid object in the way of a rapidly moving pressure front will be accelerated. If the body is fixed, then parts able to move, such as the limbs and head, will be subject to acceleration and deceleration, particularly if the pressure wave is reflected and reverses its direction.[20] This would give rise to a similar pattern of brain injury to that which occurs in a high-speed motor vehicle accident (ie, diffuse axonal injury).

Blast injuries and human studies

Studies from World War I attempted to distinguish shell shock, thought to be related to emotional impact, from commotio cerebri, a physically based syndrome,[24] with most

cases being ascribed to emotional disorders. It was accepted at the time that there were cases of death without visible wounding that were due to brain injury from a nearby explosion. Subarachnoid haemorrhage, subdural haematoma, cerebral venous congestion, leptomeningeal haemorrhage and hyperaemia of the brain and meninges are constant features of humans dying from blast injury.[11,25-27] Cerebral vasospasm following subarachnoid haemorrhage due to blast injury has been an under-recognised problem.[28]

At the milder end of the spectrum, it is instructive to review the recent literature on concussion in athletes, as this is a similar population to soldiers. There is increasing evidence of metabolic brain vulnerability following concussion, the time course of which may not correspond to the clinical recovery.[29] Recovery in athletes is significantly slower after a second concussion,[30] and sports-related concussions have adverse cumulative effects on intracortical inhibitory systems and visuomotor coordination.[31] It is unknown to what extent this corresponds to concussion due to blast injury.

Emotional disorders

The epidemiological data emerging from Iraq and Afghanistan link the occurrence of mild TBI with depression and/or PTSD,[4,6,8] although the definitions of mild TBI are more liberal than the agreed World Health Organization definition[32] and the reported numbers of individuals with TBI are small. More importantly, health and functional status were linked to depression and anxiety ratings, rather than the occurrence of TBI,[4] and these studies, by their sampling design, excluded the more severely injured soldiers.

Hoge et al surveyed 2525 US Army infantry soldiers 3 to 4 months after their return from a 1-year deployment to Iraq — 124 (4.9%) reported injuries with loss of consciousness, 260 (10.3%) reported injuries with altered mental status, and 435 (17.2%) reported other injuries during deployment.[4] Of those reporting loss of consciousness, 43.9% met criteria for PTSD, compared with 27.3% of those reporting altered mental status, 16.2% with other injuries and 9.1% with no injury.[4] Soldiers with mild TBI, especially if there was a loss of consciousness, were significantly more likely to report poor general health, missed work days, medical visits, and a high number of somatic and post-concussive symptoms than were soldiers with other injuries. However, after adjustment for PTSD and depression, mild TBI was no longer significantly associated with these physical health outcomes, except for headache.[4]

The authors concluded that mild TBI (ie, concussion) occurring in soldiers deployed in Iraq was strongly associated with PTSD and physical health problems 3 to 4 months after they returned home, and that PTSD and depression were important mediators of the relationship between mild TBI and physical health problems.[4] TBI and PTSD are invisible to the eye of other service personnel, family members and society in many cases, especially if one is not familiar with the signs to look for.

The link between mood disorders, PTSD and cognitive dysfunction is strong, with studies demonstrating hippocampal atrophy[33,34] and neuropsychological impairment.[35] There are abnormal patterns of neural activation in PTSD. Functional neuroimaging shows a reversal of the normal inhibition of the amygdala by the medial prefrontal cortex in human subjects in both basal and threat conditions.[36] There is also some evidence that some antidepressant medications exert their effects by promoting neural repair, probably by promotion of brain-derived neurotrophic factor synthesis.[33] In other forms of mild TBI, mood disorders predict poor long-term outcome, as do expectations of poor outcome and attribution of disability to permanent brain injury.[37] Jones et al argue that it is unwise to label TBI as a unique signature injury and that disorders that cross the divide between physical and psychological require a nuanced view of their interpretation and treatment.[24] The lessons of shell shock in World War I and World War II are of relevance today.[24]

Long-term effects of mild traumatic brain injury following bomb blast

Published studies that cite long-term neurological sequelae do not describe the initial severity of the brain injury.[38,39] Studies that suggest emotional causes have used very liberal definitions of mild TBI,[4] excluded individuals with lesions visible on computed tomography (CT) scanning,[40] or studied a population that continued in active duty.[4,6]

The link between mild TBI and long-term dysfunction remains speculative. The medial prefrontal cortex and amygdala are basal structures involved in emotional processing of traumatic events and regulation of fear and anxiety, and these structures may be vulnerable in blast injury. Injury to these structures may alter the brain's regulation of stress and anxiety and contribute to post-concussional syndrome (PCS) and possibly PTSD.[36,41,42] In other forms of TBI, those areas affected by acceleration–deceleration and contrecoup injuries (basal frontal lobes and temporal poles) tend to be those involved in the regulation of emotions, judgement and memory, and may mimic psychological illness or complicate the course of such illness.[42]

Mild TBI in US soldiers deployed to Iraq is strongly associated with PTSD and physical health problems,[4] and multiple blast exposures may be cumulative in their post-concussional effects. As PTSD and depression are important mediators of the relationship between mild TBI and physical health problems,[4] health problems following mild TBI may be due primarily to PCS and depression. PCS includes problems with headaches, memory, concentration, balance, sensitivity to light and sound, tinnitus, and irritability; these symptoms are also seen in PTSD. It is difficult to differentiate the symptoms of severe stress from those of mild TBI and PCS.

Depression further complicates the clinical picture, with symptoms of anergy, irritability and low motivation, which resemble the pattern seen in frontal lobe injury. Having a record of post-traumatic amnesia is important evidence that can identify a definite TBI.

Outcome studies of TBI are largely from civilian data of sports and motor vehicle accidents, with acceleration–deceleration as the usual injury mechanism.[37] In a meta-analytical study, mild TBI (defined as a Glasgow Coma Scale [GCS] score of 13–15, post-traumatic amnesia of less than 24 hours, and loss of consciousness of less than 30 minutes) showed complete recovery after 1 year;[43] however, post-concussional symptoms may persist. Mild TBI is commonly complicated by mood and anxiety disorders, with premorbid factors contributing significantly to the outcome.[44] PCS, with irritability, dizziness, memory and concentration problems and headaches, overlaps in symptomatology with mood and anxiety disorders,[37,44] and some have suggested it is not specific to TBI.[37,40]

There have been few long-term studies of the effects of blast-related TBI, other than assessing the prevalence of PTSD. Cernak et al reported on 665 casualties admitted to the Belgrade military hospital with blast injury and external wounds confined to the lower limbs.[38] At 1 year, 30% showed electroencephalographic (EEG) abnormalities; the level of occupational and social function was not described.[38] During the wars in Lebanon, a third of head-injured patients had closed injuries, with a third of these due to blast exposure, often from mines. They were described as making steady progress over the subsequent years, but a proportion had ongoing cognitive problems.[45] The significance of the prolonged EEG abnormalities reported by Cernak et al[38] and mentioned by Trudeau et al[39] is unknown. Most casualties had comorbid PTSD, which might account for the attentional problems described, although the EEG changes reported are unexpected.

Evaluation of patients with mild traumatic brain injury due to bomb blast

Clinical markers of severity at the time of injury are the GCS score, duration of coma and amnesia, focal neurological deficit, and damage to the tympanic membrane and vestibular apparatus. The circumstances of the explosion are relevant; in particular, the type of explosive (where known), the presence or absence of armour and head protection, and whether the patient was in a confined space should all be documented.

In the acute recovery phase, serial measures of brain function such as the mini-mental state examination or the abbreviated Westmead scale are very useful measures of post-traumatic amnesia and can be administered hourly by nurses.[46] It is preferable to use measures of post-traumatic amnesia to validate whether mild TBI has occurred.[41] Comparisons of pre- and post-deployment cognitive screens would be helpful in the investigation of cognitive deficits following blast injury. Assessment of balance and coordination are also useful. It is very difficult to reliably differentiate between symptoms caused by severe stress and mild TBI, which is made more difficult by retrospective accounts of injury.[41]

Plain skull x-ray may show skull fractures, especially following head impact. The availability of CT scans in field hospitals is useful to document abnormalities and plan management. Although CT scan findings are usually normal in mild concussion, there is a greater likelihood of skull fractures, cerebral contusions or small haemorrhages following blast exposure, particularly if the head has been impacted. Of patients with apparently mild TBI, 7%–20% of those scanned have CT or magnetic resonance imaging abnormalities, and the patients have morbidity similar to those with moderate TBI.[37] This correlation requires further research. Biochemical markers may also provide objective evidence of concussion and this can be assessed using ^{1}H magnetic resonance spectroscopy.[29] The serum level of S100B protein is a promising marker for evaluation of TBI in cases of mild concussion, but its specificity has been questioned and its serum levels increase following exercise.[47]

Treatment of mild traumatic brain injury including emotional disorders following bomb blast

Many service members are reluctant to report symptoms or admit to mental problems because of fear that doing so may affect their careers, fear that their peers would lose confidence in them, fear that medication may cause side effects, or a belief that their family or friends can provide more help than a mental health professional.[7] It is important that affected members feel that seeking treatment is completely confidential and is a sign of strength and interest in getting better, not a weakness. It is also important that soldiers returning from the Middle East are given a realistic picture of resolution of post-concussive symptoms and that treatment will minimise stress-related conditions.[41]

The Australian Defence Force has instigated compulsory return to Australia (RTAPS) and post-operational psychology screens (POPS) that will help detect emotional problems as a result of deployment. Particular attention should be directed to those personnel exposed to bomb blast.

An expectant approach to mild TBI, with education about likely resolution of emotional and cognitive dysfunction over the first year and assessment and treatment by a multidisciplinary rehabilitation team with active case management, is likely to have the greatest impact on functional recovery.[37] In more severe blast injuries, disfigurement, chronic pain, limb damage or loss, damage to the ear (particularly the vestibular apparatus) and psychiatric illness will likely complicate recovery.

Education programs with strategies aimed at reducing PCS and depression could relieve many post-concussive symptoms. Physical and mental rest is also an important treatment strategy for PCS. The type, duration and availability of suitable duties during rehabilitation need review with the affected member's unit, as well as education directed at relieving stigma.[48] Mild TBI may also impair cognitive resources and thus reduce the capacity to engage in cognitive strategies to manage the aftermath of psychological trauma. Poorly adapted cognitive strategies after trauma, including concussion, are a major predictor of PTSD.[41] The diagnosis and early management of PTSD have been extensively covered in the literature[49] and are beyond the scope of this article.

Cost of traumatic brain injury and post-traumatic stress disorder in the United States

The 1-year estimate of societal cost associated with treated cases of mild TBI is up to US$32 000 per case, while estimates for moderate to severe TBI range from US$268 000 to more than US$408 000 per case. Estimates of the total 1-year societal cost of 2700 cases of TBI identified in the Rand study to date range from US$591 million to US$910 million.[5,7]

The treatment of US personnel with PTSD and depression is estimated by the Rand Corporation to cost as much as US$6.2 billion in the 2 years following deployment, which includes both direct medical care and costs for lost productivity and suicide.[5,7] A recommendation of this extensive non-governmental study was that a major national effort was needed to expand and improve the capacity of the mental health system to provide effective care to service members and veterans. More mental health care providers will need to be trained to use high-quality, evidence-based treatment methods and to encourage affected service members to seek help.[5,7]

Conclusions

Health problems following mild TBI in returned service personnel may primarily be due to PCS and depression. There are likely to be increasing numbers of Australian personnel returning from the Middle East and Afghanistan who have had exposure to blast injury, and Joint Health professionals should consider and document any TBI component when assessing other aspects of their mental status. Compulsory focused cognitive and psychological screening of all personnel pre- and post-deployment into operational zones is an important strategy for identification of personnel at risk of long-term sequelae, including PTSD. More detailed assessment should be undertaken for those exposed to bomb blast, especially if there is any evidence they have suffered a mild TBI. There is still reluctance by military personnel to self-report mental health problems — mental health professionals must continue to address this problem and reduce the stigma attached to mental health problems. Education and early-treatment programs are effective in reducing PCS and depression and in treating PTSD. Strategies to mitigate the immediate blast effects to the brain are currently under investigation.

Competing interests

None identified.

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