Review

Bomb blast, mild traumatic brain injury and psychiatric morbidity: A review

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ABSTRACT

Traumatic brain injury (TBI) arising from blast exposure during war is common, and frequently complicated by psychiatric morbidity. There is controversy as to whether mild TBI from blast is different from other causes of mild TBI. Anxiety and affective disorders such as Post-traumatic Stress Disorder (PTSD) and depression are common accompaniments of blast injury with a significant overlap in the diagnostic features of PTSD with post-concussive syndrome (PCS). This review focuses on this overlap and the effects of mild TBI due to bomb blast. Mild TBI may have been over diagnosed by late retrospective review of returned servicemen and women using imprecise criteria. There is therefore a requirement for clear and careful documentation by health professionals of a TBI due to bomb blast shortly after the event so that the diagnosis of TBI can be made with confidence. There is a need for the early recognition of symptoms of PCS, PTSD and depression and early multi-disciplinary interventions focussed on expected return to duties. There also needs to be a continued emphasis on the de-stigmatisation of psychological conditions in military personnel returning from deployment.

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Introduction

The rate of survival of soldiers in war has increased from 2:1, injured to dead in WWII, to 7.4:1 in Operation Iraqi Freedom (OIF) and with it more complex rehabilitation needs to improve the level of function of those who survive their wounds. The use of improved body armour, tourniquets, advances in resuscitation and trauma systems, early damage control surgery, and critical care air transport teams account for the increased rates of survival for those injured at proximity to point sources of explosions that previously might have caused death.

Traumatic brain injury (TBI) caused by bomb blast has been described as the 'signature wound of the war on terror' and its...
Scope of the problem

An extensive non-governmental study by the Rand Corporation noted that 2726 cases of TBI were diagnosed in returned personnel between 2001 and 2007, with an estimated societal cost of $590–910 million dollars.79 The 1-year estimate of societal cost associated with treated cases of mild TBI is up to USD $32,000 per case, while estimates for moderate to severe TBI range from USD $268,000 to more than USD $408,000.70 The authors estimate that about 320,000 service members may have experienced a TBI, ranging from mild concussion to severe penetrating TBI, during deployment up to October 2007, with only 43% reporting being evaluated by a physician.70

The treatment of military personnel from the USA with PTSD and depression post-combat exposure estimated by the Rand Corporation to cost as much as USD $6.2 billion in the 2 years following deployment which includes both direct medical care and costs for lost productivity and suicide.70

A number of epidemiological studies of personnel returned home from the current conflicts have linked impairment post-deployment, with or without blast exposure, primarily to depression or anxiety related disorders such as PTSD.27,35,61,74 The experience of field hospitals is that blast-related brain injury occurs, often in conjunction with other physical injuries and that wounds to the face, head and neck are common.30,32,53,80

Studies of personnel who have returned home have defined traumatic brain injury (TBI) as any disturbance of consciousness; including being dazed or seeing stars, or not remembering the concussion itself.16,43,63,77 Population estimates of total TBI in these studies are between 10 and 15% of deployed samples, noting that 4% of non-deployed soldiers also experience a TBI during the same time period.74 Xydakis et al.79 have suggested that delay, in surveying soldiers may have lead to under-reporting of the extent or occurrence of head injury. In the heat of battle lack of documentation of a head injury or exposure to bomb blast will exacerbate this problem. In practice the US Department of Veterans Affairs uses historical data as a screen for blast exposure and then proceeds with physical, radiological and neuropsychological screening.56

Over half the veterans reported by Schneiderman et al.61 had more than one mechanism of injury, suggesting multiple concussions. Schneiderman et al.61 stratified the severity of mild TBI in their sample of veterans from Iraq and Afghanistan and found an association of persistent symptoms with more severe TBI, multiple injury mechanisms and the presence of PTSD, and noted that the findings of association between TBI and PTSD could relate to the stress of wounding and deployment or brain injury manifesting as PTSD.61

In support of this observation Chen et al.18 showed that there is neuroimaging evidence of neural dysfunction, in concussed depressed compared to non-depressed athletes with and without concussion. Concussed athletes as a group showed reduced insula gray matter, and those athletes who were also depressed showed reduced gray matter in the medial frontal and temporal areas. Chen et al.18 suggest that the changes causing depression were related to the concussion itself.

Mechanisms of injury

The types of injury arising from blast are described as primary through to quaternary, being the effect of a blast pressure wave, penetrating trauma from fragments, the direct concussive and contre-coup effects of collapsing structures and of being thrown by the blast wind and striking the head against fixed structures, and injuries due to burns, asphyxia and exposure to toxins respective-ly.25,43,48,55 The blast pressure wave or primary blast injury is unique to explosions.5,48,55 Individuals are likely to be affected by some or all of these mechanisms sustaining multiple injuries.30,32,43,63,77

Direct effects of the pressure wave

An explosion causes a high pressure wave to sweep across the immediate surrounds at about the speed of sound.25,26 The intensity of the blast wave declining as a function of distance to the third power from its source.77 Enhanced explosive devices, with secondary ignition of disseminated explosive, spread the point source from which the explosion radiates causing more damage from the primary blast effect.5,25 Possible mechanisms of brain injury from the primary blast are the primary pressure wave transiting the skull and body25,43,45, air embolism14,19 and acceleration/deceleration of the head.43 Where the wave meets tissues of different density there will be further turbulence.

Animal research has demonstrated transcranial transmission of pressure waves17 resulting in skull fractures and structural and ultra-structural damage to the brain.6,35–41 Transmission of an intravascular pressure wave up the neck may also contrib-ute.6,40,43 Protective devices provide limited protection.16,39

Air embolism

In the lung, the air gas interface creates the phenomenon of spalling where air enters the pulmonary circulation by blasting though the alveoli creating the phenomenon of blast lung.77 Air embolism is unlikely to be a factor in mild TBI following blast
exposure because the pulmonary blast injury is also likely to be mild.

**Acceleration/deceleration**

A solid object in the way of a rapidly moving pressure front will be accelerated. Body parts able to move such as the limbs and head will be subject to acceleration and deceleration, particularly if the pressure wave reverses its direction due to reflection which occurs in blast waves in close environments such as vehicles and buildings.2426 This would give rise to a similar pattern of brain injury which occurs in a high speed motor vehicle accident, i.e. diffuse axonal injury (DAI).

**Animal studies**

Microscopic damage is seen in the brain of blast exposed animals.163941 There appears a generalised inflammatory response with microglia and astrocyte proliferation and cytoskeletal disruption, primarily affecting not only the cortex but also subcortical structures, including the hippocampus.163941 There is some suggestion that repeated blasts may lead to a decreased threshold for damage, although the duration of the window of increased vulnerability is not clear.41 In cell cultures exposed to blast there is an increase in membrane permeability, persisting for some hours after blast exposure.41 Cernak et al.16 demonstrated cognitive and histological changes in rats exposed to blast with and without head protection, finding the latter more severely affected, although with a trend to improvement over the 5-day duration of the study. Similarly, Kaur et al.39 showed histological changes normalising 28 days after exposure to blast injury in rats.

**Blast injuries and human studies**

Studies from WWI attempted to distinguish ‘shell shock’ related to emotional impact, from concussion, related to nearby blast exposure, a physically based syndrome.37 That there were fatalities from a nearby explosion, without visible wounding and due to brain injury was accepted at the time. 

Bomb blast results in a range of TBI severity from mild TBI with subtle disabilities which may severely limit performance59 to severe brain swelling, intracranial haemorrhage and penetrating injury with metal fragments and other foreign bodies which are often heavily contaminated.54 Post-traumatic vasospasm, particularly where there has been subarachnoid haemorrhage, and later pseudo-aneurysm formation is likely in those who have experienced intracranial bleeding with penetrating injury.343536

In blast affected humans subarachnoid haemorrhage, subdural hematoma, cerebral venous congestion, leptomeningeal haemorrhage and hyperaemia of the brain and meninges are seen in individuals dying from blast injury.142043

In studies of military personnel, the mechanism of brain injury of blast compared to other causes such as motor vehicle accidents, does not appear predictive of outcome.56 However there is wide variability in the outcomes of personnel injured by blast which may reflect the multiple mechanisms involved.5538

At the milder end of the spectrum of TBI, the recent literature on concussion in athletes suggests there is metabolic brain vulnerability following concussion, the time course of which may not correspond to the clinical recovery.73 Recovery in athletes is significantly slower after a second concussion65 and sports related concussions have adverse cumulative effects on intracortical inhibitory systems and visuo-motor coordination.23 A similar lowering of threshold to injury may occur in repeated blast injury in animals.41 It is unknown how much this corresponds to concussion due to human blast injury, although Schneiderman et al.51 reported multiple mechanisms of injury and concussions in their group of deployed personnel.

**Emotional disorders**

The epidemiological data emerging from Iraq and Afghanistan link the occurrence of blast exposure, mild TBI and affective disturbance such as depressive or PTSD symptomatology.28366175 This data should be viewed with caution because the definitions of mild TBI are more liberal than the agreed WHO definitions and the samples although large were not complete. The occurrence of PTSD in a head-injured population has been considered problematic due to amnesia for the event and an inability to encode traumatic memories.39 However PTSD has been demonstrated in civilians following TBI and it has been suggested that encoding may occur at an implicit level, amnesic survivors of accidents may show physiological reactivity in response to reminders of their accident.13 Physiological derangement due to the impact itself has also been suggested as a contributor to depression in brain injured athletes18 and it may be that causality between psychiatric disorder and brain injury is due to both emotional and physical factors.67

Hoge et al.34 surveyed 2525 US Army infantry soldiers 3–4 months after their return from a 1-year deployment to Iraq. Psychiatric diagnoses of major depression and PTSD were made according to DSM IV criteria.12 124 (4.9%) reported head injuries with loss of consciousness (LOC), 260 (10.3%) reported injuries with altered mental status, and 435 (17.2%) reported other injuries during deployment. Of those reporting LOC, 43.9% met criteria for PTSD, compared to 27.3% of those reporting altered mental status, 16.2% with other injuries and 9.1% with no injury.35 Those soldiers with mild TBI, especially if there was a LOC, were significantly more likely to report poor general health than were soldiers with other injuries.35 After adjustment for PTSD and depression, mild TBI was no longer significantly associated with these physical health outcomes except for headache.35 Hoge et al.34 have more recently argued against the non-discriminate use of diagnoses such as TBI/concussion and ‘signature injury’, and that this has resulted in excessive diagnoses of ‘mild TBI’ following bomb blast exposure in US soldiers returning from the Middle East. They argue that this has in turn resulted in an increase in persistent PCS and compensation claims.34

The linkage between mood disorders, PTSD and cognitive dysfunction is strong with studies demonstrating hippocampal atrophy9,47 and neuropsychological impairment44 in the absence of TBI and substance misuse. 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.64 In traumatic brain injury due to acceleration/deceleration and contre-coup injuries the areas affected (basal frontal lobes and temporal poles) tend to be those involved in the regulation of emotions, judgment and memory and may mimic psychological illness or complicate the course of such illness.1822

Jones et al.37 argue that it is unwise to label TBI as a unique signature injury and that symptoms that cross the divide between physical and psychological require a nuanced view of their interpretation and treatment. 

**Evaluation of patients with blast-related TBI**

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.1278 As much information should be recorded at an early stage after blast exposure as possible so that the TBI component can be verified at subsequent assessments. This will assist with therapy but also the evaluation of compensation claims.
Clinical markers at the time of injury for TBI are the history or observation of head impact and signs of cranial trauma, GCS, duration of coma and post-traumatic amnesia, ongoing headache and focal neurological deficit (which is uncommon). The symptoms of being dazed, seeing stars, being confused and disoriented and brief amnesia for the event are not necessarily due to blast trauma and may be due to the emotional shock of the event. Skull fracture, including of the base of the skull (28) may be present if the head has been impacted. In deployed situations computed tomography (CT) or magnetic resonance (MR) scanning may be unavailable and clinical markers of skull fracture should be specifically examined. Rupture of the tympanic membrane should arouse suspicion of brain injury and damage to the auditory and vestibular apparatus may occur and cause persisting disability. Damage to the eyes may occur, although the likelihood is diminished where eye protection is worn.

The circumstances of the explosion are relevant; in particular the type of explosive, where known, the distance from the centre of the explosion, the presence or absence of armour and head protection and whether the patient was in a confined space.

In the acute recovery phase serial measures of brain function such as the abbreviated Westmead scale are very useful measure of post-traumatic amnesia and can be administered by nurses. It is preferable to use measures of post-traumatic amnesia (PTA) to validate whether mild TBI has occurred. 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 is also useful.

Plain skull X-ray may show skull fractures especially following head impact. The availability of CT scans in field hospitals is useful to document pathology and plan management. Although CT is 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. In civilians, 7–20% of apparent mild TBI patients have CT or MR abnormalities. In a civilian setting 36% of patients with blast head injury and a GCS of 15 were subsequently shown to have brain injury either by CT scan on admission or by CT scan later in the course of hospitalisation, ordered due to a deteriorating mental state. In some cases the intake CT was normal with deterioration over the next 48 h. These findings suggest a need for a high index of suspicion for TBI in blast-injured patients. MR is more sensitive than CT for detecting lesions in the brain following traumatic brain injury but is not available in remote military hospitals. Hence there are no reported MR studies in the early phase following blast injury. MR T2 Gradient Echo (T2GE) is sensitive for detecting the lesions of diffuse axonal injury. Brenner et al. studied veterans with traumatic brain injury and/or PTSD and found that those with moderate or severe TBI were more likely to have traumatic brain lesions than mild TBI patients and those with PTSD had negative neuroimaging. MR diffusion tractography imaging (DTI) may demonstrate subtle structural brain abnormalities after primary blast injury which are not seen on routine MR. An instructive case was reported recently and indicates that DTI should be used more often in patients with a mild TBI following bomb blast.

Biochemical markers may also provide objective evidence of concussion and this can be done using 1H-Magnetic Resonance Spectroscopy. The serum level of S100B protein has been evaluated in mild TBI but its specificity has been questioned and the levels increase following exercise.

Treatment of mild TBI including emotional disorders following bomb blast

Mild TBI has not been reported as a cause of psychiatric evacuation during Operation Enduring Freedom (OEF) and OIF, although emotional disorders, in particular anxiety disorders have been. However 35% of physically injured evacuees required neurosurgical consultation and management. The subtle deficits in performance that occur from blast-related concussion may impair performance in a highly demanding occupation and put individuals, and others, at risk unless adequately assessed and managed in the acute and rehabilitation phases. There is a likelihood that affective symptoms will synergise with the concussion and physical symptomatology to potentially worsen outcome. Civilian patients with TBI are at high risk of developing acute stress disorders, and the co-morbid presence of PTSD significantly worsens cognitive performance in military patients with 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. This might occur by increased efforts to liaise with commanders who should be well briefed and supportive of mental health strategies. It is important that personnel understand that seeking treatment is confidential and that seeking help is a sign of interest in getting better, not a weakness and that treatment will minimise stress related conditions.

An expectant approach to mild TBI is recommended with education about likely resolution of emotional and cognitive dysfunction over the first year. Attribution of injury source may affect outcome and the term “concussion” may be more familiar and less frightening to patients than the label of TBI. Coordination of rehabilitation efforts, with the setting of benchmarks, duties, review dates, and liaison with parent units is likely to have the most impact on functional recovery. Comprehensive rehabilitation, social support and a younger age of victims is associated with a better outcome for survivors in survivors of terrorist incidents in Israel and has been recommended in the RAND review. In more severe blast injuries disfigurement, chronic pain, limb damage or loss, damage to the ear and vestibular apparatus, and psychiatric illness will complicate recovery.

Physical and mental rest is also an important treatment strategy for PCS. Poorly adapted cognitive strategies after trauma, including concussion and are a predictor of PTSD and may hinder the outcome. The PCS is associated with PTSD similarly PTSD and depression worsen disability after physical injury. The diagnosis and early management of psychological disorders arising from trauma exposure have been extensively covered in the literature.

Long-term effects of mild TBI following bomb blast

Outcome studies of TBI are largely from civilian data, sports and motor vehicle accidents with acceleration/deceleration as the usual injury mechanism. Mild TBI (defined as a GCS of 13–15, PTA of less than 24 h and loss of consciousness of less than 30 min) in a meta-analysis showed complete recovery after 1 year, however post-concussional symptoms may persist. Mild TBI is commonly complicated by mood and anxiety disorders with premorbid factors contributing significantly to the outcome.

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 a military hospital with blast injury and external wounds confined to the lower limbs. At 1 year 30% showed electroencephalographic (EEG) abnormalities. During the wars in Lebanon one-third of head-injured patients had closed injuries, with one-third of these due to blast exposure. They were described as making progress over the subsequent years but a proportion had ongoing cognitive problems, the extent of which is unknown. The significance of the prolonged EEG abnormalities reported by Cernak et al. and...
mentioned by Trudeau et al. is unknown. Most casualties had co-
morbid PTSD which might account for the attentional
problems described, although the EEG changes reported are
unexpected.

Belanger et al. reported on a sample of blast-injured soldiers,
and compared them to TBI cases with other mechanical forms of
injury. There was no difference in neuropsychological performance
at 2 years between the two groups. At admission to a PRC there is a
trend for blast-injured patients to be more likely to have cognitive
impairment than those from other mechanisms of injury. Blast-
injured patients had a greater likelihood of psychological
symptomatology, which was attributed to the likely circumstances
of the original injury, terrifying at the time, but preceded and
followed by extreme stressors in a hostile environment Belanger et al. concluded that blast injury although more likely to
be severe showed similar behavioural manifestations to other
types of injury.

Schwartz et al. reviewed survivors of terrorist bombings with
TBI in Israel finding that despite severe injuries at intake, in
particular intracerebral lesions, and high rates of PTSD, the longer
term outcome was similar to a non-blast TBI group, with the
exception of a higher rate of post-traumatic epilepsy in the blast
TBI group.

Conclusions

Blast injury to the human brain may cause the whole spectrum of
TBI from mild TBI to severe brain injury with brain swelling,
intracranial haemorrhage and penetrating injury. The circum-
stances of the blast exposure and premorbid personality will
determine the mix of psychological and physical injury. The
pathological mechanisms of blast injury to the brain are well
understood from animal and human research. Mild TBI (concus-
sion) in soldiers deployed to Iraq and Afghanistan is strongly
associated with affective symptoms such as depression and PTSD,
and physical health problems.

It cannot be concluded that bomb blast causes PTSD and
depression by psychogenic means alone. The emotional shock of
the blast and the circumstances surrounding it may be enough in
some cases to explain the PTSD and depression. However, there is
emerging evidence that parts of the brain injured in blast TBI are
concerned with regulation of emotions and judgement and this
organic component of brain injury may contribute to the onset of
PTSD and depression.

In assessing the contribution of TBI to PTSD, affective disorders
and cognitive impairment following blast exposure, contempor-
aneous history, examination and imaging data (where available) is
essential. Duration of coma, persistent headache and post-
traumatic amnesia are particular features of TBI concussion which
should be documented. It is difficult to differentiate the symptoms
of severe stress from those of mild TBI and PCS as there is a great
overlap. This is also made more challenging by retrospective
accounts of injury. Having a record of the circumstances of the
blast, and the immediate clinical and performance picture after
blast is important in identifying a TBI. Persistent symptoms are
more likely in the patients diagnosed with mild TBI following blast
injury compared with the civilian population of mild TBI and
compensation claims and litigation are also higher in patients with
persistent concussional symptoms.

The relationship between mild TBI and blast injury awaits
further elucidation, but there is evidence that a concussion has
both physical and psychological components, the former possibly
diminishing in contribution to the overall pattern of disability as
time passes. There is likely a synergistic interaction between
the physical and psychological components of blast injury in promot-
ing disability.

Disclosure statement

The views expressed in this article are solely those of the
authors and do not represent the policy or views of the
Commonwealth of Australia Department of Defence or the
Australian Defence Force.

Conflict of interest statement

There is no conflict of interest by either author.

Appendix A. Diagnostic criteria for Post-traumatic Stress
Disorder and major depression [DSMIV]

A.1. Criteria for major depressive episode

A. Five (or more) of the following symptoms have been present
during the same 2-week period and represent a change from
previous functioning; at least one of the symptoms is either (1)
depressed mood or (2) loss of interest or pleasure.

Note: Do not include symptoms that are clearly due to a
general medical condition, or mood-incongruent delusions or
hallucinations.

(1) Depressed mood most of the day, nearly every day, as
indicated by either subjective report (e.g., feels sad or
empty) or observation made by others (e.g., appears tearful).

(2) Markedly diminished interest or pleasure in all, or almost
all, activities most of the day, nearly every day (as indicated
by either subjective account or observation made by others).

(3) Significant weight loss when not dieting or weight gain (e.g.,
a change of more than 5% of body weight in a month), or
decrease or increase in appetite nearly every day.

(4) Insomnia or hypersomnia nearly every day.

(5) Psychomotor agitation or retardation nearly every day
(observable by others, not merely subjective feelings of
restlessness or being slowed down).

(6) Fatigue or loss of energy nearly every day.

(7) Feelings of worthlessness or excessive or inappropriate guilt
(which may be delusional) nearly every day (not merely
self-reproach or guilt about being sick).

(8) Diminished ability to think or concentrate, or indecisiveness,
nearly every day (either by subjective account or as
observed by others).

(9) Recurrent thoughts of death (not just fear of dying),
recurrent suicidal ideation without a specific plan, or a
suicide attempt or a specific plan for committing suicide.

B. The symptoms do not meet criteria for a mixed episode.

C. The symptoms cause clinically significant distress or impairment
in social, occupational, or other important areas of functioning.

D. The symptoms are not due to the direct physiological effects of a
substance (e.g., a drug of abuse, a medication) or a general
medical condition (e.g., hypothyroidism).

E. The symptoms are not better accounted for by Bereavement, i.e.,
after the loss of a loved one, the symptoms persist for longer
than 2 months or are characterised by marked functional
impairment, morbid preoccupation with worthlessness, suicidal
ideation, psychotic symptoms, or psychomotor retardation.

A.2. Diagnostic criteria for 309.81 Post-traumatic Stress Disorder

A. The person has been exposed to a traumatic event in which both
of the following were present:

(1) The person experienced, witnessed, or was confronted with
an event or events that involved actual or threatened death
or serious injury, or a threat to the physical integrity of self
or others.
B. The traumatic event is persistently re-experienced in one (or more) of the following ways:
(1) recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions.
(2) recurrent distressing dreams of the event.
(3) acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated).
(4) intense psychological distress at exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event.
(5) physiological reactivity on exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event.
C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:
(1) efforts to avoid thoughts, feelings, or conversations associated with the trauma.
(2) efforts to avoid activities, places, or people that arouse recollection of the trauma.
(3) inability to recall an important aspect of the trauma.
(4) markedly diminished interest or participation in significant activities.
(5) feeling of detachment or estrangement from others.
(6) restricted range of affect (e.g., unable to have loving feelings).
(7) sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span).
D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:
(1) difficulty falling or staying asleep.
(2) irritability or outbursts of anger.
(3) difficulty concentrating.
(4) hypervigilance.
(5) exaggerated startle response.
E. Duration of the disturbance (symptoms in Criteria B, C, and D) is more than 1 month.
F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify if:
Acute: if duration of symptoms is less than 3 months.
Chronic: if duration of symptoms is 3 months or more.
Specify if: With delayed onset: if onset of symptoms is at least 6 months after the stressor.

Note: Paediatric aspects have been removed from these criteria.

References