

Management of trauma and PTSD

Up to 50% of the population will experience a significant traumatic event in their lifetime.

J J Benson-Martin, MB ChB, Dr Med (Zurich), CML (Unisa), FCPsych (SA)

Lecturer, Department of Psychiatry and Mental Health, University of Cape Town and Consultant Psychiatrist, Valkenberg Hospital, Cape Town

Janine Benson-Martin did both her undergraduate and postgraduate training at the University of Cape Town. Her interests are women's mental health and trauma, as well as electroconvulsive therapy.

Correspondence to: J Benson-Martin (j.benson-martin@uct.ac.za)

Whereas the majority of trauma survivors recover without clinical sequelae, 10 - 20% of these individuals develop the syndrome of post-traumatic stress disorder (PTSD).^[1] In the USA, the National Comorbidity Survey estimated that between 5% and 9% of the general population suffer from PTSD.^[2,3] In the primary healthcare setting the prevalence of PTSD is estimated to be two to three times higher.^[4] The South African Stress and Health Study (SASH) – the first large population-based study of mental disorders in South Africa – found anxiety disorders, as a class, to be the most prevalent 12-month and lifetime disorder, while the 12-month prevalence of PTSD was 2.3%.^[5]

Acute stress disorder (ASD) and PTSD are manifestations of mental illness in relation to exposure to a severe traumatic event.

Anxiety and trauma

Normal anxiety is universally experienced in response to a threat. It is associated with a range of cognitions and physiological responses from apprehension, intense feelings of dread, as well as hyperactive autonomic phenomena (e.g. abdominal discomfort, restlessness, perspiration, palpitations). It is a normal reaction to an abnormal or unpredictable event.

Trauma is described in the *Diagnostic and Statistical Manual IV – Text Revised* (DSM-IV-TR) as the experience or the witnessing of 'an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others'. It evokes feelings of intense fear,

horror and helplessness. The individual perceives the event as a potential threat to their or others' well-being or life and experiences clinically significant distress or impairment in functioning as a result of this distress.^[6]

Diagnosis

Mental pathology as a result of a traumatic event was first entered in the *Diagnostic and Statistical Manual III* (DSM-III), but was formerly known in soldiers as 'war neurosis'. The nosology has been refined over time, and is in the process of further change in the proposed DSM-V. Acute stress disorder (ASD) and PTSD are manifestations of mental illness in relation to exposure to a severe traumatic event.

The expected response to a severe trauma is one of shock, fear, horror and helplessness.

With time this response should disappear and the vast majority of individuals experience spontaneous recovery from these feelings. The problem arises when this spontaneous recovery fails to occur. PTSD and related pathology could therefore be conceptualised as a disorder where there is failure to recover.^[7]

- ASD is limited to the first 4 weeks after the traumatic event, with the disturbance lasting at least 2 days.^[6] It is characterised by the presence of four sets of symptoms: dissociative features (numbing, derealisation, depersonalisation); persistent re-experiencing of the event; marked avoidance of stimuli associated with the trauma; and symptoms of anxiety and hyperarousal.
- Acute PTSD is diagnosed after 4 weeks with the presence of the triad of re-experiencing, avoidance and signs and

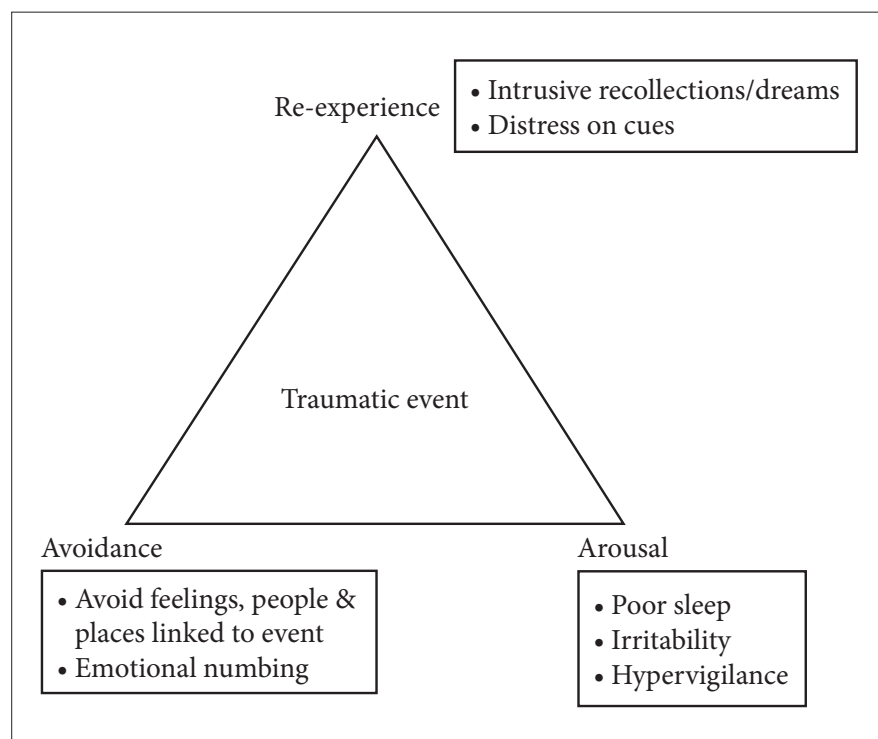


Fig.1. PTSD symptoms.

symptoms of heightened arousal (Fig. 1). There is a large overlap between ASD and PTSD, the difference being the duration and dissociative symptoms of ASD.

- Chronic PTSD refers to the presence of symptoms for 3 months or longer. It is often co-morbid with depression, substance use disorders, and other anxiety disorders. The individual remains reactive to environmental triggers or reminders and therefore symptoms often fluctuate.^[8]
- Delayed PTSD is the emergence of symptoms for the first time more than 6 months after the trauma.
- A subtype of PTSD has been described and is called 'disorders of extreme stress not otherwise specified' (DESNOS). It highlights a more chronic and disabling path and is associated with early age of onset of trauma, chronic interpersonal violence and sexual abuse.^[9]

The timelines described are important as they determine the appropriate action to be taken. The rest of this article focuses on the identification and management of ASD and PTSD.

Risk factors for the development of PTSD

A meta-analysis conducted by Brewin *et al.*^[10] found that factors such as gender, age at trauma and race predicted PTSD in some populations but not in others. Other pre-trauma predictive factors such as previous trauma, education, general childhood adversity and family psychiatric history were more uniform in their predictive effects. It was noted, however, that factors operating during the trauma and immediately afterwards had the strongest effects. Therefore emotional reactions during the event, such as panic-

like responses, dissociative responses, and pronounced distress, were associated with a higher frequency of developing PTSD, as well as trauma severity, additional life stressors and lack of support.^[11]

Assessment and treatment

Acute aftermath: Secondary prevention in PTSD – 'the golden hour/s'

Previously, the concept of debriefing, which involved the recall and rehearsing of the traumatic event, was recommended as the treatment of choice. The impetus for this was the understanding that this type of intervention could reduce continuing psychological difficulties and prevent the onset of PTSD. There is now strong evidence to suggest that psychological debriefing or critical incident stress management is no longer beneficial and in fact could delay recovery.^[12]

Recent work suggests that an alternative approach could potentially prevent the emergence of PTSD. Just as re-perfusion of an infarct-related artery in the first hour could reduce mortality, trauma researchers are suggesting that appropriate intervention immediately after trauma could prevent potential stress-related pathology such as PTSD. A distinction has been made in the literature between acute distress management and acute stress treatment, where treatment would traditionally refer to psychological debriefing and administration of medication. The aim of intervention at this stage is to assist with maintaining emotional control, restoring interpersonal communication, and helping the person to return to full functional capacity. This can be accomplished by ensuring that safety and basic needs such as shelter and nutrition are provided for.

Appropriate care for injuries, including pain control, is essential. Convey compassion and enlist basic listening skills. However, it is important not to force emotional reactions from the individual. Provide adequate information and assist in re-orientating the individual. Facilitate with mobilising support, which could involve practical processes such as contacting relatives. Lastly, affirm the expectation of returning to normality.^[11] Zohar suggests using the mnemonic ERASE (Table 1).

Normal anxiety is universally experienced in response to a threat.

The early administration of benzodiazepines to ameliorate distress or as a stress-busting prophylactic measure is no longer recommended. Studies suggest that it actually worsens outcome by interfering with the potent natural recovery process.^[13] Other novel interventions such as risperidone (an atypical antipsychotic),^[14] propanolol, and gabapentin,^[15] administered after traumatic events, all demonstrated no benefit in the

Table 1. Goals of acute stress management – ERASE

E	Reduce Exposure to stress (e.g. secure place)
R	Restore physiological needs (nutrition, pain control)
A	Provide Access to information/orientation
S	Locate source of Support (relatives, religion)
E	Emphasise Expectation of returning to normal

Adapted with permission from Zohar *et al.*^[11]

prevention of PTSD. Preliminary results from a study by Shelling *et al.* where the administration of prophylactic doses of cortisol to injured trauma survivors showed reduction in PTSD symptoms emergence is promising but needs to be replicated.¹¹⁶

However, there are certain procedures that should not be done. These are summarised as the 3 Ps: do not pathologise; do not psychologise; and do not pharmacologise (Table 2).

Acute stress disorder

ASD develops in the first 4 weeks after a traumatic event. Zohar *et al.* recommend 'watchful waiting' and reassurance during this period.¹⁷ There is no evidence for the routine use of medication, but if the patient suffers from insomnia a short-

acting hypnotic may be used. The patient must be followed up within 4 weeks to check if the symptoms have resolved, preferably by the doctor who initially saw the patient.

There is now strong evidence to suggest that psychological debriefing or critical incident stress management is no longer beneficial and could delay recovery.

The clinician should provide information to the victim and the carers about

symptoms and when to seek help, as well as of treatments available to treat symptoms if they should emerge. The ultimate aim is to normalise the experience, provide reassurance that only a minority of people will develop PTSD, but also to ensure help-seeking behaviour if symptoms should develop. If symptoms are distressing or continue, it is highly likely that the patient has developed acute PTSD.

PTSD

As a healthcare provider it would be pertinent to screen for symptoms of PTSD. Breslau and colleagues devised a 7-item short screening scale for PTSD,¹⁷ which has been shown to have validity and reliability in both primary care settings¹⁸ and general population samples¹⁹ (Table 3). A score

Table 2. What not to do – 3 Ps

Do not pathologise	i.e. 'This is a normal response to an abnormal situation'
Do not psychologise	Do not facilitate emotional reactions by e.g. group counselling or debriefing
Do not pharmacologise	Do not use benzodiazepines etc. in the first few hours

Adapted with permission from Zohar *et al.*¹⁷

Table 3. Screening PTSD

DSM-IV items that constitute the 7-item screening scale. In: Breslau N, Peterson EL, Kessler RC. Short screening scale for DSM-IV posttraumatic stress disorder. *Am J Psychiatry* 1999;156:908-911.¹⁷

C2	Did you avoid being reminded of this experience by staying away from certain places, people or activities? (Remind respondent of life event if necessary)	1. Yes 2. No
C4	Did you lose interest in activities that were once important or enjoyable? (Remind respondent of life event if necessary)	1. Yes 2. No
C5	Did you begin to feel more isolated or distant from other people? (Remind respondent of life event if necessary)	1. Yes 2. No
C6	Did you find it hard to have love or affection for other people? (Remind respondent of life event if necessary)	1. Yes 2. No
C7	Did you begin to feel that there was no point in planning for the future? (Remind respondent of life event if necessary)	1. Yes 2. No
D1	After this experience were you having more trouble than usual falling asleep or staying asleep? (Remind respondent of life event if necessary)	1. Yes 2. No
D5	Did you become jumpy or get easily startled by ordinary noises or movements? (Remind respondent of life event if necessary)	1. Yes 2. No

Based on the Diagnostic Interview Schedule for DSM-IV (DIS-IV), Washington Univ., St Louis, 1995).

The 7-item scale screens for DSM-IV PTSD in persons exposed to traumatic events as defined in DSM-IV. It is intended to be used only after establishing that the respondent has experienced a qualifying event. Please read the paper carefully. It contains all the information needed for using the scale. As we emphasise in the paper, the screening scale is not an adequate substitute for a psychiatric diagnosis.

With permission from Breslau *et al.*¹⁷

of 4 or above would identify patients who would benefit from further evaluation and management or referral to a specialty mental health service.

The expected response to a severe trauma is one of shock, fear, horror and helplessness. With time this response should disappear and the vast majority of individuals experience spontaneous recovery from these feelings.

Meta-analyses by the Cochrane Review^[20] and the National Institute of Clinical Excellence (NICE)^[21] found that trauma-focused cognitive-behaviour therapy (TF-CBT) and eye movement and desensitising reprocessing (EMDR) as psychological interventions have the strongest evidence for efficacy in PTSD. Both processes involve forms of exposure – TF-CBT with a cognitive therapy component, while EMDR involves the therapist asking the patient to visualise

aspects of the trauma while inducing saccadic eye movements.

Evidence from large randomised controlled trials support the use of selective serotonin re-uptake inhibitors (SSRIs) and the serotonin and noradrenaline re-uptake inhibitor (SNRI) venlafaxine as first-line treatment in the short- and long-term management of PTSD. Sertraline and paroxetine are FDA-approved for use in this disorder.^[22] Response can be seen as early as 2 - 4 weeks after starting medication but could take up to 12 weeks. In the long term, there is evidence that medication for the treatment of chronic PTSD should continue for at least 12 months to prevent relapse.^[23] Despite this, a large proportion of patients fail to respond to treatment with pharmacotherapy, and therefore augmentation is often needed. Furthermore, the morbidity of chronic PTSD is often high as a result of co-morbid conditions such as substance abuse, depression, suicidality and other anxiety disorders that are associated with chronicity of the condition. Findings from a Cochrane review on augmentation strategies in anxiety disorders^[24] suggest that adding a course of antipsychotics to ongoing treatment of SSRIs could be beneficial. Similarly, prazosin, an alpha-1 antagonist, holds promise.

Despite a substantive and growing body of evidence on the treatment of PTSD, there

still remains a paucity of information on the long-term treatment of PTSD, symptom remission, treatment-refractory patients and the effectiveness of treatments in naturalistic or 'real-world' settings.

Conclusion

Even though major advances have been made in PTSD research in the last few years, there still remain some pertinent unanswered questions. Currently, recommendations are the 'watch and wait' approach in the early days after a traumatic event. CBT and EMDR have the best evidence base as psychological interventions for the treatment of PTSD. However, the availability and cost-effectiveness of such interventions in a developing world setting have to be taken into consideration as both CBT and EMDR are specialised interventions usually performed by trained mental health providers. Furthermore, there is a valid place for pharmacotherapy in the management of PTSD, with the most compelling evidence for the use of SSRIs and venlafaxine.

References available at www.cmej.org.za

IN A NUTSHELL

- According to the SASH study the 12-month prevalence for PTSD is 2.3%.
- Immediate intervention after a traumatic event involves stress management rather than stress treatment.
- Acute stress management aims to assist with maintaining emotional control, restoring communication and helping the individual to return to full functionality.
- ASD is characterised by the presence of dissociative features, re-experiencing phenomena, avoidance behaviour, and symptoms of hyperarousal within 4 weeks of a traumatic event.
- There is no evidence for the efficacy of psychological debriefing or for the use of benzodiazepines after trauma.
- Acute PTSD criteria occur when re-experiencing phenomena, avoidance and hyperarousal symptoms are experienced 1 - 3 months after a traumatic event.
- Chronic PTSD is diagnosed when symptoms persist for more than 3 months.
- PTSD is often associated with multiple co-morbidities such as depression, other anxiety disorders and substance abuse.
- Trauma-focused CBT and EMDR have the most evidence base as psychological treatments.
- SSRIs and venlafaxine are used in the pharmacological management of PTSD.

symptoms of heightened arousal (Fig. 1). There is a large overlap between ASD and PTSD, the difference being the duration and dissociative symptoms of ASD.

- Chronic PTSD refers to the presence of symptoms for 3 months or longer. It is often co-morbid with depression, substance use disorders, and other anxiety disorders. The individual remains reactive to environmental triggers or reminders and therefore symptoms often fluctuate.^[8]
- Delayed PTSD is the emergence of symptoms for the first time more than 6 months after the trauma.
- A subtype of PTSD has been described and is called 'disorders of extreme stress not otherwise specified' (DESNOS). It highlights a more chronic and disabling path and is associated with early age of onset of trauma, chronic interpersonal violence and sexual abuse.^[9]

The timelines described are important as they determine the appropriate action to be taken. The rest of this article focuses on the identification and management of ASD and PTSD.

Risk factors for the development of PTSD

A meta-analysis conducted by Brewin *et al.*^[10] found that factors such as gender, age at trauma and race predicted PTSD in some populations but not in others. Other pre-trauma predictive factors such as previous trauma, education, general childhood adversity and family psychiatric history were more uniform in their predictive effects. It was noted, however, that factors operating during the trauma and immediately afterwards had the strongest effects. Therefore emotional reactions during the event, such as panic-

like responses, dissociative responses, and pronounced distress, were associated with a higher frequency of developing PTSD, as well as trauma severity, additional life stressors and lack of support.^[11]

Assessment and treatment

Acute aftermath: Secondary prevention in PTSD – 'the golden hour/s'

Previously, the concept of debriefing, which involved the recall and rehearsing of the traumatic event, was recommended as the treatment of choice. The impetus for this was the understanding that this type of intervention could reduce continuing psychological difficulties and prevent the onset of PTSD. There is now strong evidence to suggest that psychological debriefing or critical incident stress management is no longer beneficial and in fact could delay recovery.^[12]

Recent work suggests that an alternative approach could potentially prevent the emergence of PTSD. Just as re-perfusion of an infarct-related artery in the first hour could reduce mortality, trauma researchers are suggesting that appropriate intervention immediately after trauma could prevent potential stress-related pathology such as PTSD. A distinction has been made in the literature between acute distress management and acute stress treatment, where treatment would traditionally refer to psychological debriefing and administration of medication. The aim of intervention at this stage is to assist with maintaining emotional control, restoring interpersonal communication, and helping the person to return to full functional capacity. This can be accomplished by ensuring that safety and basic needs such as shelter and nutrition are provided for.

Appropriate care for injuries, including pain control, is essential. Convey compassion and enlist basic listening skills. However, it is important not to force emotional reactions from the individual. Provide adequate information and assist in re-orientating the individual. Facilitate with mobilising support, which could involve practical processes such as contacting relatives. Lastly, affirm the expectation of returning to normality.^[11] Zohar suggests using the mnemonic ERASE (Table 1).

Normal anxiety is universally experienced in response to a threat.

The early administration of benzodiazepines to ameliorate distress or as a stress-busting prophylactic measure is no longer recommended. Studies suggest that it actually worsens outcome by interfering with the potent natural recovery process.^[13] Other novel interventions such as risperidone (an atypical antipsychotic),^[14] propanolol, and gabapentin,^[15] administered after traumatic events, all demonstrated no benefit in the

Table 1. Goals of acute stress management – ERASE

E	Reduce Exposure to stress (e.g. secure place)
R	Restore physiological needs (nutrition, pain control)
A	Provide Access to information/orientation
S	Locate source of Support (relatives, religion)
E	Emphasise Expectation of returning to normal

Adapted with permission from Zohar *et al.*^[11]

prevention of PTSD. Preliminary results from a study by Shelling *et al.* where the administration of prophylactic doses of cortisol to injured trauma survivors showed reduction in PTSD symptoms emergence is promising but needs to be replicated.¹¹⁶

However, there are certain procedures that should not be done. These are summarised as the 3 Ps: do not pathologise; do not psychologise; and do not pharmacologise (Table 2).

Acute stress disorder

ASD develops in the first 4 weeks after a traumatic event. Zohar *et al.* recommend 'watchful waiting' and reassurance during this period.¹⁷ There is no evidence for the routine use of medication, but if the patient suffers from insomnia a short-

acting hypnotic may be used. The patient must be followed up within 4 weeks to check if the symptoms have resolved, preferably by the doctor who initially saw the patient.

There is now strong evidence to suggest that psychological debriefing or critical incident stress management is no longer beneficial and could delay recovery.

The clinician should provide information to the victim and the carers about

symptoms and when to seek help, as well as of treatments available to treat symptoms if they should emerge. The ultimate aim is to normalise the experience, provide reassurance that only a minority of people will develop PTSD, but also to ensure help-seeking behaviour if symptoms should develop. If symptoms are distressing or continue, it is highly likely that the patient has developed acute PTSD.

PTSD

As a healthcare provider it would be pertinent to screen for symptoms of PTSD. Breslau and colleagues devised a 7-item short screening scale for PTSD,¹⁷ which has been shown to have validity and reliability in both primary care settings¹⁸ and general population samples¹⁹ (Table 3). A score

Table 2. What not to do – 3 Ps

Do not pathologise	i.e. 'This is a normal response to an abnormal situation'
Do not psychologise	Do not facilitate emotional reactions by e.g. group counselling or debriefing
Do not pharmacologise	Do not use benzodiazepines etc. in the first few hours

Adapted with permission from Zohar *et al.*¹⁷

Table 3. Screening PTSD

DSM-IV items that constitute the 7-item screening scale. In: Breslau N, Peterson EL, Kessler RC. Short screening scale for DSM-IV posttraumatic stress disorder. *Am J Psychiatry* 1999;156:908-911.¹⁷

C2	Did you avoid being reminded of this experience by staying away from certain places, people or activities? (Remind respondent of life event if necessary)	1. Yes 2. No
C4	Did you lose interest in activities that were once important or enjoyable? (Remind respondent of life event if necessary)	1. Yes 2. No
C5	Did you begin to feel more isolated or distant from other people? (Remind respondent of life event if necessary)	1. Yes 2. No
C6	Did you find it hard to have love or affection for other people? (Remind respondent of life event if necessary)	1. Yes 2. No
C7	Did you begin to feel that there was no point in planning for the future? (Remind respondent of life event if necessary)	1. Yes 2. No
D1	After this experience were you having more trouble than usual falling asleep or staying asleep? (Remind respondent of life event if necessary)	1. Yes 2. No
D5	Did you become jumpy or get easily startled by ordinary noises or movements? (Remind respondent of life event if necessary)	1. Yes 2. No

Based on the Diagnostic Interview Schedule for DSM-IV (DIS-IV), Washington Univ., St Louis, 1995).

The 7-item scale screens for DSM-IV PTSD in persons exposed to traumatic events as defined in DSM-IV. It is intended to be used only after establishing that the respondent has experienced a qualifying event. Please read the paper carefully. It contains all the information needed for using the scale. As we emphasise in the paper, the screening scale is not an adequate substitute for a psychiatric diagnosis.

With permission from Breslau *et al.*¹⁷

of 4 or above would identify patients who would benefit from further evaluation and management or referral to a specialty mental health service.

The expected response to a severe trauma is one of shock, fear, horror and helplessness. With time this response should disappear and the vast majority of individuals experience spontaneous recovery from these feelings.

Meta-analyses by the Cochrane Review^[20] and the National Institute of Clinical Excellence (NICE)^[21] found that trauma-focused cognitive-behaviour therapy (TF-CBT) and eye movement and desensitising reprocessing (EMDR) as psychological interventions have the strongest evidence for efficacy in PTSD. Both processes involve forms of exposure – TF-CBT with a cognitive therapy component, while EMDR involves the therapist asking the patient to visualise

aspects of the trauma while inducing saccadic eye movements.

Evidence from large randomised controlled trials support the use of selective serotonin re-uptake inhibitors (SSRIs) and the serotonin and noradrenaline re-uptake inhibitor (SNRI) venlafaxine as first-line treatment in the short- and long-term management of PTSD. Sertraline and paroxetine are FDA-approved for use in this disorder.^[22] Response can be seen as early as 2 - 4 weeks after starting medication but could take up to 12 weeks. In the long term, there is evidence that medication for the treatment of chronic PTSD should continue for at least 12 months to prevent relapse.^[23] Despite this, a large proportion of patients fail to respond to treatment with pharmacotherapy, and therefore augmentation is often needed. Furthermore, the morbidity of chronic PTSD is often high as a result of co-morbid conditions such as substance abuse, depression, suicidality and other anxiety disorders that are associated with chronicity of the condition. Findings from a Cochrane review on augmentation strategies in anxiety disorders^[24] suggest that adding a course of antipsychotics to ongoing treatment of SSRIs could be beneficial. Similarly, prazosin, an alpha-1 antagonist, holds promise.

Despite a substantive and growing body of evidence on the treatment of PTSD, there

still remains a paucity of information on the long-term treatment of PTSD, symptom remission, treatment-refractory patients and the effectiveness of treatments in naturalistic or 'real-world' settings.

Conclusion

Even though major advances have been made in PTSD research in the last few years, there still remain some pertinent unanswered questions. Currently, recommendations are the 'watch and wait' approach in the early days after a traumatic event. CBT and EMDR have the best evidence base as psychological interventions for the treatment of PTSD. However, the availability and cost-effectiveness of such interventions in a developing world setting have to be taken into consideration as both CBT and EMDR are specialised interventions usually performed by trained mental health providers. Furthermore, there is a valid place for pharmacotherapy in the management of PTSD, with the most compelling evidence for the use of SSRIs and venlafaxine.

References available at www.cmej.org.za

IN A NUTSHELL

- According to the SASH study the 12-month prevalence for PTSD is 2.3%.
- Immediate intervention after a traumatic event involves stress management rather than stress treatment.
- Acute stress management aims to assist with maintaining emotional control, restoring communication and helping the individual to return to full functionality.
- ASD is characterised by the presence of dissociative features, re-experiencing phenomena, avoidance behaviour, and symptoms of hyperarousal within 4 weeks of a traumatic event.
- There is no evidence for the efficacy of psychological debriefing or for the use of benzodiazepines after trauma.
- Acute PTSD criteria occur when re-experiencing phenomena, avoidance and hyperarousal symptoms are experienced 1 - 3 months after a traumatic event.
- Chronic PTSD is diagnosed when symptoms persist for more than 3 months.
- PTSD is often associated with multiple co-morbidities such as depression, other anxiety disorders and substance abuse.
- Trauma-focused CBT and EMDR have the most evidence base as psychological treatments.
- SSRIs and venlafaxine are used in the pharmacological management of PTSD.

References

1. Kessler RC, et al. Posttraumatic stress disorder in the National Comorbidity Survey. *Arch Gen Psychiatry* 1995;52(12):1048-1060.
2. Frans O, et al. Trauma exposure and post-traumatic stress disorder in the general population. *Acta Psychiatr Scand* 2005;111(4):291-299.
3. Kessler RC, et al. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Arch Gen Psychiatry* 2005;62(6):593-602.
4. Stein MB, et al. Posttraumatic stress disorder in the primary care medical setting. *Gen Hosp Psychiatry* 2000;22(4):261-269.
5. Stein DJ, et al. Lifetime prevalence of psychiatric disorders in South Africa. *Br J Psychiatry* 2008;192(2):112-117.
6. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. Washington DC: APA, 1994.
7. Zohar J, et al. New insights into secondary prevention in post-traumatic stress disorder. *Dialogues Clin Neurosci* 2011;13(3):301-309.
8. Davidson JR, et al. Posttraumatic stress disorder: acquisition, recognition, course, and treatment. *J Neuropsychiatry Clin Neurosci* 2004;6(2):135-147.
9. Roth S, et al. Complex PTSD in victims exposed to sexual and physical abuse: results from the DSM-IV Field Trial for Posttraumatic Stress Disorder. *J Trauma Stress* 1997;10(4):539-555.
10. Brewin CR, Andrews B, Valentine JD. Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. *J Consult Clin Psychol* 2000;68(5):748-766.
11. Zohar J, et al. Can posttraumatic stress disorder be prevented? *CNS Spectr* 2009;14(1 Suppl 1):44-51.
12. Rose S, et al. Psychological debriefing for preventing post traumatic stress disorder (PTSD). *Cochrane Database Syst Rev* 2002;(2):CD000560.
13. Gelpin E, et al. Treatment of recent trauma survivors with benzodiazepines: a prospective study. *J Clin Psychiatry* 1996;57(9):390-394.
14. Stanovic JK, James KA, Vandevere CA. The effectiveness of risperidone on acute stress symptoms in adult burn patients: a preliminary retrospective pilot study. *J Burn Care Rehabil* 2001;22(3):210-213.
15. Pitman RK, et al. Pilot study of secondary prevention of posttraumatic stress disorder with propranolol. *Biol Psychiatry* 2002;51(2):189-192.
16. Schelling G, et al. Stress doses of hydrocortisone, traumatic memories, and symptoms of posttraumatic stress disorder in patients after cardiac surgery: a randomized study. *Biol Psychiatry* 2004;55(6):627-633.
17. Breslau N, Peterson EL, Kessler RC. Short screening scale for DSM-IV posttraumatic stress disorder. *Am J Psychiatry* 1999;156(6):908-911.
18. Kimerling R, et al. Brief report: Utility of a short screening scale for DSM-IV PTSD in primary care. *J Gen Intern Med* 2006;21(1):65-67.
19. Bohnert KM, Breslau N. Assessing the performance of the short screening scale for post-traumatic stress disorder in a large nationally-representative survey. *Int J Methods Psychiatr Res* 2011;20(1):e1-5.
20. Bisson J, Andrew M. Psychological treatment of post-traumatic stress disorder (PTSD). *Cochrane Database Syst Rev* 2005;(2):CD003388.
21. NICE. *Post-Traumatic stress disorder. The management of PTSD in adults and children in primary and secondary care. CG26*. London: National Institute for Health and Clinical Excellence, 2005.
22. Ipser JC, Stein DJ. Evidence-based pharmacotherapy of post-traumatic stress disorder (PTSD). *Int J Neuropsychopharmacol* 2011;Jul:1-16.
23. Bandelow B, et al. World Federation of Societies of Biological Psychiatry (WFSBP) guidelines for the pharmacological treatment of anxiety, obsessive-compulsive and post-traumatic stress disorders – first revision. *World J Biol Psychiatry* 2008;9(4):248-312.
24. Ipser JC, et al. Pharmacotherapy augmentation strategies in treatment-resistant anxiety disorders. *Cochrane Database Syst Rev* 2006;(4):CD005473.