

Editorial

QJM

Recognizing post-traumatic stress disorder

Post-traumatic stress disorder (PTSD) is a severe and complex disorder precipitated by exposure to a psychologically distressing event. PTSD first appeared in the Diagnostic and Statistical Manual of Psychiatric Disorders (DSM-III) in 1980,^{1,2} arising from studies of the Vietnam war, and of civilian victims of natural and man-made disasters.^{3,4} However, the study of PTSD dates back more than 100 years. Before 1980, post-traumatic syndromes were recognized by different names, including railway spine, shell shock, traumatic (war) neurosis, concentration-camp syndrome, and rape-trauma syndrome.^{2,3} The symptoms described in these syndromes overlap considerably with what we now recognize as PTSD. According to the most recent edition of the Diagnostic and Statistical Manual of Psychiatric Disorders (DSM-IV-TR),⁵ the essential feature of PTSD is the development of characteristic symptoms following exposure to an extreme traumatic stressor characterized by: direct personal experience of an event that involves actual or threatened death or serious injury, or other threat to one's physical integrity; or witnessing an event that involves death, injury, or a threat to the physical integrity of another person; or learning about unexpected or violent death, serious harm, or threat of death or injury experienced by a family member or other close associate. The person reacts to this event with fear and helplessness, and tries to avoid being reminded of it. Traumatic events include military combat, violent personal assault, being kidnapped, being taken hostage, terrorist attack, torture, incarceration, natural or man-made disasters, automobile accidents, or being diagnosed with a life-threatening illness. The principal symptoms of PTSD are the painful re-experiencing of the event, a pattern of avoidance and emotional numbing, and fairly constant hyperarousal.

Post-traumatic reactions fall on a continuum, and many clinicians and researchers believe it is useful to conceptualize partial symptoms as well.^{2,6–12} Clinical researchers see the need to classify such reactions as subsyndromal PTSD, or sub-threshold PTSD, or partial PTSD. Individuals who do not make

the full, strict criteria for a diagnosis of PTSD may be equally as impaired in functioning, and require the same level of care, as those who do.^{6,7} Subsyndromal PTSD may result from partial recovery from the full syndrome or from the development of subsyndromal symptoms after trauma. Subsyndromal PTSD has been noted to be highly prevalent among Vietnam war veterans, sexual abuse survivors, and other traumatized persons.^{6–12}

Among lay people, PTSD is commonly described as a normal reaction to an abnormal event, although PTSD does not affect the majority of survivors of trauma.^{4,13,14} A distinction should be made between the self-limiting distress response most people experience after exposure to severe trauma, and the persistent difficulty that some people experience in adaptation to such events. The failure in adaptation presents as psychological and biological abnormalities.

Although traumas such as rape, combat, assault, and tragic bereavement may appear different on the surface, victims of these events share what has been described as a violation of pre-existing schemata of the self and the world.¹⁵ Trauma has been characterized as breaking three basic assumptions: the belief in personal invulnerability, the perception of the world as meaningful, and the positive view of self. All victims must deal with the psychological distress caused by the violation of these basic beliefs.

Traumatic experiences are common, if we consider the preponderance of individuals exposed to sexual or non-sexual assault, natural disasters (e.g. flooding), accidents (e.g. work, motor vehicle), and war.^{5,14,16,17} Epidemiological studies have indicated a lifetime prevalence of exposure to traumatic events of 40%¹⁸ to 90%¹⁹ and a lifetime prevalence of PTSD in the community ranging from 1% to 9%.^{19–21} This indicates that not all people suffering traumatic events develop the disorder. Additional factors of personal vulnerability and post-trauma variables must be of importance.^{4,22–25} There is a strong correlation between the level of danger perceived by the individual exposed to the trauma

and the likelihood of developing PTSD.²⁴ Other predictive factors include previous exposure to trauma and a personal or family history of psychiatric disorder, particularly depressive disorder.^{4,5,25} Some persons seem to be more likely than others to develop PTSD because of their genetic composition.²² Genetic factors can influence the risk of exposure to some forms of trauma, probably, through individual differences in personality that influence environmental choices.²⁶ Many of the same genes that influence exposure to trauma appear to influence susceptibility to PTSD symptoms. It is difficult to know to what extent the increased vulnerability to PTSD in family members of trauma survivors is related to biological and genetic phenomena, as opposed to experiential ones, because of the large degree of shared environment in the families.²³

The comorbidity problems associated with PTSD are of great significance: the National Comorbidity Survey found that approximately 80% of patients with PTSD meet criteria for at least one other DSM disorder.²¹ Other studies found lifetime prevalences of major depressive disorder 30–36%, obsessive-compulsive disorder 15% and panic disorder 10–13%.^{18,20} Comorbidity between PTSD and depression is associated with greater symptom severity and higher risk for suicidal behavior.^{27–31} When comorbid psychiatric conditions are identified and treated, the intensity of PTSD symptoms usually decreases.^{32–34} Among treatment-seeking patients, 60–80% suffer from alcohol or drug abuse or dependence.^{21,34–36} Besides being associated with increased risk for depression, anxiety, and alcohol or substance use disorders, PTSD is associated with higher rates of hypertension, bronchial asthma, and peptic ulcer and with other diseases of the cardiovascular, digestive, musculoskeletal, endocrine, respiratory, and nervous systems, as well as increased rates of infectious disease for up to 20 years following exposure to major trauma.^{20,37,38}

People with PTSD might be expected to seek mental health treatment.³⁷ However, evidence suggests that this is not common and that, even in academic and community mental health settings, rates of recognition may be low, with clinical diagnosis of PTSD occurring in as few as 4% of individuals with the disorder.^{20,37,39,40} In a national cohort of Israeli primary care patients, 9% met criteria for current PTSD, but only 2% of actual cases were recognized by their treating physicians.⁴¹ Although 49% of physicians recognized the existence of psychological distress, this rate was still far less than the 88% frequency of self-rated distress in the sample. PTSD can be easily misdiagnosed and inappropriately treated.^{33,34}

How might PTSD be recognized, and what are its likely disguises? According to the DSM-IV-TR,⁵ PTSD can only be diagnosed if the existence of a traumatic event has been established. However, clinical suspicion may be warranted based on symptoms or behaviours, even if no such event has been established.³⁷ To elicit this information may in some cases be relatively straightforward. But in other cases, it will be more difficult, requiring the establishment of trust and confidence between patient and physician, and requiring the physician's unwillingness to accept initial denial as being the final answer in some cases.³⁷ Denial of PTSD may stem from a fundamental human difficulty in comprehending and acknowledging our own vulnerability.⁴² PTSD requires that physicians give patients adequate time for disclosing their stories. Psychological safety, which includes trust in the clinician and the associated ability to communicate extreme feelings and reactions, can decrease any potential that an assessment will increase self-destructive behaviour.⁴³

Findings on history-taking, including onset of symptoms as they relate to traumatic experiences, can help distinguish PTSD from other conditions.^{34,44} In some individuals, PTSD symptoms first emerge months, years, or even decades after exposure to traumatic event.¹⁴ In others, there may be a lifelong pattern of oscillating between active PTSD and remitted symptoms. Clinical presentation in PTSD is often very heterogeneous, with symptoms varying in intensity from patient to patient. Traumatic head injury, concussion, delirium, and seizure disorders must be ruled out. Alcohol and substance abuse, along with acute intoxication or withdrawal, must also be considered. PTSD must be delineated from factitious disorders, personality disorders, and malingering.^{5,34,44}

To ensure that the diagnosis is not missed, a brief trauma history should be included in all evaluations for anxiety or depression.⁴⁵ Traumatic events of adulthood can be asked about directly: for example, 'Have you ever been physically attacked or assaulted? Have you ever been in an automobile accident? Have you ever been in a war or disaster?' A positive response should alert the examiner to ask further about the relationship between the event and the current symptoms. Traumatic childhood experiences require reassuring statements of normality to put the patient at ease: 'Many people continue to think about frightening aspects of their childhood. Do you?'^{46,47}

The evaluation of PTSD frequently includes structured interviews and self-rating scales.^{49–55} The Clinician-Administered PTSD scale,⁴⁹ the Structured Interview for DSM-IV,⁵⁰ the Diagnostic

Interview Schedule,⁵¹ the Structured Interview for PTSD,⁵² the Davidson Trauma Scale,⁵³ the PTSD scale,⁵⁴ and other instruments can be used. Structured interviews and rating scales are also available for children with PTSD.^{49,55} A version of the Clinical-Administered PTSD Scale for children and adolescents has been developed.⁵⁵

Collateral assessment in PTSD is important because individuals with PTSD may have difficulties reporting on their condition due to denial, amnesia, avoidance, minimization, and/or cognitive impairment.⁴³ Collateral reports from spouses, partners, family members, or friends can provide valuable information to clinician and researchers. Prior medical, school, legal, and military records may also help to corroborate patients' reports of PTSD symptoms and prior functioning. Collateral reports can provide supplementary data that may not be seen under other assessment conditions.

The suggestion that PTSD at times may be associated with 'hypervigilance to symptoms' has been advanced and received some support from a study of abuse survivors with gastroesophageal reflux who showed lower cutaneous sensitivity thresholds and hypervigilance in labelling stimuli as being painful.^{37,56} Their pain-coping mechanisms were considered to be poor. Considerable evidence suggests that pain is one of the most commonly reported symptoms of patients with PTSD, regardless of the nature of their traumatic experience.⁵⁷ Similarly, patients who have persistent, chronic pain associated with musculoskeletal injury, serious burn injuries, and other pathologies frequently present with symptoms of PTSD. An unusually high rate of comorbid somatization disorder suggests that individuals with PTSD may be marked and persistent somatizers.^{20,37,41} Musculoskeletal, genitourinary, dermatological, respiratory, panic, sleep-related, and appetite disturbances are frequently seen. High rates of PTSD also have been reported in association with irritable bowel syndrome.⁴⁷ Thus, in the health care system, PTSD or the role of trauma should be considered in patients experiencing prominent chronic pain or persistent somatization, especially when these symptoms are not otherwise fully accounted for. Heavy smoking is much more likely in trauma survivors with PTSD, compared with those who had experienced similar trauma but did not have PTSD.⁵⁸

Evidence suggests that women experience more or different types of trauma than men, including labour and delivery, rape, and childhood sexual abuse.^{21,59} Although the prevalence of PTSD in response to miscarriage or stillbirth is unknown, some women clearly develop PTSD after pregnancy loss. Most common causes of PTSD in men are

combat and witnessing death or severe injury.²¹ Women may react to trauma more often and more robustly than men because of sex hormones, cultural gender roles, or some combination of those factors.⁵⁹

Psychological and pharmacological treatments of PTSD can produce a meaningful reduction in distress. However, an appropriate treatment can be administered only if a correct diagnosis is established. There is thus a pressing need to improve recognition of PTSD.

L. Sher

Division of Neuroscience
Department of Psychiatry
Columbia University
New York
USA
e-mail: LS2003@columbia.edu

References

1. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders. Third Edition.* Washington, DC, American Psychiatric Association, 1980:236–8.
2. Schnurr PP, Friedman MJ, Bernardy NC. Research on posttraumatic stress disorder: epidemiology, pathophysiology, and assessment. *J Clin Psychol* 2002; **58**:877–89.
3. Saigh PA, Bremner J. The history of posttraumatic stress disorder. In: Saigh PA, Bremner J, eds. *Posttraumatic Stress Disorder. A Comprehensive Text.* Boston, Allyn & Bacon, 1999:1–17.
4. Hageman I, Andersen HS, Jorgensen MB. Post-traumatic stress disorder: a review of psychobiology and pharmacotherapy. *Acta Psychiatr Scand* 2001; **104**:411–22.
5. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision.* Washington DC, American Psychiatric Association, 2000:463–8.
6. Yule W, Williams R, Joseph S. Post-traumatic stress disorder in adults. In: Yule W, ed. *Post-traumatic Stress Disorder. Concepts and Therapy.* Chichester UK, John Wiley & Sons, 1999:1–24.
7. Stein MB, Walker JR, Hazen AL, Forde DR. Full and partial posttraumatic stress disorder: findings from a community survey. *Am J Psychiatry* 1997; **154**:1114–19.
8. Carlier IVE, Gersons BPR. Partial posttraumatic stress disorder (PTSD): The issue of psychological scars and the occurrence of PTSD symptoms. *J Nerv Ment Dis* 1995; **183**:107–9.
9. Weiss DS, Marmar CR, Schlenger WE, Fairbank JA, Jordan BK, Hough RL, Kulka RA. The prevalence of lifetime and partial post-traumatic stress disorder in Vietnam theater veterans. *J Trauma Stress* 1992; **5**:365–76.
10. McLeer SV, Deblinger E, Atkins MS, Foa EB, Ralphe DL. Posttraumatic stress disorder in sexually abused children. *J Am Acad Child Adolesc Psychiatry* 1988; **27**:650–4.

11. Marshall RD, Olsson M, Hellman F, Blanco C, Guardino M, Struening EL. Comorbidity, impairment, and suicidality in subthreshold PTSD. *Am J Psychiatry* 2001; **158**:1467–73.
12. Amsel L, Marshall RD. Clinical management of subsyndromal psychological sequelae of the 9/11 terror attacks. In: Coates SW, Rosenthal JL, Schechter DS, eds. *September 11. Trauma and Human Bonds*. Hillsdale NJ, The Analytic Press, 2003:75–97.
13. Turner S. Place of pharmacotherapy in post-traumatic stress disorder. *Lancet* 1999; **354**:1404–5.
14. McFarlane AC. The prevalence and longitudinal course of PTSD. Implications for the neurobiological models of PTSD. *Ann NY Acad Sci*, 1997; **821**:10–23.
15. Solomon SD, Johnson DM. Psychosocial treatment of posttraumatic stress disorder: a practice-friendly review of outcome research. *J Clin Psychol* 2002; **58**:947–59.
16. McFarlane AC, Potts N. Posttraumatic stress disorder: prevalence and risk factors relative to disasters. In: Saigh PA, Bremner J., eds. *Posttraumatic Stress Disorder. A Comprehensive Text*. Boston, Allyn & Bacon, 1999:92–102.
17. Sullivan GM, Gorman JM. Finding a home for post-traumatic stress disorder in biological psychiatry. Is it a disorder of anxiety, mood, stress, or memory? *Psychiatr Clin N Am* 2002; **25**:463–8.
18. Breslau N, Davis GC, Andreski P, Peterson E. Traumatic events and posttraumatic stress disorder in an urban population of young adults. *Arch Gen Psychiatry* 1991; **48**:216–22.
19. Breslau N, Kessler RC, Chilcoat HD, Schultz LR, Davis GC, Andreski P. Trauma and posttraumatic stress disorder in the community: the 1996 Detroit Area Survey of Trauma. *Arch Gen Psychiatry* 1998; **55**:626–32.
20. Davidson JR, Hughes D, Blazer DG, George LK. Post-traumatic stress disorder in the community: an epidemiological study. *Psychol Med* 1991; **21**:713–21.
21. Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB. Posttraumatic stress disorder in the National Comorbidity Survey. *Arch Gen Psychiatry* 1995; **52**:1048–60.
22. True WR, Pitman R. Genetics and posttraumatic stress disorder. In: Saigh PA, Bremner J., eds. *Posttraumatic Stress Disorder. A Comprehensive Text*. Boston, Allyn & Bacon, 1999:144–59.
23. Yehuda R. Biological factors associated with susceptibility to posttraumatic stress disorder. *Can J Psychiatry* 1999; **44**:34–9.
24. Ballenger JC, Davidson JR, Lecrubier Y, Nutt DJ, Foa EB, Kessler RC, McFarlane AC, Shalev AY. Consensus statement on posttraumatic stress disorder from the International Consensus Group on Depression and Anxiety. *J Clin Psychiatry* 2000; **61** (Suppl. 5):60–6.
25. Koenen KC, Harley R, Lyons MJ, Wolfe J, Simpson JC, Goldberg J, Eisen SA, Tsuang M. A twin registry study of familial and individual risk factors for trauma exposure and posttraumatic stress disorder. *J Nerv Ment Dis* 2002; **190**:209–18.
26. Stein MB, Jang KL, Taylor S, Vernon PA, Livesley WJ. Genetic and environmental influences on trauma exposure and posttraumatic stress disorder symptoms: a twin study. *Am J Psychiatry* 2002; **159**:1675–81.
27. Acierno R, Resnick H, Kilpatrick DG, Saunders B, Best CL. Risk factors for rape, physical assault, and posttraumatic stress disorder in women: examination of differential multivariate relationships. *J Anxiety Disord* 1999; **13**:541–63.
28. Fava M, Rankin MA, Wright EC, Alpert JE, Nierenberg AA, Pava J, Rosenbaum JF. Anxiety disorders in major depression. *Compr Psychiatry* 2000; **41**:97–102.
29. Kaufman J, Charney D. Comorbidity of mood and anxiety disorders. *Depress. Anxiety* 2000; **12** (Suppl. 1):69–76.
30. Oquendo MA, Friend JM, Halberstam B, Brodsky BS, Burke AK, Grunebaum MF, Malone KM, Mann JJ. Association of comorbid posttraumatic stress disorder and major depression with greater risk for suicidal behavior. *Am J Psychiatry* 2003; **160**:580–2.
31. Sher L, Oquendo MA, Galfalvy HC, Cooper TB, Mann JJ. Age effects on cortisol levels in depressed patients with and without a history of posttraumatic stress disorder, and healthy volunteers. *J Affect Disord* 2004; in Press.
32. Eriksson NG, Lundin T. Early traumatic stress reactions among Swedish survivors of the m/s Estonia disaster. *Br J Psychiatry* 1996; **169**:713–16.
33. Grossman LS, Willer JK, Stovall JG, McRae SG, Maxwell S, Nelson R. Underdiagnosis of PTSD and substance use disorders in hospitalized female veterans. *Psychiatr Serv* 1997; **48**:393–5.
34. Khouzam HR, Donnelly NJ. Posttraumatic stress disorder. Safe, effective management in the primary care setting. *Postgrad Med* 2001; **110**:60–2, 67–70, 77–8.
35. Branchey L, Davis W, Lieber CS. Alcoholism in Vietnam and Korea veterans: a long term follow-up. *Alcohol Clin Exp Res* 1984; **8**:572–5.
36. Perkonig A, Kessler RC, Storz S, Wittchen HU. Traumatic events and post-traumatic stress disorder in the community: prevalence, risk factors and comorbidity. *Acta Psychiatr Scand* 2000; **101**:46–59.
37. Davidson JR. Recognition and treatment of posttraumatic stress disorder. *JAMA* 2001; **286**:584–8.
38. Boscarino JA. Diseases among men 20 years after exposure to severe stress: implications for clinical research and medical care. *Psychosom Med* 1997; **59**:605–14.
39. Amaya-Jackson L, Davidson JR, Hughes DC, Swartz M, Reynolds V, George LK, Blazer DG. Functional impairment and utilization of services associated with posttraumatic stress in the community. *J Trauma Stress* 1999; **12**:709–24.
40. Switzer GE, Dew MA, Thompson K, Goycoolea JM, Derricott T, Mullins SD. Posttraumatic stress disorder and service utilization among urban mental health center clients. *J Trauma Stress* 1999; **12**:25–39.
41. Taubman-Ben-Ari O, Rabinowitz J, Feldman D, Vaturi R. Post-traumatic stress disorder in primary-care settings: prevalence and physicians' detection. *Psychol Med* 2001; **31**:555–60.
42. Solomon Z. Oscillating between denial and recognition of PTSD: why are lessons learned and forgotten? *J Trauma Stress* 1995; **8**:271–82.
43. Newman E, Kaloupek DG, Keane TM. Assessment of posttraumatic stress disorder in clinical and research settings. In: Van der Kolk BA, McFarlane AC, Weisaeth L, eds. *Traumatic Stress. The Effects of Overwhelming Experience on Mind, Body, and Society*. New York, The Guilford Press, 1996:242–75.
44. Jacobs WJ, Dalenberg C. Subtle presentations of post-traumatic stress disorder. Diagnostic issues. *Psychiatr Clin North Am* 1998; **21**:835–45.

45. Lange JT, Lange CL, Cabaltica RB. Primary care treatment of post-traumatic stress disorder. *Am Fam Physician* 2000; **62**:1035–40, 1046.
46. Blank AS Jr. Clinical detection, diagnosis, and differential diagnosis of post-traumatic stress disorder. *Psychiatr Clin North Am* 1994; **17**:351–83.
47. Irwin C, Falsetti SA, Lydiard RB, Ballenger JC, Brock CD, Brenner W. Comorbidity of posttraumatic stress disorder and irritable bowel syndrome. *J Clin Psychiatry* 1996; **57**:576–8.
48. Beckham JC, Davidson JRT, March JS. Anxiety disorders: traumatic stress disorders. In: Tasman A, Kay J, Lieberman JA, eds. *Psychiatry*, 2nd edn, vol. 2. Chichester UK, John Wiley & Sons, 2003:1362–79.
49. Blake DD, Weathers FW, Nagy LM, Kaloupek DG, Klauminzer G, Charney DS, Keane TM. A clinician rating scale for assessing current and lifetime PTSD: The CAPS-1. *Behav Ther* 1990; **13**:187–8.
50. Spitzer R, Williams JBW, Gibbon M. *Structured Clinical Interview for DSM-III-R*. New York, New York State Psychiatric Institute, 1989.
51. Robins LN, Helzer JE, Croughan J, Ratcliff KS. The NIMH Diagnostic Interview Schedule, its history, characteristics, and validity. *Arch Gen Psychiatry* 1981; **38**:381–9.
52. Davidson JRT, Smith RD, Kudler HS. Validity and reliability of the DSM-III diagnostic criteria for posttraumatic stress disorder: experience with a structured interview. *J Nerv Ment Dis* 1989; **177**:330–41.
53. Davidson JRT, Book SW, Colket JT, Tupler LA, Roth S, David D, Hertzberg M, Mellman T, Beckham JC, Smith RD, Davison RM, Katz R, Feldman ME. Assessment of a new self-rating scale for posttraumatic stress disorder: The Davidson Trauma Scale. *Psychol Med* 1997; **27**:153–60.
54. Foa EB, Riggs DS, Dancu CV, Rothbaum BO. Reliability and validity of a brief instrument for assessing posttraumatic stress disorder. *J Trauma Stress* 1993; **6**:459–73.
55. Nader K, Kriegler J, Blake DD, Pynoos RS. *Clinician Administered PTSD Scale for Children (CAPS-C). Current and Lifetime Diagnosis Version and Instruction Manual*. UCLA Neuropsychiatric Institute and National Center for PTSD, 1994.
56. Scarinci IC, McDonald-Haile J, Bradley LA, Richter JE. Altered pain perception and psychosocial features among women with gastrointestinal disorders and history of abuse: a preliminary model. *Am J Med* 1994; **97**:108–18.
57. Asmundson GJG, Coons MJ, Taylor S, Katz J. PTSD and the experience of pain: research and clinical implications of shared vulnerability and mutual maintenance models. *Can J Psychiatry* 2002; **47**:930–7.
58. Beckham JC, Kirby AC, Feldman ME, Hertzberg MA, Moore SD, Crawford AL, Davidson JR, Fairbank JA. Prevalence and correlates of heavy smoking in Vietnam veterans with chronic posttraumatic stress disorder. *Addict Behav* 1997; **22**:637–47.
59. Miller LJ, Wiegartz P. Posttraumatic stress disorder. How to meet women's specific needs. *Curr Psychiatry* 2003; **2**:25–6, 35–9.



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