Women and Traumatic Events

Edna B. Foa, Ph.D., and Gordon P. Street, Ph.D.

Posttraumatic stress disorder (PTSD) gained the status of a psychiatric disorder in 1980, although the syndrome had already been recognized widely for many years. PTSD is distinguished by alternations between reexperiencing of the traumatic event that triggered the PTSD in the first place and avoidance and numbing. Increased arousal (e.g., exaggerated startle reaction) also forms part of the diagnosis. Although the majority of trauma victims recover spontaneously, more than 30% develop persistent PTSD symptoms, with women being twice as likely as men to suffer PTSD. To date, the most studied psychosocial treatments for PTSD are the cognitive-behavioral interventions. Exposure therapy (systematic exposure to the traumatic memory in a safe environment) has been demonstrated to be quite effective with adult women who were sexually or nonsexually assaulted in adulthood as well as with women who were sexually abused in childhood. Supportive counseling does not appear as effective as exposure therapy, but is better than no therapy.

(J Clin Psychiatry 2001;62[suppl 17]:29–34)

Posttraumatic stress syndrome has been recognized for more than 100 years under a variety of labels such as *compensation neurosis*,[1] *nervous shock*,[2] *hysteria*,[3] *traumatophobia*,[4] and *war neurosis*,[5] with the use of these general terms reflecting an awareness that different experiences produce similar symptoms. The official recognition of the syndrome in the diagnostic nomenclature is quite recent; it was first introduced as posttraumatic stress disorder (PTSD) in the *Diagnostic and Statistical Manual of Mental Disorders*, Third Edition (DSM-III), in 1980.[6]

PTSD is currently defined according to sets of specific diagnostic criteria established by DSM-IV[7] and ICD-10[8] (see review by Shalev[9] elsewhere in this supplement). According to these definitions, the traumatic event triggering PTSD must have been of exceptional severity, involving actual or perceived threat to physical integrity, and the person's emotional response must have included intense fear or helplessness.[7] PTSD is distinguished by (1) reexperiencing the traumatic event[10] (e.g., nightmares and flashbacks), (2) avoidance and numbing[11] (regarding thoughts and reminders of the trauma), and (3) increased arousal[12] (e.g., problems in sleeping and concentrating, exaggerated startle reaction). Alternations of reexperiencing and numbing characterize the disorder and distinguish it from other anxiety disorders,[12] although there is considerable overlap between PTSD and other anxiety disorders.

Most patients experience spontaneous reduction of PTSD symptoms within the first year after the trauma. Only a minority of traumatized victims develop PTSD, but more than 30% of all traumatized victims still have PTSD symptoms after 10 years.[13] For this minority, something has gone wrong in the natural recovery process. One can perceive effective treatment as facilitating these processes and thus encouraging recovery. Because of the recent recognition of PTSD as a formal disorder, critical issues such as predictors of failure to recover from a trauma and the development and evaluation of effective treatments have just begun to be addressed in research using rigorous methodology.

SCOPE OF THE PROBLEM

Individuals differ in their ability to recover from a traumatic experience, and traumas differ in their likelihood to produce PTSD. Furthermore, although men are more likely to be traumatized than women, women appear to be twice as likely as men to suffer from PTSD.[13] In a retrospective study[14] of the prevalence of PTSD among a representative sample of women in the United States, chronic PTSD was diagnosed in 17.8% of physical assault victims (N = 9.9 million), 12.4% of sexual assault victims (N = 13.8 million), and 3.4% of female victims of noncrime trauma (N = 32.0 million). Thus female victims of physical or sexual assaults are 4 to 5 times more likely to develop PTSD than female victims of noncrime trauma.

From the Center for the Treatment and Study of Anxiety, Department of Psychiatry, University of Pennsylvania, Philadelphia.

Presented at the advisory board meeting "Understanding Posttraumatic Stress Disorder," which was held March 8–9, 1999, in Cannes, France, and supported by an unrestricted educational grant from Janssen-Cilag.

Reprint requests to: Edna B. Foa, Ph.D., Center for the Treatment and Study of Anxiety, Department of Psychiatry, University of Pennsylvania, 3535 Market St., Philadelphia, PA 19104 (e-mail: foa@mail.med.upenn.edu).

Rape Versus Nonsexual Assault

Three prospective studies[15–17] investigated the occurrence of PTSD in assault victims (primarily women) beginning within 1 month after rape or nonsexual assault. Victims were 17 to 65 years of age, literate in English, and had not been assaulted by their current spouse or partner.

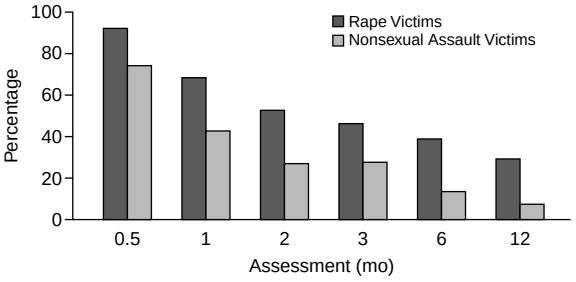
In the first study,[15] posttrauma reactions of 64 rape victims (all women) were assessed weekly for 3 months. In the initial assessment, approximately 2 weeks after assault, almost all rape victims (94%) met symptomatic criteria for PTSD. The incidence of PTSD declined steadily during the assessment period, as some victims seemed to process the trauma successfully. However, after 3 months, 47% of rape victims remained sufficiently disturbed to meet diagnostic criteria for PTSD symptoms.

In the second study,[16] posttrauma reactions of 60 victims of nonsexual assault (38 women and 22 men) were also assessed weekly for 3 months starting about 1 week after the assault. At the initial interview, 71% of the women (and 50% of the men) met symptomatic criteria for PTSD. After 3 months, 21% of the women (but none of the men) still met PTSD criteria.

The third and largest study,[17] which examined the rate of PTSD in 196 women (96 rape victims and 100 nonsexual assault victims), yielded similar results (Figure 1). About 2 weeks after the assault, 92% of rape victims showed signs of PTSD, compared with 74% of nonsexual assault victims. Again, a steady decline was observed, so that by month 3, 47% of rape victims and 27% of nonsexual assault victims had PTSD. Six months after the assault, 38% of rape victims compared with 13% of nonsexual assault victims still met diagnostic criteria for PTSD.

Across these 3 studies, lower rates of PTSD were observed in victims of nonsexual assault compared with victims of rape. Thus, a higher proportion of rape victims failed to process their rape experience successfully compared with nonsexual assault victims.

Figure 1. Incidence of Posttraumatic Stress Disorder During the Year After the Assault Among 196 Victims of Rape and Nonsexual Assault[a]



[a]Reprinted from Foa,[17] with permission.

FACTORS IMPEDING NATURAL RECOVERY

Since the 19th century, trauma theorists have postulated that deliberate avoidance of reminders of the trauma, or emotional withdrawal and dissociation, are factors that hinder natural recovery from a traumatic event.[18–21] Indeed, avoidance is included among the numbing symptoms of PTSD in DSM-IV.[7] Retrospective reports of dissociative experiences during trauma have been found related to the severity of PTSD later.[22–24]

Many trauma theorists also hold that the natural process of recovery involves special processing of traumatic memories: abilities to organize,[25] streamline,[25] and articulate[26] the trauma memory have been suggested as pointers to recovery. Clinical observations suggest that trauma narratives recounted by clients with chronic PTSD are characterized by an abundance of incomplete sentences and thought utterances that reflect confusion.[27,28]

Another popular idea among trauma theorists is that the successful processing of a traumatic experience requires adjustments in beliefs about the world and about oneself.[20,29–31] Some theorists postulate that a traumatic experience violates the beliefs inherent in human beings that the world is benevolent and that the self is invulnerable.[30] In the study described above of recent rape and nonsexual assault victims,[17] individuals who had PTSD 3 months after the trauma exhibited a less positive view about the world and themselves immediately after the trauma than did victims who recovered.

Thus, the evidence suggests that the degree to which a victim (1) is emotionally engaged with the traumatic memory, (2) organizes and articulates the traumatic memory, and (3) maintains a balanced view about the world and self determines whether he or she will recover or develop chronic disturbances.

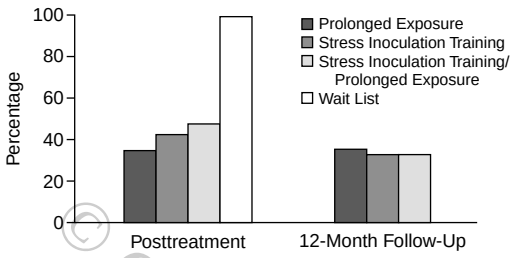
COGNITIVE-BEHAVIORAL TREATMENT

To date, the most studied psychosocial treatments for PTSD are the cognitive-behavioral interventions. Two cognitive-behavioral programs for PTSD were used with the introduction of the disorder in DSM-III[6]: exposure therapy[32] and stress inoculation training.[33] Despite many methodological flaws, studies to date confirm that these 2 procedures are effective in reducing symptoms of PTSD.[34]

Exposure Therapy

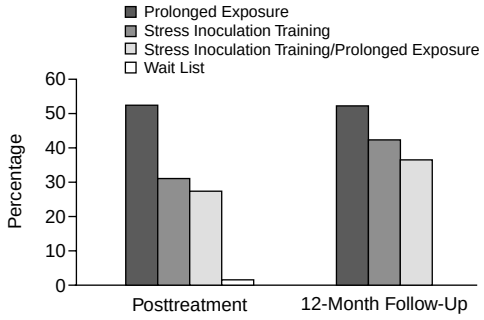
Exposure therapy is composed of a set of techniques designed to help patients confront their fears of objects, situations, memories, and images (e.g., systematic desensitization and flooding). The program developed by Foa and colleagues[35–37] includes (1) education about common reactions to trauma, (2) breathing retraining, (3) prolonged, repeated exposure to the trauma memory (reliving), and (4) repeated in vivo exposure to situations the

Figure 2. Percentage of Patients With Posttraumatic Stress Disorder Diagnosis Following Treatment and at 12-Month Follow-Up (N = 96)[a]



[a]Reprinted from Foa et al.,[35] with permission.

Figure 3. Percentage of Responders With Good End-State Functioning[a] Following Treatment (N = 96)[b]



[a]Defined as symptom scores below 20 on the PTSD Symptom Scale-Interview Version, below 10 on the Beck Depression Inventory, and below 40 on the State-Trait Anxiety Inventory.
[b]Reprinted from Foa et al.,[35] with permission.

patient is avoiding because of assault-related fear. The hypothesized mechanisms of change in exposure therapy are (1) fear activation via confrontation with object or memory matched to what the person is afraid of and (2) corrective information provided during exposure. Exposure therapy is widely believed to be the most effective approach for the treatment of phobias.

Stress Inoculation Training

Stress inoculation, or anxiety management training, teaches patients the skills to manage anxiety when it occurs. This approach does not attempt to bring about permanent change. The procedures involve (1) relaxation training, (2) thought stopping, (3) guided self-dialogue, (4) cognitive restructuring, (5) covert modeling, and (6) role-playing.

Cognitive Therapy (Cognitive Restructuring)

Cognitive therapy is based on the assumption that emotional disturbances reflect distorted, dysfunctional (unrealistic) cognitions.[38,39] It has been further proposed that the different anxiety disorders reflect specific cognitive structures that involve particular cognitive distortions.[40] For example, panic disorder is characterized by the distorted cognition that certain bodily sensations (e.g., heart palpitations) are dangerous.[41] The goal of cognitive therapy is to ameliorate the target symptoms by helping patients identify their dysfunctional, unrealistic cognitions, evaluate their validity, and replace them with functional cognitions.

Randomized Trials of Exposure Therapy, Stress Inoculation Training, and Cognitive Therapy

In a randomized trial,[35] prolonged exposure, stress inoculation training, and a combination of stress inoculation training and prolonged exposure were compared with one another and with a wait list control.[34,36,37] Treatment consisted of 9 twice-weekly individual sessions conducted over a 5-week period. Patients who were on a wait list for treatment served as control subjects.

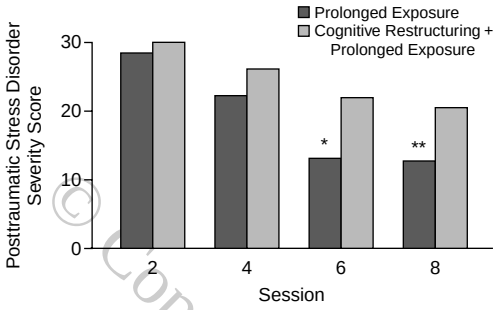
A total of 96 female patients with a mean age of 34.9 years were randomly assigned to the treatment groups. All patients met DSM-III-R criteria for PTSD.[42] Most had experienced rape (72%). The remainder (28%) had experienced nonsexual assault. All patients had chronic PTSD. Almost half (48%) reported at least one incident of physical or sexual abuse during childhood.

Following treatment, 100% of wait list controls had a PTSD diagnosis, compared with 35% after prolonged exposure treatment, 42% after stress inoculation training, and 46% after stress inoculation training plus prolonged exposure. At the 12-month postassault assessment, the respective figures for prolonged exposure, stress inoculation training, and stress inoculation training plus prolonged exposure were 35%, 32%, and 32% (Figure 2). All 3 active treatments reduced the incidence of PTSD compared with controls but did not differ significantly from each other. Overall, gains were retained throughout the follow-up period.

More stringent recovery criteria were then applied. "Good end-state functioning" was defined as scoring below 3 cutoff scores: PTSD severity below 20 on the PTSD Symptom Scale-Interview Version,[43] depression below 10 on the Beck Depression Inventory,[44–46] and a state anxiety score below 40 on the State-Trait Anxiety Inventory.[47] Immediately after treatment, more than half of the patients who received prolonged exposure met these stringent criteria (Figure 3) compared with less than one third of patients who received stress inoculation training (31%) or stress inoculation training plus prolonged exposure (27%). However, the differences between the active treatments fell short of significance at posttreatment, and by the 12-month posttreatment assessment, all 3 treatments were associated with "good end-state functioning" in at least one third of patients.

Large effect sizes were observed for all symptoms (reexperiencing, arousal, avoidance, anxiety, and depression) for all 3 treatments. However, exposure treatment tended

Figure 4. A Comparison Between Prolonged Exposure Only and Prolonged Exposure Plus Cognitive Restructuring on Self-Reported Severity of Posttraumatic Stress Disorder During Treatment (N = 96)[a]



[a]E.B.F., unpublished data, July 1999. Scale used is the PTSD Symptom Scale-Interview Version.[43]
*p < .05.
**p < .10 versus cognitive restructuring + prolonged exposure.

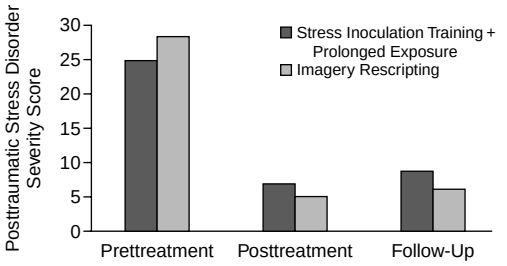
to yield superior outcome at follow-up on all measures. Curiously, the combination of prolonged exposure and stress inoculation training was not superior to either treatment alone in reducing PTSD and related symptoms. Information overload and doubled homework assignments may have been contributing factors, and more therapy time may be needed for the combined treatment program.

In an ongoing randomized trial (E.B.F., unpublished data, July 1999), the relative benefits of exposure therapy alone and exposure therapy combined with cognitive restructuring are being evaluated. Patients are randomly assigned to one of the following groups: (1) prolonged exposure, (2) exposure therapy and cognitive restructuring, or (3) wait list control. Treatment consists of 9 weekly sessions (extended to 12 for partial responders who show less than 70% improvement). A total of 96 female patients had completed the treatment phase at the time of publication. They had a mean ± SD age of 32.1 ± 9.6 years and a mean duration of 8.9 ± 9.4 years since assault. Most (65%) were victims of rape, for 20%, childhood sexual abuse was the index trauma, and 15% were treated for nonsexual assault.

Both prolonged exposure and prolonged exposure combined with cognitive restructuring significantly reduced the severity of PTSD when assessed according to independent evaluation and self-report. After treatment, prolonged exposure and prolonged exposure with cognitive restructuring were associated with lower PTSD severity scores than wait list controls ($p < .05$). The effect was sustained at the 3-month follow-up visit.

Some patients did not achieve 70% reduction of PTSD symptoms. They were classified as partial responders and received a treatment extension to 12 sessions. Only 43% of prolonged exposure patients needed such extended treatment, whereas two thirds of patients treated with prolonged exposure plus cognitive restructuring required extension, suggesting that prolonged exposure alone may be

Figure 5. A Comparison Between the Effects of Prolonged Exposure Plus Stress Inoculation and Imagery Rescripting on Severity of Posttraumatic Stress Disorder (N = 12)[a]



[a]Reprinted from Smucker et al.,[49] with permission.

the more efficient treatment program. At sessions 6 and 8, PTSD severity was reduced to a significantly greater extent for prolonged exposure alone compared with prolonged exposure combined with cognitive restructuring (Figure 4).

Further support for prolonged exposure and cognitive therapy was provided by Marks et al.,[48] who compared exposure, cognitive restructuring, the combination, and a relaxation control condition. As in the ongoing study (E.B.F., unpublished data, July 1999), good end-state functioning was observed posttreatment. Similar to our findings, exposure therapy alone yielded the greatest number of participants achieving good end-state functioning, although these differences failed to reach significance.

The studies described above were conducted primarily with trauma victims whose traumas occurred in adulthood. However, cognitive-behavioral programs were found to be successful in reducing PTSD following childhood sexual abuse. In a pilot study of 12 childhood sexual abuse survivors with PTSD, Smucker et al.[49] used a variant of exposure therapy termed *imagery rescripting*. This technique involves (1) prolonged reliving of the childhood sexual abuse memory, (2) mastery imagery (driving out the perpetrator), (3) adult-nurturing-child imagery, (4) writing a letter to the perpetrator or other family member, and (5) the adult "checking in" with the child. Patients were randomly assigned to 9 sessions of either stress inoculation training plus prolonged exposure or imagery rescripting (2 sessions lasting 120 minutes and 7 sessions lasting 90 minutes).

The 12 patients who participated in the study had experienced childhood sexual abuse at 4 to 11 years of age. The mean age of survivors enrolled in the study was 35 years, and most (64%) were rape victims. Childhood sexual abuse had been carried out by a father or stepfather in 67% of cases and had occurred at least once a week in 75% of victims.

Mean severity of all PTSD symptoms (reexperiencing, avoidance, and arousal) was markedly reduced following stress inoculation training plus prolonged exposure and

imagery rescripting treatment; the reduction was sustained at follow-up (Figure 5).

Cognitive Processing Therapy

Cognitive processing therapy (CPT) is another combined treatment approach specifically tailored to treat the symptoms of PTSD in rape victims.[50] The treatment approach is concerned with processing the traumatic memory (see Factors Impeding Natural Recovery). CPT includes exposure and cognitive components, but these differ from those described above. In CPT, exposure consists of repeatedly writing accounts of the traumatic experience. Another factor distinguishing CPT from traditional cognitive therapy is the focus of cognitive restructuring on 5 primary themes—safety, trust, power, esteem, and intimacy—believed to comprise the core difficulties in female rape victims.[31] Treatment consists of 12 weekly sessions.

In a quasi-experimental study, Resick and Schnicke[50] treated 19 rape victims with group CPT and compared their results with a wait list control group. Assessment of PTSD symptoms and depression, obtained by standardized self-report, suggested that CPT had a similar benefit to the cognitive-behavioral approach. Clients who received CPT showed greater reduction in PTSD symptoms and depression than controls (on one self-report PTSD scale, mean symptom reduction for CPT was 40% vs. 1.5% for wait list controls), and the improvement was sustained at 6-month follow-up.

CONCLUSION

Three important psychological factors involved in the successful processing of a traumatic event are emotional engagement with the trauma memory, organization and articulation of the trauma narrative, and maintaining balanced core beliefs about the world and about oneself. All 3 factors are related to natural recovery from a recent trauma. As noted below, there is also evidence that these factors are involved in successfully reducing PTSD severity via exposure therapy.[51]

A common misconception of individuals with anxiety disorders is that anxiety during confrontation with a feared situation remains indefinitely unless escape is realized and will cause "disastrous consequences" such as loss of control. In fact, anxiety ameliorates (habituates) during systematic prolonged or repeated exposures to anxiety-evoking situations. That is, when anxious individuals are exposed repeatedly to the situation they dread, they become less fearful of that situation. The mechanisms of habituation are not clear, but relate to the patients' interpretation of their anxiety reduction, i.e., patients realize that anxiety does not remain "forever" and that a confrontation with feared situations or memories brings about a reduction in anxiety rather than the anticipated loss of control or "going crazy."

Exposure therapy in particular has been demonstrated to work well for PTSD and related disturbances (e.g., depression) following adult victimization and childhood abuse. Counseling does not appear as effective as exposure therapy, but is better than no therapy. In the first author's experience, the vast majority of PTSD patients embrace exposure therapy if the rationale for treatment is clearly explained and its effectiveness delineated. The process is interactive between patient and therapist and is altered to fit the individual responses of the patient as therapy progresses.

Indications for using exposure treatment include pervasive trauma-related anxiety and avoidance, anxiety about the PTSD symptoms themselves, and fear of loss of control or "going crazy." Interpretation of the PTSD symptoms as dangerous ("something terrible is going to happen to me") is highly predictive of PTSD development and responds well to exposure therapy. Contraindications include psychosis, *severe* dissociative symptoms, substance abuse, and PTSD symptoms related to *realistic* guilt and shame, for example, when the patient was traumatized by being the perpetrator in the trauma.

Rates of traumatic events and resulting PTSD are high, and, because of the chronicity of the disorder, the PTSD population is always growing. It is therefore imperative that we use our increasing knowledge of the biological and psychological factors involved in PTSD to develop efficacious and cost-effective treatments for these individuals.

REFERENCES

1. Rigler R. Bever die Folgen der Verletzungen auf Eisenbahnen, insbesonder der Verletzungen des Ruckenmarks. Mit Hinblick auf das Haftpflichfgesetz dargestellf VIII 1879;124:80
2. Page H. Injuries of the Spine and Spinal Cord Without Apparent Mechanical Lesion. London, England: J & A Churchill; 1885
3. Putnam JJ. Recent investigations into patients of so-called concussion of the spine. Bot Med Surg J 1881;109:217
4. Rado S. Pathodynamics and treatment of traumatic war neurosis (traumatophobia). Psychosom Med 1942;42:363–368
5. Grinker RR, Spiegel JP. War Neurosis in North Africa, the Tunisian Campaign, January to May 1943. New York, NY: Josiah Macy Foundation; 1943
6. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Third Edition. Washington, DC: American Psychiatric Association; 1980
7. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. Washington, DC: American Psychiatric Association; 1994
8. World Health Organization. The ICD-10 Classification of Mental and Behavioural Disorders: Clinical Descriptions and Diagnostic Guidelines. Geneva, Switzerland: World Health Organization; 1992
9. Shalev AY. What is posttraumatic stress disorder? J Clin Psychiatry 2001; 62(suppl 17):4–10
10. Foa EB, Rothbaum BO. Posttraumatic stress disorder: clinical features and treatment. In: Peters RD, McMahon RJ, Quinsey VL, eds. Aggression and Violence Throughout the Life Span. Newbury Park, Calif: Sage; 1992: 155–170
11. Litz BT. Emotional numbing in combat-related posttraumatic stress disorder: a critical review and reformulation. Clin Psychol Rev 1993;12: 417–432
12. Foa EB, Riggs DS, Gershuny BS. Arousal, numbing and intrusion: symptom structure of post traumatic stress disorder following assault.

Am J Psychol 1995;152:116–120
13. Kessler RC, Sonnega A, Bromet E, et al. Posttraumatic stress disorder in the National Comorbidity Survey. Arch Gen Psychiatry 1995;52: 1048–1060
14. Resnick HS, Kilpatrick DG, Dansky BS, et al. Prevalence of civilian trauma and posttraumatic stress disorder in a representative national sample of women. J Consult Clin Psychol 1993;61:984–991
15. Rothbaum BO, Foa EB, Murdock T, et al. A prospective examination of post-traumatic stress disorder in rape victims. J Trauma Stress 1992;5: 455–475
16. Riggs DS, Rothbaum BO, Foa EB. A prospective examination of symptoms of posttraumatic stress disorder in victims of nonsexual assault. J Interpersonal Violence 1995;10:201–214
17. Foa EB. Failure of emotional processing: post trauma psychopathology. Presented at the World Congress of Behavioural & Cognitive Therapies; July 15, 1995; Copenhagen, Denmark
18. Janet PL. L'Automatisme Psychologique. Paris, France: Felix Alcan; 1889
19. Freud S. The New Introductory Lectures in Psychoanalysis. New York, NY: Penguin; 1973
20. Horowitz MJ. Stress Response Syndromes. 2nd ed. Northvale, NJ: Jason Aronson; 1986
21. Foa EB, Jaycox LH. Cognitive-behavioral treatment of posttraumatic stress disorder. In: Spiegel D, ed. Psychotherapeutic Frontiers: New Principles and Practices. Washington, DC: American Psychiatric Press; 1997
22. Bremner JD, Southwick S, Brett E, et al. Dissociation and posttraumatic stress disorder in Vietnam combat veterans. Am J Psychiatry 1992;149: 328–332
23. Marmar CR, Weiss DS, Schlenger WE, et al. Peritraumatic dissociation and posttraumatic stress in Vietnam theater veterans. Am J Psychiatry 1994; 151:902–907
24. Tichenor V, Marmar CR, Weiss DS, et al. The relationship of peritraumatic dissociation and posttraumatic stress: findings in female Vietnam theater veterans. J Consult Clin Psychol 1996;64:1054–1059
25. Foa EB, Riggs DS. Posttraumatic stress disorder in rape victims. In: Oldham J, Riba MB, Tasman A, eds. American Psychiatric Press Review of Psychiatry, vol 12. Washington, DC: American Psychiatric Press; 1993: 273–303
26. Amir N, Stafford J, Freshman MS, et al. Relationship between trauma narratives and symptoms in posttraumatic stress disorder. J Trauma Stress 1998;11:385–392
27. Tromp S, Koss MP, Figueredo AJ, et al. Are rape memories different? a comparison of rape, other unpleasant, and pleasant memories among employed women. J Trauma Stress 1995;8:607–627
28. Harber KD, Pennebaker JW. Overcoming traumatic memories. In: Christianson S, ed. The Handbook of Emotion and Memory: Research and Theory. Hillsdale, NJ: Lawrence Erlbaum Associates; 1992:359–387
29. Epstein S. The self-concept, the traumatic neurosis, and the structure of personality. In: Ozer D, Healy JM, Stewart AJ, eds. Perspectives in Personality, vol 3, pt A. London, England: Jessica Kingsley; 1991:63–98
30. Janoff-Bulman R. Shattered Assumptions: Towards a New Psychology of Trauma. New York, NY: Free Press; 1992
31. McCann IL, Pearlman LA. Psychological Trauma and the Adult Survivor: Theory, Therapy and Transformation. New York, NY: Brunner/Mazel; 1990
32. Keane TM, Fairbank JA, Caddell JM, et al. Implosive (flooding) therapy reduces symptoms of PTSD in Vietnam combat veterans. Behav Ther 1989;20:245–260
33. Kilpatrick DG, Veronen LJ, Resick PA. Psychological sequelae to rape: assessment and treatment strategies. In: Doleys DM, Meredity RL, Ciminero AR, eds. Behavioral Medicine: Assessment and Treatment Strategies. New York, NY: Plenum; 1982:473–497
34. Foa EB, Meadows EA. Psychosocial treatments for posttraumatic stress disorder: a critical review. Ann Rev Psychol 1997;48:449–480
35. Foa EB, Dancu CV, Hembree EA, et al. A comparison of exposure therapy, stress inoculation training, and their combination for reducing posttraumatic stress disorder in female assault victims. J Consult Clin Psychol 1999;67:194–200
36. Foa EB. Trauma and women: course, predictors, and treatment. J Clin Psychiatry 1997;58(suppl 9):25–28
37. Foa EB, Rothbaum BO, Riggs DS, et al. Treatment of posttraumatic stress disorder in rape victims: a comparison between cognitive-behavioral procedures and counseling. J Consult Clin Psychol 1991;59:715–723
38. Ellis A, Grieger R, eds. Handbook of Rational-Emotive Therapy. New York, NY: Springer; 1977
39. Beck AT. Cognitive Therapy and the Emotional Disorders. New York, NY: International Universities Press; 1976
40. Foa EB, Kozak MJ. Treatment of anxiety disorders: implications for psychopathology. In: Tuma AH, Masser JD, eds. The Practice of Psychotherapy. Washington, DC: American Psychiatric Press; 1985
41. Clark DM. A cognitive approach to panic. Behav Res Ther 1986;24: 461–470
42. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised. Washington, DC: American Psychiatric Association; 1987
43. Foa EB, Riggs DS, Dancu CV, et al. Reliability and validity of a brief instrument for assessing post-traumatic stress disorder. J Trauma Stress 1993; 6:459–473
44. Beck AT, Ward CH, Mendelsohn M, et al. An inventory for measuring depression. Arch Gen Psychiatry 1961;4:561–571
45. Beck AT, Steer RA, Garbin MG. Psychometric properties of the Beck Depression Inventory: twenty-five years of evaluation. Clin Psychol Rev 1988;8:77–100
46. Kendall PC, Hollon SD, Beck AT, et al. Issues and recommendations regarding use of the Beck Depression Inventory. Cogn Ther Res 1987;11: 289–299
47. Spielberger CD. Manual for the State-Trait Anxiety Inventory (Form Y) (Self-Evaluation Questionnaire). Palo Alto, Calif: Consulting Psychologists Press; 1983
48. Marks I, Lovell K, Noshirvani H, et al. Treatment of posttraumatic stress disorder by exposure and/or cognitive restructuring: a controlled study. Arch Gen Psychiatry 1998;55:317–325
49. Smucker MR, Dancu C, Foa EB, et al. Imagery rescripting: a new treatment for survivors of child sexual abuse suffering from posttraumatic stress. J Cogn Psychother 1995;9:3–17
50. Resick PA, Schnicke MK. Cognitive processing therapy for sexual assault victims. J Consult Clin Psychol 1992;60:748–756
51. Foa EB, Rothbaum BO. Treating the Trauma of Rape. New York, NY: Guilford Publications; 1998