

# Review and Critique of the New DSM-IV Diagnosis of Acute Stress Disorder

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**Objective:** A new diagnosis can greatly influence scientific research, access to resources, and treatment selection in clinical practice. The authors review the historical evolution, rationale, empirical foundation, and clinical utility to date of the recently introduced diagnosis of acute stress disorder. **Method:** The conceptual basis and relevant methods for identifying a psychiatric syndrome are reviewed with respect to acute stress disorder, including selection of criteria for core symptoms; considerations of sensitivity and specificity of a syndrome definition; longitudinal course; and distinctions between normative and pathological phenomena. Particular attention is devoted to two major issues: the implications of the core feature requirement of three of five dissociative symptoms, and the question of whether there should be two separate diagnoses (acute stress disorder and posttraumatic stress disorder [PTSD]) describing posttraumatic syndromes. The widely divergent approaches in DSM-IV and ICD-10 are also reviewed. **Results:** The diagnosis of acute stress disorder does not appear to achieve the important objective of providing adequate clinical coverage for individuals with acute posttraumatic symptoms. The validity and utility of requiring peritraumatic dissociative symptoms as a core feature are questionable, as is the separation of essentially continuous clinical phenomena into two disorders with different criteria sets (acute stress disorder and PTSD) based on persistence of symptoms for 30 or more days. **Conclusions:** Longitudinal studies using acute stress disorder criteria, as well as broader considerations of the clinical and scientific functions that posttraumatic diagnoses should serve, suggest a need to reevaluate the current DSM-IV approach to posttraumatic syndromes.

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“Just as Copernicus did, so all good researchers—physicians and observers and thinkers do—must do: turn the data and the method about, in order to see whether it wouldn’t fit better that way.”

—Novalis (Friedrich von Hardenberg, 1772–1801)  
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Because the etiology of most mental disorders is largely unknown, psychiatric research remains particularly dependent on the principles and process of syndrome identification. Identification of syndrome criteria with acceptable reliability can then facilitate investigation of syndrome validity with methodologies such as factor and cluster analysis, laboratory study, studies of comorbidity and distinction from other disorders, follow-up, family studies, and treatment response (1, 2). Official recognition of a clinical syndrome can also greatly stimulate scientific interest, as illustrated by the surge of research in posttraumatic stress disorder (PTSD) since its recognition in DSM-III in 1980. Finally, diagnoses serve the crucial clinical objectives of identifying individuals in need of treatment and guiding treatment selection.

Since manifestations of a given psychological disorder generally are not uniform and often are distributed in a spectrum of severity, seemingly minor variations in syndrome criteria definition can alter the detected prevalence of the diagnosis, with substantial clinical and scientific consequences (3). Given a particular hypothetical rate of disorder in a population, increasing the sensitivity of diagnostic criteria enhances the rate of true positive cases detected (diagnosing those who have the true disorder), at the risk of including more false positive cases (diagnosing those who do not have the true disorder). Increasing the specificity of diagnostic criteria enhances the rate of true negative cases detected (not diagnosing those who do not have the true disorder), at the risk of failing to identify more true positive cases (failing to diagnose individuals with the true disorder). The decision as to whether to define a disorder broadly or narrowly usually hinges on the relative consequences of doing so and has been widely debated in psychiatry with respect to psychiatric diagnosis. For example, which is worse: failing to identify some persons with PTSD who could benefit from treatment, or mistakenly diagnosing some persons who are actually experiencing a normative reaction to a traumatic event? The problem is compounded by the absence of a "gold standard" to identify true disorder.

Substantial clinical evidence was required for a new disorder to be recognized in DSM-IV (4, 5). After extensive review of the available empirical literature, a second trauma-related diagnosis, acute stress disorder, was included to describe responses to trauma occurring within the first 30 days of the event. This addressed an important limitation in DSM-III-R, since the definition of PTSD did not allow the diagnosis to be made until symptoms had been present for at least 30 days. Acute stress disorder identifies individuals with symptoms similar to those of PTSD (i.e., intrusive, avoidant, numbing, and increased arousal symptoms) but does not specify the number of symptoms required in the avoidance and increased arousal categories (unlike the definition of PTSD). Furthermore, acute stress disorder requires that at least three of the following five dissociative symptoms were experienced during or after the traumatic event: numbing, detachment, or absence of emotional responsiveness; a reduction in awareness of surroundings; derealization; depersonalization; or dissociative amnesia.

A number of complex issues are raised by this new diagnosis. This article reviews the historical development, empirical foundation, rationale, and clinical implications of acute stress disorder as defined in DSM-IV. The rationale and implications of requiring three of five dissociative symptoms in order to diagnose acute stress disorder in DSM-IV are a central focus of this review and critique. Furthermore, both conceptual and practical problems are raised by the existence of two posttraumatic diagnoses that have different criteria but that appear to describe largely continuous clinical phenomena. Finally, the related ICD-10 diagnoses of acute stress reaction and PTSD are discussed in comparison.

## HISTORICAL DEVELOPMENT OF DIAGNOSES OF TRAUMATIC STRESS SYNDROMES

There have been several important points of contention during the evolution of the diagnoses of PTSD and acute stress disorder. Some concern fundamental issues in nosological development, and some are specific to trauma research. These include the following: 1) the item content of the syndromes of acute stress disorder and PTSD, including the relative importance of dissociative symptoms; 2) the way to address longitudinal course; and 3) the importance of distinguishing between normative and pathological responses to traumatic events in order to define a "boundary" for the disorder.

The diagnosis of PTSD was first officially recognized in DSM-III in 1980 and was based on an extensive clinical literature documenting characteristic responses after a broad range of traumatic events (6). Before this, DSM-I included a diagnosis of gross stress reaction, which presented no criteria and described a reaction in a "normal personality" to severe trauma, which supposedly resolved rapidly in most cases. DSM-II contained no posttraumatic diagnostic category. The diagnosis of PTSD, created in DSM-III, required the presence of a stressor that "would evoke significant symptoms of distress in almost everyone" (criterion A). Criteria included the three symptom clusters of re-experiencing the trauma (B, one required); numbing of general responsiveness (C, one required); and increased arousal, cognitive impairment, avoidance of reminders of the trauma, reexperiencing symptoms in the presence of reminders of the trauma, and survivor guilt (D, two required). Subtypes of the diagnosis were acute (onset within 6 months and symptom duration of less than 6 months), chronic (symptom duration of more than 6 months), and delayed (onset at least 6 months after the trauma). The diagnosis could be made immediately after a traumatic event if the full syndrome were present.

The most significant revision in DSM-III-R was the addition of a minimum duration criterion requiring symptoms to have been present for at least 30 days. This constituted a response to new findings that symptoms and distress were common after a severe trauma and therefore might represent a normative process (7, 8). Since the boundary between normative and pathological responses to trauma was unclear, it was felt that trauma-related diagnoses should be narrowly defined to include only those individuals who, in essence, had not begun to show clinically significant spontaneous recovery after 30 days.

The unfortunate clinical consequence of the minimum duration criterion, however, was that the only stress-related diagnosis available in the first month after a traumatic event was the nonspecific diagnosis of adjustment disorder. Thus, survivors of severe trauma, experiencing the full PTSD syndrome, would initially receive the same diagnosis as individuals experiencing

nonspecific and relatively mild reactions to common life stressors.

During the development of DSM-IV, this deficit was recognized and addressed. Longitudinal studies showed, in fact, that a substantial proportion of individuals met criteria for PTSD in the first month after severe trauma (7, 9) and would have received the diagnosis of acute PTSD according to DSM-III. Treatment and research were curtailed by the unavailability of a diagnosis in DSM-III-R. However, the possibility of returning to the DSM-III definition—i.e., dropping the time duration requirement—was rejected for the same reasons it was instituted, out of concern for assigning a diagnosis of a mental disorder to individuals who would eventually recover spontaneously (10).

### ACUTE STRESS DISORDER

Several different diagnostic approaches were considered. The final decision was to select criteria for acute stress disorder that would theoretically identify individuals likely to meet criteria for PTSD 1 month later (11). The objective was to distinguish between normative and pathological acute stress responses by linking acute stress disorder to predictors of a relatively poor prognosis.

Four symptom clusters were created: reexperiencing, avoidance, increased arousal, and dissociative symptoms. All but the last are essentially identical to those of PTSD but are less precise in two of three criteria categories. PTSD requires the presence of at least one intrusive symptom, three of seven specific avoidance symptoms, and two of five specific arousal symptoms, whereas acute stress disorder requires one intrusive symptom and “marked avoidance” and “marked symptoms of anxiety or increased arousal” and provides lists of potential symptoms but no required minimum number. Duration of symptoms was stipulated as between 2 days and 4 weeks, with onset within 4 weeks of the traumatic event. Thus, if symptoms persist beyond 4 weeks, the diagnosis is changed to PTSD. The greatest difference between acute stress disorder and PTSD is the additional core symptom requirement of three of five dissociative symptoms during or after the traumatic event.

Other options proposed included creation of a new V code called uncomplicated posttraumatic stress reaction, in order to capture a normal reaction, similar to the V code condition of bereavement. The use of course of illness descriptors such as “acute” (onset within 1 year), “delayed” (onset after 1 year), “recurrent” (for a resurgence of symptoms after a period of recovery), and “residual” (for subthreshold symptoms in individuals who previously met criteria for the full syndrome) was discussed (4).

The possibility of reclassifying PTSD as a dissociative disorder was also considered (10). The empirical evidence linking PTSD to anxiety disorders was viewed in the end as more convincing, but this debate may

have played a role in promoting an increased emphasis on dissociative symptoms. The DSM-IV subcommittee on PTSD, in fact, recommended that syndromes clearly following a stressor be grouped together, but this recommendation was not implemented (10).

### PERITRAUMATIC DISSOCIATION AND OTHER VULNERABILITY FACTORS

Several studies have found that a dissociative response during or immediately after a traumatic experience predicted the subsequent development of PTSD (for review, see Cardena et al. [12]); these studies used both retrospective (13–18) and prospective (19–21) methodologies. This important finding generated renewed interest in the role of dissociation in posttraumatic syndromes and led to the inclusion of peritraumatic dissociation as a core feature of acute stress disorder.

Several other factors also increase the risk of development of PTSD. For example, overall severity of acute PTSD symptoms also predicts development of chronic disorder (7, 21–24). Preexisting vulnerability factors increase the risk of development of PTSD after trauma; these include neuroticism (22), personality disorder (25), history of psychiatric illness (9, 26), history of trauma or stress (27–29), genetic liability (30), and family history of psychological disorder (22, 30–32).

Thus, the risk of experiencing both peritraumatic dissociation and subsequent PTSD may be elevated by common vulnerability factors. A study of rescue workers responding to a freeway collapse after an earthquake, in fact, found that particular personality traits and coping styles increased the risk of both peritraumatic dissociation and subsequent PTSD (33). In a prospective study of female rape and criminal assault victims, dissociative symptoms in the immediate post-trauma period were correlated with prior history of sexual abuse (34). A study that retrospectively diagnosed acute stress disorder in a sample of motor vehicle accident victims with PTSD 1–4 months after the trauma similarly found acute stress disorder to be associated with higher rates of premorbid mood disorders other than major depression and premorbid axis I and axis II disorders (35). Dissociative symptoms correlate with high levels of anxiety and PTSD symptoms in all studies.

In summary, the current definition of acute stress disorder singles out one of several risk factors for PTSD—peritraumatic dissociation—and elevates it to the status of a core symptom of a new disorder. Furthermore, common preexisting vulnerability factors appear to increase the risk of both peritraumatic dissociation and subsequent PTSD, perhaps in part by increasing the intensity of the response to trauma (36). Most important, however, the current definition of acute stress disorder excludes from receiving the diagnosis those individuals who do not have prominent dissociative symptoms (criterion B) even if symptom

criteria for PTSD are met within a month of the trauma.

One of us (R.S.) observed the following illustrative case example. A 30-year-old married woman presented to the psychiatric emergency room 9 days after having been badly beaten by her husband. Since the beating she had felt depressed, tearful, sleepless, numb, and hopeless and had thoughts of killing herself. She had repeated nightmares of being beaten and dragged by her husband. During the day she was hypervigilant and anxious and experienced intrusive thoughts of the beating. The resident proposed a diagnosis of PTSD and was surprised when the attending psychiatrist explained that the diagnosis was not applicable because the duration was less than 4 weeks, nor was it acute stress disorder because of the absence of multiple dissociative symptoms. In fact, the only available diagnosis, according to DSM-IV, was adjustment disorder.

### ACUTE STRESS IN THE ABSENCE OF SEVERE DISSOCIATIVE SYMPTOMS

The studies that established peritraumatic dissociation as a predictor of PTSD (cited earlier) did not address the question of taxonomic specificity. A few later studies using acute stress disorder criteria can be examined to address the issue of whether peritraumatic dissociation can be considered a *sine qua non* or core feature of an acute stress disorder. Three prospective studies (37–39) and two retrospective studies (35, 40) are reviewed to determine the proportion of acute trauma survivors who had significant distress or impairment or who will later develop chronic disorder, who were unrecognized by the current DSM-IV definition. In addition, three prospective studies (21, 34, 41) are reviewed that used dimensional measures (but not DSM-IV criteria) to measure dissociative symptoms after acute trauma. Generalization across studies is limited by the use of different measures and methodology.

Jaycox et al. (37) assessed 43 female assault victims within 1 month of the trauma. All subjects met criteria for PTSD (with the duration criterion suspended), whereas only 43% met criteria for acute stress disorder. This suggests that acute stress disorder, as currently defined, fails to identify a large proportion of patients with significant clinical distress or impairment after serious trauma. Subjects with acute stress disorder reported significantly more posttraumatic symptoms. The authors suggested that acute stress disorder may therefore simply reflect greater severity of PTSD symptoms, rather than a distinct diagnostic entity. In a second sample of patients with chronic PTSD (mean time since assault was 3.7 years), only 58% retrospectively met criteria for acute stress disorder, and the diagnosis of acute stress disorder did not correlate with later symptom severity.

A recent study (38) of adults who sustained mild traumatic brain injury after a motor vehicle accident consecutively assessed subjects within 1 month by

structured interview and again 6 months after the trauma. Within the first month, 13.8% (N=11) met criteria for acute stress disorder, and after 6 months, 23.8% (N=15) met criteria for PTSD. Among those subjects with PTSD at 6 months, nine (60%) had met criteria for acute stress disorder, and six (40%) had not. Thus, in that study, most subjects with acute stress disorder (nine of 11) did develop chronic PTSD, but 40% of subjects who developed chronic PTSD were not identified by the acute stress disorder diagnosis.

A recent prospective study assessed acute stress symptoms (within 8 days of the trauma) and subsequent PTSD symptoms among a treatment-seeking subset of witnesses of a mass shooting (39). Symptoms were assessed with self-report forms only. The main finding was that meeting criteria for acute stress disorder predicted PTSD symptom dimensional scores, and acute stress disorder symptoms were significantly correlated with later PTSD symptoms ( $r=0.44$ ,  $p\leq 0.01$ ). Other potential predictors (such as acute PTSD symptoms or comorbid or prior psychiatric disorder) were not assessed, however. As the authors noted, in the absence of clinical interviews, it was not known whether subjects actually met diagnostic criteria for either acute stress disorder or PTSD (including the distress/impairment criterion).

Barton et al. (35) made retrospective diagnoses of acute stress disorder (since the criteria were not available at the initial data collection) in a subset of motor vehicle accident victims with PTSD. The diagnosis was based on detailed accounts of immediate responses to the accident and review with the interviewing clinician. In the original sample of non-treatment-seeking motor vehicle accident victims (N=158), 39% (N=62) had been diagnosed with PTSD on the basis of DSM-III-R criteria (42). In fact, only a minority (N=14 [23%] of 62) of the PTSD subjects retrospectively met criteria for acute stress disorder (i.e., reported at least three or more dissociative symptoms from criterion B). Individuals who met criteria for acute stress disorder were more likely to show preexisting psychopathology, as well as greater initial severity on measures of depression, state and trait anxiety, PTSD symptoms, and level of functioning. However, at 6-month follow-up, there were no differences in symptom severity or rate of chronic PTSD between the two groups, again suggesting that meeting criteria for acute stress disorder did not predict poorer outcome. The authors noted the limitation of retrospective assessments.

A second study retrospectively assessed PTSD and acute stress disorder symptoms in the Swedish survivors (N=53) of the capsizing of the Swedish car ferry *Estonia* in 1994 (40). Of more than 900 passengers, only 135 survived after several hours' struggle in near-freezing water. The authors reported a PTSD rate of 64.3% 3 months after the accident, but only 40.5% (N=17 of 42) of the subjects reported three or more DSM-IV dissociative symptoms. The authors stated that "the more dissociative symptoms the person had, the higher was the risk of post-traumatic symptoms";

however, the analyses were not reported. Since the report also failed to present data on the proportion of subjects with PTSD and fewer than three dissociative symptoms, it is of limited use in assessing the adequacy of the acute stress disorder diagnosis.

Dancu et al. (34) prospectively examined the relationship between acute dissociative symptoms (assessed within 2 weeks of trauma) and clinical outcome 3 months after trauma in three groups of women: rape victims (N=74), nonsexual assault victims (N=84), and comparison subjects (N=46). Fifty percent of rape victims and 21% of nonsexual assault victims met the criteria for PTSD 3 months after trauma. There was considerable heterogeneity of dissociative tendencies (range of scores on the Dissociative Experiences Scale [43] was 0–88.6), and the predictive power of acute dissociative symptoms varied in the two trauma groups. Among rape victims, a regression analysis of PTSD symptoms at 3 months revealed that the combination of initial symptom scores with Dissociative Experiences Scale scores accounted for only 12% of the variance, and no single variable reached significance. Among nonsexual assault victims, the same analysis showed a stronger contribution for intrusion scores, and Dissociative Experiences Scale scores approached significance as a predictor ( $p < 0.06$ ). Overall, the study does not support an emphasis on peritraumatic dissociation as the most important predictor of subsequent PTSD, at least when assessed with the Dissociative Experiences Scale.

Shalev et al. (21) assessed PTSD and dissociative symptoms within 6 days of trauma in hospitalized individuals who had been physically injured (N=51). Fifty-one percent of subjects (N=26 of 51) endorsed at least one symptom on the Peritraumatic Dissociation Experiences Questionnaire (16). At 6-month follow-up, 25.5% (N=13 of 51) met criteria for PTSD. Those who later developed PTSD had more severe initial symptoms of peritraumatic dissociation, intrusive re-experiencing, depression, and state anxiety. Peritraumatic dissociation scores were significantly correlated with initial severity of anxiety, depressive, and PTSD symptoms, again suggesting an important link between dissociative symptoms and general psychopathology.

A logistic regression analysis was conducted to identify predictors of PTSD status and included the following variables: age, gender, education, event severity, immediate response, peritraumatic dissociation, PTSD symptoms (Impact of Event Scale), state-trait anxiety, and depressive symptoms. Peritraumatic dissociation was the only significant predictor of PTSD status in this analysis. The sensitivity and specificity of the model, however, are particularly relevant to the question of whether dissociation should be viewed as a core symptom of acute stress reactions. The model possessed sensitivity of only 30.8%, with a specificity of 94.7%. At 6 months, only nine (35%) of 26 subjects who endorsed at least one peritraumatic dissociative symptom had developed PTSD, and four (16%) of 26 subjects without any dissociative symptoms still devel-

oped PTSD. These two groups showed nearly identical severity of all other PTSD symptoms at week 1 assessment. Thus, it is likely that more than four (31%) of 13 subjects who later developed PTSD would not have received the diagnosis of acute stress disorder in the first month after trauma.

Shalev et al. (41) reported a second, larger prospective study (N=239) assessing acute dissociative symptoms, PTSD symptoms, and general distress within 1 week after a range of traumatic events, with 1- and 4-month follow-up assessments. Of the subjects who completed at least the 1-week and 4-month assessments (N=207), about 30% met criteria for PTSD after 1 month, and 17% met criteria after 4 months. In that study, acute dissociative symptoms (again measured by the Peritraumatic Dissociation Experiences Questionnaire) were no better at predicting 4-month outcome than were measures of PTSD and general symptoms. In addition, assessments at 1 week were as predictive as assessments at 1 month, calling into question the DSM-IV requirement of waiting 1 month before diagnosing PTSD.

In summary, prospective and retrospective studies to date suggest that a substantial proportion of individuals with characteristic PTSD symptoms in the first month after trauma may not have experienced significant peritraumatic dissociation and thus would not meet criteria for acute stress disorder. Furthermore, acute dissociative symptoms do not emerge as a *sine qua non* of severe acute stress reactions. Finally, the more parsimonious view that the symptoms of acute stress disorder simply reflect greater severity of PTSD, rather than a separate diagnostic entity, is supported by the available data.

## DISSOCIATION AND POSTTRAUMATIC SYNDROMES

Although the above review suggests that dissociation is not a core feature of acute PTSD, higher rates of dissociative symptoms have been found in subjects with PTSD than in normal control subjects, subjects with histories of trauma but no PTSD, and subjects with psychiatric disorder but no PTSD (44–46). Dissociative symptoms have been viewed as a response to overwhelming fear and anxiety in a predisposed individual and are associated with general psychopathology. One study of the Dissociative Experiences Scale found that 61% of the variance in the scale's scores was predicted by measures of phobic anxiety, hostility, somatization, imaginative absorption, and irrational thinking (47). However, a number of studies have shown that mild dissociative experiences, such as the capacity for imaginative involvement/absorption, appear normally distributed (48, 49). Thus, it is likely that this unfortunately vague term is used to describe a broad range of phenomena, and clarification of phenomenological models of dissociation may be important in future studies of the psychobiology and clinical treatment of types of dissociation (50).

Whether dissociative symptoms are viewed as central or secondary phenomena has important clinical implications. For example, dissociative symptoms secondary to severe anxiety (as in panic disorder and PTSD) may improve with treatment of the primary disorder (51).

#### **USING SYMPTOM DURATION TO DISTINGUISH BETWEEN DISORDERS**

According to DSM-IV, an individual's diagnosis changes from acute stress disorder to acute PTSD if symptoms persist for 1 month. After 3 months of symptoms, the diagnosis changes again to chronic PTSD. This represents an effort to distinguish between three different clinical situations: a severe acute response to trauma, a time-limited disorder that eventually resolves after weeks to months, and a chronic disorder that tends to persist for months to years (52–54). However, the time point after which a chronic disorder can be identified, and the proportion of subjects who develop chronic disorder, varies considerably across studies and with the severity of the trauma. Among female rape victims, the proportion of subjects meeting criteria for PTSD diminished over time from 94% 1 week after trauma, to 65% 1 month after trauma, to 47% at 3 months, with no further significant proportional change between 3 and 9 months (7). Among 84 prospectively studied criminal assault victims, rates of PTSD declined more rapidly, from 71.1% of women and 50% of men within the first month, to 21.1% of the women and none of the men after 4 months (8). In the National Comorbidity Survey (which presents the largest data set and longest period of follow-up in the literature, albeit with retrospective assessments), the rate of PTSD declined at a relatively constant rate over 12 months, with a more gradual decline over 6 years (55).

Thus, the distinction between a time-limited form of PTSD that eventually remits and a chronic form of PTSD is supported, although studies vary as to the point at which chronic PTSD can be identified. However, research to date suggests that it makes little clinical or conceptual sense to regard the first month of symptoms as a separate disorder.

#### **SPECIAL CONSIDERATIONS FOR POSTTRAUMATIC SYNDROMES**

DSM-IV defines mental disorder as “a clinically significant behavioral or psychological syndrome or pattern that occurs in an individual and that is associated with present distress (e.g., a painful symptom) or disability (i.e., impairment in one or more important areas of functioning) or with a significantly increased risk of suffering death, pain, disability, or an important loss of freedom.” It also must not be merely an “expectable” response and must represent “dysfunction.”

Klein (56) proposed that disease implies a dysfunction of one or more evolved (naturally selected) functions within an organism. Following this line of reasoning, the DSM definition of acute stress disorder attempts to make the important distinction between a normal reaction to trauma and a symptomatic state that is more likely to involve underlying dysfunction or dysregulation.

The viewpoint that an acute stress syndrome should not include symptoms that are relatively common (and therefore “expectable”) after severe trauma, however, conflates nosological and statistical concepts. Physical injury after a serious accident (e.g., falling from a height of 20 feet) may indeed be common. By analogy, the fact that the risk of developing PTSD increases with the severity and duration of trauma suggests that there are likely limitations to the evolved normal capacity to adapt to psychological trauma. The conceptual maxim that a disorder should not be expectable is therefore called into question in the case of severe traumatic events.

The argument that many trauma survivors will eventually show gradual, spontaneous recovery and therefore should not be given a diagnosis is similarly inconsistent with nosologic principles. The natural history of many mental disorders and medical diseases is spontaneous recovery, and interventions are often directed at shortening the natural course of illness to alleviate distress and prevent further complications. That is, a disorder involving an underlying dysfunction may still resolve spontaneously over time.

Finally, the studies reviewed above suggest that initial symptom response after a traumatic event (including peritraumatic dissociative symptoms) is not a strong enough predictor to be useful for diagnostic purposes—i.e., to identify all individuals who will later develop chronic PTSD. Although individuals who develop PTSD tend to have a more severe initial response to trauma, there is considerable overlap with the group of those who eventually recover. In other words, attempts to minimize false positive cases on the basis of initial symptom profile alone appear to result in a clinically unacceptable failure to identify individuals who may be in need of clinical intervention after a traumatic experience.

#### **COMPARISON OF ICD-10 AND DSM-IV**

In ICD-10, the category “Reactions to severe stress, and adjustment disorders” groups the diagnoses with the common feature of being clearly precipitated by severe stressors. These are acute stress reaction, PTSD, and the adjustment disorders, with a residual category of “unspecified.”

The ICD-10 definition of PTSD most resembles the first DSM-III definition in that it does not require a minimum duration of symptoms. Acute stress reaction, by contrast, requires immediate onset of symptoms (within 1 hour) and relatively rapid diminution of

symptoms after the stressor is relieved (within 8 hours). If the stressor is ongoing, diminution of symptoms must begin after not more than 48 hours. Thus, this syndrome essentially describes an immediate, transient reaction to a traumatic experience.

In ICD-10, acute stress reaction can be diagnosed following "an exceptional mental or physical stressor" and provides three levels of severity with different criteria sets for each and a complex method for assigning the diagnosis. Thus, it captures a broad range of peritraumatic responses with varying degrees of dissociation, anxiety, affective instability, and confusion.

The primary strength of the ICD-10 approach is that it may provide greater diagnostic coverage for a broader range of posttraumatic responses associated with impairment and distress. This is accomplished through inclusion of both nonspecific acute responses to trauma (in the diagnoses of acute stress reaction and adjustment disorder), and the specific, well-validated constellation of symptoms included in PTSD. It differs from the DSM approach in that there is probably substantial overlap with normal emotional reactions, since the milder form of acute stress reaction describes an emotional upheaval that resolves relatively rapidly after trauma. Further research is needed to clarify this important distinction in the nosology.

### **SOCIAL IMPLICATIONS OF A TRAUMA DIAGNOSIS**

The psychological and societal implications of receiving a psychiatric diagnosis have often been considered in the DSM development process, and played a role in the decision to define acute stress disorder strictly. Since the concept of illness is subject to value-laden considerations, a psychiatric diagnosis historically has carried multiple meanings with both positive and pejorative implications (57). Critics of DSM have emphasized that individuals and society may perceive a diagnosis as stigmatizing. In the United States, there has been concern that broad definitions of posttraumatic diagnoses may be exploited in the courts, and this issue was discussed at length during the deliberation process. Alternatively, a diagnosis can often be used to validate a patient's symptoms and disability and legitimately entitle an individual to the sick role. Diagnoses are used to guide treatment selection, as well as to justify access to resources. In fact, patient advocacy groups have used a biological model of mental illness to destigmatize psychiatric disorder and encourage fair and compassionate treatment. In clinical practice, providing education about a disorder can help to counter a patient's distorted view of the self as weak or defective.

Since a psychiatric disorder evokes both positive and negative connotations, it seems unwise to allow such considerations to distort the scientific process of syndrome development. Instead, stigmatization and distortion can be addressed through individual and societal education. It should be noted that broadening the

definition of acute stress responses should not have important implications in the courts, since compensation is usually sought for severe, protracted disabilities.

### **CONCLUSIONS**

Prospective findings to date do not support the current DSM-IV requirement of three of five prominent dissociative symptoms as a core feature of acute stress disorder, since a significant proportion of individuals with an identifiable trauma-related syndrome appear to be thereby excluded from receiving a diagnosis. Under the present system, an individual who did not experience significant peritraumatic dissociation might meet criteria for PTSD after 1 month but never have met criteria for acute stress disorder. Such an inconsistency may produce clinical confusion in applying these diagnoses. Most important, the current division of posttraumatic symptoms into two separate disorders is called into question by the available data.

Given that the time course of spontaneous recovery after traumatic experiences varies considerably, the requirement of 1 month of symptoms before the diagnosis of PTSD can be made does not appear to distinguish adequately between normative and pathological posttraumatic responses and creates an illusion of pseudo-exactness regarding longitudinal course.

A more parsimonious approach that serves both clinical and scientific objectives would allow PTSD to be diagnosed at any point in time after trauma if symptom criteria are met. This would essentially represent a return to the DSM-III format and would be consistent with ICD-10. Acute and chronic subtypes might still be retained, and the duration cutoff point reviewed on the basis of further prospective data. Further study is needed to clarify the distinction between a normative response to trauma and a disorder involving dysfunction. The validity of a chronic subtype of PTSD has been established by prospective longitudinal study, the pathophysiology of which may also prove distinct from acute PTSD. The alternative of retaining the diagnosis of acute stress disorder, but broadening the definition, would also accomplish the same purpose but lacks parsimony and splits a continuous dimension arbitrarily into separate syndromes.

Further study is needed as to how dissociative symptoms might be included in future definitions of PTSD. Since peritraumatic dissociation is one of several predictors of chronic PTSD, the presence of dissociative symptoms might be recognized as an associated, but not required, feature of acute PTSD.

A recent study found significant differences in heart rate, skin conductance, and nonspecific movement between rape victims with high peritraumatic dissociation scores and those with low scores (36). High-dissociation subjects were also more likely to meet criteria for PTSD and had more severe PTSD symptoms. Thus, prominent dissociation may be associated with a physiologic subtype of severe PTSD, and recognition in the

diagnostic criteria would facilitate further research. Future research may reveal physiologic factors that assist in distinguishing between normative and pathological responses to trauma.

After acute trauma, neither dissociative symptoms nor severity of PTSD symptoms has a high sensitivity for detecting individuals who will develop chronic PTSD, since there is significant overlap with the symptoms of individuals who spontaneously recover. It is possible that initial symptom presentation is of limited use in predicting longitudinal course in the case of posttraumatic syndromes, as is the case, for example, with schizophrenia.

On balance, it may be argued an approach similar to that used in ICD-10 better addresses the above concerns with a hierarchical, algorithmic organization of the three diagnoses of PTSD, acute stress reaction, and adjustment disorder. The diagnoses are all mutually exclusive and differentiated on the basis of clinical phenomenology.

Many important clinical questions remain concerning the treatment of acute trauma survivors. An intervention may 1) shorten the course of the normal posttraumatic response, 2) reduce PTSD symptoms, 3) prevent the development of chronic PTSD, 4) help to restore functioning, and 5) prevent functional deterioration. Since research is often guided by diagnosis, the way in which posttraumatic syndromes are defined in the future will likely be greatly influential in all aspects of trauma studies.

## REFERENCES

- Robins E, Guze SB: Establishment of diagnostic validity in psychiatric illness: its application to schizophrenia. *Am J Psychiatry* 1970; 126:983-987
- Wakefield JC: Disorder as harmful dysfunction: a conceptual critique of DSM-III-R's definition of mental disorder. *Psychol Rev* 1992; 99:232-247
- Regier DA, Kaelber CT, Rae DS, Farmer ME, Knauper B, Kessler RC, Norquist GS: Limitations of diagnostic criteria and assessment instruments for mental disorders. *Arch Gen Psychiatry* 1998; 55:109-115
- Blank AS Jr: Suggested recommendations for DSM-IV on course and subtypes, in *Posttraumatic Stress Disorder: DSM-IV and Beyond*. Edited by Davidson JRT, Foa EB. Washington, DC, American Psychiatric Press, 1993, pp 237-239
- Pincus HA, Frances A, Davis WW, First MB, Widiger TA: DSM-IV and new diagnostic categories: holding the line on proliferation. *Am J Psychiatry* 1992; 149:112-117
- Kinzie JD, Goetz RR: A century of controversy surrounding posttraumatic stress-spectrum syndromes: the impact on DSM-III and DSM-IV. *J Trauma Stress* 1996; 9:159-179
- Rothbaum BO, Foa EB, Riggs DS, Murdock T, Walsh W: A prospective examination of post-traumatic stress disorder in rape victims. *J Trauma Stress* 1992; 5:455-475
- 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
- North CS, Smith EM, Spitznagel EL: Posttraumatic stress disorder in survivors of a mass shooting. *Am J Psychiatry* 1994; 151:82-88
- Davidson JRT, Foa EB, Blank AS, Brett EA, Fairbank J, Green BL, Herman JL, Keane TM, Kilpatrick DL, March JS, McNally RJ, Pitman RK, Resnick HS, Rothbaum BO: Posttraumatic stress disorder, in *DSM-IV Sourcebook*, vol 2. Edited by Widiger TA, Frances AJ, Pincus HA, Ross R, First MB, Davis WW. Washington, DC, American Psychiatric Press, 1996, pp 577-606
- Rothbaum BO, Foa EB: Subtypes of posttraumatic stress disorder and duration of symptoms, in *Posttraumatic Stress Disorder: DSM-IV and Beyond*. Edited by Davidson JRT, Foa EB. Washington, DC, American Psychiatric Press, 1993, pp 23-35
- Cardeña E, Lewis-Fernández R, Bear D, Pakianathan I, Spiegel D: Dissociative disorders, in *DSM-IV Sourcebook*, vol 2. Edited by Widiger TA, Frances AJ, Pincus HA, Ross R, First MB, Davis WW. Washington, DC, American Psychiatric Press, 1996, pp 973-1005
- Wilkinson CB: Aftermath of a disaster: the collapse of the Hyatt Regency Hotel skywalks. *Am J Psychiatry* 1983; 140:1134-1139
- Carlson EB, Rosser-Hogan R: Trauma experiences, posttraumatic stress, dissociation, and depression in Cambodian refugees. *Am J Psychiatry* 1991; 148:1548-1551
- Bremner JD, Southwick S, Brett E, Fontana A, Rosenheck R, Charney DS: Dissociation and posttraumatic stress disorder in Vietnam combat veterans. *Am J Psychiatry* 1992; 149:328-332
- Marmar CR, Weiss DS, Schlenger WE, Fairbank JA, Jordan BK, Kulka RA, Hough RL: Peritraumatic dissociation and posttraumatic stress in male Vietnam theater veterans. *Am J Psychiatry* 1994; 151:902-907
- Tichenor V, Marmar CR, Weiss DS, Metzler TJ, Ronfeldt HM: The relationship of peritraumatic dissociation and posttraumatic stress: findings in female Vietnam theater veterans. *J Consult Clin Psychol* 1996; 64:1054-1059
- Weiss DS, Marmar CR, Metzler TJ, Ronfeldt HM: Predicting symptomatic distress in emergency services personnel. *J Consult Clin Psychol* 1995; 63:361-368
- Koopman C, Classen C, Spiegel D: Predictors of posttraumatic stress symptoms among survivors of the Oakland/Berkeley, Calif, firestorm. *Am J Psychiatry* 1994; 151:888-894
- Holen A: The North Sea oil rig disaster, in *International Handbook of Traumatic Stress Syndromes*. Edited by Wilson JP, Raphael B. New York, Plenum, 1993, pp 471-478
- Shalev AY, Peri T, Canetti L, Schreiber S: Predictors of PTSD in injured trauma survivors: a prospective study. *Am J Psychiatry* 1996; 153:219-225
- McFarlane AC: The aetiology of post-traumatic morbidity: predisposing, precipitating, and perpetuating factors. *Br J Psychiatry* 1989; 154:221-228
- Perry S, Difede J, Musngi G, Frances AJ, Jacobsberg L: Predictors of posttraumatic stress disorder after burn injury. *Am J Psychiatry* 1992; 149:931-935
- McFarlane AC: Avoidance and intrusion in posttraumatic stress disorder. *J Nerv Ment Dis* 1992; 180:439-445
- Schnurr PP, Friedman MJ, Rosenberg SD: Premilitary MMPI scores as predictors of combat-related PTSD symptoms. *Am J Psychiatry* 1993; 150:479-483
- Helzer JE, Robin LN, McEvoy L: Post-traumatic stress disorder in the general population: findings from the Epidemiological Catchment Area survey. *N Engl J Med* 1987; 317:1630-1634
- Bremner JD, Southwick SM, Johnson DR, Yehuda R, Charney DS: Childhood physical abuse and combat-related posttraumatic stress disorder in Vietnam veterans. *Am J Psychiatry* 1993; 150:235-239
- Zaidi LY, Foy DW: Childhood abuse and combat-related PTSD. *J Trauma Stress* 1994; 4:325-343
- Resnick HS, Kilpatrick DG, Best CL, Kramer TL: Vulnerability-stress factors in development of posttraumatic stress disorder. *J Nerv Ment Dis* 1992; 180:424-430
- True WR, Rice J, Eisen SA, Heath AC, Goldberg J, Lyons MJ, Nowak J: A twin study of genetic and environmental contributions to liability for posttraumatic stress symptoms. *Arch Gen Psychiatry* 1993; 50:257-264

31. Breslau N, Davis GC, Andreski P, Peterson E: Traumatic events and post-traumatic stress disorder in an urban population of young adults. *Arch Gen Psychiatry* 1991; 48:216-220
32. Davidson J, Swartz M, Storck M, Krishnan RR, Hammett E: A diagnostic and family study of posttraumatic stress disorder. *Am J Psychiatry* 1985; 142:90-93
33. Marmar CR, Weiss DS, Metzler TJ, Delucchi K: Characteristics of emergency services personnel related to peritraumatic dissociation during critical incident exposure. *Am J Psychiatry* 1996; 153 (July Festschrift suppl):94-102
34. Dancu CV, Riggs D, Hearst-Ikeda D, Shoyer BG, Foa EB: Dissociative experiences and posttraumatic stress disorder among female victims of criminal assault and rape. *J Trauma Stress* 1996; 9:253-267
35. Barton KA, Blanchard EB, Kickling EJ: Antecedents and consequences of acute stress disorder among motor vehicle accident victims. *Behav Res Ther* 1996; 34:805-813
36. Griffin MG, Resick PA, Mechanic MB: Objective assessment of peritraumatic dissociation: psychophysiological indicators. *Am J Psychiatry* 1997; 154:1081-1088
37. Jaycox LH, Johnson KM, Foa EB: Acute stress disorder in female assault victims: Concurrent and retrospective reports, in *Proceedings of the 31st Annual Convention of the Association for the Advancement of Behavior Therapy*. New York, AABT, 1997
38. Bryant RA, Harvey AG: Relationship between acute stress disorder and posttraumatic stress disorder following mild traumatic brain injury. *Am J Psychiatry* 1998; 155:625-629
39. Classen C, Koopman C, Hales R, Spiegel D: Acute stress disorder as a predictor of posttraumatic stress symptoms. *Am J Psychiatry* 1998; 155:620-624
40. Eriksson NG, Lundin T: Early traumatic stress reactions among Swedish survivors of the *m/s Estonia* disaster. *Br J Psychiatry* 1996; 169:713-716
41. Shalev AY, Freedman S, Peri T, Brandes D, Sahar T: Predicting PTSD in trauma survivors: prospective evaluation of self-report and clinician-administered instruments. *Br J Psychiatry* 1997; 170:558-564
42. Blanchard EB, Hickling EJ, Vollmer AJ, Loos WR, Buckley TC, Jaccard J: Short-term follow-up of post-traumatic stress symptoms in motor vehicle accident victims. *Behav Res Ther* 1995; 33:369-377
43. Bernstein EM, Putnam FW: Development, reliability, and validity of a dissociation scale. *J Nerv Ment Dis* 1986; 174:727-735
44. Yehuda R, Elkin A, Binder-Brynes K, Kahana B, Southwick SM, Schmeidler J, Giller EL Jr: Dissociation in aging Holocaust survivors. *Am J Psychiatry* 1996; 153:935-940
45. Carlier IVE, Lamberts RD, Fouwels AJ, Gersons BPR: PTSD in relation to dissociation in traumatized police officers. *Am J Psychiatry* 1996; 153:1325-1328
46. Warshaw MG, Fierman E, Pratt L, Hunt M, Yonkers KA, Massion AO, Keller MB: Quality of life and dissociation in anxiety disorder of patients with histories of trauma or PTSD. *Am J Psychiatry* 1993; 150:1512-1516
47. Norton GR, Ross CA, Novotny MF: Factors that predict scores on the dissociative experiences scale. *J Clin Psychol* 1990; 46:273-277
48. Sanders B, Green JA: The factor structure of the dissociative experiences scale in college students. *Dissociation* 1997; 7: 23-27
49. Carlson EB, Putnam FW: An update on the dissociative experiences scale. *Dissociation* 1993; 6:16-27
50. Waller NG, Putnam FW, Carlson EB: Types of dissociation and dissociative types: a taxometric analysis of dissociative experiences. *Psychol Methods* 1996; 1:300-321
51. Marshall RD, Schneier FR, Knight CBG, Abbate LA, Fallon BA, Goetz D, Campeas R, Liebowitz MR: An open trial of paroxetine in patients with noncombat-related chronic PTSD. *J Clin Psychopharmacol* 1998; 18:10-18
52. Shore JH, Vollmer WM, Tatum EI: Community patterns of posttraumatic stress disorders. *J Nerv Ment Dis* 1989; 177: 681-685
53. Mellman TA, Randolph CA, Brawman-Mintzer O, Flores LP, Milanes FJ: Phenomenology and course of psychiatric disorders associated with combat-related posttraumatic stress disorder. *Am J Psychiatry* 1992; 149:1568-1574
54. McFarlane AC: The longitudinal course of posttraumatic morbidity: the range of outcomes and their predictors. *J Nerv Ment Dis* 1988; 176:30-39
55. Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB: Posttraumatic stress disorder in the National Comorbidity Survey. *Arch Gen Psychiatry* 1995; 52:1048-1060
56. Klein DF: A proposed definition of mental illness, in *Critical Issues in Psychiatric Diagnosis*. Edited by Spitzer RL, Klein DF. New York, Raven Press, 1978, pp 41-71
57. Marshall RD, Klein DF: Diagnostic classification of anxiety disorders: historical context and implications for neurobiology, in *Neurobiology of Mental Illness*. Edited by Charney DS, Nestler EJ, Bunney BS. New York, Oxford University Press, 1999, pp 437-450