Acute Stress Disorder as a Predictor of Posttraumatic Stress Disorder: A Systematic Review

Richard A. Bryant, PhD

Objective: The utility of the acute stress disorder diagnosis to describe acute stress reactions and predict subsequent posttraumatic stress disorder (PTSD) was evaluated.

Data Sources: A systematic search was conducted in the PsycINFO, MEDLINE, and PubMed databases for English-language articles published between 1994 and 2009 using keywords that combined *acute stress disorder and posttraumatic stress disorder*.

Study Selection: Studies were selected that assessed for acute stress disorder within 1 month of trauma exposure and assessed at a later time for PTSD, using established measures of acute stress disorder and PTSD.

Data Extraction: For each study, capacity of the acute stress disorder diagnosis to predict PTSD was calculated in terms of sensitivity, specificity, and positive and negative predictive power. For studies that reported subsyndromal acute stress disorder, the same analyses were calculated for cases that initially satisfied subsyndromal acute stress disorder criteria.

Data Synthesis: Twenty-two studies were identified as suitable for analysis (19 with adults and 3 with children). Diagnosis of acute stress disorder resulted in half the rate of distressed people in the acute phase being identified relative to including cases with subsyndromal acute stress disorder. In terms of prediction, the acute stress disorder diagnosis had reasonable positive predictive power (proportion of people with acute stress disorder who later developed PTSD). In contrast, the sensitivity (proportion of people who developed PTSD who initially met criteria for acute stress disorder) was poor.

Conclusions: The acute stress disorder diagnosis does not adequately identify the majority of people who will eventually develop PTSD. There is a need to formally describe acute stress reactions, but this goal may be achieved more usefully by describing the broad range of initial reactions rather than by attempting to predict subsequent PTSD.

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Corresponding author: *Richard A. Bryant, PhD, School of Psychology, University of New South Wales, Sydney, NSW 2052, Australia (r.bryant@unsw.edu.au).*

Acute stress disorder was introduced in the *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition (*DSM-IV*) as a new diagnosis to describe acute stress reactions that may precede posttraumatic stress disorder (PTSD). As discussions commence concerning the definitions of posttraumatic stress disorders in *DSM-5*, it is timely to consider the conceptual and empirical bases of the acute stress disorder diagnosis. The acute stress disorder diagnosis was introduced in *DSM-IV* for 2 reasons: to describe acute stress reactions that occur in the initial month after trauma exposure,[1] and to identify trauma survivors who are high risk for developing subsequent PTSD.[2] At the time of its introduction, there was little evidence to support the definition of the diagnosis.[3] Since its introduction, however, there has been an explosion of research into acute stress disorder over the past decade.[4,5] This review (1) outlines the definition and rationale of the acute stress disorder diagnosis, (2) considers its utility as a description of acute psychological reactions to trauma, (3) evaluates the evidence for its predictive ability in identifying trauma survivors who will develop PTSD, and (4) offers suggestions for how acute stress reactions could be defined in *DSM-5*.

The *DSM-IV* stipulates that acute stress disorder can occur after a fearful response to experiencing or witnessing a threatening event (cluster A). The requisite symptoms to meet criteria for acute stress disorder include 3 dissociative symptoms (cluster B), 1 reexperiencing symptom (cluster C), marked avoidance (cluster D), marked arousal (cluster E), and evidence of significant distress or impairment (cluster F). The disturbance must last for a minimum of 2 days and a maximum of 4 weeks (cluster G), after which time a diagnosis of PTSD can be considered. The primary differences between the criteria for acute stress disorder and PTSD are the time frame and the former's emphasis on dissociative reactions to the trauma. In terms of dissociation, a diagnosis of acute stress disorder requires that the individual has at least 3 of the following: (1) a subjective sense of numbing or detachment, (2) reduced awareness of one's surroundings, (3) derealization, (4) depersonalization, or (5) dissociative amnesia either during the traumatic event or in the following month.

One major reason for the introduction of the acute stress disorder diagnosis into *DSM-IV* was to fill a nosologic gap, because the PTSD diagnosis did not address posttrauma symptoms experienced in the first month posttrauma. Since *DSM-III-R*,[6] diagnosing PTSD within a month of the trauma was precluded because of concerns that this early diagnosis would unnecessarily pathologize transient and normative stress reactions. This issue is a significant one for health care, because trauma survivors will typically not be eligible for health care under existing insurance systems unless they are given a recognized diagnosis. Survivors of rape, natural disaster, accidents, or terrorist acts may benefit from mental health assistance in the initial month but could be denied mental health services because they needed to wait 1 month until a diagnosis of PTSD could be made. To overcome this potential barrier to care, *DSM-IV* described acute stress responses in the form of acute stress disorder.

The second goal of the acute stress disorder diagnosis was to identify people who are unlikely to adapt after the trauma but who will develop longer-term PTSD. Discriminating between people who are experiencing a transient stress reaction and those who are suffering the prodromal phase of chronic PTSD has been difficult, because of the robust finding that the majority of people who experience initial posttraumatic stress adapt in the following months.[7–10] It was argued that the inclusion of dissociative responses in the acute stress disorder diagnosis would enhance prediction of subsequent PTSD, because acute dissociative reactions are purportedly a crucial mechanism in posttraumatic adjustment.[11] This view can be traced back to the work of Janet,[12] which suggested that people who are overwhelmed by traumatic experiences may minimize emotional pain of the trauma by restricting awareness of the traumatic experience. Janet argued that, although this splitting of traumatic memories from awareness led to short-term reduction in distress, there was a loss of mental functioning, because mental resources were not available for other processes.[12] This view attracted much attention in the years leading up to *DSM-IV* and led to a commonly held conclusion that dissociation is a pivotal trauma response.[13] In terms of dissociation that occurs at the time of trauma (termed *peritraumatic dissociation*), it has been proposed that dissociating trauma memories and their associated affect from normal awareness impedes processing of these reactions and thereby leads to subsequent PTSD.[2] Support for the inclusion of dissociative symptoms in the acute stress disorder diagnosis to predict subsequent PTSD came from evidence demonstrating an association between peritraumatic dissociation and subsequent levels of PTSD, a finding that has been replicated across many longitudinal studies.[14–18]

This review provides a synthesis of all published prospective studies of acute stress disorder and PTSD in order to determine (1) the utility of the acute stress disorder diagnosis as a description of acute stress responses, and (2) the capacity of the acute stress disorder diagnosis to identify trauma survivors who will subsequently develop PTSD.

METHOD

Data Sources

Prospective studies of acute stress disorder and PTSD were located through searches in PsycINFO, MEDLINE, and PubMed for English-language articles published between 1994 and 2009 using keywords that combined *acute stress disorder and posttraumatic stress disorder.* Studies were then included if they assessed acute stress disorder within 1 month of trauma exposure and prospectively followed the same participants over time to assess PTSD diagnosis. Inclusion required studies to use measures of acute stress disorder and PTSD that permitted diagnostic prevalence rates based on *DSM-IV* criteria. Although there were many studies assessing the relationship between acute stress reactions (and particularly peritraumatic dissociation) and PTSD, these studies were not included if they did not formally diagnose acute stress disorder. In addition, studies that assessed acute stress disorder but did not report PTSD diagnostic results were excluded. Twenty-two studies were identified: 19 with adults and 3 with children. The duration between initial assessment and follow-up assessments ranged from 2 to 24 months, with a mean duration of 7.54 months.

RESULTS

Acute Stress Disorder as a Descriptor of Acute Stress Response

Table 1 summarizes the results of the 22 published longitudinal studies that have assessed acute stress disorder within a month of trauma exposure and determined the relationship between acute stress disorder and subsequent PTSD.[16,19–39] Table 1 initially presents the prevalence rates of acute stress disorder in these studies; the rates of full acute stress disorder range from 7% to 59%, with a mean rate of 17.41%. It is noteworthy that a proportion of these studies has also reported the prevalence rates of subsyndromal acute stress disorder, which is typically defined as satisfying at least 3 (rather than 4) of the symptom clusters. Including subsyndromal cases of acute stress disorder together with cases that meet full acute stress disorder criteria results in markedly higher rates of identified cases, with the range from 10% to 66% and a mean rate of 26.33%. Across some of these studies, the rates of trauma survivors displaying acute stress are increased primarily by not requiring the dissociative criteria to be met.

Acute Stress Disorder as a Predictor of PTSD

Table 1 also presents the relationship between acute stress disorder and PTSD in the 22 longitudinal studies. Table 1 summarizes 4 key outcomes of these studies: (1) sensitivity of the acute stress disorder diagnosis (defined as the proportion of people who initially met criteria for acute stress disorder who eventually developed PTSD), (2) specificity of acute stress disorder (the proportion of people who initially did not meet criteria for acute stress disorder who did not develop PTSD), (3) positive predictive power (the proportion of people who developed PTSD who initially met criteria for acute stress disorder), and (4) negative predictive power (proportion of people who did not develop PTSD who did not initially meet criteria for acute stress disorder). Perusal of these studies leads to several key findings.

First, there is considerable variability in the predictive ability of the acute stress disorder diagnosis.

Second, the positive predictive power of studies of adults was reasonable, with most studies of adults indicating that at least half of those trauma survivors with acute stress disorder subsequently met criteria for PTSD. In the context of most longitudinal studies indicating that the majority of trauma survivors adapt in the 6 months after exposure, these studies suggest that people who do meet criteria for acute stress disorder are at higher risk for persistent PTSD.

Third, the sensitivity across most studies was poor, indicating that the majority of trauma survivors who eventually developed PTSD did not meet the full criteria for acute stress

Table 1. Summary of Prospective Studies of the Relationship Between Acute Stress Disorder (ASD) and Posttraumatic Stress Disorder (PTSD)

Study/Type	Trauma	N	Follow-Up, n	Follow-Up Duration, mo	ASD, % (+ subsyndromal ASD, %)	PTSD, %	Sensitivity[a,b]	Specificity[a,c]	Positive Predictive Power[a,d]	Negative Predictive Power[a,e]
Adults										
Harvey and Bryant, 1998[19]	Motor vehicle accident	92	71	6	13 (21)[f,g]	25	0.39 (0.89)	0.96 (0.85)	0.78 (0.67)	0.85 (0.89)
Bryant and Harvey, 1998[20]	Brain injury	79	63	6	14	24	0.60	0.96	0.82	0.88
Brewin et al, 1999[21]	Assault	157	138	6	19	20	0.57 (0.79)	0.89 (0.76)	0.57 (0.46)	0.89 (0.93)
Holeva et al, 2001[22]	Motor vehicle accident	434	265	6	21	23	0.59	0.93	0.72	0.88
Fuglsang et al, 2004[23]	Motor vehicle accident	122	90	6–8	28	17	0.47	0.76	0.28	0.88
Balluffi et al, 2004[24]	Parental distress	272	161	2	32	21	0.61	0.78	0.42	0.89
Staab et al, 1996[25]	Typhoon	385	322	8	7 (22)	6	0.37	0.95	0.30	0.95
Kangas et al, 2005[26]	Cancer	82	63	6	23 (32)	22	0.61 (0.32)	0.89 (0.96)	0.53 (0.93)	0.84 (0.45)
Harvey and Bryant, 1999[27]	Motor vehicle accident	92	56	24	13 (21)[f,g]	25	0.29 (0.70)	0.92 (0.84)	0.62 (0.66)	0.75 (0.86)
Harvey and Bryant, 2000[28]	Brain injury	79	50	24	14	22	0.72	0.94	0.80	0.92
Elklit and Brink, 2004[29]	Assault	214	114	6	24	22	0.44	0.88	0.50	0.85
Hamanaka et al, 2006[30]	Motor vehicle accident	100	100	6	9 (10)	9	0.43 (1.0)	0.94 (0.87)	0.33 (0.37)	0.96 (1.0)
Bryant et al, 2008[31]	Injury	597	507	3	7 (20)[f]	10	0.31 (0.40)	0.96 (0.90)	0.46 (0.31)	0.93 (0.93)
Ginzburg et al, 2006[32]	Cardiac	196	116	7	18	16	0.39	0.86	0.33	0.88
Fullerton et al, 2004[33]	Disaster	207	116	13	26	17	0.58	0.85	0.42	0.91
Murray et al, 2002[16]	Motor vehicle accident	146	128	6	10	24	0.34	0.97	0.77	0.83
Kühn et al, 2006[34]	Injury	58	52	6	7 (17)	6	0.33 (0.67)	0.92 (0.86)	0.25 (0.20)	0.96 (0.98)
Kassam-Adams et al, 2009[35]	Parental injury	334	251	6	12 (25)	8	0.47 (0.79)	0.92 (0.68)	0.32 (0.17)	0.96 (0.98)
Elklit and Christiansen, 2010[36]	Rape	148	148	3	59 (66)	35	0.72 (0.92)	0.56 (0.43)	0.63 (0.63)	0.65 (0.82)
Children										
Dalgleish et al, 2008[37]	Motor vehicle accident	367	285	6	9 (32)[f]	7	0.24 (0.68)	0.93 (0.80)	0.26 (0.25)	0.93 (0.96)
Kassam-Adams and Winston, 2004[38]	Motor vehicle accident	243	177	3	8 (22)[f]	6	0.20 (0.40)	0.93 (0.80)	0.14 (0.11)	0.95 (0.96)
Bryant et al, 2007[39]	Injury	76	62	6	10 (28)[f]	13	0.25 (0.50)	0.93 (0.76)	0.33 (0.24)	0.89 (0.91)

[a]Numbers in parentheses refer to calculations based on cases defined by meeting at least subsyndromal ASD in the acute phase.
[b]Proportion of people who initially met criteria for acute stress disorder who eventually developed PTSD.
[c]Proportion of people who initially did not meet criteria for acute stress disorder who did not develop PTSD.
[d]Proportion of people who developed PTSD who initially met criteria for acute stress disorder.
[e]Proportion of people who did not develop PTSD who initially did not meet criteria for acute stress disorder.
[f]Denotes subsyndromal ASD defined as requiring reexperiencing, avoidance, and arousal clusters (not dissociation).
[g]Denotes subsyndromal ASD; included 15/18 participants who did not meet dissociative criteria.

disorder. This conclusion suggests that, if a major goal of acute stress disorder is to predict people who will subsequently develop PTSD, it is failing to identify the majority of those who will meet criteria for PTSD at some later time.

Fourth, although there are only 3 published studies of children, it is curious that the acute stress disorder diagnosis appears to have very poor capacity to predict PTSD in injured children. It is possible that children experience different trajectories of posttraumatic adjustment than adults, that the key markers of psychological impairment are distinctive in children, or that the definition of acute stress disorder or PTSD may not adequately capture the nature of stress reactions in children.[40]

Fifth, 12 studies reported data that permitted calculation of the predictive capacity of subsyndromal acute stress disorder, defined as satisfying only 3 of the acute stress disorder symptom clusters. These data are reported in parentheses in Table 1. Overall, these analyses indicate that the sensitivity is generally better when one adopts a subsyndromal approach. Seven of these studies specified that subsyndromal acute stress disorder was defined as not requiring the dissociative cluster, whereas the other 5 studies did not specify the cluster that was not satisfied. These findings suggest that focusing on general posttraumatic stress symptoms, rather than the more restrictive requirement of dissociation, results in more people who eventually develop PTSD being identified in the acute phase. It should be noted, however, that even this approach resulted in significant proportions of trauma survivors who developed PTSD not being identified in the acute phase.

DISCUSSION

The issue of whether acute stress disorder is retained in *DSM-5* depends on the purpose of the diagnosis. To adequately answer this question, it is useful to discern between the goals of (1) describing acute stress reactions and (2) predicting subsequent PTSD. It is apparent that there is a need for a diagnosis to describe the responses that can occur in the initial month. Although there may be criticisms of applying

a psychiatric diagnosis to a condition that may be transient,[41] provision of a diagnosis can facilitate access to mental health services for many trauma survivors in the acute phase.

The available evidence suggests that the current definition is overly restrictive in describing people who are acutely distressed, because requiring 3 (rather than 4) symptom clusters identifies more distressed trauma survivors. If the intention of describing these people is to identify those who currently experience distress that may benefit from some form of mental health intervention, it seems unwise to exclude those people who suffer posttraumatic stress but do not experience dissociative responses. The dissociative symptoms were included in the acute stress disorder diagnosis primarily to increase the capacity of the diagnosis to predict subsequent PTSD (see below); however, the dissociative symptoms requirement has resulted in many distressed people not qualifying for a diagnosis in the initial month. In this context, it is worth comparing the acute stress disorder diagnosis with the description of acute stress reactions in the tenth edition of the *International Statistical Classification of Diseases and Related Health Disorders* (*ICD-10*).[42] Rather than considering acute stress reaction a precursor of subsequent psychopathology, the *ICD-10* conceptualizes acute stress reaction as a transient reaction (that occurs in the initial 48 hours after a trauma) but encompasses an array of anxiety and depressive reactions. It has been argued that a more clinically useful approach to describing the broad range of emotional disturbances that may occur in the month after trauma and that may benefit from mental health services would be to allow for anxiety, depression, and other distressing reactions.[41,43]

A major issue for *DSM-5* is whether the acute stress disorder diagnosis should be retained as a predictor of subsequent PTSD. The available evidence suggests that the acute stress disorder diagnosis is not adequately identifying the majority of people who eventually develop PTSD. Various studies have attempted to modify the emphasis placed on initial symptoms to improve the acute prediction of PTSD, including emphasizing reexperiencing,[21] insomnia,[44] emotional numbing,[19] or overall level of acute symptoms.[45] Overall, none of these attempts provide adequate sensitivity or positive predictive power. In contrast, a more promising approach to acute prediction of subsequent PTSD is emerging from other acute biologic and cognitive markers.[4] For example, there is evidence that chronic PTSD is associated with a range of acute markers, including elevated resting heart rate,[46–48] elevated respiration rate,[49] low γ-aminobutyric acid plasma levels,[50] and maladaptive appraisals about the experiences and one's responses.[14,51,52] It appears that we have better scope for identifying people who are at high risk for PTSD in the acute phase by developing formulae that encompass these specific risk factors than by limiting prediction to diagnostic categories. There is no doubt that identifying individuals who are high risk for PTSD shortly after trauma is worthwhile because of the accumulating evidence that early intervention can limit subsequent PTSD in many cases.[53–57] At this point in time, it seems that early identification can be achieved with greater flexibility by not requiring the acute stress disorder diagnosis to function as a predictive tool. On this basis, it appears that acute stress disorder should not be retained in *DSM-5* as a predictor of subsequent PTSD.

One of the emerging suggestions from these data is that the role of peritraumatic dissociation is not as straightforward as previous studies have suggested. Despite the overwhelming evidence that peritraumatic dissociation is associated with subsequent PTSD,[18] recent meta-analyses have highlighted the fact that the majority of studies suggest that this relationship is indirect; that is, peritraumatic dissociation is not an independent predictor of PTSD.[58,59] Several possible mechanisms may account for the indirect relationship between peritraumatic dissociation and PTSD. Peritraumatic dissociation may be associated with PTSD because it is associated with other known risk factors for PTSD development. For example, there is a documented relationship between a history of childhood trauma and subsequent dissociation tendencies[60]; childhood trauma is also a risk factor for adult PTSD.[61] It has been suggested that peritraumatic dissociation may be linked to PTSD because of its association with childhood trauma.[62] Alternately, peritraumatic dissociation may be a consequence of elevated arousal that occurs during trauma.[63] Fear conditioning models posit that extreme fear and panic at the time of trauma conditions with associated stimuli subsequently lead to PTSD symptoms.[64] The possibility that peritraumatic dissociation may reflect panic responses is indirectly supported by evidence that dissociative phenomena occur in PTSD individuals with yohimbine-induced panic,[65] that dissociative reactions are commonly reported during panic attacks,[66] that more than half of trauma survivors report panic attacks during the trauma,[67] and that dissociative responses can be induced in recently trauma-exposed individuals with hyperventilation.[68] Of particular relevance is the finding that level of panic symptoms in the acute phase influences the relationship between fear and dissociation.[69,70]

Another problematic aspect of the current definition of peritraumatic dissociation in the acute stress disorder diagnosis is the time frame, which states that dissociative responses may be present during the traumatic experience or in the month following the event. The ambiguity concerning the time frame for dissociation is potentially problematic, because transient (peritraumatic) dissociation may represent impaired encoding of the traumatic experience, which may be protective because it can limit the degree to which aversive experiences are processed. In contrast, persistent dissociation may involve ongoing avoidance that impedes emotional processing of the experience and can lead to psychopathological responses.[71] This issue is an important one, because evidence suggests that the majority of trauma survivors experience transient dissociative reactions that do not persist beyond the trauma.[72] More important, there is emerging evidence that persistent dissociation is more predictive of both acute stress reactions[73] and subsequent PTSD[16,74] than dissociation that occurs only at the time of the traumatic experience. These data suggest that future

studies need to discriminate between initial alterations in awareness that may not be linked to maladaptive responses and responses that persist beyond the immediate phase of the traumatic experience.

It needs to be noted that there was marked variability in the prevalence rates of acute stress disorder and PTSD across studies. The prevalence rates of acute stress disorder varied from 7% to 28%, and rates of PTSD varied from 6% to 25%. This variability may be attributed, in part, to use of measures with varying psychometric strengths, different retention rates from the initial to the follow-up assessment, use of questionnaire rather than clinical interviews in some studies, "opt-in" procedures that may bias the sampling, and variable prevalence rates of acute stress disorder and PTSD. This variability is problematic, because prevalence rates will strongly influence the extent to which acute stress disorder may predict subsequent PTSD. Sensitivity can drop markedly when prevalence rates decrease,[75] and this difference in sensitivity has been shown across studies of PTSD in populations that have varying prevalence rates.[76] One consistent finding was the strong negative predictive power found across studies, which suggests that absence of acute stress disorder is suggestive of not developing PTSD. This observation probably has little clinical utility, however, because rates of chronic PTSD are generally low in trauma-exposed populations, and the goal for early intervention is to identify the minority of people who will develop a subsequent disorder.

A basic premise underpinning the acute stress disorder diagnosis is that there is a linear relationship between initial trauma reactions and subsequent PTSD that permits prediction of PTSD on the basis of acute stress reactions. There is increasing evidence that chronic PTSD can be influenced by a range of factors that occur after the acute response. Delayed-onset PTSD explicitly refers to PTSD reactions that develop at least 6 months after the trauma, which clearly impairs the capacity for prediction by acute symptoms. Although there has been much debate over the operational definition of delayed-onset PTSD, there appears to be evidence that it does occur in certain cases, especially in military settings.[77] There is also evidence that rates of PTSD can increase over time; one study that found that rates of PTSD following Hurricane Katrina increased suggested that increased prevalence rates may reflect the effects of cumulative stressors in New Orleans in the years after Katrina.[78] These patterns highlight the fact that there are limitations on the extent to which acute stress reactions can predict subsequent PTSD.

Options for *DSM-5*

Despite the problems associated with the predictive ability of acute stress disorder, there is a need to describe acute stress reactions in a manner that allows trauma survivors to receive needed mental health care. This goal could be achieved in several ways. First, it is possible to describe these transient reactions with a V-code, which *DSM-IV* uses to describe conditions that are not mental disorders but require clinical attention. This option can be problematic, because many health insurance companies may not recognize a V-code as a formal diagnosis. Second, it is possible to describe these reactions by describing them as an adjustment disorder. Although there are many similarities between the definition of adjustment disorder and acute stress disorder, there are several reasons to argue against using the adjustment disorder diagnosis for this purpose. First, acute posttraumatic stress reactions are qualitatively different from other adjustment problems in terms of the biologic, psychological, and cognitive responses. Second, there are clearly defined treatment protocols for acute posttraumatic stress reactions, and it may be beneficial in treatment planning to describe acute stress reactions as a specific type of reaction.[79] The third option is to remove the 1-month minimum duration criterion for PTSD and allow PTSD to be described in the initial month. This option is problematic, because it will inevitably result in many temporary stress reactions being labeled as PTSD, thereby increasing the risk of pathologizing transient responses. The fourth option is to retain a diagnosis, possibly termed acute stress disorder, that is less prescriptive than the *DSM-IV* definition, encompasses a broad range of common severe posttraumatic reactions (including anxiety, depression, and anger), and explicitly describes initial (and possibly transient) reactions. The latter option appears the most consistent with the available evidence, and it also serves the purpose of increasing access to care for many trauma survivors who may seek mental health services in the initial month after trauma. The exact definition of such a diagnosis should be based on available data sets that allow (1) identification of the most distressing symptoms and (2) the minimum time frame in which the diagnosis should be made.

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