Posttraumatic Stress Disorder: A Model of the Longitudinal Course and the Role of Risk Factors

Alexander C. McFarlane, M.D.

Posttraumatic stress disorder (PTSD) differs from other anxiety disorders in that experience of a traumatic event is necessary for the onset of the disorder. The condition runs a longitudinal course, involving a series of transitional states, with progressive modification occurring with time. Notably, only a small percentage of people that experience trauma will develop PTSD. Risk factors, such as prior trauma, prior psychiatric history, family psychiatric history, peritraumatic dissociation, acute stress symptoms, the nature of the biological response, and autonomic hyperarousal, need to be considered when setting up models to predict the course of the condition. These risk factors influence vulnerability to the onset of PTSD and its spontaneous remission. In the majority of cases, PTSD is accompanied by another condition, such as major depression, an anxiety disorder, or substance abuse. This comorbidity can also complicate the course of the disorder and raises questions about the role of PTSD in other psychiatric conditions. This article reviews what is known about the emergence of PTSD following exposure to a traumatic event using data from clinical studies.

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Naturalistic observations about the onset and course of posttraumatic stress disorder (PTSD) can inform theoretical hypotheses about PTSD. One of the main issues that emerges from such observations is that PTSD involves a series of transitional states and that progressive modification of the phenomenology of the disorder occurs with the passage of time. Examining the longitudinal course of PTSD raises a series of conceptual issues about the relationships between PTSD and other disorders and the way in which risk factors influence vulnerability to the disorder and its remission.[1] These issues are the template onto which emerging neurobiological findings need to be superimposed.

A problem exists concerning the role of risk factors related to the question of comorbidity, which has been largely ignored in the epidemiologic literature. When a traumatic event such as a natural disaster affects a community group or when an event occurs randomly to an individual, there will be people within that population who already have a psychiatric disorder, given that the prevalence of psychiatric disorder in a random community sample is approximately 15%.[2,3] Therefore, there will be 2 groups of people who develop a disorder in this setting. First, there will be those who had a preexisting disorder at the time of the traumatic event that may have acted as a risk factor to the onset of PTSD. The phenomenology, etiology, course, and pattern of comorbidity may be affected by this preexisting disorder. The second group will be those who develop the disorder de novo. The role and nature of the risk factors and the course of PTSD in the second group may be significantly different. To date, few studies have been able to make this separation because they have not begun in sufficient proximity to the event. This discussion will, however, focus on the latter group and present a model for the emergence of PTSD following exposure to an event.

A MODEL OF ONSET

PTSD needs to be thought of as having a series of stages. It does not begin in the immediate aftermath of the event. Rather, there is a critical phase in which the stress response in some individuals will settle, whereas in others, there will be a progressive dysregulation. In other words, the response at the time of the traumatic event may be only one determinant of who develops PTSD, with the ability of the individual to modulate the acute stress response and restore psychological and biological homeostasis being of equal importance. Thus, PTSD may be as much a disorder of transition as a specific stress disorder. Once the disorder has emerged, it will resolve in approximately 60% of subjects.[3] This raises the question as to whether the acute form is different from the chronic, unremitting form of PTSD.

From the Department of Psychiatry, University of Adelaide, Queen Elizabeth Hospital, Woodville, South Australia, Australia.

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Reprint requests to: Alexander C. McFarlane, M.D., Department of Psychiatry, University of Adelaide, Queen Elizabeth Hospital, Woodville, South Australia, Australia (e-mail: amcfarla@pulse.health.adelaide.edu.au).

There are compelling neurobiological reasons to believe that PTSD emerges in the aftermath of a traumatic event, given the importance of traumatic memory to the etiology of PTSD.[4] For example, Gold and McGaugh[5] have noted that the susceptibility of memory storage to processes and modulating influences occurring after learning provides the opportunity for emotional activation to regulate the strength of memory transfers representing important experiences. A model of risk factors therefore needs to consider both determinants of the initial reaction and factors that might influence learning after exposure to events.

In other words, PTSD may be a disorder in which there is a failure of the resolution of the acute stress response or, in the more toxic forms of posttraumatic adaptation, a progressive recruitment of instabilities of the underlying neurobiological systems.[6] This possibility raises many interesting questions about the relationship between the acute psychological state of an individual, his or her neurobiological systems, and the capacity for preventative treatment interventions to be of benefit in the immediate period after a traumatic experience. The ability of an individual to modulate his or her distress at these times may be critical in determining long-term outcomes. It may be that during this phase there is a secondary series of modifications of underlying memory structures that further increases the risk of chronic outcomes.

LONG-TERM OUTCOMES OF TRAUMATIC EXPOSURE

In understanding the longitudinal consequences of trauma, information should be derived from a range of victim groups because the outcomes of different types of traumas may vary substantially. For example, clinical experience suggests that the long-term consequences of child abuse are very different from the experience of a natural disaster or other circumscribed trauma in adult life.[7] Just as the prevalence of PTSD varies following different traumas,[3] so might its longitudinal course.

Blank[8] has highlighted the multiple variations of the longitudinal course of PTSD, namely, acute, delayed, chronic, intermittent, residual, and reactivated patterns. Longitudinal studies like the National Vietnam Veterans Readjustment Study (NVVRS)[9] and the study by Lee et al.[10] suggest a need to define a posttraumatic syndrome where the full PTSD criteria are not met, since there is a significant associated social disadvantage in the fulfillment of roles. An important methodological issue emerges in the way populations are studied. The results from the retrospective lifetime history method are different from the results of longitudinal observation. Subjects are not especially accurate when retrospectively recalling the course of their illness and tend to be influenced by the more recent state of that illness. A matter requiring exploration is the possibility that the etiology of an unremitting chronic disorder might be different from that of an intermittent disorder. At this time, the data do not exist to answer this question. Before the range of psychological consequences of trauma is examined, the issue of physical health will be discussed. The range of long-term effects of trauma on health is an important observation because PTSD is only one vector of the adverse consequence of trauma, and it provides insights into individuals' neurobiology.

Physical Health

Although a number of studies have noted an increased reporting of physical symptoms in persons with PTSD, the reason for this association is unclear.[11] Escobar et al.[12] reported on the development of new physical symptoms 1 year after a natural disaster in Puerto Rico. Victims of the disaster were more likely to report new gastrointestinal or pseudoneurologic symptoms than persons not exposed to the disaster. Although these symptoms may have been indicators of psychopathology, no correlation was made with the presence of psychiatric illness.

Lee et al.[10] examined the impact of combat in a group of sophomores recruited at Harvard University by following their health until the age of 65 years. These men were selected for their physical and psychological health and high levels of achievement at university. Although 72 had a high level of combat exposure, only 1 retrospectively satisfied the diagnostic criteria for PTSD in 1946, with another 4 having a PTSD-like syndrome. However, combat exposure predicted early death, independent of PTSD. Fifty-six percent of the men who had experienced heavy combat were dead or chronically ill by the age of 65 years.[10] The length of follow-up in this study makes the results especially noteworthy as these long-term effects of trauma may emerge only in old age when the risk of physical illness is increased. The investigation of Askoveld et al.[13] of Norwegian merchant mariners from World War II similarly showed that they had an increased mortality and earlier onset of dementia, findings that would emerge only in old age.

Modified Vulnerability

Some concepts developed in the literature about the outcome of the treatment of depression[14] can be usefully applied to the description of the longitudinal course of PTSD. It is important to distinguish between remission and recovery from a disorder, because recovery defines the end of an illness episode and presumes that a further episode is a recurrence of the disorder rather than relapse of the current one. In medicolegal circles, where the prognosis of PTSD and its long-term outcome are of particular relevance, there is an assumption that once the symptoms of PTSD have resolved, the disorder does not recur. This is based on the idea that PTSD is an adaptational response to an event,[15] beginning with an acute stress reaction which then follows a predictable course that eventually resolves without sequelae. However, emerging evidence suggests that this is

not the case. In particular, Solomon et al.[16] described 35 soldiers who had several exposures to combat and multiple episodes of PTSD. The original PTSD may have been reactivated in some soldiers, whereas in others, the second episode may have been substantially independent of the first. The ability of traumatic exposure to alter the neurobiology of the acute stress response was also demonstrated by Resnick et al.,[17] who found altered cortisol responsiveness in women who had been raped a second time.

Thus, the question remains of how trauma modifies vulnerability. If somebody recovers, no longer satisfying the diagnostic criteria, is that person left with residual deficits? If a person does relapse, is this because there is a progressive sensitization that occurs on subsequent exposures? In particular, if a person does have a second trauma, does that individual develop a new case of PTSD or experience recurrence of the previous one?

Comorbid Disorders

The range of specific trauma-related disorders has received increasing attention, as has the nonspecific role of trauma as a trigger for a variety of psychiatric disorders.[18] There is a consistent finding across a range of traumatic events that PTSD is only one of a number of psychiatric disorders that occur in such settings. In fact, in the majority of cases, even in community samples, PTSD is usually accompanied by another disorder such as major depression, an anxiety disorder, or substance abuse.[3,9] In general psychiatric patient populations with a range of disorders, there has been surprisingly little research examining the extent to which trauma plays a role in the onset and maintenance of disorders. However, a series of investigations have looked at the prevalence of child abuse in clinical samples and found prevalence rates in the order of 18% to 60%.[19] Davidson and Smith[20] and McFarlane[21] have found that the lifetime rates of PTSD in general patient samples are also significantly underestimated.

The rates of traumatic events in seriously mentally ill patients are much greater than observed in community samples.[22] The course of PTSD and a comorbid condition may be independent, and trauma in itself may be an important lifetime risk factor for a range of other psychiatric conditions. This evidence about very high rates of traumatization in populations with serious mental illness raises challenging questions about the role of environmental factors in psychotic disorders. This idea is not a fashionable one in the current world of the ascendancy of biological models of schizophrenia.

EVIDENCE ABOUT THE LONGITUDINAL COURSE OF PTSD FROM COHORT STUDIES

The most informative studies of the longitudinal course of PTSD have focused on specific types of events, and the studies of combat veterans are a particularly fruitful source of information. The NVVRS found that 19 years after combat exposure, 15% of veterans still had PTSD,[9] whereas the lifetime prevalence was more than double this figure. A similar Australian study[23] found a current prevalence of 12%, which also represented about one third of the lifetime prevalence. It is especially noteworthy that the results of both studies confirm the remission rate found in the National Comorbidity Survey (NCS)[3] because many of the NVVRS veterans had received more focused treatment than occurred in the NCS community sample. These data suggest that there is a group of people with PTSD who have a chronic course despite treatment.

The relationship between the acute effects of combat and long-term outcome has been investigated most thoroughly in veterans of the 1982 Lebanon War.[24] This study found that soldiers who become acutely distressed at the time of combat have a much higher risk of PTSD and that the disorder emerges from combat stress reactions. On the other hand, the rate of PTSD among those who cope at the time of the combat is significantly less. This study also provided valuable insights into the pattern of symptom emergence.

It was found that intrusive symptoms had low diagnostic specificity in contrast to the combination of intrusive symptoms and avoidance symptoms. In addition, the prominence of intrusive symptoms decreased over a 2-year period, while avoidance increased.[8] The relationship between acute reactions and other types of traumatic events has not been investigated systematically because it is uncommon for people to present for treatment in the immediate aftermath of disasters and accidents. Retrospective studies have examined these issues in 824 Dutch resistance fighters[25] and found that 5 decades later, 27% of men and 20% of women were currently experiencing PTSD. Thus, there is evidence from these cohort studies of combat to suggest the potential chronicity of PTSD, and these data raise the possibility that the phenomenology of these disorders changes with time.

Studies of disaster come to similar conclusions. Longitudinal studies of disaster victims suggest a similar picture in which delayed PTSD is uncommon, and the typical course of PTSD begins in the immediate aftermath of the trauma and then continues. A study of 469 firefighters who had an intense exposure to a major Australian bushfire disaster[26] found that in the majority with a chronic course of PTSD, the symptoms fluctuated significantly with the passage of time, a picture that does not emerge from most retrospective studies. Delayed onset of PTSD was rare, and some who reported such a pattern of symptoms failed to recall their acute posttraumatic symptoms. As in the NCS,[3] only 15% had PTSD in the absence of an anxiety disorder or major depression,[26] indicating that PTSD is only one of a number of psychiatric disorders that arise in such settings.[3]

Forty-two months after the Australian disaster, symptoms remained in 56% of those who had PTSD immediately after the disaster.[26] However, when followed up 8 years af-

ter the disaster, only 4% continued to meet criteria for a diagnosis of PTSD. At this stage, 60% still had significant intrusive symptoms, and symptoms of disordered arousal were as common as at 42 months. Failure to reach the diagnostic threshold of avoidance and estrangement represented the main reason that those firefighters did not qualify for PTSD. At 8 years, disordered arousal was the most prominent clinical feature, suggesting that anxiety and depressive symptoms were the most prominent residual symptoms of the disorder. This finding contrasted with those in a clinical population who were followed after the same disaster in which there tended to be a much greater stability of intrusive and avoidance symptoms.[27] This comparison suggests that quite different pictures can emerge from community and clinical samples studied after the same event. It may be the case that there is a different longitudinal course in PTSD according to the initial severity. In the most severe forms, the symptoms may be relatively stable with the passage of time, whereas with the less intense forms, the specific trauma-related symptoms of intrusion and avoidance decrease while the disordered affect and arousal remain.

The longest follow-up of those with PTSD was conducted after the 1972 Buffalo Creek Dam collapse that caused a devastating flood disaster. Grace et al.[28] conducted a 14-year follow-up of the victims that included 121 (32%) of the original sample of 381 subjects who participated in the study. Forty-four percent had PTSD in 1974; this prevalence rate had decreased to 28% in 1986. The symptoms in this population fluctuated with the passage of time, explaining the emergence of what otherwise might have been considered delayed-onset cases.

THE RELATIONSHIP BETWEEN THE ACUTE STRESS RESPONSE AND PTSD

When examining the acute stress response, a critical issue to consider is the inability to modulate the initial reaction; vulnerability and protective factors are both relevant. If there were a role for protective factors in the etiology of PTSD, this is the domain in which they would most probably exert their influence. How people can recreate a sense of safety and control in their lives is paramount. Investigation of the transitional stage from the acute stress response may also be of particular importance in understanding why some individuals develop PTSD after traumatic events, whereas others develop conditions such as major depression and panic disorder. There may be general risk factors for psychiatric disorders and specific risk factors for individual disorders operating in this transitional phase. In other words, there may be a general vulnerability to dysregulation, the pattern of which is then determined by specific underlying vulnerability markers or risk factors that can be defined at the time of the traumatic experience.

In a study of the acute patterns of reaction within 24 hours of admittance of 200 subjects to hospital following motor vehicle accidents,[29] a range of diagnostic outcomes was examined. These data have demonstrated that the subjects who develop PTSD with or with out associated disorders (N = 36) or major depressive disorder (MDD) (N = 12) cannot be easily differentiated from the subjects who have no disorder according to the nature of their symptomatic reactions on the first day after the accident. The existence of this MDD group raises the possibility that there are 2 types of depression, 1 that is trauma related and the other that is not.

Differences emerged by the 10th day, when anxiety in the PTSD group was starting to increase, whereas, in the depression group and the no disorder group, it was starting to decrease. This finding was generally true for the other groups of symptoms as well.[29]

In 42 of these motor accident victims, the relationship between acute cortisol rise and the development of psychiatric disorder at 6 months was examined.[29] These measurements were made from compulsory blood alcohol samples taken a mean of 2 hours postaccident. Preliminary findings were that the subjects who went on to develop PTSD had a lower cortisol rise than those with no disorder or MDD.[29] These findings suggest that the people who went on to develop PTSD had an abnormal acute stress response. We did not find any difference on psychological measures on the first day among the 3 groups, apart from avoidance in the people who went on to develop MDD.

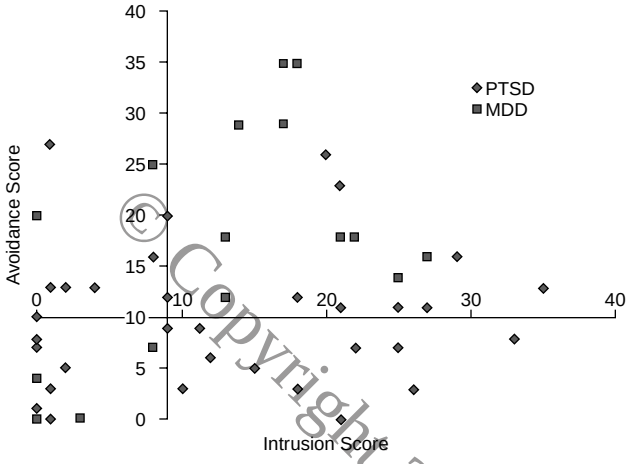
When we look at group data, we can often create too simplistic a statistical model. To investigate this issue, we looked at the individual subjects with PTSD and those with MDD and plotted them according to their avoidance and intrusion scores on the first day (Figure 1). The fascinating finding is that some people who develop PTSD have extremely low scores of avoidance and intrusion on the first day, and the individuals who go on to develop MDD tend to congregate around high intrusion and high avoidance (A.C.M., unpublished data). Thus, this finding is not due to lack of variance.

The relationship between the acute stress response and the outcome has also been investigated by other groups,[30] including Blanchard et al.,[31] who have specifically focused on motor vehicle accident victims. They found that acute stress disorder is common, and vulnerability factors to acute stress disorder can be demonstrated including previous mood disorder and a lifetime history of other Axis I and Axis II disorders. However, acute stress disorder did not predict outcome in relation to PTSD. In conclusion, these combined data suggest the existence of a complex matrix of influences determining the emergence of an acute stress and its relationship to the long-term outcome.

THE EMERGENCE OF PTSD

If the trajectory of the intrusive phenomena is tracked in the 6 months following the initiating accident, the pro-

Figure 1. Avoidance and Intrusion Scores in Patients With Posttraumatic Stress Disorder (PTSD; N = 36) or Major Depressive Disorder (MDD; N = 14)[a]



[a]A.C.M., unpublished data. Data for some patients missing at different points.

gressive dysregulation characteristic of PTSD becomes apparent. In the PTSD group, the severity of the intrusions increases with time, whereas in the no disorder group severity decreases and in the MDD cohort there is a small reduction in severity.[29]

The process that leads to this augmentation of intrusion is of practical and theoretical significance and appears to be influenced by 3 factors. First, the distress caused by the recurring traumatic memories leads to a process of retraumatization; patients have reported that the experience of flashbacks is worse than the initiating traumatic experience because any sense of control or behavioral choice is removed in the memory. Second, in an animal model, Pynoos et al.[32] demonstrated that exposure to traumatic triggers is as important to the emergence of PTSD as the initial stress exposure. Finally, the observation that the symptoms of PTSD are maintained and triggered by day-to-day adverse life experience suggests that a progressive modification of the individual's stress responsiveness may be an important aspect of the dysregulation that is central to the psychobiology of PTSD.[33–35]

This effect is similar to the transformations of stress responsiveness via kindling[36] that are thought to operate in affective disorders. There appears to be an initial period of cognitive appraisal of the traumatic experience and the associated self-regulation, during which time the traumatized individual processes and reworks the experience, elicits social support, and tries to integrate the horror of the experience and the losses suffered. Over a period of several weeks, the typical symptom constellation of PTSD begins to congeal. These observations are not new. In 1918, Rivers[37] commented:

> I hope to show that many of the most trying and distressing symptoms from which the subjects of war and neurosis suffer are not the necessary result of the strains and shocks to which they have been exposed to in warfare, but are due to the attempts to banish from the mind distressing symptoms of warfare or painful affective states which have come into being as a result of their war experience.(p173)

In 1945, Grinker[38] stated:

> It is to our astonishment, the majority of the neuroses that are hospitalized today in the convalescent hospitals are people who have developed either the first signs of neurosis on return to this country or have become worse after landing on these shores.[39(p188)]

The surprise to which he refers is a result of the belief that once the ongoing stress of war or threat of combat ceased, all the disorders observed during the war would go into spontaneous remission.[39] This belief in the role of the acute stress reaction as a determinant of chronic PTSD may be the reason why PTSD was not included in DSM-I or DSM-II, in contrast to the inclusion of diagnostic categories such as "transient situational disturbance."

THE NATURE OF CHRONIC PTSD

The challenging question of whether the process and nature of the disorder change in the more chronic forms of PTSD remains. This model exists in schizophrenia: the disease changes as it merges into the chronic form in which the negative symptoms become more prominent. Kardiner[40] made an interesting comment about chronic traumatic neurosis akin to this notion: "Some subjects undergo a deterioration that is not dissimilar to that which occurs in schizophrenia. In fact the subject suffers from the kind of delusion that the world becomes an unbearably hostile place."(p249) The increasing dominance of numbing and withdrawal in chronic PTSD has many similarities with the affective blunting and social decline in schizophrenia, although this idea has received little attention in the more recent literature.

CONCLUSION

The difference between PTSD and other psychiatric disorders is that, by definition, it follows the experience of a traumatic event, and such an event is necessary for the onset of the disorder. What appear to differentiate these events, which are characterized by horror and helplessness, from other types of life events are the quality and nature of the emotional memory that they generate. The stressor initiates the traumatic memories, and whether PTSD emerges depends on the ability of the individual to modify the associated hyperarousal and neurobiological cascade. This transition phase is likely to be the period when risk

factors and protective factors are of greatest significance. The nature of the acute biological stress response is the springboard from which the psychological adaptation is launched. The role of vulnerability factors is important, as only a small percentage of the people exposed to a traumatic event develop PTSD. The challenge is to set up a matrix model that takes account of vulnerability factors at the different temporal points, i.e., onset and spontaneous remission of the disorder. Current knowledge factors that need to be considered include prior trauma, prior psychiatric history, family psychiatric history, peritraumatic dissociation, the severity of the acute stress symptoms, the nature of the biological stress response, and autonomic hyperarousal. The exploration of such models will assist in developing a better typology and prediction of the variable course of this disorder.

A further challenge is to better understand the problem of the margins of PTSD and the related problem of comorbidity. Both the high prevalence of comorbid MDD and the fact that this phenomena is not ubiquitous raise the question of whether there is a problem with the inclusion criteria of the diagnosis, or whether this separation is artificial and more generic features would better group the ranges of responses to trauma. The research task is to maintain a sufficiently broad base of observation to allow exploration of these issues.

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