Anxiety Disorders

Post-Traumatic Stress Disorder, Conditioning, and Network Theory

By ROGER K. PITMAN, MD

Time is functional, it can be referred to as action; we say a thing is "brought about" by time. What sort of thing? Change! Now is not then, here not there, for between them lies motion. But the motion by which one measures time is circular, is in a closed circle; and might almost equally well be described as rest, as cessation of movement—for the there repeats itself constantly in the here, the past in the present.

Thomas Mann, The Magic Mountain, 1924

Time does not heal all wounds. We don’t expect time to heal defects, but we do expect it to heal wounds. Hence, when a psychological wound does not heal, we may be inclined to believe that it actually represents some kind of defect. This accounts for much of the resistance that the diagnosis post-traumatic stress disorder (PTSD) has encountered prior to and following its introduction into the official American psychiatric nomenclature in 1980. For some time this debate was cast between two competing sociological theories: stress evaporation versus residual stress. The former implies that in a healthy person a stressful event may cause temporary adjustment problems and discomfort, but that eventually the bad effects of the stress will fade away. The latter, conversely, admits the possibility of long-term adverse psychological consequences of stress.

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The search for defect in PTSD should not be confused with the possibility of premorbid vulnerability. As physically healthy per-
sons may differ in their resistance to an infectious agent, so may mentally healthy persons differ in their resistance to a psychologically stressful event. The life history of the individual may provide clues as to vulnerability; frequently, it does not. The intriguing possibility that vulnerability to PTSD may be constitutional is yet to be explored in genetic or physiological studies. The orthodox psychoanalytic approach to PTSD postulates that those who develop a mental disorder as the result of a stressful event must have preexisting unconscious developmental conflict or fixation, and the event activates this conflict. Unfortunately, the plastic nature of such explanations means that one can generally be found in many or even most cases. This hypothesis has never been tested in any rigorous research design.

The importance of considering the meaning of an event within the life continuity of the individual is a psychological truism applicable to PTSD. However, the current definition of a PTSD-producing event as "outside the range of usual human experience" suggests that such events are discontinuous with, and consequently not readily understandable in the context of, the morbid lives of most PTSD sufferers. The clinician who resorts to an idiosyncratic psychodynamic explanation of the meaning of an event in order to establish its stressful nature is probably best advised to seek a diagnosis other than PTSD. Recently, analytically oriented theorists have become more willing to accept the traumatic event in PTSD at face value, and have redirected psychodynamic efforts toward understanding the conflicts, personality changes, and compensatory defenses deriving from the trauma.

**CONDITIONING AND PTSD**

As the A criterion (Table) in the DSM-III-R points to the traumatic event as the primary etiological factor in PTSD, certain of the other DSM-III-R criteria provide clues as to PTSD's pathogenesis. For example, the symptoms of "intense psychological distress" (B.4 in the Table) and "physiological reactivity" (D.6) "... upon exposure to events that symbolize or resemble an aspect of the traumatic event" (subcriteria that were previously joined in DSM-III) both suggest a role for Pavlovian (or classical) conditioning in the disorder. Within the Pavlovian framework, the traumatic event may be likened to an intense unconditioned stimulus (UCS) evoking in the individual an unconditioned (traumatic) response (UCR). Subsequently, events that may themselves be emotionally neutral, (D.6) through their association with the traumatic UCS may come to serve as conditioned stimuli (CS) that evoke the original trauma and emotional responses such as fear and anger, now referred to as the conditioned response (CR). Kolb has discussed the notion of PTSD as a conditioned emotional response.

A strength of the conditioning model of PTSD is that it has lent itself to testing in the laboratory. Prior to work in our laboratory described below, there were four major published psychophysiological studies of combat-related PTSD (reviewed in references 11 and 12). Each attempted to document the DSM-III-R criterion of physiological reactivity to events connected to the traumatic event. In these studies, PTSD and control subjects were exposed to recorded combat sights and sounds while heart rate, electrodermal, and other physiological responses were measured. In each study, the PTSD group showed greater autonomic arousal in response to the combat stimuli compared with the various control groups; the ability of the physiological measures to discriminate PTSD from control subjects has ranged from 80% to 95%.

While a primary role for Pavlovian conditioning may be seen in the pathogenesis of PTSD, a secondary role may also be seen for operant conditioning. If exposure to events resembling the stressor leads to the activation of unpleasant emotions, behaviors that avoid such exposure may be expected to be reinforced. This can be viewed as the mechanism behind the DSM-III-R criteria, "persistent avoidance of stimuli associ-
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about responding in this context, including expressive verbal behavior, overt acts, and the visceral and somatic events that mediate arousal and action (response propositions); and 3) information that defines the meaning of the stimulus and response data (meaning propositions). These propositions are organized into an associative network which, when a critical number of propositions are accessed, is processed as a unit. Pitman and Orr have hypothesized that PTSD may be conceptualized as consisting of one or more pathological emotional networks which, when activated, produce its characteristic reexperiencing symptomatology. The unitary nature of network processing in PTSD is best illustrated by the flashback phenomenon (B.3 in the Table), in which a full-blown, complete experiential memory of the traumatic event may be activated with a loss of reality testing.

In research in our laboratory, we have attempted to instigate PTSD emotional networks through the means of mental imagery. Our approach, based on Lang’s methodology, has been to expose medication-free combat PTSD patients and controls, matched for age, educational level, combat exposure, and event severity, to self-generated mental imagery of their own specific traumatic events. This is accomplished by reading to subjects individualized scripts portraying these events and instructing them to imagine the events while physiological measures are recorded. As in the psychophysiological studies mentioned above, our results have shown that PTSD subjects produce significantly and dramatically (up to fivefold) higher physiological responses to their own traumatic imagery stimuli than do controls.17

The following is an example of an individualized combat traumatic script constructed from the experience of a Vietnam veteran with PTSD.

You have just received a signal for a hasty ambush. You sit in the elephant grass trying to figure out your field of fire. Then you hear them coming, talking and laughing and making jokes. You hold your breath and your heart stops. You freeze, like you can’t move. Their voices keep getting louder and louder. When they get right in front of you, you can see them from the waist down, their AKs slung. You count them as they pass. When you get to four, all shit breaks loose. You pull your trigger and hold it down. The next thing you know, you’re staring at a dead Gook’s feet and your teammates are yelling “Get up. We gotta go.” Now your heart is pounding and you feel jittery all over, like you want to run but there’s no place to go. You stand up and see the top of the Gook’s head blown off, his brains glaring in the sun. You’ve never seen blood and guts before. You feel sick to your stomach and in a state of shock.

The Figure schematizes the above experience as a theoretical emotional network utilizing the format suggested by Lang. The stimulus propositions (elephant grass, hand signal, AKs, Gooks, brains) appear in shaded circles. The meaning propositions (field of fire, enemy, dead) appear in light circles. The response propositions (freeze, pull trigger, heart pounds, run) appear in heavy circles.

The applicability of network theory to the PTSD flashback phenomenon is illustrated by the following case anecdote. A 57-year-old Korean veteran with severe PTSD described continued on page 187
the occurrence of one of his flashbacks. He was sitting at the kitchen table having coffee and watching the morning news. While someone was being interviewed, helicopter sounds were heard in the background on the TV. The patient's wife reported that he became dazed, complained of nausea, and shouted "they're bringing more in." He then went into a deep sleep and when he awoke he was lucid.

This patient had served aboard a hospital ship with the daily duty of unloading severely wounded GIs who had been freshly evacuated from the battlefield via helicopter. He often got little or no sleep for long periods. The smell of burnt human flesh had made him nauseous on many occasions. Following his naval service, he had frequent nightmares in which helicopters would fly over and dump bloody body parts on him.

In the anecdote, the stimulus proposition "helicopter sound" may be considered to belong to two networks, i.e., the patient's present emotionally neutral experience (sitting at the kitchen table watching TV), and the emotionally charged traumatic memory (receiving wounded on the hospital ship). Furthermore, "helicopter sound" served as a bridge, or nodal point, between the two networks. Because of the strength of the traumatic network, this one proposition was capable of activating it in entirety, leading to its being processed as a unit. The patient's mentation thereby became derailed from the present into the past. His nausea represented a physiological response proposition properly connected with the past traumatic network, but not with the present. The perception "they're bringing more in" represented a second (hallucinatory) stimulus proposition connected with the activated traumatic network as well. During subsequent interview, the patient commented that he had seen and heard helicopters on TV before without going into a flashback. He thought that hearing the helicopter noise without seeing it on the screen might have made the difference.

Thus, present experience and traumatic memory may be seen as competing for the patient's attention. The sight of the helicopter on TV could have comprised a stimulus proposition properly connected with the present, but not with the past traumatic network ("I'm seeing the helicopter on TV so I can't really be back on the hospital ship"), which had it been available might have tipped the balance in favor of the present, preventing the flashback.

Although attractive, the emotional network theory of PTSD still requires that the network be accessed to be activated, making it difficult to account for the occurrence of spontaneous symptoms in PTSD such as intrusive recollections and nightmares. One way of addressing this problem is to consider that PTSD networks may be capable of spontaneous activation by sheer virtue of their neurological strength. PTSD memories seem to have become deeply etched into the neural template of the organism. It is quite possible, even likely, that the brain is primed for such etching by the extreme state of arousal brought about by a life-or-death or other traumatic situation. Pursuit of this metaphor leads to the question of what the "acid" might be that is capable of "etching" traumatic experience into the brain.

Miller has pursued a neurological network theory suggesting that Pavlovian conditioning may be conceptualized as a process by which cortical synapses are strengthened either through repetition, or through a facilitating action exerted by the ascending nonspecific monoaminergic pathways. Thus, it is possible that extreme activation of the locus ceruleus, for example, with a consequent massive and diffuse release of norepinephrine into the cortex may, through a process that we might term "superconditioning," etch the stimulus, meaning, and response propositions that are active at the time of the traumatic event into a durable pathological network that forms the basis for subsequent PTSD. Other candidates for the role of "etching" agents may be neuropeptides such as vasopressin and ACTH, which in animals appear to be released in stress, increase NE turnover rates, modify CNS neurotransmission, enhance acquisition and resistance to extinction of conditioned avoidance responses, and
enable memory consolidation and/or retrieval. However, support for this superconditioning theory of PTSD at present is only indirect.

Along with the intrusion and avoidance phenomena, persistent symptoms of increased arousal (the D criteria in the Table) complete the DSM-III-R triad of PTSD symptomatology. In addition to possible modifications in cortical synaptic connectivity, it is likely that traumatic experience can produce a lasting propensity to subcortically mediated hyperarousal. In this regard, a recent study reported that controlled double-blind administration of sodium lactate produced panic attacks in six of seven Vietnam veteran PTSD patients, and flashbacks in all seven.

The co-occurrence of panic (intensely anxious emotion) and flashback supports an emotional network theory of PTSD. Van der Kolk et al have suggested that “long-term augmentation of locus ceruleus pathways following trauma underlies the repetitive intrusive recollections and nightmares that plague patients with PTSD.” In an alternative psychological model, Kolb proposes that the observed vulnerability to excessive arousal in PTSD in secondary to failure of the protective cortical stimulus barrier originally proposed by Freud, as the consequence of excessive and prolonged sensitizing traumatic stimulation, with secondary release of hindbrain arousal centers.

**CLINICAL IMPLICATIONS**

**Assessment**

Because of its etiological primacy, the history of the traumatic event is the most important factor in the diagnostic assessment of PTSD. One might suppose that this history would be readily obtainable, given the event’s importance in the mental life of the patient. Curiously, experience has indicated that the opposite is often the case. Probably the foremost reason for this is the patient’s efforts to avoid recollection of the event because of the discomfort it induces. The interviewer may consciously or unconsciously collaborate with this avoidance. Unless the clinician specifically asks about a potentially traumatizing event in the patient’s past, it is likely that the patient will not volunteer it.

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**Prevention**

PTSD is obviously a disorder in which the proverbial ounce of prevention is worth a pound of cure. In the event that reliable vulnerability factors to PTSD are discovered, thought should be given to selecting out highly vulnerable individuals from exposure to potentially traumatic events such as combat (if such events themselves cannot be prevented). An intriguing possibility for the future might be the administration, at the time of an anticipated or actual traumatic event, of a drug designed to prevent the formation of a pathological PTSD network by aborting a “superconditioning” reaction in the victim. In this regard, the opioid peptides and oxytocin have been found to exert effects opposite to those discussed above for vasopressin and ACTH and to function as endogenous amnestic agents, suggesting possible models for pharmacological intervention.

**Treatment**

Foa and Kozak have proposed a model of psychotherapy that incorporates network theory and is based upon the concept of emotional processing. This formulation is relevant to PTSD, in that it is widely accepted that PTSD symptomatology results from failure to successfully process a traumatic event. Foa and Kozak propose that successful emotional processing requires activation of the pathological network, achieved through the means of “therapeutic exposure,” followed by its modification through the incorporation of corrective information.
An important matter for research concerns whether persistent emotional responses to extreme traumatic events obey the same laws of habituation and extinction assumed to be the curative factors in the therapeutic exposure treatment of disorders such as phobias. There is evidence that a critical element in the successful treatment of PTSD is adequate duration and intensity of exposure to traumatic content. Although anecdotal and case reports suggest that PTSD does respond to exposure treatment, the clinician invariably encounters PTSD patients who complain that talking about their trauma makes them worse. Whether to persist with exposure in the face of such patient complaints can be one of the most difficult decisions facing the therapist.

The plethora of medications currently in use in the treatment of PTSD, including tricyclics, monoamine oxidase inhibitors, benzodiazepines, lithium carbonate, neuroleptics, beta-adrenergic blockers, and alpha-adrenergic agonists, clearly indicates that the superior efficacy of none has been established. That several of these agents functionally affect CNS monoamine activity is of theoretical interest.

CONCLUSIONS

Perhaps the most striking feature of PTSD emotional networks is their timelessess. This is most dramatically illustrated by the flashback, in which the patient may be observed to relive an event of many years ago, as if it were recurring in the present. Even in PTSD patients without flashbacks, the clinician is often struck by the uncanny present reality of the event in the patient's mental life, as if it had undergone what Thomas Mann has called "hermetic enchantment within the timeless." In this sense, the therapeutic task in PTSD may be viewed as helping time to bring about change, enabling the patient to live fully in the present.

REFERENCES