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Use of Neuropsychological Assessment in Posttraumatic Stress Disorder

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This article addresses issues related to neuropsychological assessment in posttraumatic stress disorder (PTSD). Specifically, it is proposed that neuropsychological evaluation offers valuable methods for objectively assessing complaints of cognitive dysfunction in patients with this disorder. Various psychological and organic conditions often associated with PTSD are discussed and their impact on cognitive status is reviewed. The general conditions for using neuropsychological testing with PTSD patients are outlined. The article also includes an overview of future assessment directions in this field, emphasizing the diversity of variables associated with PTSD and how they are likely to affect both clinical presentation and related test performance.

Posttraumatic stress disorder (PTSD) is a complex disorder that requires use of comprehensive evaluation procedures to assess it (Wolfe & Keane, in press; Wolfe, Keane, Lyons, & Gerardi, 1987). On the basis of evaluation models, one especially important area to consider is the assessment of cognitive status. Reports from patients with PTSD often indicate frequent complaints about a variety of cognitive disturbances, including memory, learning, attention, and concentration difficulties. Deficits in planning, organization, and judgment have also been noted during clinical evaluation. For some individuals, these difficulties represent a source of considerable interference in their daily functioning. Although the relationship between neuropsychological deficits and disorders of mood appears complex (Caine, 1986; Lishman, 1978), the frequency of patients' complaints suggest that detailed cognitive assessment may help define the nature of these symptoms and clarify their contribution to general psychological functioning.

Several other sources suggest that evaluation of cognitive factors may play an important role in PTSD. A range of cognitive symptoms are described in current diagnostic criteria for the disorder, including memory disturbance, psychogenic amnesia, and concentration problems (American Psychiatric Association, 1987). Although these deficits span two major diagnostic categories for PTSD—numbing/avoidance and physiological dysregulation—there are few empirical data available on the definitive existence or the scope of these deficits. Hence, to date, reports of these changes have been used primarily for descriptive and classification purposes. Empirical evidence of cognitive changes in PTSD would contribute to efforts at discriminating the disorder from closely related syndromes (e.g., major depression), helping to resolve questions of diagnostic validity (Wolfe & Keane, 1990).

More complex models of the etiology of PTSD also implicate cognitive factors and call for comprehensive assessment of neuropsychological functions. Although earlier models relied heavily on behavioral formulations (Keane, Fairbank, Caddell, Zimering, & Bender, 1985), newer models of the disorder reflect a growing interest in the contribution of information-processing, physiological, and psychobiological factors (e.g., Blanchard, Kolb, Pallmeyer, & Gerardi, 1982; Krystal et al., 1989; Watson, Hoffman, & Wilson, 1988). These models are predicated on the mental processing of information and traumatic stimuli in the determination of the disorder's phenomenology. Data from a series of biologically oriented PTSD studies in both clinical and preclinical populations strongly suggest that cognitive functions are of distinct importance for understanding how psychological and biological factors converge in the genesis and mediation of this disorder. As a result, psychological evaluations that systematically address cortically mediated functions can contribute to the formulation of more comprehensive models of PTSD.

A final area in which cognitive assessment may be relevant to PTSD is in evaluation of the interface between neurological insult and subsequent cognitive and behavioral stress-related symptoms. Because trauma can occur under a broad range of external conditions, PTSD may co-occur with overt brain damage in some cases. These instances make the evaluation of cognitive status especially critical. For example, an individual suffering a head injury may incur neurological damage and accompanying cortical dysfunction; these may produce isolated performance deficits or a combination of psychological and behavioral alterations. Depending on the nature and location of the insult, these changes may be exclusively brain-based or may reflect secondary emotional reactions to the experience of trauma. In some cases, neurological damage itself will directly

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affect the ability of the individual to deal with the emotional aftermath that follows trauma. Hence, a number of situations can arise in which physical and psychological components exert distinct effects on performance or where they combine to influence diagnostic presentation or outcome. In both of these cases, neuropsychological assessment is potentially useful in sifting through the myriad of behavioral and cognitive symptoms and in linking clinical presentation to a particular etiology.

Application of neuropsychological assessment may be useful as one component of systematic clinical and research exploration of the etiology and expression of stress-related phenomena. On a practical level, neuropsychology can enhance diagnostic acumen by delineating whether certain cognitive features are potentially associated with different forms and degrees of life trauma. By linking certain brain functions to behavioral and emotional correlates (Heilman & Valenstein, 1985), findings from neuropsychological assessment can also guide treatment planning. For example, in cases in which abilities to recall and retain information about traumatic experiences are required (Lyons & Keane, 1989), cognitive assessment offers data about the individual's capacity to function in those areas. Beyond its practical application, careful cognitive assessment can contribute to theoretical conceptualizations of PTSD. On this level, data from cognitive evaluations can assist in the development and testing of models integrating behavioral, psychodynamic, and psychobiological constructs because they are increasingly defined in this disorder.

The applications proposed here raise various questions for conducting assessments in PTSD. First, can cognitive dysfunction be demonstrated in patients with PTSD and, if so, what are its characteristics? Do the cognitive profiles of PTSD patients differ from those of depressed and anxious patients despite some similarities in overt symptomatology? Second, under what behavioral, biological, or environmental conditions do cognitive changes typically appear? What is the role of discernible organic insult; if it is present, how will the contribution of psychological trauma be assessed? Third, is cognitive performance related to the presence of particular PTSD symptom patterns (e.g., reexperiencing phenomena)? Finally, what are the diagnostic and treatment implications of alterations in cognitive status? How do findings from neuropsychological assessment potentially affect diagnosis and treatment of this disorder?

In raising these questions, this article reviews possible roles for neuropsychological assessment in PTSD and discusses how its use can advance both applied and scientific knowledge in this area. An argument is made for the fact that, even in the absence of overt brain damage, patients with PTSD may benefit from neuropsychological examination as a way of improving conceptualization of overall functional abilities and for enhancing selection of treatment formats. Certain distinctive factors associated with trauma disorders are reviewed and their effects on performance are discussed. The implications of various psychobiological, neurological, and cognitive perspectives for PTSD assessment are specifically explored. Although the general use of neuropsychological assessment is the focus of this article, consideration is also given to the evaluation of certain cortical functions, including planning, organization, attention, memory, and learning, for their role in the acquisition and manifestation of severe stress reactions.

The Psychobiology of PTSD

Overview of the Model

Current biological models of PTSD are based on Selye's seminal writings on stressor exposure (van der Kolk, Greenberg, Boyd, & Krystal, 1985) as well as Pavlov's research on the mechanisms leading to the establishment of conditioned reflexes (Silverman, 1986). A number of recent preclinical and clinical studies (Anisman, deCatanzaro, & Remington, 1978; Anisman, Ritch, & Sklar, 1981; Krystal, 1978; van der Kolk, Boyd, Krystal, & Greenberg, 1984) suggest that distinct behavioral and neurobiological effects follow exposure to severe stress, particularly when the stress is uncontrollable in nature. Paradigms with laboratory animals have contributed further to the development of models of inescapable shock or stress (IS) for PTSD (van der Kolk, 1987). These theories postulate that severe uncontrollable stress triggers a preliminary response of strong alarm followed by the relatively rapid conditioning of more chronic, generalized alarm states. In animal studies reported to date, lack of control over the stressor has been shown to be an especially strong predictor of the development of postexposure behavioral and learning deficits. The appearance of these deficits, which often include impairments in escape and exploratory behaviors along with diminished motivation for the learning of new contingencies, is frequently associated with sensitization to subsequent stressors (i.e., the probability that exposure to a subsequent stressor will produce a similar pathological response; Antelman, 1988). Findings such as these implicate the central nervous system in stress disorders, probably at the level of neuronal receptors (Krystal, 1978; Pitman, 1988), and suggest that learning and behavioral performance may be associated with the induction of neuronal change. Kolb (1987) and McGaugh (1990) have described similar models, proposing that exposure to such stressors produces excessive stimulation of brain receptor sites in both animals and humans and results in observable changes in their behavior and learning. Because of the neuroanatomical pathways involved, these neuronal changes potentially have the capacity to disrupt established patterns in learning, habituation, and emotional regulation that are normally mediated at discrete cortical and limbic levels.

Other recent biological studies have focused more directly on the effects of uncontrollable stress in the dysregulation of a wide range of neurotransmitter and neuropeptide systems also implicated in cognitive and behavioral functioning (Charney & Heninger, 1986). These pathways, including the noradrenergic, dopaminergic, serotonergic, benzodiazepine, opioid, and hypothalamic-pituitary-adrenal systems, may have distinct implications for the production of certain PTSD symptoms. For example, some of the more chronic symptoms of the disorder—in particular, anxiety, insomnia, and hyperarousal—may be associated with changes in noradrenergic and benzodiazepine functions (Krystal et al., 1989; Mellman & Davis, 1985; Rainey et al., 1987). In other investigations, alterations in serotonin found after the induction of IS in animals suggest that the pathophysi-

ology of depressive and anhedonic symptoms in PTSD may relate in part to changes in serotonin function (Charney, Delgado, Price, & Heninger, 1991; Charney, Woods, Krystal, & Heninger, 1990). Studies showing that uncontrollable stress has an impact on endogenous opiate functions, especially the production of stress-induced analgesia (SIA; Pitman, van der Kolk, Orr, & Greenberg, 1990) suggest that opiate-system dysfunction may be a partial determinant of the numbing seen in many individuals following exposure to severe stress (Amit & Galina, 1986; van der Kolk et al., 1984). All of these data are consistent with the hypothesis that uncontrollable stress produces profound and long-lasting changes in several different brain neurochemical systems and that a wide variety of biological and biochemical phenomena contribute to symptoms associated with PTSD.

Implications for Cognitive Assessment

The findings discussed here have various implications for understanding etiologic models and the role of learning and cognition in PTSD. Certain findings suggest that strict acceptance of biological interpretations of PTSD are insufficient for explaining the disorder's symptomatology and its diverse presentation in humans; rather, cognitive functions appear to play a pivotal role in understanding the etiology and manifestation of stress-related phenomena. For example, clinical observations indicate that biological mechanisms do not fully explain some patterns in PTSD, especially the chronicity and delayed symptom onset often found in long-term cases. Hence, mechanisms other than biological processes seem to be operational. Second, although receptor sensitization can produce multiple behavioral effects, sensitization alone does not account for the broad generalizability of stimulus cues and responses shown by many PTSD patients long after exposure to the stressor has ended. Thus, biological models themselves raise questions of the roles of learning and cognition in the development of PTSD.

A specific assessment question raised by psychobiological studies is whether PTSD patients acquire a type of "learning dysfunction" as a result of exposure to severe and typically uncontrollable stress. This question potentially links psychobiological models to the need for cognitive psychological assessment in several ways. Animal models of noradrenergic transmission suggest the involvement of the locus coeruleus and amygdala in the mediation of uncontrollable stress. Both of these brain structures are known to be involved in learning and memory functions in humans. In addition, the locus coeruleus has been shown to innervate both cortical and limbic areas directly involved in the perception and discrimination of meaningful and specifically fear-related stimuli (Redmond, 1979; Sara, 1985), and the amygdala has been implicated in both aversive learning paradigms and in the integration of information across multiple channels (Krystal et al., 1989; Mellman & Davis, 1985; Rainey et al., 1987). Together, functioning of these brain systems suggests a distinct role for cognitive, perceptual, and memory processes in the genesis of certain PTSD symptoms (e.g., unwanted reexperiencing of traumatogenic stimuli) and thus the need to evaluate their functional status as components of feedback mechanisms.

Additional evidence suggests that cognitive mechanisms may be implicated in the production of assorted types of PTSD symptomatology (Weiss, Simson, Knight, & Kilts, 1987). Dysregulation in serotonergic pathways, for example, has been found in conjunction with cognitive deficits in depressive illness. The presence of similar serotonin alterations and depressive symptoms in PTSD raises the question of whether serotonergic changes mediate depressive behavior and cognitions following trauma (Weingartner & Silberman, 1984). Moreover, distinct changes in serotonin regulation in PTSD may be tied to the emergence of reexperiencing symptoms, one of the more treatment-resistant features of the disorder. In terms of linkages between other neurochemical systems and cognitive mechanisms in PTSD, research on brain opioids indicates that cognitive processes in humans play a critical role in determining production of stress-induced analgesia (SIA; Amit & Galina, 1986), a biobehavioral phenomenon found in PTSD. It has been observed that cognitively mediated variables such as novelty, level of behavioral control, and use of suggestion predict the appearance of SIA beyond characteristics of the aversive stimulus alone (Melzack & Wall, 1982). Hence, although centrally mediated biochemical changes play a role in the evolution of PTSD, data indicate that multiple cognitive processes, including learning, memory, perception, and appraisal, are highly influential in the formation of reactions to severe stressors. Consequently, these functions require careful assessment and monitoring for both descriptive and diagnostic purposes.

The Interaction of Physical and Psychological Trauma

Findings From Neurological Studies

Studies of cases in which there are physical components of trauma have yielded equivocal results on the influence of cognitive factors in PTSD. Certainly in cases in which there is known cortical or subcortical damage, changes in emotional state have often been shown to relate distinctively to the area and type of brain damage and less directly to the stressor experience per se (Starkstein, Robinson, & Price, 1987). Nonetheless, the interaction between psychological and physical components of trauma may be subtle and worthy of consideration because of the large number of cases in which both types of insults occur.

Studies on individuals suffering behavioral and cortical trauma under conditions of extreme physical hardship provide some direction about potential uses of cognitive assessment in PTSD. Data from a series of studies conducted on former prisoners of war and civilian refugees have yielded some evidence of distinct alterations in memory, learning, and concentration abilities following conditions of incarceration or torture (Klonoff, McDougall, Clark, Kramer, & Horgan, 1976; Kral, Pazder, & Wigdor, 1967). These findings suggest that central nervous system compromise can occur as a function of multiple forms of brain insult during captivity and that these may contribute to long-lasting neuropsychological dysfunction (Arthur, 1982; Goldfeld, Mollica, Pesavento, & Faraone, 1988; White, 1983). Although the contribution of psychological traumatization to cognitive performance is difficult to ascertain, the possibility exists that deficits reflect a combination of factors and require

more systematic studies controlling for the impact of selective brain damage.

To test hypotheses on the relationship of sustained severe trauma and cognitive dysfunction, Sutker, Allain, and Winstead (1987) examined the performances of former prisoners of war (POWs) on standardized intellectual tests (e.g., the Wechsler Adult Intelligence Scale–Revised [WAIS–R Wechsler, 1981]). They found performance deficits consistent with underlying dysfunction in concentration, attention, and memory, suggesting the possibility of subtle central nervous system sequelae from the prisoner of war (POW) experience. Sutker, Galina, West, and Allain (1990) subsequently tested former POWs from the Korean Conflict and World War II on more extensive standardized neuropsychological measures of intelligence and memory. Dividing veterans into groups on the basis of severity of weight loss during confinement, these researchers found that veterans who lost more than 35% of their precaptivity body weight showed significant cognitive compromise on tasks involving attentional, concentration, memory, and problem-solving skills compared with nonincarcerated combat control subjects. By contrast, POWs who had low weight loss differed from control subjects only on measures of immediate memory recall. These findings suggest the effects of malnutrition on central nervous system functioning with a differential impact determined by the severity of the physical stressor. Although effects of psychological traumatization were not determined, it is possible that they contributed to the clinical picture along with the more obvious impact of biological stressors. This notion is consistent with findings from a study by Speed, Engdahl, Schwartz, and Eberly (1989), who noted two significant predictors of PTSD in an ex-POW sample: (a) the proportion of body weight lost during captivity and (b) the experience of torture during incarceration. Although certain aspects of torture are likely to influence biological status, these data nonetheless suggest that additional consideration of the combined effect of psychological and physical trauma is needed.

In another study, Levy (1988) administered a series of standardized neuropsychological tests of immediate and delayed memory and perceptual organization to Vietnam veterans. Compared with a matched veteran control group, veterans exposed to the herbicide Agent Orange showed increased cognitive impairment and a correspondingly higher rate of PTSD. These results conflict with findings from a recent Centers for Disease Control study (1988) that found no adverse impact of dioxin exposure on cognitive performance. However, it is likely that a number of difficult-to-obtain measures were not systematically controlled in these studies (e.g., premorbid cognitive abilities, emotional responses to trauma) and require attention before definitive conclusions can be drawn on the interaction between psychological trauma and organic insult.

Assessment Considerations

One particular concern in the cognitive assessment of PTSD patients is that traumatic brain injury may mimic, rather than cause, symptoms of PTSD or related mood disorders. Studies that do not control for differential effects of brain damage through the inclusion of groups with varying lesions sites (as well as non-brain-damaged control subjects) will be likely to have difficulty in addressing this issue. Furthermore, in instances of organic amnesia, cases of apparent PTSD may represent organically derived depression, psychological responses, or both, constituting secondary reactions to loss of behavioral function. Conversely, cases involving mild traumatic brain injury (e.g., minor vehicular accidents) in which no loss of consciousness or amnesia resulted may be more conducive to assessing directly the relationship of cognitive changes following life trauma (Levin, Eisenberg, & Benton, 1989).

The influence of more subtle organic factors on cognitive performance in PTSD also needs to be considered. Recent research shows that patients with PTSD have high rates of comorbid disorders, particularly substance abuse (Keane, Gerardi, Lyons, & Wolfe, 1988). Depending on its severity, the substance abuse disorder may produce global cognitive disturbance or a more limited impairment in the recall of traumatic memories. In addition, patients with these comorbid disorders may be at increased risk for other forms of physical damage affecting cognition (e.g., head injury secondary to alcoholic black-outs). All of these factors suggest that clinicians need to be especially conscientious in obtaining background histories from trauma survivors before basing their findings on any one etiology.

Information-Processing Models and Assessment in PTSD

Review of Models

Information processing is an area that provides conceptual models for PTSD and directly implicates cognitive processes in the establishment–acquisition and maintenance of this disorder. Information processing emphasizes the utility of assessing both quantitative and qualitative components of cognitive processes in PTSD. Research in information processing has recently begun to demonstrate that, independently of physical insult, certain perceptual and processing parameters may be specifically affected in trauma disorders.

One cornerstone of the information-processing model in anxiety disorders is the establishment of an affectively charged, semantic memory network that preferentially processes trauma- or threat-related stimuli and may directly contribute to the persistence of symptomatology (Chemtob, Roitblat, Hamada, Carlson, & Twentyman, 1988; Foa, Steketee, & Rothbaum, 1989; Pitman, 1988). Based on Lang's (1985) theory of emotion, current information-processing theories in PTSD predict that this network comprises certain types of information, including details of the context in which trauma-relevant stimuli are present, response events that mediate arousal and action, and information concerning the meaning of stimulus and response acts.

In information-processing theory, the associational network is thought to form the basis for the recall of functional experience in humans. When extreme threat or fear (e.g., panic) is present, this network becomes "colored" by distinctive individualized associations to fear-potentiated cues. It is posited that this model of interconnected informational modules is readily activated in PTSD (i.e., fear-based conditioning and generalization generate multiple-threat connotations among previously neutral associations). These cognitive associations are main-

tained and probably reinforced by a temporary reduction in anxiety afforded by the operation of selective processing, attentional bias, and constant autonomic readiness (Gray, 1982). Hence, an elaborate cognitive network may exist that is readily potentiated following trauma.

Recent studies on information processing in PTSD have generated some empirical evidence that PTSD patients selectively process and retain fear- or threat-related associations in a manner different from that of normal control subjects and those with other psychiatric disorders (McNally et al., 1987; Trandel & McNally, 1987). This selectivity has been observed under conditions involving presentation of autobiographical and "generic" trauma stimuli (Pitman, Orr, Forgul, de Jong, & Claiborn, 1987). The data substantiate the notion that distinct semantic networks exist in PTSD and that they are involuntarily activated under certain proprioceptive and interoceptive cues.

To date, studies of baseline cognitive functioning in PTSD have not succeeded in yielding significant differences among these patients. However, application of "higher order" tests such as the Stroop (Lezak, 1983) have preliminarily demonstrated subtle cognitive-processing changes in traumatized patients in a manner that differs from the performance of normal subjects and patients with general anxiety disorders. Specifically, PTSD patients appear to respond to threat-relevant content with greater reactivity and decreased latencies, processing threat-content cues preferentially, compared with neutral stimuli (see Litz & Keane, 1989, for review). Findings such as these suggest that the use of specialized neuropsychological tests, especially those requiring selective perceptual discrimination and inhibition, can serve as valuable methods for differentiating among some patient groups as well as for assessing how certain types of information are selectively processed following trauma. Furthermore, use of these tests can provide quantitative and qualitative data on a number of high-level processing abilities, permitting comparison of patients' performance with normative data from nonpsychiatrically disordered populations as well as those with defined neurological damage.

The Function of Memory in PTSD

Existing models of memory disorders (Cermak, 1982) offer other rationales for considering the application of neuropsychological assessment in PTSD. Similar to information-processing models, memory research provides valuable methodologies for defining cognitive characteristics potentially associated with stressor exposure, especially as they relate to recall, amnesia, and intrusive imaginal experiences. Extensive memory research with various neurologically disordered patients has demonstrated the existence of multiple memory systems in the brain (Atkinson & Shiffrin, 1978; Squire, 1987; Tulving, 1983), which process and integrate information at a number of levels with varying degrees of verbal and perceptual elaboration. Operation of these systems now appears to be susceptible to modification by an array of factors ranging from cortical and subcortical neurochemical elements to destructive brain lesions to external environmental conditions.

One relevant application of memory functions and their assessment relates to amnesia following intense emotional trauma (Schacter & Kihlstrom, 1989). Assessment methods within this area—specifically, recall and recognition paradigms—may be used to examine the effects of trauma on mental processing and retention. Yet, few empirical investigations have actually studied the phenomenon of psychogenic amnesia or have applied methodologies from organic amnesias to the study of trauma-related memory problems. Instead, explanations of memory dysfunction or forgetting have often relied on existing traditional psychiatric and dynamic interpretations (Nemiah, 1979; Schacter & Tulving, 1982). The possibility exists, however, that functional amnesias reflect selective effects of stressor experiences on certain memory systems. If this is the case, neuropsychological assessment may be used to explore basic parameters of memory processes as well as the conditions under which they are idiosyncratically stimulated or redirected. Thus, in PTSD, assessment of several types of memory may be relevant.

In the past, clinically based studies of differences in recall and recognition skills have aided differential diagnosis in a range of neurological and psychiatric disorders with known or suspected memory disturbance (Butters, Wolfe, Martone, Granholm, & Cermak, 1985; Wolfe, Granholm, Butters, Saunders, & Janowsky, 1987). The assessment of short- and long-term recall and recognition abilities in traumatized patients may provide useful indexes of basic cortical integrity and aid treatment planning. More innovative assessment formats involve tests of explicit versus implicit memory (Graf & Schacter, 1985; Schacter, 1987). *Explicit memory* represents the ability to recollect consciously earlier experiences; *implicit memory*, on the other hand, refers to the retrieval of such information without conscious intention or awareness on the part of the subject. Explicit memory can be assessed using traditional tests of recall and recognition, whereas implicit memory requires testing under conditions that obviate the subject's awareness of the topic or stimulus being investigated (e.g., word-stem completion tasks). Research has shown that patients with clear-cut, organically determined, amnestic syndromes are, in fact, capable of certain types of recognition memory despite the presence of significant deficits in explicit recall (Schacter, 1987; Squire, 1987; Weiskrantz, 1987).

Recently, studies on functional amnesias indicate that similar patterns of memory dysfunction exist in psychologically distressed patients who have experienced traumatizing events. In these patients, implicit memory abilities appear to be strikingly preserved despite the presence of explicit memory deficits (Kaszniak, Nussbaum, Berren, & Santiago, 1988). Such findings suggest the possibility of a dissociation between these two memory systems in patients without neurological lesions. On the basis of this work, some authors (Schacter & Kihlstrom, 1989) have proposed that similar memory assessment paradigms be used to search for dissociations between explicit and implicit memory systems in disorders such as PTSD, in which recall, processing, and retrieval of certain types of information are purportedly disrupted. These assessments could provide important clinical information on the fluctuating course of intrusive traumatic recollections as well as on the conditions under which trauma-related stimuli are seemingly perceived or "forgotten." Neuropsychological paradigms can thus address how cortical processes relate to a variety of emotional experiences. In cases in which structural cortical insult may have oc-

curred, these assessments can combine information on the cortical integrity of PTSD patients with data on the effects of explicit and implicit memory stores.

Further Issues in Assessment

As reviewed here, psychobiological and behavioral models of PTSD predict that cognitive experience is both mediated by, and contributes to, regulatory functions of the central nervous system. Neuropsychological evaluation offers the opportunity to investigate conditions under which cognitive abilities are affected by extreme emotional experience and the ways in which these changes mediate subsequent behavioral response. Specifically, neuropsychological assessment can be used to evaluate (a) how basic mental processes (e.g., problem-solving, attention, and memory) are affected in PTSD, and (b) whether changes in these functions play a part in the etiology or maintenance of symptoms found in this disorder.

One strength of neuropsychological assessment is that it does not require an emphasis on diagnosis to be useful; in fact, neuropsychological paradigms can be used to assess brain-based cognitive processes relatively independently from diagnostic status. Inferences can be made about functional brain processes even in the absence of observable pathology, allowing detection of seemingly subtle impairments even when evidence of gross dysfunction is lacking. Neuropsychological paradigms therefore allow for qualitative as well as quantitative examination of processes or mechanisms associated with experience as well as investigation into the existence of potential deficits or abnormalities in a given functional area (Kaplan, 1990).

Certain conditions or uses for neuropsychological testing and their limitations should be reiterated. These include the use of neuropsychological assessment (a) to determine baseline functioning after psychological or physical trauma, or their combination; (b) to determine functioning under conditions of changes in symptomatology (e.g., heightened arousal); (c) to assess cognitive capacities for certain forms of PTSD treatment (e.g., imaginal or exposure-based approaches); (d) to conduct experimental investigations between patients with this and potentially related disorders; and (e) to conduct experimental analyses of cognitive functions and their relationship to purported neurobiological components of PTSD (e.g., serotonin dysregulation).

Limitations of the above uses involve a number of issues. First, baseline functions in PTSD, like symptom pictures, may be influenced by a variety of psychological, neurological, and metabolic factors. As previously discussed, numerous factors can affect their presentation. Furthermore, an apparent correlation between psychological disturbance and concomitant cognitive disturbance does not imply that the two are causally related (Caine, 1986). Hence, causal relationships between PTSD symptoms and cognitive changes may be difficult to ascertain. Second, PTSD is a phasic disorder, and the appearance of cognitive deficits at a particular time may not reflect a static situation. In addition, trauma victims may present an inability to disclose full details of their background experiences; this presentation requires careful differential diagnosis of psychogenic from organic amnesias.

Investigations into neuropsychological aspects of psychiatric disorders suggest that other guidelines be kept in mind when assessing cognitive functions in relation to any psychological disturbance. First, there is some evidence that the study of a disorder per se has limitations. Research on major mood and anxiety disorders has often yielded equivocal results about cognitive and functional deficits associated with diagnoses (Berenbaum, Kerns, & Taylor, 1990, for a review) and has contributed only marginally to etiological models for those disorders. Second, like any form of assessment paradigm, neuropsychological tests have their own limitations. Even when there are obvious deficits in performance, performance-based abnormalities may have no definitive causal implication or may reflect subtle factors such as fluctuation in mood or effects of motivation. Certainly, medical and neurological status of PTSD patients are factors to be considered at all times. Furthermore, tests of many higher level neuropsychological functions (e.g., problem-solving and organizational skills), which are likely to be of interest in non-brain-damaged individuals, often do not have clear-cut localizing value. Consequently, assessment may provide information on qualitative aspects of performance unique to the individual rather than data that serves a distinct diagnostic or localizing function.

Despite these caveats, a number of other cognitive abilities apart from basic cognitive, memory, and language functions may warrant systematic evaluation in the assessment of PTSD. These include functions mediated by the frontal lobes, such as attention, set establishment and maintenance, set shifting, and general problem-solving abilities, and relate to descriptive behavioral symptoms of impairments in judgment, planning, and flexibility. Although the presence of PTSD does not necessarily imply frontal-lobe pathology, examination of frontal functions can serve as an indicator of overall cortical intactness, helping to rule out effects of earlier brain trauma that may be associated with prior life events. In terms of the PTSD diagnosis, data from frontal-functions tests can help to define the degree to which regulatory and attentional processes may be disrupted (e.g., whether affective components of the disorder cause decreased motivation; whether autonomic reactivity produces disordered attention) and whether the obtained profiles differ from those found in depressed, non-PTSD patients. Widely used neuropsychological tests such as the Wisconsin Card Sorting Test (Lezak, 1983) and others involving timed or complex combinations of motor and cognitive skills (e.g., Trailmaking Test; Army Individual Test Battery, 1944) can potentially clarify the impact of behavioral and affective symptoms on cognition and whether they are specific to the emotional aftermath of trauma, accompany focal changes in higher brain functions, or are generic to a variety of psychiatric disturbances. In these cases, repeated testing under varying conditions of PTSD symptoms (e.g., hyperarousal) can also help to define whether evidence of cognitive alterations represents a static or state-dependent phenomenon.

Conclusion

This article suggests that the clinical and research description of stress-related symptoms of PTSD may be enhanced by testing of cognitive functions. In the majority of cases in which the traumatic event has been psychological in nature, informa-

tion gleaned from cognitive assessment can provide both the clinician and the researcher with baseline estimates of the individual's ability to process, organize, and retain both nonmeaningful and meaningful material. These data may inform the psychologist about the formation of particular symptoms and the patient's capacity to discuss and modify them through both insight-oriented and more behaviorally based exposure therapies. Furthermore, knowledge of cognitive status can affect treatment planning that may call on the patient's ability to analyze and modify responses to a complex and changing external environment. Where traumatic exposure is associated with central nervous system damage, neuropsychological evaluation can help to distinguish symptoms that are more emotionally based from those that represent sequelae of known brain damage. Assessment of both quantitative and qualitative changes in cognitive performance can aid in the determination of multiple diagnoses and define the need for a variety of rehabilitation efforts. By adding to the available evaluation methodologies, neuropsychological assessment can add significantly to advances in the clinical work and science of PTSD.

References

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

Amit, Z., & Galina, Z. H. (1986). Stress-induced analgesia: Adaptive pain suppression. *Psychological Review, 66,* 1091–1120.

Anisman, H., deCatanzaro, D., & Remington, G. (1978). Escape performance following exposure to inescapable shock: Deficits in motor response maintenance. *Journal of Experimental Psychology: Animal Behavior Processes, 4,* 197–218.

Anisman, H., Ritch, M., & Sklar, L. S. (1981). Noradrenergic and dopaminergic interactions in escape behavior: Analysis of uncontrollable stress effects. *Psychopharmacology, 74,* 263–268.

Antelman, S. M. (1988). Time-dependent sensitization as the cornerstone for a new approach to pharmacotherapy: Drugs as foreign/stressful stimuli. *Drug Development Research, 14,* 1–30.

Army Individual Test Battery. (1944). *Manual of directions and scoring.* Washington, DC: War Department, Adjutant General's Office.

Arthur, R. J. (1982). Psychiatric syndromes in prisoner of war and concentration camp survivors. In C. T. H. Friedman & R. A. Faguet (Eds.), *Extraordinary disorders of human behavior* (pp. 47–63). New York: Plenum Press.

Atkinson, R. C., & Shiffrin, R. M. (1978). Human memory: A proposed system and its control processes. In K. W. Spence & J. T. Spence (Eds.), *The psychology of learning and motivation: Vol. 2* (pp. 89–105). New York: Academic Press.

Berenbaum, S. A., Kerns, K., & Taylor, M. A. (1990). A neuropsychological perspective on comorbid disorders. In J. D. Maser & C. R. Cloninger (Eds.), *Comorbidity of mood and anxiety disorders.* Washington, DC: American Psychiatric Press.

Blanchard, E. B., Kolb, L. C., Pallmeyer, T. P., & Gerardi, R. J. (1982). The development of a psychophysiological assessment procedure for post-traumatic stress disorder in Vietnam veterans. *Psychiatric Quarterly, 54,* 220–229.

Butters, N., Wolfe, J., Martone, M., Granholm, E., & Cermak, L. S. (1985). Memory disorders associated with Huntington's disease: Verbal recall, verbal recognition, and procedural memory. *Neuropsychologia, 23,* 729–743.

Caine, E. D. (1986). The neuropsychology of depression: The pseudodementia syndrome. In I. Grant & K. M. Adams (Eds.), *Neuropsychological assessment of neuropsychiatric disorders* (pp. 221–243). New York: Oxford University Press.

Centers for Disease Control. (1988). Health status of Vietnam veterans. *Journal of the American Medical Association, 259,* 2701–2724.

Cermak, L. S. (Ed.). (1982). *Human memory and amnesia.* Hillsdale, NJ: Erlbaum.

Charney, D. S., Delgado, P. L., Price, L. H., & Heninger, G. R. (1991). The receptor sensitivity hypothesis of antidepressant action: A review of antidepressant effects on serotonin function. In S. Brown & H. van Praag (Eds.), *The role of serotonin in psychiatric disorders* (pp. 27–57). New York: Brunner/Mazel.

Charney, D. S., & Heninger, G. R. (1986). Abnormal regulation of noradrenergic function in panic disorder. *Archives of General Psychiatry, 43,* 1042–1058.

Charney, D. S., Woods, S. W., Krystal, J. H., & Heninger, G. R. (1990). Neurobiological mechanisms of human anxiety. In R. Pohl & D. Gershon (Eds.), *Bases of psychiatric treatment* (pp. 242–283). New York: Karger.

Chemtob, C., Roitblat, H. C., Hamada, R. S., Carlson, J. G., & Twentyman, C. T. (1988). A cognitive action theory of post-traumatic stress disorder. *Journal of Anxiety Disorders, 2,* 253–275.

Foa, E. B., Steketee, G., & Rothbaum, B. (1989). Behavioral/cognitive conceptualizations of post-traumatic stress disorder. *Behavior Therapy, 20,* 155–176.

Goldfeld, A. E., Mollica, R. F., Pesavento, B. H., & Faraone, S. V. (1988). The physical and psychological sequelae of torture: Symptomatology and diagnosis. *Journal of the American Medical Association, 259,* 2725–2729.

Graf, P., & Schacter, D. L. (1985). Implicit and explicit memory for new associations in normal and amnesic subjects. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 11,* 501–518.

Gray, J. A. (1982). *The neuropsychology of anxiety.* New York: Oxford University Press.

Heilman, K. M., & Valenstein, E. (1985). *Clinical neuropsychology* (2nd ed.). New York: Oxford University Press.

Kaplan, E. (1990). The process approach to neuropsychological assessment of psychiatric patients. *Journal of Neuropsychiatry and Clinical Neurosciences, 2,* 72–87.

Kaszniak, A. W., Nussbaum, P. D., Berren, M. R., & Santiago, J. (1988). Amnesia as a consequence of male rape: A case report. *Journal of Abnormal Psychology, 97,* 100–104.

Keane, T. M., Fairbank, J. A., Caddell, J. M., Zimering, R. T., & Bender, M. E. (1985). A behavioral approach to assessing and treating PTSD in Vietnam veterans. In C. R. Figley (Ed.), *Trauma and its wake.* New York: Brunner/Mazel.

Keane, T. M., Gerardi, R. J., Lyons, J. A., & Wolfe, J. (1988). The interrelationship of substance abuse and PTSD: Epidemiological and clinical considerations. In M. Galanter (Ed.), *Recent developments in alcoholism: Vol. 6.* New York: Plenum Press.

Klonoff, H., McDougall, G., Clark, C., Kramer, P., & Horgan, J. (1976). The neuropsychological, psychiatric, and physical effects of prolonged and severe stress: Thirty years later. *Journal of Nervous and Mental Disease, 163,* 246–252.

Kolb, L. C. (1987). A neuropsychological hypothesis explaining post-traumatic stress disorders. *American Journal of Psychiatry, 144,* 989–995.

Kral, V. A., Pazder, L. H., & Wigdor, B. T. (1967). Long-term effects of a prolonged stress experience. *Canadian Psychiatric Association Journal, 12,* 175–181.

Krystal, H. (1978). Trauma and affects. *Psychoanalytic study of the child, 33,* 81–116.

Krystal, J. H., Kosten, T. R., Southwick, S., Mason, J. W., Perry, B. D., & Giller, E. L. (1989). Neurobiological aspects of PTSD: Review of clinical and preclinical studies. *Behavior Therapy, 20,* 177–198.

Lang, P. J. (1985). The cognitive psychophysiology of emotions: Fear and anxiety. In A. H. Tuma & J. Maser (Eds.), *Anxiety and the anxiety disorders.* Hillsdale, NJ: Erlbaum.

Levin, H. S., Eisenberg, H. M., & Benton, A. L. (1989). *Mild head injury.* New York: Oxford University Press.

Levy, C. L. (1988). Agent orange exposure and posttraumatic stress disorder. *Journal of Nervous and Mental Disease, 176,* 242–245.

Lezak, M. D. (1983). *Neuropsychological assessment* (2nd ed.). New York: Oxford University Press.

Lishman, W. A. (1978). *Organic psychiatry: The psychological consequences of cerebral disorder.* Oxford, United Kingdom: Blackwell Scientific Publications.

Litz, B. T., & Keane, T. M. (1989). Information processing in anxiety disorders: Application to the understanding of post-traumatic stress disorder. *Clinical Psychology Review, 9,* 243–257.

Lyons, J. A., & Keane, T. M. (1989). Implosive therapy for the treatment of combat-related PTSD. *Journal of Traumatic Stress, 2,* 137–152.

McGaugh, J. L. (1990). Significance and remembrance: The role of neuromodulatory systems. *Psychological Science, 1,* 15–25.

McNally, R. J., Luedke, D. L., Besyner, J. K., Peterson, R. A., Bohn, K., & Lips, O. J. (1987). Sensitivity to stress relevant stimuli in post-traumatic stress disorder. *Journal of Anxiety Disorders, 1,* 105–116.

Mellman, T. A., & Davis, G. C. (1985). Combat-related flashbacks in post-traumatic stress disorder: Phenomenology and similarity to panic attacks. *Journal of Clinical Psychiatry, 46,* 379–382.

Melzack, R., & Wall, P. (1982). *The challenge of pain.* Middlesex, U.K.: Penguin.

Nemiah, J. (1979). Dissociative amnesia: A clinical and theoretical recantioration. In J. F. Kihlstrom & F. J. Evans (Eds.), *Functional disorders of memory* (pp. 303–324). Hillsdale, NJ: Erlbaum.

Pitman, R. K. (1988). Post-traumatic stress disorder, conditioning, and network theory. *Psychiatric Annals, 18,* 182–189.

Pitman, R. K., Orr, S. P., Forgul, D. F., de Jong, J. B., & Claiborn, J. M. (1987). Psychophysiologic assessment of post-traumatic stress disorder imagery in Vietnam combat veterans. *Archives of General Psychiatry, 44,* 970–975.

Pitman, R. K., van der Kolk, B. A., Orr, S. P., & Greenberg, M. S. (1990). Naloxone-reversible analgesic response to combat related stimuli in posttraumatic stress disorder. *Archives of General Psychiatry, 47,* 541–544.

Rainey, J. M., Aleen, A., Ortiz, A., Yeragami, V., Pohl, R., & Bereliou, R. (1987). A laboratory procedure for the induction of flashbacks. *American Journal of Psychiatry, 144,* 1317–1319.

Redmond, D. E., Jr. (1979). New and old evidence for the involvement of a brain noradrenergic system in anxiety. In W. E. Fann, I. Karacan, A. D. Pakorney, & R. L. Williams (Eds.), *Phenomenology and treatment of anxiety* (pp. 153–203). New York: Spectrum.

Sara, S. J. (1985). The locus coeruleus and cognitive function: Attempts to relate noradrenergic enhancement of signal/noise in the brain to behavior. *Physiological Psychology, 13,* 151–162.

Schacter, D. L. (1987). Implicit memory: History and current status. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 13,* 501–518.

Schacter, D. L., & Kihlstrom, J. F. (1989). Functional amnesia. In F. Boller & J. Grafman (Eds.), *Handbook of neuropsychology.* Amsterdam: Elsevier Publications.

Schacter, D. L., & Tulving, E. (1982). Memory, amnesia, and episodic/semantic distinction. In R. L. Isdacson & N. E. Spear (Eds.), *The expression of knowledge* (pp. 33–65). New York: Plenum Press.

Silverman, J. J. (1986). Post-traumatic stress disorder. *Advances in psychosomatic medicine, 16,* 115–140.

Speed, N., Engdahl, B., Schwartz, J., & Eberly, R. (1989). Posttraumatic stress disorder as a consequence of the POW experience. *Journal of Nervous and Mental Disease, 177,* 147–153.

Squire, L. R. (1987). *Memory and brain.* New York: Oxford University Press.

Starkstein, S. E., Robinson, R. G., & Price, T. R. (1987). Comparison of cortical and subcortical lesions in the production of poststroke mood disorders. *Brain, 110,* 1045–1059.

Sutker, P. B., Allain, A. N., & Winstead, D. K. (1987). Cognitive performances in former WWII and Korean-Conflict POWs. *VA Practitioner, 4,* 77–78, 81, 85.

Sutker, P. B., Galina, Z. H., West, J. A., & Allain, A. N. (1990). Trauma-induced weight loss and cognitive deficits among former prisoners of war. *Journal of Consulting and Clinical Psychology, 58,* 323–328.

Trandel, D. V., & McNally, R. J. (1987). Perception of threat cues in post-traumatic stress disorder: Semantic processing without awareness? *Behavior Research and Therapy, 25,* 469–476.

Tulving, E. (1983). *Elements of episodic memory.* Oxford, United Kingdom: Oxford University Press.

van der Kolk, B. A. (1987). *Psychological trauma.* Washington, DC: American Psychiatric Press.

van der Kolk, B. A., Boyd, H., Krystal, J., & Greenberg, M. (1984). Post-traumatic stress disorder as a biologically based disorder: Implications of the animal model of inescapable shock. In B. A. van der Kolk (Ed.), *Post-traumatic stress disorder: Psychological and biological sequelae* (pp. 123–134). Washington, DC: American Psychiatric Press.

van der Kolk, B. A., Greenberg, M., Boyd, H., & Krystal, J. (1985). Inescapable shock, neurotransmitters and addiction to trauma: Toward a psychobiology of post traumatic stress. *Biological Psychiatry, 20,* 314–325.

Watson, L. P. H., Hoffman, L., & Wilson, G. V. (1988). The neuropsychiatry of post-traumatic stress disorders. *British Journal of Psychiatry, 152,* 164–173.

Wechsler, D. A. (1981). *Wechsler Adult Intelligence Scale–Revised.* New York: Psychological Corporation.

Weingartner, H., & Silberman, E. (1984). Cognitive changes in depression. In R. M. Post & J. C. Ballenger (Eds.), *Neurobiology of the mood disorders* (pp. 121–135). Baltimore: Williams and Wilkins.

Weiskrantz, L. (1987). Neuroanatomy of memory and amnesia: A case for multiple memory systems. *Human Neurobiology, 6,* 93–106.

Weiss, J. M., Simson, P. E., Knight, J. A., & Kilts, C. D. (1987). Hyperresponsivity of locus coeruleus is associated with stress-induced behavioral depression. *Social Neuroscientific Abstracts, 13,* 32.

White, N. S. (1983). Posttraumatic stress disorder [Letter to the editor]. *Hospital and Community Psychiatry, 34,* 1061–1062.

Wolfe, J., Granholm, E., Butters, N., Saunders, E., & Janowsky, D. (1987). Verbal memory deficits associated with major affective disorders: A comparison of unipolar and bipolar patients. *Journal of Affective Disorders, 13,* 83–92.

Wolfe, J., Keane, T. M., Lyons, J. A., & Gerardi, R. J. (1987). Current trends and issues in the assessment of PTSD. *The Behavior Therapist, 10,* 27–32.

Wolfe, J., & Keane, T. M. (1990). The diagnostic validity of post-traumatic stress disorder. In M. Wolf & A. Mosnaim (Eds.), *Post-traumatic stress disorder: Etiology, phenomenology, and treatment.* Washington, DC: American Psychiatric Press.

Wolfe, J., & Keane, T. M. (in press). New perspectives in the assessment of combat-related post-traumatic stress disorder. In J. Wilson & B. Raphael (Eds.), *The international handbook of traumatic stress syndromes.* New York: Plenum Press.

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