

Published by:

The National Center for PTSD
VA Medical and Regional
Office Center (116D)
215 North Main Street
White River Junction
Vermont 05009-0001 USA

☎ (802) 296-5132
FTS (700) 829-5132
FAX (802) 296-5135
FTS FAX (700) 829-5135
Email: ptsd@dartmouth.edu

Subscriptions are available
from the Superintendent of
Documents, P.O. Box 371954,
Pittsburgh, PA 15250-7954.

Editorial Director
Matthew J. Friedman, MD,
PhD
Scientific Editor
Paula P. Schnurr, PhD
Managing Editor
Fred Lerner, DLS
Production
Sharon Liebert, MLS
Graphics
Margaret J. Pearson
Circulation Manager
Jan Clark

In this issue:

- A "Hot-System/
Cool-System" View of
Memory Under Stress
- Research Activities
in FY 1995
- PILOTS Update

National Center Sites
Executive Division
White River Junction
VT 05009

Behavioral Science
Division
Boston MA 02130

Clinical Laboratory
and Education Division
Menlo Park CA 94304

Clinical Neurosciences
Division
West Haven CT 06516

Evaluation Division
West Haven CT 06516

Pacific Islands Division
Honolulu, HI 96813

Women's Health Sciences
Division
Boston MA 02130

The National Center for Post-Traumatic Stress Disorder PTSD RESEARCH QUARTERLY

VOLUME 7, NUMBER 2

ISSN 1050-1835

SPRING 1996

A "HOT-SYSTEM/COOL-SYSTEM" VIEW OF MEMORY UNDER STRESS

Janet Metcalfe, PhD¹

Columbia University

W. J. Jacobs, PhD²

University of Southern California

The following review is based on an article that has been submitted for publication, "The Effects of Stress on 'Cool' and 'Hot' Memory Systems," by W.J. Jacobs and J. Metcalfe.

In the systems approach to human memory, the mind/brain is assumed to be multifaceted, with semi-autonomous specialized subsystems operating in parallel. The component subsystems function to highlight their own parts of the representational tableau, which includes the phenomenological experience, the actions, the preferences, and the memories of the person. A number of component systems have been identified or hypothesized, each with its distinctive functions, representations (both in terms of modality and format), and operating principles. Within this systems view, the mind can be likened to a chamber orchestra: Each subsystem or instrument contributes its own special qualities to the melody and harmonies of mind, providing an unparalleled complexity and richness. The music may be altered when individual instruments are either muted or amplified. Similarly, depending on the response of the individual subsystems to factors such as stress (or other factors such as age, hormonal levels, and cognitive set), different aspects of human experience may be accentuated or silenced. While there are undoubtedly many subsystems that contribute to human experience, in this summary, we shall focus on two that are of fundamental importance for our understanding of human memory under acute stress.

The first system we shall consider is the "cool" cognitive system; the second, the "hot" emotional-fear system. We proposed that the "cool" hippocampal memory system records, in an unemotional manner, well-elaborated autobiographical events, complete with their spatial-temporal context. In contrast, the "hot" amygdala system responds to unintegrated fragmentary fear-provoking features of events, which become hooked directly to fear responses. The hot system is direct, quick, highly emotional, inflexible, and fragmentary. The cool system is cognitive and complex, informationally neutral, subject to control processes, and integrated. Hot-system memories are stimulus-driven and entail a sense of reliving—more like simple responses (often fearful) than like recollections. Cool-system memories are narrative, recollective, and episodic. The person knows that the events occurred in his

personal past, and there is no sense of reliving or of mistaking the memory for a current percept. Normally, encoding in the two systems is thought to operate in parallel, with the cool system encoding the contextual panorama and the hot system contributing a "highlighting" of the specifically fear-provoking (or emotional) aspects of the experience.

One implication of this theory is that it provides a natural explanation for the often-observed phenomenon of weapon focus—the finding that people selectively attend to and remember threatening objects, such as weapons, to the detriment of more neutral objects in the scene. In addition, as Burke and colleagues (1992) have shown, they selectively encode (and show enhanced memory on) the aspects of a story that have emotional impact. Presumably the hot amygdala system contributes a special emphasis or highlighting to such objects and events. Christianson (1992) and Egeth (1994) have provided reviews of the literature on the experimental effects of emotion on eyewitness memory. One criticism of the experimental literature in this area is that experimental studies are irrelevant to understanding traumatic stress, since they are conducted under minimally stressful conditions, and that the kinds of situations studied in the laboratory are radically different from naturally occurring situations (Yuille & Cutshall, 1989).

A converging literature is accumulating that points to the separability of a hot, amygdala-based, and a cool, hippocampus-based system. LeDoux (1995) and Davis (1992), having both conducted extensive research on the amygdala, have reviewed the neural circuitry underlying fear and anxiety, showing that lesions to the amygdala in rats eliminate the expression of fear and the ability of the animals to learn conditional fear. LeDoux argues that the fear-related function of the amygdala holds in all vertebrates, including humans. He has traced the informational pathways leading into and out of the amygdala by observing the results of selective lesions on fear conditioning. One pathway is a quick, uncognitive route, in which stimulation of the amygdala results directly from the thalamus. A second pathway is more circuitous and cognitive, and involves feedback from both the hippocampus and the cortex. Results indicate that once fear is conditioned, it is virtually indelible, although the connection to the frontal lobes and other cortical regions—parts of what are here called the cool sys-

¹Address for Dr. Metcalfe: Department of Psychology, 401B Schermerhorn Hall, Columbia University, New York, NY 10027. Internet: metcalfe@paradox.psych.columbia.edu. ²Address for Dr. Jacobs: Department of Psychology SGM 501, University of Southern California, Los Angeles, CA 90089-1061. Internet: jakej@alnitak.usc.edu.



tem—allow suppression of fear responding. It follows, from this research in animals, that procedures used in the clinic, such as implosion, flooding, exposure, and systematic desensitization may inhibit the expression of fear without eliminating the underlying neural basis.

Aggleton and Passingham (1981) have summarized the literature on amygdala lesions in monkeys. Wild monkeys who have suffered bilateral amygdala lesions exhibit extremely distorted and inappropriate social behavior. They do not show normal fear responses, nor do they behave appropriately in the face of real threat. They appear to be apathetic, and soon become isolated from the tribe. Being cut off from the social structure of the group, the wild monkeys soon die. The cause appears to be maladaptive emotional responding rather than direct physical repercussions of the lesion.

Nahm et al. (1993) described a woman with selective bilateral amygdala damage resulting from Urbach-Wiethe disease. Her IQ was low average; she had no difficulty remembering new information, including new objects and new faces; she could keep track of and remember temporal relations. However, she could not learn or remember associations involving fear or reward or discern fear in facial expressions. Her social behavior also showed mild improprieties. Such a selective deficit in emotional responding is unlike the kind of amnesia seen with cool-system (hippocampal or frontal-lobe damage).

To investigate the characteristics of the cool system, we may look at the role of the hippocampus and related structures in animals and humans. Nadel et al. (1985) and Rudy and Sutherland (1994) have summarized the literature on the function of the hippocampus in rats, and related these memorial and spatial functions to the human literature. Hippocampally lesioned rats are unable to remember spatial locations. Such deficits may be attributable to a failure of the rats to encode and remember relational information—information that appears needed for both spatial recall in rats, and for episodic or explicit memory in humans (Kroll et al., in press; Metcalfe et al., 1994).

Scoville and Milner (1957) and Zola-Morgan and colleagues (1986) have studied patients with hippocampal damage. Patient R.B., who had sustained damage to the CA1 region of the hippocampus, showed a selective deficit on episodic memory tasks including free recall, recognition, serial recall, picture reproduction, and story recall. However, other mental functions such as general intellectual functioning, attention, IQ, and presumably emotional responding, were unimpaired. R.B. suffered no personality distortion from the cool-system lesion. Bechara and colleagues (1995) have shown a double dissociation between fear conditioning and episodic memory with two patients, one of whom has an amygdala lesion and the other a hippocampal lesion.

In the hot/cool framework, we put much emphasis on the findings, extensively reviewed by de Kloet et al. (1993), that the cool system and the hot system respond differently to increasing stress. The cool system shows a non-monotonic response to increasing stress, much like the classic

Yerkes-Dodson Law. At low levels of stress, mineralocorticoid receptors in the hippocampus produce an increase in responsiveness, but at higher levels of stress the successive occupation of glucocorticoid receptors, in addition to the mineralocorticoid receptors, causes the hippocampus to become less responsive, and eventually, at extremely high levels, dysfunctional. In contrast, the hot system shows a simple increase in responsiveness to increasing stress, at least within physiological boundaries (McGaugh, 1989).

It follows that at low levels of stress, both the fear-evoking features (hot) and the contextual and narrative features (cool) of a situation show enhanced encoding with increasing stress (or arousal). However, at traumatic levels of stress, the cool system becomes dysfunctional, while the hot system becomes hyper-responsive. This means that the encoding under such conditions should be fragmentary rather than spatio-temporally bound, replete, and coherent. At high levels of stress the individual will focus selectively and, at traumatic levels, exclusively, on the fear-evoking features that are peculiar to the hot system. These hot features or triggers provoke fear reactions, and condition such reactions to them. Memories and reactions that are attributable to the isolated hot-system encoding may seem irrational both to the individual him- or herself, and to the therapist, since such fragments are ungrounded by the kind of narrative and spatio-temporal contextual anchors that tie our ordinary experience to reality. Such memories are disturbing, not only because of the direct fear they evoke but also because of their strangeness.

The oft-reported irrational fears, fragmented memories, and dissociated experience in PTSD patients are summarized by Spiegel and Cardeña (1991). Van der Kolk (1994) has contributed extensive discussions of how people with post-traumatic stress disorder fail to integrate traumatic experiences into the narrative of their lives. Such people, according to van der Kolk's analysis, initially perceive such experiences differently than do normal people under non-traumatic conditions—processing the traumatic event at a somatosensory level rather than autobiographically. He suggested that recall of the traumatic experience can be segregated into two phases. The first involves sensorimotor hypermnesia, extreme sensitivity to fragmentary trauma-related stimuli, and intrusive reliving of parts of the traumatic event. The second involves a blunting of emotional experience in general, avoidance of any intrusive stimuli, and anhedonia.

We suggest that narratizing such fragments, in the course of therapy, could reweave them back into a cool-system framework and defuse some of the anxiety surrounding the fragments. This idea bears a strong resemblance to the historical therapeutic ideas of Pierre Janet (see van der Hart et al., 1989) and provides a theoretical grounding for modern therapies (Foa et al., 1993). This hot/cool hypothesis also provides a modern physiologically-based interpretation of the repercussions of trauma that relates to the classic work of Breuer and Freud (1896/1955). In this, the first mention of the concept of repression, they discuss the idea that the person may be able to consciously and inten-

tionally push remembrance of painful events out of his or her consciousness. Fragments later emerge because keeping the remembered events out of consciousness requires effort, and such effort may occasionally flag, resulting in disturbing, apparently unconnected, fear-evoking fragments. Although the fragmentary nature of the intrusive ideation is acknowledged by Breuer and Freud, its etiology is thought to be different than in the hot/cool framework.

Interestingly, in this early paper, Breuer and Freud (1896/1955) also allude to the construct that later became known as state-dependent memory, that is, if a person encoded an event under a severely altered mood-state he or she may be unable to retrieve that event in his or her normal state. However, it may reemerge when the original mood-state is reinstated (Eich, 1995). By this view, the entire event encoded under trauma may be available in memory, but inaccessible unless the individual is again in a state of trauma. Within the hot-cool framework, by contrast, only the fear-provoking fragments were assumed to be encoded in the first place, and so the spatio-temporal context, not having been encoded, cannot be retrieved from episodic memory.

This theory, unlike the state-dependent account (provided the memory surfaces only under the appropriate mood state, of course) and unlike the repression notion, is challenged to explain how it is that people sometimes seem to be able to provide a detailed narrative of traumatic events. However, there is abundant evidence that people will indeed fill in gaps to impose a meaning and a schematic structure, even when no such structure is given in the

materials (Alba & Hasher, 1983).

Finally, Jacobs and Nadel (1985) spelled out some of the implications of the hippocampal-stress functions for the reemergence of childhood phobias. They noted that the hippocampus is fully myelinated and functional only relatively late in development. Before this system becomes functional, though, the infant may experience fear-evoking stimuli. Once the cool system is fully functioning, it suppresses the more primitive infantile systems—except under conditions of extreme high stress when the cool system becomes dysfunctional and control reverts to the more infantile systems. They suggested that this impairment of cool-system processing under high stress may explain the reemergence of childhood phobias under stress in adulthood.

REFERENCES

- ALBA, J.W. & HASHER, L. (1983). **Is memory schematic?** *Psychological Bulletin*, 93, 203-231.
- BREUER, J. & FREUD, S. (1896/1955). *Studies on hysteria* (J. Strachey, Ed. & Trans.). *The standard edition of the complete psychological works of Sigmund Freud: Volume 2*. London: Hogarth Press.
- EICH, E. (1995). **Searching for mood dependent memory.** *Psychological Science*, 6, 67-75.

SELECTED ABSTRACTS

AGGLETON, J.P. & PASSINGHAM, R.E. (1981). **Syndrome produced by lesions of the amygdala in monkeys (*Macaca mulatta*)**. *Journal of Comparative and Physiological Psychology*, 95, 961-977. Behavioral effects of subtotal amygdaloid lesions were investigated in an attempt to dissociate some of the abnormalities seen after total amygdectomy. 12 monkeys received bilateral stereotaxic lesions centered in the basolateral amygdala, lateral amygdala, dorsal amygdala, or the temporal white matter lying adjacent to the lateral amygdala. These monkeys were compared with others with control operations. The control monkeys then received total amygdaloid lesions (AMX). The AMX monkeys exhibited the typical amygdaloid syndrome of hypoemotionality, meat eating, coprophagia, and excessive exploration. In contrast, the monkeys with subtotal amygdaloid lesions would not eat meat or feces, though they were more willing than control monkeys to investigate inanimate objects. Although minor changes in affect were observed, the extreme emotional changes seen after total amygdectomy were found only in the monkey with the largest subtotal lesion. Only those animals that were hypoemotional showed a deficit in learning successive reversals of an object discrimination. This close association suggests that both the hypoemotionality and the successive reversal deficit arise from the same underlying dysfunction.

CHRISTIANSON, S.Å. (1992). **Emotional stress and eyewitness memory: A critical review.** *Psychological Bulletin*, 112, 284-309. The eyewitness literature often claims that emotional stress leads to an impairment in memory and, hence, that details of unpleasant emotional events are remembered less accurately than details of neutral or everyday events. A common assumption behind this view is that a decrease in available processing capacity occurs at states of high emotional arousal, which, therefore, leads to less efficient memory processing. The research reviewed here shows that this belief is overly simplistic. Current studies demonstrate striking interactions between type of event, type of detail information, time of test, and type of retrieval information. This article also reviews the literature on memory for stressful events with respect to two major theories: the Yerkes-Dodson law and Easterbrook's cue-utilization hypothesis. To account for the findings from real-life studies and laboratory studies, this article discusses the possibility that emotional events receive some preferential processing mediated by factors related to early perceptual processing and late conceptual processing.

DE KLOET, E.R., OITZL, M.S., & JOËLS, M. (1993). **Functional implications of brain corticosteroid receptor diversity.** *Cellular and Molecular Neurobiology*, 13, 433-455. Corticosteroids readily

enter the brain and control gene expression in nerve cells via binding to intracellular receptors, which act as gene transcription factors. In the rat brain corticosterone binds to mineralocorticoid receptors (MRs) with a 10-fold higher affinity than to glucocorticoid receptors (GRs). As a consequence, these MRs are extensively occupied under basal resting conditions, while substantial GR occupation occurs at the circadian peak and following stress. Both receptors are colocalized in most, but not all, hippocampal neurons. In addition, some neurons contain aldosterone-selective MRs, if corticosterone is enzymatically inactivated. These aldosterone target neurons are presumably localized in the anterior hypothalamus, where they underlie central control of salt appetite and cardiovascular regulation. The data show that MR- and GR-mediated effects proceed in a coordinate and often antagonistic mode of action: (1) in hippocampus MR activation maintains excitability, while GR occupancy suppresses excitability, which is transiently raised by excitatory stimuli; (2) central MRs participate in control of the sensitivity of the neuroendocrine stress response system, while GRs are involved in termination of the stress response; (3) MRs in the hippocampus have a role in regulation of behavioral reactivity and response selection. GR-mediated effects facilitate storage of information. On the basis of these data, we propose that a relative deficiency or excess of MR- over GR-mediated neuronal effects may lead to a condition of enhanced or reduced responsiveness to environmental influences, alter behavioral adaptation, and promote susceptibility to stress. The findings may serve development of novel therapeutic strategies for treatment of stress-related brain diseases.

JACOBS, W.J. & NADEL, L. (1985). **Stress induced recovery of fears and phobias.** *Psychological Review*, 92, 512-531. Accounts of human fears and phobias based on current conditioning models using data from adults are examined and found wanting. Instead, the characteristics of human phobias resemble the kind of learning found during the amnesic period of infancy. As certain neural systems mature, conditioning begins to exhibit adult characteristics: context dependency, sharp generalization, and rapid extinction. Although direct behavioral control by the early learning systems wanes, the adult learning system seems to be structured at least partially through the lasting influence of infantile experience. Under (hormonal) stress, residues of early experience are reinstated and incorporated into adult memory where they directly control behavior. This control exhibits infantile characteristics. The evidence suggests that once acquired, such conditional fears might never be eliminated using traditional extinction or counterconditioning procedures. The view leads to a renewed emphasis upon the role of experience in human development, accepting the disproportionate importance of infant experience as the foundation upon which subsequent learning and cognitive function rest.

LEDOUX, J.E. (1995). **Emotion: Clues from the brain.** *Annual Review of Psychology*, 46, 209-235. Despite the obvious importance of emotion to human existence, scientists concerned with human nature have not been able to reach a consensus about what emotion is and what place emotion should have in a theory of mind and behavior. This review examines the neural basis of emotion and considers how research on brain mechanisms can potentially help us to understand emotion as a psychological process. Topics covered include the neural basis of fear, neural pathways mediating fear conditioning, cellular mechanisms involved in fear conditioning, extinction of conditioned fear, conditioned fear and instrumental action, and the relation of the neural basis of fear to other emotions. Implications for psychopathology and other topics are discussed. [Adapted from Text]

MCGAUGH, J.L. (1989). **Involvement of hormonal and neuromodulatory systems in the regulation of memory storage.** *Annual Review of Neuroscience*, 12, 255-287. The findings reviewed here clearly indicate that retention of recently acquired information can be altered by a variety of posttraining treatments affecting hormonal and neuromodulatory systems. The findings provide strong support for the general hypothesis that endogenous hormonal and neuromodulatory systems activated by learning may play an important role in regulating the storage of information. Further, recent research has begun to provide some evidence concerning the brain systems through which hormone and neuromodulatory systems may act to influence memory. It seems clear from these recent findings that an understanding of the neural process underlying lasting memory will require knowledge of how memory storage processes are orchestrated by these modulatory systems. [Adapted from Text]

NADEL, L., WILLNER, J., & KURZ, E.M. (1985). **Cognitive maps and environmental context.** In P.D. Balsam & A. Tomie (Eds.), *Context and learning* (pp. 385-406). Hillsdale, NJ: Lawrence Erlbaum Associates. We attempt an integration of ideas about the role the hippocampus plays in creating cognitive maps by outlining neural processes that could realize the psychological functions we have attributed to cognitive maps. Our use of the term cognitive map is similar in spirit to that of Tolman, though it is more restricted in that we stress the explicitly spatial content of the representations underlying the maps. Each form of contextual information pertains to separate biological problems. There is no good reason to assume that these various kinds of context are all represented within the same processing system. We posit that the differing "contextual" systems reflect the action of separate representational mechanisms, realized with different neural modules. Because of their special role in maintaining the "connectivity" among elements representing everything occurring in an environment, spatial maps play an extended role in the dynamic reorganization of memory with the passage of time after an event. Rehearsals, reactivations, and reminders all effect a retrieval of memories that can activate appropriate hippocampal-neocortical circuitry, leading to improved consolidation. In order to specify the precise details of this interaction between hippocampal maps and neocortical associative links, we need more research on the anatomical and physiological machinery. [Adapted from Text]

NAHM, F.K.D., TRANEL, D., DAMASIO, H., & DAMASIO, A.R. (1993). **Cross-modal associations and the human amygdala.** *Neuropsychologia*, 31, 727-744. The role of the human amygdala in cross-modal associations was investigated in two subjects: SM-046, who had bilateral damage circumscribed to the amygdala; and the patient known as Boswell, whose damage in both temporal lobes included the amygdala and surrounding cortices. Neither subject was impaired on Tactile-Visual or Visual-Tactile cross-modal tasks using the Arc-Circle test, suggesting that the amygdala is not involved in cross-modal associations involving perceptually "equivalent" basic stimulus properties. On the other hand, the results are compatible with the amygdala's involvement in higher-order associations between exteroceptive sensory data and interoceptive data concerned with correlated somatic states.

VAN DER KOLK, B.A. (1994). **The body keeps the score: Memory and the evolving psychobiology of posttraumatic stress.** *Harvard Review of Psychiatry*, 1, 253-265. Ever since people's responses to overwhelming experiences have been systematically explored, researchers have noted that a trauma is stored in somatic memory and expressed as changes in the biological stress

response. Intense emotions at the time of the trauma initiate the long-term conditional responses to reminders of the event, which are associated both with chronic alterations in the physiological stress response and with the amnesias and hypermnesias characteristic of PTSD. Continued physiological hyperarousal and altered stress hormone secretion affect the ongoing evaluation of sensory stimuli as well. Although memory is ordinarily an active and constructive process, in PTSD failure of declarative memory may lead to organization of the trauma on a somatosensory level (as visual images or physical sensations) that is relatively impervious to change. The inability of people with PTSD to integrate traumatic experiences and their tendency, instead, to continuously relive the past are mirrored physiologically and hormonally in the misinterpretation of innocuous stimuli as potential threats. Animal research suggests that intense emotional memories are processed outside of the hippocampally mediated memory system and are difficult to extinguish. Cortical activity can inhibit the expression of these subcortically based emotional memories. The effectiveness of this inhibition depends, in part, on physiological arousal and neurohormonal activity. These formulations have implications for both the psychotherapy and the pharmacotherapy of PTSD.

VAN DER HART, O., BROWN, P., & VAN DER KOLK, B.A. (1989). **Pierre Janet's treatment of post-traumatic stress disorder.** *Journal of Traumatic Stress, 2*, 379-395. Pierre Janet's therapeutic approach to traumatized patients was the first attempt to create a systematic, phase-oriented treatment of post-traumatic stress. Janet viewed the trauma response basically as a disorder of memory which interfered with effective action. Relying heavily on the use of hypnosis, he taught that the treatment of post-traumatic psychopathology consisted of forming a stable thera-

peutic relationship; retrieving and transforming traumatic memories into meaningful experiences; and taking effective action to overcome learned helplessness. Most of his observations and recommendations are as challenging today as when he first made them, starting a century ago.

ZOLA-MORGAN, S., SQUIRE, L.R., & AMARAL, D.G. (1986). **Human amnesia and the medial temporal region: Enduring memory impairment following a bilateral lesion limited to field CA1 of the hippocampus.** *Journal of Neuroscience, 6*, 2950-2967. During the past 100 years clinical studies of amnesia have linked memory impairment to damage of the hippocampus. Yet the damage in these cases has not usually been confined to the hippocampus, and the status of memory functions has often been based on incomplete neuropsychological information. Thus, the human cases have until now left some uncertainty as to whether lesions limited to the hippocampus are sufficient to cause amnesia. Here we report a case of amnesia in a patient (R.B.) who developed memory impairment following an ischemic episode. During the 5 years until his death, R.B. exhibited marked anterograde amnesia, little if any retrograde amnesia, and showed no signs of cognitive impairment other than memory. Thorough histological examination revealed a circumscribed bilateral lesion involving the entire CA1 field of the hippocampus. Minor pathology was found elsewhere in the brain (e.g., left globus pallidus, right postcentral gyrus, left internal capsule), but the only damage that could be reasonably associated with the memory defect was the lesion in the hippocampus. To our knowledge, this is the first reported case of amnesia following a lesion limited to the hippocampus in which extensive neuropsychological and neuropathological analyses have been carried out.

ADDITIONAL CITATIONS Annotated by the Editors

BECHARA, A., TRANEL, D., DAMASIO, H., ADOLPHS, R., ROCKLAND, C., & DAMASIO, A.R. (1995). **Double dissociation of conditioning and declarative knowledge relative to the amygdala and hippocampus in humans.** *Science, 269*, 1115-1118.

Studied conditioning and the acquisition of declarative knowledge in 4 controls and in 3 people with lesions to either, or both, the amygdala and the hippocampus. The patient with amygdala damage but no damage to the hippocampus acquired declarative knowledge but did not acquire conditioning. In contrast, the opposite pattern was observed in the patient who had damage only to the hippocampus. A patient who had damage to both the amygdala and the hippocampus acquired neither conditioning nor declarative knowledge.

BURKE, A., HEUER, F., & REISBERG, D. (1992). **Remembering emotional events.** *Memory & Cognition, 20*, 277-290.

Performed two experiments to reconcile conflicting findings regarding the effects of emotional arousal on memory encoding. Results indicated that effects depend on the nature of the to-be-remembered information. Emotion, induced by exposure to a distressing story, improved memory for gist, basic-level visual information, and detail irrelevant to a plot but associated with an event's center. Emotion impaired memory for details not associated with an event's center.

DAVIS, M. (1992). **The role of the amygdala in fear and anxiety.** *Annual Review of Neuroscience, 15*, 353-375.

Reviews data showing that the amygdala and its efferent projections may represent a central fear system that is involved in the expression and acquisition of conditioned fear. For example, electrical stimulation of the amygdala elicits a pattern of behaviors that mimic natural or conditioned states of fear and lesions of the amygdala block innate or conditioned fear.

EGETH, H. (1994). **Emotion and the eyewitness.** In P.M. Niedenthal & S. Kitayama (Eds.), *The heart's eye: Emotional influences in perception and attention* (pp. 245-267). New York: Academic Press.

Reviews literature on the effects of arousal and on the presence of a weapon on the accuracy of eyewitness testimony. The author concludes that the presence of a weapon may impair memory but that data are inconsistent. He suggests caution among psychologists testifying about weapon focus in jury trials.

FOA, E.B., ROTHBAUM, B.O., & STEKETEE, G.S. (1993). **Treatment of rape victims.** *Journal of Interpersonal Violence, 8*, 256-276.

Provides a framework for treating rape victims, first, by critiquing outcome studies, next, by reviewing traditional interventions and then cognitive behavioral interventions. The approach sug-

gested relies on a conceptualization based on fear networks. The authors propose that successful treatment is provided when fear networks are activated and then information incompatible with the fear structure is provided and incorporated into the fear structure.

KROLL, N.E.A., KNIGHT, R.T., METCALFE, J., WOLF, E.S., & TULVING, E. (in press). **Cohesion failure as a source of memory illusions.** *Journal of Memory and Language*.

Tested recognition memory of visual patterns and compound words in patients with focal damage to the left and right hippocampus. Patients showed no deficits, compared to normals, on the old and new items, but they were significantly impaired on rejecting the recombined items, that is, those consisting of old parts in new configurations. The authors suggest that the specific failure observed is attributable to a hippocampally-controlled breakdown in within-event binding.

METCALFE, J., MENCL, W.E., & COTTRELL, G.W. (1994). **Cognitive binding.** In D. L. Schacter & E. Tulving (Eds.)

Memory Systems 1994 (pp. 369-394). Cambridge: MIT Press.

Reviews models and findings on explicit/episodic and implicit memory. The authors argue that the two types of memory are distinguishable on the basis of how tightly the parts of the representation are interconnected. Episodic events are proposed to be those in which features are perceived and remembered as a particular, unique configuration, and not just as distinctive fragments. They propose that implicit and explicit memory systems are distinguished by a special memory-binding function in explicit memory that coalesces parts of an event into a coherent whole.

RUDY, J.W. & SUTHERLAND, R.J. (1994). **The memory coherence problem, configural associations, and the hippocampal system.** In D.L. Schacter & E. Tulving (Eds.), *Memory Systems 1994* (pp. 119-146). Cambridge: MIT Press.

Reviews the authors' theory about the role of the hippocampal system in learning and memory, focusing on the issue of memory

coherence. The authors argue that memory coherence is lost when the hippocampal system is damaged because it is essential to the configural-association system. They further argue that normal memory following hippocampal damage is facilitated by the elemental-association system.

SCOVILLE, W.B. & MILNER, B. (1957). **Loss of recent memory after bilateral hippocampal lesions.** *Journal of Neurology, Neurosurgery and Psychiatry*, 20, 11-21.

Performed memory and intelligence testing in 10 patients who had undergone bilateral medial temporal lobe resections. Results indicated the importance of the hippocampus in normal memory function. Any damage to the hippocampus or hippocampal gyrus was associated with some kind of memory deficit. Specific information is provided about each patient.

SPIEGEL, D. & CARDEÑA, E. (1991). **Disintegrated experience: The dissociative disorders revisited.** *Journal of Abnormal Psychology*, 100, 366-378.

Discusses proposed changes to the dissociative disorders section of DSM-IV. The authors review the concept of dissociation and empirical findings on the relationship between traumatic events and dissociative phenomenology. They also describe the dissociative disorder and argue that the study of dissociation is promising because it integrates both emotion and cognition and clinical and experimental approaches.

YUILLE, J.C. & CUTSHALL, J. (1989). **Analysis of the statements of victims, witnesses and suspects.** In J.C. Yuille (Ed.), *Credibility assessment* (pp. 175-191). Dordrecht, The Netherlands: Kluwer.

Examines the appropriateness of statement analysis in the evaluation of eyewitness testimony in adults. The authors distinguish between the cognitive aspects of statement analysis, which focus on detail and accuracy, and motivational aspects, which focus on the credibility of an account. They note that memory for actual crimes may be remarkably persistent, in contrast to event memories usually studied in laboratories.

PILOTS UPDATE

As we approach the addition of the 10,000th record to the PILOTS database, we are working to expand not only the size of the database but also its scope. We are not changing the subject-matter covered by the database, but we are working to push back the linguistic and chronological frontiers that have bounded our bibliographical work until now.

For several years we have been collecting foreign-language publications on PTSD. Many of these are in French and German, but we now have got material in over twenty languages, including Arabic, Finnish, Georgian, Korean, and Slovenian. Our in-house linguistic expertise does not stretch quite that far, so we have had to resort to the expedient of "limited indexing" to provide rudimentary coverage of these publications in the PILOTS database.

By "limited indexing" we mean the presentation of the basic bibliographical facts about the document, an abstract if an English-one appears in the publication, and descrip-

tors assigned on the basis of our best understanding of its content. In many cases we are able to provide only a broad sense of the document's subject-matter; but it is often possible to assign more specific descriptors to aspects of the population under study or of the treatment provided. We can identify internationally-recognized pharmaceutical nomenclature, or demographic information presented in tabular form, even when we cannot follow the authors' discussions of their work. We have assembled a reference collection of foreign-language dictionaries, lists of drug names, and specialized encyclopedias to help us in our efforts; and we are not above taking hints from the MeSH headings assigned to journal articles by the National Library of Medicine.

PILOTS database users will recognize these publications by the phrase "Limited indexing applied," which appears in the "Notes" field of each affected record. It is our hope someday to be able to revise our treatment of these papers,

so that their indexing will conform to the standards we have tried to maintain for the database as a whole. We hope to learn from PILOTS users, especially those in countries where English is not the common language, whether our experiment with "limited indexing" will in fact help to improve access to PTSD work reported in other languages. And, as always, we would be delighted to hear from any organization or individual with an interest in helping us collect and index foreign-language literature on traumatic stress.

Another area in which we are working to expand our coverage is the historical literature of traumatic stress. While historians have found descriptions of PTSD symptoms in Homer and the Bible, in Shakespeare and Samuel Pepys, formal publications in the scientific literature first appeared in the mid-19th century. Da Costa's paper on "irritable heart" in Civil War soldiers, published in 1871, is the earliest that we have in our PTSD Resource Center.

We recently purchased a collection of over one hundred papers extracted or reprinted from American, Canadian, European, and Australian publications during and immediately after World War I, and we have been promised copies of several dozen papers from the 1920s, '30s, and '40s. After taking action to preserve their content—they were often printed on low-quality newsprint which literally crumbles when touched—we shall index them in the same way that we do modern publications.

We feel that this material will be of interest, not only to historians, but also to those whose concern is with today's

PTSD patients. There will be few cases in which a diagnostic or treatment approach used eighty years ago will be applicable today. But there may well be situations in which it will be useful to know that a particular set of circumstances or a particular constellation of symptoms was presented to practitioners several decades ago.

Those who do not wish to include historical or foreign-language materials in the results of their PILOTS database searches can use the LIMIT command to exclude them. After completing your search, the System will inform you how many items have been retrieved. You may then type:

```
LIMIT LANG ENGLISH
```

to remove foreign-language items from the result set, or type:

```
LIMIT YEAR GT 1950
```

to remove anything published in 1950 or before. You can also use the LIMIT command after you have used DISPLAY MEDIUM or DISPLAY LONG to examine your initial search results. For further information on this, see page 21 of the PILOTS Database User's Guide, or see the page on "Displaying Your Search Results" on our World Wide Web site at:

<<http://www.dartmouth.edu/dms/ptsd/>>

NATIONAL CENTER FOR PTSD: RESEARCH ACTIVITIES IN FY 1995

The National Center for PTSD remains a world leader in research on psychological, psychophysiological, and neurobiological aspects of PTSD. Such activities include the development of diagnostic assessment tools, identification of biological markers, and testing of effective treatment for PTSD. Specific projects have focused on female, elderly, and minority veterans. In addition to war zone trauma, specific stressors investigated include sexual trauma, natural disasters, and experimental exposure to mustard gas during World War II. There is a new emphasis on treatment outcome research in which separate investigations have been designed to test cognitive-behavioral, drug, and group treatments for PTSD.

During FY 1995, 93 scientific publications and 112 in press articles were written by National Center authors. Currently, Center staff hold 56 research grants that have a total (multi-year) funding of over \$43,000,000, of which almost \$8,000,000 came in during the past year. Funding for all National Center research comes from both Center funds and peer-reviewed grants from VA, PHS, and private foundations. These funds are crucial to maintaining the Center's research program.

Major research activities during FY 1995 included the following:

- **Ethnic Minorities Study.** The Ethnic Minorities (or Matsunaga) Study, directed by Dr. Friedman, is a Congressionally mandated epidemiological survey of the prevalence of PTSD among Native American and Asian-Pacific Islander American Vietnam veterans. During FY 1995, data collection for the entire project (1,600 interviews) was completed. It is anticipated that a report of results will be available in 1996.

- **Cooperative Study on Psychophysiological Assessment.** This study, directed by Drs. Terence Keane and Lawrence Kolb, recruited 1,200 subjects from VAMCs across the United States. The primary purpose of the study is to determine the accuracy of psychophysiological measures for making a PTSD diagnosis. Analysis of data was completed for presentations at two major North American conferences and one international conference.

- **Women Veterans.** The Women's Health Sciences Division continued to conduct a variety of projects related to the study of gender and PTSD. One project found differences between men and women with PTSD in psycho-

physiological reactions to trauma exposure.

- **PTSD Treatment Research.** Drs. Matthew Friedman and Paula Schnurr submitted to the VA Cooperative Studies Program in July 1995 a proposal to conduct a 10-site randomized clinical trial of trauma focus group therapy for PTSD. This 3.5-year project will involve a total of ten sites closed from both Center and non-Center VAMC PTSD programs. The study was approved and is scheduled to begin late in FY 1996.

- **Neurobiological Research.** Under the direction of Dr. Dennis Charney, the Clinical Neurosciences Division continued to generate ground-breaking research that advances our emerging understanding of the complex pathophysiology of PTSD. This is accomplished through an integrated program of laboratory and clinical investigation on animals and humans. These include brain imaging studies, challenges with pharmacological probes, psychoendocrine research, and clinical drug trials.

- **Persian Gulf War Veterans.** The National Center has maintained an active programmatic and methodologic involvement in research initiatives concerning health problems of Persian Gulf returnees, especially at the recently funded Boston VA Environmental Hazards Research Center. Dr. Jessica Wolfe and her staff at the Women's Health Sciences Division continued to expand the Ft. Devens study, which is now in Year 5. This dataset continues to represent one of the largest, earliest, and most comprehensive databases on American military personnel who served in the Persian Gulf War.

- **Follow-Up of Military Personnel from Somalia.** Dr. Brett Litz, working in collaboration with other Behavioral Science Division staff, with the Readjustment Counseling Service, and with the Department of Defense, continued a follow-up project designed to examine the longitudinal mental health outcome associated with United Nation peacekeeping and peacemaking missions for US men and women who had been deployed to Somalia.

- **Mustard Gas.** Drs. Friedman, Schnurr, and Ford of the Executive Division, in collaboration with Dr. Bonnie Green of Georgetown University Medical Center, launched the

second phase of a project to study PTSD among men who participated in secret mustard gas tests during World War II. The second phase is a phone survey of 500 men who will be randomly sampled from a roster developed by VA's Office of Environmental Medicine and Public Health.

- **Program Evaluation.** The Evaluation Division continued its evaluation of the effectiveness of the inpatient treatment of PTSD. Four SIPUs, four EBTPUs, and three non-specialized General Psychiatry Units were compared in terms of the characteristics of the veterans being served, the operation of the treatment programs themselves, and the outcomes of treatment over a one-year period following discharge from the programs. A preliminary report was presented at the annual meeting of the International Society for Traumatic Stress Studies in Chicago in November 1994. Data collection is now complete, and a final report of the results is in preparation.

Future issues of the PTSD Research Quarterly will describe specific research activities at each National Center site.

"Books Received" Now on the World Wide Web

"Books Received in the PTSD Resource Center" is an annotated listing of books for professional and lay readers. Formerly appearing in our sister periodical, the *NCP Clinical Quarterly*, it is now a feature of our World Wide Web site (<http://www.dartmouth.edu/dms/ptsd>). This allows us to update the list as new titles come in, and also allows us to include as few or as many titles as we receive without restrictions imposed by deadlines or space limitations.

We encourage authors to ensure that we receive copies of their publications. This will ensure that information reaches potential readers and purchasers quickly, without the long delay usually experienced before a formal book review appears. Books may be sent to: Fred Lerner, National Center for PTSD (116D), VA Medical Center, 215 North Main Street, White River Junction, Vermont 05009-0001.

National Center for PTSD (116D)
VA Medical and Regional Office Center
215 North Main Street
White River Junction, Vermont 05009-0001