Deficits in Short-Term Memory in Posttraumatic Stress Disorder

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Objective: The purpose of this study was to compare the memory function of patients with posttraumatic stress disorder (PTSD) to that of matched comparison subjects. Method: Vietnam veterans with combat-related PTSD (N=26) were compared to physically healthy comparison subjects (N=15) matched for age, race, sex, years of education, handedness, socioeconomic status, and alcohol abuse. Memory and intelligence were assessed with a battery of neuropsychological tests, including the Russell revision of the Wechsler Memory Scale, the Selective Reminding Test, and subtests of the Wechsler Adult Intelligence Scale—Revised (WAIS-R). Results: The PTSD patients scored significantly lower than the comparison subjects on the Wechsler Memory Scale logical memory measures for immediate recall (mean=11.6, SD=3.3 versus mean=20.9, SD=6.6) and delayed recall (mean=8.0, SD=3.3 versus mean=17.8, SD=6.4). The PTSD patients also scored significantly lower on the total recall, long-term storage, long-term retrieval, and delayed recall measures for the verbal component of the Selective Reminding Test and on the recall, long-term storage, long-term retrieval, and continuous long-term retrieval measures for the visual component of the Selective Reminding Test. There was no significant difference between the PTSD patients and comparison subjects in prorated full-scale IQ as measured by the WAIS-R. Conclusions: Patients with PTSD may have deficits in short-term memory. Counseling and rehabilitation that address these deficits may be of value for PTSD patients.

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Studies of war veterans suggest an association between the extreme stress of combat and alterations in memory function. Since the time of the first world war, military psychiatrists have treated soldiers who wander off the battlefield, having forgotten their names or other pieces of important personal information (1–6). In a study from World War II (7) it was found that, immediately after a major campaign, about 5% of the soldiers who had been combatants in the campaign had no memory for events that had just occurred. Follow-up studies of veterans of World War II have found that many of these veterans suffer from episodes of "blackouts" or loss of memory (8). Disturbances of memory and concentration also have been found to be frequent complaints in studies of prisoners of war (POWs) from World War II (9–12) and the Korean conflict (13). One study of Korean POWs (13) performed 30 years after the war showed that these subjects had poorer short-term verbal memory, as measured with the logical memory component of the Wechsler Memory Scale, than did Korean combat veterans without a history of containment. Most of the patients (68%), however, had experienced weight loss of greater than 35% of baseline weight, which has been found to be associated with memory deficits in POWs (10). A higher than normal rate of psychogenic amnesia has also been reported in Vietnam veterans with combat-related posttraumatic stress disorder (PTSD) (14). These studies suggested that alterations in memory may be a component of the constellation of symptoms associated with exposure to combat stress, although they did not assess possible organic etiologies of memory alterations.

Stress may be associated with alterations in memory. Animals exposed to inescapable stress show deficits in learning and memory, manifested by impairment in maze-escape behaviors, in comparison to animals exposed to escapable stress (15–17). In another study, nonhuman primates exposed to the chronic stress of im-

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proper housing developed damage to the hippocampus (18), a brain structure involved in memory (19–23). Glucocorticoids, which are released at high levels during stress, appear to have neurotoxic effects on the CA2 and CA3 regions of the hippocampus (24–30). These studies suggest that exposure to stress may result in alterations in memory, possibly through effects on the hippocampus and adjacent areas.

The importance of the hippocampus and adjacent cortical areas in learning and memory is demonstrated by data from neuroanatomical and neuropsychological studies. Neuroanatomical evidence from studies of the hippocampus and adjacent cortical areas in nonhuman primates shows deficits in learning and memory following lesions in these areas (31–34). Neuropsychological studies show deficits in short-term memory in patients with epilepsy following surgical removal of the temporal lobe (35) and in epileptic patients with intractable temporal lobe seizures and associated decreased hippocampal volume (36–39). Studies of human amnestic subjects, such as the well-known case of H.M., also support a role for the hippocampus in memory (40, 41).

We recently reported lower hippocampal volume in patients with combat-related PTSD than in matched comparison subjects (42). Since lesions of the hippocampus have been associated with deficits in short-term memory, the purpose of the present study was to compare short-term memory function in patients with combat-related PTSD and in matched comparison subjects. We selected a group of healthy comparison subjects for this purpose who were matched for alcohol abuse, age, sex, years of education, handedness, and other factors that could affect memory function.

METHOD

Subjects

The patient group consisted of 26 Vietnam veterans with combat-related PTSD. They were recruited from the inpatient unit of the National Center for Post-Traumatic Stress Disorder, Division of Clinical Neurosciences, West Haven Veterans Affairs Medical Center. The diagnosis of PTSD was based on the DSM-III-R criteria for PTSD as determined with the Structured Clinical Interview for DSM-III-R (SCID) (43), consensus diagnosis by three research psychiatrists, and a score of greater than 107 on the Mississippi Scale for Combat-Related Posttraumatic Stress Disorder (44). None of the patients had a history of traumatic brain injury, neurological disorder, current alcohol abuse as defined by DSM-III-R criteria, or psychosis. Alcohol and drug abuse were monitored for 2 months before the study while the patients were on an inpatient PTSD unit, where they received random toxicology screens for substance abuse. The patient group comprised 26 of 28 consecutively admitted patients who met the criteria for the study. The effect of loss of consciousness was controlled for by selecting patients without extensive loss of consciousness. Of the 26 patients, 25 did not have a history of sustained loss of consciousness (defined as loss of consciousness for longer than 10 minutes), and one patient had a loss of consciousness for 1 hour (not in the past year); this patient was matched with a comparison subject who had a similar history. None of the patients or comparison subjects had a history of loss of consciousness of any duration within the past year. The patients had undergone a medication washout period before participation and were medication free for 1 month or more before participation in the study.

The 15 comparison subjects were physically healthy and were matched for a number of factors that may affect memory function. They were recruited largely from a crew of construction workers and also included hospital orderlies, other nonprofessional workers, and unemployed individuals. The comparison subjects did not have a history of combat exposure and were chosen from the 20 individuals who were contacted and were eligible for participation in the study. The comparison subjects and the patients were paid for their participation in the study. The comparison subjects were matched with the patients for age, sex, race, handedness, height, weight, years of education, years of parental education, and years of alcohol abuse. Subjects with a history of traumatic brain injury, meningitis, neurological disorder, current alcohol abuse according to DSM-III-R criteria and the SCID interview, physical illness, or psychiatric disorder were excluded from the study. Subjects were also excluded if they had a history of loss of consciousness of greater than 10 minutes, except for the single subject who was matched with the PTSD patient who had a similar history.

All of the subjects were men. The mean ages of the patients and comparison subjects were 45.7 (SD=3.8) and 42.3 (SD=10.3) years, respectively. Their mean education levels were 12.9 (SD=1.9) and 13.5 (SD=2.9) years. Twenty (77%) of the patients and 14 (93%) of the comparison subjects were right-handed. The patient group comprised 25 white subjects (96%) and one black subject (4%), whereas the comparison group comprised 12 white (80%), two black (13%), and one Hispanic (7%) subjects. There were no significant differences between the groups in any of these variables.

Assessment

The Addiction Severity Index interview was used to assess lifetime alcohol abuse. The Addiction Severity Index evaluates the total number of years an individual has abused alcohol over a lifetime; abuse is defined as drinking to the point of intoxication, consuming three or more drinks per day, drinking on a regular basis, and drinking on three or more days per week (45). The PTSD patients with a history of alcohol abuse were matched on a case-by-case basis with comparison subjects who had a history of alcohol abuse according to the Addiction Severity Index interview. The mean durations of alcohol abuse in the two groups were 11.5 (SD=8.4) and 8.1 (SD=10.9) years ($t=0.88$, $df=39$, $p=0.38$).

The PTSD patients were evaluated with instruments designed to assess level of current PTSD symptoms and degree of exposure to combat in Vietnam. The patients were evaluated with the Mississippi Scale for Combat-Related Posttraumatic Stress Disorder, a self-report measurement of current PTSD symptom severity with acceptable reliability and validity (44). Level of combat exposure was evaluated with the Combat Exposure Scale, another validated and reliable self-report instrument for measuring exposure, in order to establish a history of combat exposure (46).

A battery of neuropsychological tests was also administered to each subject, as follows.

1. To estimate an intellectual level for each subject, we administered four tests of the Wechsler Adult Intelligence Scale—Revised (WAIS-R): the arithmetic, vocabulary, picture arrangement, and block design tests.

2. Two subtests of the Wechsler Memory Scale were administered according to the Russell revision (47). The subtests included logical memory—the free recall of two story narratives, which is felt to be a test of verbal memory—and figural memory—which is felt to represent visual memory and involves the reproduction of designs after a 10-second presentation. For both subtests, immediate and delayed reproduction were tested and a percentage of retention was computed (delayed recall divided by immediate recall multiplied by 100).

3. Two components of the Selective Reminding Test (48, 49) were completed. The verbal task is a measure of verbal learning in which 12 words are presented for immediate recall. On subsequent trials only the words not recalled on the prior trial are presented. The task is complete after two consecutive perfect recall trials or 12 presentations.

The visual component of the Selective Reminding Test (48, 49) is modeled on the verbal test; 12 designs are presented one at a time for 3 seconds each, followed by an opportunity to draw all from memory.

Each design that is not accurately reproduced on a given trial is shown again until perfect recall is attained or 12 trials are reached.

Five indexes of learning and memory are obtained from each of the selective reminding tasks: total recall, long-term retrieval, long-term storage, list learning (continuous long-term retrieval), and delayed recall.

Statistical Analysis

We used t tests to compare the patients and comparison subjects on each of the subcomponents of the Wechsler Memory Scale, Selective Reminding Test, and WAIS-R. Two-tailed tests of significance were used throughout, and Fisher's exact tests were used when there were fewer than five observations in a single cell. Pearson's product correlations were performed on the neuropsychological test scores and clinical variables, including the Mississippi scale score. Analysis of covariance (ANCOVA) was performed to assess differences in scores on the neuropsychological tests of memory while controlling for differences in alcohol abuse between the two groups. The hypothesis of this study was that the scores on the scales for measuring memory function would be lower for the PTSD patients than for the comparison subjects. Since multiple comparisons were performed with 16 different tests of memory, the Bonferroni correction for multiple comparisons was applied to both the t tests and the correlations. Significance was defined as $p<0.05$, and the significance level with the Bonferroni correction was determined to be $p<0.0016$.

RESULTS

As shown in table 1, the PTSD patients and comparison subjects had similar intelligence levels. The PTSD patients scored significantly lower on the Wechsler Memory Scale logical memory (verbal) component measures for immediate and delayed memory. Scores on the figural memory (visual) component of the Wechsler Memory Scale were slightly lower in the patients than in the comparison subjects, although the difference was not significant. After ANCOVA was used to control for differences in alcohol abuse between the two groups, the difference in scores on the logical memory component persisted for immediate memory (F=15.29, df=1, 38, p=0.0004) and delayed recall (F=42.62, df=1, 38, p<0.0001), and there was a trend toward a difference in percentage of retention after multiple comparisons were controlled for (F=4.94, df=1, 38, p=0.03).

The PTSD patients scored significantly lower than the comparison subjects on most of the measures for the verbal and visual components of the Selective Reminding Test. The patients had significantly lower scores on the total recall, long-term retrieval, long-term storage, and delayed recall measures of the verbal component and on the total recall, continuous long-term retrieval, long-term retrieval, and long-term storage measures for the visual component (table 1). The difference was greatest for verbal continuous long-term retrieval, on which the patients scored 36% lower than did the comparison subjects. After ANCOVA was used to control for differences in alcohol abuse between the two groups, the differences in the verbal test measures persisted for total recall (F=14.09, df=1, 38, p=0.0006), long-term retrieval (F=15.35, df=1, 38, p=0.0004), long-term storage (F=13.90, df=1, 38, p=0.0006), and continuous long-term retrieval (F=12.78, df=1, 38, p=0.001). The differences in the scores on the visual component of the Selective Reminding Test also persisted for total recall (F=14.00, df=1, 38, p=0.0006), long-term storage (F=11.75, df=1, 38, p=0.002), long-term retrieval (F=14.34, df=1, 38, p=0.0005), and continuous long-term retrieval (F=15.55, df=1, 38, p=0.0003).

TABLE 1. IQs and Memory Test Scores of Patients With Combat-Related PTSD and of Matched Healthy Comparison Subjects

Measure	PTSD Patients (N=26) Mean	SD	Comparison Subjects (N=15) Mean	SD	Two-Tailed t Test (df=39) t	p
WAIS-R IQ						
Verbal	101.0	10.2	101.7	16.4	0.16	0.87
Performance	100.9	16.3	107.7	19.4	1.15	0.26
Full scale	101.0	11.2	105.8	17.9	1.01	0.32
Wechsler Memory Scale						
Logical memory						
Immediate recall	11.6	3.3	20.9	6.6	5.07	0.001[a]
Delayed recall	8.0	3.3	17.8	6.4	5.58	0.0001[a]
Percent retention	67.0	18.2	82.4	20.2	2.51	0.02
Figural memory						
Immediate recall	9.6	2.7	11.3	2.8	1.88	0.07
Delayed recall	7.8	4.1	9.6	3.9	1.34	0.18
Percent retention	76.5	30.5	82.1	20.9	0.64	0.52
Selective Reminding Test						
Verbal						
Total recall	94.5	17.3	114.0	14.1	3.71	0.0006[a]
Long-term storage	82.2	26.1	109.9	16.9	3.67	0.0007[a]
Long-term retrieval	72.4	26.6	103.0	20.1	3.85	0.0004[a]
Continuous long-term retrieval	49.0	26.5	76.7	31.4	3.11	0.003
Delayed recall	6.5	3.1	10.0	2.2	3.78	0.0006[a]
Visual						
Total recall	117.5	14.9	132.8	6.6	4.55	0.0001[a]
Long-term storage	115.1	17.4	131.1	8.6	3.95	0.0003[a]
Long-term retrieval	110.5	18.8	130.3	9.0	4.54	0.0001[a]
Continuous long-term retrieval	96.7	26.8	126.7	11.4	5.00	0.0001[a]
Delayed recall	9.7	3.0	11.7	0.5	2.87	0.009

[a]Significant difference after multiple comparisons were controlled for (p<0.002).

Percentage of retention for the Wechsler Memory Scale figural memory component was correlated with current level of PTSD symptoms as measured by the Mississippi Scale for Combat-Related Posttraumatic Stress Disorder (r=–0.62, df=40, p=0.001).

DISCUSSION

The patients with PTSD in this study scored lower on neuropsychological tests of memory than did comparison subjects matched for variables that could affect memory. The PTSD patients scored significantly lower on the logical (verbal) memory component of the Wechsler Memory Scale—44% lower on immediate recall and 55% lower on delayed recall.

The percentage of retention for the figural memory component of the Wechsler Memory Scale was negatively correlated with PTSD symptom severity as assessed with the Mississippi Scale for Combat-Related Posttraumatic Stress Disorder. There may be a relationship between visual memory function level and PTSD symptom severity, although we did not find a significant difference in percentage of retention on the Wechsler Memory Scale figural memory component between the patients and comparison subjects. It is possible that alterations in visual memory are relevant to the symptoms of PTSD. Data from our study suggest that the deficit is relatively broad based and reflects problems at several levels of information processing, including both acquisition and retention/retrieval, although the patient and comparison groups did not differ significantly in overall cognitive ability as reflected by IQ.

The PTSD patients in our study displayed memory problems comparable to those of other clinical populations with clearly documented temporal lobe damage and hippocampal involvement. Our finding of 67% retention on the logical memory component of the Wechsler Memory Scale is comparable to the 53% retention in patients with left temporal lobe epilepsy and 74% in patients with right temporal lobe epilepsy previously observed (36), while our comparison subjects' performance was comparable both to the performance of the comparison subjects in that study and to normative data (47). The figural memory component of the Wechsler Memory Scale may be a less sensitive measure, as has previously been observed, and the findings for the PTSD patients are comparable to those for patients with temporal lobe seizures (36). In addition, our PTSD patients' performance on the verbal component of the Selective Reminding Test is similar to that of patients previously studied after temporal lobe and hippocampal resection for intractable epilepsy (50, 51).

There are several possible explanations for our finding of alterations in memory function in patients with PTSD. Studies in animals of the neuroanatomical correlates of memory provide evidence for a role of the hippocampus in memory. Procedures on monkeys involving the H+ lesion, which ablates hippocampus and surrounding parahippocampal gyrus, entorrhinal cortex, and perirhinal cortex, result in significant impairments in short-term memory (23, 32, 33). Lesions involving the amygdala alone do not result in a similar degree of impairment (31). The hippocampus has also been shown to have a specific function in object-place memory, or memory of an object's position in space (52). We recently reported that PTSD may be associated with low hippocampal volume (42). These studies suggest that alterations in the hippocampus and possibly other brain structures involved in memory, such as the amygdala, prefrontal cortex, and temporal lobe, may result in deficits in memory function. Another possible explanation is that deficits in concentration in patients with PTSD are responsible for impairment on neuropsychological tests of memory. Our findings of deficits in immediate recall are consistent with this explanation.

Several aspects of this study may limit generalization of the results of this study. Although we attempted to control for the effects of alcohol abuse by matching for the total number of years of alcohol abuse with the Addiction Severity Index, we may not have adequately controlled for the effects of alcohol on memory function. In addition, the fact that our patients were seeking treatment for their psychiatric condition may limit the extent to which our findings can be generalized to non-treatment-seeking populations or to patients with non-combat-related PTSD. The fact that the comparison subjects did not have a history of combat exposure suggests that deficits in memory may not be ascribed to PTSD per se but, rather, to the effects of combat exposure. In this regard, however, it should be pointed out that comparing PTSD patients to healthy comparison subjects matched for factors that have been suggested to affect memory function is an appropriate first step and can be followed up by future studies.

Our findings may have implications for the diagnosis and treatment of PTSD. Neuropsychological testing may represent an objective assessment of patients with combat-related PTSD and should be considered in the assessment of patients with the diagnosis. Rehabilitation strategies oriented toward compensation for deficits in memory may be of value for PTSD patients. Proper counseling with a full knowledge of the deficits these patients are facing may be beneficial in helping these patients succeed in occupations for which they have the most potential.

REFERENCES

1. Geleerd ER, Hacker FJ, Rapaport D: Contributions to the study of amnesia and allied conditions. Psychoanal Q 1945; 14:199–220
2. Grinker RR, Spiegel JP: War Neuroses in North Africa. New York, Josiah Macy Jr Foundation, 1943
3. Grinker RR, Spiegel JP: Men Under Stress. New York, Blakiston, 1945
4. Henderson JL, Moore M: The psychoneurosis of war. N Engl J Med 1944; 230:274–278
5. Jaff R: Dissociative phenomena in concentration camp inmates. Int J Psychoanal 1968; 49:310–312
6. Sargent W, Slater E: Amnesic syndromes in war. Proc R Soc Med 1941; 34:757–764
7. Torrie A: Psychosomatic casualties in the Middle East. Lancet 1944; 29:139–143
8. Archibald HC, Tuddenham RD: Persistent stress reaction after combat. Arch Gen Psychiatry 1965; 12:475–481
9. Goldstein G, van Kammen W, Shelly C, Miller DJ, van Kammen DP: Survivors of imprisonment in the Pacific theater during World War II. Am J Psychiatry 1987; 144:1210–1213
10. Sutker PB, Allain AN, Motsinger PA: Minnesota Multiphasic Personality Inventory (MMPI)-derived psychopathology subtypes among former prisoners of war (POWs): replication and extension. J Psychopathology and Behavioral Assessment 1988; 10:129–140
11. Thygesen P, Hermann K, Willanger R: Concentration camp survivors in Denmark: persecution, disease, compensation. Dan Med Bull 1970; 17:65–108
12. Eitinger L: The concentration camp syndrome and its late sequelae, in Survivors, Victims, and Perpetrators: Essays on the Nazi Holocaust. Edited by Dimsdale JE. Washington, DC, Hemisphere, 1980
13. Sutker PB, Winstead DK, Galina ZH, Allain AN: Cognitive deficits and psychopathology among former prisoners of war and combat veterans of the Korean conflict. Am J Psychiatry 1991; 148:67–72

14. Bremner JD, Southwick S, Brett E, Fontana A, Rosenheck R, Charney DS: Dissociation and posttraumatic stress disorder in Vietnam combat veterans. Am J Psychiatry 1992; 149:328–332
15. Sherman AD, Sacquitne JL, Petty F: Specificity of the learned helplessness model of depression. Pharmacol Biochem Behav 1982; 16:449–454
16. Seligman ME, Maier SF: Failure to escape traumatic shock. J Exp Psychol 1967; 74:1–9
17. Drugan RC, Ryan SM, Minor TR, Maier SF: Librium prevents the analgesia and shuttlebox escape deficit typically observed following inescapable shock. Pharmacol Biochem Behav 1984; 21: 749–754
18. Uno H, Tarara R, Else JG, Suleman MA, Sapolsky RM: Hippocampal damage associated with prolonged and fatal stress in primates. J Neurosci 1989; 9:1705–1711
19. Amaral DG, Witter MP: The three-dimensional organization of the hippocampal formation: a review of anatomical data. Neuroscience 1989; 31:571–591
20. Witter MP, Amaral DG: Entorhinal cortex of the monkey, V: projections to the dentate gyrus, hippocampus, and subicular complex. J Comp Neurol 1991; 307:437–459
21. Funahashi S, Bruce CJ, Goldman-Rakic PS: Mnemonic coding of visual space in the monkey's prefrontal cortex. J Neurophysiol 1989; 61:331–351
22. Friedman HR, Goldman-Rakic PS: Activation of the hippocampus and dentate gyrus by working-memory: a 2-deoxyglucose study of behaving rhesus monkeys. J Neurosci 1988; 8:4693–4706
23. Goldman-Rakic PS, Selemon LD, Schwartz ML: Dual pathways connecting the dorsolateral prefrontal cortex with the hippocampal formation and the parahippocampal cortex in the rhesus monkey. Neuroscience 1984; 12:719–743
24. Sapolsky RM, Packan DR, Vale WW: Glucocorticoid toxicity in the hippocampus: in vitro demonstration. Brain Res 1988; 453: 367–371
25. Sapolsky R, Pulsinelli W: Glucocorticoids potentiate ischemic injury to neurons: therapeutic implications. Science 1985; 229: 1397–1400
26. Sapolsky RM, Uno H, Rebert CS, Finch CE: Hippocampal damage associated with prolonged glucocorticoid exposure in primates. J Neurosci 1990; 10:2897–2902
27. Uno H, Lohmiller L, Thieme C, Kemnitz JW, Engle MJ, Roecker EB, Farrell PM: Brain damage induced by prenatal exposure to dexamethasone in fetal rhesus macaques, I: hippocampus. Brain Res Dev Brain Res 1990; 53:157–167
28. Muhlen K aus der, Ockenfels H: Morphologische Veränderungen im Diencephalon und Telencephalon nach Störungen des Regelkreises Adenohypophyse—Nebennierenrinde, III: Ergebnisse beim Meerschweincen nach Verabreichung von Cortison und Hydrocortison. Z Zellforsch 1969; 93:126–138
29. Armanini MP, Hutchins C, Stein BA, Sapolsky RM: Glucocorticoid endangerment of hippocampal neurons is NMDA-receptor dependent. Brain Res 1990; 532:7–12
30. Wooley CS, Gould E, McEwen BS: Exposure to excess glucocorticoids alters dendritic morphology of adult hippocampal pyramidal neurons. Brain Res 1990; 531:225–231
31. Zola-Morgan S, Squire LR, Amaral DG: Lesions of the amygdala that spare adjacent cortical regions do not impair memory or exacerbate the impairment following lesions of the hippocampal formation. J Neurosci 1989; 9:1922–1936
32. Zola-Morgan SM, Squire LR: The primate hippocampal formation: evidence for a time-limited role in memory storage. Science 1990; 250:288–290
33. Zola-Morgan S, Squire LR, Amaral DG, Suzuki WA: Lesions of perirhinal and parahippocampal cortex that spare the amygdala and hippocampal formation produce severe memory impairment. J Neurosci 1989; 9:4355–4370
34. Squire LR, Zola-Morgan S: The medial temporal lobe system. Science 1991; 253:1380–1386
35. Milner B: Psychological aspects of epilepsy and its neurosurgical management, in Advances in Neurology 8. Edited by Purpura DP. New York, Raven Press, 1975
36. Delaney RC, Rosen AJ, Mattson RH, Novelly RA: Memory function in focal epilepsy: a comparison of non-surgical, unilateral temporal lobe and frontal lobe samples. Cortex 1980; 16: 103–117
37. Lencz T, McCarthy G, Bronen RA, Scott TM, Inserni JA, Sass KJ, Novelly RA, Kim JH, Spencer DD: Quantitative magnetic resonance imaging in temporal lobe epilepsy: relationship to neuropathology and neuropsychological function. Ann Neurol 1992; 31:629–637
38. Sass KJ, Spencer DD, Kim JH, Westerveld M, Novelly RA, Lencz T: Verbal memory impairment correlates with hippocampal pyramidal cell density. Neurology 1990; 40:1694–1697
39. Sass KJ, Lencz T, Westerveld M, Novelly RA, Spencer DD, Kim JH: The neural substrate of memory impairment demonstrated by the intracarotid amobarbitol procedure. Arch Neurol 1991; 48:48–52
40. Milner B: Visual recognition and recall after right temporal-lobe excision in man. Neuropsychologia 1968; 6:191–209
41. Scoville W, Millner B: Loss of recent memory after bilateral hippocampal lesions. J Neurol Neurosurg Psychiatry 1957; 20:11–21
42. Bremner JD, Seibyl JP, Scott TM, Southwick SM, Delaney RC, Mason JW, Scanley E, McCarthy G, Charney DS, Innis RB: Decreased hippocampal volume in posttraumatic stress disorder, in New Research Program and Abstracts, 145th Annual Meeting of the American Psychiatric Association. Washington, DC, APA, 1992
43. Spitzer RL, Williams JBW, Gibbon M: Structured Clinical Interview for DSM-III-R (SCID). New York, New York State Psychiatric Institute, Biometrics Research, 1987
44. Keane TM, Caddell JM, Taylor KL: Mississippi Scale for Combat-Related Posttraumatic Stress Disorder: three studies in reliability and validity. J Consult Clin Psychol 1988; 56:85–90
45. McClellan AT, Luborsky A, Cacciola J, Griffith J, Evans F, Barr HL, O'Brien CP: New data from the Addiction Severity Index: reliability and validity in three centers. J Nerv Ment Dis 1985; 173:412–423
46. Keane TM, Fairbank JA, Caddell JM: Clinical evaluation of a scale to measure combat exposure. Psychol Assessment 1989; 1:53–55
47. Russell E: A multiple scoring method for the assessment of complex memory functions. J Consult Clin Psychol 1975; 43:800–809
48. Buschke H, Fuld PA: Evaluating storage, retention, and retrieval in disordered memory and learning. Neurology 1974; 24:1019–1025
49. Hannay HJ, Levin HS: Selective Reminding Test: an examination of the equivalence of four forms. J Clin Exp Neuropsychol 1985; 7:251–263
50. Prevey ML, Delaney RC, Mattson RH, Tice DM: Feeling-of-knowing in temporal lobe epilepsy: monitoring knowledge inaccessible to conscious recall. Cortex 1991; 27:1–13
51. Loring DW, Lee GP, Meador KJ, Smith JR, Martin RC, Ackell AB, Flanigan HF: Hippocampal contribution to verbal recent memory following dominant-hemisphere temporal lobectomy. J Clin Exp Neuropsychol 1991; 13:575–586
52. Parkinson JK, Murray EA, Mishkin M: A selective role for the hippocampus in monkeys: memory for the location of objects. J Neurosci 1988; 8:4159–4167