

MEMORY AND ITS REORGANIZATION IN TEMPORAL LOBE EPILEPSY

Preserved Verbal Memory Function in Left Medial Temporal Pathology Involves Reorganization to Right Medial Temporal Lobe

Richardson MP, Strange BA, Duncan JS, Dolan RJ

Neuroimage 2003;20:S112–S119

The left hippocampus and related structures mediate verbal memory function. The mechanism underlying preserved verbal memory function in patients with left hippocampal damage is unknown. Temporal lobe epilepsy, a common disease, is frequently the consequence of a characteristic hippocampal pathology termed *hippocampal sclerosis*, which also may affect the amygdala. In this setting, mapping the sites of memory function is a vital component of planning for surgical treatment for epilepsy. Using event-related functional magnetic resonance imaging, we studied 24 right-handed nonamnesic patients with left hippocampal sclerosis and 12 normal controls, performing a verbal encoding task. The patients were subdivided into two groups according to presence or absence of additional left amygdala pathology. Analysis of the data used a two-level random-effects design, examining the main effects of subsequent memory in each group, as well as the differences between the groups. Additional effects of emotionality of the remembered words also were examined. Verbal memory encoding involved activation of left hippocampus in normal subjects, but was associated with reorganization to right hippocampus and parahippocampal gyrus in the patients. The additional presence of left amygdala sclerosis resulted in reorganization for encoding of emotional verbal material to right amygdala. Retained verbal memory function in the presence of left medial temporal lobe pathology is mediated by recruitment of a parallel system in the right hemisphere, consistent with adaptive functional reorganization. The findings indicate a high degree of plasticity in medial temporal lobe structures.

COMMENTARY

A favorable *cognitive* outcome of left (speech dominant) anterior temporal lobectomy (ATL) includes minimal decline in verbal memory. Verbal learning, especially memory, is known to be dependent on a distributed neuronal system, with a particularly important role played by the left hippocampus. Verbal memory ability may be depressed preoperatively in candidates for left ATL, especially in the context of hippocampal sclerosis. But these patients are not *amnesic* for verbal information, suggesting that some functional capacity remains. In addition, an ensuing lack of change in verbal memory after complete left hippocampectomy clearly suggests that reorganization of function may have taken place. But reorganized to where?

One might immediately suspect that reorganization is apt to occur ipsilaterally within the speech dominant hemisphere. But the interesting functional imaging study of Richardson et al. suggests that in the context of a damaged left mesial temporal system, the right mesial temporal system, including hippocampus and parahippocampal gyrus, may contribute to the encoding of verbal information. In addition, the amygdala is known to play an important role in helping to remember affective-laden information. Again, Richardson et al. demonstrate that the right amygdala appears to assume a role in the mediation of emotional verbal material in the context of damage (sclerosis) to the left amygdala.

The highly creative paradigm developed by Richardson et al. is quite different from conventional, clinical verbal memory test procedures in which the input is auditory (i.e., words or passages are read to the patient for later recall) not visual, as is the case in this study. It will be important to know whether similar results, regarding reorganization of function, could be obtained by using more conventional clinical memory-assessment procedures with auditory input. Other interesting conceptual questions arise. If the right hippocampus becomes a mediator of verbal information, is there a “crowding out” of its presumed ability to mediate retention of nonverbal material? Is age specificity present in this process, with less reorganization of function appearing in late-onset, left temporal lobe epilepsy patients? Although the patients of Richardson et al. were right-handed, they did not undergo intracarotid amobarbital testing to demonstrate unequivocally



left-hemisphere speech dominance. Did these subjects have some degree of right-hemisphere speech function underlying the reorganization of verbal memory encoding to the right mesial region? All in all, this very carefully performed and cautiously interpreted investigation raises a host of interesting con-

ceptual, clinical, and methodologic issues that can be pursued by using either functional imaging or conventional clinical testing procedures.

by Bruce Hermann, Ph.D.