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Task-Specific Interictal Spiking: Adding to the Complexity

Spatial and Episodic Memory Tasks Promote Temporal Lobe Interictal Spikes

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Reflex epilepsies have been demonstrated to exploit specific networks that subserve normal physiological function. It is unclear whether more common forms of epilepsy share this particular feature. By measuring interictal spikes in patients with a range of epilepsies, we show that 2 tasks known to specifically engage the hippocampus and temporal neocortex promoted increased interictal spiking within these regions, whereas a nonhippocampal dependent task did not. This indicates that interictal spike frequency may reflect the processing demands being placed on specific functional–anatomical networks in epilepsy.

Commentary

Memory impairment is a major consequence of epilepsy and is now recognized as a core feature of many epileptic syndromes.[1] Although traditional neuropsychological testing provides a means to grossly identify the presence and location of abnormal networks underlying memory and cognitive disturbances in clinical samples, it is limited in its ability to characterize underlying mechanisms of memory disturbance. The field has turned to functional imaging and electrophysiological studies to investigate the neurophysiological basis of memory and to help determine how and why memory is impaired in patients with epilepsy.

There has been a surge of interest in studying the effects of interictal epileptiform discharges (IEDs) on memory functioning and their influence on the impairment observed in patients with epilepsy.[2] However, the results of investigations performed thus far have been somewhat conflicting. While some studies have demonstrated that IEDs can have a negative influence on cognitive performance,[3] others have shown that cognitive performance can exert a negative influence on rate of IEDs.[4] As a result, it has been difficult to determine what role, if any, IEDs play in various stages of memory processing. What has emerged from the existing literature is a complex picture involving likely interactions among testing paradigms, temporal factors, neuroanatomic location, and underlying disease processes.

The study by Vivekananda and colleagues[5] addresses the question of whether specific cognitive task demands influence expression of IEDs in specific neuroanatomic networks. The investigators studied 2 groups of 12 patients undergoing intracranial electroencephalographic monitoring for clinical purposes. They chose a set of spatial and episodic memory tasks shown in other studies to dependent on hippocampal functioning. Recordings from the hippocampus, lateral temporal lobe, and amygdala obtained during performance of those 2 tasks were compared to those obtained during an attentional bias task not considered to be dependent on hippocampal functioning.

The results showed increasing frequency of IEDs from the hippocampus during performance of the spatial memory task with similar increases in frequency observed in recordings from both the hippocampus and lateral temporal lobe during the episodic memory task. No increase in IEDs from any temporal lobe recording sites was obtained during the attentional bias task. In terms of processing stages, there was no difference in IED frequency during encoding or retrieval trials of the spatial memory task. However, an increased frequency of discharges from the hippocampus and lateral temporal lobe was observed during the encoding stage of the episodic memory task. The authors found no association between IED frequency and performance on either task and no relationship with the laterality of the recording or the anatomic location of the seizure focus.

It is noted that this is the first study reporting task-related increases in IED activity in specific anatomic regions, suggesting that this activity might reflect network engagement in cognitive tasks in patients with epilepsy. The authors emphasize the importance of controlling for the effects of these task-related changes in analysis of any higher order changes in oscillatory power with the implication that this finding represents yet another factor to address when attempting to decipher the ultimate role that IEDs might

play in explaining the occurrence of memory dysfunction associated with epilepsy.

The authors indicate that they cannot rule out the possibility that the task-dependent increases in IED frequency observed in their study are the result of other factors, including cognitive effort or the nature of the test stimuli. It is important to note that the tasks used in this study were experimental measures chosen for their specificity to hippocampal engagement, which makes them far different from the types of tests traditionally used in clinical settings and in prior studies investigating the effects of IEDs. Their reporting of task-specific effects might also reflect decreased power to detect changes in the much smaller subset of individuals completing the attentional bias task.

The ultimate goal is to come to a better understanding of the electrophysiological basis of memory functioning and to produce findings that will inform strategies to improve our ability to assess and treat memory disturbances in patients with epilepsy. This study's immediate contribution to that goal is difficult to ascertain, given the fact that there was no observed relationship between IEDs and task performance or the anatomic location of the epileptic focus. However, the finding of task-specific effects on IED frequency make a significant contribution from a methodological standpoint, indicating that this is something that clearly needs to be taken into account in analyses performed in future studies.

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