Epileptic seizures provide a complex contrast to lesions. Destructive lesions typically cause functional deficits. However, the activities of preserved cortical and subcortical areas, released from the influences of the destroyed or impaired tissue, can paradoxically cause focal hyperactivities. Seizures result from excessive synchronous neural discharges that can cause positive (hyperactive such as jerking) or negative (functional impairment such as weakness) changes. Lesions and seizures both provide valuable, but often not straightforward, insights into brain function. While the correlation between disease and abnormal function is often easily made, the extrapolation from disease to normal function is much more tenuous.

Hughling Jackson’s localization of focal motor seizures to primary motor cortex was a remarkable leap for neurology, but his localization of déjà vu, olfactory hallucinations, and dreamy states to the mesial temporal lobe was a larger jump (Jackson, 1931). He recognized in the dreamy states the dual nature of a seizure’s effects on mind. There is not always loss, but there is, I believe, always, at least defect, of consciousness coexisting with over-consciousness (“dreamy state”). Just as motor seizures can be excitatory and cause clonic or tonic activity, seizures arising from limbic and associative cortices can cause positive or negative symptoms. When a seizure evokes fear, the paroxysmal occurrence of the emotion without an environmental context is readily identified by the patient. However, a transient impairment of the experience of fear would be much more difficult to detect. It would require that a fearful stimulus occur during the seizure and that the lack of responsiveness be recognized. Not surprisingly, many ictal deficits are often difficult to recognize. Motor and language deficits are most commonly detected, yet negative motor seizures may impair only complex movements, similar to effects of electrical stimulation (Luders et al., 1988). Thus, if the seizure occurs while