Abstract

For five or six decades we have known that small dominant thalamic hemorrhages or infarcts cause aphasia and that stimulation of the dominant pulvinar or ventral anterior nucleus interrupts naming. Acutely, dominant thalamic lesion tends to cause (1) fluent output with frequent paraphasias, primarily semantic in nature and sometimes deteriorating into jargon, (2) unimpaired or minimally impaired repetition, and (3) auditory-verbal comprehension less impaired than this kind of output usually would indicate, especially in posterior thalamic hemorrhagic lesions. Within three to six months, recovery of language functions is usually good, though incomplete. After presenting cases of thalamic aphasia, mechanisms that may account for this pattern of symptoms and recovery will be discussed, and the importance of understanding the role of the thalamus in language will be addressed.

