

COMMENT

The results point toward a possible deleterious effect on memory of standard anticholinergic therapy in parkinsonian patients, reaching statistical significance in one test of intermediate memory and a similar trend in another. The digit span test for immediate memory was unaffected. Although the investigation was not double blind, the fact that medication apparently affected only some of the tests and not others may be taken to imply that observer bias was not crucial in evaluating results. The contention that reduced cerebral levels of acetylcholine are associated with impaired memory function seems to be supported.

Comparison with a group of normal

age-matched subjects shows that parkinsonian patients are at an initial disadvantage in intermediate memory capacity, although immediate capacity is not impaired. This is of interest in view of the observation that in normal subjects physostigmine injections enhanced performance in tests of long-term memory and retrieval while not affecting short-term results.²

It has also been noted that the beneficial effects on memory of measures presumed to increase cerebral acetylcholine levels, as well as the deleterious effects of administering anticholinergic drugs, are more marked in normal persons whose pretreatment scores were relatively low.⁴ In light of their poor basic performance compared with that of normal

subjects, it may be concluded that the use of anticholinergic drugs should be carefully considered in parkinsonian patients, especially those still engaged in occupations in which the function of memory is important.

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Aphasia After Right Thalamic Hemorrhage

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● A 53-year-old hypertensive, converted left-handed man manifested aphasia after a right thalamic hemorrhage. The syndrome of mild, fluent aphasia, with naming difficulty and occasional paraphasic errors, but intact comprehension and repetition, is similar to that of previously reported patients with left thalamic lesions. The occurrence of aphasia after right thalamic hemorrhage suggests that right-hemisphere language dominance in left-handed subjects can extend even to the level of the thalamus.

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Increased attention has been directed in recent years to aphasic syndromes resulting from subcortical lesions, especially left thalamic hemorrhage,^{1,7} left thalamotomy,^{8,12} and most recently ischemic strokes in the vicinity of the left basal ganglia and internal capsule.¹³⁻¹⁵ These lesions spare the perisylvian, frontotemporal language cortex, the traditional site of damage in aphasia. The involvement of left-hemisphere subcortical centers

in language processes has thus changed our understanding of the organization of language in the brain. We recently encountered a left-handed patient with aphasia secondary to spontaneous hemorrhage into the right thalamus.

REPORT OF A CASE

A 53-year-old, college-educated man was admitted to a local hospital after the abrupt onset of numbness and weakness of his left arm and leg. He had been in excellent health except for hypertension, with no medical history of stroke or transient ischemic attacks. He wrote with his right hand but described being "converted" from left-handedness in childhood. At the local hospital, he was hypertensive (BP, 200/120 mm Hg), with a flaccid left hemiparesis, somnolence, persistent headache, and nausea with frequent retching, requiring placement of a nasogastric tube.

On transfer to the Nashville (Tenn) Veterans Administration Medical Center two days after onset, he was lethargic but arousable, spoke fluently with word-finding pauses and circumlocutions, and followed simple commands. He read sentences aloud, performed simple calculations, copied geometrical figures, drew a clock, and showed no neglect of the left side. He had good insight and normal emotional expression. The cranial nerve examination disclosed only moderate weakness and decreased sensation of the left side of the face. There was no visual field defect to confrontation. He had a flaccid left hemiplegia, except for slight

movement of the left toes, and a dense hemisensory loss to pin and touch involving the left side of the face, extremities, and trunk to the midline. Vibration and position sense were nearly absent in the left arm and leg. Strength and sensation were normal on the right. The left plantar response was extensor, the right flexor.

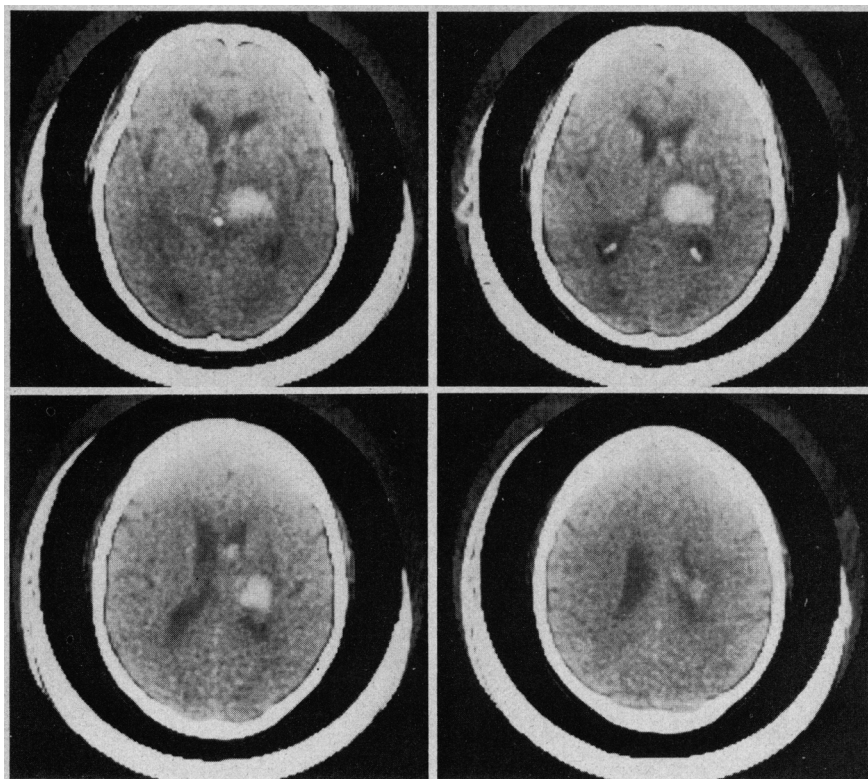
During the next two days, the patient became more alert and began to regain some use of his left extremities. A computed tomographic scan five days after onset (Figure) showed a large right thalamic hemorrhage. He was given dexamethasone, and his condition continued to improve. Subsequent examinations disclosed occasional paraphasic errors (eg, "belt" for "watchband") and persistent but improving difficulty in naming, of which the patient himself complained. Comprehension and repetition were normal. There was no longer any drowsiness or psychomotor retardation. The left hemiparesis improved very gradually to the point of the patient's walking with assistance, but the profound left hemisensory deficit persisted largely unchanged to the time of discharge, six weeks after admission.

The patient was evaluated with the Porch Index of Communicative Ability (PICA)⁶ and the Boston Diagnostic Aphasia Examination (BDAE)¹⁷ 17 and 28 days after onset, respectively. There was a mild, fluent aphasia, with intact auditory comprehension and mild deficits in spontaneous speech, naming, and writing. Performance on the PICA was at the 92nd percentile for aphasics with left-hemisphere lesions, with mean scores showing only mild deficits on all tasks except the writing

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Computed tomographic scan of head shows large right thalamic hemorrhage.

subtests. On the BDAE, occasional literal and paraphasic errors were recorded. Hesitancy and delays were noted on the naming subtests, but responsive naming, body part naming, and picture naming were nearly errorless. There was decreased fluency on animal naming (nine animals in 60 s; normal, 18). Subtests for fluency, auditory comprehension, repetition, oral reading, automatic speech, reading comprehension, and singing and rhythm all gave normal results or showed mild impairment, except for long latencies and occasional paraphasic errors. Writing was mechanically deficient, but intact for spelling and grammatical constructions.

COMMENT

Aphasic syndromes with lesions involving the deep structures of the left hemisphere have been clearly established. Several reports²⁻⁷ have described a syndrome of fluent aphasia with paraphasic errors, dysnomia, and preserved comprehension and repetition in patients with left thalamic hemorrhage. Mohr et al³ called attention to the fatigability of such patients; there is a rapid fluctuation between an alert state, with nearly normal language function, and an obtunded state, with marked paraphasias and perseveration. Luria¹⁸ suggested that the language disorder of left thalamic lesions may be more closely related to a "partial disturbance of vigilance" involving verbal

processes than to a true aphasia. Some authors^{1,3} have also suspected that thalamic hemorrhages may cause aphasia by pressure on adjacent structures such as the temporal isthmus.

A specific role of the thalamus in language function is suggested by the occurrence of aphasia in other left thalamic lesions including thalamic tumors,¹⁹ thalamotomy,⁸⁻¹² and surgical removal of a thalamic arteriovenous malformation.¹⁸ Postmortem studies of thalamotomy patients have confirmed that even small lesions restricted to the left ventrolateral nucleus can be associated with mild but persistent aphasia.¹¹ Disruption of short-term memory and object naming have also been noted during electrical stimulation of the left pulvinar²⁰ and ventrolateral nucleus.²¹ Some authors,^{2,4} indicating the known connections between the ventrolateral nucleus and the frontal cortex and between the pulvinar and posterior cortical areas, have suggested that thalamic lesions might produce aphasia by disrupting the activation of cortical language centers.

To the best of our knowledge, our patient is unique among reported cases of aphasia secondary to thalamic hemorrhage in that the lesion was in the right hemisphere. Alexander and LoVerme⁷ found no such cases

either in their own experience or in their review of aphasia with intracerebral hemorrhages. Reported cases of aphasia after thalamotomy have also included either unilateral left or bilateral lesions, with the single exception of a left-handed woman described by Bell,¹⁰ in whom a probable hemorrhage complicated a right cryogenic thalamotomy. This patient had severe aphasia, impairment of reading and writing, left hemiplegia, and visual inattention to the left. No evidence was presented to exclude damage to cortical structures.

The right thalamic hemorrhage in our patient is analogous in size and location to the left thalamic hemorrhages causing aphasia in dextrals.^{3,5,7} We presume that our patient was one of the minority of sinistrals who manifest language dominance in the right hemisphere.²² The hemorrhage could produce aphasia either by local disruption of the thalamus or by pressure on the adjacent right temporal lobe. In the case of left thalamic hemorrhage, as already reviewed, the presence of similar aphasias after surgical lesions⁸⁻¹² affecting the thalamus would suggest that direct damage to the thalamus is at least partially responsible for the aphasia. An additional pressure effect on the right temporal isthmus, however, cannot be excluded.

The occurrence of aphasia after right thalamic hemorrhage in our patient implies that language dominance in the right hemisphere of sinistrals may extend even to the thalamic level. The right-hemisphere language cortex in sinistrals could theoretically make connections to subcortical centers by one of two routes: either directly to right-hemisphere subcortical centers, or indirectly, via the corpus callosum to the left-hemisphere language cortex, and then to left subcortical structures. The former pathway appeared to be operative in our patient. Aphasia has not yet been reported with lesions of other right-hemisphere subcortical structures, but only recently has attention been drawn to aphasia after ischemic infarction of the left caudate, putamen, and internal capsule.¹³⁻¹⁵ Ross et al²³ recently reported a case of infarction of the right internal capsule with "motor aprosodia," or loss of the emotional tone of expressive speech. They concluded that, because the lesion lay below the level of corticocortical connections via the corpus callosum, the integration of left hemisphere-mediated propositional speech and right hemisphere-mediated affective

speech must occur at a subcortical, perhaps brain stem level.

A better understanding of the importance of subcortical centers to the organization of language function in the brains of both sinistrals and dextrals awaits further study of patients with subcortical lesions. Investigation of those uncommon patients with subcortical infarcts or surgical lesions, rather than hemorrhages, should help to clarify the relative importance of local subcortical damage *v* indirect pressure effects on the temporal lobes in producing aphasic disturbances.

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Bent-Knee Pulling in the Diagnosis of Upper Lumbar Root Lesions

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● The pain pattern of patients with upper lumbar root lesions can be elicited by a simple diagnostic test called bent-knee pulling, in which the examiner pulls the half-prone patient's bent knee backward while putting forward pressure on the buttock. The technique was tested in seven patients with either clinically, myelographically, or electromyographically demonstrated L-3 and/or L-4 root lesions, and it successfully reproduced the pain in all seven patients. In all patients the result of straight-leg raising was negative.

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The usefulness of the straight-leg-raising test for the bedside diagnosis of L-5 and S-1 root lesions has been

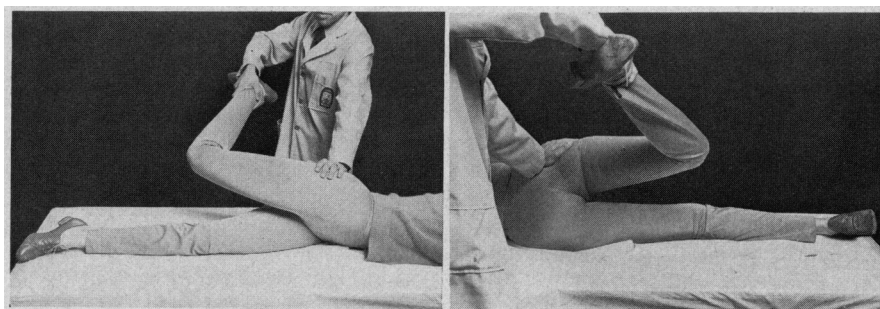
long recognized. Little attention has been given, however, to bedside tests for the diagnosis of L-2, L-3, and L-4 root lesions. Such tests have been described but have not gained wide acceptance and are infrequently mentioned in discussions of upper lumbar root lesions. We discuss the reasons that these tests have not been more widely used, assess their specificity,

and describe a technique that is easier to perform and that puts more stress on these roots.

METHODS

The patient lies half prone with his back to the examiner. Holding the leg by the foot, the examiner pulls the leg back with the knee flexed. With his other hand, he pushes forward on the patient's buttock, so that the hip is thrust anteriorly (Figure).

Technique of bent-knee pulling: left, anterior view; right, posterior view.



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