

Case Report

Aphasia in Thalamic Stroke: CT Stereotactic Localization

Carol R. Archer, Igor A. Ilinsky, Peggy R. Goldfader, and Kenneth R. Smith, Jr.

Abstract: We present a patient in whom dysphasia followed suddenly upon an apparently discrete thalamic infarct proven by computed tomography (CT). Detailed psychometric data of the patient's speech and memory disorder obtained during the acute and chronic stages were correlated with the evaluation of a focal thalamic lesion as demonstrated by serial CT. The findings and deductions drawn from selective reports in the literature form the basis of this presentation. **Index Terms:** Aphasia—Brain, infarction—Computed tomography.

Although opinions had polarized sufficiently to provoke lively and controversial debate between the localizationists and the holistic school of thought, systematic investigation into the causes of aphasia did not begin until 1861. In that year Paul Broca published a case report describing a patient who suddenly became speechless and died. Post-mortem examination of the brain demonstrated a large frontal lobe lesion. Then in 1874 Wernicke's work linked receptive aphasia to a lesion in the posterior temporoparietal area. Further studies by neurologists and neuroanatomists explored the subcortical connections between these areas and incriminated lesions in fasciculus arcuatus. As a challenge to the growing preoccupation with the deficit-anatomic premise, Hughlings Jackson reintroduced the holistic concept of brain function. This concept wielded more and more influence under the directions of such important advocates as Pierre Marie, Head, Pick, Wilson, Weisenburg and McBride, and many others.

As Benson so ably points out (1), "Many influential workers are linked theoretically with one approach but produce meaningful work in the other." This insight, shared by a number of workers, has stimulated further research efforts into areas as, for example, the role of subcortical areas in the production of speech. Reports of aphasic disorders in

patients with left thalamic tumors (2), left thalamic hemorrhage (3-9), and left thalamotomies (10,11) would appear to link the thalamus to the mechanisms of speech. Electrical stimulation studies of the left ventral lateral nucleus of the thalamus may evoke alterations in object naming and verbal recall (12-14).

We present a patient in whom dysphasia and a mild right hemiparesis followed suddenly upon an apparently discrete thalamic infarct proven by computed tomography (CT). Detailed psychometric data of the patient's speech and memory disorder obtained during the acute and chronic stages were correlated with the evolution of a focal thalamic lesion as demonstrated by serial CT scans. The findings and deductions drawn from selective reports in the literature form the basis of this presentation.

HISTORY

On March 20, 1978, a 57-year-old right-handed man experienced sudden difficulty speaking and transitory right-sided weakness of 20 min duration. He remained alert and experienced no visual or sensory symptoms. Detailed CT and psychometric examinations were first performed 2 weeks after the onset of this illness and repeated 6 months later.

RESULTS

Computed Tomography Examination

All CT examinations were obtained with the orbitomeatal line perpendicular to the table top. Slice thickness was 5 mm, and 3 mm overlap technique

From the Departments of Radiology (C. R. Archer), Anatomy and Surgery (I. A. Ilinsky), Neurology (P. R. Goldfader), and Surgery, Section of Neurosurgery (K. R. Smith, Jr.), St. Louis University Medical Center, 1325 South Grand Boulevard, St. Louis, Missouri 63104. Address correspondence and reprint requests to Dr. Archer.

was used in order to obtain rearrangement of the data in the sagittal plane.

Acute Phase: Two Weeks After Onset

Computed tomography examination. The pre-contrast examination was normal. The postcontrast examination showed an area of enhancement in the anterior one-half of the left thalamus. Computer rearrangement of the data was obtained in the sagittal plane beginning at the midline and passing to the left by 1 mm increments to the lateral margin of the lesion.

Stereotactic localization. The three-dimensional coordinate system used to localize specific thalamic nuclei is based on two lines, the intercommissural line (CA-CP) and the midline. These reference lines and coordinates are common to several stereotactic atlases (15–17).

On the axial transverse CT scan, the midline was drawn as a line bisecting the third ventricle (Figs. 1 and 3). From this line, the mediolateral coordinate (ML) of the lesion was determined in millimeters by direct measurement from the midline to the center of the lesion. This measured 6.5 mm from the midline. An appropriate plate representing a plane 6.5 mm from the midline was then selected from the stereotactic atlas of Schaltenbrand and Wahren (17). This was used for localization of the lesion as described below.

The anteroposterior (AP) and superoinferior (H) coordinates of the lesion were obtained from the sagittal reconstruction of the CT scan. The CA-CP line is a line connecting the anterior and posterior commissures. The midline sagittal CT scan with the outline of the third ventricle and the CA-CP line were superimposed on the CT scan in a sagittal plane demonstrating the lesion at its maximal size. The AP coordinate was determined in millimeters from the anterior commissure and the H coordinate in millimeters from the CA-CP line. The center of the lesion was located at a distance 6.5 mm from the midline. The lesion was spherical and extended medially to the wall of the third ventricle (Fig. 1). The anterior margin of the lesion was 3.0 mm from the anterior commissure and extended 14.0 mm posteriorly, thus occupying the anterior part of the thalamus above the CA-CP line (Fig. 1).

According to the sagittal plate 6.5 mm from the midline (Fig. 2) in the atlas of Schaltenbrand and Wahren (17), which utilized Hassler's classification of the thalamus, the nuclei involved are as follows: (a) nucleus lateropolaris thalami (Lpo), which corresponds to the nucleus ventralis anterior (VA); (b) a small part of the nucleus ventro-oralis anterior (Voa) or anterior subdivision of the ventral lateral nucleus (VL); and (c) nucleus reticularis thalami (RT) and adjacent part of the genu and posterior limb of the internal capsule (IC).

Chronic Phase: Two and Six Months After Onset

Computed tomography examination. The CT examinations were performed before and after contrast medium administration and revealed an area of decreased density in the left thalamus. No contrast enhancement was noted. Compared to the acute phase, the lesion was smaller and occupied a more lateral and posterior position.

Stereotactic localization. Localization of the lesion was accomplished using the same technique as described above. The center of the lesion in the mediolateral direction was located 11.0 mm from the midline (Fig. 3). The lesion was again located above the CA-CP line and was 7.0 to 16.0 mm from the anterior commissure (Fig. 3). An appropriate sagittal atlas plate was selected for localization of the lesion (Fig. 4). The lesion was found to occupy the following sites: (a) the nucleus ventro-oralis anterior (Voa) and nucleus ventro-oralis posterior (Vop), and nucleus reticularis (RT). In other words, the lesion occupied the entire ventrolateral nucleus (VL), as well as the adjacent part of the posterior limb of the internal capsule (IC).

Psychometric Evaluation

Acute Phase: Two Weeks After Onset

The test data (Table 1) revealed an overall decrement in higher cortical functions characterized by fluent paraphasic and perseverative speech, reduction in auditory comprehension, inability to repeat material read, impaired visual and verbal recall, and mild difficulty with visuospatial organization. Moment-to-moment fluctuations in attention were striking.

Chronic Phase: Two and Six Months After Onset

Over the ensuing 2 months, the patient gradually improved. On formal testing, auditory comprehension and spontaneity had improved remarkably, and perseveration and moment-to-moment fluctuations in attention were less marked. The patient could name objects with greater facility but continued to have difficulty in naming their component parts. He remained unable to relate the contents of short paragraphs (logical sequences) and was still unable to learn related word pairs such as metal/iron. Visual recall and constructional abilities were approaching premorbid levels. Thus, at 2 months, the overall decrement in higher cortical functioning noted initially had resolved into a mild anomia and impaired verbal recall. Improvement continued and testing 6 months after onset revealed only impairment of verbal recall.

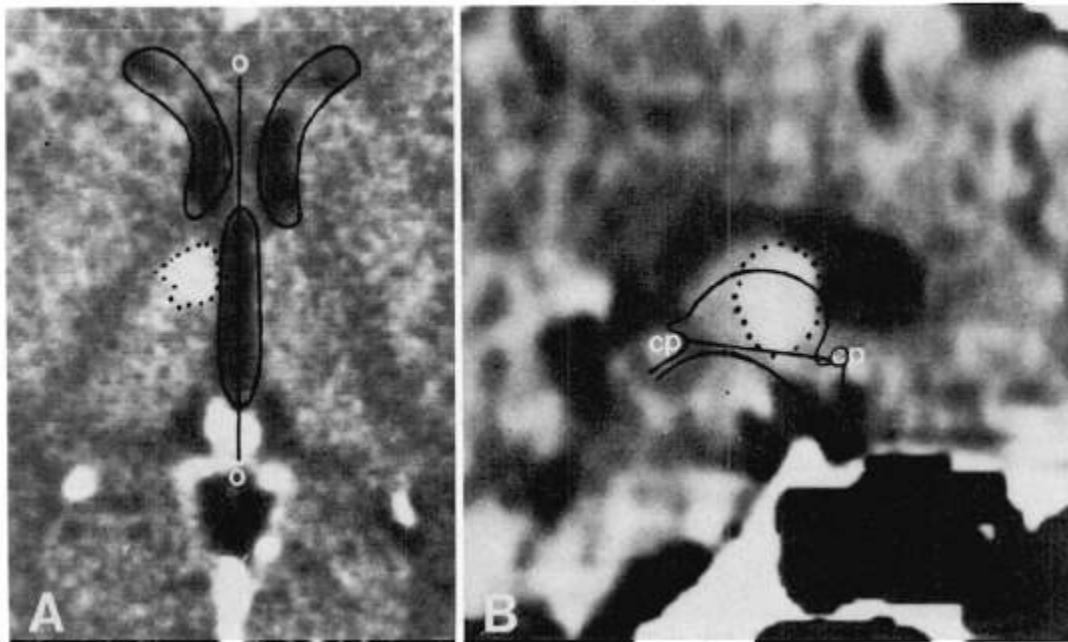


FIG. 1. CT scans in axial transverse (a) and sagittal (b) planes performed 2 weeks after thalamic infarct. The ventricles are outlined in the axial transverse scan. The outlines of the third ventricle as seen on the midline scan are superimposed on the 6.5 mm sagittal scan (see text for explanation). Dots outline the lesion. Magnification, 1:1

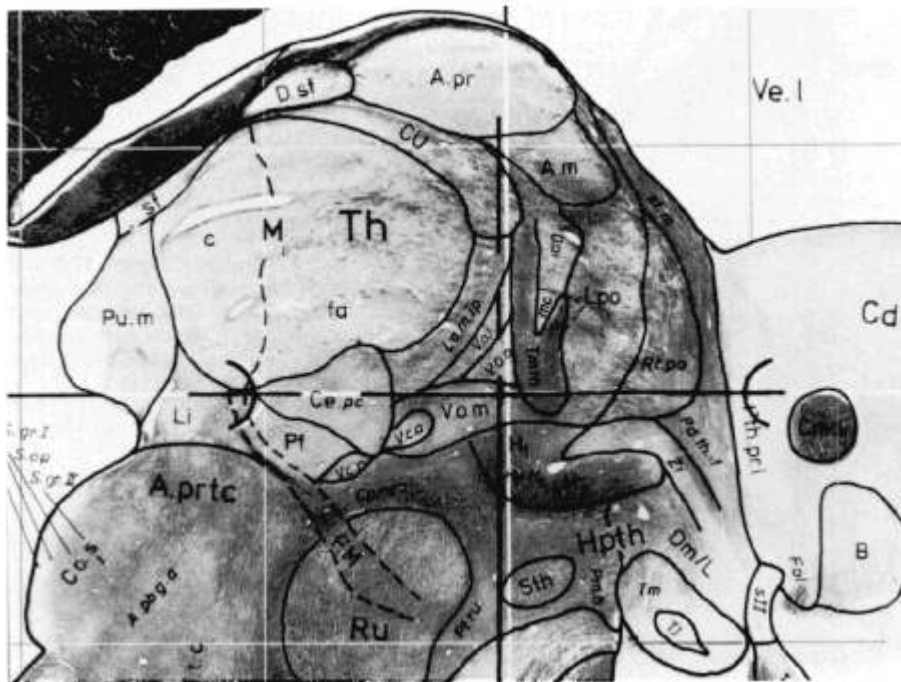


FIG. 2. The sagittal plate 6.5 mm from the midline from the stereotactic atlas of Schaltenbrand and Wahren (17), which corresponds to the sagittal CT scan in Fig. 1b.

DISCUSSION

Disorder of speech and verbal recall have been reported in patients with thalamic lesions (5,6,13, 18). Although cerebral hemorrhage or tumor may appear confined to the thalamus, the secondary effects on adjacent tissues due to edema, direct pressure, or hypoperfusion are difficult, if not impos-

sible, to measure accurately and therefore have not been taken into account.

Electrode stimulation of specific thalamic nuclei prior to stereotactic thalamotomies provides a more controlled experimental situation (11,13,14). Anomia, perseveration, and impairment of verbal recall have been produced by stimulation of the ventrolateral nucleus and pulvinar of the left (dominant)

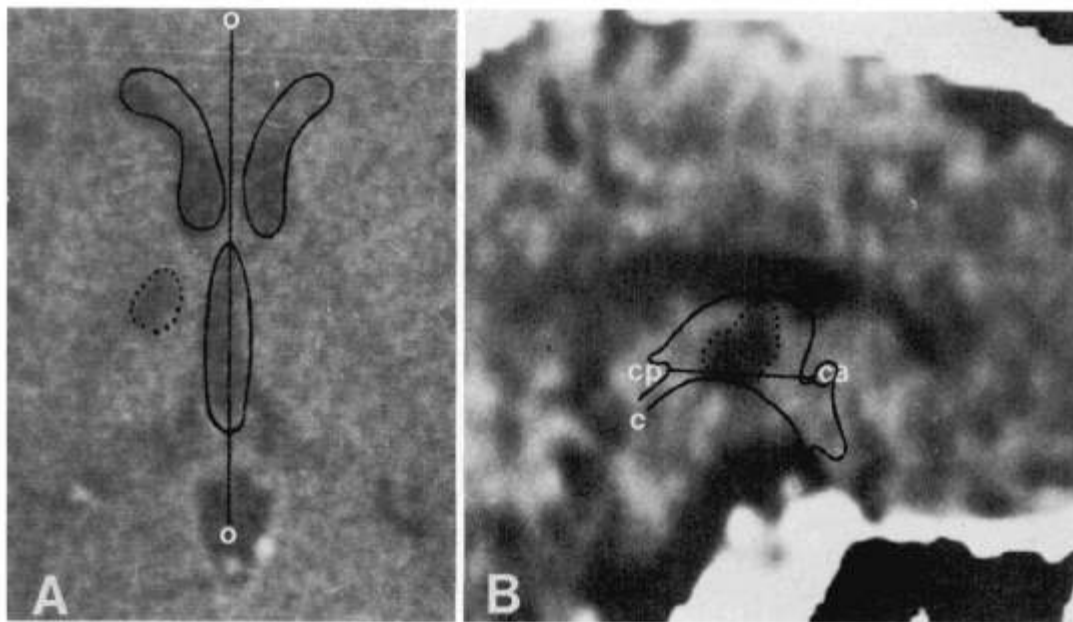


FIG. 3. CT scans in axial transverse (a) and sagittal (b) planes performed 6 months after thalamic hemorrhage. Designations are the same as in Fig. 1.

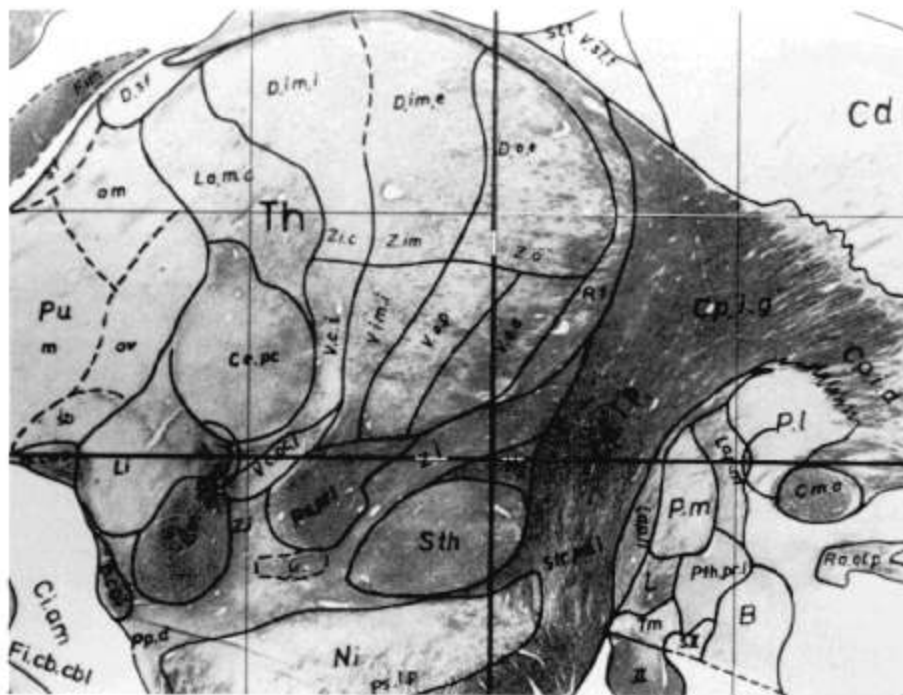


FIG. 4. The sagittal plate 12.0 mm from the midline from the stereotaxic atlas of Schaltenbrand and Wahren (17), which corresponds to the sagittal CT scan in Fig. 3b.

thalamus, while similar speech disorders were not produced by stimulation of the same areas on the right (8). It should be mentioned that discrete focal lesions in the dominant thalamus, however, usually produce only a temporary impairment of speech (8). This raises the question as to whether the aphasia is due to stimulation of the thalamus *per se* or to

blocking of impulses received by the cortex from the thalamus (18).

During the acute phase of the illness of our patient, a discrete lesion was identified on CT. The failure to detect evidence of cerebral edema or hypoperfusion involving areas adjacent to the left thalamus or more diffusely in the left cerebral hemi-

TABLE 1. Boston Diagnostic Aphasia Examination and a supplementary naming task

Task	Errors and types	
	2 Weeks postinfarct	6 Months postinfarct
Responsive naming	Errors: 3/10 items: unable to name items	Errors: 0/10
Visual confrontation naming	Errors: 4/6 objects: substitutions or no name	Errors: 0/6
Spontaneous animal naming	No names given	Named 10 animals
Naming fine parts of objects	Errors: 9/13 parts: semantic substitutions, perseveration, or no name given	Errors: 5/13 parts: semantic substitutions or no name given
Articulation	WNL	WNL
Repetition	WNL	WNL
Fluency	WNL	WNL
Reading	Substitutions, additions, omissions, perseveration, or unable to relate material	Unable to relate material read
Writing	Perseverations	WNL
Comprehension		
Word discrimination	Errors: 11/48: perseveration or no response	Errors: 0/48
Complex ideational problems	Errors: 9/12 problems	Errors: 3/12 problems
Wechsler Memory Scale		
Memory quotient	M.Q. = 63 (90–109 average)	M.Q. = 74
Logical passages	Recall: 0/46 facts	Recall: 2/46 facts
Visual reproductions	Recall: 5/14 points	Recall: 8/14 points
Paired associate learning	Meaningful pairs learned after three trials: 2/6	Three trials: 2/6
Wechsler Adult Intelligence		
Scale Scaled Score		
Verbal tests		
Similarities	0	0
Digit span	4	4
Vocabulary	4	7
Performance tests		
Block design	8	7
Object assembly	8	9

sphere, does not preclude their presence nor can we exclude the fact that they may have played a role in the production of the global impairment of intellectual functioning. Results of psychometric testing in the chronic stage can more reliably be attributed to the effects of the lesion itself. The most striking residual deficit was impairment of verbal recall. Mild anomia present 2 months after the ictus had resolved when the patient was tested 6 months later. It must not be concluded, however, that there is necessarily a causal relationship between the lesion in the ventrolateral nucleus of the thalamus and the final neuropsychological deficit. Disruption of thalamocortical pathways may be the mechanism by which these signs are produced (18).

In conclusion, CT combined with stereotactic localization allows for identification of specific

thalamic nuclei. Combined with careful neurological and neuropsychological evaluation, this technique may further the understanding of the function of the thalamus, specifically the role of the thalamus in speech and memory.

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