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Aphasia after Left Thalamic Infarction

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Medicine, Miami, provided expertise in determining the metabolic studies in case 1.

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Aphasia After Left Thalamic Infarction

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• We examined a 70-year-old woman who became aphasic after a left thalamic infarction. Computed tomographic scan showed injury that was largely limited to the ventral anterior and rostral ventral lateral thalamic nuclei. Speech was characterized by reduced voice volume, impaired auditory and reading comprehension, perseverations, intermittent use of jargon, fluctuations in the ability to perform confrontation naming, extraneous intrusions, verbal paraphasia, intact repetition skills, and fluent speech that was laconic but grammatically correct. We propose that the deficits after left thalamic injury can be grouped into the following four large clusters: *extrapyramidal deficits* (decreased or fading voice volume), deficits in *lexical access* (anomia, verbal paraphasia), deficits in *vigilance* (neologisms, intrusions, fluctuating performance, jargon, perseverations), and *comprehension* defects.

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Aphasia following dominant hemisphere thalamic injury has been reported with infarction,[1-4] hemorrhage,[1,5-14] tumor,[15-18] stereotaxic surgery,[19-21] electrostimulation,[22-24] arteriovenous malformation,[25] and abscess.[26] Descriptions of aphasic syndromes are varied. Doubters have argued that the resulting disturbance is an elemental speech disorder rather than true language dysfunction.[18] Pathologic and computed tomographic studies indicate that some lesions may extend beyond the thalamic borders to other subcortical and cortical language structures. We describe an infarction limited largely to the lateral-ventral thalamus associated with features of transcortical aphasia.

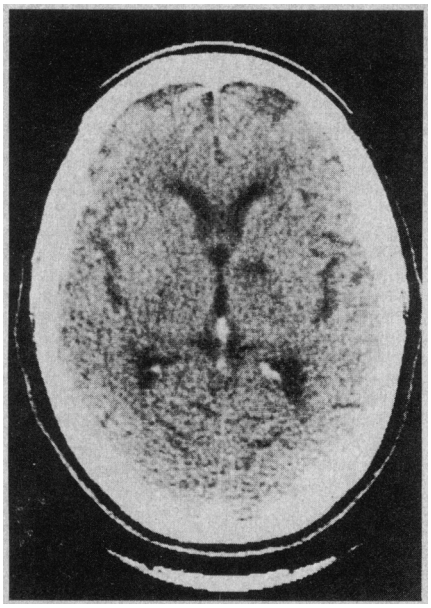
REPORT OF A CASE

A 70-year-old, right-handed woman was found wandering aimlessly on a street corner by the police in the early hours of the morning. She was last observed in her podiatrist's waiting room at 4 PM the day before. The police were notified when a relative who was to chauffeur her from the doctor's office reported her missing. Two days before the incident she had "snapped" angrily at her son, an uncharacteristic act for this individual. The night before her speech "did not sound exactly right." However, the following morning, she spoke normally. The police officer who found her described her speech as incoherent.

For the past several years, she had been a widow who lived independently in a retirement home for senior citizens. She rode the bus by herself to visit relatives and performed her own cooking, washing, and cleaning. Although she possessed only a grammar school education, she read daily newspapers and magazines. Recently her vision had been failing due to cataracts. She had insulin-dependent diabetes as well as hypertension treated with hydrochlorothiazide and methyldopa. She did not smoke cigarettes or drink alcohol and had no history of headaches.

On physical examination her BP was 180/100 mm Hg and the radial pulse rate was 80 beats per minute. There were no bruits of the head or neck and facial pulses were symmetrical. The results of the general physical examination were unremarkable.

The initial examination showed a paucity of spontaneous speech. When prodded, she produced fluent empty speech contaminated by neologisms, verbal and literal paraphasias. Voice volume was reduced. She could repeat "boy," "dog," "no *if*'s *and*'s, or *but*'s about it," and "he and she and I are here." She was able to follow commands such as "point to the ceiling," "close your eyes," and "bend your neck." When instructed to place three different-sized pieces of tissue paper on three separate articles of furniture, she picked up each individual tissue paper and touched it to her nose. She correctly named a watch, a pen, and a spoon but called a cup "another." When a fork was displayed, the patient picked it up, studied it, and then placed it down on her lunch plate. When asked "What is your daughter's name?" she replied, "Every name to your daughter." She referred to her telephone number as "Willis" and stated "Who is in your family?" when asked to name her family members. She could read only two words from a



Computed tomographic scan on third hospital day demonstrates nonenhancing lucency of left lateral-ventral thalamus.

simple paragraph at a third-grade level. When shown the word *herd* on an index card, she responded "rd." She was unable to write a sentence to dictation or to copy a model sentence. When asked to salute or show how a hammer is used, she did not respond. She imitated the examiner saluting when asked to do so. A demonstration by the examiner of hammering (without a hammer in hand) was followed by repetitive saluting with the right hand. Attempts at figure copying produced illegible marks that did not resemble the model. She was unable to describe the inside of a watermelon, the color of a fire engine, or the color of the grass. When asked to group similar pictured faces of unfamiliar individuals, she correctly matched two pairs but attempted no more.

Cranial nerves II through XII, visual fields, and results of funduscopic examinations were normal. An iridectomy scar was noted on the right side and a cataract in the left lens. Pinprick and other noxious stimuli were localized. With eyes closed, the patient correctly named a ball, a fork, and a comb when placed in either hand. Both upper extremities could be raised spontaneously above the head. She reached for objects without any difficulty. There was no weakness of the upper or lower extremities. There was no limb ataxia. Reflexes were present but diminished in the upper and lower extremities. The Babinski sign was not present.

An ECG demonstrated left-sided ventricular hypertrophy. Cardiomegaly was noted on chest roentgenogram. Slowing in the left temporal and right frontal and temporal areas was noted on an EEG. Computed tomography (CT) on the third hospital day demonstrated a nonenhancing lucency in the left lateral-ventral thalamus (Figure). A CT scan repeated on the 12th hospital day was unchanged. On the

Speech Profile (Boston Diagnostic Aphasia Examination)*

Characteristic	Z Score
Melodic line	+1
Phrase length	+1
Articulatory agility	+1
Grammatical form	+1
Paraphasia in running speech	−1.5
Word finding	−1.5
Auditory comprehension (mean)	−1.7
Naming (mean)	−1.0
Repetition (mean)	0.0

*Negative scores indicate greater impairment.

17th hospital day, bilateral carotid and left vertebral angiography was performed. The findings included the following: a shallow plaque with ulceration at the junction of the left internal and common carotid arteries; a nonulcerating, shallow plaque in the distal right common carotid artery and proximal right internal carotid artery; and an area of focal narrowing in the right angular branch of the middle cerebral artery.

During the patient's hospitalization, the Boston Diagnostic Aphasia Examination (BDAE) was administered. Spontaneous speech was fluent, easily articulated with normal intonation, phrase length, and grammatical form. Moderate to severe auditory comprehension deficits were noted. Word-retrieval problems were manifested as a lack of content words. Verbal output was characterized by frequent verbal paraphasias, extended English jargon, and neologisms. Repetition of single words and high-probability sentences was generally intact, although occasional phonemic paraphasias were observed. Repetition of low-probability sentences was poor, with frequent neologistic distortion. Reading and writing skills were severely impaired. the BDAE Z-score profile was consistent with transcortical sensory aphasia (Table).

The patient was discharged to a nursing home on the 18th hospital day without improvement in her language abilities. Aspirin, dipyridamole, insulin, and diuretic therapy were prescribed.

Three months after discharge, a family member reported that the patient's condition had improved substantially. However, she was still "mixed-up" at times. Verbal paraphasias occurred on naming commonplace objects, eg, an oncoming bus would be identified as a train or car. Reading skills were steadily improving. She was again able to read the newspaper.

During the sixth month following hospitalization, the patient was reexamined. She spoke softly, fluently, and spontaneously. Semantic paraphasia was infrequently noted. Writing to dictation was marked by perseveration and poor grammatical construction. She counted backward from 20 to 18 in 15 s when asked to count from 20 to 0 rapidly. One of three pictured presidents was recalled after five minutes. The other two were identified correctly after visual clues were given. The following were preserved: comprehension of three-step commands; naming (ball, key, spoon, rubber band, wristwatch, buckle and sewing needle); repetition ("he and she and I" and "no *if*'s *and*'s or *but*'s about it"); reading (newspaper); right-left orientation; oral-buccal-lingual and limb praxis; finger identification; eye-hand coordination; simultaneous visual perception; and revisualization ability. There was a subjective increase to pinprick sensation on the left side of the face and trunk and the left arm, leg, and ear; a tremor of the outstretched left upper extremity; slight ptosis of the right eyelid; and a tendency to veer and stumble to the right on gait testing.

A CT scan of the thalamus at 1.5-mm intervals was obtained. Estimated anatomic reconstructions disclosed that the infarction involved the lateral nuclear group, ventral anterior nucleus (VA), ventral lateral nucleus (VL), anterior portion of the posterior limb of the internal capsule, and reticular nucleus bordering VA and VL. There was slight extension into the globus pallidus.

COMMENT

This case and others from the literature suggest that injury to the left thalamus is followed by a constellation of deficits that may include reduced or fading voice volume, neologisms, verbal and phonemic paraphasic errors, laconic speech, contaminations, and intrusions. Repetition is nearly always intact. Comprehension is variably affected. Perseveration is often prominent. Several cases, including our own, have shown unexplained fluctuations in or fatigability of speech performance, ie, deterioration of grammatically correct speech into unintelligible jargon. We have divided these deficits into four major clusters: (1) *extrapyramidal*, (2) *lexical access*, (3) *vigilance*, and (4) *comprehension*.

Decreased or fading voice volume (extrapyramidal cluster) is common with thalamic injury[1,6,19,20] and appears to reflect an elemental speech disturbance rather than an aphasic disorder.[18] The anatomic basis for this defect may be interruption of extrapyramidal inputs into the thalamus. Fibers from the medial globus pallidus and pars reticularis of the substantia nigra enter the ansa lenticularis to end in VA nucleus and the rostral portion of VL nucleus.[27-29] In our case the latter two nuclei were involved.

A variety of phenomena that occur after left thalamic injury include decreased spontaneous speech (laconic or sparse speech), anomia, verbal paraphasia, neologisms, and jargon, suggesting marked difficulties in *lexi-*

cal access. Anatomic areas that may be important in this process include the supplementary motor region, orbital cortex, and anterior insular cortex.[30-32] The VA nucleus projects to each of these regions. Furthermore, orbitofrontal cortical projections to insular temporal cortex and the amygdala[32] give VA nucleus influence over limbic activity that may affect lexical access. Insular connections with supplementary motor area and cortical association areas[32] (including supratemporal plane, frontal, parietal, and occipital regions) may also influence this process.

The large numbers of contaminations, perseverations, semantic paraphasias, intrusions, and neologisms emitted by patients with thalamic injury may reflect more than a defect in lexical access. The failure to suppress incorrect, even outrageous, words suggests a prominent defect in self-regulation of speech (*vigilance*). This defect could account in part for the neologisms, perseverations, and jargon, as well as for the frequent observations of fluctuations in speech performance.[7] Others have suspected that the thalamus may play a role in maintaining vigilance. Ojemann[24,33] has suggested a specific "alerting" function for the VL nucleus. Similarly, Luria[25] has suggested that altered vigilance may explain quasiaphasic speech disturbances after thalamic injury. Mohr et al[7] have speculated that the logorrheic paraphasia and marked speech fluctuations that occur after thalamic injury reflect deregulation of the cortical surface.

Vigilance deficits may reflect interruption of inputs from arousal systems. The VA nucleus receives inputs from the ascending mesencephalic reticular system via the intralaminar and midline thalamic nuclei, which also project diffusely to the cortex and globus pallidus.[34,35] The VA nucleus is also reciprocally connected with intralaminar nuclei[29] and the reticular nucleus.[36] These connections help account for the characteristics of the nonspecific thalamic system exhibited by this nucleus.[37] In physiologic studies, VA stimulation evokes widespread cortical response.[38-40] The VA is also considered essential for recruiting response.[40,41]

Comprehension is variably disturbed in thalamic injury. The anatomic basis for this defect may reflect dysfunction of those thalamic projections also necessary for lexical access. In addition, the VA nucleus has a modulating or driving influence on Broca's area. Disruption of this interconnection might produce a comprehension defect by an effect on Wernicke's area via the superior longitudinal fasciculus.[30]

The exact neuroanatomical basis for the language deficits following left thalamic infarction remains speculative. Physiologic and anatomic evidence suggest that VA nucleus plays a major role. We have proposed that the deficits following dominant lateral-ventral thalamic injury can be divided into four major clusters. Although it may be argued that these categories are subject to overlap, are too broad in scope, or constitute deficits that are nonspecific, the clusters do help to organize the analysis of deficits following thalamic injury and do provide a framework for clinical-pathologic correlation. These clusters also emphasize the preservation of repetition skills after thalamic injury.

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