

Significant Case Reports

"Semantic" Conduction Aphasia from a Posterior Insular Cortex Infarction

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ABSTRACT

A unique infarction limited to the posterior insula and intrasylvian parietal opercular cortex produced a subtype of conduction aphasia, characterized by a predominance of semantic paraphasias. Temporal lobe hypoperfusion seen on hexamethylpropyleneamineoxime single-photon emission computed tomography in the absence of any signs of ischemia suggested that cortical diaschisis played a role in the emergence of this syndrome.

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The role of the insula in language has been difficult to assess clinically because it is rarely injured in isolation. Because the insula is supplied by the proximal middle cerebral artery (MCA), MCA infarction usually damages also the frontal, parietal, or temporal cortex, or the subcortical white and gray matter [1]. Perisylvian infarction that includes the insula can produce motor [1], global [2], sensory [3], and conduction aphasia [4], but even Wernicke's original report

of conduction aphasia described large posterior lesions involving much of the parietotemporal cortex [5]. The patient described here had a unique subtype of conduction aphasia, produced by infarction restricted to the posterior insula and intrasylvian parietal opercular cortex.

Case Report

A 54-year-old, left-handed woman with a viral cardiomyopathy was found on the floor

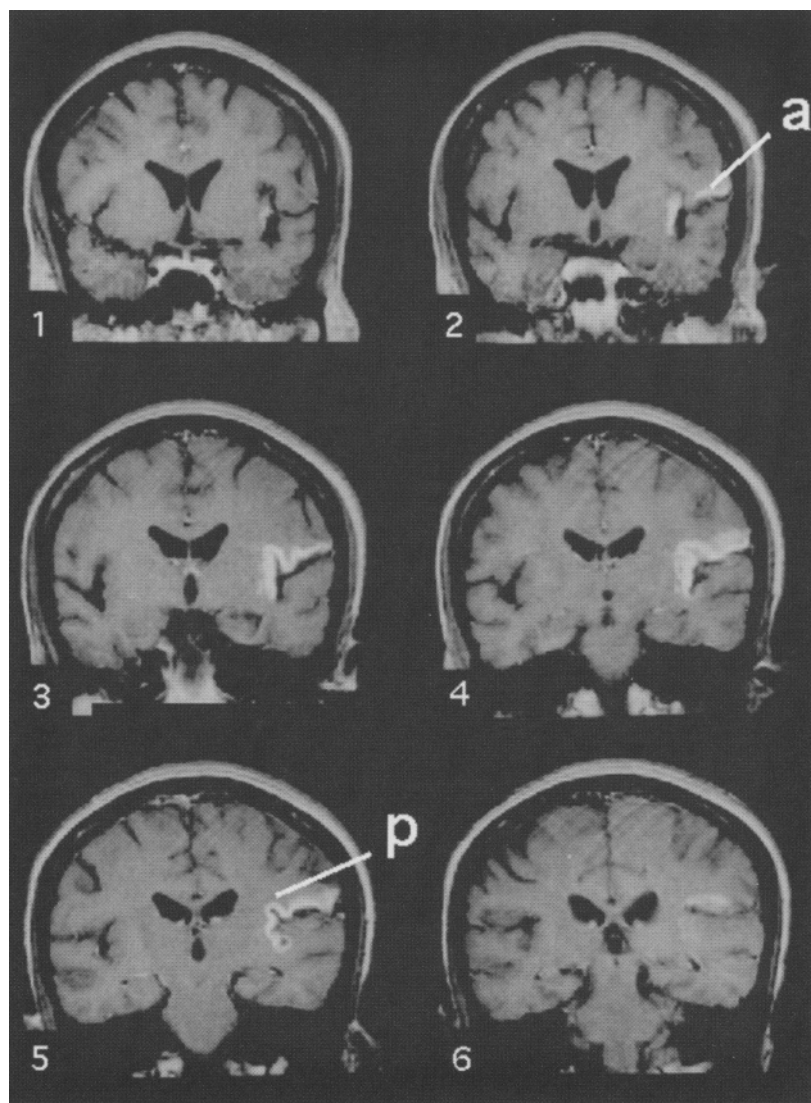


Fig 1. Coronal MRI (T1-weighted with gadolinium enhancement) showing infarction in the insular cortex and intrasylvian parietal opercular cortex. a = anterior extent of the infarct in the central insula; p = posterior extent of the infarct in the posterior insula.

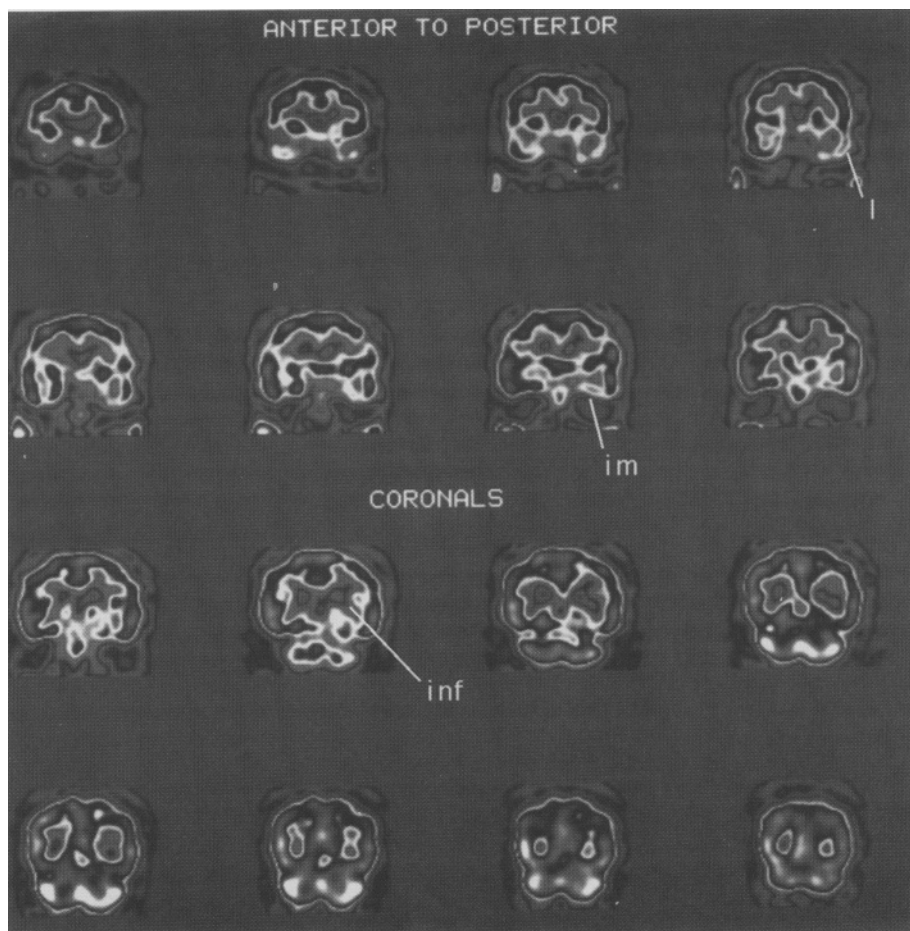


Fig 2. Coronal HMPAO-SPECT showing lateral and inferomedial left temporal lobe hypoperfusion. l = lateral temporal lobe; im = inferomedial temporal lobe; inf = site of infarct in the left posterior insula.

of her apartment with “garbled speech” and right-sided weakness. In the emergency room she was alert and attentive, able to follow commands, but answered questions in brief, stereotyped phrases. She was unable to repeat short sentences. She wrote her signature accurately, but when asked to write

“Today is a nice day,” she wrote, “also. . . so. . . locate. . . locate. . . at. . . theis. . . locate. . . that. . . is. . . the. . .” There was a mild right hemiparesis and right-sided hyposthesia. Computerized tomography scans appeared normal, but T1-weighted, gadolinium-enhanced magnetic resonance imaging

(MRI) performed at 1 week revealed cortical infarction limited to the posterior insula and the adjacent intrasylvian parietal operculum (Fig 1). A technetium 99m-hexamethylpropyleneamineoxime single-photon emission computed tomography (HMPAO-SPECT) scan showed regional hypoperfusion in the inferomedial and lateral regions of the left temporal lobe (Fig 2).

At 1 week, language testing revealed fluent speech except for word-finding difficulties. She generated only eight animal names in 1 minute (normal, 20–22). No phonemic or semantic paraphasias were noted during spontaneous speech. She named correctly 39 of 60 pictured items on the Boston Naming Test (normative mean, 55.8; standard deviation, 2.63) [6]. Nineteen of her errors were semantic substitutions. Auditory comprehension was intact to multiple-step commands. Reading comprehension was normal. Repetition was halting and effortful, with paraphasias on longer sentences. She was unable to imitate a tapped rhythm accurately. Verbal agility was impaired. She struggled with automatized sequences (e.g., alphabet, months of the year), stopping repeatedly to ask, “What was the last one I said?” Moderate dysgraphia was present. Her paraphasias were striking for a predominance of semantic substitutions, noted on naming, repetition, reading aloud, and writing (Table).

Follow-up examination 1 month later showed substantial improvement. Except for minor hesitations, there was resolution of the repetition deficit. She generated 20 animal names in 1 minute. She named correctly 48 of 60 pictured items, with seven semantic paraphasic errors.

Discussion

An aphasia characterized by poor repetition and frequent semantic paraphasias was produced by an infarct limited to the gray matter

Examples of Semantic Paraphasias

Task	Target	Patient's Response
Picture naming	Globe	Atlas
	Harmonica	Accordian. . . not the accordian, it's the small one
	Rhinoceros	Hippopotomous, armadillo
	Unicorn	Pegasus
	Sphinx	Pyramid, pharaoh
Repetition	Near the table in the dining room.	Near table in the living room
	The lawyer's closing argument convinced him.	The lawyer's closing argument persuaded him.
	I stopped at his front door and rang the bell.	I stopped at his front door and rang his bell.
	The phantom soared across the foggy heath.	The ph. . . The phan. . . The phantom went across the marsh.
	The quarterback threw the football down the field on Saturday.	The qu. . . The quarterback threw the f. . . The quarterback threw the baseball into the field.
Oral reading	Beautiful	Pretty
Writing to dictation	Today is a sunny day.	Today a cleary. . .
	The quarterback threw the football down the field on Saturday.	A little. . . A Quarterbock was e. . . A Quarterback was take. . .

of the posterior insula and intrasylvian parietal operculum. The syndrome fit a classic picture of conduction aphasia, except for the predominance of semantic errors.

The insula is complex and heterogeneous in cytoarchitectonic structure. Modern anatomical studies provided ample evidence for insular connections with multimodal association cortex, including language areas [7]. At its anteroventral border, an agranular cytoarchitecture is coextensive with the pyriform cortex and the claustrum. Posterodorsally, the cellular structure becomes more differentiated: Islets of granule cells appear in the "dysgranular" midsection, and then become well laminated in the parietoopercular cortex with which the insula merges posteriorly. The anatomical connectivity of the insula reflects its cytoarchitectonic organization. The anterior insula projects reciprocally to agranular olfactory, amygdaloid, and paralimbic areas of the cortex. In contrast, the posterior insula has extensive connections with isocortical, higher-order association regions, predominantly with somatosensory, auditory, and motor association areas. Anatomical connections with the insula that support language include not only projections into the fasciculus arcuatus, but also direct connections to the temporal lobe and to the frontal opercular paramotor region. A recent case report described an infarct in the anterior insula that produced poor speech initiation, poor repetition, and phonemic paraphasias [8]. The dysfunction in speech initiation was proposed to reflect the anterior insula's projections to the limbic and reticular systems. To the present authors' knowledge, this is the first report of the effects of a posterior insular infarct.

The classic defining characteristics of conduction aphasia are fluent speech, normal auditory and printed word comprehension, impaired repetition, and paraphasic errors that contaminate both spontaneous speech and repetition [9]. Paraphasias in naming, reading aloud, and writing can occur also. Phonemic paraphasias, which predominate with conduction aphasia, are defined as a substitution of one phoneme for another (e.g., "telebone" is uttered for the target word "telephone"). Semantic paraphasias, rarely reported with conduction aphasia, are defined as the substitution of a semantically related word for the target word (e.g., "radio" for "telephone"). Paraphasias are explained most often as a phonological dysfunction, either at an early decoding step or as a phonetic mistargeting during the execution of articulatory output [10]. This might explain phonemic errors, but not semantic errors. Goldstein, however, explained the generation of paraphasias in conduction aphasia (which he termed "central apha-

sia") as a disorder of "inner speech" [11]. He believed that when inner speech was disturbed, "the word was dissolved into different parts." Under such dysfunctional conditions, word parts would be individually altered, omitted, or produced out of sequence, and literal paraphasias would emerge. He argued further that as a consequence of the struggle to produce a word as a sum of its parts, the patient might abandon the effort in favor of an alternative word that lay within the same "sphere" or semantic class. Semantic paraphasias would result. Interestingly, among the early aphasiologists, Goldstein promoted the insula's role in conduction aphasia, in contrast to the connectionists' assertion that disruption of white matter tracts (e.g., the fasciculus arcuatus) explained the condition [5, 12].

It is unclear why semantic paraphasias do not occur more frequently with conduction aphasia. In the present patient, the temporal lobe hypoperfusion observed on SPECT, which persisted even as the patient began to recover clinically, may have contributed to the clinical findings. Because the patient's temporal lobe appeared normal on MRI, the hypoperfusion could not be explained by infarction. Hemodynamic stenosis in the MCA and posterior cerebral artery was possible, but unlikely as transcranial Doppler ultrasound showed normal flow velocity in these vessels. Because the hypoperfusion encompassed both posterior cerebral artery and MCA vascular territories, a functional metabolic effect may have occurred. Cortical "diaschisis," defined as the "deactivation of intact brain regions remote from but connected to the area of primary injury" [13], has been proposed to explain some aphasias [14, 15]. In the present patient, hypoperfusion in the left temporal lobe may have reflected a functional disconnection between the posterior insula and language areas in the temporal lobe, including inferomedial and basal temporal areas [3, 16]. The unusual lesion anatomy and the temporal lobe hypoperfusion may have combined to produce this "semantic conduction aphasia." The mechanisms underlying rapid recovery still need to be explained in the face of permanent infarction and persistent temporal lobe hypoperfusion. Future studies with functional activation imaging may disclose an anatomical redistribution of function in these cases.

It is possible that because the present patient was left-handed, there were atypical anatomical connections. Nonetheless, isolated injury to the posterior insula and intrasylvian parietal operculum may produce a unique subtype of conduction aphasia in which semantic dysfunction predominates, contrasting with the phonological dysfunction that

characterizes conduction aphasia seen with temporoparietal lesions or anterior insular injury.

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