Insular Stroke Causing Unilateral Auditory Processing Disorder: Case Report

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Abstract
This case presentation describes a patient with a strategically placed lesion involving the right insula and adjacent white matter. The most remarkable finding associated with this case is the presence of a unilateral auditory processing disorder when presenting speech materials to the left ear. Intervention for this patient is described in addition to a discussion of possible explanations for the unique pattern of auditory dysfunction.

Key Words: Auditory agnosia, insula, speech discrimination, stroke

The reception and comprehension of spoken language is a very complex function that requires analysis at numerous synaptic junctions along the ascending pathway. The importance and function of some central auditory nervous system structures has been elusive, especially for multimodal areas of the central nervous system (Hicks et al, 1988). Although attempts have been made to model structural and physiologic components in animals, the methods and results do not always permit direct translation for applications in humans, due, in part, to basic differences in anatomy and morphology (Augustine, 1985). Consequently, detailed, functional descriptions of some auditory structures in humans have been difficult to affirm.

Among the structures with functions that are least understood is the insula. Previous studies have shown that the insula is a multisensory area displaying increased levels of activity to auditory, somatosensory, visual, and gustatory stimulation (Hicks et al, 1988). Its specific role in audition is poorly understood. The insula receives afferent auditory fibers from the medial geniculate body in a separate pathway from the neural tract proceeding to the transverse gyri of Heschl (Sudakov et al, 1971; Streitfeld, 1980). The insula also receives projections that originate in the primary auditory cortex of the temporal lobe (Augustine, 1985). Neural tracts that originate in the insula project to the frontal lobe cortex, the cortex adjacent to the primary motor area, the parietal lobe cortex adjacent to the somatosensory area, and the parietal operculum (see Augustine, 1985). Consequently, the insula is a very complex structure with numerous interconnected pathways. It is, in part, due to the complexity of the insula that its precise function, especially for auditory stimuli, has been difficult to define. The following case presentation may provide some insight into this structure, especially as it relates to speech understanding.

CASE HISTORY

D A was a 60-year-old, right-handed, white male who arrived at the emergency room on October 9, 1987 with the primary complaint of a right frontal headache. Approximately 6 hours after the headache began, he developed slurred speech, increased difficulty performing fine motor tasks with both hands, and an unsteady gait. He arrived at the emergency room approximately 13 hours after the headache began and was alert and cooperative. No evidence of aphasia was noted during the initial examination or the following period of hospitalization. The admitting physical examination found left facial paresis, oral apraxia, decreased pinprick sensation in his left upper extremity, and difficulty with fine motor movements of the left hand. The examination also revealed that his left extremities were generally weaker than those on the right side, and there existed bilat-
eral coordination problems. The remaining aspects of the physical examination were unremarkable. His previous medical history included diabetes mellitus, which was managed with insulin, pancreatitis, nonoccluding plaque formations in his major arteries, a previous blood clot in his right leg, and a myocardial infarction that occurred 4 months earlier.

A diagnosis was made of cerebral vascular accident, for which he was admitted into the special care unit of the hospital. Shortly after the hospital admission, the symptoms of bilateral coordination and fine motor movements of the right hand resolved. Over the course of his hospital stay, there was little change in his symptoms of left facial paresis, decreased body sensations of the left upper quadrant, and fine motor movements of the left hand. He was referred for a neuropsychologic examination, which documented decreased attention to objects approaching him from the left side.

A computerized tomography (CT) scan was obtained on October 12, 1987; it showed an ischemic area in the inferior aspect of the temporoparietal junction and included the insula, extreme capsule, claustrum, and external capsule (Fig. 1A-1C). Except for a small portion of the posterior limb, the internal capsule, globus pallidus, and putamen did not appear to be directly involved with the primary locus of the lesion. The CT scan did suggest possible involvement of the anterior portion of the auditory radiations (thalamocortical fibers). The lesion appeared to be medial and posterior to the transverse (Heschl's) gyri, which, at the level of these scans, are slightly anterior to and along the lateral fissure (DeArmond et al, 1976). The lesion is also anterior and inferior to the region usually associated with Wernicke's area.

There was no evidence of hemorrhaging or brainstem infarct. He was discharged 12 days after admission, to be followed as an outpatient.

Prior to discharge, DA complained of decreased auditory function in the left ear. The admitting neurologic examination found no changes in gross sensitivity or sound awareness in either ear. A re-examination prior to discharge also found "normal" sensitivity and sound awareness in both ears. Immediately post discharge, he was referred for otologic and audiological evaluations.

**Audiologic Findings**

The first audiologic evaluation was performed less than 1 week post discharge in October of 1987 (Fig. 2). Pure-tone thresholds revealed normal sensitivity from 250 through 2000 Hz and moderate to severe high-frequency sensorineural hearing loss at 3000 Hz and above. These results were consistent with a pre-existing loss of sensitivity associated with military duty. Speech identification scores for the right ear were 96 percent at 80 dB for monosyllabic words and 80 percent at 80 dB for SSI-ICM (synthetic sentence identification with ipsilateral competing messages) sentences (0-MCR [message-to-competition ratio]). In contrast, speech results for the left ear were abnormal, with a maximum monosyllabic word score of 32 percent and a maximum SSI-ICM score of 10 percent.

The next audiologic evaluation was completed in December of 1987 (Fig. 3), 2 months post-CVA. No changes were noted in pure-tone thresholds for either ear. Similarly, speech results were unchanged for the right ear. Speech identification levels for the left ear showed marked improvement from the previous evalu-

*Figure 1A-1C* Computerized tomography scans at successive 5 mm cuts showing an area of ischemia involving the posterior-inferior temporoparietal region.
ation. The maximum monosyllabic word score was 64 percent, and the maximum SSI-ICM score was 30 percent.

The final audiologic evaluation was performed in February of 1988 (Fig. 4). The patient stated that his hearing was back to "normal" and demonstrated clinical behavior consistent with that impression. Again, the audiologic evaluation showed no change in pure-tone thresholds for either ear or in speech results for the right ear. On this date, his word and sentence identification scores for the left ear were within the range of normal performance, with a 92 percent monosyllabic word score and a 90 percent SSI-ICM score.

**Intervention Strategy**

Following the initial audiologic evaluation in October of 1987, the possibility of auditory amplification was considered. Although he had no change in sensitivity in the left ear, his speech understanding in that ear was sufficiently poor to produce functional difficulties in various environmental situations. A wireless behind-the-ear (BTE) CROS (contralateral routing of the signal) hearing aid, routing sound to the right ear, was fitted to him. The rationale for this type of fitting was twofold: first, to treat the left ear as if it were nonfunctional, and second, to produce a possible Stenger-type effect in the right ear from the mildly increased sound pressure level delivered to that ear. DA stated that he perceived substantial improvement with speech understanding with the hearing aid in contrast to listening without it. Due to the uncertainty of possible spontaneous recovery, the hearing aid was loaned to him.

When he was evaluated in December of 1987, DA reported that he perceived improve-
ment in speech understanding in the left ear. Consequently, he used the CROS hearing aid less than previously. At the February 1988 evaluation date, DA stated that he no longer appreciated benefit from the CROS hearing aid and had discontinued using the device.

**DISCUSSION**

DA's condition mimics, in many respects, the more classic descriptions of auditory agnosia, except that, in this case, the clinical findings reflect a unilateral processing disorder. By many indications, the lesion that DA experienced was on the nondominant side and confined to the region of the insula and adjacent white matter. Interestingly, many of the acoustically activated cells appear to be located in the posterior portion of the insula (Cleary and Irvine, 1986), the area commensurate with DA's primary site of involvement. However, it is noteworthy that DA displayed many symptoms, not only those in the auditory realm.

In general, lesions affecting the insula and adjacent white matter produce a variety of symptoms, including hemianesthesia (Hyman and Tranel, 1989), contralateral multimodal field neglect (Berthier et al, 1987), oral apraxia (Tognola and Vignolo, 1980), and degradation of fine motor control (Hyman and Tranel, 1989). In some more severe forms, insular lesions may produce behavior changes, due to the interruption of neural pathways to the frontal, temporal, and parietal lobes (Metter et al, 1988), mutism, and severe dysarthria without aphasia. Insular lesions may also produce opercular syndrome, characterized by loss of the voluntary function of the muscles of the face, the tongue, mastication, and swallowing, with preservation of reflexive functions (Bruyn and Gathier, 1969; Starkstein et al, 1988). Previous reports have indicated that insular lesions do not produce problems with comprehension and writing (Schiff et al, 1983; Hyman and Tranel, 1989), although in one case, a deficit in the contralateral ear was detected using dichotic speech (Hyman and Tranel, 1989). Jerger and Jerger (1981) presented a case of cerebrovascular disease of the right fronto-temporoparietal area. This patient maintained normal hearing sensitivity bilaterally but demonstrated abnormal performance-intensity functions for the left ear for both phonetically balanced (PB) words and SSI-ICM sentences.

Insight into the function of one particular part of the brain is difficult for two reasons. First, the brain is a very complex structure in which many parts interact for the analysis, association, and comprehension of verbal language. Second, the area of influence of a lesion may extend beyond the anatomic boundaries defined by CT scan. Extension of the sphere of influence may be due to the interruption of neural pathways in a manner that adversely influences input or metabolic activity to other, structurally normal parts of the brain (Metter et al, 1988). Moreover, local edema can produce a mass effect on nearby neural tracts (Chollet et al, 1991), thereby disrupting the metabolic activity of the involved fibers (Chambers et al, 1973; Waxman, 1988).

The patient described in this report had several symptoms that implicated adjacent white matter involvement. These symptoms included...
facial paresis, hemianesthesia, and fine motor deficits of the left hand. The oral apraxia implicated involvement of the insula, thus supporting the conclusion that the patient manifested symptoms of a mixed insular/white matter lesion. The site of involvement for this lesion offers several possible explanations for the unilateral auditory agnosia.

The first possibility is that afferent fibers terminating in the transverse gyri of the right hemisphere were disrupted, thus preventing information from proceeding to Heschl's area. This prospect could hypothetically account for the breakdown in understanding speech while preserving auditory sensitivity. Under this scenario, auditory thresholds would remain unchanged, due to the ability of the central nervous system to detect and process simple stimuli at multiple synaptic levels. Unchanged thresholds could also reflect the redundancy of the auditory system contained in the bilateral ascending pathways to each temporal lobe. Because more complex speech stimuli appear to require higher levels of analysis typically found at the temporal lobe level, disruption of the afferent neural pathway would "disconnect" the right temporal lobe, resulting in an inability to recognize words. Two problems exist with this scenario: First, it does not fully acknowledge the redundancy of the auditory system and, second, the patient was unable to recognize nondegraded speech samples in quiet. One would expect the bilateral afferent representation to provide sufficient input to the ipsilateral hemisphere for word identification in quiet. This is based on clinical observations that nonaphasic temporal lobe lesions become most apparent with degraded or competing speech stimuli, not monosyllables in quiet.

A second possibility is that the sphere of influence extended beyond the anatomic region noted on the CT scan, due to acute edema disrupting Heschl's gyrus and the callosal fiber tract. The functional effect would be to prevent all incoming information from arriving at the temporal lobe and inhibit any output from traveling along the callosal pathway to the left temporal lobe. The argument against this is the same as stated above. That is, one would expect better speech understanding in quiet than what DA demonstrated, due to the bilateral afferent representation of auditory input going to each temporal lobe.

Because the insula is densely populated with auditory fibers, especially in the posterior portion, one would expect that damage to this area would produce a specific type of deficit in the central auditory nervous system. Consequently, the third possibility proposes that the insular region may be a secondary association area for the auditory system. Such has been suggested for somatosensory function (Roland et al, 1980) based on bilateral activation of the insula when fingers were active. Using positron emission tomography, Kushner et al (1987) found that acoustic stimulation of either ear produced bilateral activation of the insula. Moreover, glucose uptake was greater for the left insula than the right insula, regardless of which ear received the stimulation. Since the insula receives neural input from a primary thalamocortical auditory pathway, it is plausible that the insula may be involved in preprocessing auditory stimuli prior to Heschl's gyri and Wernicke's area. If the orderly preprocessing were disrupted, then final processing and recognition would be sufficiently distorted to render speech stimuli unintelligible. The cerebral degradation of the input signal would be most noticeable in the contralateral ear, due to the influence of pathway dominance. At present, this hypothesis is most consistent with the clinical findings for DA.

It would not be unusual for vascular lesions in the posterior part of the insula to also affect fiber tracts of the internal capsule, due to their close proximity. Such lesions may produce hemianesthesia of the contralateral head, trunk, and limbs, and contralateral hemiplegia. These additional symptoms are the result of the disruption of thalamocortical fibers and corticospinal tracts (Carpenter, 1976). Indeed, the presenting physical symptoms were consistent with posterior internal capsule involvement.

The magnitude and rapidity of spontaneous recovery can be attributed to the CT scan findings indicating an ischemic area with an absence of hemorrhaging. The hallmark signs of a vascular problem begin with the rapid onset of symptoms. In the case of ischemia without hemorrhage, the site of the vascular lesion is focal and well circumscribed by virtue of the distribution of affected vessels. The clinical progression of symptoms, however, is common during the first few hours. This may be attributable to acute edema of the primary site of involvement producing compression, or mass effects, on adjacent neural tracts, which, in turn, compromise their functional abilities. During the histologic states of attempted repair and corresponding changes in neural morphologies at the initial site, the acute onset edema resolves over
time, thereby relieving the compression of adjacent structures. The clinical observation typically corresponds to a notable, albeit gradual, diminution of many functional deficits (Daube et al, 1978). The time period during which DA recovered his word and sound identification abilities was approximately 3 to 4 months post insult.

The clinical management of this type of patient must be balanced to meet short-term needs, without being overly aggressive or pessimistic regarding long-term prognosis. This is due to the probability of spontaneous recovery, which is variable and extremely difficult to predict (Stillbeck et al, 1983). In the present case, intervention was designed to consider the clinically impaired ear as nonfunctional, taking advantage of the reasonably normally functioning contralateral ear. A CROS hearing aid was loaned to the individual until the degree of spontaneous recovery could be determined. As recovery proceeded, the patient found less need of the CROS hearing aid, and after approximately 4 months post onset, the hearing aid was no longer needed.

**SUMMARY**

This case presentation describes a patient with a strategically placed lesion involving the right insula and adjacent white matter. The most remarkable finding associated with this case was the presence of a transient, unilateral auditory processing disorder when presenting speech materials to the left ear. Intervention consisted of a CROS hearing aid to route speech sounds to the noninvolved ear. This intervention proved to be temporary and was unnecessary after approximately 4 months, when the patient displayed evidence of excellent recovery of speech understanding.

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**REFERENCES**


