

Clinical and Anatomic Findings in a Case of Auditory Agnosia

David R. Oppenheimer, DM, Freda Newcombe, PhD

• A case is reported of severe agnosia for verbal and nonverbal sounds in a setting of well-preserved intelligence and transient dysphasic symptoms. The lesions responsible for this disorder were bilateral cerebral infarcts, one of which had virtually destroyed the areas of Wernicke and Broca. The relation between lesions and disabilities is discussed and comparisons made with other published cases.

(*Arch Neurol* 35:712-719, 1978)

Auditory agnosia, the inability to interpret the meaning of sounds unaccompanied by other major disabilities is uncommon. (Some authors¹ restrict the meaning of the term "auditory agnosia" to the interpretation of *nonverbal* sounds. In this article, we use the term to include *word deafness*.) Even less common are cases with a well-documented disorder of sound recognition and in which the brains have shown clearly defined lesions, undisturbed by recent vascular disasters. During the past century, a number of case reports have been published, with and without anatomic findings. These have been the subject

of comprehensive reviews by Vignolo,¹ Lhermitte and colleagues,² Gazzaniga et al,³ and others.

We describe a patient who, having previously recovered from an episode of right-sided hemiplegia with dysphasia, suddenly lost the ability to interpret sounds of all kinds, although he could still hear noises and could still talk, read, and write. He survived in this state for nearly six months and died of extracerebral causes.

REPORT OF A CASE

The patient was born in 1907, served as a regular soldier, and was later a welder in the motor industry. He was fully right-handed. Slight left-handed tendencies were noted only in one of his three children. He had enjoyed good health and was not hypertensive. On July 10, 1971, he collapsed suddenly, having felt unwell for the previous two weeks. On admission to the Radcliffe Infirmary, Oxford, England, he was fully conscious but almost speechless, with a right-sided hemiparesis and a right-sided homonymous hemianopia. Hearing and common sensation were intact. A lumbar puncture yielded normal fluid at normal pressure.

During the next two days, power in the right arm and leg was regained, though the right plantar response remained extensor; the visual fields were full. On July 15, language functions were assessed. Spontaneous speech was very slow and hesitant; he responded to spoken instructions, although there was some perseveration. He could tell the time, and reading aloud was good. Writing was poor: he could only print

and could hardly write his own name. He had difficulty in reciting the alphabet, repeating words, and naming objects. The salient feature of his condition was a severe expressive dysphasia, with mild receptive loss for spoken language.

He was discharged home after 13 days in the hospital, and on examination seven weeks after discharge, his speech had improved. His wife, however, reported that he sometimes did not understand what she had said to him. Within a few months, he could speak normally and read a newspaper.

The second episode was on April 28, 1972. During the afternoon, he had pain in the chest after exertion. During the night, he became confused and very agitated and he was admitted to hospital, where he was found to have a right-sided hemiparesis and hemianopia, with expressive dysphasia. These symptoms rapidly improved, and a few hours later, when seen by a neurologist, the patient was able to converse normally and give a clear account of the previous day's events. The only positive sign was a minimal right-sided hemiparesis, present since the first episode. Cardiac muscle enzymes were found to be increased; the diagnosis of a myocardial infarct, followed by transient cerebral ischemia was made.

Nine days after admission, on May 8, there was a third episode. He had awakened that morning feeling confused "as if there was something wrong in (his) head." He was oriented in place, but could not understand why the time on his watch seemed to have changed. He did not respond to speech, but obeyed gesture and read print well. His speech was garrulous but rational. Past and recent memory was

Accepted for publication Jan 20, 1978.

From the Departments of Neuropathology (Dr Oppenheimer), Radcliffe Infirmary, and Neuropsychology Unit (Dr Newcombe), Churchill Hospital, Oxford, England.

Reprint requests to Neuropsychology Unit, Churchill Hospital, Headington, Oxford OX3 7LJ, England (Dr Newcombe).

good. Two days later, it was noted that very few spoken words were recognized; he sometimes caught a word or guessed the purpose of a question from vision, gesture, or context, but complained that his visitors were not speaking clearly enough. There was a minimal nominal dysphasia, calculation was fair, and right-left orientation was not grossly impaired. He could read with comprehension and responded appropriately to written instructions and mime. He recognized the buzzing of a tuning fork but not the ticking of a watch or a hand clap. On May 16, he was seen by an ear, nose, and throat registrar who reported that clinical examination of the ears was normal. He considered that quantitative audiometric assessment was impracticable, but added that the patient had no difficulty in hearing the spoken word: "the problem . . . is more central." A brain scan on May 13, showed an abnormal uptake in the right posterior temporal region.

No improvement in word or sound recognition was noted during his stay in hospital and he was discharged home on May 18. A month later, he was still unable to understand the spoken word, but his wife thought that lip reading had improved. Bone conduction was better than air conduction on the right side, whereas air conduction was better on the left. Weber was referred to the right. From then until his death, on Oct 26, 1972, his condition remained much the same except for an improvement in speech, reading, and writing. According to his wife, his spontaneous speech seemed normal to outsiders, but she noticed slurring when he was excited. He read newspapers and discussed their content with his wife in the evening when she returned home from work. He spent most of the day on his own, reading, walking, and shopping.

On occasion, he could hear the telephone ringing; he usually told callers that he could not hear what was being said and that they should telephone his daughter. He thought of himself as deaf, but his wife noted that he disliked the noise of their television set and compelled her to turn the volume so low that she herself could not hear what was being said. He did not, however, react to noises outside the room; he did not respond to people knocking on the front door or ringing the door bell, or to the sound of his grandchild crying in the next room.

His remote memory remained remarkably good and he had no difficulty in recalling the events of daily life. Two or three days before his death, he negotiated the sale of a watch for an appropriate sum of money, and a few hours before his death, reminded his wife of the transaction and

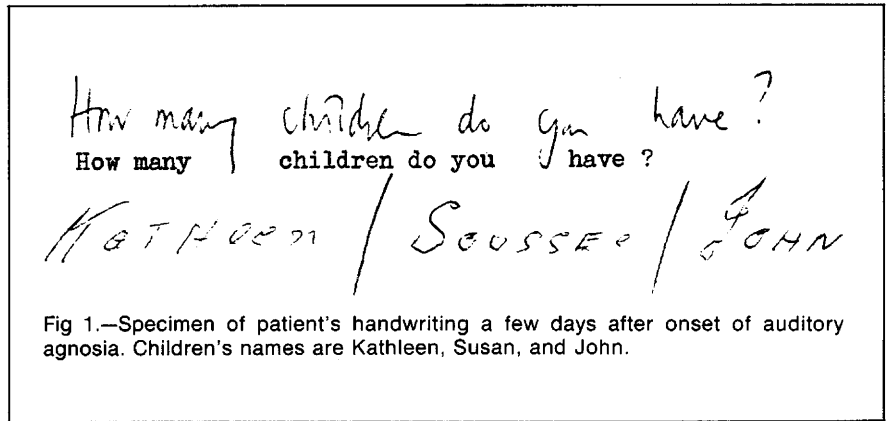


Fig 1.—Specimen of patient's handwriting a few days after onset of auditory agnosia. Children's names are Kathleen, Susan, and John.

the fact that the buyer was due to call to collect it. Topographical memory and orientation were unimpaired; he traveled alone by bus for his errands or to visit the hospital. He usually had a small shopping list and had no difficulties with change, either at local shops or at the neighboring supermarket. He was thought to have become childish and irritable, giving away small but valueless trinkets to visitors, and occasionally shouting at his wife. But she considered this behavior to be an exaggeration of previous personality traits; he had always been quick-tempered and somewhat extravagant. An ex-regular guardsmen, he was well-groomed until the day of his death.

PSYCHOLOGICAL AND AUDITORY TESTS

These tests were carried out between May 8 and May 18, 1972. The salient feature was the disorder of word and sound recognition to be described. This disorder occurred in a setting of mild dysphasia. Speech was fluent but included repetitions and paraphasias. Words were clearly articulated, but the amplitude of his voice varied unpredictably and tended to be loud. He had no difficulty in reading single words, but his attempts to read aloud paragraphs of a modern novel were distorted by repetition, omission, and a very occasional neologism. Comprehension of written instructions was fairly good, but test instructions sometimes had to be paraphrased before he fully understood them. The gist of his own written messages was usually clear, but they included a number of perseverative and spelling errors (Fig 1).

For example, the following is a typed copy of a letter written by the

patient to his wife within 24 hours of the onset of auditory agnosia and word deafness:

you would had
brought too to the Sister
my suit too the Sister
my Suit and Brought my
CLOWES too the HOSPITAL
NOT without asking the
Doctor without asking
the Doctor IF the Doctor
then I bring them If the
Doctor Ill I WILL WHAT
the doctor TELLS ME WHAT
The Doctor I WILL NOT The
Doctor tells me Ill I WILL
DOO WHOT the Doctor tells
me want me too Doo.
[8 : v : 1972]

In contrast, some nonverbal abilities were well preserved, eg, block design, cube counting, and nonverbal span (see Milner¹ for a description of the latter task).

The patient was rarely able to understand speech, although he consistently maintained that some voices were easier to understand (eg, one of the examiners who spoke slowly); he sometimes complained if speakers raised their voices. His ability to repeat single words was tested on May 16. Fifty-five short words were presented in three different conditions: (1) on tape through earphones (condition T), (2) spoken by the examiner whose lip movements were visible (condition Sp+V), and (3) spoken by the examiner whose lip movements were concealed (condition Sp). The patient usually attempted a response to the auditory signal, but there were a few, relatively long sequences (in conditions T and Sp) during which he

Condition	Initial Consonant, N = 55 (%)	Vowel, N = 55 (%)	Final Consonant, N = 53 (%)
T	5 (9.3)	18 (30.9)	9 (17.0)
Sp + V	24 (42.6)	32 (56.4)	9 (17.0)
Sp	7 (13.0)	15 (27.3)	2 (3.8)

Condition	Initial Consonant		Final Consonant: Labial, N = 37 (%)
	CA, N = 17 (%)	Labial, N = 16 (%)	
T	7 (41.2)	5 (31.3)	11 (29.7)
Sp + V	14 (82.4)	14 (87.5)	22 (59.5)
Sp	5 (29.1)	6 (37.5)	11 (29.7)

Stimulus	Response								
	p	t	k	b	d	g	m	Fricative	Omission
p	3
t	...	5	1
k	3	1	1
b	1	1	...	4	...	2
d	...	1	1	1
g	...	1
m	1

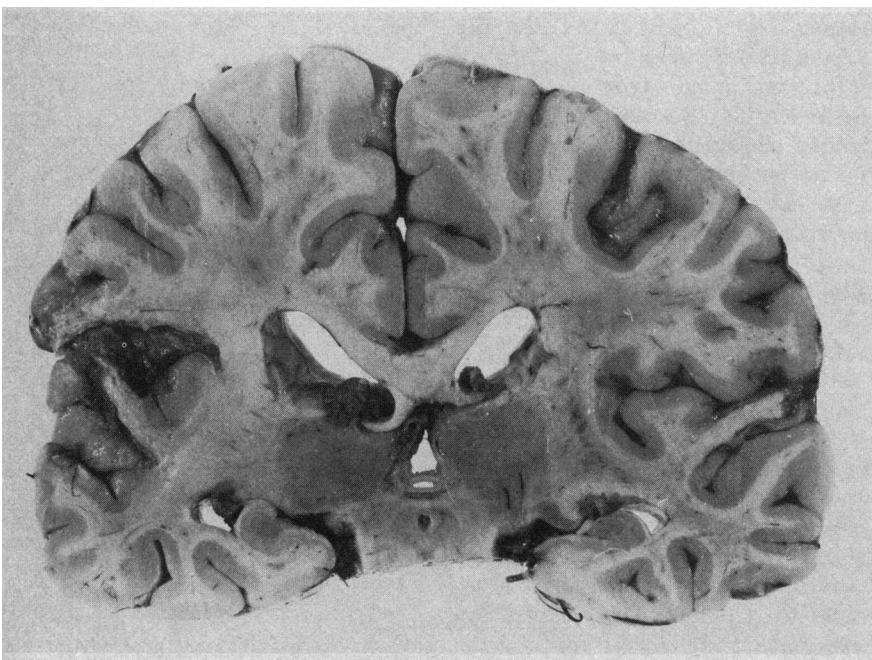


Fig 2.—Coronal slice at level of geniculate bodies, passing through transverse (Heschl's) gyri. On left, gyrus is completely destroyed; on right, only partially so.

produced only a puzzled, stereotyped "what?" or "please?"

There were 20, 47, and 25 scorable responses in conditions T, Sp + V, and Sp, respectively (Table 1). He was more successful at repeating vowels than initial or final consonants; his errors were often drawn from the same phonetic category. Table 2 gives his ability to reproduce features of the initial and final consonants, in the case of labials and coronal obstruents (Smith³ gives a description of these categories). His performance was notably better in the Sp + V condition; place of articulation of the initial consonant was incorrect in only two of the 47 responses. In this condition also, the pattern of response to the initial stop and nasal consonants (Table 3) showed consistent trends ($p \rightarrow b$; $b \rightarrow m$) rather than random error.

His ability to recognize familiar, nonverbal sounds was examined on May 18. A tape (O. Spreen and A. L. Benton, oral communication, 1963) comprising 26 environmental sounds (eg, coughing, miaowing, gunfire, and bird song) was relayed binaurally in three conditions. First, the subject was asked to describe what he had heard. In the two subsequent conditions, he was asked to match each sound with a picture selected from an array of three pictures for the 13 sounds in set A and an array of two pictures for the 13 sounds in set B (The pictures were drawn by one of the examiners, since the recognition test cards of Spreen and Benton were not available at the time. The same technique and drawings were used to examine a 10-year-old child with a severe disorder of speech sound production who made no errors in this version of the test). Before using the recognition procedures, the patient was asked to name and describe all the pictures used in the array; he had no difficulty in doing so.

From tape alone, the subject failed to identify any of the sounds but always interpreted them as speech, saying repetitively "please," "where," "yes," and occasionally a neologism "nar." When asked to match sound with picture, his performance was no better than chance: four and nine

correct choices in sets A and B, respectively. When the matching task was repeated with arrays of three pictures on June 12, there had been no improvement. He made four and six correct choices for sets A and B, respectively. He did, however, respond whenever an auditory stimulus was relayed on the tape. Since his capacity to discriminate speech and nonspeech sounds was in doubt, the melodies (without words) of 14 songs, very familiar to his generation, were played binaurally and he was asked what he had heard. On one occasion only did he describe an orchestra; he otherwise interpreted the sound as speech.

NECROPSY FINDINGS

The heart showed fibrosis and focal thinning of the left ventricular wall. There was adherent antemortem thrombus near the apex of the left ventricle. All the main coronary arteries were narrowed, and the circumflex branch of the left coronary was completely occluded by atheroma. The aortic arch, common and internal carotids, and vertebral arteries were healthy. The liver showed changes of chronic venous congestion, and there was a large old infarct in the right kidney. Other viscera were unremarkable.

The brain (1,480 g) showed externally two areas of old cortical infarction with shriveling. These involved the left inferior frontal gyrus and inferior parietal lobule, and the right superior temporal gyrus. The pattern of the basal vessels was normal, and the arteries were healthy except for a few plaques of atheroma. In coronal slices (Fig 2 and 3), the lesion on the left side extended from near the frontal pole, some 10 cm back to behind the splenium of the corpus callosum, destroying part of the inferior frontal gyrus, the whole of the frontal and parietal operculum, insula, and the superior surface of the posterior half of the temporal lobe. On the right, the lesion lay more posteriorly, starting at the level of the mamillary bodies, where it involved the superior temporal gyrus and temporal operculum, and extending 8 cm backward to end in the inferior parietal lobule. The insula was not involved.

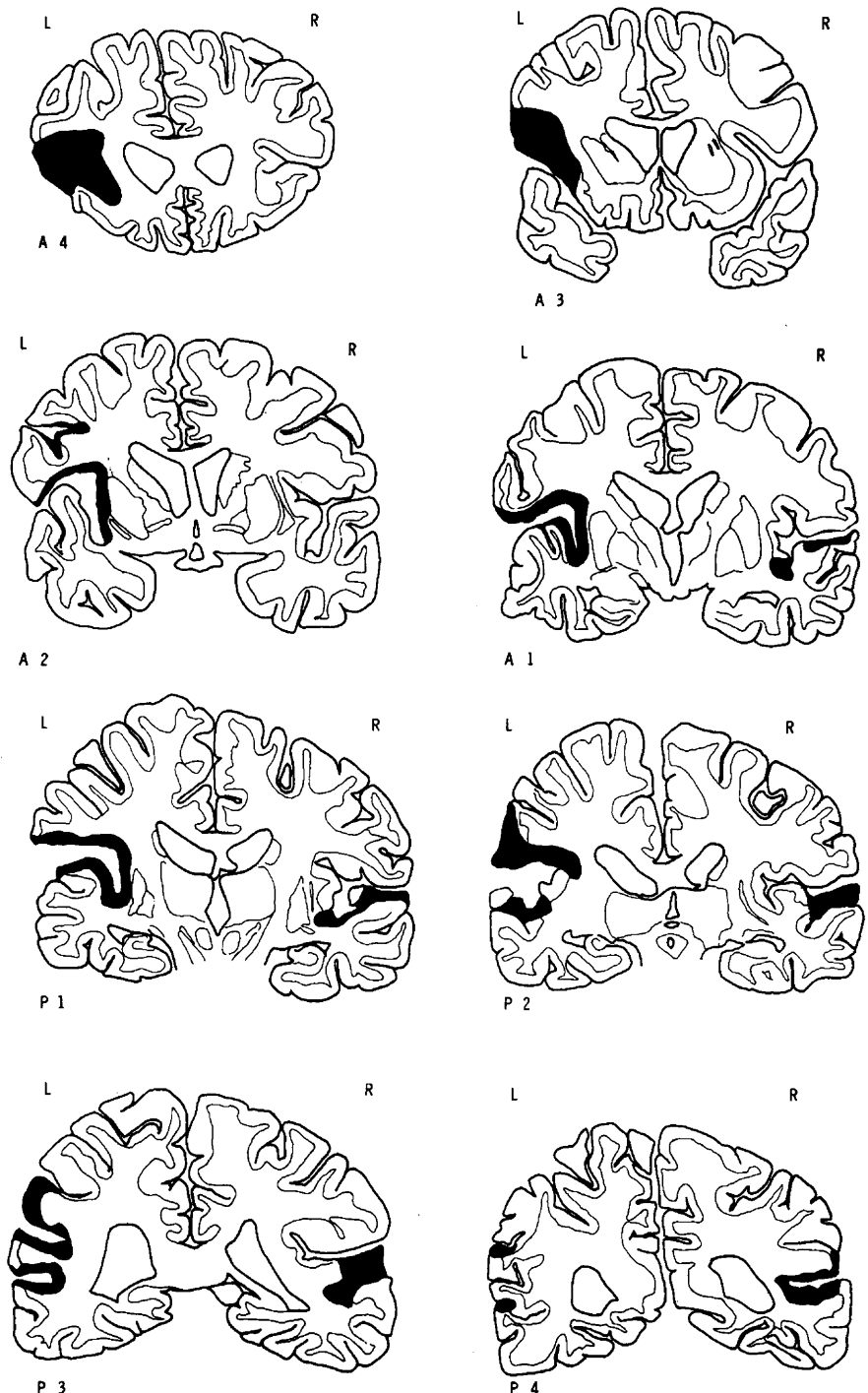


Fig 3.—Tracings from coronal slices. Areas of destruction shown in black. Convention: A1 indicate level of mamillary bodies; A2, A3, and A4 lie 1, 2, and 3 cm, respectively in front of A1; and P1, P2, P3, and P4 lie 1, 2, 3 and 4 cm, respectively behind A1. Lateral ends of Heschl's gyri lie at level A1, their medial ends at level P3.

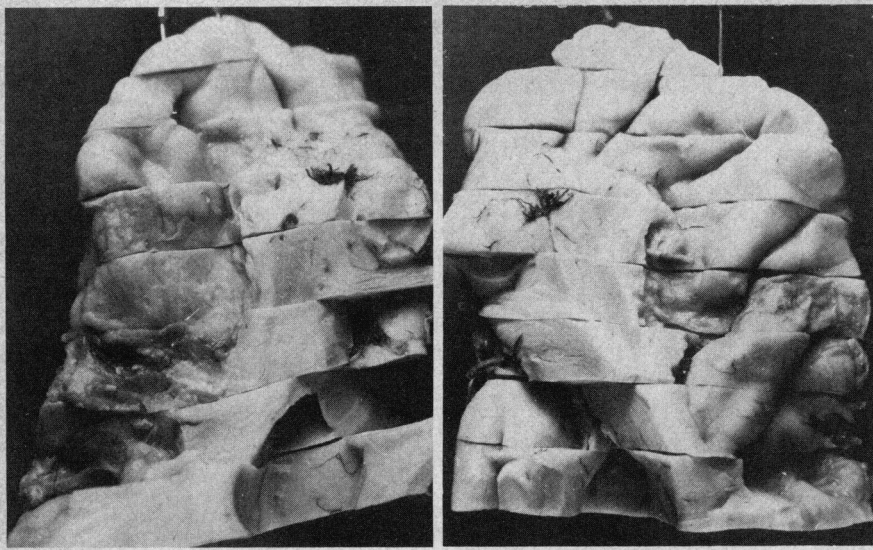


Fig 4.—Upper surfaces of temporal lobes; left and right. Temporal lobes have been reassembled from centimeter-thick coronal slices and skewered together.

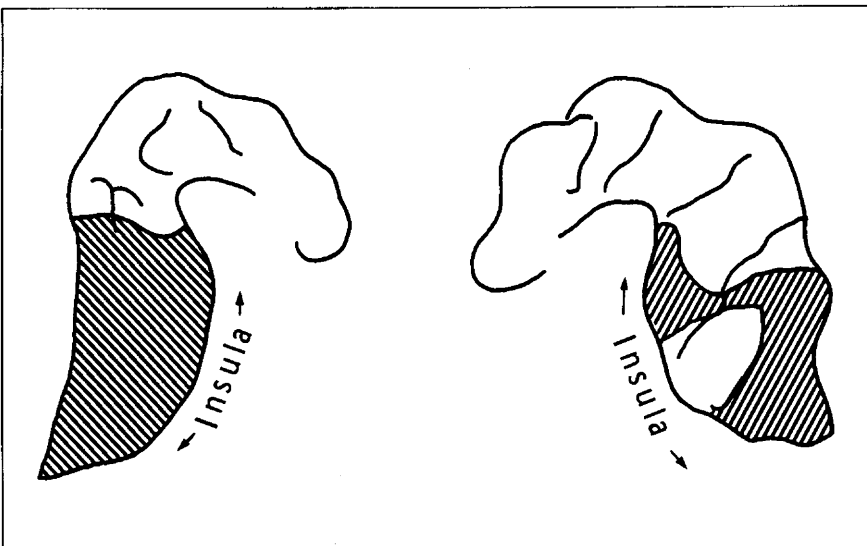


Fig 5.—Tracings from photographs, showing areas of destruction of superior temporal cortex.

The left and right temporal lobes were cut out and reassembled as shown in Fig 4 and 5. On the left, the posterior two thirds of the upper surface of the temporal lobe was destroyed; on the right, there was a band of infarction, crossing and partially destroying the transverse (Heschl's) gyrus and also destroying the posterior part of the planum temporale. The rest of the cerebral hemispheres seemed normal apart

from a mild (probably compensatory) dilation of the ventricles. There were no lesions in the central gray matter or in the brain stem and cerebellum.

Histology

Sections of the softened areas (left frontal and temporal; right temporal and parietal) showed the usual late features of anemic cortical infarction. The affected cortex consisted of an

outer shell of solid glial tissue and an inner zone composed of small blood vessels and phagocytes. Deep to this was degenerate white matter, with variable loss of myelin and axis cylinders, and conspicuous swollen-bodied astrocytes. There was no obvious difference between the appearance on the left side, where the presumed age of the lesion was 15 months, and that on the right, with a presumed age of five months. On both sides, there were local variations, as easily attributable to variations in the intensity of the insult as to differences of timing.

The thalamus and tegmentum of both sides were examined in serial sections. Here, there was a noticeable difference between the two sides. On the right, there was a small patch of nerve cell loss in the medial geniculate body, with slight glial proliferation among the fibers approaching it from above and laterally. On the left, there was severe (though not complete) cell loss in the medial geniculate body, with heavy gliosis in a band lying between the lateral geniculate body and the reticular nucleus of the thalamus (Fig 6); also gliosis, less conspicuous, in Forel's field and in the fronto-pontine part of the cerebral peduncle. The splenium of the corpus callosum showed some diffuse loss of myelin, with gliosis. No acute lesions, of any kind, were observed.

COMMENT

The Nature of the Psychological Disturbance

Patients with auditory agnosia, including the present case, can distinguish noise from silence; they have described it as "rattles and squeaks,"⁶ "a continuous humming,"⁷ or an uncomprehending "ça crie."⁸ A well-studied patient of Lhermitte and his co-workers² reported: "Lorsqu'on me parle, c'est comme si c'était loin . . . et trop près en même temps; comme si il y avait un léger voile tout près de mes oreilles, j'entends aoua . . . aoua . . . aou . . . ça roule; la musique, ça fait tout un brouhaha, un boucan." In contrast, our patient was not able to distinguish between speech and music.^{1,8,9}

The question arises as to how patients with this disorder occasional-

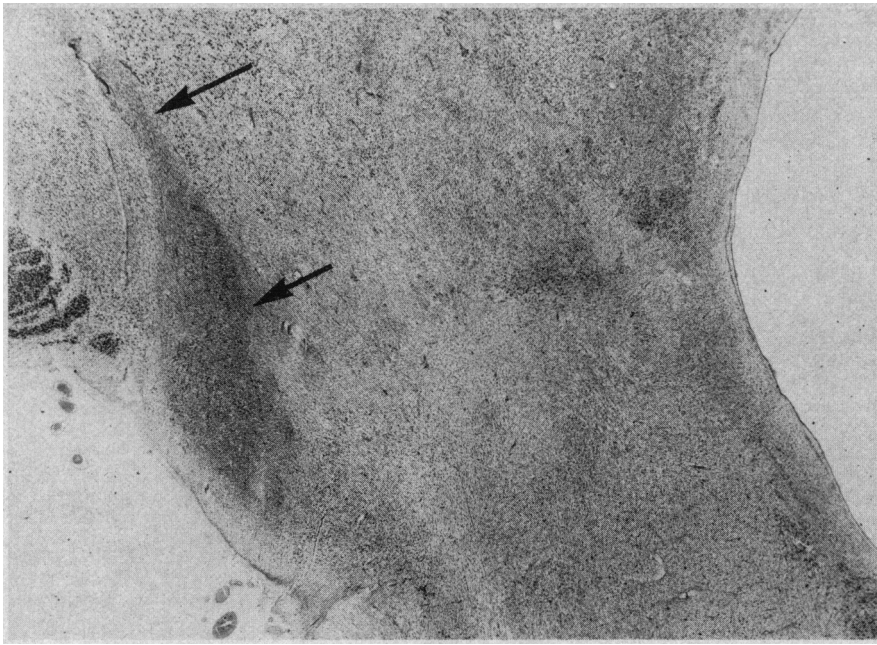


Fig 6.—Left geniculate bodies. Severe loss of nerve cells in medial geniculate body, and glial proliferation here and in auditory radiation (arrows [Nissel stain, $\times 10$]).

ly recognize a word or an environmental sound. They have been described as hearing doors opening and shutting,⁸ a softly rung bell,¹⁰ or the telephone. Wohlfart et al⁸ noted of their patient that "he could tell the difference between the jingle of a bunch of keys and knocks against the wall, provided he had been told the noise must be one of those two"; the patient described by Jones and Dinoli¹¹ heard the word "glasses" when his attention was focussed on reading. It is possible that context and the narrow range of probabilities against recognition in the way that patients with visual agnosia may recognize beds, tables, chairs, and eating utensils in an appropriate setting.

The recognition disorder in our patient cannot be attributed to gross sensory loss. In addition to the clinical examination, the patient's attempts at repetition showed that some phonetic features were detected and correctly reproduced, especially the vowel sounds; the trend to regularity in the pattern of error (Table 3) is not characteristic of deaf subjects,¹² but resembles in some respects the performance of dysphasic patients.^{13,14} Our patient's errors, including some interesting cluster responses ($p \rightarrow br;d \rightarrow tr;/ \text{ } \text{ } \rightarrow tr$) suggested that

he may have had a specific deficit in the discrimination of voicing and nasality (Smith, personal communication, 1977). This capacity to detect some features of an auditory signal is implied by the errors of other patients, eg, elevator \rightarrow vibration of a tuning fork⁸ and tinkling glass \rightarrow jingling keys.¹³ These recognition disorders may occur without peripheral hearing loss² or with only a slight diminution of acuity.^{8,15,16} Other patients, like ours, have been difficult to test⁸ or they may show such puzzling variation in standard audiometry and Bekesy tests¹⁵ as to make a single chart of dubious importance. The role of auditory inattention, noted by Lichtheim¹⁰ and several other workers, notably Hemphill and Stengel,¹⁷ has yet to be explored; the need for more refined tests of central auditory processing is too patent to require discussion.

The severe auditory recognition disorder of our patient was initially accompanied by milder disturbances of speech, reading, and writing; most cases of auditory agnosia, where language has been more than cursorily examined, show at least mild difficulties in reading,^{18,19} writing,³ and spelling.⁷ Our patient's spontaneous speech was fluent after the vascular

accidents, but there were occasional paraphasias and stereotyped, repetitive phrases. This perseveration, more noticeable in his writing during the acute phase (see aforementioned writing sample), has often been described in patients with auditory agnosia.^{2,3,8} Klein and Harper⁷ commented on their patient with word deafness that the most striking feature of his speech was the constant repetition of words and whole phrases, almost as if he was not sure what he said. Other patients find their own voices unclear or slightly noisy; their speech, however, may be unaffected by delayed auditory feedback.³

Our patient's dysphasic symptoms resolved during the few months preceding death, during which (as Lichtheim¹⁰ noted of such patients) he resembled a deaf person, whereas patients with aphasic comprehension difficulties for both spoken and written language may be misdiagnosed as confused or demented. His speech was by then normal, but his voice was louder than before; we note that changes in voice quality have sometimes been associated with auditory agnosia: raised tone²⁰; a "rather loud and monotonous tone"³; rising inflections^{19,21}; and high pitch, even where there was little if any evidence of sensory loss.²² In turn, our patient complained that some of his visitors spoke too loudly; there are similar reports in the literature: "it was easier to follow quiet talk"⁷; children made too much noise singing in the same room¹⁰; and the breaking of an egg shell was heard as a loud crash.⁸ These phenomena suggest changes in the normally sharply tuned properties of the cochlear nerve.²³

Anatomic Correlations

Cases such as the one we are reporting have attracted interest because of their relevance to problems of localization of function within the cerebrum. These problems have been debated, with particular reference to the condition of word deafness, since the last century; Freud's²⁴ monograph on aphasia being an outstanding example of critical reasoning on this subject. Recent reviews include those of Vignolo,¹ Lhermitte et al,² Gazzani-

ga et al,³ and Goldstein.²⁵

There seems to be general agreement that the auditory fibers from the medial geniculate bodies end in Brodmann's areas 41 and 42, that is, in the transverse (Heschl's) gyri, lying across the upper, buried, surfaces of the temporal lobes.^{26,27} In theory, bilateral destruction or undercutting of these areas should result in total deafness. In a massive review of the existing literature Henschen²⁶ claimed to have found nine cases of so-called cortical deafness due to bilateral lesions of Heschl's gyrus. Comparing these nine cases with 14 of pure word deafness (ie, loss of comprehension of the spoken word without loss of hearing and without gross disturbance of other language functions) Henschen concluded that the second group was able to hear because at least a part of one or other Heschl's gyrus had been preserved. On this view, our own patient owed his residual hearing to the small intact remnant of his right Heschl's gyrus. It is now suspected¹ that many published cases of cortical deafness were inadequately tested and were suffering from auditory inattention rather than deafness. Perhaps the most convincing case report is that by Clark et al,²⁸ in which the insulae and temporal opercula were "disconnected" by bilateral hemorrhages in the external capsules (Lhermitte et al² question the propriety of the term "cortical deafness" in this case. They suggest that the patient's deafness was caused by bilateral retrograde degeneration of the medial geniculate bodies).

In auditory agnosia, two main ingredients are commonly recognized; word deafness and the inability to interpret nonverbal sounds. The two usually go together, as in our case, and in nearly all cases with necropsy there have been bilateral temporal lobe lesions, almost always involving the planum temporale, which lies immediately behind Heschl's gyrus. Apart from the cases listed by Henschen²⁶ and his own case, reports on autopsy cases of word deafness include those of Liepmann and Storch,²⁹ Barrett,³⁰ Schuster and Taterka,¹⁵ Misch,³¹ Pötzl,^{32,33} Wohlfart et al,⁶ Mahoudeau et al,⁹ Lhermitte et al,² and Kanshe-

Date	Anatomic Changes	Clinical Effects
June-July 1971	Coronary atheroma leading to myocardial ischemia; infarct with thrombus in left ventricle	Patient felt unwell
July 10, 1971	Embolism of thrombus to a branch of the left middle cerebral artery	"Stroke," with right-sided hemiparesis, dysphasia, and homonymous hemianopia
July 1971	Subsidence of edema surrounding infarct	Rapid recovery from hemiparesis and hemianopia; slower and incomplete recovery from dysphasia
April 28, 1972	Further episode of myocardial infarction, causing transient arterial hypotension	Recurrence of right-sided hemiparesis, hemianopia and dysphasia, with recovery to previous state within a few hours
May 8, 1972	Embolism of thrombus from heart to branches of the right and (?) left middle cerebral arteries	Patient felt confused; unresponsive to speech, but rational and able to talk and read; not deaf, but failed to understand nonverbal sounds
May-October 1972	Retrograde degeneration in medial geniculate bodies	Auditory agnosia and word-deafness persisted unchanged
October 26, 1972	Further myocardial infarction	Patient died

polsky et al.³⁴ In all these cases there was a lesion of the left temporal lobe, and in all but two, a lesion on the right as well. The exceptions are the cases of Liepmann and Storch²⁹ and of Schuster and Taterka.¹⁵ In the latter, there was total inability to comprehend the spoken word, with exceptionally good preservation of all the other language functions, along with a severe but incomplete loss of comprehension of nonverbal sounds. The lesions lay on the left, involving claustrum, external capsule, and white matter deep to the temporal operculum. The cortex itself was intact.

In nearly all cases of word deafness, there is also a severe or mild nonverbal auditory agnosia as well; as far as we know, there is only one report of nonverbal auditory agnosia with good understanding of the spoken word. In this case,¹⁸ there was a single, unilateral lesion affecting the right insula, inferior parietal lobule, and the medial part of the temporal operculum.

Turning to our own case, the correlation of clinical disturbances with observed lesions is fairly clear in some respects, obscure in others (Table 4). The episode of July 10, 1971, is attributable to an embolus coming from the left side of the heart and infarcting an area supplied by the left middle cerebral artery. The episode of April 28, 1972, with symptoms lasting only a few hours, was probably due to tran-

sient ischemia of an area in the left frontal and temporal lobes that had escaped infarction but suffered from reactive edema after the first episode. This could have been caused by a short spell of arterial hypotension consequent on a myocardial infarct.

What happened in the final episode of May 8 is harder to envisage. If it was simply an embolism to the right middle cerebral artery, it is difficult to see why this produced word deafness, of which there was no trace on the previous day. It may well be that emboli lodged in both left and right middle cerebral branches at about the same time, the one on the left enlarging the already-existing infarct.

Still harder to explain is the fact that the patient, with most of Wernicke's area and the whole of Broca's area having been infarcted, could still talk, read, and write, although the left hemisphere was dominant for speech, as it seems from the details of the first episode of dysphasia with right-sided hemiplegia. A similar problem faced Boller,³⁵ who described a right-handed patient with no aphasic disturbances at the time of death, in whom necropsy showed an old softening of the posterior third of the left superior temporal gyrus, including the planum temporale. Boller argues that the patient's "speech area" must have been on the right side, and regards the case as

providing negative evidence of crossed aphasia in a dextral patient. This view would not easily account for the preservation of speech and writing in our own patient, and we think that in both cases alternative explanations can be found, for instance, individual variations both in the organization of language within the left hemisphere and in the capacity for reorganization and recovery of function after damage to the language areas.

There are good reasons for supposing that such variability exists. The fact that after left-sided hemispherectomy in early life a child learns (or relearns) to talk shows that areas other than Wernicke's area are potential "speech centers."^{36,37} It seems that whereas the motor cortex and the primary visual cortex can be accurately defined and are constant in their location, areas concerned with speech are less precisely localized.³⁸ This, if true, need not cause surprise.

Vision and the cerebral control of movement, are as old as or older than the mammalian class itself, and the anatomic bases for these have become stereotyped. Speech is a recent acquisition, and it is reasonable to suppose that its neural basis may vary from subject to subject. Very little is known at present about such individual variations.³⁹ Although it may be legitimate to say that lesions of Broca's area tend to interfere with expressive speech, lesions of the left posterior and superior temporal area interfere with understanding of the spoken word, and lesions of the left supramarginal and angular gyri interfere with disturbances of reading and writing, it is unwise to suggest a constant correlation of bits of cortex with isolated language functions. (Recent evidence on the broad localization of language functions is found in Kertesz et al.⁴⁰ There is still a need for detailed mapping of lesions in pa-

tients with language disorders; but if we are to learn more about variations between individuals, these need to be supplemented by studies of brains with lesions in the "speech areas" from patients whose language functions were intact or nearly so.

The clinicians at the Radcliffe Infirmary and Churchill Hospital made their case notes available. Dr Mark Haggard, Dr Neil Smith, and Dr Philip Smith commented on the patient's performance on auditory tests.

References

1. Vignolo LA: Auditory agnosia: A review and report of recent evidence, in Benton AL (ed): *Contributions to Clinical Neuropsychology*. Chicago, Aldine Publishing Co, 1969, pp 172-231.
2. Lhermitte F, Chain F, Escourolle R, et al: Étude des troubles perceptifs auditifs dans les lésions temporeles bilatérales. *Rev Neurol* 124:329-351, 1971.
3. Gazzaniga MS, Glass AV, Sarno MT, et al: Pure word deafness and hemispheric dynamics: A case history. *Cortex* 9:136-143, 1973.
4. Milner B: Interhemispheric differences in the localization of a psychological process in man, in Summerfield A (ed): *Cognitive Psychology*. *Br Med Bull* 27:272-277, 1971.
5. Smith NV: *The Acquisition of Phonology*. London, Cambridge University Press, 1973.
6. Reinhold M: A case of auditory agnosia. *Brain* 73:203-223, 1950.
7. Klein R, Harper J: The problem of agnosia in the light of a case of pure word deafness. *J Ment Sci* 102:112-120, 1956.
8. Wohlfart G, Lindgren Å, Jernelius B: Clinical picture and morbid anatomy in a case of "pure word deafness." *J Nerv Ment Dis* 116:818-827, 1952.
9. Mahoudeau D, Lemoine J, Foncin JF, et al: Considérations sur l'agnosie auditive. *Rev Neurol* 99:454-471, 1958.
10. Lichtheim L: On aphasia. *Brain* 7:433-484, 1885.
11. Jones J, Dinolt R: Pure word deafness. *Laryngoscope* 62:194-203, 1952.
12. Erber NP: Auditory, visual, and auditory-visual recognition of consonants by children with normal and impaired hearing. *J Speech Hear Res* 15:413-422, 1972.
13. Fry DB: Phonemic substitutions in an aphasic patient. *Lang Speech* 2:52-61, 1959.
14. Saffran EM, Marin OSM, Yeni-Komshian GH: An analysis of speech perception in word deafness. *Brain Lang* 3:255-265, 1976.
15. Schuster P, Taterka H: Beitrag zur Anatomie und Klinik der reinen Worttaubheit. *Z Gesamte Neurol Psychiatr* 105:494-538, 1926.
16. Liepmann H: Über die agnostischen Störungen. *Neurol Centralblatt* 27:664-675, 1908.
17. Hemphill RE, Stengel E: A study on pure word-deafness. *J Neurol Psychiatr* 3:251-262, 1940.

18. Spreen O, Benton AL, Fincham RW: Auditory agnosia without aphasia. *Arch Neurol* 13:84-92, 1965.

19. Horenstein S, Bealmeur K: Concerning the nature of word deafness. *Trans Am Neurol Assoc* 98:265-267, 1973.

20. Laignel-Lavastine M, Alajouanine T: Un cas d'agnosie auditive. *Rev Neurol* 37:194-198, 1921.

21. Albert ML, Sparks R, von Stockert T, et al: A case study of auditory agnosia: Linguistic and non-linguistic processing. *Cortex* 8:427-443, 1972.

22. Tanaka Y, Taguchi K, Sakabe N, et al: Pure auditory agnosia associated with alteration of voice and personal character. *Folia Phoniat* 17:185-194, 1964.

23. Evans EF: Normal and abnormal functioning of the cochlear nerve. *Symp Zool Soc London* 37:133-165, 1975.

24. Freud S: *Zur Auffassung der Aphasien*. Vienna, Deuticke, 1891. (English translation, *On Aphasia*. London, Imago, 1953).

25. Goldstein MN: Auditory agnosia for speech ("pure word-deafness"): A historical review with current implications. *Brain Lang* 1:195-204, 1974.

26. Henschen SE: Über die Hörsphäre. *J Psychol Neurol* 22:319-474, 1918.

27. Brodal A: *Neurological Anatomy in Relation to Clinical Medicine*, ed 2. Oxford, Oxford University Press, 1969.

28. Clark WE, Russell WR: Cortical deafness without aphasia. *Brain* 61:375-383, 1938.

29. Liepmann H, Storch E: Der mikroskopische Gehirnbefund bei dem Fall Gorstelle. *Monatsschr Psychiatr Neurol* 11:115-120, 1902.

30. Barrett AM: A case of pure word-deafness with autopsy. *J Nerv Ment Dis* 37:73-92, 1910.

31. Misch W: Über corticale Taubheit. *Z Gesamte Neurol* 115:567-573, 1928.

32. Pözl O: Zur Pathologie der Amusie. *Z Gesamte Neurol Psychiatr* 165:187-194, 1939.

33. Pözl O: Bemerkungen zum Problem der korticalen Vorgänge bei der akustischen Wahrnehmung. *Monatsschr Ohrenheilkd* 77:422-430, 1943.

34. Kanshepolsky J, Kelley JJ, Waggener JD: A cortical auditory disorder. *Neurology* 23:699-705, 1973.

35. Boller F: Destruction of Wernicke's area without language disturbance: A fresh look at crossed aphasia. *Neuropsychologia* 11:243-246, 1973.

36. Wilson PJE: Cerebral hemispherectomy for infantile hemiplegia. *Brain* 93:147-180, 1970.

37. Smith A, Sugar P: Development of above normal language and intelligence 21 years after left hemispherectomy. *Neurology* 25:813-818, 1975.

38. Bogen JE, Bogen GM: Wernicke's region: Where is it? *Ann NY Acad Sci* 280:834-843, 1976.

39. Marshall JC: Some problems and paradoxes associated with recent accounts of hemispheric specialization. *Neuropsychologia* 11:463-470, 1973.

40. Kertesz A, Lesk D, McCabe P: Isotope localization of infarcts in aphasia. *Arch Neurol* 34:590-601, 1977.