

PURE WORD DEAFNESS  
ANALYSIS OF A CASE WITH BILATERAL LESIONS  
AND A DEFECT AT THE PREPHONEMIC LEVEL

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INTRODUCTION

PURE word deafness is a rare syndrome first described by Kussmaul (1877) when he used the term '*reinen Worttaubheit*' to describe a patient unable to comprehend spoken words with otherwise intact speech production and reading abilities. Clinically, the patient with pure word deafness is supposed to be free of any aphasic difficulties, although many of the reported cases are marked by elements of Wernicke's aphasia (Brown, 1974; Rubens, 1979; Benson, 1979). The syndrome is distinct from the symptom of word deafness which may appear as a component of Wernicke's aphasia. Some authors have preferred the term 'verbal auditory agnosia' or 'auditory agnosia for linguistic material' (Ulrich, 1978), but the term 'pure word deafness' has retained wide clinical acceptance (Brown, 1974; Hécaen and Albert, 1978; Rubens, 1979; Benson, 1979).

There was uncertainty that pure word deafness was a distinct disorder of cerebral function. Wernicke and Freidlander (1893) believed Kussmaul's case represented an incomplete description of his sensory aphasia. Freud (1891) suspected that a peripheral hearing loss was necessary for the development of an isolated word deafness. On the basis of two cases with labyrinthine dysfunction, Freund (1895) concluded that the syndrome was primarily due to hearing loss. Marie (1906) also doubted the existence of such a syndrome. However, the subsequent description of several well-documented cases which came to autopsy established the syndrome as a distinct entity (Goldstein, 1974).

In recent years, investigators have attempted to characterize the nature of this deficit, and have emphasized the role of auditory processing (Jerger *et al.*, 1969; Kanshepol'sky *et al.*, 1973; Albert and Bear, 1974; Chocholle *et al.*, 1975; Gordon

and Naeser, 1977), temporal resolution (Albert and Bear, 1974); phonemic discrimination (Naeser, 1974; Chocholle *et al.*, 1975; Denes and Semenez, 1975; Saffran *et al.*, 1976), and contextual cueing (Saffran *et al.*, 1976). None of these authors addressed the possibility that this syndrome could present in more than one clinical form and that conclusions based on case studies could be contradictory. Such a possibility is implicit in the observation that this disorder has been seen in cases with either unilateral or bilateral lesions (Geschwind, 1965). Furthermore, the patterns of phonemic discrimination in patients with bilateral lesions (Naeser, 1974; Chocholle *et al.*, 1975) differ from those in patients with unilateral lesions (Denes and Semenza, 1975; Saffran *et al.*, 1976).

We have recently had the opportunity to study a patient with the syndrome of pure word deafness associated with amusia and a milder auditory agnosia for environmental sounds. On the basis of a detailed neurophysiological, neurolinguistic and neuropsychological study, we found a disordered pattern of linguistic discrimination that could be attributed to the patient's disorder in auditory processing.

In light of our own findings, and a review of the literature, we conclude that pure word deafness exists in two forms. The first is a disorder of prephonemic temporal auditory acuity and has been associated with bilateral temporal lobe lesions. The second is a disorder in phonemic discrimination that cannot be attributed to a difficulty in temporal auditory acuity and has been associated with left unilateral lesions. The former represents an apperceptive disorder secondary to pathology involving the auditory cortices whereas the latter may be a fragment of Wernicke's aphasia, especially the word-deaf type.

#### CASE REPORT

The patient is a 58-year-old, fully right-handed white male of French Canadian descent admitted to the Neurobehavioral Unit of the Boston Veterans Administration Medical Center on August 28, 1979 for investigation of an inability to understand spoken words.

In April of 1978 he had the sudden onset of a left hemiparesis and slurred speech. At that time, both spoken words and music sounded like noise. Initial evaluation at another hospital revealed no speaking, reading or writing difficulties. Although he could hear and recognize sounds, he could not differentiate spoken words. There was a left-sided sensory loss and a mild left hemiparesis. An isotope brain scan demonstrated abnormal uptake in the right parietal cortical and subcortical regions and an EEG was consistent with a structural lesion in the right hemisphere. He did well with conservative treatment and was able to walk easily within forty-eight hours. He was engaged in language therapy and despite great difficulty, he eventually learned to lip read.

He was admitted to the Neurology Service of the Boston Veterans Administration Center in July 1978 for investigation of right-sided amaurosis fugax. At first, his difficulty with auditory comprehension was attributed to a chronic hearing loss. There was no evidence of aphasia and only some mild constructional difficulties. He had a left-sided sensory loss with a mild left hemiparesis primarily affecting the arm and hand. Fibrin plaques were noted in the right ocular fundus. Computerized axial tomography (CT scan) demonstrated an enhancing right middle cerebral artery infarct, extending

from the right middle and posterior superior temporal region to the parietal lobe, including the post-central and supramarginal gyri; and a small, older, nonenhancing left posterior temporal lobe infarct including part of Wernicke's area (fig. 1). An electroencephalogram was within normal limits. A cerebral arteriogram demonstrated marked stenosis of the right internal carotid artery and minimal intracranial and left internal carotid artery disease. The patient underwent an uneventful right carotid endarterectomy and was discharged.

On the basis of a relatively normal pure tone audiogram, his physician suspected pure word deafness and referred him to the Aphasia/Behavioral Neurology Unit. In describing his symptoms, the patient stated that the speech of others often sounded like a 'foreign language'. Music was incomprehensible and he was unable to pick out familiar music or recognize instruments as he formerly did. Noise was bothersome and background music in a restaurant required ear plugs. He was dependent on lip reading and said, 'If I go blind, I won't hear anything.' Nevertheless, hearing the associated voices greatly augmented his lip reading ability, even though loud voices were bothersome to him. When asked specific questions relating to disorders of temporal resolution, he responded that people's voices seemed faster, that it helped when people spoke slower, and that words came too quickly (Albert and Bear, 1974), and would run together (Klein and Harper, 1956).

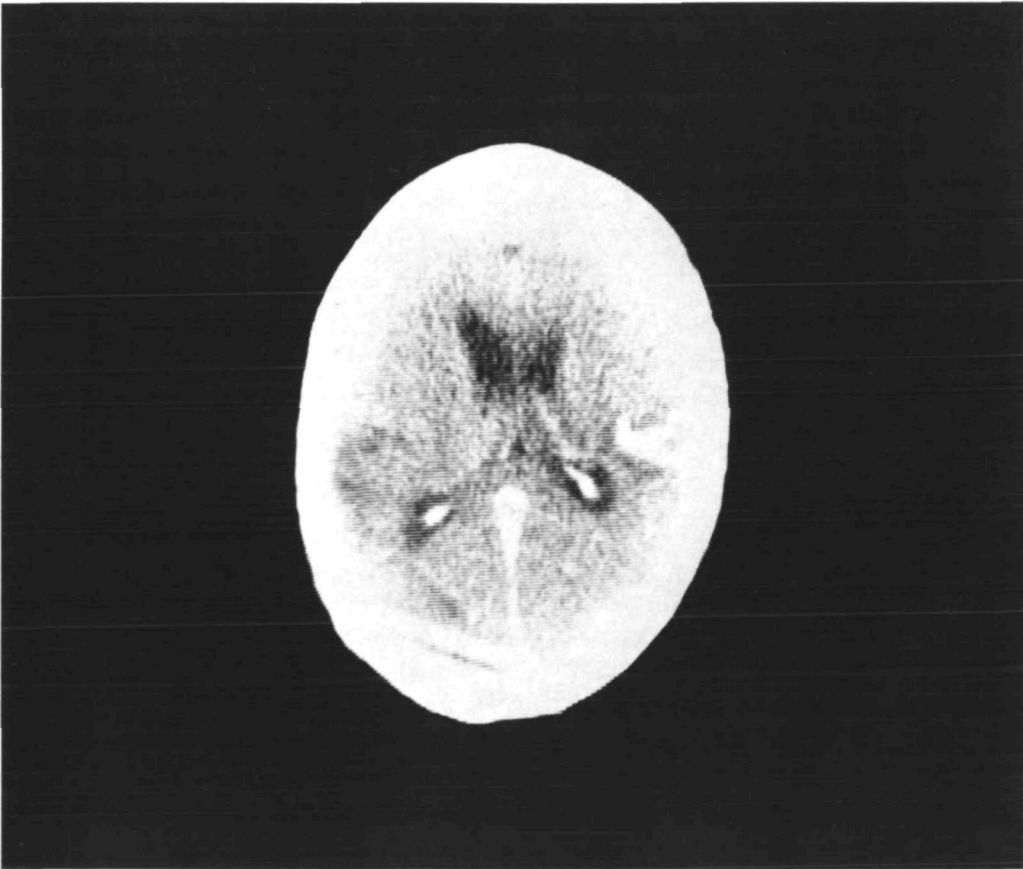


FIG. 1. Early CT scan performed in 1978 showing enhancement of the right posterior temporal lobe lesion and non-enhancement of the left posterior temporal lobe lesion.

He did not acknowledge changes in the quality of other people's voices, but he felt that his own voice sounded slightly different. He could still recognize an individual by voice even in a crowd, although he was unable to determine what language was being used even when it was a familiar language such as French or English.

### *Background*

The patient spoke both French and English as a child. In the Army, he served as a French interpreter and spoke Parisian French. Since World War II he has spoken little French. He completed one year of high school before leaving when aged 16 years. He had played the violin for about ten years as a child, but had not played an instrument since that time. Prior to the onset of symptoms, he had enjoyed listening to a wide range of music. After the war, he had a variety of jobs before becoming a machinist. He retired because of medical problems in 1971.

### *Past Medical History*

The patient had a history of atherosclerotic cardiovascular disease with angina pectoris and an old myocardial infarction. He suffered from peripheral vascular disease with intermittent claudication, chronic obstructive pulmonary disease, and chronic sinusitis. Medications on admission included theophylline, digitalis, aspirin, nitroglycerin.

### *Examination*

His general medication examination was remarkable for a right carotid bruit and findings consistent with pulmonary disease and peripheral vascular disease. Cranial nerve examination revealed left facial weakness. Visual fields were full to confrontation. He had a mild increase in tone in the left upper extremity with a mild left hemiparesis, more severe distally than proximally, left-sided sensory loss to all modalities and left-sided hyperreflexia with a left Babinski response. There were no frontal release signs. Cerebellar testing was normal.

### *Mental state*

He was alert and oriented in time and place. He understood his problem but was not disturbed by it and considered it a challenge. He was jocular and quite animated in conversation.

His speech was slightly slurred with occasional stuttering but was otherwise fluent and grammatically correct with normal phrase length. He made extremely rare paraphasic errors (panaapple for panhandle, maroon-blue for blue). He could distinguish environmental sounds when tested at the bedside with common objects (keys, crumpling paper, etc.). He was able to localize sounds and recognize voices. He could augment auditory comprehension with lip reading although he could not lip read without hearing the voice. Care was taken to test his auditory comprehension without visual cues. The patient was able to distinguish broad categories of sounds such as speech, music, environmental sounds. However, he was unable to detect whether the speaker was using French, English or Hebrew. He could not recognize familiar tunes and was unable to sing or carry a melody. He had particular difficulty perceiving initial consonants, but did extremely well with vowels. He used his ability to recognize vowels in listening to letters and numbers. Reading aloud and reading for comprehension, writing spontaneously and naming to confrontation were normal. He was able to generate a word list of 28 animals in one minute. He was able to reproduce a pointing sequence of three items reliably and four items approximately 50 per cent of the time.

The results of the standard administration of the Boston Diagnostic Aphasia Examination (Goodglass and Kaplan, 1972) are found in Table 1. These subtests were performed without restrictions on lip reading. It was noted that his performance on word discrimination, body part discrimination, written words to dictation and repetition of words deteriorated when lip reading was not allowed.

TABLE 1. RESULTS FROM THE BOSTON DIAGNOSTIC APHASIA EXAMINATION

<i>Subtest</i>	<i>Score</i>	<i>Max. possible score</i>
Fluency		
Articulation rating	3	5
Phrase length	6	7
Verbal agility	7	7
Auditory comprehension		
Word discrimination	72	72
Body part identification	20	20
Commands	6	15
Complex material	2	12
Naming		
Responsive naming	14	30
Confrontation	105	105
Animal	16	25
Body part	30	30
Oral reading		
Word	30	30
Sentence	9	10
Repetition		
Words	8	10
High probability phrases	1	8
Low probability phrases	0	8
Paraphasia		
Neologisms	0	12
Literal	0	16
Verbal	0	24
Extended	0	16
Automatized speech		
Automatized sequences	8	8
Reciting	1	2
Reading comprehension		
Symbol discrimination	10	10
Word recognition	7	8
Comprehension oral spelling	7	8
Word picture match	10	10
Reading sentences, paragraphs	10	10
Writing		
Mechanics	3	3
Serial writing	46	47
Primer dictation	15	15
Written confrontation naming	9	10
Spelling to dictation	8	10
Sentences to dictation	0	12
Narrative writing	3	4
Music		
Singing	0	1
Rhythm	1	2

Praxis was normal, as were finger recognition and right-left discrimination. Calculations were fairly good. Constructions were remarkable for loss of three dimensionality. Map drawing was fair. When asked to draw a clock with the hands at '10 after 11' he drew a clock with the numbers 5 and 10 substituted for 1 and 2, respectively. Memory for verbal material and hidden objects was intact. Remote memory was normal.

## INVESTIGATIONS

The following investigations were performed: (1) neuroanatomical, (2) neurophysiological, (3) neurolinguistic, and (4) neuropsychological.

### 1. *Neuroanatomical Investigation*

A repeat CT scan with 7 mm slices was obtained in May 1980 (approximately two years after onset of language problems). The following represents a summary of the detailed anatomical study (fig. 2).

*Left hemisphere lesion.* The left temporal lobe lesion was small and patchy at the surface of the middle portion of the left superior temporal gyrus (slice 1). Heschl's gyrus was probably intact, although a lesion was present in the white matter inferior to Heschl's gyrus (slice 2). At the level of the third ventricle (slice 2) a small lesion was present (cortical and deep) in the middle portion of the more inferior half of the superior temporal gyrus. A lesion was also present in the left temporal isthmus where geniculotemporal auditory radiations pass from the left medial geniculate body (primarily right ear) towards the left Heschl's gyrus and Wernicke's area (left arrow, slice 2). The left temporal isthmus was spared 7 mm below, at slice 1, as well as 7 mm above, at slice 3. The major part of the left temporal lobe lesion was visualized in the left posterior superior temporal gyrus area (Wernicke's area) on slice 3. There was only a very small, patchy left parietal lobe lesion in the area of the left supramarginal gyrus at slice 4.

The number of pixels (1 mm<sup>2</sup> picture elements) in the lesion at each slice was computed at the cathode ray tube viewer screen using the Ohio Nuclear CT scanner MAP function. There were 596 lesion pixels in Wernicke's area at slice 3 (2.4 × 2.4 cm). This was small compared to a mean of 737 pixels (2.7 × 2.7 cm) for Wernicke's aphasics (range, 500 to 1344, SD 406) (Naeser *et al.*, 1981). This patient's Wernicke's area lesion extended minimally into the left supramarginal gyrus area (slice 4). This left supramarginal gyrus area lesion was also smaller than that usually observed with Wernicke's aphasics, i.e. 552 lesion pixels at slice 4 in this case, vs. 675 mean number lesion pixels for Wernicke's aphasics (range 272 to 1592, SD 615) (Naeser, *et al.*, 1981). Hence, from the small Wernicke's area lesion size, it is possible this patient could have presented with a mild conduction aphasia before the second stroke, had he been tested for it.

*Right hemisphere lesion.* The right Heschl's gyrus was intact at slice 1. At slice 2, there was more destruction of Heschl's gyrus on the right than on the left, although some minimal sparing was probably present. There was considerable destruction of white matter inferior to the right Heschl's gyrus (slice 2). At this third ventricle level (slice 2) a lesion was also present (patchy cortical and deep) in the middle portion of the more superior part of the right superior temporal gyrus. A lesion was also present in the right temporal isthmus where geniculotemporal auditory radiations pass from the right medial geniculate body (primarily left ear) towards the right Heschl's gyrus (right arrow, slice 2). The right temporal isthmus was spared 7 mm below at slice 1, but there may have been some superior extension of the deep temporal isthmus lesion 7 mm above, at slice 3.

The major part of the right temporal lobe lesion was visualized in the right posterior superior temporal gyrus area on slice 3. The number of lesion pixels in this right posterior superior temporal

gyrus lesion was 588, approximately the same size as the left posterior superior temporal gyrus lesion (596 pixels or  $2.4 \times 2.4$  cm). This right hemisphere lesion was much larger at slices 4, 5, 6 and 7 in contrast to the left hemisphere lesion, which was minimally present at slice 4 and totally absent at slices 5, 6 and 7. The right hemisphere lesion extended into the right frontal lobe deep to the precentral gyrus at slice 4, and was cortical and deep to the postcentral gyrus and anterior part of supramarginal gyrus at slices 4, 5, 6 and 7. This large right parietal lobe lesion including the right pre- and postcentral gyri and right supramarginal gyrus areas extended from the cortex, deep to periventricular white matter areas. It was approximately  $4 \times 4$  cm at each higher slice.

*Comment.* There were two lesions. The lesion on the right enhanced four months after onset on the first CT scan. The lesion on the left never enhanced. Therefore, it appears that the left temporal lesion was an older, previously silent infarct.

Auditory fibres were presumably interrupted as they passed through the temporal isthmus on the right and the left. In addition, the right parietal lobe lesion in the deep

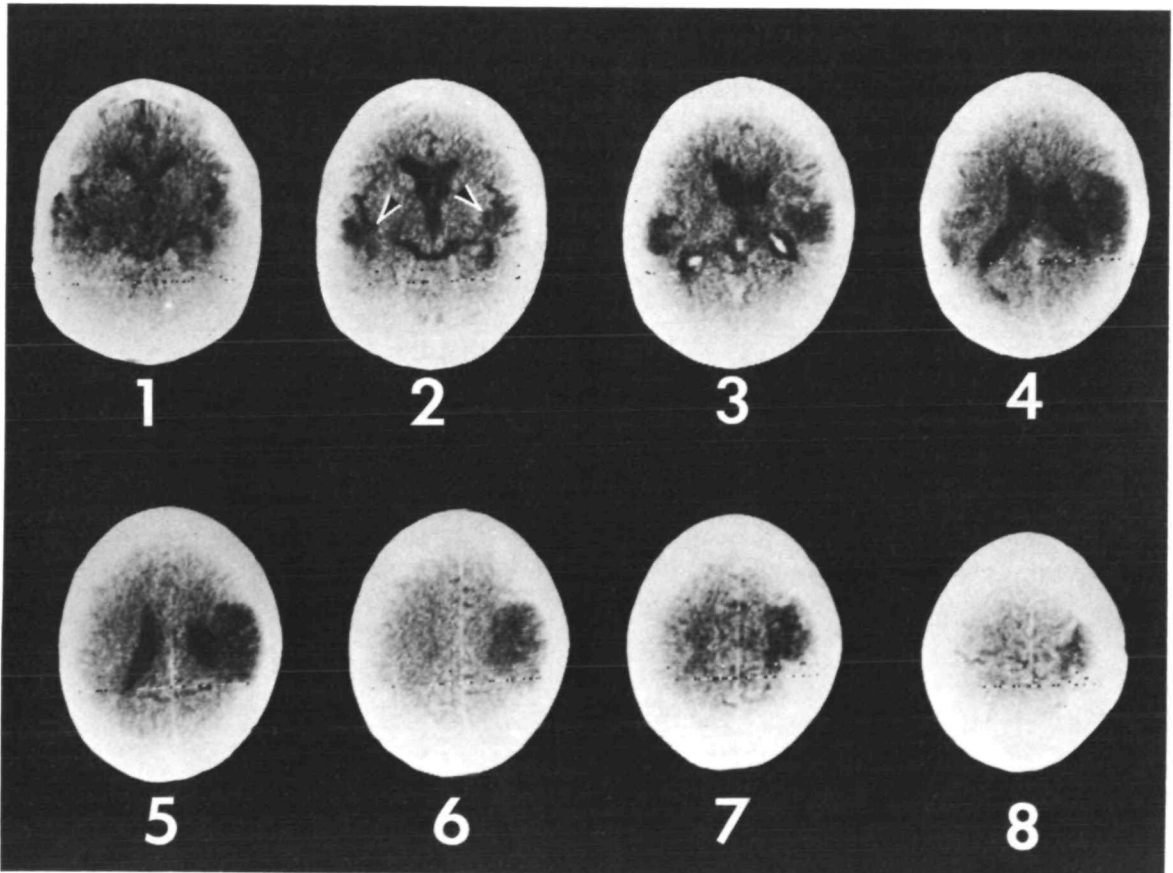


FIG. 2. Late CT scans performed two years after onset in 1980 showing bilateral posterior temporal lobe lesions with extension into the temporal isthmus on each side (arrows, slice 2). The right hemisphere lesion was larger than the left, with extension into precentral, postcentral and supramarginal gyrus areas, cortical and deep to border of lateral ventricle.

posterior periventricular white matter at slices 4 and 5 may have interrupted the interhemispheric auditory pathway from the right temporal lobe to the left (Damasio and Damasio, 1979).

We concluded from these observations that (1) the left hemisphere lesion was clinically silent prior to the onset of pure word deafness and therefore insufficient to account for this syndrome; (2) at the time of our investigation, the auditory radiations on the right were almost completely interrupted. Not only was primary cortical auditory processing interrupted in the right geniculotemporal tracts, but the interhemispheric auditory pathway from the right temporal lobe to the left was also probably interrupted; and (3) at the time of our investigation, cortical auditory processing occurred only in the left temporal lobe.

## 2. *Neurophysiological Investigation*

This investigation was undertaken in the following sequence: pure tone audiometry to provide a standard measure of peripheral auditory function; brainstem auditory evoked responses to study the intactness of brainstem auditory pathways; dichotic listening studies to assess functional hemispheric lateralization; cortical auditory evoked potentials to provide another measure of cortical function and to address the issue of potential paradoxical responses; and measures of auditory acuity (to be defined below) to assess the nature of the auditory processing deficit.

Our review of the relevant literature suggested that we would find relatively normal function through the brainstem. We anticipated that our data on hemispheric function would be consistent with our neuroanatomical study and demonstrate predominant left hemispheric function and that this observation would differ from other reports of pure word deafness patients with predominantly left hemisphere lesions. Finally, based on studies of auditory cortical lesions (reviewed by Baru and Karasseva (1972) and others) we anticipated finding a major disorder in temporal auditory acuity.

(a) *Pure tone threshold audiometry.* Standard pure tone threshold audiometry was performed (Table 2) and revealed a mild, bilateral sensorineural hearing loss at frequencies up to 2000 Hz and a moderate high frequency loss.

*Comment.* This degree of hearing loss is not sufficient to explain such a profound comprehension deficit.

(b) *Brainstem auditory evoked responses.* Brainstem auditory evoked responses were obtained using a Grass Model 10 ERS-8 Evoked Response System. Each trial utilized 0.1 ms clicks at 11 Hz and 60 dB above threshold. Contralateral masking was used for all trials. Each trial represented at least 1024 responses. Trials were obtained on several occasions. Representative tracings are demonstrated in fig. 3. Waves I-V are within normal limits for our laboratory.

*Comment.* Our patient has normal waves I-V suggesting intact brainstem auditory function in response to clicks (McCutchen and Iragui-Madoz, 1979).



TABLE 2. PURE TONE THRESHOLD AUDIOMETRY

Frequency (Hz)	Pure tone threshold (dB)	
	Left ear	Right ear
250	45	45
500	40	40
1000	35	35
2000	40	30
3000	65	45
4000	80	65
8000	80	65

(c) *Dichotic listening studies.* Dichotic listening techniques have been widely used in the study of speech discrimination and temporal lobe function. Their use has a basis in studies of speech perception lateralization (Kimura, 1961) and in phonemic analysis (Studdert-Kennedy and Shankweiler, 1970). The technique has been useful in lesion localization (Sparks and Geschwind, 1968; Sparks *et al.*, 1970). Using this technique in temporal lobectomy patients, Berlin *et al.* (1972) concluded that the anterior temporal lobes play a critical role either in preliminary speech analysis or in the relay of speech information to the posterior temporal cortex *via* association pathways. Furthermore, they suggested that information passing from the right anterior temporal lobe to the left posterior temporal lobe does not necessarily pass through the left anterior temporal lobe.

Dichotic listening techniques had been used in two other patients with pure word deafness (Albert and Bear, 1974; Saffran *et al.*, 1976). Both these patients had presumed left unilateral lesions and both demonstrated strong right ear extinction. Our patient had bilateral lesions with a predominant right hemisphere lesion and a different result was anticipated.

There is an extensive literature on dichotic listening studies of speech and nonspeech stimuli. Unfortunately, conclusions based on right or left ear advantage in normal subjects are not necessarily applicable to a study of a brain-damaged patient. For example, a right ear advantage might suggest a left hemisphere dominance but does not exclude the possibility that the right hemisphere cannot or does not perform the task that is in question.

*Method.* The dichotic listening tapes (one verbal, digit triads; one musical, hummed nursery tune pairs) were administered to the patient by Dr Joan Borod. A free recall procedure was used. Because of the patient's bilateral hearing loss, the volume for the dichotic input was augmented equally for the two ears.

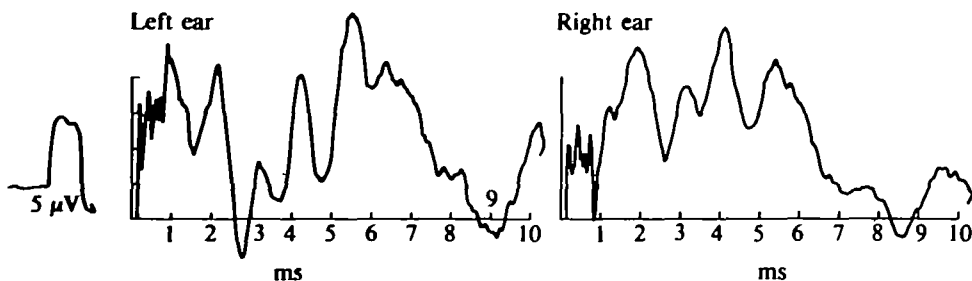


FIG. 3. Brainstem auditory evoked potentials. Recordings are from Cz to the left or right ear, respectively; 2048 signals were averaged. Filters were set at 30 and 3K. Waves I-V were intact in both tracings.

**Results.** For the digit triads, 6 practice trials were first administered: 2 dichotic trials and 4 monaural trials (2 from each ear). Twenty-eight dichotic trials were next presented. The patient correctly recalled 69/84 digits from his right ear and 6/84 digits from his left ear. Since awareness of the left ear input was questioned, 9 monaural trials were administered to the left ear. The patient correctly reported 20/27 digits. An additional 11 dichotic pairs were presented, for which the patient again attended almost exclusively to his right ear and reported 29/33 from the right and 1/33 from the left. His total scores for 39 dichotic triads were 7/117 correct with the left ear, 98/117 correct with the right ear.

With the nursery tunes, the patient was never able to identify correctly the five tunes monaurally. The patient was told that the pairs would be made from these five tunes. When the 24 dichotic pairs were presented, he correctly identified 9 of the possible 48 tunes, a finding at the chance level.

*Comment.* His ability to understand digits may seem surprising. It is likely that this is related to the earlier observation that he was able to identify vowels in spoken language. All spoken numerical digits have a distinctive vowel except for five and nine which share a similar vowel. This ability was not useful with the nursery tunes.

The results from the dichotic listening study are consistent with our anatomical data.

(d) *Cortical auditory evoked potentials.* Late component evoked potentials to nonspeech stimuli were obtained using a Grass Model 10 ERS-8 Evoked Response System. Several trials were performed on several different occasions. Two techniques were used. On the initial studies, the stimulus was always a 2 Hz click, 60 dB above threshold with contralateral masking. Filters were varied with the low filters set at 1 to 10, the high filters 100 to 300. Time sweep was 200 ms and each trial represented at least 256 responses.

In a later study, the stimulus was changed to 50 ms trains of 100/s clicks presented at the rate of 1/2 s. Filters were set at 1 and 30, time sweep was 1000 ms and each trial represented 128 responses. Stimuli again were presented at 60 dB above threshold with contralateral masking.

All trials produced consistent results. Stimuli presented to the right ear produced a prominent response largely seen over the left hemisphere. P<sub>1</sub>, N<sub>1</sub> and P<sub>2</sub> were easily identified with latencies at about 45, 85 and 190 ms, respectively. Stimuli presented to the left ear produced a minimal response over either hemisphere. Representative trials are found in fig. 4.

*Comment.* The above investigations indicate a disorder of auditory processing above the brainstem. The presence of late responses suggests some intact cortical auditory activity, at least in response to nonspeech stimuli (Vaughan and Ritter, 1970; Goff *et al.*, 1980). The marked asymmetry in response to right ear stimulation is consistent with the apparently complete posterior peri-Sylvian lesion noted in the anatomical study (Auerbach and Naeser, 1981). Furthermore, the trials with left ear stimulation demonstrated an abnormal response bilaterally.

The observed auditory evoked potentials, however, are consistent with our anatomical and dichotic listening study data, suggesting a disruption of auditory processing in the right hemisphere. Paradoxical responses have been reported in dichotic listening studies (Damasio and Damasio, 1979), but the auditory evoked potentials are consistent with the interpretation that the contralateral cortical pathways from the right ear to left temporal cortex are relatively preserved. Furthermore, the contralateral response to right ear stimulation distinguishes this case from cortical deafness (Michel *et al.*, 1980).

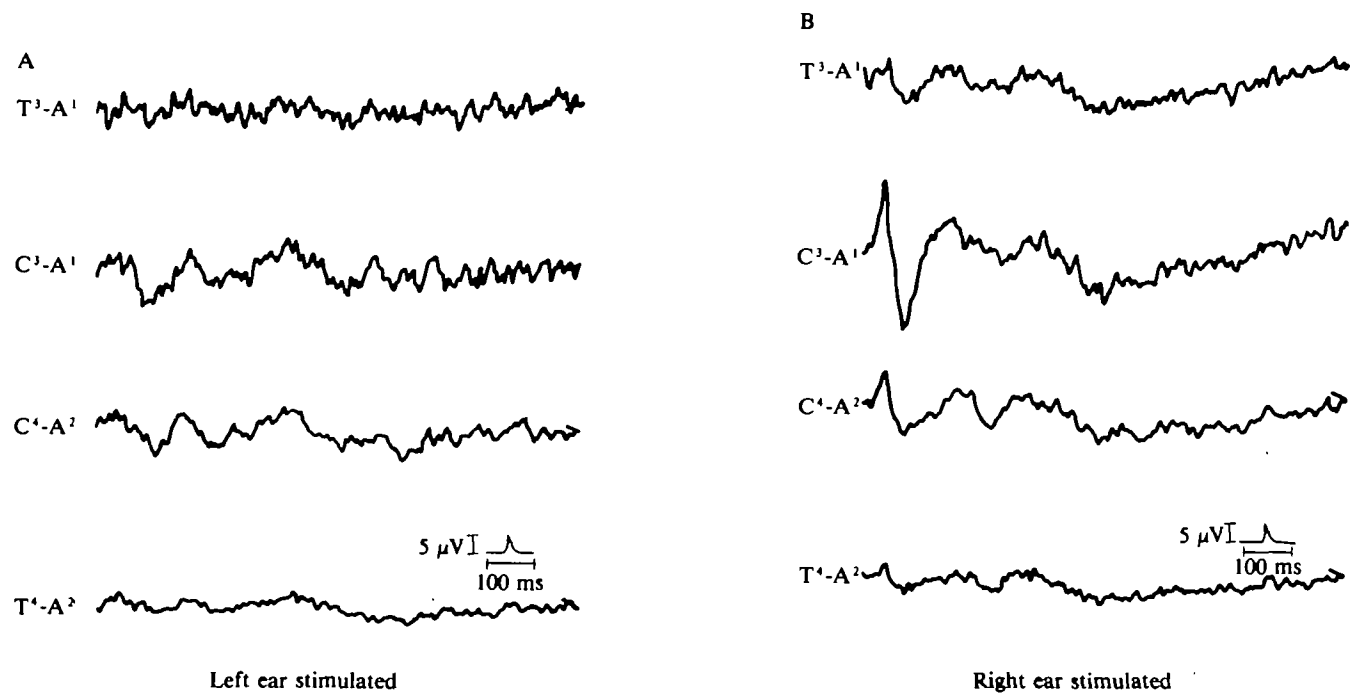


FIG. 4. Representative tracings of cortical evoked potentials. Stimuli were trains of 100/s clicks, 50 ms in duration, presented at the rate of  $\frac{1}{4}$  s at 60 dB above threshold with contralateral masking. Filters were set at 1 and 30. Each tracing represents an average of 128 responses. Tracings represent trials with either left ear stimulation (A) or right ear stimulation (B).

A word of caution is warranted in the interpretation of the cortical auditory evoked potentials to nonspeech stimuli. There is insufficient evidence available to state that normal cortical auditory evoked potentials to clicks imply normal auditory processing. However, the implication employed here is that abnormal cortical auditory evoked potentials imply abnormal auditory processing. Such logic is the basis for the development of the term 'hemiacousia' to denote hemispheres deprived of auditory input (Michel *et al.*, 1980). It is assumed that the abnormal auditory processing demonstrated in this manner for nonspeech stimuli will apply to speech stimuli. There are no reports, to our knowledge, of patients with abnormal cortical auditory evoked potentials for nonspeech stimuli where the evoked potentials for speech stimuli were normal.

(e) *Temporal auditory acuity measures.* Temporal auditory acuity refers to the ability to discriminate short durations: it is a measure of the temporal resolution of the auditory system and is analogous to the spatial resolution of the visual system. In this instance, temporal refers to time and not to temporal lobe.

Several studies have documented the presence of a temporal auditory acuity disorder in auditory cortex lesions. Neff (1961) studied auditory cortex lesions in animals and found significant impairments in duration discrimination. Studies in humans have demonstrated disorders of temporal auditory acuity in patients with unspecified lesions (Van Allen *et al.*, 1966), aphasics with old left hemisphere lesions (Lackner and Teuber, 1973), patients with superior temporal lobe lesions (Karasseva, 1972); and more specifically, with lesions of either auditory cortex (Baru and Karasseva, 1972), and aphasics with lesions of the auditory cortex or auditory radiations (Auerbach and Naeser, 1981). Finally, Tallal and Newcombe (1978) have suggested that a deficit in temporal auditory acuity may account for a disorder in phonemic discrimination.

Various measures of duration discrimination and temporal auditory acuity have been developed. We selected click fusion thresholds and click counting thresholds, measures also utilized by Albert and Bear (1974).

(i) *Click fusion threshold.* Procedure. The interval between two brief binaural pulses was varied and the patient was asked to report either two clicks or one. Clicks were generated by a Grass S8 Stimulator and were each 0.1 ms duration and delivered binaurally with earphones. A click fusion time was established by the method of ascending and descending limits.

Results. Our patient reliably fused at intervals of 30 ms. Below this interval he could rarely detect distinct clicks. Normals can distinguish two clicks at 1 to 3 ms separations (Hirsh, 1975).

(ii) *Click counting.* This technique is a variation of the one used by Albert and Bear (1974). Binaural presentation of clicks was given using the click generator of the Model 10 Grass ERS-8 Evoked Response System. The patient was asked to count the clicks presented in a 1 to 2 s interval. A range of accurate counting in normals in our laboratory is 9 to 11 clicks per second. Our patient was inaccurate for rates greater than 2 per second.

*Comment.* The results of both click fusion and click counting show that our patient had a severe deficit in temporal auditory acuity.

In summary, we performed a series of neurophysiological investigations designed to delineate the nature of the auditory processing deficit. We demonstrated (a) a mild to moderate deficit (approximately 40 dB) in the speech frequency range with a more severe high frequency deficit in pure tone audiometry, (b) normal brainstem auditory click evoked responses suggesting intact brainstem auditory function, (c, d) abnormal dichotic listening studies and cortical auditory evoked potentials

indicating a major right hemisphere deficit, and (e) abnormal click fusion and click counting thresholds consistent with a temporal auditory acuity disorder. We concluded that our patient had a severe temporal auditory acuity disorder based on bilateral damage to auditory cortical systems which was more severe on the right side.

### 3. *Neurolinguistic Studies*

Our neurophysiological investigation had demonstrated a temporal auditory acuity disorder; however, it was still necessary to demonstrate the possibility of a relationship of this disorder to our patient's difficulty in the comprehension of spoken words. We therefore investigated the neurolinguistic dimensions of his deficit. Our evaluation of the patient's linguistic deficit was directed towards a possible disorder in discrimination of phonemes. Such an approach had been taken in other patients with pure word deafness (Naeser, 1974; Chocholle *et al.*, 1975; Denes and Semenez, 1976; Saffran *et al.*, 1976).

Vowels and consonant-vowel combinations (CV) have received particular attention in studies of phonemic discrimination. When plotted on a frequency spectrogram, vowels are represented by steady state characteristic frequencies. In natural speech, vowel durations usually average 100 to 150 ms but may last as long as 400 ms. CV combinations formed with stop consonants such as ba, pa, da, ta, ga or ka all contain early rapid formant transitions. In these CV combinations, the vowels are characterized by steady state formants whereas the consonant is characterized by rapid frequency changes within the first 40 ms of onset of stimulus.

Two distinctive features have been used further to distinguish these CV combinations—place of articulation and voicing. Place of articulation refers to the place of vocal tract occlusion at the release of the consonant: labial (ba, pa); alveolar (da, ta); and velar (ga, ka). Voicing measured acoustically with voice onset time (VOT) refers to the time between the onset of vocal cord vibration and the initial burst release and, therefore, distinguishes the pairs: ba and pa, da and ta, ga and ka. On the other hand, the triads ba, da, ga and pa, ta, ka each have a similar VOT. If the vowel or steady state frequencies used in the CV stimuli (ba, pa, da, ta, ga, ka) is the same, then the distinguishing information is contained in the spectrum of the burst and the brief, early, changing, rapid formant transitions.

Linguistic analysis has potential value because of data suggesting laterality of function in phonemic discrimination (dichotic listening studies have provided much of this data). There is a right ear advantage for perception of speech stimuli (Kimura, 1961) and in particular for phonemic analysis (Studdert-Kennedy and Shankweiler, 1970). Even though auditory processing may occur bilaterally, linguistic processing appears to occur in the dominant hemisphere (Studdert-Kennedy and Shankweiler, 1970; Oscar-Berman *et al.*, 1975).

A final characteristic makes these CV combinations attractive to study. When two target phonemes are selected that differ by a single distinctive feature, the existence of a continuum along which there is a categorical boundary can be demonstrated.

For example, /da/ and /ta/ share the same place of articulation (alveolar) and differ in voicing. If all acoustic parameters are held constant except VOT, a continuum can be artificially created. When subjects listen to the items in this continuum, a 'categorical' boundary is observed with /da/ always perceived on one side and /ta/ on the other. Discrimination is much simpler across boundaries compared to discrimination within boundaries (Abramson and Lisker, 1967; Stevens, 1975).

We wanted to establish that the pattern of phonemic discrimination in pure word deafness could be predicted on the basis of a temporal auditory acuity disorder in a patient with otherwise intact language function. Such a pattern would demonstrate (1) a superior performance in vowel discrimination as observed in our initial clinical assessment; (2) improvement with an increase in the number of distinctive features distinguishing two target phonemes; (3) a particular vulnerability of the distinctive feature place compared to VOT (Miller and Nicely, 1955); (4) relative preservation of categorical boundaries on synthetic speech tests. If such a pattern existed then it would meet our criteria for being considered prephonemic.

The term 'prephonemic' is used in a specific manner in this discussion. Aphasia is the term used to refer to acquired disorders of language. The existence of an aphasic disorder is determined independently of nonlanguage input or output functions. For example, patients with peripheral deafness are not considered aphasic. In general, a phonemic discrimination disorder can be considered prephonemic if it can be attributed to a disorder in acoustic processing. A prephonemic disorder, therefore, should not be considered an aphasic disorder. In our patient we identified the disorder in acoustical processing to be a deficit in 'temporal auditory acuity' as defined above. We excluded a peripheral auditory disorder. Therefore, in this case a prephonemic disorder could be demonstrated if the deficit in phonemic discrimination could be attributed to a temporal auditory acuity disorder.

(a) *Vowel identification—natural speech.* The patient was asked to identify the long vowels, a, e, i, o, u. One hundred randomly ordered vowels were presented by a native English-speaking male voice. Special care was taken to avoid cues from lip reading or other facial gestures.

*Results.* The patient identified all the stimuli correctly.

(b) *Phonemic discrimination/identification—natural speech.* All the following tasks were performed with a native English-speaking female voice. Special care was taken to avoid cues from lip reading or other facial gestures. All items were in a consonant-vowel (CV) format. This means that there is an initial consonant paired with a vowel. In this case, the vowel was always /a/. The initial stop consonants differed according to place of articulation (labial, alveolar, velar) or voicing (voiced, voiceless). These tasks are performed by normal subjects without error.

(i) Binary decision tasks.

(a) Binary decision with single distinctive feature. The patient was told that each item in each test represented one of two possible choices which had been shown to him in written form. The condition refers to the possible choices in each test. All items were in a CV presentation method with the vowel /a/. Nine tests of 100 items each were administered. He performed above chance on each of the 9 tests with a very consistent error rate across all tests.

## Results.

Distinction	Condition			Nature of distinction
	/ba/-/pa/	/da/-/ta/	/ga/-/ka/	
% correct	71	72	67	Voiced/ voiceless
Distinction	/ka/-/ta/	/pa/-/ta/	/pa/-/ka/	Place of articulation for voiceless stops
% correct	78	66	71	
Distinction	/da/-/ga/	/da/-/ba/	/ba/-/ga/	Place of articulation for voiced stops
% correct	84	76	78	

Average per cent correct across all 9 tests = 74. A 60 item spot check was done a month later and yielded a 73 per cent correct response rate.

(b) Binary decision with two distinctive features. Three tests of 60 items each were given in exactly the same way as the tests above. The only difference was that the forced choice was between two phonemes that differed by two features instead of one.

## Results.

Distinction	/ka/-/da/	/ga/-/pa/	/ba/-/ta/
% correct	98	83	97

Average per cent correct across all 3 tests = 93.

*Comment.* When two target phonemes differed by two instead of one distinctive feature the patient responded correctly at a rate of 93 per cent as compared to 74 per cent. This suggested that the patient was able to take advantage of the additional phonemic information.

(ii) Three way decision for place of articulation. Two tests of 150 items each were given in a similar CV presentation. In each test the patient was told that the stimulus would be one of these possible target CV combinations. The three possible targets were varied between tests. The patient was asked to identify each stimulus item. In each test, a triad of stimuli was chosen which shared similar voicing and differed by place of articulation.

## Results.

Distinction	/ba/-/da/-/ga/	/pa/-/ta/-/ka/
% correct	69	53

(iii) Ten way decision. The patient was given a choice of 10 consonants and asked to identify each of 100 items as one of these consonants /b, p, d, t, g, k, m, n, s, z/ were used in CV and VC combinations with the vowel /a/. For the stop consonants there were 11 voicing errors, 33 place errors and 3 other errors.

*Comment.* There is a particular vulnerability for errors in the discrimination of place of articulation. This is probably part of a more general vulnerability for errors in the discrimination of the initial rapid modulation of both speech and nonspeech sounds.

Comment on the linguistic investigation with natural speech stimuli. An initial

evaluation indicated no difficulty in vowel discrimination. Two additional characterizations are derived from the above studies: an improved performance when the number of distinguishing features increased and particular vulnerability for the distinctive feature of place of articulation. Therefore, all of our criteria for a prephonemic disorder were met except a demonstration of preserved categorical boundaries.

Two additional features are noteworthy: (1) the disorder of phonemic discrimination in our patient represents a relative impairment; the patient scored considerably below normal, but still above chance on our tests of phonemic discrimination; (2) the patient performed quite well on the Word Discrimination subtest of the Boston Diagnostic Aphasia Examination despite a poor performance on our tests of phonemic discrimination. This probably reflects the fact that it is impossible to make a strict one-to-one correspondence between these tests. In the Word Discrimination subtest, it is likely that the patient was able to rely on his superior vowel discrimination (*see* section on dichotic listening) and he could rely on contextual cues (Saffran *et al.*, 1976; Caplan, 1978).

(c) *Phonemic discrimination/identification synthetic speech.* Synthetic speech stimuli are useful because they allow for the careful control of acoustic parameters. If two target phonemes differ by a single distinctive feature then a continuum between them can be used for careful study. Particular attention has been paid to the categorical boundaries in these continua across which a jump in perception occurs (Liberman *et al.*, 1967; Abramson and Lisker, 1967).

(i) Identification tasks.

(a) VOT continuum. A voice onset time (VOT) continuum of 11 stimuli ranging in value from -20 ms to +80 ms in 10 ms steps was synthesized to simulate an alveolar progression from /da/ to /ta/. An identification tape consisted of 146 randomly ordered stimuli separated by an interstimulus interval of 4 s: each VOT value was presented at least 10 times.

(b) Place of articulation continuum. A second identification tape, provided by D. Klatt, consisted of 14 blocks of 10 randomly ordered synthetic stimuli representing 14 values along a place of articulation continuum from /ba/ through /da/ to /ga/ holding VOT constant; stimuli were presented every 3 s within each block.

(ii) Discrimination. A discrimination tape comprised stimulus pairs with either identical elements or elements that differed from each other by 20 ms VOT. The members of each pair were separated by 1 s of silence, and the intertrial interval was 3.5 s. Three blocks of 47 discrimination trials were recorded in separate random orders. Each block contained 11 pairs composed of two identical elements and 36 pairs composed of two different elements (*see* Blumstein *et al.* (1977) for details of VOT test materials).

Results. The identification of a synthetic VOT continuum (fig. 5) and place of articulation continuum (fig. 6) were within normal limits. Categorical boundaries appeared in the VOT continuum at about 40 ms. The place of articulation continuum (fig. 6) also demonstrates the clear boundaries seen in normals (S. Blumstein, personal communication).

The discrimination task (fig. 7) revealed a 'blunted' response. When both stimuli were from the same side of a categorical boundary the patient recognized them as the same. When the patient had to make fine discriminations across the boundary, he made frequent errors.

*Comment.* The investigation with synthetic speech stimuli demonstrated preserved categorical boundaries as determined by the labelling tasks, and difficulties with fine discriminations along the categorical boundaries. We had anticipated the relative



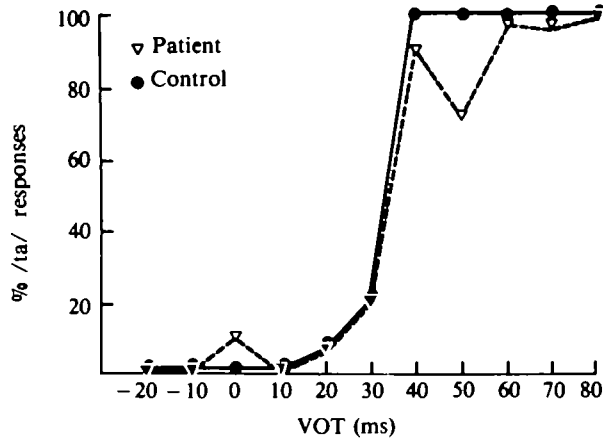


FIG. 5. Results of labelling task for a VOT continuum. The identification function for a synthetic VOT continuum for the word deaf patient and a normal bilingual control subject (R.R.). The ordinate represents the percentage of '/ta/' responses for each stimulus value from -20 to +80 ms VOT.

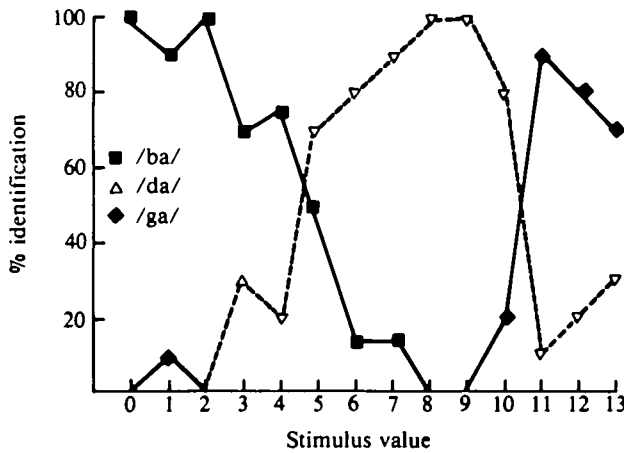


FIG. 6. Results of labelling task for a place of articulation continuum. The identification function for a synthetic place of articulation continuum for the word deaf patient. Each curve represents the percentage of '/ba/', '/da/' or '/ga/' responses at each stimulus value on the continuum.

preservation of categorical boundaries; the difficulty with fine discrimination would be predicted on the basis of his temporal auditory acuity disorder. Saffran *et al.* (1976), on the other hand, found no evidence of preserved categorical boundaries in their patient with a unilateral lesion.

We cannot exclude the possibility that there is some deficit in the understanding of spoken words that cannot be attributed to the prephonemic disorder. Were the deficit in temporal auditory acuity to show some or complete recovery, this would enable one to determine whether there was an independent residual deficit in comprehension, but such a possibility at this stage is hypothetical.

To make a final comment on the neurolinguistic investigation, we established certain criteria for determining a prephonemic deficit related to a temporal auditory acuity disorder and all of them were observed in our patient.

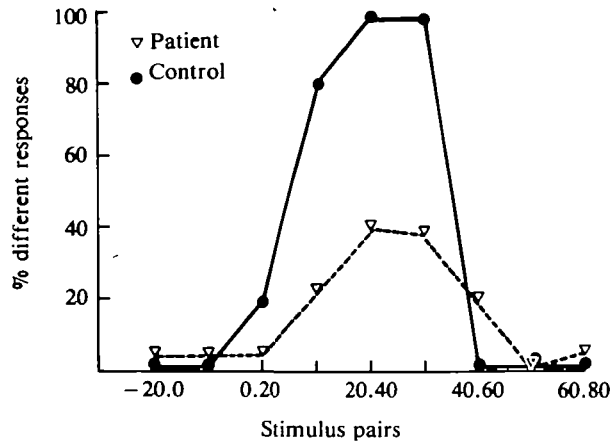


FIG. 7. Results of discrimination task. The discrimination function for a synthetic VOT continuum for the word deaf patient and a normal bilingual control (R.R.). The ordinate represents the percentage of 'different' judgements for each different comparison value; stimulus pairs made up of identical elements are not represented in this figure.

#### 4. Further Neuropsychological Studies

After studying his deficits in auditory and linguistic processing we wanted to examine other aspects of his deficits. In particular, we examined his musical abilities, abilities to distinguish broad categories of sounds, abilities to distinguish the language of a speaker and abilities to detect environmental sounds.

(a) *Music testing.* When asked to sing a familiar song, the patient produced no familiar melody or rhythm. In addition, he was unable to repeat single notes. He was also unable to recognize any familiar melodies. When provided with a three way multiple choice he was able to choose correctly 2/6 songs.

In another test, the patient was asked to determine whether a tape-recorded sequence of either 2 or 4 items was ascending or descending. Each test was administered 3 times and all scores were combined.

Results.

Two tones		Four tones	
Interval	% correct	Interval	% correct
1/2 tone	53	1/2 tone	53
Whole	31	Whole	78
Third	89	Third	85
Fourth	75	Irregular, spanning more than one octave	89
Fifth	92		
Octave	97		

*Comment.* The patient displayed a profound amusia which is not uncommon in pure word deafness. The fact that he was able to determine whether a tonal sequence was ascending or descending for longer intervals is of some interest.

(b) *Distinguishing categories of sounds.* Method. The patient was told that he would hear an item on a tape recorder from one of three categories:

- He knew the tape contained
- A—music      singing  
                     humming  
                     instrumental
- B—noises
- C—language    English  
                     French  
                     German

Results. He was able to distinguish between the three broad categories A, B, C almost perfectly. He was very good at identifying singing vs. humming (i.e. the presence vs. absence of language) vs. instruments.

(c) *Distinguishing between different languages.* In a separate test, the patient was told that he would hear short segments of speech in one of 4 possible languages (French, English, Russian or Chinese) and that he would have to identify the language. He scored 10/30 correct.

*Comment.* His inability to distinguish languages may reflect his inability to utilize either the semantic information or the musical qualities that distinguish foreign languages.

(d) *Recognition of environmental sounds.* In a natural setting, using objects in the examining room the patient did quite well, scoring 17/17 correct. A score of 19 out of a possible 39, however, was obtained on the Spreen and Benton Sound Recognition Test (Spreen and Benton, 1969) (See Table 3).

When asked to produce vocal approximations to sounds, he produced acceptable versions (i.e. a motor cycle engine, the ocean waves . . .).

*Comment.* Although his agnosia for environmental sounds was not initially apparent, it was elicited by finer testing. The data collected in our linguistic studies were not comparable to the data collected in the sound recognition tests. One cannot compare the ability to discriminate /ba/ and /pa/ to the ability to distinguish whether a sound represents a telephone ringing or a door slamming. A more detailed analysis of auditory agnosia for environmental sounds would be quite interesting but it was

not directly related to the main point of our paper. Unfortunately, the methods required for an analysis of his auditory agnosia for environmental sounds that would be comparable to the methods used in our study of his word deafness are not readily available.

TABLE 3. RESULTS OF SPREEN AND BENTON SOUND RECOGNITION TEST

<i>Stimulus</i>	<i>Response</i>	<i>Score</i>
1. Cat	Girl singing	0
2. Coughing	Man speaking in a different language	0
3. Bell	Bell, clock of a steeple tower	3
4. Applause	Clapping	3
5. Machine gun	Somebody climbing steps	0
6. Fog horn	A car running	0
7. Airplane	Sounds like seashore waves	0
8. Telephone ringing	Telephone ringing	3
9. Piano	Music (could not determine instrument)	2
10. Woman speaking English	Man making instructions to someone (when asked to specify language: English)	2
11. Birds	Tea kettle whistle	0
12. Door slamming	Closing doors	3
13. Trumpet	Two different musical instruments playing. When asked to specify: a trumpet and drums, like snare drums	3

#### SUMMARY OF MAJOR FINDINGS

Our patient is a 58-year-old right-handed man of French Canadian descent with the syndrome of pure word deafness. One and a half years prior to the study, he had a sudden left-sided sensory loss and mild left hemiparesis that was associated with an inability to understand spoken words or music. There were no aphasic difficulties except for the question of rare paraphasic errors. He learned to lip-read but this ability was dependent on continued auditory input.

Computerized tomography demonstrated that he had a small previously silent, left temporal lobe infarct just lateral to the temporal isthmus, presumably interrupting part of the auditory radiation pathways; and a more recent right temporoparietal infarct, more completely interrupting the temporal isthmus and the auditory radiation pathways.

A sequence of studies was performed to characterize the deficit in auditory processing: (a) pure tone threshold audiometry revealed a mild to moderate deficit in the speech frequency range which could not account for his difficulties; (b) brainstem auditory evoked potentials were normal and consistent with intact brainstem auditory function in response to clicks; (c) dichotic listening studies revealed a strong left ear extinction; (d) auditory evoked potentials revealed little or no elicitable response with left ear click stimulation and a diminished right hemisphere response

with right ear click stimulation; (e) measures of temporal auditory acuity as determined by click fusion and click counting were abnormal.

Linguistic studies with natural speech indicated that he had good recognition of vowels, but greater difficulties with initial stop consonants in CV presentations. In particular, he had greater difficulties with the distinctive feature 'place' as compared to 'voicing'. His performance improved with an increase in the number of distinctive features that differentiated two target stimuli. Synthetic speech studies further confirmed preserved perception of phonemic categories for place and voice but demonstrated difficulties discriminating small differences at the boundaries on a voice onset time (VOT) continuum.

Further neuropsychological testing demonstrated his amusia and difficulty in discriminating the language of a speaker. Identification of broad categories of sounds was relatively intact and only a mild auditory sound agnosia was demonstrated.

## DISCUSSION

We have presented the case of a patient with pure word deafness, amusia and mild auditory sound agnosia. Our studies document a temporal auditory acuity disorder that may bear on difficulty in phonemic discrimination at a prephonemic level. The discussion will consider clinico-anatomical, neurophysiological, neurolinguistic, and neuropsychological aspects of this disorder. These factors will then be considered with respect to a new classification of pure word deafness.

### *I. Clinico-anatomical Considerations*

Kussmaul (1877) believed that pure word deafness resulted from destruction of the left temporal lobe. Lichtheim (1885) later described the syndrome as a disconnection of the pathway from the auditory reception centre to the auditory word impression centre. Bastian (1897) similarly thought of pure word deafness as arising from isolation of the auditory word centre rather than its destruction, and Liepmann (Liepmann, 1898; Liepmann and Storch, 1902) supported this hypothesis on the basis of his case with a unilateral lesion. Geschwind (1965) in his review of the disconnection syndromes noted that most cases have had bilateral lesions, usually cortical-subcortical lesions in the anterior part of T1, leaving Heschl's gyri essentially intact; and that the less common unilateral cases involve subcortical lesions that interrupt auditory radiation fibres as well as callosal fibres from the contralateral auditory cortex. He concluded that the syndrome ultimately represented a disconnection of the auditory cortex from Wernicke's area.

Our case had bilateral lesions. The left temporal lesion was small and previously silent: it was lateral to the temporal isthmus and probably partially involved the auditory radiation fibres. Since this lesion did not enhance on the initial scan, it was considered to predate the onset of pure word deafness and was insufficient to account for the syndrome. The lesion on the right was larger and completely

interrupted the temporal isthmus, and involved primarily the posterior superior temporal lobe and parietal lobe.

Anatomical study demonstrated that in our patient auditory input had no access to right cortical auditory areas and only partial access to left cortical areas. The presence of a previously silent small, but critical, infarct raises the possibility that some clinical reports of patients with presumed unilateral lesions may have had bilateral lesions. Another consideration is that the association of unilateral/bilateral and cortical/subcortical lesions suggests the possibility that more than one type of pure word deafness may exist. Our study is consistent with the disconnection hypothesis (Geschwind, 1965) but offers no definite proof.

## II. *Neurophysiological Assessments—Auditory Processing Deficits*

Previous studies of pure word deafness have included neurophysiological measures: standard pure tone audiometry, measures of central auditory processing, dichotic listening studies and evoked potential studies. These areas were assessed in our patient. We conclude that (1) standard measures of audiological function such as pure tone thresholds or brainstem auditory evoked potentials are usually unaffected in pure word deafness; (2) measures of hemispheric function such as dichotic listening and cortical auditory evoked potentials may vary across patients and reflect the fact that some cases have had primarily unilateral left hemisphere lesions and others may have bilateral lesions with a larger right hemisphere lesion; and (3) the essential neurophysiological deficit for pure word deafness as in our patient is a disorder of temporal auditory acuity.

(a) *Audiometric studies.* After the initial description of pure word deafness, there was some concern that this syndrome was related to a peripheral hearing loss (Freud, 1891; Freund, 1895). Most of the subsequent studies demonstrated normal or near normal pure tone thresholds with the exception of occasional deficits in the high frequency range usually attributed to the patient's age (*see review by* Goldstein, 1974). These observations have some support in auditory cortex ablation studies in animals (Diamond and Neff, 1953; Jerisen and Neff, 1953; Brugge, 1975), and clinical studies in man (Baru and Karasseva, 1972, and others).

Our patient, like other patients with pure word deafness, did not have audiometric deficits that could account for his syndrome. He had only a mild hearing loss which may have been related to noise exposure. There is no evidence from studies of pure word deafness or from animal studies to suggest that a deficit in standard pure tone thresholds plays a major role in pure word deafness.

(b) *Brainstem auditory evoked potentials.* Evoked potentials have been previously reported in two other cases of pure word deafness (Albert and Bear, 1974; Stockard and Rossiter, 1977). Both patients had normal brainstem averaged evoked potentials with intact waves I–VII suggesting preserved function up to the level of the auditory radiations (McCutchen and Iragui-Madoz, 1979). Our patient had similar normal results.

(c) *Dichotic listening studies.* Dichotic listening has been studied in two other patients with pure word deafness (Albert and Bear, 1974; Saffran *et al.*, 1976). Both patients had a strong right ear extinction, suggesting predominant left hemisphere disease. Our patient had a strong left ear extinction. This finding suggests either that pure word deafness may be independent of major left temporal lobe dysfunction or that there may be two somewhat different mechanisms underlying a common clinical presentation.

(d) *Cortical auditory evoked potentials.* Auditory evoked potentials have been performed in several patients with pure word deafness. Michel *et al.* (1980) recently reviewed these data and noted cases with abnormal responses on the right, left or bilaterally. They also referred to another of their cases with normal responses bilaterally. Our patient had responses indicative of predominantly right hemisphere pathology which was consistent with the anatomical and dichotic listening studies. Results of the auditory evoked potentials serve to rule out the possibility of paradoxical responses (Damasio and Damasio, 1979) and cortical deafness (Michel *et al.*, 1980).

The variability in the results from studies with auditory evoked potentials in patients with pure word deafness is consistent with the variability in pathology. These data support the hypothesis that more than one type of pure word deafness exists.

(e) *Temporal auditory acuity measures.* Two previous studies have examined the role of temporal auditory acuity in pure word deafness. Albert and Bear (1974) found abnormalities in click fusion and click counting thresholds in a patient with probable unilateral left hemisphere damage. Chocholle *et al.* (1975) found deficits related to duration discrimination in their patient with bilateral temporal lobe lesions. We demonstrated clear abnormalities in click fusion and click counting and consider this a disorder of temporal auditory acuity. We suspect that this deficit occurs in patients with pure word deafness with either unilateral left or bilateral damage.

Several measures of temporal auditory acuity exist (click fusion, click counting, duration discrimination, etc.). For the purposes of this discussion we have considered all these measures together. It is possible, however, that important distinctions may emerge between these measures and provide yet another means for subclassifying pure word deafness. In particular, Chocholle *et al.* (1975) found impaired perception of brief sounds with relatively unimpaired fusion thresholds.

### III. Linguistic Considerations

(a) *Discrimination.* Our patient demonstrated an impaired pattern of discrimination of linguistic material that we have described as prephonemic. According to our model: (1) a disorder in temporal auditory acuity would cause particular difficulty with rapid formant transitions, leaving vowel discrimination relatively preserved;

(2) increasing the number of target distinctions would improve performance; (3) place of articulation would be the vulnerable feature; and (4) categorical boundaries would be preserved.

The few previous attempts to analyse patterns of phoneme discrimination of natural speech stimuli in pure word deafness are summarized in Table 4. Naeser (1974) and Chocholle *et al.* (1975) both studied patients with bilateral temporal lobe lesions and found a deficit in the discrimination of minimally distinct phonemes with a particular vulnerability for the distinctive feature of place of articulation when compared to the feature of voicing. These results are comparable to the findings in normals when the signal to noise ratio is lowered (Miller and Nicely, 1955). Naeser (1974) also suggested that since her patient had more problems with stop consonants than fricatives or vowels that the selective problems may have actually been for phonemes of brief duration, a finding consistent with other data presented by Chocholle *et al.* (1975).

TABLE 4. STUDIES OF PHONEMIC DISCRIMINATION IN PURE WORD DEAFNESS

Case	Naeser (1974)	Chocholle <i>et al.</i> (1975)	Denes and Semenza (1975)	Saffran <i>et al.</i> (1976)	Our case
Lesion	Bilateral	Bilateral	Left	Left	Bilateral
Dichotic listening	—	—	—	Right ear extinction	Left ear extinction
Temporal auditory acuity disorder	—	+	—	—	+
Selective vulnerability of distinctive features	Place of articulation	Place of articulation	No pattern	Voicing	Place of articulation
Preservation of categorical boundaries	—	—	—	Not present	Present

— = not examined in these particular reports.

There are two other studies with natural speech stimuli available in patients with presumed unilateral left hemisphere pathology (Denes and Semenza, 1975; Saffran *et al.*, 1976). In an abbreviated examination of phonemic discrimination patterns, Denes and Semenza (1975) found a significant deficit but with no particular pattern of error. Saffran *et al.* (1976), on the other hand, found that voicing errors exceeded place errors. They concluded that this pattern of phonemic discrimination could not be explained on the basis of a reduced signal to noise ratio and suggested that this pattern represented the potential ability of the right hemisphere to detect place features.

In summary, there are four previous case studies of phonemic discrimination utilizing natural speech in pure word deaf patients. Two patients with bilateral temporal lobe lesions (Naeser, 1974; Chocholle *et al.*, 1975) demonstrated a particular vulnerability for the feature of place. The other two represented studies of patients with presumed unilateral left disease. Either a reverse pattern was demonstrated with the distinctive feature of voice being most vulnerable (Saffran *et al.*, 1976), or no particular pattern was discerned (Denes and Semenza, 1975). Our data are consonant with the two of pure word deaf cases with bilateral temporal lobe



lesions. The inconsistency with the unilateral cases suggests that their patients do not simply have prephonemic temporal auditory acuity disorders. These patients may indeed represent another type of pure word deafness, perhaps a fragment of Wernicke's aphasia.

Our synthetic speech investigation emphasized the prephonemic nature of our patient's disorder. It also provided another contrast between our patient with bilateral lesions and the patient of Saffran *et al.* (1976) with a unilateral lesion.

#### IV. Other Neuropsychological Considerations

(a) *Amusia*. Our patient also had a profound amusia. Henschen (1926) reviewed 65 published cases of pure word deafness and found that about one-third had an associated deficit in receptive musical ability. Similarly, he collected several cases of patients with musical agnosia but without language deficits. Despite some contradictory evidence, there seems to be agreement that language and musical functions are independently organized in the brain (Hécaen and Albert, 1978). There are several meaningful factors in music. Each of these factors may rely on an interaction between right and left hemisphere functions which is not easily predicted (Brust, 1980). For instance, melody recognition may rely on either spatial or temporal factors (Gordon, 1974; Hécaen and Albert, 1978); rhythm recognition could depend on the recognition of temporal order and duration or on an overall pattern (Gates and Bradshaw, 1977).

We believe that the amusia in our patient represents an interaction of his right and left hemisphere lesions. Impaired temporal auditory acuity may be relevant to amusia, but there are insufficient data to draw any further conclusions.

Our patient was able to discriminate broad categories of sounds: voices, music, environmental sounds; however, he was unable to distinguish the particular language being spoken. In this latter instance, our patient was deprived of much of the semantic information. On the basis of dichotic listening experiments, Blumstein and Cooper (1974) demonstrated a left ear advantage for perception of the intonation contours of speech. They suggested that the right hemisphere is responsible for this latter aspect of speech processing which may be similar to melody recognition. It seems that when deprived of the semantic information our patient was unable to distinguish foreign languages by their different contours of speech because of his large right hemisphere lesion.

(b) *Auditory sound agnosia*. Auditory sound agnosia is a frequent companion of pure word deafness and was present in a mild form in our patient. Auditory sound agnosias have been described as either associative or apperceptive in type. Our case seems to be of an apperceptive type. Vignolo (1969) suggested that with careful testing, such a deficit could be detected in many patients with right hemisphere lesions. It seems reasonable to interpret our patient's deficit as a consequence of a temporal auditory acuity deficit combined with a disorder in perception of melody.

(c) *Influence of contextual cues.* Certain factors tended to improve our patient's performance. For instance, the smaller the set of phonemes to differentiate, the better his performance; once told the language of the speaker, his auditory comprehension improved considerably; he was able to use lip reading which offers considerable information for place of articulation, and so on. Saffran *et al.* (1976) similarly concluded that contextual cues played an important role in the auditory comprehension of pure word deafness. This ability to use contextual cues has been described in other perceptual disorders of cerebral origin (Caplan, 1978) and is not restricted to pure word deafness.

#### SUMMARY

We have demonstrated that the deficit in speech perception in our patient with pure word deafness is secondary to a prephonemic temporal auditory acuity disorder. We delineated the nature of the auditory processing deficit in our patient with bilateral lesions and then demonstrated the presence of a predicted deficit in phonemic discrimination. This pattern is comparable to previous cases with bilateral lesions and distinct from other cases with unilateral lesions. Review of previous reports suggests that there are two distinct types of pure word deafness: type 1, in which the deficit is prephonemic and related to a temporal auditory acuity disorder, and type 2, a form that is independent of a temporal auditory acuity disorder and has a deficit in linguistic discrimination that does not adhere to a prephonemic pattern (Denes and Semenza, 1975; Saffran *et al.*, 1976). The former has been associated with bilateral temporal lobe lesions (Naeser, 1974; Chocholle *et al.*, 1975), the latter with left unilateral lesions (Denes and Semenza, 1975; Saffran *et al.*, 1976). The first form is an apperceptive disorder, whereas the second represents a higher disorder in phonemic discrimination and may be considered a fragment of Wernicke's aphasia.

#### ACKNOWLEDGEMENTS

The authors wish to acknowledge the assistance of Ann Berg who aided in the collection of much of the neurolinguistic and neuropsychological data and Maria Gabriel who aided in the collection of the evoked potential data. Jean M. Pieniadz assisted with quantitative analysis of the CT scan lesion sizes. Don Osterberg first recognized the syndrome in this patient and referred him for further evaluation. Materials for the synthetic speech studies were provided in part by Sheila Blumstein and Dennis Klatt.

T.A. is indebted to Suzanne Corkin for her valuable support and discussion. Harold Goodglass, Sheila Blumstein and Howard Gardner are thanked for their extremely valuable review and critique of the manuscript. Barbara Barresi and Nancy Helm Estabrooks administered the Boston Diagnostic Aphasia Examination. Melody Eisenberg performed the audiogram and assisted in its interpretation.

T.A. was supported by NIH training Grant 5T 32 GMO 7484. This work was supported in part by NIH Grant NINDCDS NS11408.

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(Received January 20, 1981)