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WORD DEAFNESS AND AUDITORY CORTICAL FUNCTION

A CASE HISTORY AND HYPOTHESIS

by PETER PRAAMSTRA,[1] PETER HAGOORT,[2] BEN MAASSEN[1] *and* THOM CRUL[3]

(*From the* [1]*Institute of Neurology, University Hospital Nijmegen, Nijmegen,* [2]*Max-Planck-Institut für Psycholinguistik, Nijmegen, and* [3]*Institute of Otorhinolaryngology, University Hospital Nijmegen, The Netherlands*)

SUMMARY

A patient who already had Wernicke's aphasia due to a left temporal lobe lesion suffered a severe deterioration specifically of auditory language comprehension, subsequent to right temporal lobe infarction. A detailed comparison of his new condition with his language status before the second stroke revealed that the newly acquired deficit was limited to tasks related to auditory input. Further investigations demonstrated a speech perceptual disorder, which we analysed as due to deficits both at the level of general auditory processes and at the level of phonetic analysis. We discuss some arguments related to hemisphere specialization of phonetic processing and to the disconnection explanation of word deafness that support the hypothesis of word deafness being generally caused by mixed deficits.

INTRODUCTION

Pure word deafness, or auditory verbal agnosia, is a rare disorder of auditory language comprehension. It is characterized by a selective impairment of auditory verbal comprehension with preservation of other language functions. However, both the selectivity for verbal material and the preservation of other language functions are matters of dispute (Goldstein, 1974; Buchman *et al.*, 1986). Given the rare occurrence of pure cases, Buchman *et al.* dropped the adjective 'pure' and adopted 'word deafness' to designate 'a disparity between auditory verbal comprehension and other linguistic functions'. Thus defined, it still remains a distinctive syndrome which has drawn the attention of many neurologists and neuropsychologists.

Opinions diverge as to the functional locus of the auditory verbal recognition disorder in word deafness. Agnosias were originally conceived of as central recognition disturbances, although a distinction between apperceptive and associative types was made in its history by Lissauer (1890) and later by others (Geschwind, 1965; Vignolo, 1969; Bauer and Rubens, 1985). In the last two decades attempts to characterize impairments in individual patients have focused on speech perceptual deficits. Most authors have emphasized impairments of low level general auditory processes (Jerger *et al.*, 1969; Albert and Bear, 1974; Chocholle *et al.*, 1975; Auerbach *et al.*, 1982; Miceli, 1982;

Correspondence to: Dr P. Praamstra, Institute of Neurology, University Hospital Nijmegen, PO Box 9101, 6500 HB Nijmegen, The Netherlands.

Coslett *et al.*, 1984; Motomura *et al.*, 1986; Tanaka *et al.*, 1987; Yaqub *et al.*, 1988; Buchtel and Stewart, 1989). In contrast, Saffran *et al.* (1976), Caramazza *et al.* (1983) and Metz-Lutz and Dahl (1984) implicated phonetic processes, that is, a stage of auditory processing specific to speech.

In theories of speech perception, the distinction between phonetic and auditory processes indicates that some aspects of speech perception are specifically linguistic (phonetic processes), while others are not (general auditory processes) (Liberman *et al.*, 1967; Repp, 1982; Kuhl, 1986; Pisoni and Luce, 1987). Clearly, the extraction of phonetic structure from the physical speech signal requires the operation of basic processes that register, for example, intensity, frequency, and duration. But how these general auditory processes relate to specialized phonetic processes is unresolved. It is, for instance, not known what kind of auditory processes are crucial for the perception of speech. What is known, though, is that phonetic analysis is probably a left hemisphere-based competence (Studdert-Kennedy and Shankweiler, 1970; Studdert-Kennedy *et al.*, 1972; Tartter, 1988). Hence left hemisphere lesions, especially of the auditory cortex, might have a disruptive effect on speech perception. This expectation is, however, only partially met. Recent studies have confirmed that phonetic analysis may be deficient in aphasia, but these phonetic impairments are not strongly correlated with auditory language comprehension impairments (for a review, *see* Blumstein, 1987). Also, studies comparing left and right hemisphere-damaged patients have not contributed much to the identification of the auditory processes underlying speech perception (*see*, e.g., Divenyi and Robinson, 1989). Given these unresolved questions it is surprising that so many authors of recent case reports have assumed deficits of general auditory processes as the cause of word deafness (Albert and Bear, 1974; Auerbach *et al.*, 1982; Miceli, 1982; Coslett *et al.*, 1984; Tanaka *et al.*, 1987; Yaqub *et al.*, 1988; Buchtel and Stewart, 1989).

We report a case of word deafness, which was studied using neurolinguistic and speech perception investigations similar to those used by earlier authors. These investigations helped to delimit the nature of the patient's disturbance and to generate hypotheses regarding the contribution of auditory and phonetic deficits. In the case we studied, an unusual circumstance provided an additional source of evidence bearing on the role of auditory versus phonetic deficits. As a Wernicke's aphasic, the patient had already participated in various neurolinguistic experiments when he sustained a second cerebral infarct, which was located temporoparietally in the right hemisphere and made him word deaf. This sequence of events provided the opportunity to repeat earlier tests and to perform new experiments in order to chart in detail the differences between the old and the new patterns of deficits. The newly acquired deficit appeared to be limited to a disorder of speech perception and was difficult to reduce to either an acoustic or a phonetic type of deficit (Auerbach *et al.*, 1982).

CASE DESCRIPTION

The patient is a 57-yr-old right-handed man. He was first admitted in December 1984, when he had suddenly experienced difficulties in speaking. His past medical history included myocardial infarct at age 44, from which he fully recovered. He is university educated and held a position as professor. His native language is Dutch. General physical examination was unremarkable. Neurological examination revealed mild right-sided facial weakness. There was no visual field loss on confrontation. A very slight right hemiparesis and mild right hyperreflexia were noticed. The right plantar response was extensor. There

was no hemi-inattention or apraxia. A cerebral CT scan (fig. 1) demonstrated a left middle cerebral artery infarction. Left carotid angiography was normal. Electrocardiography showed signs of old anteroseptal infarction. Echocardiography revealed no evidence for a cardiac source of emboli.

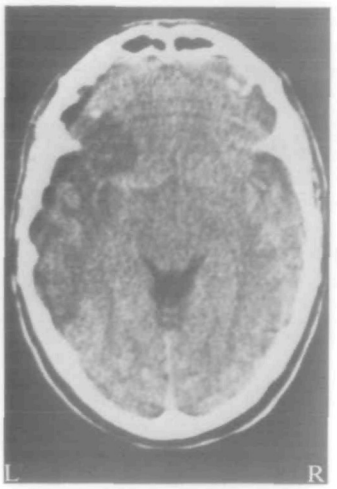

FIG. 1. Unenhanced CT scan performed on admission after first stroke.

The patient had fluent speech, mainly consisting of jargon and empty phrases. Repetition was severely disturbed, although individual sounds could be repeated to some extent. Auditory and written language comprehension were both affected. He was tested formally using the Dutch version of the Aachen Aphasia Test (AAT) (Huber *et al.*, 1984) and classified as a severe Wernicke's aphasic. As such he participated in a number of neurolinguistic experiments. In the course of 16 months he improved to the extent that retesting with the ATT demonstrated a shift, on all subtests, from severely disturbed to a disturbance of average degree.

In January 1988 the patient consulted his neurologist because of a hearing disturbance, which had been preceded by mild complaints of headache and nausea. On admission he reported that sounds seemed louder and noisy. It had occurred to him that he did not recognize the sound of an airplane passing and that music sounded strange. Before the onset of this disturbance he often played the piano, but he had recently stopped. His speech seemed unchanged, but he did not understand any spoken language. The interview with the patient as well as his communication with relatives and nurses proceeded with written messages.

General physical examination revealed normal heart rate, rhythm, and blood pressure. There were no bruits. Neurological examination showed no abnormalities of cranial nerve function, no visual field defect, and normal motor and sensory functions. The tendon reflexes were symmetric, but the left plantar response was extensor.

A CT scan revealed a hypodense area in the distribution of the right middle cerebral artery having the appearance of a fresh ischaemic infarct. Digital subtraction angiography of the extracranial cerebral vessels showed no abnormalities. There was right temporoparietal theta activity in an EEG. Electrocardiography and echocardiography contributed no new information.

A provisional diagnosis of auditory agnosia caused by bilateral destruction of auditory cortical structures due to ischaemic infarcts was made. Investigations over the next one-and-a-half years confirmed this diagnosis and showed the auditory agnosia to be of a predominantly verbal type. The patient experienced no improvement of auditory language comprehension and continued to be dependent on written communication.

INVESTIGATIONS

The following investigations are reported: neuroradiological, audiological, and neurophysiological; language status and nonverbal sound recognition; neurolinguistic and speech perceptual investigations.

Neuroradiological investigation

CT scans of the left and right hemisphere infarcts (fig. 2) were classified by comparing the individual slices with standard sections from an atlas for computerized tomography of the brain (Matsui and Hirano, 1978). Both the left and the right hemisphere lesions were middle cerebral artery infarcts, albeit with considerable differences in size and localization. The left hemisphere lesion involved the superior temporal gyrus and extended superiorly and anteriorly into the parietal lobe and the frontal lobe. The anterior extension overlay the central sulcus and the precentral and postcentral gyri. Posteriorly the lesion involved the supramarginal gyrus of the inferior parietal lobule and extended just onto the angular gyrus. The lesion included both the superior and middle temporal gyri. Involvement of the supratemporal plane with Heschl's gyri could be inferred reliably. The temporal isthmus containing geniculotemporal radiations was spared. The parietal part of the lesion did not extend deep enough to involve the area lateral to the posterior part of the lateral ventricle where the interhemisphere auditory pathways traverse (Rubens *et al.*, 1978; Damasio and Damasio, 1979).

The right hemisphere lesion was largely contained within the temporal lobe. The superior temporal gyrus carried the brunt of the lesion. Like the left hemisphere lesion it extended posteriorly onto the angular gyrus, but it was less well demarcated. Therefore, a massive destruction of the supratemporal plane including Heschl's gyri was uncertain. The geniculotemporal radiation through the temporal isthmus was not affected.

Comment. Given the nature and extent of the left and right hemisphere lesions, the patient's remaining auditory function is probably subserved for the most part by the right hemisphere auditory cortex. Note that this dependence on the right hemisphere already existed before the second stroke.

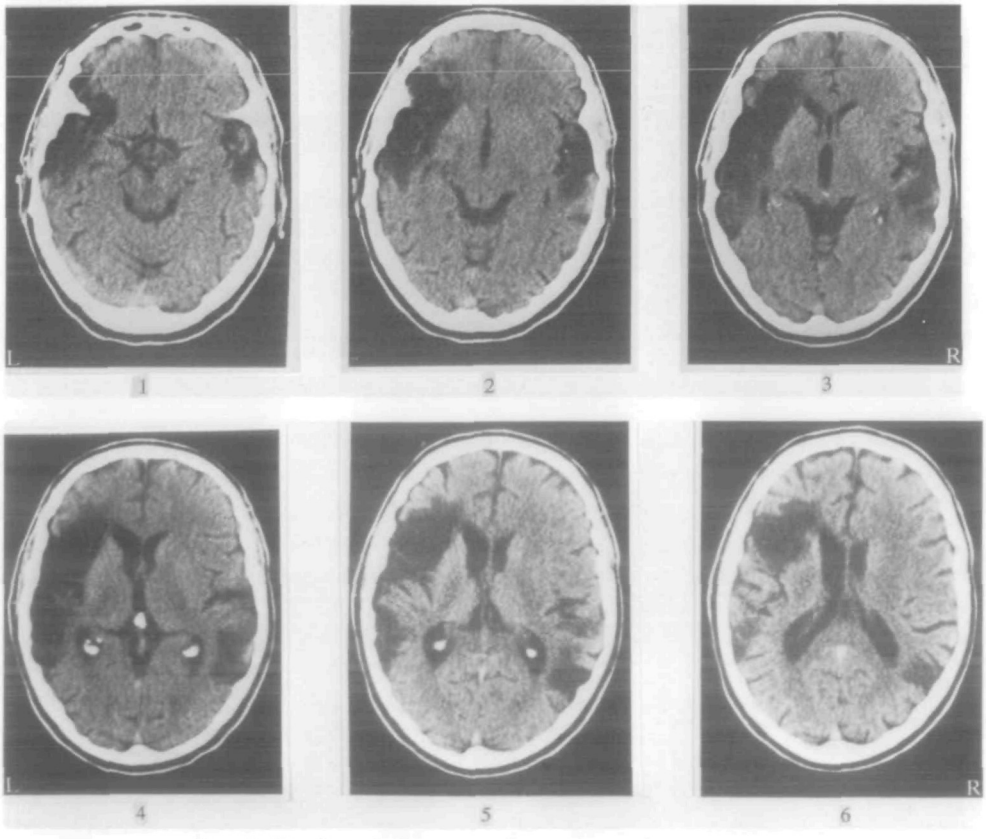

FIG. 2. Unenhanced CT scans performed 2 yrs after second stroke.

Audiological investigation

Standard pure tone audiometry demonstrated a minor bilateral sensorineural hearing loss, which exceeded 20 dB only at frequencies above 4000 Hz (Table 1).

TABLE 1. PURE TONE AUDIOMETRY

	Pure tone threshold (dB)	
Frequency (Hz)	*Left ear*	*Right ear*
250	10	20
500	10	15
1000	5	10
2000	5	10
4000	20	20
8000	40	45

Comment. The patient's mild hearing loss for higher frequencies was obviously not the cause of his auditory comprehension deficit.

Brainstem auditory evoked potentials

BAEPs were recorded on a Nicolet Pathfinder II. Stimuli were 0.1 ms clicks of 80 dB (SPL) presented monaurally at a rate of 11/s. The contralateral ear was masked with white noise. The filter setting allowed a bandpass of 150−3000 Hz. The potentials were recorded ipsilateral to the ear of stimulation from bipolar derivations Cz-A1 and Cz-A2.

Results. Left and right ear stimulation produced waveforms with identifiable peaks I−V, all latencies being within normal limits.

Comment. The results of tone audiometry and BAEPs indicate that the auditory afferent pathways up to the inferior colliculi were functioning normally.

Middle and long-latency auditory evoked potentials

Recordings of MLAEPs and LLAEPs were aimed at an evaluation of midbrain and telencephalic auditory pathways and structures. They were obtained with the same apparatus and stimuli and from the same recording sites as the BAEPs. The stimulation rate was 4.5/s and the bandpass 5−1000 Hz for MLAEPs and 0.25/s and 1−250 Hz for the LLAEPs.

Results. The Na component of the MLAEP was clearly present in the derivations contralateral and ipsilateral to the separately stimulated ears. The latencies were normal. This component was of smaller amplitude with right ear stimulation. The Pa component was also present in both derivations, but of smaller amplitude in the Cz-A1 derivation. Components P1, N1 and P2 of the LLAEP had normal amplitudes and latencies in all recordings. Ipsilateral and contralateral derivations showed nearly identical waveforms. The N2 and later components could not be identified reliably.

Comment. The generator sites of middle and long-latency auditory evoked potentials are still controversial. The study of patients with unilateral or bilateral temporal lesions has contributed little to resolve this issue. Bitemporal lesions have been found compatible with normal or nearly normal MLAEPs (Parving *et al.*, 1980; Woods *et al.*, 1984), while they can also be absent (Özdamar *et al.*, 1982). There is a similar lack of consistency regarding the LLAEPs, which have been reported normal (Parving *et al.*, 1980; Woods *et al.*, 1984; Tanaka *et al.*, 1987), delayed and of smaller amplitude (Albert *et al.*, 1972; Miceli, 1982), absent unilaterally (Auerbach *et al.*, 1982), and absent bilaterally (Michel and Peronnet, 1980) in patients with bitemporal lesions.

In an extensive review of reports of AEPs in patients with temporal lobe lesions, Woods *et al.* (1987) concluded that cortical lesions only affect the Pa generator if they extend beyond the classical auditory

areas. They further observed that the N1 component was usually not abolished with lesions restricted to the superior temporal plane. The authors suggested that this component probably also has generators in the inferior parietal lobe. There is, however, evidence from neuromagnetic recordings (Hari *et al.*, 1980; Sams *et al.*, 1985) and from EP dipole source analysis (Scherg and von Cramon, 1986, 1990) that the exogenous middle and long-latency AEPs are generated in the auditory cortex.

The remarkably preserved AEPs in our case may be due to the presence of remnants of intact auditory cortex. Such remnants can produce normal evoked potentials in experimentally lesioned animals (Diamond and Neff, 1957). In addition, the posterior parts of the inferior parietal lobes, which according to Woods *et al.* (1987) have to be destroyed to annihilate the AEPs, may have contributed to their generation. Alternatively, it may be the case that the scalp-recorded potentials, although symmetric in distribution, are generated in the right hemisphere auditory cortex, which is obviously the least affected side. In summary, only the modest conclusion can be drawn that the presence of middle and late AEPs attests to the integrity of geniculotemporal radiations and at least some auditory cortical structures. Thus the evidence obtained from AEP investigations is compatible with the CT evidence.

LANGUAGE STATUS AND NONVERBAL SOUND RECOGNITION

Aachen Aphasia Test

The patient was tested using the Dutch version of the Aachen Aphasia Test (AAT) (Huber *et al.*, 1980, 1984; Weniger *et al.*, 1981; Willmes *et al.*, 1980, 1983). In addition to eliciting and recording a sample of spontaneous speech, the AAT has subtests for word and sentence repetition, written language, object and situation naming, and word and sentence comprehension. In the subtest on comprehension, patients have to select 1 picture out of 4 on the basis of auditory or visual input. The AAT further includes the shortened version of the Token Test (TT) (De Renzi and Vignolo, 1962; Orgass, 1976). The TT is usually presented auditorily. The TT was also presented visually using a test procedure introduced by Poeck and Hartje (1979). This procedure involves the test materials being screened off from view during the reading of each instruction. After 10 s (parts I–III) or 15 s (parts IV and V) the instruction was covered and the test materials were uncovered.

On several occasions before the second cerebrovascular lesion the patient had shown a stable pattern of performance on the AAT. The patient was tested again 3 months after his second stroke. In Table 2 these results are shown together with the last AAT before the second stroke. The interval between the tests was 23 months. There was an interval of 10 months between the auditory and the visual administrations of the TT after the second stroke.

Comment. The preliminary diagnosis of an auditory agnosia, in addition to the preexisting Wernicke's aphasia, was supported by the AAT results. The qualitative assessment of spontaneous speech, which takes into account semantic, syntactic and phonemic structure, articulation and prosody, automatized speech and communicative behaviour, found no changes compared with his status before the second stroke. Repetition changed dramatically. The performance deteriorated with growing complexity of the presented items (*see* Table 2). On written language the patient also performed worse than before the second stroke. But, as is shown in Table 2, this was almost entirely due to his inability to spell and to write to dictation, both mediated by the auditory modality. The comprehension part of the AAT demonstrated a dissociation between an almost unchanged written language comprehension and a considerably lower score on oral language comprehension. The performance on the auditory sentence comprehension part did not exceed chance level.

The pattern of results on the AAT obtained after the second stroke, in comparison with that before it, shows a selective disturbance of tasks that are dependent on the auditory modality. This modality bias also emerges from a discrepancy between the visual and auditory presentations of the Token Test (Table 3). The results obtained in the visual version show an error score that, according to the interpretation of the standard auditory test, is nonaphasic. Although the TT, like other subtests of the AAT, demonstrates a modality bias, it must be interpreted with caution. The performance on the visual TT was not only much better than the auditory TT results after the second stroke, but it was also superior compared with the auditory TT results before the second stroke. Since the visual TT is not a standardized test we consider the unambiguous change in performance on the comprehension part of the AAT the more reliable evidence of a modality specific change.

TABLE 2. AACHEN APHASIA TEST

	April 1986	*March 1988*	*Max. score*
Repetition	87	30	150
Phonemes	26	18	30
One-syllable words	25	9	30
Foreign words	21	3	30
Composits	6	0	30
Sentences	9	0	30
Written language	44	24	90
Oral reading	25	24	30
Spelling to dictation	8	0	30
Words/sentences to dictation	11	0	30
Naming	85	91	120
Objects (singular nouns)	23	30	30
Colours	29	30	30
Objects (composit nouns)	16	19	30
Situations (descriptions)	17	12	30
Comprehension	91	73	120
Auditory (word)	23	20	30
Auditory (sentence)	24	10	30
Written (word)	25	26	30
Written (sentence)	19	17	30

TABLE 3. TOKEN TEST

	Error score (max. 50)	
Date	*Auditory*	*Visual*
April 1986	45	–
March 1988	42	–
January 1989	–	3

In sum, the AAT showed changes in comparison with the test results before the second stroke that can be attributed to a disturbance in the auditory reception of language. The patient's Wernicke's aphasia was essentially unchanged but compounded by an auditory deficit. Given its apparent central origin, this deficit was characterized as an auditory agnosia.

Nonverbal sound recognition

To test the patient's ability to recognize nonverbal sounds we applied a sound recognition test modelled after Spinnler and Vignolo (1966). It consisted of 20 environmental sounds recorded on tape (e.g., ambulance siren, electric razor, canary, trumpet, etc.). Each sound was presented in conjunction with 4 pictures representing the response alternatives. The correct response alternative pictured the actual source of the sound. The other 3 pictures represented the sources of an acoustically similar sound, an object or event semantically related to the source of the presented sound, or something unrelated. Failures to identify the correct picture were accordingly characterized as discriminative, semantic or odd failures. The mean time of presentation of each sound was 15 (range 8–28) s. Ten healthy control subjects (mean age 56, range 43–65 yrs) made between 1 and 5 incorrect responses (mean 3.2/20; 91% discriminative, and 9% semantic errors).

The original test of Spinnler and Vignolo was designed to investigate the nature of sound recognition deficits in aphasia. It consisted of 10 different sounds and was administered twice, the maximum score being 20. An abnormal score was established to be lower than 16. Spinnler and Vignolo detected sound

recognition deficits mainly in severe Wernicke's aphasia. Semantic errors were far more frequent than discriminative errors. They concluded that sound recognition deficits in aphasia are associated with disorders of auditory verbal comprehension and are not perceptual discriminative, but associative in nature.

Results. Our patient was tested 7 wks after the occurrence of the second stroke. He performed 14/20 correct. Of the incorrect responses 4 were discriminative and 2 odd.

Comment. Our patient's score was slightly below normal performance. As the incorrect responses were mainly discriminative, we conjecture that the disturbed performance was due to the deficit acquired after the second stroke and not due to the pre-existing Wernicke's aphasia which, according to Spinnler and Vignolo, generally causes semantic errors.

NEUROLINGUISTIC INVESTIGATIONS

The specific changes in the AAT test profile demonstrated a clear dissociation between language comprehension in the auditory and visual modalities. In the presence of relatively preserved nonverbal sound recognition, this warranted the diagnosis of auditory verbal agnosia or word deafness. Further investigations were directed at the characterization of the underlying processing deficits.

Although the pure form of word deafness may be a fictitious entity (Buchman *et al.*, 1986), some of the most important studies on word deafness were carried out on relatively pure cases (Saffran *et al.*, 1976; Auerbach *et al.*, 1982) or on cases in which aphasic disturbances were temporary (Albert and Bear, 1974; Miceli, 1982; Tanaka *et al.*, 1987). In our case, however, Wernicke's aphasia already existed before the signs of word deafness developed. Is it also possible to attribute these later signs to the aphasia? The AAT results suggest that such is not the case. We contend that 'central' language mechanisms did not suffer further damage to the second cerebral infarct. This is supported by the results of a lexical decision task performed before and after the second stroke. These results are reported below, together with those of a syllable matching task and a task testing the ability to distinguish between sequences of Dutch and English words.

Lexical decision

Both before and after his second stroke the patient participated in a lexical decision experiment (Hagoort, 1989, 1990). The lexical decision task requires subjects to decide whether strings of letters or sounds are words of their language or not. In the original experiment words and nonwords were presented auditorily. The word targets consisted of Dutch words ranging from 1 to 4 syllables. Nonwords were matched in length and consisted of randomly selected Dutch syllables. The targets were preceded by primes, which were always words. A subset of the primes was semantically related to the word targets, while the remainder was unrelated. For our case, however, the primes are irrelevant. We will focus on the patient's number of correct and incorrect responses to the word and nonword targets. After the patient's second stroke the experiment was repeated. In addition, a visual version of the experiment was administered using the same target words and nonwords presented in written form, without primes.

Method. The targets consisted of 128 Dutch words and 128 nonwords, which were pronounceable according to the rules of the sound system in Dutch. Primes and targets were spoken by a female voice, tape recorded, and presented via headphones. The test session began with 20 starter trials to acquaint the subject with the procedure. For each target the patient had to decide whether it was a word or a nonword by pushing one of two buttons, a 'yes' button for words and a 'no' button for nonwords. For the visual version of the experiment the same targets were presented as a list, written in capitals, and the patient was asked to mark each target as a word or nonword.

Results. Before his second stroke the patient made errors on 4% of the auditorily presented targets. For the word targets, he always gave the correct response. For 7% of the nonwords, he incorrectly decided that they were words. After the second stroke the overall error percentage increased to 49% (30% for the words and 69% for the nonwords). In contrast, when the same targets were presented visually he responded without hesitation and made no errors.

Comment. The results indicate that the second stroke caused a specific deficit in accessing the auditory input lexicon, while access to the orthographic input lexicon was normal.

Distinguishing between languages

To investigate whether he was able to discriminate Dutch from a nonnative language on the basis of the auditory word form information, the patient, who had an active knowledge of English, was subjected to a test of randomly presented sequences of Dutch and English words.

Method. Twenty-four sequences of 15 bisyllabic and trisyllabic words each were presented. Twelve sequences consisted of Dutch words, and 12 of English words. The first sequence, consisting of Dutch words, was not scored. The stimuli were spoken by a female voice, tape recorded, and presented via headphones. After every sequence the tape was stopped by the experimenter, and the patient was asked to point to one of two cards, saying 'Dutch' and 'English'.

Results and comment. The results are presented in Table 4. The overall performance was above chance, the response being accurate in 17 out of 23 word sequences. However, closer inspection revealed that the patient was only accurate in identifying the English sequences (11/12), but performed at chance level for the Dutch ones (6/11). One explanation of this dissociation is that he used a response strategy in which uncertainty was resolved by choosing the response alternative 'English'. Alternatively, the patient might still have been able to extract certain salient phonetic characteristics of English that are not part of Dutch. Whatever the correct explanation for the dissociation between his performance for Dutch and English, the results suggest that he is no longer able to derive adequate phonetic representations from verbal acoustic input.

TABLE 4. ENGLISH-DUTCH LANGUAGE DISCRIMINATION

	Language of sequence		
	English	*Dutch*	*Total*
Response			
English	11	5	16
Dutch	1	6	7
Total	12	11	

Syllable matching

The patient's performance on the auditory lexical decision task established that he is no longer able to access the mental lexicon via the auditory route. This prevents him from recognizing spoken words. In the process of spoken word recognition the listener has to contact stored word forms in the auditory input lexicon. For this purpose, the listener has to extract contact representations from the speech signal (Frauenfelder and Tyler, 1987). These contact representations allow the activation of stored word forms that are consistent with the input. A number of different proposals have been made concerning the nature of these contact representations (e.g., Klatt, 1979; Mehler, 1981; Pisoni and Luce, 1987). Most likely they include information about the consonants and vowels composing a word and about the number of syllables and the stress pattern (Pisoni and Luce, 1987). To investigate whether the patient was still able to extract such a representation from the acoustic input, we applied a syllable matching task (P. Hagoort and A. Witjes, unpublished). This task requires a subject to construct some sort of phonological representation of presented word pairs and match the specified segments.

Method. In this task, pairs of bisyllabic words were presented. The list of word pairs consisted of two blocks, each including 60 target and 40 filler pairs. The first block contained 20 target word pairs with an identical first syllable (e.g., *fortuin-formaat*) (fortune-format) and 40 target pairs with no syllables

overlapping (e.g., *laken-zebra*) (sheet-zebra). In the second block, 20 target word pairs were identical in their second syllable (e.g., *boeket-raket*) (bouquet-rocket). In addition, each block contained 40 filler pairs, 20 with identical first or second syllable and 20 without identical syllables. These pairs were constructed by combining the words from the target pairs in a different way. In this way, response strategies on the basis of the list structure were excluded (Hagoort, 1990). Each block started with 8 practice items. For the first block the patient was required to decide for each pair whether the words had the same first syllable. The pairs in the second block had to be judged for identity of the second syllable. The words were spoken by a female voice and played over closed headphones. There was an interval of 500 ms between the two words of a pair. The patient was given instructions in written form. He was asked to listen to each word pair and to indicate whether or not the words matched with respect to the specified syllable, by pointing to one of two cards saying 'same' and 'different'.

Results. The results for the two blocks were analysed separately. Only the responses on the 60 target items were analysed. To separate the patient's response bias from his sensitivity in this two-alternative forced-choice task, the nonparametric index of sensitivity A′ was computed. This measure is derived from signal detection theory (Green and Swets, 1966; Grier, 1971). The A′ value (e.g., 0.90) can be interpreted as the expected percentage correct (e.g., 90%) on a forced two-choice procedure. For the first block the patient's index of sensitivity (A′) was 0.86 (15/20 hits and 8/40 false alarms). For the second block his A′ was 0.87 (12/20 hits and 2/40 false alarms).

Comment. The patient turned out to be fairly accurate in matching the specified syllables of the auditorily presented word pairs. This implies that he is still able to extract at least a partial phonological representation from verbal acoustic input. Adequate performance in the syllable matching task probably only requires the presence of a very global phonological representation without detailed phonetic specifications (e.g., whether the onset consonant is a /b/ or a /p/). Given his severely impaired performance on the auditory lexical decision task the representation derived from the input, however, seems insufficiently specified to zoom in successfully on the appropriate word forms in the auditory lexicon.

In conclusion, the patient is clearly deficient in the ability to extract from the speech waves phonological representations specified well enough to allow him to recognize the words of his language. A deficit at the acoustic-phonetic level could be responsible for the impairment in constructing a fully specified phonological representation. The subsequent investigations thus focused on the patient's auditory verbal discrimination and identification abilities.

SPEECH PERCEPTUAL INVESTIGATIONS

The perception of speech sounds is usually tested using the 6 stop consonants /b/, /d/, /g/, /p/, /t/ and /k/ paired with a vowel (Saffran *et al.*, 1976; Auerbach *et al.*, 1982; Miceli, 1982; Tanaka *et al.*, 1987). These speech sounds have also been extensively used in dichotic listening research on hemisphere specialization of speech perception (Studdert-Kennedy and Shankweiler, 1970; Studdert-Kennedy *et al.*, 1972). The stop consonants tax the auditory-phonetic analysis in a particular way. Whereas vowels have a long steady state that makes them relatively easy to discriminate, the stop consonants are differentiated from one another by rapid formant transitions within about 50 ms from onset of the speech signal (Lieberman and Blumstein, 1988). This could make the perception of stop consonants particularly vulnerable to impairments of auditory temporal resolution. It is precisely this mechanism by which some authors have sought to connect impairments of general auditory processes to speech perception deficits (Tallal and Newcombe, 1978; Auerbach *et al.*, 1982; Miceli, 1982; Coslett *et al.*, 1984).

The contrasts between the stop consonants can be defined by two phonetic features, as illustrated in Table 5. Normal subjects discriminate more easily between speech sounds distinguished by two phonetic features (e.g., /b/ vs /t/) than between sounds distinguished by one feature (e.g., /b/ vs /d/) (Lieberman and Blumstein, 1988). If the discrimination of speech sounds by a patient demonstrates such a feature effect this might be interpreted as evidence that speech sound discrimination is still supported by phonetic processing capacities (Auerbach *et al.*, 1982).

We performed speech perceptual investigations very similar to those used in earlier case studies (Saffran *et al.*, 1976; Auerbach *et al.*, 1982; Tanaka *et al.*, 1987; Yaqub *et al.*, 1988). The results should provide a measure of the severity of the speech perceptual deficit and might also provide clues to its nature.

TABLE 5. ARTICULATORY FEATURES OF THE STOP CONSONANTS

	Place-of-articulation		
	Front (bilabial)	*Middle (alveolar)*	*Back (velar)*
Voicing			
Voiced	/b/	/d/	/g/
Unvoiced	/p/	/t/	/k/

1. *Discrimination of monosyllabic words*

As a first attempt at characterizing a possible speech perceptual deficit underlying the dissociation between auditory and written language comprehension, an auditory discrimination test was administered (Crul and Peters, 1976). The test consisted of 50 pairs of monosyllabic words, spoken by a female voice and recorded on tape. All words began and ended in consonants or consonant clusters. The words constituting a pair differed either in the word-initial or the word-final consonant (cluster) (27 pairs), or in the vowel (8 pairs). There were 15 pairs with identical words. The patient had to mark the response alternatives 'same' or 'different' on a response sheet.

Results. The test was performed at 3 and at 18 months after the second stroke with scores of 25/50 and 23/50 incorrect responses. Conversion into a nonparametric index of sensitivity gave A′ values of 0.67 and 0.58, respectively. The contingency tables (Table 6) illustrate an especially poor performance on words differing in vowel.

TABLE 6. DISCRIMINATION OF MONOSYLLABIC WORDS—CONTINGENCY TABLES*

	Stimulus			*Stimulus*		
	Different	*Identical*	*Total*	*Different*	*Identical*	*Total*
Responses						
Different	13 (2+11)	3	16	19 (2+17)	7	26
Identical	22 (6+16)	12	34	16 (6+10)	8	24
Total	35	15		35	15	

* The left and right-hand table represent, respectively, the performance at 3 and at 18 months after the second stroke. The numbers in parentheses indicate the responses on word pairs differing in vowel and the responses on pairs differing in consonant, respectively.

2. *Vowel identification*

We tested vowel identification in a similar way to Auerbach *et al.* (1982). The patient was presented with a series of 100 vowels, /a/,/e/, /i/, /o/ and /u/, which were spoken by a male experimenter in random order. The lower half of the experimenter's face was covered to prevent lip-reading. On each trial, the patient marked 1 of the 5 response alternatives on an answer sheet.

Results. /a/, /e/, /i/,/o/ and /u/ were identified correctly, 5, 6, 8, 11 and 13 times out of 20, respectively. The total score of 43% correct identification was above chance (20%).

3. *Vowel identification; binary choice*

The patient was also tested with a forced binary choice procedure. A tape was prepared with 200 tokens of the 5 vowels /a/, /e/, /i/, /o/ and /u/ in random order. The interval between successive stimuli was 4 s. The stimuli were spoken by a female voice and presented through headphones. The length of the vowels ranged between 290 and 323 ms. The patient had an answer sheet with 200 numbered vowel pairs, one of each pair being the presented vowel. On the answer sheet, each vowel was paired with each of the other stimuli 40 times. The correct response alternative was randomly the first or the second member of the pair. The patient answered by marking one of the response alternatives.

Results. The patient's performance on this task is represented in Table 7. The average performance was 72.5% correct, which is above chance (50%).

TABLE 7. CONFUSION MATRIX OF VOWEL IDENTIFICATION—BINARY CHOICE

	Percentage responses				
	/a/	*/e/*	*/i/*	*/o/*	*/u/*
Stimulus					
/a/	87.5	7.5	2.5	2.5	0
/e/	7.5	80.0	7.5	5.0	0
/i/	7.5	12.5	65.0	2.5	12.5
/o/	15.0	7.5	2.5	62.5	12.5
/u/	7.5	5.0	10.0	10.0	67.5

4. *Vowel discrimination*

Vowel discrimination was tested employing the same stimuli as used in the vowel identification task. A tape was prepared with 200 vowel pairs. Between pairs there was an interval of 4 s and between members of a given pair an interval of 1 s. Half of the vowel pairs were identical. The stimuli were presented through headphones. The patient responded by marking 'same' or 'different' on an answer sheet after each presentation of a vowel pair.

Results. Results are presented in Table 8. The patient had a percentage of 92.0% correct responses. An A′ of 0.97 was computed from the overall proportions of hits (89/100) and false alarms (2/100).

TABLE 8. PROPORTION CORRECT DISCRIMINATION OF VOWELS

	/a/	*/e/*	*/i/*	*/o/*	*/u/*
/a/	1.00	1.00	1.00	1.00	1.00
/e/		1.00	0.30	1.00	1.00
/i/			0.95	1.00	0.90
/o/				0.95	0.70
/u/					1.00

5. *Consonant identification; binary choice*

Consonant identification and discrimination were evaluated using the stop consonants /b/, /d/, /g/, /p/, /t/ and /k/, paired with the vowel /a/. For the identification task a tape was prepared of 300 tokens of these 6 CV syllables, spoken by a female voice. There were pauses of 4 s between trials. The test procedure was similar to the one used in the vowel identification task.

Results. Table 9 presents the patient's performance on the consonant identification task. The average performance was 68.7% correct, which is above chance (50%).

TABLE 9. CONFUSION MATRIX OF CONSONANT IDENTIFICATION—BINARY CHOICE

	Percentage responses					
	/ba/	*/da/*	*/ga/*	*/pa/*	*/ta/*	*/ka/*
Stimulus						
/ba/	74	10	0	10	4	2
/da/	10	58	8	4	16	4
/ga/	0	0	82	4	6	8
/pa/	8	12	0	72	6	2
/ta/	4	2	0	4	84	6
/ka/	10	12	8	8	20	42

6. *Discrimination of stop consonants*

The tape for the discrimination task was constructed using the same stimuli as used in the CV identification task. The tape contained 420 pairs of CV syllables, half of which were identical. The 30 possible pairs of different stimuli were presented in random order, each 7 times. The test procedure was the same as the procedure used for the discrimination of vowels.

Results. Results are presented in Table 10. The average performance was 80.0% correct. The A′ computed was 0.87 (proportion hits 170/210, false alarms 45/210).

Comment. In speech perceptual tasks using isolated vowels or CV syllables, the perception of vowels was better than the perception of consonants. The difference in performance between the vowel and the CV discrimination tasks (tasks 4, 6) was significant at the 5% level, using the Mann-Whitney U test ($Z = 2.15$, $P<0.05$; two-tailed). Here, as elsewhere in this section, t tests performed on arcsine transformed scores yielded comparable results. There was a considerably smaller and not significant difference in performance on the corresponding identification tasks (tasks 3, 5) ($Z = 0.32$; $P = 0.75$; two-tailed). While, probably due to their longer duration and steady state, vowels were better discriminated than CV stimuli, the assignment of phonetic labels to vowels was apparently not better than to CV stimuli. If the patient's speech perceptual disorder was entirely due to an acoustic deficit, it would be expected that the advantage of vowels in the discrimination task would be accompanied by a better identification of vowels compared with consonants. Since this was not so, an additional phonetic deficit seems likely.

TABLE 10. PROPORTION CORRECT DISCRIMINATION OF CV SYLLABLES

	/ba/	*/da/*	*/ga/*	*/pa/*	*/ta/*	*/ka/*
/ba/	0.91	0.86	0.93	0.36	1.00	0.93
/da/		0.71	1.00	1.00	0.36	0.79
/ga/			0.77	1.00	0.93	1.00
/pa/				0.71	0.86	0.79
/ta/					0.83	0.36
/ka/						0.77

The assumption that the acoustic properties of isolated vowels (i.e., their longer steady-state portion) accounted for their better discrimination is compatible with the finding that monosyllabic words differing in vowel did not seem to be discriminated at all (task 1). The perception of vowels in consonant contexts

depends partly on the extraction of phonetic attributes of the signal, while isolated vowels can be processed on the basis of their acoustic attributes alone (Liberman *et al.*, 1967). Due to the additional phonetic specification, vowels in consonantal contexts are normally better identified than isolated vowels, in spite of a shorter steady-state portion (Strange *et al.*, 1976, 1979). The breakdown on vowels in a consonant context implies that the patient was highly sensitive to the steady state duration of vowels. But this sensitivity in turn might indicate that he was not able to use the phonetic cues adduced by the context, as in normal subjects duration is not decisive (Dwyer *et al.*, 1982). These results, therefore, cannot easily be reduced either to an acoustic or to a phonetic deficit, but rather suggest an interaction.

In testing consonant perception we used a CV identification task with a forced binary choice procedure similar to the task used by Auerbach *et al.* (1982), Tanaka *et al.* (1987), and Yaqub *et al.* (1988). Auerbach *et al.* and Yaqub *et al.* observed better performance in their patients when the two choice alternatives differed in two features than when they only differed in one. Presumably, to choose one of two response alternatives on hearing 1 CV syllable, a phonological representation is generated and compared with the phonological representations of the syllables that constitute the response alternatives. Feature effects may arise from this comparison (Baker *et al.*, 1981). If our patient's performance demonstrates a feature effect, this might indicate that he can still draw on phonetic processing capacities and is not only dependent on nonlinguistic auditory capacities. However, the difference between the performance on one-feature and on two-feature contrasts (65.0% vs 74.2%) was not significant ($Z = 0.86$, $P = 0.20$; two-tailed). The absence of a significant feature effect also suggests a deficit at the phonetic level.

In contrast to other authors who used the CV identification task with binary choice, we also used a discrimination task. Although in this task the percentage correct responses were significantly higher for stimulus pairs differing in two features than for the one-feature contrasts (95.3% vs 72.0%; $Z = 2.2$, $P < 0.05$; two-tailed), the performance between different one-feature contrasts diverged even more than between one and two-feature contrasts (*see* Table 11). Even an unambiguous feature effect, however, would not necessarily imply that the patient is able to process speech at the phonetic level, since in discrimination tasks this effect may also be attributed to the acoustic differences underlying the phonetic identity of the speech signals.

TABLE 11. SUMMARY OF CV IDENTIFICATION AND DISCRIMINATION (TASKS 5 AND 6)

Nature of phonetic distinction/ no. of contrasting features	*Identification (binary choice)*	*Discrimination*
Voiced/voiceless (1 distinctive feature)	56.7%	57.3%
Place-of-articulation for voiceless stops (1 distinctive feature)	61.7%	67.0%
Place-of-articulation for voiced stops (1 distinctive feature)	76.7%	93.0%
Voiced/voiceless and place-of-articulation (2 distinctive features)	74.2%	95.3%

A note is in order on the patient's poor discrimination of monosyllabic words and his fairly good detection of identical syllables in bisyllabic words. Most likely, this difference is explained by the fact that each of the monosyllabic word pairs differed in a single feature or sound contrast specially selected to detect difficulties in auditory discrimination (Crul and Peters, 1976). In the syllable matching task the words were not selected to have minimal sound contrast, but only such that they had (or had not) an identical first or second syllable. The difference in performance indicates that phonological representations can be

constructed that include a syllabic breakdown of words, but that lack sufficiently distinctive phonetic specifications.

7. *CVC syllable identification and discrimination; synthetic speech*

In addition to the previous tasks using natural speech, the patient was investigated using synthetic speech. Identification and discrimination tests using synthetic speech stimuli have been developed to systematically investigate auditory and phonetic processes in speech perception (Liberman *et al.*, 1967). Similar tests have been used to investigate speech perception deficits in aphasia (Basso *et al.*, 1977; Blumstein *et al.*, 1977*b*, 1984; Yeni-Komshian and Lafontaine, 1983). The synthetic nature of the stimuli allows for control of the physical/acoustical dimensions that underlie the characterization of speech signals in terms of phonetic features. Whereas the phonetic feature voice, for example, distinguishes between /b/ (voiced) and /p/ (unvoiced) in a dichotomous way, the acoustical property voice-onset-time (VOT) underlying the distinction can be varied continuously. This principle is put to use in identification and discrimination tasks on VOT and place of articulation continua. Such tests consist of stimuli that span the acoustical distance between, for instance, /b/ and /p/ (VOT continuum) or /b/ and /d/ (place-of-articulation continuum) in a stepwise fashion. These tests have demonstrated that there is a remarkably well-defined boundary between stimuli on such a continuum labelled as one or the other speech signal by normal subjects. Normal subjects only discriminate accurately between stimuli that they can assign to different phonetic categories. Two stimuli from the same phonetic category but separated by a certain distance on the continuum are not discriminated at better than chance level, while 2 stimuli separated by the same distance and from different categories are. This phenomenon of categorical perception is central to the issue of whether or not the perception of speech requires special mechanisms (Repp, 1984; Kuhl, 1986). Our patient was submitted to synthetic speech tests for one VOT continuum (/b/-/p/) and two place-of-articulation continua (/p/-/t/ and /b/-/d/).

Materials and procedure. Starting from a naturally spoken Dutch CVC word /bak/, 4 meaningful Dutch CVC words were constructed. The procedure involved low-pass filtering (cut-off frequency 4 kHz, slope 24 dB/octave), digitization (10 kHz sampling rate), and storage in computer memory. Subsequently formants were extracted with LPC-analysis and root-solving procedure using the ILS programming package (ILS, 1978). Next, the signal was resynthesized, applying a pitch synchronous energy correction (Maassen and Povel, 1985). Because plosives and vocal murmur deteriorate after resynthesis from formants, /ak/ was resynthesized from reflection coefficients, and the /b/ plus vocal murmur of the original spoken /bak/ was spliced in initial position in the resynthesized /bak/. Total stimulus duration was 320 ms.

The VOT continuum (/bak/-/pak/) was constructed by changing VOT in 9 steps from −80 ms (i.e., 80 ms vocal murmur preceding the plosive) to +12 ms (i.e., no vocal murmur and a silent gap of 12 ms between plosive and vowel onset). Stimulus 7 had VOT = 0 ms. It should be noted that in Dutch the phoneme boundary between voiced and voiceless plosive cognates is different from that in English. The value around −20 ms that we found in normal subjects (*see* fig. 3) is consistent with previous reports (Lisker and Abramson, 1964; Slis and Cohen, 1969).

Two place-of-articulation continua (/bak/-/dak/ and /pak/-/tak/) were constructed by manipulating the second (F2) and third (F3) formant transitions. F2 started at 1000 Hz for the bilabial and at 2000 Hz for the alveolar plosive, and ended at 1200 Hz in the /a/. F3 transition starting values were 2500 Hz and 3100 Hz, respectively, and ending value 2000 Hz. By linear interpolation of F2 and F3,9 steps on the place continua were constructed. Transition duration was 40 ms for both F2 and F3. F1 increased from 200 Hz to 750 Hz in a much shorter transition duration (14 ms) in order to make the plosive character of the initial consonant more prominent, and to emphasize the voiceless cognate. The analysed signal was subsequently resynthesized as described above.

Identification tapes consisted of 90 stimuli for each continuum. Each stimulus was presented 10 times. The interval between successive stimuli was 3.3 s. Discrimination tapes contained 78 pairs, 42 of which differed by two steps on the continuum, and 36 that differed by 3 steps. In the /bak/-/pak/ and /pak/-/tak/ continua the intervals between pairs were 4.0 s and intervals between stimuli within pairs 2.3 s. In the second place-of-articulation continuum (/bak/-/dak/) the interstimulus interval within pairs was 960 ms.

The patient was tested in a quiet room on three different occasions. The stimuli were presented through headphones. In discrimination tasks, the patient responded by marking on an answer sheet the response alternative 'same' or 'different'. In the identification tasks he had to mark the words constituting the response alternatives.

Results. The results are shown in fig. 3. The discrimination conditions shown are those with 3 steps between pair members. Except possibly for the /pak/-/tak/ continuum identification performance seems random. Discrimination performance is relatively preserved in the VOT continuum.

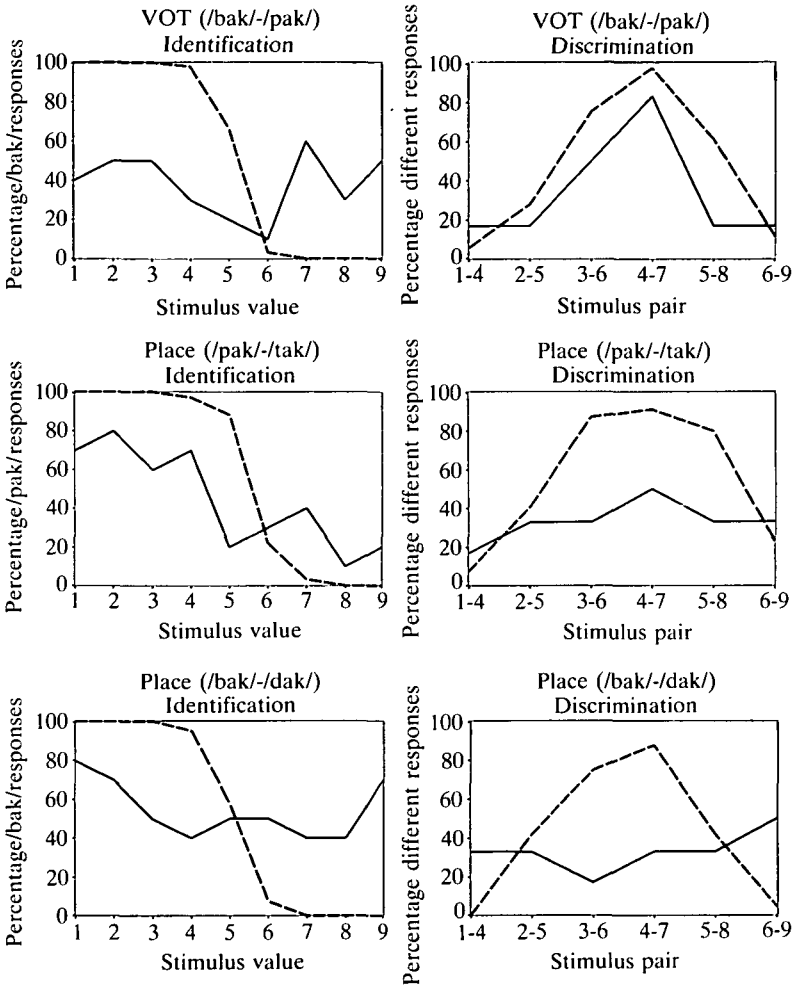

FIG. 3. Performance in identification and discrimination tasks on three different speech continua. Solid lines represent the patient's performance, dashed lines the averaged performance of 10 normal subjects.

Comment. From the severe impairment of both discrimination and identification performance, we inferred that there is probably a phonetic as well as an auditory component to the patient's speech perceptual disturbance. It could be argued that the impaired identification is fully determined by an inability to discriminate the stimuli, which might be attributed to an auditory deficit. This is not an adequate account, however, because discrimination on the VOT continuum was relatively preserved, while identification was random. The contrasting hypothesis that the pattern of results is due to a phonetic deficit only is equally unlikely. This possibility would be supported if the disturbance in identification performance had been

accompanied by an intact discrimination (Yeni-Komshian and Lafontaine, 1983). Our patient's overall discrimination performance, however, was evidently impaired, which rules out this possibility.

The selectively preserved discrimination on the VOT continuum might be due to the perception of voicing differences being more readily mediated by general auditory mechanisms, as the feature voice is more closely tied to acoustically invariant properties than place-of-articulation (for critical discussion, *see* Summerfield, 1982). This explanation implies a deficit at the phonetic level. However, while in the synthetic speech tasks the voicing distinction was better perceived than the place contrast, the reverse was true using natural speech. Although differences between results obtained with natural speech and results obtained with synthetic speech are not uncommon in patients (Miceli *et al.*, 1982; Caramazza *et al.*, 1983), the discrepancy in our results cautions us not to overstate this argument (*see also* Addendum).

The speech perceptual investigations may be summarized by the conclusion that the patient's level of performance was comparable with that found in other cases of word deafness attributed to a speech perceptual deficit (Saffran *et al.*, 1976; Miceli, 1982; Auerbach *et al.*, 1982; Yaqub *et al.*, 1988). As to the pattern of performance, several findings argued against a reduction of the speech perceptual disorder to an auditory processing deficit only. (1) Discrimination of vowels was superior to the discrimination of consonants, while identification of vowels and consonants was not significantly different, though more severely impaired. This dissociation between discrimination and identification performance argues for a phonetic deficit. (2) The better discrimination of isolated vowels compared with consonants was not mirrored in the discrimination of monosyllabic words differing either in (medial) vowel or in a consonant. Since vowels in a consonantal context have a shorter steady-state portion, this might be explained in terms of an auditory deficit. But in view of evidence that consonantal context provides additional phonetic specification that aids vowel recognition, a phonetic deficit has to be assumed to explain the apparent inability to use these cues and, thereby, the strong dependence on steady-state duration of vowels. (3) The absence of a significant feature effect in CV identification with binary choice and the lack of an unambiguous feature effect in CV discrimination implies that recognition of these sounds is mainly dependent on nonlinguistic auditory capacities. (4) In the presence of a relatively preserved discrimination on the VOT continuum, the otherwise severe disturbance of identification and discrimination on synthetic speech continua indicates that both auditory and phonetic levels of speech perception are affected.

Taken together, these results point to a combination of auditory and phonetic impairments. The strength of this conclusion, however, is limited by the fact that auditory and phonetic deficits cannot be assessed independently given that the auditory processes essential to speech perception are largely unknown. Also, the results do not allow any firm statement with respect to the relative contributions of the two types of impairment.

SUMMARY OF MAJOR FINDINGS

The patient we studied suffered a left hemisphere stroke and acquired a Wernicke aphasia at the age of 54 yrs. Three years later, a right hemisphere stroke caused severe deterioration of auditory language comprehension while the patient's language expression remained unchanged. The recognition of environmental sounds was only mildly impaired.

The left hemisphere infarct was located temporoparietally and involved most of the superior temporal gyrus. It probably left the geniculotemporal radiation intact. The right hemisphere infarct was smaller. It also involved the superior temporal gyrus and extended posteriorly, like the left hemisphere infarct, onto the angular gyrus. The auditory radiation through the temporal isthmus was spared.

Pure tone audiometry and BAEPs established intact auditory afference up to the midbrain. Middle and long-latency auditory evoked potentials could be elicited both with left and right ear stimulation. A slight asymmetry in the Pa component was consistent with CT evidence of greater destruction of the left hemisphere auditory cortex. The P1, N1 and P2 deflections were essentially normal, suggesting that the auditory cortices might contain some intact remnants.

Comparison of patient's performance on the Aachen Aphasia Test before and after his second stroke confirmed that only auditory comprehension had suffered, while performance on tasks not dependent on the auditory input channel was unchanged. The performance on an auditory lexical decision task was nearly normal prior to the second stroke, but random after it. When the same targets were presented visually,

he made no errors. Furthermore, the patient could not distinguish auditorily presented Dutch words from English words very well. On a syllable matching task, however, he showed a fairly good performance. This suggested that he was still able to extract some sort of phonological representation from the acoustic-phonetic input, including a syllabic breakdown of the words.

Further investigations focused on speech perceptual abilities, revealing an impairment of phoneme identification and discrimination using monosyllabic words, vowels and CV syllables. The voicing contrast was perceived less well than the place-of-articulation contrast. The performance on synthetic speech tasks using place-of-articulation and voice-onset-time continua was also severely disturbed. Only discrimination of stimuli on the VOT continuum showed a preserved phoneme category boundary. The overall pattern of results provided arguments for a speech perceptual disturbance to which both general auditory and phonetic deficits seem likely to contribute.

GENERAL DISCUSSION

In many cases of pure word deafness that have been reported, the syndrome was accompanied by aphasic disturbances (Goldstein, 1974; Buchman *et al.*, 1986). Most case reports, however, do not explicitly take issue with the presence of aphasic symptoms and treat them as merely contaminating the word deafness. The possibility of an underlying deficit that contributes both to the aphasic disturbance and to the word deafness in these patients is rarely considered (*but see* Caramazza *et al.*, 1983; Franklin, 1989). The patient we studied is a rare instance of the coexistence of aphasia with word deafness, in that the aphasia already existed and was documented at the time the patient acquired word deafness. The word deafness could, therefore, clearly be distinguished from the aphasia. This does not preclude a deficit related to the aphasia also being relevant to the word deafness. Surely the word deafness of our patient would not have occurred if the right hemisphere stroke had not been preceded by one in the left hemisphere.

The nature of the left hemisphere's contribution to word deafness will be our main focus in this discussion. In the analysis of our case, we chose to distinguish auditory and phonetic stages of speech perception, since earlier case reports claimed a specific deficit at either one or the other level (Saffran *et al.*, 1976; Auerbach *et al.*, 1982; Yaqub *et al.*, 1988). While we suggested a mixed deficit, the arguments we provided are not incontrovertible, given the fact that there is no generally accepted account of how auditory and phonetic processes are related, let alone a way to assess auditory and phonetic deficits independently. However, as we will discuss below, the left hemisphere specialization of phonetic processing capacities and the explanation of word deafness as a disconnection syndrome favour a mixed deficit rather than a pure auditory or phonetic deficit. We further discuss how this ties in with present knowledge on speech perceptual deficits in aphasia.

Auditory vs speech modes of perception

The distinction between auditory and phonetic levels or modes of perception originates partially in research on categorical perception (Liberman *et al.*, 1967). Recent developments in categorical perception research, such as the use of animal models and the demonstration that nonspeech sounds can be categorically perceived, have changed the field, but not eliminated the distinction (Repp, 1984; Kuhl, 1986). Of particular interest to the present discussion is the support for the distinction from studies on

hemispheric specialization using dichotic presentation of speech stimuli (for a review, *see* Tartter, 1988). Ear asymmetries obtained in dichotic studies have been interpreted as due to hemisphere asymmetries in processing capacities. Since stop consonants display a right ear advantage not displayed by vowels, it has been suggested that consonants require a speech mode of perception, which is left hemisphere based, while vowels can be processed in both hemispheres by general auditory mechanisms (Studdert-Kennedy and Shankweiler, 1970; Studdert-Kennedy *et al.*, 1972). Evidence from patients with hemisphere lesions and evidence from split-brain research also indicate that phonetic processing capacities are, indeed, unique to the left hemisphere (Oscar-Berman *et al.*, 1975; Zaidel, 1976; Levy and Trevarthen, 1977). Finally, animal data suggesting a left hemisphere specialization for species-specific calls provide circumstantial evidence for the engagement of left hemisphere specialized processes in speech perception (Petersen *et al.*, 1978; Miller and Jusczyk, 1989).

The distinction between auditory and speech modes of perception has been invoked by several authors to explain the dissociation between verbal and nonverbal auditory agnosia (Albert *et al.*, 1972; Albert and Bear, 1974; Lambert *et al.*, 1989). Although most patients that have been reported displayed difficulties in both verbal and nonverbal sound recognition (Gazzaniga *et al.*, 1973; Chocholle *et al.*, 1975; von Stockert, 1982; Miceli, 1982; Buchtel and Stewart, 1989), the occurrence of relatively pure cases does support a double dissociation (Lambert *et al.*, 1989). The distinction between auditory and speech modes of perception might indeed be relevant to the explanation of this dissociation. However, the way it was applied so far merely restated the observation that the processing of speech and nonspeech sounds diverge at some level, and did not elucidate the functional loci of verbal and nonverbal auditory agnosia.

The distinction between auditory and speech modes of perception has also been invoked to account for different patterns of deficits in word deafness. Auerbach *et al.* (1982) argued that the speech perceptual deficit encountered in word deafness must be considered prephonetic if it can be attributed to a disorder in acoustic processing. He specified this acoustic deficit as a disorder of temporal auditory acuity, to be demonstrated by psychoacoustic measures like click-counting and click-fusion. While this deficit is localized at a prelinguistic level, it has its major manifestation at a linguistic level, as the auditory temporal resolution suffices for the perception of nonverbal sounds but is insufficient to resolve the rapid acoustic transients characteristic of certain speech sounds. Auerbach *et al.* formulated a number of criteria that purportedly distinguish patients with a deficit at the acoustic (prelinguistic) level from patients with a deficit at the phonetic (linguistic) level. While they described a case of the former type, a well-documented case that fits the latter type has been reported by Saffran *et al.* (1976).

The model proposed by Auerbach *et al.* (1982) connects the distinction between phonetic and acoustic deficits in word deafness with the lesion type being unilateral or bilateral. It associates the acoustic type of deficit with bitemporal lesions ('type I word deafness') and the phonetic deficit with unilateral left hemisphere lesions ('type II word deafness'). Thus the model offers an explanatory scheme that relates deficits at different stages of processing to distinct lesion patterns. We want to argue in the next section, however, that the distinction between acoustic and phonetic deficits in word deafness must be dissociated from the distinction between unilateral and bilateral cases.

The explanation of pure word deafness as a disconnection syndrome

Pure word deafness has long been recognized as a sequel either to a left hemisphere temporal lobe lesion or to bilateral temporal lobe lesions (Schuster and Taterka, 1926; Geschwind, 1965). The explanation of pure word deafness as a disconnection syndrome (Geschwind, 1965) unifies unilateral and bilateral cases in that it assumes that in unilateral cases the lesion extends deep subcortically and involves the interhemispheric auditory pathways. This explanation is strengthened by evidence from dichotic listening studies which elucidated the functional significance of interhemispheric auditory connections. Sparks and Geschwind (1968) established that sectioning the corpus callosum results in an almost complete suppression of left ear dichotic verbal information. This finding confirmed that left ear dichotic verbal information, arriving in the right hemisphere auditory cortex, has to be transferred to the left hemisphere by commissural auditory fibres in order to be processed as language.

The disconnection explanation of pure word deafness also derives support from neuroradiological studies (Rubens *et al.*, 1978; Damasio and Damasio, 1979), which demonstrated that patients with left hemisphere lesions and a left ear extinction in verbal dichotic listening tasks have a consistent pattern of lesions extending deep to the wall of the lateral ventricle. It can be concluded that unilateral left hemisphere lesions may indeed cause damage to the interhemispheric auditory pathways and deprive the left hemisphere of auditory input via this pathway. According to Geschwind's disconnection theory, a lesion of this type may have the same effect on language function as bilateral temporal lobe lesions.

Most of the case reports published in the last decade, including Auerbach *et al.* (1982), subscribe to a disconnection type of explanation of pure word deafness. But whereas the disconnection hypothesis unifies unilateral and bilateral cases in a single explanation, Auerbach *et al.* chose to subdivide the syndrome again into two distinct types. It is unclear how Auerbach *et al.* intended their adherence to the disconnection hypothesis to be reconciled with their subdivision of pure word deafness into different types. Since the disconnection hypothesis implicates both auditory cortices in the causation of word deafness (either by direct lesion involvement or by a disconnection), a classification of word deafness into auditory and phonetic types cannot be based on the differential specialization of left and right auditory cortex. This does not imply that unilateral and bilteral cases must be identical in all respects. Whereas only bilateral cases seem to go through an initial episode of cortical deafness (Mendez and Geehan, 1988), unilateral cases may be more frequently accompanied by aphasic symptoms, purely contingent on the considerable extension of a lesion which, according to the disconnection hypothesis, must involve the interhemispheric auditory connections and the auditory cortex at the same time.

Mixed deficits in word deafness

In our view, a classification of word deafness as acoustically or phonetically based bears no relation to the lesion being unilateral or bilateral. In addition, the dichotomous classification itself may be criticized on several grounds.

First, the acoustic deficit documented in bilateral cases might also be present in patients with unilateral lesions. We know of only one unilateral case in which the presence of acoustic deficits was investigated (Albert and Bear, 1974). This patient evidenced an

impairment of auditory temporal resolution only slightly milder than that displayed by the bilateral case of Auerbach *et al.* (1982).

A second reason for questioning the proposed classification of word deafness is the improbability of an acoustic deficit as the only cause of the syndrome in all bilateral cases. A recent case report carefully documented a severe psychoacoustic deficit in a patient with bilateral lesions (Tanaka *et al.*, 1987). We suspect that even the severe deficit of this case only caused word deafness because it occurred together with a higher level deficit. This patient had gradually recovered from Wernicke's aphasia due to a left temporal lobe lesion when she experienced a right hemisphere stroke that caused word deafness. The authors documented deficits in temporal resolution (Albert and Bear, 1974) and in temporal sequencing (Efron, 1963; Swisher and Hirsh, 1972). However, these deficits were not limited to the auditory modality, but also encompassed the visual and somatosensory modalities. A supramodal temporal sequencing disorder seems to be associated with left hemisphere lesions that cause aphasia, though not with left hemisphere lesions as such (Efron, 1963). The presence of this supramodal deficit suggests persistent effects due to the left hemisphere lesion, in spite of the patient's reported recovery from aphasia. Furthermore, for an acoustic deficit one would predict vowel perception to be better than consonant perception, consistent with the criteria formulated by Auerbach *et al.* (1982). The patient's performance was indeed slightly better on vowels, but at the same time the recognition of nonverbal sounds was only mildly disturbed. We would have expected that an auditory processing disorder that severely disrupts vowel perception would also strongly affect the recognition of environmental sounds. In sum, these features of the case reported by Tanaka *et al.* (1987) provide reasons to express doubts on an auditory processing deficit as the only cause of this patient's word deafness.

The main thrust of this discussion is that most patients are not sufficiently documented to allow a classification according to the dichotomous scheme proposed by Auerbach *et al.* (1982). Moreover, evidence can be gathered from recent literature amounting to counter-examples to the proposed explanatory scheme. This prepares the ground for an alternative hypothesis that implies the involvement of auditory as well as phonetic processes in the causation of word deafness. The findings in our own case, indicating a mixed deficit rather than an isolated acoustic or phonetic deficit, are consistent with the proposed hypothesis. The way word deafness evolved from Wernicke's aphasia in our patient may further elucidate the hypothesis.

A remarkable feature of our patient is the fact that the change in his language status, caused by the right hemisphere infarct, was limited to the auditory input modality, as demonstrated by the AAT results before and after the second vascular accident. The second stroke did not even cause temporary changes outside the domain of the auditory recognition disturbance. This indicates that the patient's residual language capacities after his first stroke were still subserved by the damaged left hemisphere, which was only dependent on the right hemisphere for transfer of auditory information. Presumably the severe involvement of the left hemisphere auditory structures compromised the left hemisphere specialized processes that support phonetic analysis. This did not result in specific speech perceptual difficulties as long as general auditory processes subserved by the right hemisphere functioned adequately, given that intact interhemispheric connections allowed an interplay between the two hemispheres. The subsequent

involvement of the right hemisphere auditory cortex, then, sufficiently degraded the auditory input to linguistic processing stages that a specific speech perceptual deficit, that is, word deafness, resulted. We thus suppose that, before the second stroke, the patient was already partially dependent on nonlinguistic auditory capacities to compensate for deficient phonetic processing.

The interpretation we propose of our case might apply more generally. We suggest that word deafness is caused by deficits both of phonetic processes and of nonlinguistic general auditory capacities. This hypothesis strongly relies, first, on the disconnection hypothesis as an account of the underlying neurological events, and, secondly, on the left hemisphere's specialization for phonetic analysis. These two elements do not in all respects fit together, however. Whereas the disconnection hypothesis makes word deafness conditional on bilateral lesions or a disconnection, it is unclear why, given the existence of specialized phonetic processes for speech perception, a selective disturbance of these processes by a left hemisphere lesion does not suffice to produce severe speech perceptual problems. Evidence relevant to this issue can be obtained from the study of speech perceptual deficits in aphasia.

Speech perceptual deficits in aphasia

In spite of the evidence we have already cited in favour of left hemisphere specialized capacities for phonetic analysis, the disconnection explanation of word deafness could make one more inclined to take both hemispheres for equipotential in this respect, or even doubt the existence of special mechanisms for phonetic analysis. The question to be dealt with in this section is therefore whether studies on speech perceptual deficits in aphasia support left hemisphere specialized phonetic processes.

One study by Oscar-Berman *et al.* (1975) explicitly addressed phonetic processing in left and right hemisphere-damaged patients. It was found that left hemisphere patients, in contrast to right hemisphere patients, were impaired in the use of a phonetic code in identification tasks using dichotically presented CV syllables. These results support a distinction between auditory and phonetic levels of speech processing and strongly suggest that it is the left hemisphere that is specialized for the conversion of auditory parameters of speech into phonetic attributes.

The majority of studies pertinent to the issue have focused on the question to what extent speech perceptual deficits contribute to auditory language comprehension impairments in aphasia. These studies have in large part been motivated by the claim by Luria (1970) that the comprehension deficit of Wernicke's aphasia should be attributed to a specific impairment of phonetic analysis. A number of studies using phoneme identification and discrimination tasks have found perceptual difficulties but have failed to show a consistent relation between speech perception and auditory language comprehension (Basso *et al.*, 1977; Jauhiainen and Nuutila, 1977; Blumstein *et al.*, 1977*a*, *b*, 1984). Other studies found only a weak relation (Varney and Benton, 1979; Varney, 1984; Miceli *et al.*, 1980; Yeni-Komshian and Lafontaine, 1983; Riedel and Studdert-Kennedy, 1985). It thus appears that impairments in phonetic perception that can be demonstrated in aphasia are not a major factor in aphasic disturbances of auditory language comprehension. However, the results do not refute the original assumption that there are specialized left hemisphere mechanisms for phonetic analysis. This is supported, first, by the fact that right hemisphere-damaged subjects evidenced no deficit

in phoneme perception (Goldblum and Albert, 1972; Basso *et al.*, 1977; Blumstein *et al.*, 1977*b*; Varney and Benton, 1979). In addition, a number of studies consistently found Wernicke's aphasics worse than other aphasia types on phoneme perception tasks (Goldblum and Albert, 1972; Blumstein *et al.*, 1977*b*; Baker *et al.*, 1981). This might be related to temporal lobe damage and concurrent involvement of auditory cortical structures that presumably underlie phonetic processing capacities. Finally, in some studies that explored categorical perception in aphasia, phoneme discrimination was better preserved than identification performance (Blumstein *et al.*, 1977*b*; Riedel and Studdert-Kennedy, 1985), that is, a dissociation that argues for an impairment of phonetic perception (Yeni-Komshian and Lafontaine, 1983; Riedel and Studdert-Kennedy, 1985). In short, these studies on speech perception and auditory language comprehension in aphasia are compatible with the existence of a specialized phonetic competence subserved by the left hemisphere.

The puzzling fact that remains to be explained, then, is why a disturbance of phonetic perception by a left hemisphere lesion contributes so little to impairments of auditory language comprehension in aphasia. Part of the answer is provided by work conducted by Blumstein and coworkers. Whereas most investigations referred to in the last paragraph set out to isolate speech perceptual deficits from higher level language impairments, Baker *et al.* (1981) and Milberg *et al.* (1988) also addressed the question of whether speech perceptual deficits interacted with semantic impairments and the process of lexical access. These studies suggested an intimate interaction between phonological and semantic factors in auditory word processing. More specifically, the first study indicated that perceptual deficits may become manifest with increased semantic demands, even if they are not evident in perceptual tasks. Thus there might be a small but more pervasive influence of speech perceptual deficits on auditory language comprehension in aphasia than earlier investigations suggested.

Within a modular framework, Blumstein (1987) summarized the investigations referred to above by stating that the speech perception system, that is, the phonetic/phonological processor, is not functionally autonomous since it can be affected by higher level language impairments. Our conjecture that word deafness might be due to an interaction of auditory and phonetic deficits emphasizes the obvious possibility that it can also be affected by lower level auditory impairments. This proposal is in some sense complementary to Blumstein's view. The relatively selective disturbance of speech perception in word deafness seems difficult to account for in purely auditory terms, given that neither auditory temporal resolution, nor any other auditory process can be singled out as crucial to intact speech perception. The evidence from aphasia on the other hand makes clear that, while left hemisphere lesions can cause speech perceptual deficits, these deficits alone do not account for major impairments of auditory language comprehension. Therefore, the hypothesis of a mixed deficit in word deafness might be in better agreement with our present knowledge of auditory cortical function than the hypothesis of isolated acoustic or phonetic deficits.

CONCLUSIONS

The present conception of word deafness is far removed from the original conception of the agnosias as central disturbances of recognition (Bauer and Rubens, 1985). Like

many patients reported in recent decades, our patient had a speech perceptual disturbance of sufficient severity to explain the word deafness. The documentation on his language status before and after his second stroke rules out that changes occurred other than those related to the disturbance of the auditory input channel. The very discrete change in the language status, occasioned by the right temporal lobe infarction, strongly suggests that, apart from auditory input via the right hemisphere, there had been no compensation of any importance of left hemisphere language functions by the right hemisphere, since in this case a more general change should be expected.

The nature of the speech perception impairment in our patient does not fit in well with the hypothesis of Auerbach *et al.* (1982), stating that speech perceptual disturbances in word deafness can be classified as due to either a prephonetic, acoustic deficit or a deficit at the linguistic level of phonetic perception. In contrast to the proposal by Auerbach *et al.*, we conjecture that word deafness is due to an interaction between an impairment of general auditory processes and a deficit at the level of phonetic analysis. The presence of deficits at both these levels is coherent with the left hemisphere specialization of phonetic processing capacities and with the necessary involvement of both auditory cortices (by direct lesion involvement or by a disconnection) in word deafness.

Evidence from aphasia, reviewed by Blumstein (1987), suggests that a deficit at the level of phonetic analysis gives rise to a disturbance of auditory language comprehension only in the presence of a higher level language deficit. Similarly, according to our hypothesis, a deficit at the phonetic level gives rise to a manifest disorder of speech perception only if it is compounded with a lower level deficit of general auditory processes, that is, when a left temporal lobe infarct extends deep enough to involve interhemispheric auditory fibres, or when it is accompanied by a right temporal lobe infarct. This implies that speech perceptual processes, in spite of their left hemisphere specialization, are quite robust in the face of left hemisphere damage.

ADDENDUM

An anonymous reviewer proposed that the preserved discrimination of /bak/-/pak/ might be due to an artificial cue caused by the fact that the voiced and voiceless stimuli had different proportions of original and resynthesized speech. We investigated the possibility that voiceless stimuli sounded more unnatural than voiced stimuli by presenting 8 normal subjects with the same stimuli as used in the identification tasks on the VOT continuum and the two place-of-articulation continua. The subjects, who were naive with respect to synthetic speech continua, were asked to rate the 'naturalness' of the stimuli on a 5 point scale, ranging from −2 to +2. Prior to analysis, the 5 response categories were combined into 3 categories (unnatural, neutral, natural). The results demonstrated no difference in the percentage 'neutral' responses on the three continua (6%, 4% and 4%). The percentage 'natural' responses for each stimulus of the three continua is represented in fig. 4. The response patterns on the two place-of-articulation continua show, not unexpectedly, that the stimuli at the ends of the continua were most frequently rated as natural. The VOT continuum diverges from this pattern, but the stimuli at the voiced extreme of the continuum are not rated more frequently as natural than the stimuli at the voiceless extreme. Therefore, the pattern that would be expected if the suggested explanation for the preserved discrimination on the VOT continuum were right, was not obtained.

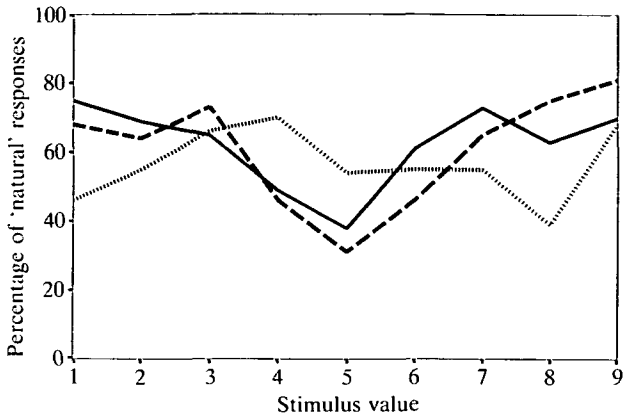

FIG. 4. Naturalness ratings by 8 normal subjects of the stimuli used in 1 VOT continuum and 2 place-of-articulation continua. Hatched line = bak-pak; broken line = pak-tak; continuous line = bak-dak.

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