Auditory Agnosia: Apperceptive or Associative Disorder?

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Neuropsychological testing of a patient with auditory agnosia showed that certain difficulties in the initial analysis of sounds may be the cause of his inability to understand spoken words and other sounds. Abnormalities included a slow reaction time to brief auditory stimuli (but not to equally brief visual stimuli or to longer auditory stimuli) and the need for approximately 1/2 sec of silence between two tones before the patient was able to hear them as separate. He could identify words and word associations if he was able to view the object whose name or word associate he was hearing. The findings imply that this patient's deficit in comprehending speech was probably apperceptive rather than associative in origin.

A preliminary version of this paper was presented to the 90th Annual Convention of the American Psychological Association, August 1982, Washington DC. We are grateful to Dr. Brenda Milner for consultation and for the use of test facilities and materials. We thank Dennis Rains for helping with part of the testing, Steve Burgida and Dr. Henry Ilecki of the Royal Victoria Hospital for their help in attempting to measure click separation thresholds in this patient, Dr. Braxton Milburn of the Audiology and Speech Pathology Service of the Ann Arbor V.A. Medical Center for help with control studies of reaction time to auditory stimuli near threshold, and Dr. Terry Allard and two anonymous referees for valuable comments and suggestions on the manuscript. Address correspondence and reprint requests to Henry A. Buchtel, Ph.D., Psychology (116B), Ann Arbor V.A. Medical Center, 2215 Fuller Road, Ann Arbor, MI 48105.

1 Supported in part by Grant MT2624 from the Medical Research Council of Canada to Brenda Milner, Sc.D. Preparation of the manuscript was supported by the Ann Arbor V.A. Medical Center and by the Department of Psychiatry, University of Michigan Medical School. Dr. Buchtel is currently at the Ann Arbor V.A. Medical Center and the Departments of Psychiatry and Psychology, University of Michigan, Ann Arbor.
INTRODUCTION

Auditory agnosia is a rare condition in which the person has a relatively isolated defect in auditory comprehension despite a normal or nearly normal audiogram. Such patients are unable to recognize either spoken words or environmental sounds. When the disorder affects only the comprehension of spoken language, the person is said to have verbal auditory agnosia or "pure word deafness." If only environmental sounds are affected, the person is said to have nonverbal auditory agnosia. Whether one or both kinds of auditory comprehension are affected, such patients are able to read, write, name objects, converse intelligently (Vignolo, 1969; Goldstein, 1974; Bauer & Rubens, 1985), and, at least in the case of pure word deafness, use intonation in a speaker's voice to identify the emotional content (Coslett, Brashear, & Heilman, 1984). The lesions responsible for these disorders are usually caused by cerebrovascular accidents and involve the midportion of the first temporal gyrus bilaterally (Goldstein, 1974; Buchman, Garron, Trost-Cardamone, Wichter, & Schwartz, 1986), or, if unilateral, a deep lesion in the posterior temporal lobe of the hemisphere dominant for speech (e.g., Kanter, Day, Heilman, & Gonzalez-Rothi, 1986). In the latter case, the individual may show signs of depending on right-hemispheric linguistic abilities (Metz-Lutz & Dahl, 1984). Coslett et al. (1984) have suggested that damage to the primary auditory cortex produces pure word deafness while damage to the auditory association cortex produces difficulties with nonspeech sounds. Damage to both regions would produce global auditory agnosia.

The pattern of disabilities in auditory agnosia differs from that of the more common Wernicke syndrome in that the receptive language disturbance is limited to the auditory domain, and the patient's spoken language is relatively good. Early accounts of the syndrome assumed that the basic auditory analysis (apperception) was normal while the ability of the person to link the sound with its meaning (association) was lost (e.g., Wernicke, 1908). This interpretation can still be found in current texts (e.g., "The patient can hear the language sounds but they remain meaningless, as if spoken in a foreign tongue" Benson, 1979, p. 134). Geschwind (1965) has classed the syndrome as a disconnection of auditory cortex from Wernicke's area, with the implication that both areas may be capable of normal functioning.

Nevertheless, evidence has been accumulating that the audiogram in patients with auditory agnosia is not an adequate measure of their ability to analyze the kinds of sounds that characterize speech: With more sophisticated auditory tests, several abnormalities have emerged. For instance, many such patients have difficulty determining the temporal order of sounds (Jerger, Weikers, Sharbrough, & Jerger, 1969) and many of them require a greater than normal sound intensity to make very brief sounds audible (Kanshepolsky, Kelley & Waggener, 1973; but see also
Chocholle, Chedru, Botte, Chain, & Lhermitte, 1975). Some but not all patients also have difficulty discriminating the loudness of stimuli and their location in space (right ear affected, Jerger et al., 1969; left ear affected, Jerger, Lovering, & Wertz, 1972). Disturbances of temporal resolution (Albert & Bear, 1974; Auerbach, Allard, Naeser, Alexander, & Albert, 1982; Tanaka, Yamadori, & Mori, 1987) and two-click discrimination (Chocholle et al., 1975) have also been described. The case we present here adds further evidence in support of the conclusion that a loss of comprehension of speech may be apperceptive in origin. In our patient the disorder may be the consequence, in part at least, of a very slow analysis of brief sounds and an independent or derivative disorder that manifests itself as the need for an unusually long period of silence between successive sounds before they are appreciated as distinct.

CASE REPORT

The patient is a 51-year-old right-handed normotensive man who had three myocardial infarctions when aged 41 and 42 years. At age 49, he woke with a partial weakness of the right limbs, the arm more so than the leg, and difficulty in speaking. The hemiparesis resolved within a few days but the speech deficit persisted. It consisted of a moderate to severe impairment in understanding spoken and written words; spontaneous speech was characterized by many paraphasias and word repetition. He was very dysnomic for common objects, and repetition of short sentences was poor. A CT scan done on the first day of his illness showed a large hypodense area in the region supplied by the left middle cerebral artery, consistent with an infarction. In the following months, his dysphasia improved and a year later he was able to obey complex commands, he could express himself clearly with only mild word-finding difficulty, and he had no dysnomia. There was, however, a great deal of difficulty in sentence repetition. Neurologic examination was normal except for very slight right lower facial weakness. There was no reflex asymmetry, visual field deficit, or sensory or visual inattention. He subsequently returned to work and his speech difficulties eventually improved to the point that they were no longer noticed by his fellow workers.

Seventeen months after the left cerebral infarction, he suddenly developed markedly abnormal speech. Neurological examination 2½ hr after the onset showed no understanding of spoken commands. There was clear but inappropriate spontaneous speech interspersed with incomprehensible jumbled words. He could not repeat simple phrases, nor could he read aloud or write. He had good power and normal reflexes in all limbs but the left plantar response was extensor. Within 4 hr, he developed mild left limb weakness, his eyes deviated to the right, and he tended to ignore his left side. By 6 hr after the onset of the symptoms, he had a dense flaccid left hemiparesis including a left upper motor
neuron-type facial weakness. By 15 hr, there was some return of power in the left limbs, and by 48 hr his limb strength had returned to normal. His marked language impairment persisted: He could understand written but not spoken commands and he claimed to have become deaf. His spontaneous speech was intelligible but repetitious, with an unmodulated cadence and occasional paraphasias.

A CT scan performed within 12 hr was of poor quality due to motion artifacts but showed low density areas in the left frontotemporal regions attributable to his previous infarction. Three months later a repeat CT scan showed these same abnormalities in the left cerebral hemisphere, but also a similar small area of low density in the right posterior temporal region (Fig. 1). These areas did not enhance with infusion of pertechnetate and had the appearance of infarctions in the territories supplied by both middle cerebral arteries. An audiogram showed good hearing with the left ear, and mild to moderate hearing loss with the right ear (Fig. 2).

In the 6 months following the second cerebral infarction, he was repeatedly examined, and this showed a mild left lower facial weakness, mild left hyperreflexia, and normal visual fields. Sensation was normal in the face and limbs, but there was extinction of pin-prick in the right face, arm, and leg on bilateral simultaneous testing. There was no extinction of bilaterally presented visual stimuli.

Neuropsychological Examination and Language Tests

The patient obtained a Full Scale IQ of 103 on the Wechsler–Bellevue Intelligence Scale (Verbal IQ 92; Performance IQ 114) and obtained a Wechsler Memory Quotient of 110 on the Wechsler Memory Scale (Form I). The examiner used written test instructions for communicating with the patient and presented numbers visually in the Digit Span subtest of the Intelligence and Memory Scales. On the basis of his best scores it

Fig. 1. CT scan showing two lesions in the left hemisphere (frontotemporal and parietotemporal) and a small lesion in the right posterior temporal area (left infarcts, October 1978; right infarct, March 1980. CT scan, June 1980).
is estimated that his premorbid intelligence was in the high average range (110–119).

His Verbal IQ is almost certainly below its premorbid level but it is likely that it had already been at this relatively low level after the left-hemisphere lesion and prior to the occurrence of the right hemisphere infarct. The Information, Digit Span, and Arithmetic subtest scores were not strikingly different from their presumed premorbid levels (+0.1, −0.6, and +0.6 SDs from the means for his age group, respectively) but his scores on Comprehension and Similarities were quite poor (1.8 and 1.3 SDs below the means for his age group, respectively).

Among the Performance subtests, only the Picture Arrangement score was lower than expected (1.1 SD below the mean for his age group) The other subtest scores were between 0.7 and 1.6 SDs above the mean for his age group.

The patient also had difficulty generating words starting with a particular letter of the alphabet (Chicago Word Fluency Test, Thurstone & Thurstone, 1943; see Milner, 1964). Since the patient is strongly right-handed, the left hemisphere is probably dominant for speech. Thus the verbal cognitive deficits are consistent with the left-hemispheric lesions (frontal and parietal) seen in the CT scan and inferred from the physical signs after the first stroke. Reasonably good scores on tests of immediate and delayed recall of verbal material (visually presented stories and paired verbal associates) suggest that the mesial structures of the temporal lobe of the hemisphere dominant for speech were relatively intact at the time of testing (see Milner, 1972).

His writing was fluent but contained occasional grammatical irregularities. His reading was entirely adequate for understanding task instructions but when tested in a systematic way (visually presented Token Test from
De Renzi & Vignolo, 1962) he obtained a score at the lower end of the mildly deficient range (50 correct responses in 62 trials); on an auditory version of the first three sections of this task he was correct on only six trials, which is barely above chance. His naming of objects (Oldfield & Wingfield, 1965) was normal. Finally, his speech was good enough to allow him to obtain the normal scores reported on certain verbal tests above; nevertheless, it was by no means unaffected. It had an explosive quality and was often inappropriately loud. His grammar was not always correct and occasional words could not be understood by the examiner. One may speculate that some of the oddities of his speech were caused by abnormal auditory feedback, or perhaps by an inability to monitor his own speech. Other errors appeared to be dysphasic in nature, though insignificant compared to his receptive disabilities.

The presence of a temporal lobe lesion on the right side of the brain may be the cause of relatively poor scores on memory for simple and complex drawings (Wechsler Memory Scale drawings; Rey-Osterrieth Figure).

**Auditory Comprehension**

In contrast to his relatively intact ability to perform tasks that do not depend on auditory comprehension, his impairment in understanding speech was severe. Comprehension of nonspeech sounds was also defective, as will be described below. When tested without a defined context, he was never observed to understand a single spoken word. He could not repeat words spoken to him and this inability extended, with rare exceptions, to single letters, numbers, phonemes, and nonsense sounds whether short or polyphonemic. He could not tell whether an utterance was a real word or nonsense: Overall, he was correct on 58% of the trials, which was not different from chance ($\chi^2 = 0.063; df = 1; p > .80$). Of 23 real words, he identified 14 as “real” (anchor, be, clock, comb, dice, folder, house, lock, map, needle, octopus, piano, picture, telephone) and 9 as “not real” (book, Dick, ear, horseshoe, pencil, scissors, toothbrush, trip, truck). Of 12 nonwords, he identified 6 as “real” (Standard IPA symbols: blig, klågøri", delà, mip, seif'øg, trik'bræk) and 6 as “not real” (blaek'rik, blig'læk, ðæk, klæg, prog, trib). He was also somewhat impaired, although better than chance, at determining whether 2 letters were the same or different. Of 27 pairs judged as “same” or “different,” he was correct on 17 (63%). Of 7 identical stimuli, he correctly identified 5 as “same” (L-L, N-N, N-N, G-G, B-B) and was unsure of 2 (A-A, presented at two different times). Of 20 mixed pairs, he correctly identified 7 after the first presentation (G-L, M-N, G-A, B-T, B-G, B-O, M-A) and a further 5 after a repeat presentation (D-F, P-L, N-R, X-I, Q-L). Three mixed pairs were called “same” after the first presentation (P-B, N-M, L-M) and 1 after a repeat presentation
(C-S). He was unsure about 4 pairs (N-L, Q-X, O-R, G-P). Finally, he also had considerable trouble telling whether a sound consisted of a single prolonged letter or 2 letters spoken 1 after the other without a break: Of 8 single letters, he incorrectly identified 1 ("X") as being a double letter and of 14 double letters, he incorrectly identified 6 as being single letters (A-L, B-A, L-M, T-O, M-N, L-O).

If the conversational context was well defined, he would respond appropriately, perhaps in part because of a rudimentary use of lip-reading. Frequently, however, it was clear that he was guessing at the meaning of the message. He would reply appropriately to a greeting if the circumstances were correct, but if asked to point to an object in the room or carry out any other action, he simply looked perplexed.

Interestingly, if three, six, or even eight drawings of common objects (Oldfield-Wingfield Pictures) were spread out on the table in front of him, and the name of one was spoken by the examiner, the patient pointed confidently and usually without error to the named drawing, repeating the word correctly. This observation is similar to the facilitation by context described by Saffran, Marin, and Yeni-Koshian (1976). To our surprise, he was frequently able to choose the correct drawing when an associated word was spoken, e.g., "secretary" to indicate the typewriter; "gamble" to indicate dice; "woman" to indicate a high-heel shoe (for examples see Fig. 3 and Table 1).

He had no particular difficulty generating words that rhyme with words that he read. This finding, in conjunction with the other test results, suggests that his internal speech may have been intact.

Environmental Sounds

He was unable to identify any items in a recognition task consisting of 20 tape-recorded sounds. On 7 items he could give no answer (lion roaring, water pouring, children playing, tractor engine running, bowling ball rolling, squeaking door, and screen door shutting) and 6 sounds were identified as people talking (car starting, effervescent bubbling, buzzer, water draining, power mower, and sandpaper rubbing). Other errors consisted of "music" for shower sounds and for a car idling, "start a car" for a manual lawn mower noise, "open the door" for telephone dialing, "radio" for telephone dial tone, "key in a lock" for windshield

Fig. 3. Example of stimuli for recognition from spoken word. Even associations ("gamble" for dice) elicited correct responses among three items.
APPRECEPTIVE DISORDER IN AUDITORY AGNOSIA

TABLE 1

<table>
<thead>
<tr>
<th>Targets</th>
<th>Verbal stimulus</th>
<th>Patient's response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pencil, piano, watch</td>
<td>Watch</td>
<td>(No response)</td>
</tr>
<tr>
<td>Pencil, piano, watch</td>
<td>Watch</td>
<td>Watch</td>
</tr>
<tr>
<td>Telephone, lamp, comb</td>
<td>Telephone</td>
<td>Telephone</td>
</tr>
<tr>
<td>Toothbrush, boat, scissors</td>
<td>Boat</td>
<td>Boat</td>
</tr>
<tr>
<td>Bed, tap, microscope</td>
<td>Bed</td>
<td>Bed</td>
</tr>
<tr>
<td>Book, cigarette, windmill</td>
<td>Cigarette</td>
<td>Cigarette</td>
</tr>
<tr>
<td>Metronome, chair, anchor</td>
<td>Holding the boat</td>
<td>Anchors</td>
</tr>
<tr>
<td>Key, stethoscope, clock</td>
<td>Open the door</td>
<td>Key</td>
</tr>
<tr>
<td>Octopus, drum, screw</td>
<td>Parade</td>
<td>(No response)</td>
</tr>
<tr>
<td>Octopus, drum, screw</td>
<td>Noise</td>
<td>Drum</td>
</tr>
<tr>
<td>Tuning fork, bagpipe, gyroscope</td>
<td>Scottish</td>
<td>That's not the word...</td>
</tr>
<tr>
<td></td>
<td></td>
<td>this one here (correct)</td>
</tr>
<tr>
<td>Horseshoe, dice, anvil</td>
<td>Gambling</td>
<td>(No response)</td>
</tr>
<tr>
<td>Horseshoe, dice, anvil</td>
<td>Gambling</td>
<td>Dice</td>
</tr>
<tr>
<td>Syringe, shoe, xylophone</td>
<td>Medicine</td>
<td>Needle (points to syringe)</td>
</tr>
<tr>
<td>Typewriter, glove, basket</td>
<td>Secretary</td>
<td>Typewriter</td>
</tr>
</tbody>
</table>

wiper and ‘‘to stop, she said’’ for a dog growling. A similar difficulty with environmental sounds has been described with some other patients having bilateral lesions (e.g., Jerger et al., 1969).

*Reaction Time to Sound and Light*

It has been suggested that patients with pure word deafness are less aroused by auditory stimuli than normal (Vignolo, 1969). This patient’s simple reaction times to tones were, in fact, unusual (Fig. 4, left section). The patient listened to 1500-Hz tones generated on a loud speaker located directly ahead at 60 dB SPL, or looked into a tachistoscope (Scientific Prototype Mfg. Co.) at a dark background onto which was projected a rectangle of bright light 7.8° by 5.6°. With 100-msec tones, the patient’s manual reaction times were probably not very different from their pre-morbid levels (mean of two separate tests: 256 ± 88 msec with 25 stimuli; 221 ± 52 msec with 15 stimuli) and were in the same range as the reaction times to a light of the same duration (229 ± 68 msec with 23 stimuli). With a tone lasting only 30 msec reaction times became significantly longer and somewhat more variable (mean of two separate tests: 393 ± 116 msec with 39 stimuli; 363 ± 175 msec with 25 stimuli) whereas the reaction time to a light stimulus of the same duration was only slightly slower than in the case of the 100-msec stimulus (245 ± 96 msec with 22 stimuli).

Since the patient had a moderate hearing loss at 1500 Hz in both ears (Fig. 2), the intensity of the 60-dB SPL tone stimuli was approximately 25 dB above threshold. For this reason, it is important to demonstrate
that his relatively slow reaction times to 30-msec tones did not simply represent responses to stimuli that, by their brevity, were close to threshold for that duration. This possibility was tested in two ways. First, we established thresholds for 1500-Hz tones in three normal individuals with varying degrees of hearing loss (15, 20, and 23 dB). These subjects then responded to the same number of presentations of 30 and 100-msec tones at an intensity 25 dB above their threshold (i.e., at 40, 45, and 48 dB SPL, respectively). The mean reaction times to these tones are shown in the right section of Fig. 4. It is clear that the reaction times to the brief tones at this intensity level are not statistically slower than the reaction times to the longer tones; in fact there is a tendency in the opposite direction.

A second approach was to study a single subject (the individual among the three above who had 23-dB loss at 1500 Hz) using stimuli at various levels above threshold. The results of this study are shown in Fig. 5. It is apparent that only at 10 dB above threshold is there a slowing of the reaction times to the 30-msec tones ($t = 5.45, df = 108; p < .0001$). An incidental observation is that the subject made 9 non-responses to the 30-msec tone at 10 dB above threshold but no non-responses at higher values. The patient made no non-responses during the test of his reaction times to 30-msec tones. This finding, in addition to the patient's own comment that he did not have trouble hearing the 30-msec tones, adds credence to the conclusion that the stimuli were not close to threshold.
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Two-Tone Discrimination

Because of the unusually slow response latency to 30-msec tones, a further test was administered which involved the discrimination between the presentation of one and two 30-msec tones separated by blank intervals of 20–500 msec. The patient was asked to judge whether one or two stimuli were heard, with the intervals between the two tones or lights varying pseudorandomly. The frequency of identifying two tones correctly as a function of the intertone stimulus is shown in Fig. 6. The patient needed a gap of approximately 250 msec before he could determine that
two stimuli rather than one had been presented. The result of administering this test with normal subjects indicates that the presence of a second tone should have been evident after an interval of approximately 15 msec, although two distinct tones may be heard only after an interval of approximately 50 msec.

**DISCUSSION**

This patient presented with the syndrome of relatively pure auditory agnosia following two cerebral infarctions involving the temporal regions bilaterally. This resulted in a virtually complete and persisting loss of auditory comprehension in ordinary conversation. While other speech functions were not entirely spared, these deficiencies were overshadowed by his inability to understand speech and most environmental sounds. In addition, and despite an essentially normal audiogram, the patient demonstrated very striking deficits in simple reaction time to brief tones and was unable to discriminate a single tone from two tones that were separated by less than 250 msec. His difficulty in the latter task does not depend on motor deficits or on a misunderstanding of the task since his performance with visually presented stimuli was essentially normal. There is evidence from other studies with word-deaf patients that auditory stimuli of short duration have to be unusually loud in order to be heard (see under Introduction). This may also be true in the present case since, for example, we were unable to carry out tests with “click” stimuli (<1 msec in duration) because the patient was unable to hear them. Nevertheless, he reported no difficulty in hearing the 30-msec duration tones in the reaction time test and the variance in reaction times was on the same order as the variance with visual stimuli, suggesting that there was no more uncertainty with the 30-msec tones than with visual flashes. His problem may derive rather from a slowness in the initial neural activation (perhaps recruitment) after the onset of the tone, or in a lengthening of the aftereffects of the initial tone such that the onset of a second tone is masked for almost a quarter of a second.

The presence of a slow initial analysis or of an abnormally long persistence would clearly interfere with the understanding of speech sounds. Many speech elements last only 40–200 msec and are followed immediately by another element that also has to be analyzed correctly for proper comprehension. With abnormally long auditory persistence, sounds would “avalanche” on top of each other, resulting in a jumbled, roaring sound, which is how the patient once characterized speech. While slowing of speech did not appear to help our patient’s comprehension (see also, e.g., Tanaka, Yamadori, & Mori, 1987), it is clear from this study and others that slowing of nonspeech sounds may improve performance. The hypothesis of abnormal persistence also suggests the presence of mechanisms that lengthen the physiological effect of sounds beyond their
physical duration and that these mechanisms are kept under control by an inhibitory mechanism. Since the present patient was unable to hear clicks, the function of such a prolongation is probably not to render very brief stimuli audible. Rather, it may be part of a system that serves to modulate transients in speech and other sounds.

One may ask why the patient was able to identify a word when its picture (among others) was placed before him. There are precedents for this observation in other reports. For example, the patient described by Saffran et al. (1976) performed better when the spoken words were drawn from a particular category and also when context was provided by placing the word at the end of a meaningful sentence (e.g., “boat” in the sentence, “The boy sailed the boat.”). One may hypothesize that the disturbed apperception in this patient caused speech to become jumbled, but it did not entirely mask all acoustic information in the message. Thus partial information, inadequate for identification of the word without a restrictive context, could be used to distinguish between the possible words being spoken, e.g., overall length of the word, place of emphasis, and certain preserved sounds that characterize only one of the possible alternatives. It might be possible to test this hypothesis by presenting artificially lengthened and superimposed speech sounds to normal subjects and testing if they can identify the distorted words with and without the presence of visual representations. The fact that the patient was also able to identify pictures from spoken words when the word was an associate of the picture (“gamble” for dice) is of interest because it suggests that the picture was capable of activating or facilitating the internal representation of words other than the simple name of the drawing. It should be noted that this was not a foolproof procedure: the patient did not always understand which picture was being indicated by the associative word. Nevertheless, if the word was the actual name of the object depicted, there were practically no uncertainties or errors. This observation may have an application in rehabilitation efforts with such patients and in producing conditions that favor communication in everyday interactions.

One may conclude from the findings described above that bilateral brain damage can lead to apperceptive deficits that render speech and certain other auditory stimuli incomprehensible. It is not necessary to postulate a higher-order deficit of an associative kind to explain the patient’s word deafness, and other patients with comprehension deficits may share some of the same deficiencies. An attempt is currently underway to assess patients with milder comprehension deficits from unilateral lesions to determine the extent to which their difficulties in understanding speech may include problems with temporal resolution and speed of response to unstructured tone stimuli. If deficits are found, they will possibly allow an objective study of the status of the disturbance and
thereby a method for assessing and documenting the recovery or lack of recovery in patients with disturbed comprehension.

REFERENCES


