Word deafness in Wernicke’s aphasia

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SUMMARY Three patients with otherwise typical Wernicke’s aphasia showed consistent superiority of visual over auditory comprehension. The precedents for and anatomical basis of a selective auditory deficit in Wernicke’s aphasia are discussed, including the relationship to pure word deafness. One implication of spared visual language function may be the use of gesture in language therapy for such patients.

Traditional descriptions of Wernicke’s aphasia have emphasised the disruption of central, supramodal mechanisms of language comprehension, together with paraphasic language output and impaired naming. Specific sensory modalities of language input, for example auditory comprehension and reading, have been described as equally impaired.\(^1\) \(^2\) Reading, by this formulation,\(^3\) takes place by association of visual symbols with previously stored auditory word images in Wernicke’s area. Damage to Wernicke’s area thus prevents processing of visual as well as auditory language. A few recent studies,\(^4\)\(^-\)\(^7\) however, have demonstrated differential involvement of auditory and visual comprehension in Wernicke’s and global aphasia, usually with relative sparing of the ability to read.

Selective impairment of auditory language processing has long been recognised in the syndrome of “pure word deafness”, in which isolated deficits of auditory comprehension and repetition occur in the absence of primary hearing loss or abnormalities of spontaneous speech, writing, naming, or reading. Most reported cases of pure word deafness, however, have manifested some degree of paraphasic speech.\(^8\) The language deficit of such cases thus resembles that of cases of Wernicke’s aphasia with disproportionate impairment of auditory comprehension. The following three cases exemplify the phenomenon of Wernicke’s aphasia with predominant word deafness.

Methods and report of cases

Three aphasic patients underwent a battery of language tests. The evaluation included a bedside language examination\(^1\) and the Boston Diagnostic Aphasia Examination (BDAE).\(^9\) The Token Test,\(^10\) a sensitive measure of comprehension, was administered both in the standard auditory format and also with the commands printed on cards. A battery of naming and matching-to-sample tests, modified from those described by Sidman et al,\(^11\) was also given. Naming tests included oral and written naming of letters, words, pictures, and colours. Matching tests included letter-letter and word-picture tasks, with samples presented in auditory and visual forms. Choices appeared in visual displays containing the correct and eight incorrect stimuli.

Case 1 This 55 year old right handed male former school principal abruptly developed paraphasic speech in March, 1979. He had a history of rheumatic heart disease and was maintained on warfarin for a prosthetic mitral valve. Despite his inability to understand conversational speech, he read the newspaper avidly and with apparent comprehension. When first examined two months later, he was noted to have a marked Wernicke’s aphasia, with verbal paraphasias, neologisms, and severely impaired auditory comprehension. He named only a few, simple objects. Repetition was intact for some single words but marked by literal paraphasias and perseverations. He followed six of eight oral commands of increasing difficulty but eight of eight of the same commands in printed form. He performed the Marie Three-Paper Test promptly and accurately. Writing was paragraphic and limited to single words and phrases. There were no discernible cranial nerve, motor, sensory, or cerebellar deficits. The BDAE showed deficits in spontaneous speech, naming, repetition, and auditory comprehension typical of Wernicke’s aphasia. The auditory comprehension deficit extended to yes/no questions as well as complex ideational material. Reading comprehension, by contrast, was remarkably preserved, including the sentence and even paragraph items. Writing was limited to his name, address, and some simple words. He was able to sing a melody and to imitate tapping patterns.

On the Token Test, the patient correctly performed 68% of the written and 46% of the oral directions (table). On the naming and matching tests, the patient named...
eight of 10 capital letters, seven of 10 small letters, seven of 20 pictures, and four of six colours, though the correct name was often embedded in paraphasic phrases. He wrote the names of only five of 20 pictures. On the matching tests (table), he matched 10 of 10 printed capital letters, 10 of 10 printed small letters, and four of 10 dictated letters to printed capital letters. He correctly matched 10 of 10 printed, eight of 10 dictated, and five of 10 dictated, spelled picture names to pictures. A computed tomographic scan (CT scan) (fig 2) showed a large area of reduced attenuation, consistent with infarction, in the left temporal region. As analysed by the diagrams of Gado, \textit{et al},\textsuperscript{12} the infarct involved most of the superior temporal gyrus and extended superiorly into the supramarginal gyrus. The angular gyrus, superior parietal lobule, and occipital lobe were spared.

The patient's course was one of gradual but incomplete improvement. When retested in August, 1979, he named 10 of 10 pictures, seven of seven colours, and 10 of 10 letters. His matching was without error on the word-picture matching tasks, except for a seven of 10 performance with the dictated, spelled word samples. His letter matching was also perfect except for a single error in the auditory-visual letter matching task. His Token Test results again showed superior performance with printed letters.

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<td>auditory form</td>
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<td>visual form</td>
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**Fig 1** CT scan (case 1) showing a low density area, consistent with infarction, in the left temporal lobe.
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Fig 2 CT scan (case 2) showing an area of reduced absorption in the left temporal region. On higher cuts this did extend into the inferior parietal lobe.

tion, and copying of geometric figures. His speech was hesitant, with occasional literal and verbal paraphasic errors, and with a marked tendency to add extra syllables to words. Naming of pictures, colours, letters, and objects was performed well, except for the added syllables. Auditory comprehension was deficient; he required numerous repetitions to perform even one-step commands, while he invariably missed two- and three-step commands. In contrast, he correctly followed eight of eight written commands and promptly carried out the Marie Three-Paper Test. Repetition was intact for short, high-probability phrases but impaired for complex phrases, sentences, and polysyllabic nonsense words. He wrote short sentences to dictation with some errors, and he copied accurately.

The BDAE revealed prominent difficulties with auditory comprehension and repetition, while his reading comprehension was intact for all but complex paragraphs. His speech output, both spontaneously and in response to repetition and reading tasks, showed numerous literal paraphasic errors, with palilalic suffixing of syllables and often complete unintelligibility. He named objects, pictures, and body parts as well. Writing was well formed but hesitant and contained numerous spelling errors. Rhythm, melody, and automatic sequences were also severely impaired. Auditory and visual forms of the short form of the Token Test were administered in October, 1979, and again in March, 1980. The patient correctly performed 32% of the auditory and 80% of the visual directions on the first test, and 26% and 94%, respectively, on the second. On the matching-to-sample test battery, his oral naming was 100% for visual letters, words, pictures, and colours, but only six of 10 for repetition of single syllable, three-letter picture names. Written naming was of 10 of 10 for pictures. He matched printed small letters and dictated letter names to printed capital letter choices without errors. He matched both printed words and spoken words to pictures without errors but scored eight of 10 with the dictated, spelled word samples (table).

The patient underwent neuropsychological testing by Dr Denton Buchanan. Right hemisphere tasks were performed at above-average level for the patient's age; he showed no constructional apraxia and demonstrated above-normal memory for visual designs. Estimated IQ based on visual organisation was 115. In contrast to this performance, left hemisphere functions were significantly impaired. CT scans performed in November, 1978, April, 1979, and March, 1980, all showed moderate cerebral atrophy and ventricular dilatation (fig 3). There were no focal areas of reduced density or of abnormal enhancement with contrast infusion.

The patient was reevaluated clinically and with repeat BDAEs in January, 1979, October, 1979, and March, 1980. The overall pattern of the language deficit had not changed, but progressive deterioration of auditory comprehension, speech intelligibility, and writing became evident. Reading comprehension remained stable, and he remained fully oriented, appropriate, and able to perform well on copying of figures, drawing a clock, and other visual-spatial tasks.

Fig 3 CT scan (case 3) showing only generalised cortical atrophy. No focal lesion could be identified.

Discussion

All three of our cases showed a language impairment characteristic of Wernicke's aphasia, with fluent, paraphasic speech, deranged auditory comprehension, and variably impaired naming and repetition. All showed partially preserved comprehension of printed language. Cases 1 and 2 had infarctions localised by CT scan to the left temporal region, while case 3 represents a progressive language deterioration of uncertain aetiology, associated with cerebral atrophy. In all three cases, the superiority of visual over auditory comprehension was mani-
fested in bedside testing of oral and written commands, in the BDAE subtests, in matching-to-sample tests involving auditory and visual samples, and in the auditory and visual forms of the Token Test. All cases had some impairment in visual comprehension tasks, naming and writing.

Most descriptions of Wernicke’s aphasia have considered comprehension to be affected equally in auditory and visual modalities. Wernicke, and later Lichtheim, envisaged reading as taking place by association of visual word images with previously stored auditory word images in Wernicke’s area. Such compulsory phonetic transcoding of visual images would explain the disruption of both visual and auditory comprehension with lesions of the auditory word association area. This traditional formulation of the reading process has been echoed by Benson and Geschwind, though more recently Benson has recognised variations in the degree of auditory versus visual comprehension deficit in Wernicke’s aphasia. Wernicke himself noted that well-educated individuals who develop Wernicke’s aphasia sometimes retain the ability to read.

A few recent authors have noted a disparity between auditory and visual comprehension, or between oral and written expression, in cases of Wernicke’s aphasia. Hécaen described a subsyndrome of Wernicke’s aphasia in which word deafness and inability to repeat exist as a nearly pure deficit, with relative preservation of reading, save for paralexic errors. Lhermitte and Dérouesné reported two patients with fluent aphasia whose writing did not share the paraphasic errors of their speech. In the first case, comprehension was superior for written over spoken language; the second patient had neologistic speech but normal auditory and visual comprehension. Basso, Taborelli, and Vignolo likewise found two cases of Wernicke’s aphasia in their material in whom writing was less paraphasic than spontaneous speech. Comprehension in the two modalities is not mentioned in their report. Hier and Mohr described a patient with Wernicke’s aphasia secondary to temporal lobe encephalitis in whom written naming was superior to oral naming, and visual comprehension was “modestly superior” to auditory comprehension, as measured by the matching of auditory and visual words to pictures.

Two other studies have attempted to correlate modality-based subgroups of Wernicke’s aphasia with specific sites of pathology. Mohr, Hier, and Kirshner presented in abstract form a retrospective analysis of CT scans in patients with Wernicke’s aphasia. In six cases with disproportionate auditory comprehension impairment, lesions appeared restricted to the temporal lobe, with only slight extension into the anterior portion of the inferior parietal lobe. In two cases with disproportionate visual comprehension impairment, lesions were more posterior, involving the supramarginal and angular gyri, and even portions of the superior parietal lobule and occipital lobe. In 23 cases with no modality disparity, the lesions tended to be large, encompassing or lying between the two sub-syndrome areas. The authors concluded that posterior parts of the left hemisphere could subserve reading even in the absence of a functional Wernicke’s area. Heilman, et al. presented three cases of Wernicke’s and global aphasia without alexia. One of these patients, who had not only retained reading but also learned to communicate by sign language, lost the ability both to read and to understand signs when he suffered a second stroke, localised to the right temporoparietal region by isotope brain scan. These authors postulated that preserved reading ability, performed without association to auditory word images, might be a function of the right hemisphere.

Kirshner and Webb recently reported a case of bitemporal infarction with preserved reading, writing, and sign language ability, but poor auditory comprehension and oral expression. In this patient both temporal lobes had sizeable infarcts on CT scan, while the posterior parieto-occipital regions of both hemispheres were intact. The presence of a right temporal lesion in this patient is evidence against the right hemisphere auditory word association area playing a role in the patient’s preserved reading ability.

The anatomical localisation of our patients’ lesions does not permit a definite conclusion regarding the site of preserved reading ability in patients with Wernicke’s aphasia. Cases 1 and 2 had infarcts largely restricted to the temporal lobe, though the lesion in Case 2 did extend into the angular and supramarginal gyri. Both lesions spared the superior parietal lobule, occipital lobe, and portions of the angular gyrus of the left hemisphere. Case 3 had no focal lesions identified, though the degree of perisylvian atrophy is such that a small infarct could easily be missed. Aphasia has been reported as a presenting symptom in dementia, and Horenstein has noted the frequent occurrence of auditory agnosia in demented patients. The patient’s course has been progressive, but as yet his behavioural deficits have not become sufficiently generalised to justify a diagnosis of dementia.

The phenomenon of Wernicke’s aphasia with disproportionate involvement of auditory as compared to visual comprehension is clearly defined as a behavioural syndrome. Formerly, such cases might have been classified as pure word deafness. The paraphasic speech, impaired naming, and imperfect
reading ability, however, leave no doubt as to the presence of fluent aphasia, in addition to word deafness, in these patients. As reviewed by Goldstein,1 most reported cases of pure word deafness have manifested some degree of paraphasic speech. The best studied cases have involved bilateral temporal lesions, presumably isolating the two Heschl's gyri from Wernicke's area. Cases of unilateral temporal pathology have been described, but are more difficult to explain on the basis of anatomic disconnection of Wernicke's area from both primary cortical auditory areas. We suggest that many of these unilateral cases may represent Wernicke's aphasia with predominance of auditory language impairment.

The importance of recognizing selective impairment of modalities in aphasia, aside from theoretical interest, lies in the use of spared modalities to improve communication. Our patients 1 and 3 have spontaneously communicated in writing; patient 2, whose deforming arthritis prevented writing, made substantial gains with the use of gestures. Heilman, et al.,7 and Kirshner and Webb26 have also described the ability of aphasic with spared visual language functions to learn sign language. Gestural communication, by sign language or pantomime, shares with reading the visual modality, and evidence has been presented23 that these two communication functions may correlate better with each other than with auditory comprehension. Further studies on the use of gestural communication in modality-selective aphasia syndromes are clearly needed.

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