Word deafness in Wernicke’s aphasia

HOWARD S KIRSHNER, WANDA G WEBB, AND GARY W DUNCAN

From the Departments of Neurology and Hearing and Speech Sciences, Vanderbilt University School of Medicine and Nashville Veterans Administration Medical Centre Nashville, Tennessee, USA

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. Reading, by this formulation, 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 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. 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 aphasis patients underwent a battery of language tests. The evaluation included a bedside language examination1 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, et al, 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 selections.

Case 2. This 60 year old right handed male developed a right hemiparesis and aphasia in September, 1979. Upon transfer to the Nashville VA Medical Centre four weeks later, his speech output consisted largely of repetitive jargon syllables and inappropriate words (examples were “shovel,” “peanut butter,” and “pepper,” uttered repetitively). He occasionally replied “I don’t know” to questions. He was unable to name objects or body parts. Repetition was limited to one-syllable words. He could count aloud to ten but could not recite the days of the week. Auditory comprehension was unreliable for yes/no or nonsense questions. He followed two of eight dictated and six of eight printed commands. He frequently attempted to read the printed commands aloud, uttering only meaningless monosyllables while performing the command accurately. Writing was not testable because of hand deformities secondary to rheumatoid arthritis. He was able to point to printed words appropriately on a communication board (for example “hungry”, “cold”, “thirsty”), and he spontaneously gestured the meanings of several of these.

On the BDAE, the marked paraphasic speech, inability to name, and impaired repetition fit the diagnosis of Wernicke's aphasia, but the relative sparing of reading comprehension was striking. The patient was also noted to be alert to gestural input, with comprehension of spoken phrases and commands improving markedly when accompanied by a gesture. He was able to learn a few simple gestures of Amerind Sign Language, though his hand deformities limited his ability to perform these.

On the Token Test, his performance with auditory commands was 0% while with printed directions it improved to 39%. In matching-to-sample test, he matched only one of 10 dictated letters to printed capital letter choices, while he correctly matched seven of 10 printed small letters to capital letters (table). On the word-picture task, he matched two of 20 dictated picture names, 0 of 10 dictated spelled picture names, and 15 of 20 printed picture names to pictures. Similarly, he matched one of six dictated but six of six printed colour names to colours. A CT scan showed an area of decreased density involving the superior temporal gyrus, but extending superiorly and posteriorly into the supramarginal and angular gyri. The superior parietal lobule and occipital lobe appeared spared.

Case 3. This 61 year old right handed male first noted a “stutter” and difficulty expressing himself during a stressful situation in June, 1976. Over the next two years the impaired fluency became more evident, and his wife noted auditory comprehension difficulty as well. An audiogram revealed only slight bilateral high frequency hearing loss. Neurologic evaluation in November, 1978 revealed an alert, oriented man who performed normally on bedside tests of memory, calculations, general informa-

![CT scan (case 1) showing a low density area, consistent with infarction, in the left temporal lobe.](http://jnnp.bmj.com/)

Table Matching to sample tests

<table>
<thead>
<tr>
<th></th>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Capital letter—</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>visual-visual</td>
<td>10/10</td>
<td>10/10</td>
<td>10/10</td>
</tr>
<tr>
<td>Small letter—</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>visual-visual</td>
<td>10/10</td>
<td>7/10</td>
<td>10/10</td>
</tr>
<tr>
<td>Letter-letter</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>auditory-visual</td>
<td>4/10</td>
<td>1/10</td>
<td>10/10</td>
</tr>
<tr>
<td>Word-picture</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>visual-visual</td>
<td>10/10</td>
<td>15/20</td>
<td>10/10</td>
</tr>
<tr>
<td>Word-picture</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>auditory-visual</td>
<td>8/10</td>
<td>2/20</td>
<td>10/10</td>
</tr>
<tr>
<td>dictated sample</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Token test</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>auditory form</td>
<td>46%</td>
<td>0%</td>
<td>32%</td>
</tr>
<tr>
<td>Token test</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>visual form</td>
<td>68%</td>
<td>39%</td>
<td>80%</td>
</tr>
</tbody>
</table>
Word deafness in Wernicke's aphasia

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 lobule.

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,15
and later Lichtheim,16 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,1 though more recently
Benson17 has recognised variations in the degree
of auditory versus visual comprehension deficit in
Wernicke’s aphasia. Wernicke himself18 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. Hécaen4 described a sub-
syndrome of Wernicke’s aphasia in which word
defauness and incaentability to repeat exist as a nearly
pure deficit, with relative preservation of reading,
save for paraphasic errors. Lhermitte and Dérécuesné18
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 Vignolo19
likewise found two cases of Wernicke’s
aphasia in their material in whom writing was less
paraphasic than spontaneous speech. Comprehen-
sion in the two modalities is not mentioned in their
report. Hier and Mohr5 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
Kirshner6 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 re-
stricted to the temporal lobe, with only slight ex-
tension into the anterior portion of the inferior
parietal lobule. 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
lobe and occipital lobe. In 23 cases with no modality
disparity, the lesions tended to be large, encom-
passing or lying between the two sub-syndrome
areas. The authors concluded that posterior parts of
the left hemisphere could subservive reading even in
the absence of a functional Wernicke’s area. Heilman,
et al.7 presented three cases of Wernicke’s and global
aphasia without alexia. One of these patients, who
had not only retained reading but had 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 postula-
ted that preserved reading ability, performed
without association to auditory word images, might
be a function of the right hemisphere.

Kirshner and Webb20 recently reported a case of
bihemispheric 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 regard-
ing 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 peri-
sylvian atrophy is such that a small infarct could
easily be missed. Aphasia has been reported as a
presenting symptom in dementia,21 and Horenstein
has noted the frequent occurrence of auditory
agnosia in demented patients.22 The patient’s
course has been progressive, but as yet his behavoural
deficits have not become sufficiently generalised
to justify a diagnosis of dementia.

The phenomenon of Wernicke’s aphasia with
disproportionate involvement of auditory as com-
pared 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, 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 Heschel'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. and Kirshner and Webb have also described the ability of aphasics 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 presented 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.

This research was supported by NINCDS Teacher Investigator Development Award I KO7 NS 00429-01 to Dr Kirshner and also by the Veterans Administration. The authors thank Mrs Laura Peterson and Mrs Suzanne Allen for technical assistance, Dr Denton Buchanan for the neuropsychologic testing of Case 3 and Miss Beth Gehrke for assistance in preparation of the manuscript.

References

8 Goldstein MN. Auditory agnosia for speech ("pure word-deafness"). Brain Lang 1974; 1:195–204.
10 Spreen O, Benton A. Token Test. From the Neurosensory Center Comprehensive Examination for Aphasia, Neuropsychiatry Laboratory, University of Victoria, 1969.
20 Kirshner HS, Webb WG. Selective impairment of the auditory modality in an acquired communication disorder. Brain Lang, in press.
Word deafness in Wernicke's aphasia.

H S Kirshner, W G Webb and G W Duncan

*J Neurol Neurosurg Psychiatry* 1981 44: 197-201
doi: 10.1136/jnnp.44.3.197

Updated information and services can be found at:
http://jnnp.bmj.com/content/44/3/197

**Email alerting service**

Receive free email alerts when new articles cite this online article. Sign up in the box at the top right corner of the article.

**Notes**

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/