

Atypical Conduction Aphasia

A Disconnection Syndrome

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● Conduction aphasia was originally proposed to result from separation of the posterior language comprehension area and the anterior motor speech area of the left hemisphere. The arcuate fasciculus has been the most frequently suggested site of such a disconnection, but the syndrome has been reported in cases in which the abnormality involved the dominant Wernicke's area. This challenges the arcuate fasciculus theory, and it has been suggested that a cortical lesion, not a disconnection, is the crucial factor. Three new cases in which the lesion does not lie in the arcuate fasciculus are reported, two in left-handed patients with left temporoparietal lesions and one in a right-handed patient with a right temporoparietal infarct, a "crossed" aphasia. While atypical, these cases offer evidence that disconnection of the circuit linking language comprehension to motor speech output, not damage to a specific cortical region, underlies the syndrome of conduction aphasia.

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The existence of conduction aphasia, a disorder characterized by disproportionate difficulty in the repetition of spoken language, was originally proposed by Wernicke¹ on purely theo-

retical grounds. He postulated that any lesion disrupting the principal pathways connecting the site of auditory language comprehension and the site of motor expression of speech should result in a characteristic aphasic disorder. The postulated language disturbance was subsequently described clinically,^{2,5} and although initially considered rare,⁶ recent reports suggest that conduction aphasia may constitute up to 10% of classifiable aphasias.⁷ Wernicke's original explanation for the aphasic syndrome has not been universally accepted, however.⁸ Several alternate explanations, eg, Goldstein's⁹ theory of a disturbance of a central language area or recent presentations that suggest that damage to a specific language center¹⁰ or to the cortex of the parietal operculum,⁸ have been suggested for this syndrome.

One important step toward understanding the mechanism underlying conduction aphasia is location of the neuroanatomic site or sites of lesions that produce the syndrome. Most cases reported in the literature show abnormality near the posterior rim of the left sylvian fissure, either temporal or parietal.^{9,11} The usual lesion has been suprasylvian, involving the parietal opercular area (supramarginal gyrus) with many cases showing disturbance of the arcuate fasciculus, a fiber bundle coursing between temporal and frontal association cortices. Arcuate fasciculus abnormality appears consistent with Wernicke's¹ disconnection hypothesis. Based on this and the frequency of its occurrence with this disorder, interruption at the

arcuate fasciculus can be considered the "typical" model of conduction aphasia.

Other locations of neuroanatomic damage have resulted in this language disturbance. Destruction of the left temporal region, including Wernicke's area, a lesion that would characteristically produce impaired comprehension, has been reported in a number of cases in which a defect in repetition was the major finding.¹²⁻¹⁴ As one of the authors (D.F.B.) has previously pointed out,¹¹ a lesion involving a single neuroanatomic location cannot explain all cases of conduction aphasia. Three additional cases of patients with conduction aphasia who do not have a lesion in the arcuate fasciculus are reported. They provide an opportunity to reexamine the validity of the disconnection theory of conduction aphasia and to compare it with other hypotheses.

REPORT OF CASES

CASE 1.—A 60-year-old left-handed man with a strong family history of sinistrality presented with an acute onset of speaking disturbance and right arm and leg numbness of 12 hours' duration. There was a well-documented history of recurrent supraventricular tachycardia secondary to Wolff-Parkinson-White syndrome, a history of hypertension, and a left nephrectomy performed 15 years previously for renal cell carcinoma.

Blood pressure was 120/80 mm Hg, pulse rate was 80 beats per minute and regular, and respirations were 24/min with some respiratory distress secondary to congestive heart failure (CHF). There was an S₃ gallop, jugular venous distention, and moist rales heard at the lung bases.

Neurologic examination revealed an

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alert man with a mild aphasia. The corneal reflex was decreased on the right and deep tendon reflexes were slightly increased on the right compared with the left. Primary sensory modalities were mildly impaired in the right upper extremity.

Language examination demonstrated a fluent verbal output with word-finding pauses, circumlocutions, and increasing approximations in approaching the missed word. There was a strong tendency for phonemic substitutions (literal paraphasia), eg, "bundle" for "buckle" and "klist-er" for "crystal." The patient made many attempts to self-correct his paraphasic errors. Spoken language comprehension was totally normal, even for complex multistep commands and the understanding of relational words. Repetition of spoken language was considerably abnormal with marked hesitations and multiple literal paraphasias. Number repetition revealed frequent semantic substitutions. Confrontation naming was done well, except for paraphasic errors. Comprehension of written language was excellent, but both reading aloud and writing were contaminated by phonemic substitutions. All verbal commands were carried out without difficulty, the patient showing no evidence of ideomotor apraxia or body part substitution.

The admitting diagnosis was embolic stroke. Initial laboratory data were unremarkable except for left ventricular hypertrophy on electrocardiogram and evidence of mild congestive heart failure on chest roentgenogram. Computed tomographic (CT) scan was normal at the time of hospital admission, but a repeated scan a few days later demonstrated a low-density area involving much of the left temporoparietal region (Fig 1). Heparin sodium anticoagulation and digoxin therapy were initiated. The hospital course was complicated by hypertensive episodes, runs of supraventricular tachycardia, and the discovery of mild hyperthyroidism.

Reexamination at the time of discharge from the hospital and again as an outpatient one month after onset demonstrated improved language function with fewer word-finding difficulties and literal paraphasias on spontaneous speech, but continued difficulty in repetition.

CASE 2.—A 26-year-old strongly left-handed man noted the sudden onset of word-finding difficulties and frequent mispronunciation of words unaccompanied by any other symptoms. Three to four days later he noticed tingling paresthesias of the distal part of the right arm and leg and was subsequently admitted to the hospital for evaluation of his condition. Medical history proved unremarkable, except for a recent dental abscess. There was no family history of left-handedness.

Results of general physical examination at the time of hospital admission were normal, but the neurologic examination revealed a right upper quadrantopsia and a flattened right nasolabial fold. Motor testing demonstrated mildly decreased fine motor movements of the right hand. Deep tendon reflexes were slightly more active on the right side, but the toes were

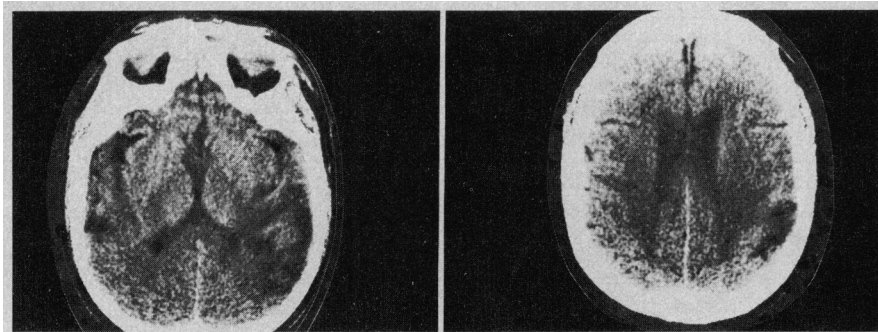


Fig 1.—Case 1. Computed tomographic scans showing large left temporoparietal hypodense area.

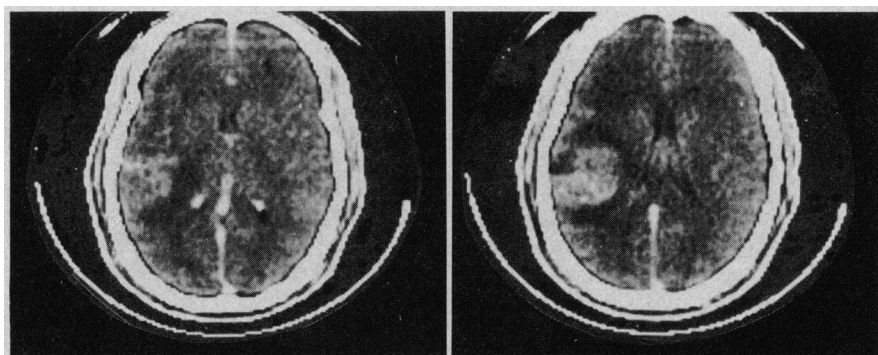


Fig 2.—Case 2. Computed tomographic scans demonstrating hyperdense, enhancing area with surrounding area of edema, ultimately demonstrated as arteriovenous malformation. Left, parietal involvement; right, temporoparietal junction involvement.

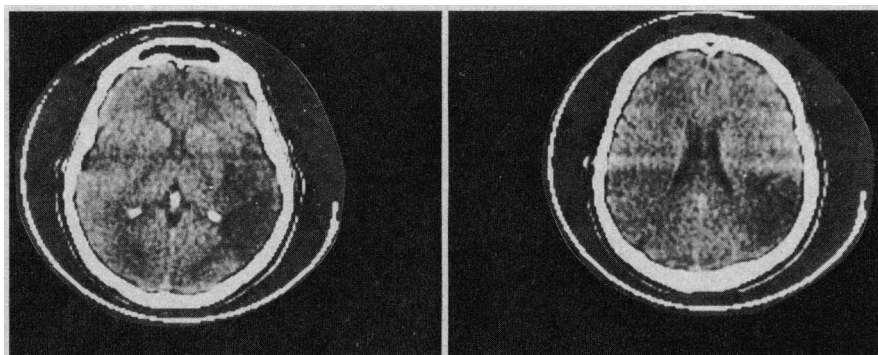


Fig 3.—Case 3. Computed tomographic scan demonstrating large right parietal hypodense area with extension to posterior temporal area.

downgoing. No sensory deficits were found.

Language was strikingly abnormal. Word-finding pauses and phonemic substitutions were present in conversational speech, confrontation naming, and reading aloud, but the greatest difficulty occurred when he was asked to repeat words or phrases. There was a strong tendency to self-correct paraphasic errors, especially by increasing phonemic approximations. Typical examples of his paraphasic errors included "walt" for "walk" and "rick" for "wrist." Comprehension of both oral and written language was tested carefully and was unimpaired. He understood complex sentences, grammatical words, and multi-

step commands. His writing showed misspellings and paragraphic substitutions. A mild difficulty copying three-dimensional drawings was demonstrated, but no cognitive deficits were found; he performed flawlessly on all praxis tests.

A CT scan on the day of admission disclosed a hyperdense and peripherally enhancing lesion with surrounding edema located in the left temporoparietal area (Fig 2). Because of the recent tooth infection the patient was treated with intravenously administered penicillin and metronidazole until an angiogram was performed. Angiography showed a left middle cerebral artery terminal branch arteriovenous malformation that had bled. After much

discussion it was the decision of the patient and his family not to proceed with any therapeutic procedure but to continue with close clinical follow-up and serial radiologic studies. He was discharged from the hospital one month after onset having improved to the point of only a mild residual language disturbance.

CASE 3.—A 58-year-old right-handed man, visiting at a friend's house, experienced the acute onset of a right temporal headache and an inability to speak properly. His friends noted garbled speech and nonsensical phrases but believed this to be an effect of alcohol. The next morning he became frustrated when unable to speak coherently over the telephone and he drove himself 64 km to a hospital for evaluation. The patient was strongly right-handed and favored the right foot as well. There was no family history of any left-handedness.

General examination revealed a well-developed man in no acute distress. No bruits were heard but the left carotid pulse was less forceful than the right. Peripheral pulses were absent in the right lower extremity. Neurologic examination showed a left hemi-inattention with extinction on double simultaneous stimulation and decreased swing of his left arm when walking.

Language output was fluent with normal grammar and prosody but contaminated by multiple literal paraphasias and frequent word-finding pauses. Comprehension of both oral and written material was partially compromised at the time of hospital admission (he was unable to perform two-step commands), but this improved rapidly so that only minimal difficulty in the comprehension of complex material could be demonstrated within a few days. In contrast, repetition was disproportionately impaired and remained abnormal during hospitalization. He often failed even single-word repetitions. Attempts to name were characterized by pauses and literal paraphasias, although he recognized his errors and tried to correct them. Examples included "botel-butel-buckle," "caid" for "pain," and "pim" for "pills." Reading comprehension was considerably better than reading aloud, which was also contaminated by literal paraphasias, and his writing contained multiple paragraphic errors and misspellings.

There was a notable left-sided neglect including a "dressing disturbance" of the hemineglect variety, a failure to dress his left side. He was unable to copy three-dimensional drawings nor could he locate areas on a map. He showed persistent difficulty in finger recognition and an acalculia. In contrast, there was no evidence suggesting visual agnosia, prosopagnosia, or amnesia. Results of all tests for ideomotor apraxia were normal.

A CT scan demonstrated a large, nonenhancing, hypodense lesion in the right parietal area with extension to involve some portions of the posterior temporal region (Fig 3). Three serial CT scans did not show any lesion in the left hemisphere. Arteriography revealed absence of posterior branches of the right middle cerebral

artery with some enlargement of collateral vessels. The aphasia improved during the hospital course, with comprehension returning to normal, but the marked inability to repeat spoken words or phrases remained.

COMMENT

These patients met established clinical criteria for conduction aphasia⁷; they showed fluent paraphasic conversational speech, fairly normal oral comprehension, and a disproportionate disturbance in the ability to repeat spoken words. Spontaneous speech, confrontation naming, reading aloud, and particularly repetition were contaminated by literal paraphasias. In contrast to many reported cases, these three patients demonstrated considerable awareness of their errors, attempted self-correction, and the *conduit d'approche* phenomenon of increasing phonemic approximations was notable. Repetition of numbers was often failed because of semantic or whole-number substitutions. Reading comprehension was good, but writing had spelling and grammatical errors.

In contrast to the typical clinical picture, these three cases were atypical in location of the lesion and none could be explained by an interruption of the arcuate fasciculus pathway. The first two involved Wernicke's area of the left hemisphere, similar to the case reported by Kleist,¹⁴ while the third is a case of "crossed" conduction aphasia.

Wernicke's original theory for the mechanism of conduction aphasia proposed a separation of the left-sided temporal auditory comprehension area (Wernicke's area) from the left frontal motor speech area (Broca's area).¹ He suggested that this could result from damage to the white matter tracts that form a direct pathway between the major language zones.¹ The localization of lesions of cases reported in the literature has often been in accord with this formulation.^{10,15} A lesion involving these tracts in and of itself does not prove, however, that the white matter lesion is causative because a lesion of the adjoining gray matter might be responsible. The theory that arcuate fasciculus lesions are one cause of conduction aphasia as proposed by Wernicke,¹ Lichtheim,² Kleist,¹⁴ and more recently, Geschwind,¹⁶ Benson et al.,¹¹ and Damasio and Damasio,¹⁵ has thus been contested. None of the recent investigations, however, have proposed arcuate fasciculus damage as the *only* explanation for conduction

aphasia. Alternate postulations have been proposed on the basis of new information available concerning the pathophysiology of the disorder. Our three atypical cases, along with others reported in the literature, offer data that can be used to substitute, modify, or refute the disconnection model. Any explanation of conduction aphasia must account for the atypical cases.

Establishment of the disconnection theory for conduction aphasia could be of importance. It would make conduction aphasia one of the few language disorders with a truly understandable theoretic mechanism. It would also lend support to the basic Wernicke¹ model for the organization of language in the brain: two major language centers, one for verbal comprehension and the other for verbal expression, linked by a direct pathway with additional circuits for the mediation of semantic meaning.

One additional neurobehavioral observation, the presence or absence of accompanying ideomotor apraxia, deserves attention in this discussion. The ability to carry out actions following verbal command has been said to depend on pathways connecting the auditory language comprehension area with the pertinent motor areas.^{17,18} While apraxia is frequently reported in conjunction with conduction aphasia,¹¹ dissociation of the two disorders is also reported,¹¹ suggesting that separate routes may be available from language comprehension to motor areas.

Cases 1 and 2 of this report closely resemble Kleist's early description.¹⁹ Both subjects were left-handed and suffered acute vascular lesions that involved the posterotemporal cortex, including Wernicke's area plus adjoining parietal cortex of the left hemisphere. Comprehension of spoken language was apparently performed through the right auditory association area, while motor speech was performed by the left frontal cortex. Repetition of spoken language thus demanded an interhemispheric connection that was damaged. In contrast, the motor activities performed to verbal command (praxis) apparently could be performed via a right temporal to right motor cortex and then anterior callosal connection. In other studies good correlations have been reported between the types of aphasia and localization of the lesion by CT scan.^{20,21} The areas of pathologic involvement demonstrated by x-ray CT scans in these two cases and the pathways theorized for both repeti-

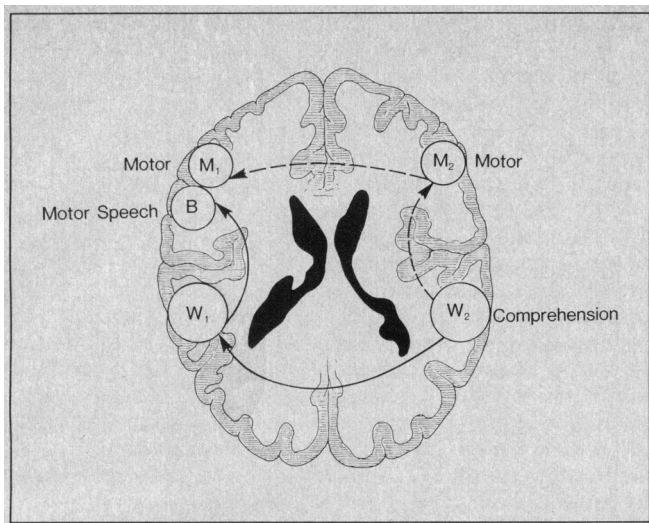


Fig 4.—Diagram based on disconnection theory for cases 1 and 2, demonstrating interruption of language pathways from lesion in left temporoparietal area, including Wernicke's area. Language pathways are indicated by solid line. Praxis pathways are indicated by dotted line. M₁ indicates left motor area; M₂, homologous area on right; B, Broca's area or left motor speech area; W₁, location of Wernicke's classic area; W₂, homologous area for language comprehension in right hemisphere.

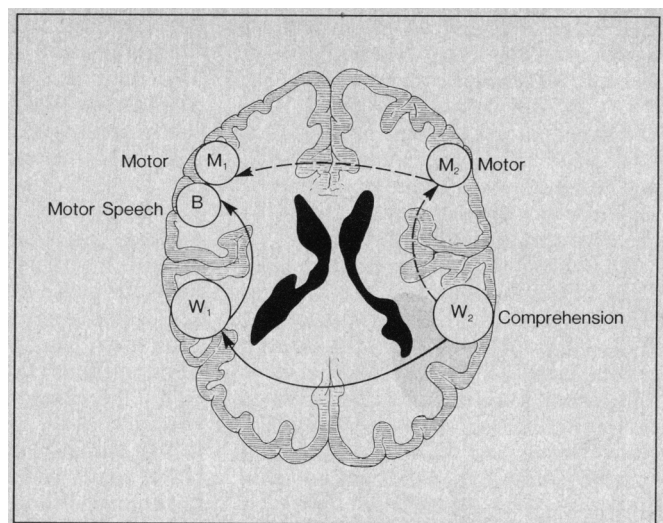


Fig 5.—Diagram for case 3 based on disconnection theory. Language pathways are interrupted by lesion in right temporoparietal area as they cross posteriorly to homologous area on left. Praxis pathways that travel anteriorly in right hemisphere are spared. Abbreviations are same as in Fig 4.

tion and ideomotor apraxia are outlined in Fig 4.

These two cases replicate neuropathologic observations that Wernicke's area may be destroyed without production of a severe comprehension deficit.^{4,11,22} Kleist¹⁴ promoted the explanation that conduction aphasia could result from a disconnection of a locus for comprehension located in the right temporal area from the more consistently located left frontal motor speech area. Interhemispheric transfer across the corpus callosum to the homologous structure in the left hemisphere would be interrupted by damage to the left hemisphere Wernicke's area. Such an explanation is clearly compatible with the disconnection theory.

Our first patient had a strong familial history of left-handedness, while the second had no family history of sinistrality. Patient 2 may have shifted hand dominance because of the arteriovenous malformations, with a partial shift of language comprehension to the right hemisphere as well. Pathologic left-handedness of this nature is well recorded,²³ and a hemispheric shift of language with left temporal and/or parietal developmental abnormality has also been demonstrated.²⁴

The third case is a rare crossed aphasia, a language disturbance resulting from a right hemisphere lesion in a dextral. Both clinical and CT scan evidence indicated significant

right hemisphere temporoparietal damage. Comprehension appears to be subserved by the right temporal auditory area as substantiated by the initial comprehension difficulty correlated with the presence of a limited degree of damage in the right posterior temporal cortex. The possibility that a small second lesion in the left hemisphere underlies the aphasia seems highly unlikely, given the temporal relationship of his right hemisphere stroke with the aphasia and the total absence of any left hemisphere abnormality on repeated neurological examinations and three serial CT scans.

Figure 5 presents a theoretical explanation of case 3. Right temporoparietal damage disconnects the right temporal area (comprehension) from the left temporal area and, hence, the pathway to the left frontal motor area. Right temporal to right frontal connections are again used to carry out motor activities in this patient who also lacked any evidence of ideomotor apraxia. The model is identical to that of the first two cases, even though the causative abnormality is in the opposite hemisphere.

A less likely schema would be interruption of a pathway for motor speech running forward in a fully language-dominant right hemisphere. With this explanation the pathway for carrying out verbally commanded motor activities must travel separately, either in the same hemisphere or through a

posterior connection. A third possibility would be an exact reversal of the Kleist¹⁴ diagram, with comprehension in the left hemisphere and motor speech in the right, an even less likely possibility given the initial comprehension deficit.

Other theories have been put forward to account for the clinical findings in conduction aphasia. One prominent rival model is "central aphasia." Goldstein⁹ proposed the existence of a central cortical language area to relate "inner speech" to external speech. With disruption of this process a language disturbance characterized by a disproportionate problem in correctly repeating spoken language can be theorized. Regardless of some strengths of the Goldstein model (eg, the ability to account for more variability in the clinical presentation), it requires a "central" area connected to a speech decoding site and a speech encoding site and is really only a modification of the Wernicke¹ model featuring a "language center" rather than a pathway. Location of this language center is difficult; conduction aphasia has been reported with lesions occupying a semicircle around the posterior rim of the left sylvian fissure, including abnormalities individually or in combinations in the temporal auditory complex, supramarginal gyrus, insula, the white matter deep to these regions, and possibly even the adjacent posterior frontal cortex.²⁵⁻²⁸ Of 25 cases with neuro-

anatomic information reviewed by Green and Howes,^{10, 13} had mixed lesions extending from temporal gyrus to supramarginal gyrus, nine had supramarginal gyrus involvement with sparing of the temporal area, and three involved the temporal auditory cortex without abnormality in the supramarginal gyrus.

Our third case suggests that even right hemisphere lesions can be associated with this language disorder. While it is possible that Goldstein's central area could have such a variable location, a disconnection between the major centers appears more plausible. Green and Howes proposed a specific center to synthesize semantic meaning with linguistic form, a hypothesis poorly compatible with the number of reported neuropathologic sites.

More recently, Levine and Calvanio⁸ proposed another hypothesis for conduction aphasia. They postulated a gradient of overlapping auditory speech and motor speech cortices extending from Broca's area to Wernicke's area and suggested that abnormality anywhere along this course could produce a repetition deficit. They further suggested that the varying concentrations of neurons for speech perception and speech production determine the clinical features of the syndrome. Thus, the most posterior lesions result in a clinical picture approaching Wernicke's aphasia, while the more anterior lesions result in a more nonfluent conduction aphasia. They also suggested that the disproportionate repetition deficit is actually a milder form of auditory perceptual deficit in comparison with Wernicke's aphasia. This model has obvious difficulty accounting for the occurrence of conduction aphasia with large temporoparietal lesions that encompass Wernicke's area such as in our first two patients and fails to account for the almost perfect comprehension reported in some patients with conduction aphasia. Many patients with conduction aphasia have been shown to have correct memory of the phrases they fail to repeat, again suggesting adequate to excellent auditory comprehension.

The Levine-Calvanio⁸ hypothesis of cortical damage in the supramarginal gyrus does not differ markedly from the arcuate fasciculus theory. In fact, Geschwind^{16(p29)} clearly points out that "since there is considerable evidence that the arcuate fasciculus consists in great part of short fibers . . ." it is likely that purely cortical lesions of the supramarginal gyrus would

destroy part of the pathway linking Wernicke's and Broca's areas.

Various neurolinguists have interpreted the language difficulties of conduction aphasia as either a speech encoding or a speech decoding disorder. Dubois et al²⁹ proposed that the essential problem involved the production of phonemes. Conduction aphasia became an expressive aphasia with a marked tendency to transpose and substitute phonemes resulting in prominent phonemic substitutions in all aspects of language. This theory would be compatible with the often reported ability of conduction aphasics to monitor and self-correct. Repetition would be expected to show more paraphasias than spontaneous speech, because paraphasias have a wider selection of alternative words. Other investigators have defined the problem as one of decoding. Stengel and Lodge-Patch³⁰ regarded conduction aphasia as an incomplete form of receptive aphasia with a decreased ability to focus attention on words. Luria and Hutton³¹ hypothesized a failure to attend to the phonologic structure of words sufficiently to analyze their structures. Warrington and Shallice³² suggested that selective impairment of auditory verbal short-term memory produced the repetition disturbance, and Tzotzis and Albert³³ proposed a specific impairment of memory for sequences.

Strub and Gardner³⁴ suggest that the repetition deficit actually occurs between these major processes, several steps after phonologic processing but before phonemic encoding. This formulation appears neurolinguistically more satisfactory¹⁰ and is consistent with an interruption of transmission of correctly decoded speech from the posterior comprehension area to the anterior speech encoding region.

One assumption critical for use of the disconnection theory in our three atypical cases is the presence of auditory language comprehension ability in the right hemisphere. This is in accord with neuropathologic observations of conduction aphasia with severe involvement of Wernicke's area, Heschl's gyrus, and surrounding structures in the left hemisphere.^{4,22} In the left-handed there is considerable evidence for a right hemisphere role in language, particularly in auditory processing.^{35,36} Most patients whose cases were reported in the literature conforming to Kleist's¹⁴ formulation were either left-handed or had a family history of sinistrality.

There is further evidence for a right hemisphere role in comprehension.

Adult patients who have undergone left hemispherectomy show better preservation of comprehension than fluency,³⁷ and a similar pattern has been reported in patients with large left hemisphere strokes.³⁸ Kanschepolsky et al³⁹ reported the case of an individual who developed conduction aphasia following a left hemisphere lesion and then pure-word deafness after a subsequent lesion in the right temporal area. Damasio and Damasio⁴⁰ have shown right ear extinction with dichotic listening in conduction aphasia. Studies on patients who have undergone corpus callosum commissurotomy also demonstrate some degree of comprehension in the right hemisphere.⁴¹ Rasmussen and Milner's⁴² data, using amobarbital sodium injected unilaterally into one carotid artery to selectively anesthetize one hemisphere (Wada test), revealed that among 262 patients (right-handed, left-handed, or mixed) without evidence of early damage to the left hemispheres, 16% had evidence of right hemisphere or bilateral language representation. In those cases with bilateral representation, language testing demonstrated a dissociation of naming from repetition in half with one language function in one hemisphere and the other in the opposite hemisphere. Kinsbourne,⁴³ performing Wada testing of a right-handed patient with conduction aphasia, induced complete interruption of language with a right carotid injection. It could be suggested that the atypical conduction aphasia of patient 3 represented a disconnection of interhemispheric pathways from a right temporal language reception area to the more routine left frontal motor speech area.

At least six other cases of crossed conduction aphasia are recorded in the literature.⁴⁴⁻⁴⁷ It is remarkable that, although most patients with crossed aphasia are nonfluent with initial mutism and agrammatism,⁴⁸ the predominant type of fluent crossed aphasia has been the conduction type.⁴⁹ We are aware of only one neuropathologically proved case of crossed Wernicke aphasia.⁴⁹ These findings are not likely to be simply due to the proportional occurrence of the aphasic syndromes in dextrals with total homologous language representation in the right hemisphere. There seems to be a specific tendency to develop crossed conduction aphasia in cases of fluent aphasia from right-hemisphere lesions as predicted by disconnection theory.

"Atypical" conduction aphasia,

then, is a syndrome with fluent paraphasic verbal output, relatively good auditory comprehension, and significantly disturbed ability to repeat with variable sites of abnormality inconsistent with the arcuate fasciculus interruption model. Atypical conduction aphasia apparently occurs when language comprehension is at least partially subserved by the right hemisphere. The variety of sites of abnormality for atypical conduction aphasia,

both as reported in the literature and as demonstrated by our three cases, the neurolinguistic data, and the evidence for right-hemisphere auditory language comprehension, are all harmonious with the original mechanism postulated by Wernicke, a separation of auditory language comprehension and motor language output areas. Finally, the absence of an apraxia in our three cases, showing that the ability to carry out verbally

commanded motor acts can be successful even though the patient fails to repeat spoken language, supports the existence of more than one route from auditory comprehension to anterior motor areas. Atypical conduction aphasia can be explained by the disconnection concept, which remains the most tenable model for this language disturbance.

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