THE ANATOMICAL BASIS OF CONDUCTION APHASIA

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INTRODUCTION

THE concept of conduction aphasia has been central to the understanding of language disorders since the early writings of Wernicke (1874) and the controversy regarding its definition, anatomical basis and pathophysiological mechanism has continued to our times. But while the current definition provided by the Boston school of aphasia (Geschwind, 1965; Goodglass and Kaplan, 1972), seems to have clarified the question of the existence and clinical essence of the syndrome, Geschwind's interpretation of the disturbance as a disconnection syndrome (1965) has not been universally accepted (see Warrington and Shallice, 1969; Brown 1972). Such controversy often obscures the fact that the pathological substrate on which the disconnection hypothesis is based never has been challenged and appears fundamentally correct whatever stand you take on the issue of interpreting its functional significance. In this article we present anatomical findings in six cases of conduction aphasia, obtained with a method for localization of lesions in computerized tomography scans (Damasio and Damasio, 1979; Damasio, Damasio, Hamsher and Varney, 1979). We reconsider the anatomical correlates of conduction aphasia and discuss their bearing on the production of the syndrome.

METHOD

Neuropsychological Assessment

Patients were given the MAE (Multilingual Aphasia Battery; Benton, 1969; 1976) which consists of a series of standardized and validated tests (for example, Visual Naming, Sentence Repetition, Aural Comprehension, Reading Comprehension, Token Test). The diagnosis of conduction aphasia was made on the basis of results obtained in those tests but according to the Boston Diagnostic Classification of the Aphasias (Goodglass and Kaplan, 1972). The operational definition of conduction aphasia was: an aphasic syndrome with fluent, meaningful speech (in which phonemic paraphasias predominate over global ones), profound verbal repetition defect (with either entire blocking of repetition or repetition with phonemic paraphasias), partial or complete preservation or repetition of words which stand for digits, and preserved or minimally impaired aural comprehension defect. Additional neuropsychological evaluation was carried out with standardized tests of the Benton Laboratory of Neuropsychology.

Anatomical Study

Computerized tomography was performed by EMI scanners (Mark III or Whole Body Scanner). The anatomical study was conducted on the basis of CT scan films of consecutive parallel cuts with a depth of 0.8 cm.

Templates of the cuts as described previously (Damasio and Damasio, 1979; Damasio et al., 1979), were prepared on the basis of brain sections and of available atlases of computerized tomography and of neuroanatomy. In the standard template of each consecutive level, cortical and subcortical areas of special interest in terms of higher nervous function were indicated by the numbers of Brodmann's map or by appropriate initials. By means of identifiable anatomical landmarks, lesions were plotted on the best fitting template and the results of the record read as a three-dimensional reconstruction.

SUBJECTS

Six patients with conduction aphasia due to stroke were selected. Only patients who could be evaluated within three months of the onset of aphasia and who were native English speakers were included in the study. All patients had right-handed ancestry and all but one were right-handed. Age range was 35 to 55 years and educational level varied between 10 and 16 years. Pertinent details of each case are summarized below.

Case 1

The patient is a 43-year-old man, right-handed, college graduate and owner/manager of a factory. In December 1978, he suffered a thrombotic stroke which produced aphasia, right central facial paralysis and right brachial paralysis. The facial and brachial weakness improved remarkably over the ensuing weeks but aphasia persisted. Speech was fluent and entirely meaningful but with occasional phonemic paraphasias generally recognized as errors by the subject. Repetition of digits was normal (Forward Digit Span = 7) but repetition of verbal material was severely impaired with phonemic paraphasias and omission of phonetic and lexical components (in the MAE Sentence Repetition Test he was unable to repeat a single sentence correctly, e.g. nil percentile). Word finding ability was defective, though less so than repetition (the patient scored in the 18th percentile in the MAE Visual Naming Test, which was low normal for his age and education). Aural comprehension was normal for simple conversational items but impaired for syntactically organized material. In the MAE Test for Aural Comprehension of Words and Phrases he scored in the 20th percentile, low normal for his age and education. The Token Test was defective (he scored 27/44). Reading comprehension was mostly normal (59th percentile in the MAE Test of Reading Comprehension of Words and Phrases) but reading aloud was marred by phonemic paraphasias. Recall of recent and remote events was excellent as were calculations and visuospatial abilities. Dichotic word listening revealed a complete extinction of the right channel.

Case 2

The patient is a 45-year-old woman, right-handed, a housewife with a ten-year education. In March 1978, she suffered a thrombotic stroke which produced an aphasia, right central facial paralysis and right brachial paralysis. Speech was fluent and meaningful. The major disturbance was in repetition of words and sentences, particularly the latter, with frequent blocking and phonemic paraphasias (in the MAE Sentence Repetition Test she was profoundly defective, e.g. nil percentile). Repetition of digits was normal (Forward Digit Span = 7). Word finding ability was within normal range (in the MAE Visual Naming Test she scored in 75 per cent percentile) as was aural

comprehension for syntactically complex items but not for simple conversational items (in the MAE Test for Aural Comprehension of Words and Phrases she scored in the 20th percentile). The Token Test was defective (34/44). Reading comprehension was normal but reading aloud elicited numerous literal paraphasias. Memory was excellent in all spheres. Constructional praxis and calculation were normal. Dichotic word listening revealed a complete extinction of the right channel.

Case 3

The patient is a 47-year-old man, left-handed but with a dextral family background, a high school graduate. In November 1976, he suffered a thrombotic stroke and as a result had aphasia and right-sided paralysis of the face and upper extremity. The latter cleared while aphasia persisted. The patient was always fully orientated and with fluent meaningful speech, his major difficulty being verbal repetition of words and sentences but not of digits (in the MAE Sentence Repetition Test the patient repeated none but the simplest phrase, a score of 1, below the 2nd percentile; he could repeat five digits forward). Aural and reading comprehension were impaired for complex material but preserved otherwise. (In both the MAE Test for Aural Comprehension of Words and Phrases and the MAE Test for Reading Comprehension of words and phrases he scored in the 75th percentile). The Token Test was defective (he scored 32/44). Written calculation and visuospatial abilities were preserved.

Case 4

The patient is a 35-year-old woman, right-handed, college graduate. In April 1979, she suffered a thrombotic stroke which produced aphasia, right central facial paralysis and right brachial paralysis. Spontaneous and conversational speech was fluent and meaningful but with frequent phonemic paraphasias and occasional global paraphasias. Repetition of words and sentences was severely impaired with profuse phonemic paraphasias. Often repetition was entirely blocked and when attempted, phonetic and lexical components were omitted. At the time of admission her score in the MAE Sentence Repetition Test was 0 but then improved to 1 and 2 (three word sentences). Her Digit Span Forward initially was 6 and then improved to 7. Aural and reading comprehension were normal (in both the Aural and Reading Comprehension Tests of the MAE, the patient performed in the 65th percentile) but reading aloud was disturbed by phonemic paraphasias and omission of lexical elements. The Token Test was mildly defective (she scored 40/44). Calculation, constructional ability and general memory functions were normal.

Case 5

The patient is a 46-year-old woman, right-handed. In October 1976, she suffered a hæmorrhagic stroke. At the time she had a ten-year history of untreated systemic hypertension. After an initial period of diminished alertness and confusion she was noted to have a fluent aphasia with severely impaired verbal repetition for words and sentences but not for digits. Paraphasias were mostly phonemic but some were verbal as well and occurred frequently. Aural and reading comprehension were impaired although she was able to understand the less complex stimuli. As spontaneous speech and comprehension improved the pronounced repetition defect stood out more clearly. Her score in the MAE Sentence Repetition Test was 0. Her Digit Span Forward was 4.

Case 6

The patient is a 55-year-old woman, right-handed, a secretary. In August 1979, she suffered a stroke which manifested itself by sudden onset of difficulty in finding words and a mild paresis of the right arm and right side of the face. Her difficulty in word finding cleared and at the time of admission, other than for occasional phonemic paraphasias, her speech had normal content, articulation and rate. But her repetition, other than for digits, was severely impaired (MAE Sentence Repetition Test in nil percentile; Digit Span = 6). Aural and reading comprehension were intact (in both the Aural and Reading Comprehension Tests of the MAE she scored in the 65th percentile). Calculation, constructional ability and general memory function were normal.

In sum, these 6 patients best fit the diagnosis of conduction aphasia, given the fluency and meaningfulness of their speech, the marked defect in verbal repetition, and the comparatively preserved aural comprehension which allowed them to grasp the meaning of sentences which they could not repeat. The scores obtained in the MAE validate the clinical impression.

ANATOMICAL STUDY

Case 1

The CT scan shows an area of decreased density encompassing four consecutive cuts. Plotting of this area on the corresponding templates suggests that it involves the insular cortex and some white matter directly subjacent to it (fig. 1). The lesion extends laterally and posteriorly to involve structures of the temporal lobe particularly in the supratemporal plane, namely, part of cortical areas 22, 41 and 42, as well as white matter directly underlying them. Although the lesion does not damage the parietal lobe extensively it involves the cortex of the parietal operculum lying over the supratemporal plane (areas 3, 1, 2 and 40) as well as its corresponding white matter.

Case 2

The CT scan shows an area of decreased density which encompasses six consecutive cuts. Plotting of this area suggests that it involves the insula in most of its extent compromising its cortex and underlying white matter. It extends into the temporal lobe (areas 22, 41 and 42 and subjacent white matter) and also involves parietal cortex (area 40 and subjacent white matter). (See fig. 2).

Case 3

The CT scan shows an area of decreased density which encompasses four consecutive cuts. The area involves the insula in most of its extent, cortically and subcortically, and extends into the temporal lobe compromising a portion of area 22, 41 and 42 and subjacent white matter. It extends also into area 40 and corresponding white matter (fig. 3).

Case 4

The CT scan shows an area of decreased density located in approximately the same region involved in the three previous cases (fig. 4). A significant difference regards the amount of involvement of the insular region, in this case limited to its posterior portion. The involvement of the auditory complex is similar to the other cases as is the compromise of the supramarginal gyrus which is quite pronounced.

Case 5

The CT scan shows an area of increased density located deep in the basal ganglion region of the left hemisphere and encompassing three consecutive cuts. This area corresponds to the lenticular nucleus, mainly the putamen, and involves white matter underlying the insula and possibly its cortex as well as that of areas 41 and 42. Areas 22 and 40 do not appear to be directly involved but are in all likelihood displaced due to the space-occupying nature of the lesion while their white matter is undercut by the lesion (fig. 5).

Case 6

The CT scan shows areas of decreased density in the insular region, the temporal lobe (areas 22, 41 and 42 and subjacent white matter) and also in the parietal cortex (area 40 and subjacent white matter) (fig. 6).

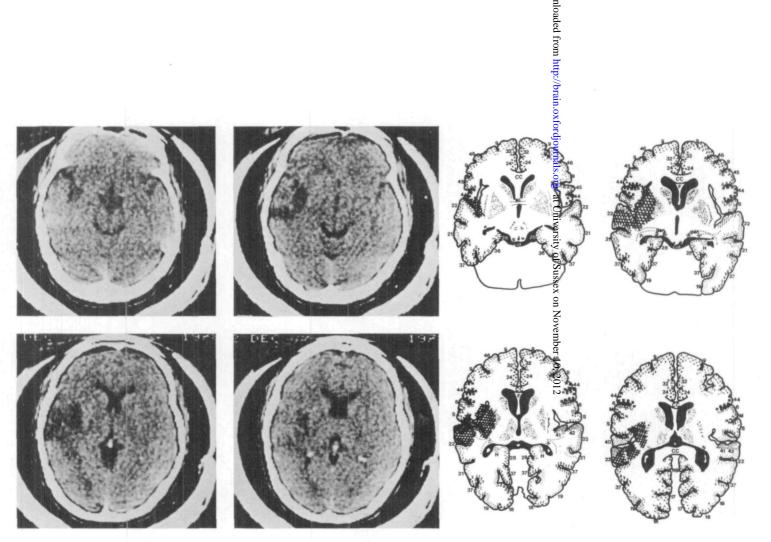


FIG. 1. CT scan and templates of Case 1. Note involvement of the left insula, auditory areas and supramarginal gyrus.

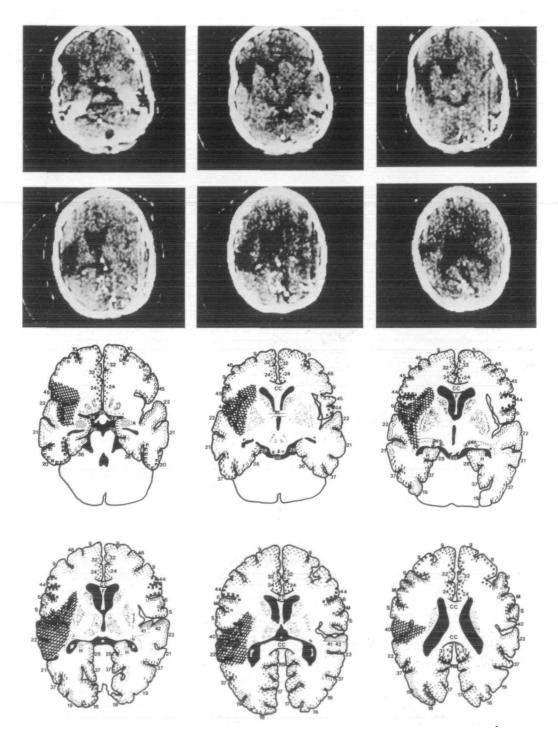
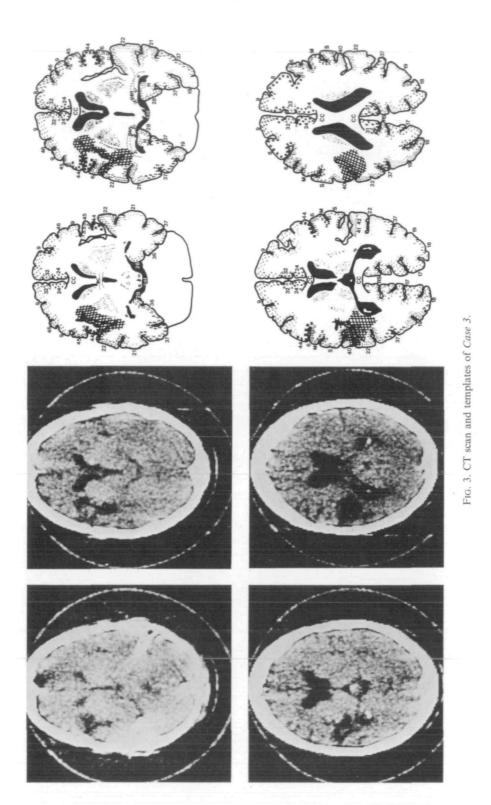


FIG. 2. CT scan and templates of Case 2. Note the similarity with the lesions in Cases 1 and 3.



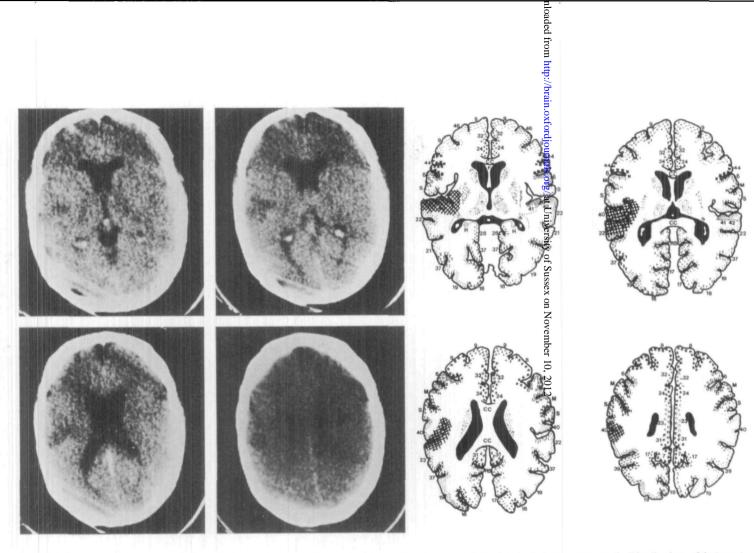


FIG. 4. CT scan and templates of *Case 4*. In comparison with previous cases, involvement of the insula is less pronounced. Distribution of lesions is otherwise comparable.

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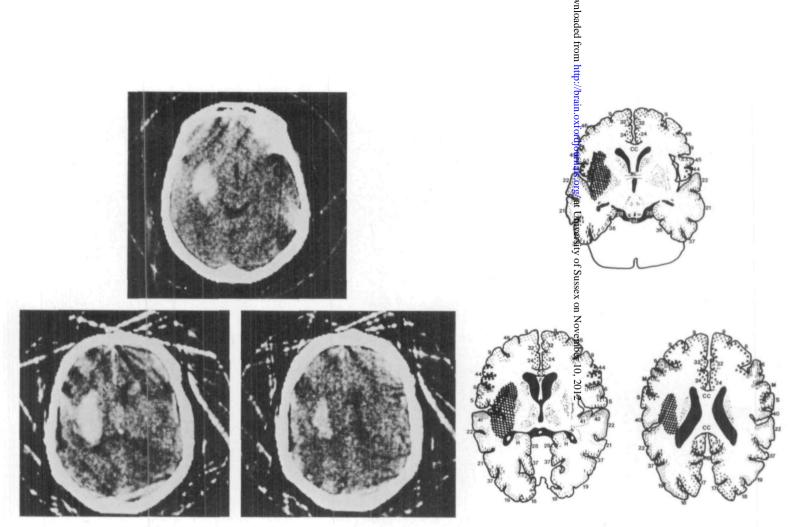


FIG. 5. CT scan and templates of Case 5. The area of increased density corresponds to white matter underlying insular, auditory and supramarginal cortex.

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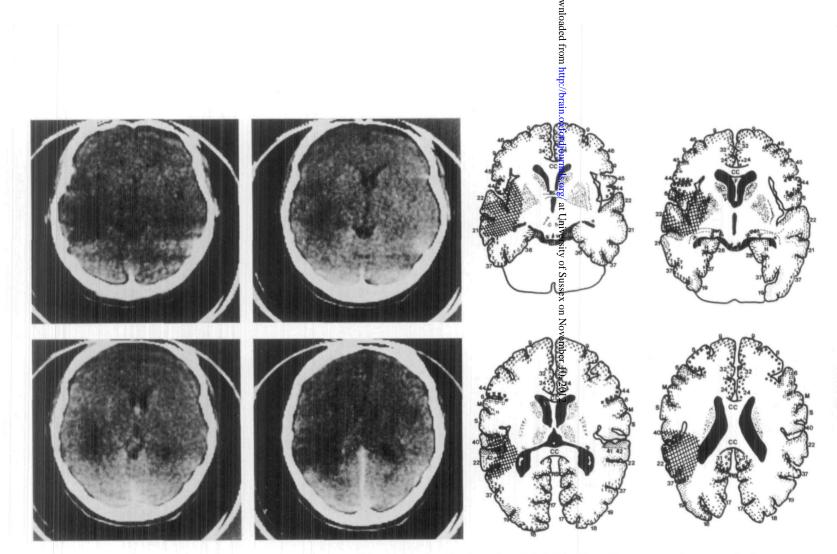


FIG. 6. CT scan and templates of Case 6. The areas of decreased density are placed in the region of the left insula, auditory areas and supramarginal gyrus.

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In summary, 5 cases show compromise of the insular region, and 5 cases show variable degree of involvement of the superior temporal gyrus and an extension of the lesion into the inferior parietal region. In all cases the lesion is deep and compromises the white matter corresponding to the aforementioned regions. In no case is there involvement of the angular gyrus (area 39), or of the posterior portion of the second and third temporal gyri (area 37) or of Broca's area (areas 44 and 45). These neuroradiological findings are consonant with those reported by Kertesz, Harlock and Coates (1979).

DISCUSSION

Consideration of the above findings supports the notion that conduction aphasia is associated with a highly distinctive structural pattern, and that damage to the arcuate fasciculus is strongly associated with the syndrome. As defined in classical terms, that pathway originates in the cortex of the supratemporal plane, arches around the back end of the sylvian fissure and courses in the superior longitudinal fasciculus over the extreme capsule and insula, to reach the lower frontal region. There is little doubt that, in one way or another, at its origin or while coursing in the parietal operculum, the pathway is damaged in our cases. But our data also show that with the exception of Case 5, damage was not confined to the arcuate fasciculus as it involved the left auditory complex, the left insular region and the supramarginal gyrus also.

These findings merit special attention. Let us consider the ones related to the auditory complex first. The lesion In Wernicke's area was not as extensive or posteriorly placed as that seen in our cases of Wernicke's aphasia in which the posterior portion of the supratemporal plane, the parietal component of area 22 (as defined by Galaburda, Sanides and Geschwind, 1978) and part of areas 39 and 37 are always involved (Damasio et al., 1979). On the other hand, a part of areas 41 and 42 appeared consistently compromised in the conduction cases, and that seems to be an inconstant feature of Wernicke's aphasia (Damasio et al., 1979). The finding of involvement of the auditory complex is in line with the majority of pathological reports in the literature. Its replication suggests that conduction aphasia should probably be defined, in anatomical terms, by the pattern of intactness or damage of certain portions of the left auditory complex as much as by damage to the left arcuate fasciculus. In fact, since damage to both auditory complex and arcuate fasciculus is shared by other aphasias (for example, Wernicke's, Broca's) it is the specific pattern of compromise to either structure that renders conduction aphasia distinctive, anatomically and behaviourally.

The finding of damage to the insula is thought-provoking. In recent years damage to this region has not been considered a pertinent component of the anatomical basis of conduction aphasia. For instance, in a report on conduction aphasia (Sheremata, Andrews and Pandya, 1974) a lesion in the insula, extreme

capsule, claustrum and external capsule was noted but the syndrome was interpreted as resulting from another lesion which damaged the more superiorly placed rostral arcuate fasciculus. In their review of the anatomical findings in conduction aphasia, Benson, Sheremata, Bouchard, Segerra, Price and Geschwind (1973) emphasized the importance of damage to three main structures: (1) Wernicke's area, a lesion of which was present in their Case 2 and in almost all of the few cases previously recorded; (2) the supramarginal gyrus, damaged in their Cases 1 and 3; and (3) the arcuate fasciculus, damaged deep underneath the supramarginal gyrus as could be inferred from the distribution and amount of white matter destruction in Cases 1 and 3. On discussing possible mechanisms for the genesis of conduction aphasia they suggested that two mutually exclusive anatomical patterns appear capable of producing the syndrome: (a) the supramarginal gyrus/arcuate fasciculus lesion, and (b) the auditory region lesion. They did not comment on the role of damage to the insula which was involved in their Case 2. By comparison our cases best resemble their Case 2 but with elements of Cases 1 and 2, suggesting that a combination of the two basic anatomical patterns proposed by Benson et al. (1973), coupled with damage to the insula also occurs in association with the syndrome. Such a combination was also present in the well documented cases of Goldstein (1911; 1948) and of Kleist (1962) and in the case of Voisin (1912).

The exact functional significance of damage to the insula may not be easy to settle. Wernicke initially predicted that the insula would be the locus for the 'sensorimotor' disconnection underlying conduction aphasia (1874) and the case of Lichtheim (1885) supported that view. But by 1908 he felt the insula was not crucial and the case of Liepmann and Pappenheim (1914), a conduction aphasia in which the insula was spared, now supported his new opinion (he maintained, however, that the syndrome resulted from disconnection although, by then, following Von Monakow, he had turned to the arcuate fasciculus as the responsible 'disconnecting' structure). Goldstein, on the other hand, again related the insula to conduction aphasia ('central aphasia' in his nomenclature) though he rejected the disconnective interpretation proposed by Wernicke. It could be argued that the finding is incidental, an addition to the more crucial damage in the arcuate fasciculus and auditory cortex. We suspect, however, that damage to the insula and to its underlying matter can be a pertinent factor in the production of the syndrome. More than the insular cortex itself, the decisive pathological element must be the destruction of white matter in the extreme capsule which, most certainly, contains vital cortico-cortical connections linking temporo-parietal structures with frontal pre-motor structures. D. N. Pandya (1979) has demonstrated that connections from auditory cortex to the pre-motor frontal lobe travel in the extreme capsule, underneath the insular cortex. Such a course is in fact quite logical although classic anatomical texts always portray those connections as part of the superior longitudinal fasciculus, travelling above the extreme capsule and insular cortex. The latter concept is derived from dissection anatomy and appears

incorrect, being accurate only in regard to connections of the most posterior aspect of the auditory association cortex but not in relation to the remainder of the auditory complex. In sum, it appears that damage to a specific set of temporoparietal connections, be it in the parietal operculum or underneath the insula, can be associated with impaired repetition, just as the relative intactness of Wernicke's area can be associated with considerable sparing of aural comprehension.

Anatomical data may help resolve the issue of behavioural interpretation. The variety of structures potentially involved makes it necessary to cast doubts on explanations based on a single disruptive factor (for example, mnestic defect). The act of immediate oral repetition of a heard sentence presupposes a variety of operations: phonological identification, immediate memory for phonemes and for their sequence, and open access to an intact sensorimotor effector. In all like-lihood, those steps depend on function of the left auditory cortex and of its connections to lower parietal and frontal cortex and possibly to the basal ganglia also. As we have just seen, the anatomical patterns associated with conduction aphasia are such that in any given instance more than one functional defect may be at play, for instance, impairment in immediate memory for phonemes combined with impaired access to a sensorimotor effector.

The issue of whether abnormal repetition with phonemic paraphasias is the result of left or right hemisphere function (raised by Liepmann and Pappenheim (1914)) and discussed by Goldstein (1948), Kleist (1962), and Kinsbourne (1971), can not be satisfactorily resolved by anatomical data. But the remarkable preservation of a considerable portion of left speech area seen in most cases would probably support the contention that whatever abnormal linguistic production is available can be the result of residual left hemisphere function, even if auditory information would have to arrive in the right auditory cortex first and from there be transferred to left auditory association cortex. Finally, the present anatomical evidence supports, in good measure, both Wernicke's original prediction and Geschwind's now classical anatomical account of the syndrome.

REFERENCES

- BENSON, D. F., SHEREMATA, W. A., BOUCHARD, R., SEGARRA, J. M., PRICE, D. and GESCHWIND, N. (1973) Conduction aphasia: a clinicopathological study. Archives of Neurology, Chicago, 28, 339-346.
- BENTON, A. L. (1969) Development of a multilingual aphasia battery: progress and problems. Journal of the Neurological Sciences, 9, 39-48.
 - ----- (1976) Multilingual Aphasia Examination. University of Iowa.

BROWN, J. (1972) Aphasia, Apraxia and Agnosia. Springfield: Charles C. Thomas, chapter 5.

- DAMASIO, H. and DAMASIO, A. (1979) 'Paradoxic' ear extinction in dichotic listening: possible anatomic significance. *Neurology, Minneapolis*, 29, 644-653.
 - HAMSHER, K. and VARNEY, N. (1979) CT scan correlates of aphasia and allied disorders. *Neurology, Minneapolis*, 29, 572.
- GALABURDA, A. M., SANIDES, F. and GESCHWIND, N. (1978) Human Brain. Cytoarchitectonic leftright asymmetries in the temporal speech region. Archives of Neurology, Chicago, 35, 812-817.
- GESCHWIND, N. (1965) Disconnexion syndromes in animals and man. Brain, 88, 237-294 and 585-644.
- GOLDSTEIN, K. (1911) Uber die amnestische und centrale aphasie. Archiv für Psychiatrie und Neurologie, 48, 408.
 - (1948) Language and Language Disturbances. New York: Grune and Stratton, chapter VII.
- GOODGLASS, H. and KAPLAN, E. (1972) The Assessment of Aphasia and Related Disorders. Philadelphia: Lea and Febiger.
- KERTESZ, A., HARLOCK, W. and COATES, R. (1979) Computer tomographic localization, lesion size, and prognosis in aphasia and nonverbal impairment. *Brain and Language*, **8**, 34-50.
- KINSBOURNE, M. (1971) 'Conduction aphasia'. Read before the annual meeting, American Academy of Neurology, New York.
- KLEIST, K. (1962) Sensory Aphasia and Amusia. London: Pergamon Press, chapter 3.
- LICHTHEIM, L. (1885) On aphasia. Brain, 7, 433-484.
- LIEPMANN, H. and PAPPENHEIM, M. (1914) Uber einem fall von sogenannter Leitungsaphsie mit anatomischer befund. Zeitschrift für die Gesamte Neurologie und Psychiatrie, 27, 1-41.
- PANDYA, D. N. (1979) Personal communication. Observations to be published.
- SHEREMATA, W. A., ANDREWS, R. and PANDYA, D. N. (1974) Conduction aphasia from a frontal lobe lesion. In: Transactions of the American Neurological Association, 99, 249-252.
- VOISIN, R. (1912) Cited by Goldstein, K. (1948), p. 240.
- WARRINGTON, E. K. and SHALLICE, T. (1969) The selective impairment of auditory verbal short-term memory. *Brain*, 92, 885-896.

WERNICKE, C. (1874) Der Aphasische Symptomencomplex. Breslau: Cohn and Weigert.

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