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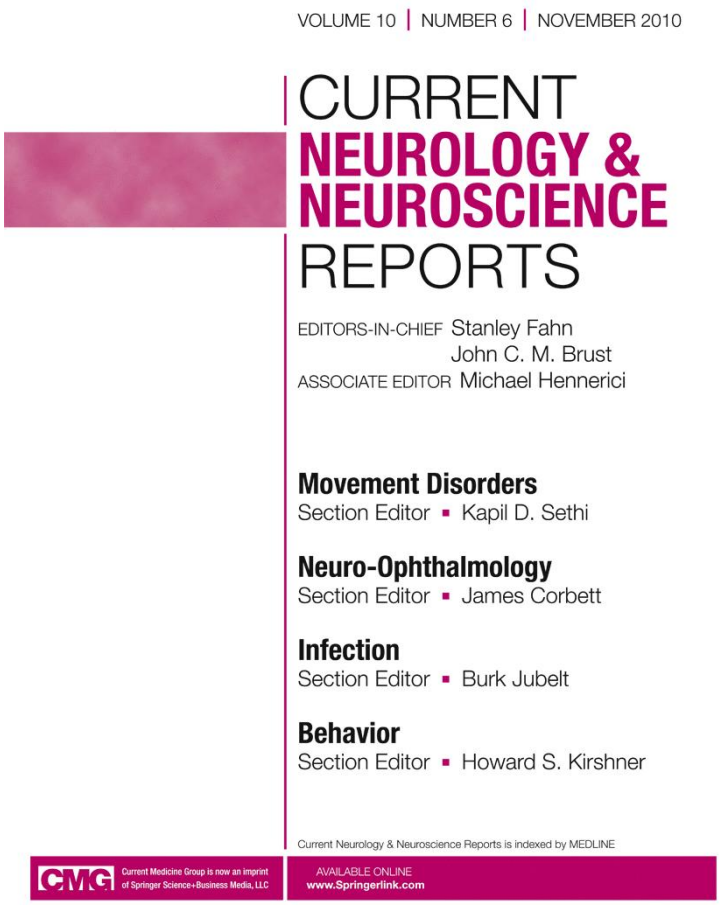

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A Review of Conduction Aphasia

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Abstract In this paper, a historical overview of the interpretation of conduction aphasia is initially presented. It is emphasized that the name conduction aphasia was proposed by Wernicke and was interpreted as a disconnection between the temporal and frontal brain language areas; this interpretation was re-taken by Geschwind, attributing the arcuate fasciculus the main role in speech repetition disturbances and resulting in the so-called Wernicke-Geschwind model of language. With the introduction of contemporary neuroimaging techniques, this interpretation of conduction aphasia as a disconnection syndrome due to an impairment of the arcuate fasciculus has been challenged. It has been disclosed that the arcuate fasciculus does not really connect Wernicke's and Broca's areas, but Wernicke's and motor/premotor frontal areas. Furthermore, conduction aphasia can be found in cases of cortical damage without subcortical extension. It is concluded that conduction aphasia remains a controversial topic not only from the theoretic point of view, but also from the understanding of its neurologic foundations.

Keywords Conduction aphasia · Arcuate fasciculus · Language repetition · Parietal lobe

Introduction

Conduction aphasia is usually defined as a language disturbance characterized by relatively fluent (yet paraphasic) spontaneous speech, intact auditory comprehension, and poor speech repetition [1–7]. In his classic aphasia diagram, Wernicke [8] assumed that a disconnection between the two major brain speech systems (motor and sensory language areas) would result in a unique disturbance, which he named *Leitungsaphasie* (conduction aphasia). He also described the first case of conduction aphasia. The name "conduction aphasia" was accepted, and it became extensively used during the following decades.

During the mid-20th century, some attempts to re-interpret conduction aphasia are found. Goldstein [3] considered that conduction aphasia was the result of a central, core language disturbance and proposed the term "*central aphasia*." He supposed that conduction aphasia may be explained as a disturbance in inner speech, "the central phenomenon of instrumentalities of speech." Luria [9, 10] used the name "afferent (or kinesthetic) motor aphasia" assuming conduction aphasia to be rather interpreted as a segmental ideomotor apraxia; according to him, paraphasias in conduction aphasia are articulatory-based (i.e., they are errors in the articulatory patterns of the phoneme productions, or articulemes), not really phonological abnormalities. Nonetheless, the name conduction aphasia remained in use by most researchers in the area.

Benson et al. [11] pinpointed the major characteristics of conduction aphasia. They proposed that conduction aphasia presents three basic and five secondary characteristics. The three basic characteristics are: 1) fluent conversational language; 2) almost normal comprehension; and 3) significant impairments in repetition. Secondary characteristics include: 1) impairments in naming; 2) reading impairments; 3) variable writing difficulties (apraxic agraphia); 4) ideomotor apraxia; however, ideomotor apraxia is not associated with temporal lobe lesions; and 5) additional neurologic impairments. Bartha and Benke [7] reported that conduction aphasia patients

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present as relatively homogeneous in their aphasic manifestations: severe impairment of repetition and fluent expressive language functions with frequent phonemic paraphasias, repetitive self-corrections, word-finding difficulties, and paraphrasing. Repetitive self-corrections frequently result in the so-called *conduit d'approche*.

The possibility of several mechanisms, each of which is capable of giving rise to deficient repetition, led to the postulation of two different forms of conduction aphasia named as efferent and afferent [4, 5]; or reproduction and repetition [12, 13]; or supra- and infrasylvian [14]; or simply parietal and temporal [7]. The efferent-reproduction type involves the phonemic organization and representation of words and is correlated with parietal and insular damage, whereas the afferent-repetition conduction aphasia involves short-term memory defects and affects the repetition of large strings of material [15]. This second subtype of conduction aphasia has been described more frequently with lesions of the temporal lobe [16].

Of note, language repetition impairments are not restricted to conduction aphasia and can be observed in different aphasia syndromes. Ardila and Rosselli [17] analyzed 38 aphasic patients divided into six groups (transcortical motor, Broca's, conduction, Wernicke's, anomic, and global aphasia). Repetition errors were generally associated with perisylvian aphasias (Broca's, conduction, and Wernicke's). However, in all aphasic groups some repetition errors were observed. These errors were not only quantitatively but also qualitatively different. It was concluded that, depending on the specific repetition task, errors may be evident or unnoticed in a particular aphasic group. The authors proposed that different mechanisms may underlie repetition deficits in aphasia: limitation of auditory-verbal short-term memory, difficulties at the level of phonological production, impairments in phoneme recognition, and semantic and syntactic comprehension.

Conduction Aphasia as a Disconnection Syndrome

Since Wernicke [8], conduction aphasia has usually been interpreted as a result of a disconnection between the superior temporal gyrus (Wernicke's area) and the inferior frontal gyrus (Broca's area). Wernicke's interpretation was supported by Geschwind in the 1960s (the so-called Wernicke-Geschwind model of language), who put it in terms of modern anatomic nomenclature, attributing to the arcuate fasciculus the main role in the speech repetition disturbances. According to Geschwind [18], disconnection syndromes were higher function deficits that resulted from white matter lesions or lesions of the association cortices; conduction aphasia was usually presented as the prototypal example of a disconnection syndrome. Since then, it has been generally assumed that, in conduction aphasia, the lesion affects the arcuate fasciculus [19, 20] and sporadically an indirect pathway passing through the inferior parietal cortex [21••].

With the introduction of contemporary neuroimaging techniques, the emphasis in disconnection began to decrease, and the focus of interest moved to the functional systems involved in different intellectual functions, including language [22]. More recently, new techniques that study connections in the living human brain allow testing the applicability of the disconnection model to different syndromes, including conduction aphasia. MRI techniques that allow complex mathematical data analysis, such as diffusion tensor imaging and computer-based postprocessing, have made fiber tracking possible [23, 24]. It is understandable that one of the tracts that has received the most attention is the arcuate fasciculus, due to its potential implication on speech and language brain organization [25•].

Neuroimaging Studies of Conduction Aphasia

The arcuate fasciculus is a brain association tract composed of arched fibers that is assumed to connect the posterior temporal language understanding area (Wernicke's area) and the anterior frontal language production area (Broca's area). The arcuate fasciculus is the main part of a larger tract located lateral to the corticospinal tract, known as the superior longitudinal fasciculus**.** The superior longitudinal fasciculus includes four different types of connecting fibers [26], all of them with a frontal terminus in the posterior part of the frontal lobe. However, their origin is different: the superior horizontal bundle originates in the parietal lobe; two more bundles originate in the angular and supramarginal gyri; and the inferior portion includes long arched fibers originating in the ipsilateral superior and middle temporal gyri. Several authors [24, 27•] refer to two major anteroposterior connections of the superior longitudinal fasciculus, namely the horizontal bundle (parieto-opercular), and its arched part (the arcuate fasciculus) constituting the bulk of the superior longitudinal fasciculus.

The really crucial question becomes: Is it invariably the arcuate fasciculus affected in cases of conduction aphasia? To answer this apparently simple question, however, is not so easy. One major limitation in the conduction aphasia cases reported with arcuate fasciculus lesions is that they are usually due to infarcts and tumors, and therefore, lesions are not limited within the arcuate fasciculus boundaries. It is not easy to find cases of conduction aphasia with a lesion limited to the arcuate fasciculus. Although the arcuate fasciculus is often reported to be involved in conduction aphasia [19, 20, 28], cortical lesions alone without subcortical extension may also

produce conduction aphasia [29, 30]; furthermore, patients with lesions of the arcuate fasciculus may retain the language repetition ability [31, 32], suggesting that the arcuate fasciculus is not crucial for language repetition.

Thus, the interpretation of conduction aphasia as a disconnection syndrome due to an impairment of the arcuate fasciculus has recently been challenged. Nonetheless, new studies have continued pointing to a central role of the arcuate fasciculus in conduction aphasia. For example, Zhang et al. [33] selected 10 individuals with conduction aphasia and 10 normal volunteers and analyzed their arcuate fasciculus using diffusion tensor imaging; fractional anisotropy values were obtained. Then, the results of the left hemisphere were compared with those of the right hemisphere, and the results of the conduction aphasia cases were compared with those of the volunteers. It was found that there were significant differences in the fractional anisotropy values between the left and right hemispheres of volunteers and conduction cases. In the volunteers, there was an increase in fiber in the left hemisphere compared with the right hemisphere, whereas there was an increase in fiber in the right hemisphere compared with the left hemisphere in conduction aphasia patients. The results of diffusion tensor tractography suggested that the configuration of the arcuate fasciculus was different between conduction aphasia patients and normal volunteers, suggesting that there was damage to the arcuate fasciculus in conduction aphasia cases. The authors concluded that a disconnection between Broca's area and Wernicke's area is likely to be one mechanism of conduction aphasia repetition impairment.

Some Recent Advances

Bernal and Ardila [34••] observed that contemporary research supports the following: 1) unusual cases of interhemispheric dissociation of language lateralization (e.g., Broca's area in the left and Wernicke's area in the right hemisphere) can be found without evident repetition defects; 2) electrocortical studies have found that the arcuate fasciculus not only transmits information from temporal to frontal areas, but also in the opposite direction [35]; 3) transferring of speech information from the temporal to the frontal lobe uses not only one but two different streams (the arcuate fasciculus and an indirect pathway passing through the inferior parietal cortex) [21••]; and 4) conduction aphasia can be found in cases of cortical damage without subcortical extension [30]. Together, all these observations conclude that the arcuate fasciculus is not required for repetition, but it could have a subsidiary role in it. Bernal and Ardila [34••] further propose a new language network model emphasizing that the arcuate fasciculus connects posterior brain areas with Broca's area via a relay station in the premotor/motor areas (BA6 and BA4). Thus, the connection with Broca's area would not be direct, but indirect.

Bernal and Altman [36••] reported the data and subsidiary results of 12 normal right-handed volunteers who participated in a diffusion tensor imaging study. The superior longitudinal fasciculus fibers were obtained bilaterally, placing an area of interest at the triangular-shaped region lateral to each of the corticospinal tracts in a coronal plane along the rostral aspect of the corpus callosum. A sagittal fractional anisotropy image was used to determine the rostral end point of the superior longitudinal fasciculus in the white matter pertaining to a specific frontal lobe area. The superior longitudinal fasciculus projection to Broca's area was ranked qualitatively (none, minimal, most, or all). It was found that the superior longitudinal fasciculus (SLF) projection to Broca's areas was absent in seven subjects (58.3%) and minimal in five (41.6%). The SLF's rostral end points were found uniquely or mainly in the precentral gyrus in 100% of cases. The authors concluded that the superior longitudinal fasciculus connects the posterior language areas to the precentral gyrus and only marginally in some cases to the canonical Broca's area.

Ardila [37•] noted that the term "aphasia" has been used to refer both to primary language disturbances affecting the language system itself (phonology, lexicon, semantics, grammar), and also to other impairments not affecting the language system itself, but disturbing some abilities required for using language. He proposes that a major distinction in aphasia can be established between primary language disturbances ("central aphasias": Wernicke-type aphasia and Broca's type aphasia) and secondary language disturbances resulting from "peripheral" impairments (secondary or "peripheral" aphasias: conduction aphasia and supplementary motor area aphasia). Occasionally, the language is not impaired, but the patient cannot use it appropriately because of executive control impairments (dysexecutive aphasia). Ardila [37•] proposes that, in conduction aphasia, language itself is not affected, but rather there is an impaired ability to reproduce aloud the auditory information that is heard. However, the mechanisms required for language comprehension and language production are well preserved. He further suggests that conduction aphasia be interpreted as a "secondary" (or "peripheral") language disturbance, rather than a primary (or "central") form of aphasia. Language knowledge is unimpaired in conduction aphasia, but there is a limitation in a particular language function (i.e., repetition). Clearly, if some animals can repeat, that means that language repetition cannot be considered a primary linguistic ability.

Other recent advances in the understanding of conduction aphasia should be highlighted. Harnish et al. [38] analyzed the role of intensity of aphasia therapy using

functional MRI (fMRI) to document changes in neural activation patterns associated with therapy in an individual with chronic conduction aphasia. They found that task improvement across fMRI testing sessions corresponded with increases in the fMRI blood oxygenation level dependent (BOLD) signal. Significant behavioral improvement and BOLD signal increases occurred after intensive therapy in the left basal ganglia and right hemisphere frontotemporal cortex. Robin et al. [39] examined the visuomotor tracking abilities of patients with apraxia of speech or conduction aphasia, compared with normal healthy subjects. Tracking performance was correlated with perceptual judgments of speech accuracy. Participants tracked predictable (sinusoidal) and unpredictable signals using jaw and lip movements transduced with strain gauges. Tracking performance in participants with apraxia of speech was poorest for predictable signals, but tracking of the unpredictable signal by participants with apraxia of speech was performed as well as for other groups. Performance of the individuals with apraxia of speech on the predictable tracking task was found to strongly correlate with perceptual judgments of speech. The authors concluded that these findings suggest that motor control capabilities are impaired in apraxia of speech but not in conduction aphasia. Kimura et al. [40] reported the case of a woman with primary progressive conduction aphasia associated with corticobasal degeneration, suggesting that progressive conduction aphasia may be a feature of corticobasal degeneration.

Conclusions

Until recently, most authors accepted the explanation proposed by Wernicke and reinforced by Geschwind that conduction aphasia represents a disconnection syndrome due to the damage of the fibers connecting Wernicke's area with the Broca's area, specifically the arcuate fasciculus. Other alternative explanations for the language repetition defect observed in cases of left parietal damage (e.g., Goldstein, Luria) had a lower impact. Recently, with the introduction of contemporary neuroimaging techniques, the disconnection hypothesis has been challenged. It has been reported that conduction aphasia can be found in cases of cortical damage without subcortical extension; and furthermore, the arcuate fasciculus does not connect Wernicke's and Broca's areas, but Wernicke's and motor/premotor frontal areas. Some authors have even suggested that repetition is not really a primary language ability and that so-called conduction aphasia does not represent a primary language disturbance.

It is evident that conduction aphasia remains a controversial topic not only from the theoretic point of view, but also from the understanding of its neurologic foundations. Doubtless, the controversy will continue, but we can anticipate that toward the future a progressively better understanding of the language repetition defects associated with left parietal damage will be obtained.

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