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The role of the arcuate fasciculus in conduction aphasia

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In aphasia literature, it has been considered that a speech repetition defect represents the main constituent of conduction aphasia. Conduction aphasia has frequently been interpreted as a language impairment due to lesions of the arcuate fasciculus (AF) that disconnect receptive language areas from expressive ones. Modern neuroradiological studies suggest that the AF connects posterior receptive areas with premotor/motor areas, and not with Broca's area. Some clinical and neurophysiological findings challenge the role of the AF in language transferring. Unusual cases of inter-hemispheric dissociation of language lateralization (e.g. Broca's area in the left, and Wernicke's area in the right hemisphere) have been reported without evident repetition defects; electrocortical studies have found that the AF not only transmits information from temporal to frontal areas, but also in the opposite direction; transferring of speech information from the temporal to the frontal lobe utilizes two different streams and conduction aphasia can be found in cases of cortical damage without subcortical extension. Taken altogether, these findings may suggest that the AF is not required for repetition although could have a subsidiary role in it. A new language network model is proposed, emphasizing that the AF connects posterior brain areas with Broca's area via a relay station in the premotor/motor areas.

Keywords: arcuate fasciculus; conduction aphasia; language repetition; tractography; vocal imitation
Abbreviations: AF = arcuate fasciculus

Introduction

Conduction aphasia, initially described by Wernicke in 1874, is usually defined as a language disturbance characterized by relatively fluent spontaneous speech, good comprehension, but poor repetition associated with abundant phonological (literal) paraphasias (e.g. Goldstein, 1948; Kertesz, 1979, 1985; Benson, 1988; Kohn, 1992; Benson and Ardila, 1994, 1996; Bartha and Benke, 2003). Patients with conduction aphasia may also have: (i) impairments in naming (from literal paraphasic contamination to total inability to produce the appropriate word); (ii) reading disturbances (comprehension is much better than reading aloud); (iii) writing disturbances (from mild spelling difficulties to profound agraphia); (iv) ideomotor apraxia (buccofacial and limb); and (v) elementary neurological abnormalities (some right hemiparesis and cortical sensory loss) (Benson *et al*., 1973). Language comprehension (auditory and reading) is only mildly impaired. Paraphasias are mainly due to phoneme substitutions and deletions, and they usually result in switches in phoneme manner and place of articulation (Ardila, 1992). Patients with phonological paraphasias due to posterior cortical lesions are usually unaware of their mistakes; in contrast, patients with conduction aphasia are aware of their paraphasic errors. The attempt to correct these errors results in the so-called *conduit d'approche*

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(successive attempts to self-correct their mispronunciations). Luria (1976) assumed that paraphasias in this type of aphasia are articulatory based (that is, errors in the articulatory patterns of the phoneme productions). Based on these differences, some authors have even considered conduction aphasia to be rather a segmental ideomotor apraxia (e.g. Brown, 1972, 1975; Luria 1976, 1980; Ardila and Rosselli, 1990). It is noteworthy that the recovery in conduction aphasia is usually good, and sometimes complete (Kertesz and McCabe, 1977).

The possibility of several mechanisms, each of which is capable of giving rise to deficient repetition has led to the postulation of two different forms of conduction aphasia, described earlier as efferent/afferent (Kertesz, 1979, 1985); reproduction/repetition (e.g. Shallice and Warrington, 1977; Caplan *et al*., 1986); supra and infrasylvian (Axer *et al*., 2001); or simply parietal and temporal (e.g. Bartha and Benke, 2003). 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 (e.g. Caramazza *et al*., 1981); this second subtype of conduction aphasia has been described more frequently with lesions of the temporal lobe (Hickok *et al*., 2000). In this article, we will exclusively refer to the first type of conduction aphasia, i.e. suprasylvian or parietal (Luria's afferent motor aphasia).

Since the time of Wernicke (1874), the aetiology of conduction aphasia has been attributed to a disconnection between the superior temporal gyrus (*'the centre of the acoustic image')* and the inferior frontal gyrus (*'the centre of motor image'*). Wernicke's view was backed up by Geschwind in the 1960s, who put it in terms of modern anatomical nomenclature, attributing the arcuate fasciculus (AF) the main role in the speech repetition problem. Nonetheless, most patients diagnosed with conduction aphasia do have some anomia, and reading difficulties, etc. and hence, usually conduction aphasia is not a pure repetition disorder.

In addition to the disconnection hypothesis, conduction aphasia has also been explained by verbal memory defects, and as a form of segmental ideomotor apraxia. It is important to underline that many features of conduction aphasia relate more to a cortical deficit than a pure disconnection mechanism, as pointed out by different authors (e.g. Levine and Calvanio, 1982). Brown (1975) emphasized that conduction aphasia is not seen in pure white matter disorders such as multiple sclerosis, as would be expected if disconnection were the principal problem, thereby suggesting a cortical involvement. Likewise, putaminal haemorrhages rarely cause conduction aphasia, in spite of the fact that they often disrupt the AF (Hier, 1977). Goldstein (1948) used the name 'central aphasia' to refer to conduction aphasia, implying a cortical rather than a white matter anomaly. He supposed that conduction aphasia may be explained as a disturbance in inner speech, *'the central phenomenon of instrumentalities of speech'*. However, Feinberg *et al*. (1986) tested this hypothesis in five conduction aphasia patients and found that the inner speech explanation may have been correct for only a subgroup of conduction aphasics. Boller and Marcie (1978) proposed a possible disturbance in auditory feedback in conduction aphasia. They describe the case of a 63-year-old man with conduction aphasia who, after being exposed to Delayed Auditory Feedback, spoke faster and with fewer errors than controls and patients with other types of aphasia (Boller *et al*., 1978; Boller and Marcie, 1978). They felt that this paradoxical decreased Delayed Auditory Feedback effect in conduction aphasia only makes sense if the system that supports auditory–motor interaction is disrupted in that syndrome.

The great deal of confusion brought by the term 'conduction aphasia' arises from the attempts to harmonize the heterogeneous presentation of lesions, and hence the clinical findings, with a well-defined small structure such as the AF, that is located in an area where critical language functions exist. According to semantics, 'conduction' should mean only a white matter problem. However, many authors (e.g. Levine and Calvanio, 1982) have described cortical lesions with repetition problems a key symptom of disconnection. Geschwind (1965) proposed that disconnection syndromes could also arise from lesions of the association cortex. Hence, it could raise the question: is conduction aphasia a topographic diagnosis (implying the AF) or is it a syndromatic diagnosis? If conduction aphasia is instead a syndromatic diagnosis, many cases can be accommodated under the same umbrella even with cortical lesions. In those cases, anomia, agraphia and other language problems could be found, depending on the extension of the cortical lesion. If it were just a subcortical disconnection, we would expect a more limited clinical picture, probably with only phonological defects, correlating with findings of functional AF disruption produced by intra-operative electrical stimulation (Mandonet *et al*., 2007).

It is noteworthy that language repetition defects are observed not only in conduction aphasia, but also in other aphasia syndromes, particularly in Broca's and Wernicke's aphasia. Involved mechanisms, however, are different. Language repetition deficits in Broca's aphasia are the result of the speech apraxia and agrammatism associated with this syndrome; phonetic deviations and phonological paraphasias (particularly phoneme omissions) are observed during repetition tasks; furthermore, repetition is agrammatical and patients tend to omit grammatical elements in repetition tests (Li and Williams, 1990; Ardila and Rosselli, 1992; Benson and Ardila, 1996; Martin, 2001). In Wernicke's aphasia, severe and frequently persistent repetition deficits can result from two sources: (i) associated phoneme discrimination defects disturbing the ability for recognition of spoken language; and (ii) verbal memory defects impairing the repetition of long strings of information. The significance of these verbal memory defects and repetition difficulties has lead to the proposal that temporal damage is sometimes associated with a conduction type of aphasia (Shallice and Warrington, 1977; Kertesz, 1979, 1985; Caramazza *et al*., 1981; Caplan *et al*., 1986; Hickok *et al*., 2000; Axer *et al*., 2001; Bartha and Benke, 2003).

Modern neuroimaging techniques have renewed the interest of the neuroscience community in understanding brain connectivity, due to its ability to depict the distinct white matter tracts of the brain. Magnetic Resonance Imaging (MRI) techniques, such as Diffusion Tensor Imaging, and computer-based post-processing that allow complex mathematical data analysis, have made fibre tracking possible. In recent years, an increasing number of publications have appeared in medical journals reviewing the anatomy

of the association tracts and their particular features, asymmetries and variations (e.g. Xia *et al*., 2005; Catani and Thiebaut de Schotten, 2008; Mori *et al*., 2002). Undoubtedly, one of the tracts that has received most attention is the AF due to its potential implication on speech and language brain organization (Catani and Mesulam, 2008). The AF is a brain association tract composed of arched fibres (hence its name) that supposedly connects the Wernicke's and Broca's areas (Fig. 1). The AF is the main part of a larger tract located lateral to the corticospinal tract, termed the superior longitudinal fasciculus. Four different types of connecting fibres have been recently proposed to be included in the superior longitudinal fasciculus (Makris *et al*., 2005). All of them have a frontal terminus in the posterior part of the frontal lobe, but they differ in their origin: the superior horizontal bundle originating in the parietal lobe; two more bundles in the angular and supramarginal gyri and the inferior portion consisting of long arched fibres originating in the ipsilateral superior and middle temporal gyri. Most authors publishing information on the AF (e.g. Duffau, 2008; Catani and Thiebaut de Schotten, 2008) refer to two major anterior–posterior connections of the superior longitudinal fasciculus: (i) the horizontal bundle (parieto-opercular); and (ii) its (inferior) arched part, i.e. the AF that constitutes by far the bulk of the track. Even though the AF is just a component of the superior longitudinal fasciculus, their names are often interchanged. For example, in the atlas of single tracts presented by Catani and Thiebaut de Schotten (2008), the authors include an image of the superior longitudinal fasciculus as the AF (Fig. 11 of their work).

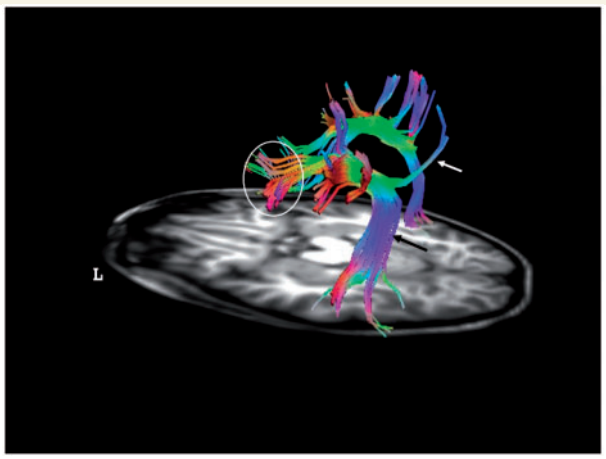

Figure 1 The superior longitudinal fasciculus. Diffusion Tensor Imaging-fibre tractography of the superior longitudinal fasciculus of both sides, over an axial T1 MRI-cut at the level of the temporal lobe. Left lateral view. Tracts are directional colour coded for better tracking. Blue colour indicates top–down; green indicates anterior–posterior and red indicates right to left directions. Reversed directions are encoded within the same colours. White arrow = parieto-frontal fibres (dorsal part of the superior longitudinal fasciculus); black arrow = AF. Within the circle, the opercular endpoint is depicted. Notice the asymmetry of the fibres for more prominent left AF. (Images courtesy of Miami Children's Hospital, Department of Radiology).

Role of the AF in conduction aphasia

As previously mentioned, conduction aphasia has been classically explained as a disconnection syndrome between Wernicke's and Broca's areas (e.g. Wernicke, 1874; Geschwind, 1965; Damasio and Damasio, 1980) due to a lesion affecting the AF. It is noteworthy that one major problem of the cases reported with AF lesions is that they are usually due to infarcts and tumours, and therefore they are not limited within the AF boundaries. Infarcts and tumours usually affect directly and indirectly other adjacent bundles, and also the cortex, including the temporal, parietal and insular cortices. Indeed, it is extremely unlikely to find a case of conduction aphasia with a lesion limited just to the AF. So far, the majority of cases describing clinical findings associated with AF lesions are in reality findings in cases in which the AF was damaged the most amongst other white and grey matter structures. Actually, Wernicke (1874) described the very first case of conduction aphasia in a patient with an insular lesion. Certainly, the AF is often reported to be involved in conduction aphasia (e.g. Tanabe *et al*., 1987; Geldmacher *et al*., 2007; Yamada *et al*., 2007), but cortical lesions alone without subcortical extension may also produce conduction aphasia (Anderson *et al*., 1999; Quigg *et al*., 2006); furthermore, patients with lesions of the AF may retain the ability to repeat (Shuren *et al*., 1995; Kreisler *et al*., 2000). Consequently, the AF does not seem to be crucial for repetition.

Taking these particularities into account, it is difficult to be completely certain of the AF's specific role in conduction aphasia. The question that a disconnected Broca's area can explain repetition problems in the absence of any other major language abnormalities remains unsettled. But the fact that conduction aphasia has been mostly characterized by language repetition problems makes it worthy to ascertain the neurophysiology of vocal repetition in both animals and humans.

What supports vocal repetition?

To account for vocal repetition, at least two neural subsystems should be assumed: one for vocalization, i.e. for the programming, production and control of vocal sounds; and the other for transferring phonological cues decoded in the auditory areas towards areas of motor programming, representation and execution. The former relates to the brainstem (vocal nuclei) network; the latter to the AF or another similar connecting pathway (Ghazanfar and Rendall, 2008). This mechanical interplay between phonological schemes and motor sequences; however, is not necessarily linked to meaning extraction or semantic awareness. One can repeat a foreign language word or even entire sentences without knowing their meaning. Furthermore, repetition of phonemes and words is not exclusively a human ability either. For example, some birds can repeat human speech (e.g. parrots, macaws, cockatoos), but presumably they do not make sense of it.

This observation implies that repetition should not be necessarily regarded as language ability but rather as speech ability. Consequently, conduction 'aphasia' may, strictly speaking, be interpreted as a speech disturbance rather than as a language disturbance; and therefore not as an aphasia, or at least not as a 'primary aphasia' (Ardila, 2009).

It can be conjectured that any vocal repetition system (human or animal) also needs a subsystem to carry information from auditory receptive areas to executive areas. Here is the point where the AF or any similar bundle may play a crucial role.

Unfortunately, the AF has not been investigated in talking birds. Monkeys, and in general mammals, without a vocal repetition ability, do not have an AF (Schmahmann *et al*., 2007). Instead, they posses three different bundles connecting areas of the parietal lobe with the frontal opercular, premotor and supplementary motor areas. The lack of connection between the auditory and motor processing areas should be associated in monkeys with problems of vocal repetition and speech learning due to the absence of phonological awareness. This lack of connection is not surprising: monkeys do not repeat, and monkeys do not use spoken language.

The neuroanatomy of the arcuate fasciculus

The type of function a given tract transfers may be inferred from its anchor points. For example, motor function may be inferred from a tract whose endpoints are located in the precentral gyrus and the pons. Visual function may be inferred from a tract whose endpoints connect the eyes and the occipital lobe. Hence, the type of transferring information that the AF subserves can be examined and inferred by determining its endpoints by means of diffusion tensor imaging tractography, a novel technique based on the anisotropy of water diffusion (Catani and Mesulam, 2008). This technique may answer the crucial question of where exactly the rostral terminus of the AF is located.

Usually, in the neurology literature, the rostral terminus of the AF has been assigned to Broca's area, that is, the pars opercularis of the frontal lobe. This view was first stated by Wernicke in 1874 and has been usually maintained to date by neuroanatomists and clinicians alike (Obler and Gjerlow, 1999; Aminoff *et al*., 2005; Bear *et al*., 2006). The pars opercularis is a narrow top-to-down oriented grey matter strip located in the posterior third of the inferior frontal gyrus. The posterior terminus of the AF is not limited to a well-defined anatomical territory with precise landmarks. It mostly encompasses the posterior middle and inferior temporal gyrus, but some fibres may be seen reaching the middle third of the same gyri.

If the AF plays a major role in language, anatomical asymmetry can be anticipated. Certainly, utilizing fibre tractography with diffusion tensor imaging, the AF has been found to be asymmetric in a number of recent studies, with dominance in the left hemisphere (Nucifora *et al*., 2005; Parker *et al*., 2005; Powell *et al*., 2006; Catani *et al*., 2007). This asymmetry and the fact that the cortical endpoints are supposedly located in canonical areas of language (Wernicke's and Broca's areas) seem to support the idea of a language transferring function.

In spite of the abundant literature that accepts the Wernicke–Broca connection, new findings do not necessarily support that point of view. Bernal and Altman (2008) studied 12 normal right-handed subjects. The AF fibres were obtained bilaterally by placing a region of interest at the triangular-shaped region lateral to each of the corticospinal tracts, in a coronal plane along the rostral aspect of the splenium of the corpus callosum. A sagittal fractional anisotropy image was used to determine the rostral endpoint of the AF fibres in the white matter pertaining to specific gyri or pars of the frontal lobe. These authors found that the AF projection to Broca's areas was absent in 10 subjects (83.3%), and minimal in 2 (16.6%). However, the AF's rostral endpoints were found in the precentral gyrus in 100% of cases. The general conclusion was that the AF does not connect with Broca's area, but instead, it connects to the pre-central gyrus (premotor and primary motor areas) (see Fig. 2)

This finding apparently conflicts with the established view in which Broca's area is thought of as the rostral endpoint of the AF. Moreover, a critical review of the most recent reports of the arcuate connectivity with diffusion tensor imaging, particularly of the images that appear in those reports, seems to support the precentral gyrus as the endpoint of the AF. Indeed, Catani and Thiebaut de Schotten (2008) published an atlas of the main brain tracts based on diffusion tensor imaging fibre tractography of 12 right-handed subjects. Figure 1B of their work (page 1107) depicts the entire superior longitudinal fasciculus reaching the

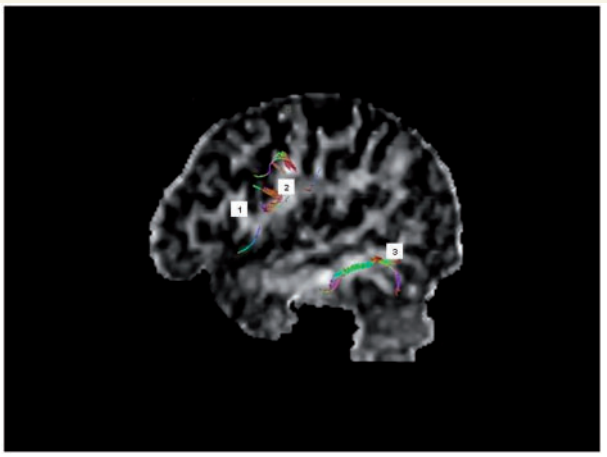

Figure 2 Localizing the endpoints of the AF. Diffusion Tensor Imaging-fibre tractography of the superior longitudinal fasciculus with fibres directional-colour-coded depicted on a left parasagittal brain fractional-anisotropy MR image. The frontal lobe appears on the left side. The following gyrus white matter banks are numbered for easy landmark identifications: 1 = inferior frontal gyrus, pars opercularis (Broca's area); 2 = Precentral gyrus; 3 = middle temporal gyrus. Notice the fibres emerging in the middle temporal gyrus and in the precentral gyrus. No emerging fibres are seen in the Broca's area (Images courtesy of Miami Children's Hospital, Department of Radiology).

pre-central gyrus and not the inferior frontal gyrus. Likewise, Glasser and Rilling (2008) report a probabilistic connectivity of the two main parts of the superior longitudinal fasciculus in 20 right-handed subjects. The anterior isocentre of the image presented in their work (Fig. 1A, p. 2473) is located in the inferior third of the precentral gyrus and not in the inferior frontal gyrus, which is only just tangentially involved.

Theoretically, the rostral connection of the AF with premotor/motor areas does not necessarily conflict with any established neurological or neuroanatomical rule. In its inferior third, the precentral gyrus is divided into two parts. The anterior bank and part of the convexital aspect of the gyrus is the seat of Brodmann's Area (BA) 6. The rest of the gyrus is represented by BA4, corresponding to the somatotopic representation of the tongue, lips and pharynx. Recent evidence suggests that the left BA6 is involved in speech programming (Fox *et al*., 2000; Shuster and Lemieux, 2005) and phonological and lexical processing (Price *et al*., 1994; McDermott *et al*., 2003). Thus, the involvement of BA6 in speech programming makes it a very suitable target for the AF.

Unidirectional, posterior to anterior, language transferring by the AF has also been recently challenged. Electrocortical studies have found that the AF not only transmits information from temporal to frontal areas, but also in the opposite direction (Matsumoto *et al*., 2004). Similar results have been observed using structural modeling functional (f)MRI (Bullmore *et al*., 2000). This unexpected observation suggests that the role of the AF may be notoriously more complex than simply transmitting information between the Wernicke's and Broca's areas. This bi-directional information flow of the AF seems to support the AF's connection to premotor areas, since certain feedback about articulation may be necessary, accordingly with the so-called motor theories of language understanding (Liberman *et al*., 1967). These theories suggest that the same processes that are involved in production, also participate in the perception of speech. Therefore, 'the objects of speech perception' are the intended phonetic gestures of the speaker, represented in the brain as invariant motor commands that call for movements of the articulators through certain linguistically significant configurations (Liberman and Mattingly, 1985; p. 2). In other words, the bi-directional transferring of information would suggest that, to some extent, information about language production is important for language understanding. It may play a dual role in speech as well; monitoring speech production and informing about verbal output.

Several findings seem to point to an accessory function of the AF in speech repetition, and not to a unique, segregated and necessary function—as in the case of the corticospinal pathway. The following are examples demonstrating that in some cases, the AF is not necessary for speech repetition.

Language lateralization, as determined by fMRI, has been found contralateral to the hemisphere in which the AF appears dominant for language in 5 of 13 left-handed volunteers (Vernooij *et al*., 2007), and in 3 of 24 patients using Wada exams (Matsumoto *et al*., 2008). Usually, the AF is lateralized to the left regardless of the right dominance of language (Vernooij *et al*., 2007). So, the coincidence of the AF and language lateralization is not essential.

Unusual patterns of brain organization of language have been reported (e.g. Basso *et al*., 1985; Vignolo *et al*., 1986; Ferro and Madureira, 1997). Subjects with normal language function rarely have inter-hemispheric dissociated Broca's–Wernicke's area lateralization (one area in the left hemisphere and the other one in the right). In those unusual dissociated conditions, Broca's and Wernicke's areas are located in different hemispheres. This condition has been reported in rare cases of stroke (Paparounas *et al*., 2002), in 2 of 87 cases by fMRI (Kamada *et al*., 2007), and in <1% of large intra-carotid amobarbital studies in epileptic patients (Risse *et al*., 1997; Lee *et al*., 2008). None of these authors report any abnormality that would suggest language repetition defects in these cases, albeit a direct communication between Wernicke's and Broca's areas is very unlikely. At the Miami Children's Hospital, we have had the opportunity to see three cases of language representation dissociation as mapped with fMRI. None of these cases had overt speech or language repetition problems. Figure 3 illustrates one of these unusual cases. Finally, only 50% of the patients with conduction aphasia show hypometabolism of Broca's areas, although all of them show reduced metabolism in the Wernicke's area (Kempler *et al*., 1988).

It is hard to harmonize these findings with a model that has the AF as a bundle necessarily required for language or speech transferring. Taking into account the clinical findings of conduction aphasia and the results of functional and neuroimaging studies it seems reasonable to speculate that the AF may function as a supplementary aid to facilitate repetition or to monitor speech at phonological level. Perhaps it may also play an important role in speech development and language learning.

Synthesis: re-interpreting the role of the arcuate fasciculus

All things considered, it could be suggested that a model that defines the AF as an accessory aid in transferring information from the temporal lobe to premotor/motor areas, and not directly to Broca's area, may be more congruent with the neuroimaging results and clinical findings. A connection like this may suggest that auditory representation of speech plays a direct role in verbal motor planning. Interestingly, that was the initial hypothesis of Wernicke (cited by Hickok, 2000), and it also favours the explanation by Luria (1976) who sees the repetition problems of conduction aphasia as an ideomotor verbal apraxia. Moreover, it seems easier to explain the phenomenon of *conduit d'approche* as having a disconnection of the premotor area. Indeed, a premotor disconnection may impair the proper motor sequencing necessary to utter word components (word segments or syllables). As a result, a phonological paraphasia is generated. Since purely language areas are not affected, phonological awareness is not impaired and the patient attempts to correct the output by exhibiting the typical *conduit d' approche*, in the same manner as an apraxic patient tries over and over to find the proper movements to allow him/her to perform a particular action. This contrasts with the truly language phonological paraphasia in which

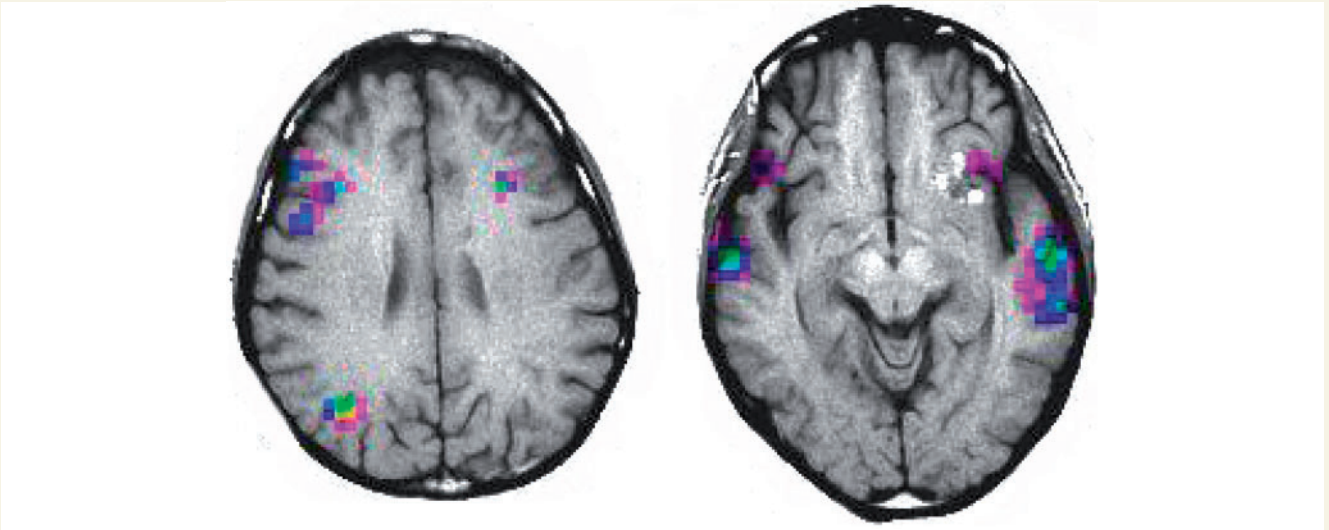

Figure 3 Functional MRI of language. Example of a subject with inter-hemispheric dissociated Broca's–Wernicke's area lateralization (one area in the left hemisphere and the other one in the right). MRI displayed with radiological convention: Left hemisphere is located in the right. Left image: activation obtained with a paradigm of semantic fluency. Right image: activation in primary and secondary auditory areas from a passive listening task. Notice frontal areas associated with expressive language located in the right hemisphere while posterior receptive areas are dominant on the left hemisphere. Activation is colour coded for signal intensity from magenta (weak) to blue to green to yellow (strong). (Images courtesy of Miami Children's Hospital, Department of Radiology).

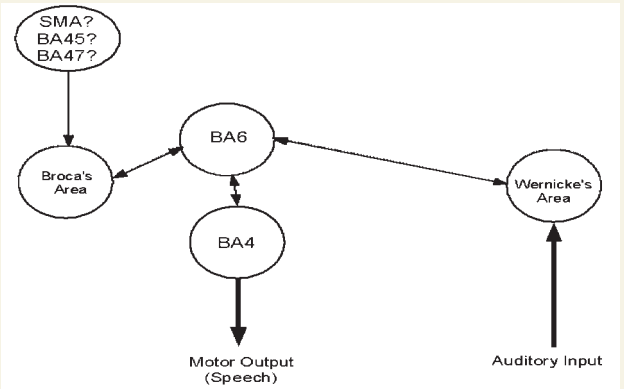

Figure 4 Language network model. Language cortical modules or systems are represented by circles. Arrows depict the flow of information from auditory input to speech and viceversa. The standard model interconnects comprehension (Wernicke) and production (Broca) areas in a direct manner through the AF. Recent findings suggest a relay station located in the precentral gyrus, represented here by the circle labeled BA6, an area with premotor functions. Input to Broca's area may have several sources. Lesions in any module or connection should produce a specific type of language or speech syndrome. Several connections are shown with bi-directional flow representing a theoretical point of view of the authors, supported for some recent articles (see text).

the patient is not usually aware of his/her errors, and therefore, the fluency of the speech is not impacted. The AF, nonetheless, may still connect to Broca's area through a relay station located in the premotor or motor cortex (Fig. 4).

A model like this can explain why the AF has a role that seems closer to speech (motor function) than to language (cognition), and may support clinical views suggesting that conduction aphasia may be explained as a type of speech apraxia (e.g. Brown, 1975; Luria, 1976; Ardila, 1992). It could be speculated that the role of the AF in language is contingent and not necessary, and that it could play a significant role in speech programming and learning with an interconnection between BA6 and BA44. A connection like this would provide a structural substrate for the imitation of speech, a first step necessary for language learning and acquisition.

It is important to mention that other paths have been described to be involved in language transferring. Particularly, a ventral path has been reported connecting the temporal lobe with frontal opercular areas (most likely Broca's area) (Duffau *et al*., 2005). This transferring pathway, however, has been found to be related to semantic cues (Parker *et al*., 2005; Saur *et al*., 2008). Its function is also challenged by the same cases described above of brain language inter-hemispheric dissociation.

Conclusions

Classically, conduction aphasia has been interpreted as a disconnection syndrome, isolating the Wernicke's and Broca's areas. Conduction aphasia is usually considered to be a result of damage of the AF, subjacent to the parietal portion of the supramarginal gyrus, and the upper part of the insula. However, conduction aphasia seems to be a rather transient phenomenon, and not a condition that lasts in time as other neurological deficits do due to structural damage.

The function of language transferring assigned to the AF is still elusive. Several clinical and functional findings suggest that the AF is not required for repetition but it does seem to have a subsidiary role in it. It could be speculated that the AF serves an important role in language development by facilitating the repetition of phonological clues, and therefore helping in learning language and monitoring speech. These observations could re-install the question whether conduction aphasia is indeed a 'language' disconnection syndrome due to damage to the AF; or rather, it should be understood as a defect in 'speech' repetition that is sometimes, but not always, associated with AF damage.

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