Chapter

A NEW CLASSIFICATION OF APHASIAS

Alfredo Ardila*

Department of Communication Sciences and Disorders
Florida International University, Miami, FL, US

ABSTRACT

In this chapter it is emphasized that there are only two fundamental forms of aphasia, which are linked to impairments in the lexical/semantic and grammatical systems of language (Wernicke-type aphasia and Broca-type aphasia, respectively). Other aphasic syndromes do not really impair language knowledge per se, but rather either some peripheral mechanisms required to produce language (conduction aphasia and aphasia of the supplementary motor area), or the executive control of the language (extra-Sylvian or transcortical motor aphasia). A new classification of aphasic syndromes is suggested. In this proposed classification a distinction is established between primary (or ‘‘central’’) aphasias (Wernicke’s aphasia—three subtypes—and Broca’s aphasia); secondary (or ‘‘peripheral’’) aphasias (conduction aphasia and supplementary motor area aphasia); and dysexecutive aphasia.

* 11200 SW 8th Street, AHC3-431B
Florida International University
Miami, Florida 33199
Email: ardilla@fiu.edu

INTRODUCTION

Aphasia represents the most studied cognitive syndrome associated with brain pathology. As a matter of fact, the analysis of aphasia represents the initial question and departing point in modern cognitive neuroscience. Understanding aphasia is consequently most crucial in our interpretations about brain organization of cognition.

Diverse aphasia classifications have been proposed since Broca's first description of a language disturbance associated with brain pathology (Broca, 1863). There are, however, two most influential aphasia classifications, that have significantly guided the area during the last decades: the Boston Group classification (Geschwind, Benson, Alexander, Goodglass, Kaplan, and others); and Luria's aphasia interpretation. The first one has been particularly influential in the US and western European countries; the second one has been mostly used in eastern European countries and Latin America.

Boston Group classification represents a further development of Wernicke's ideas about brain organization of language, and includes two basic distinctions: (1) aphasias can be fluent or non-fluent; and (2) aphasias can be cortical, subcortical, or transcortical (e.g., Albert, Goodglass, Helm, Rubers, & Alexander, 1981; Alexander & Benson, 1991; Benson, 1979; Benson & Geschwind, 1971, 1985; Geschwind, 1965; Goodglass, 1993; Goodglass & Kaplan, 1972). Conduction aphasia (initially proposed by Wernicke in 1874 and described by Lichtheim in 1885) was introduced to account for the language repetition impairments frequently found in left parietal (or insular) damage.

Luria (1966, 1970, 1974, 1976, 1980) proposed, initially six, but later seven aphasia subtypes: motor efferent or kinetic, motor afferent or kinesthetic, acoustic-agnosic, acoustic-amnesic, amnesic, semantic, and dynamic). Luria assumed that in each aphasia subtype there is a particular language processing defect. In Luria's approach, aphasia subtypes and names refer to the specific level of language that is impaired.

Benson and Ardila (1996) attempted to integrate both points of view and proposed a classification based on two different anatomical criteria: (1) aphasia can be pre-Rolandic (anterior, non-fluent) or post-Rolandic (posterior, fluent); and (2) aphasia can be associated with pathology in the peri- Sylvian language area (peri-Sylvian aphasias); or aphasia is due to damage beyond this area (extra-Sylvian). Subtypes were introduced for some aphasia syndromes. Aphasias were also regarded as anatomical syndromes

(Table 1). This classification is currently used by different authors (e.g., Basso, 2003).

Table 1. Two major parameters are used in aphasia classification: (a) Aphasia can be pre-Rolandic or post-Rolandic; (b) Aphasia can be peri-Sylvian or extra-Sylvian. Clinical syndromes are related to anatomical syndromes

	Pre-Rolandic	**Post-Rolandic**
Peri-Sylvian	Broca's Type I (triangular syndrome)	Conduction (parietal-insular syndrome)
	Broca's Type II (triangular-opercular-syndrome)	Wernicke's Type I (posterior insular-temporal isthmus syndrome)
		Wernicke's Type II (superior and middle temporal gyrus syndrome)
Extra-Sylvian	Extra-Sylvian Motor Type I (left prefrontal dorsolateral syndrome)	Extra-Sylvian Sensory Type I (temporal-occipital syndrome)
	Extra-Sylvian Motor Type II (supplementary motor area syndrome)	Extra-Sylvian Sensory Type II (parieto-occipital angular syndrome)

According to Benson & Ardila, 1996.

During the last decades significant advances in the understanding of brain organization of language has been obtained. Contemporary neuroimaging techniques, such as fMRI (e.g., Meinzer, Harnish, Conway & Crosson, 2011; Zahn et al., 2000), PET (Cao, George, Ewing, Vikingstad & Johnson, 1998) and tractography (Song et al., 2011; Yamada et al., 2007), have significantly extended our understanding of the organization of language in the brain under normal and abnormal conditions (Lee, Kannan & Hillis, 2006; Small & Burton, 2002); a significantly better understanding of the brain circuitries supporting language has been developed (e.g., Ullman, 2004); and a re-analysis of the classical language areas (Broca's and Wernicke's) has been developed (e.g., Grodzinky & Amunts, 2006); and new scientific discoveries, such as the ''mirror neurons'', have changed our

understanding of the functioning of the human brain, including language organization (e.g., Rizzolati & Arbib, 1998).

In this chapter a further attempt is made to integrate these new advances; a new classification of aphasia syndromes will be proposed. This new aphasia classification was recently presented (Ardila, 2010) and has been discussed by several authors (e.g., Buckingham, 2010; Kertesz, 2010; Marshall, 2010).

THERE ARE ONLY TWO MAJOR APHASIC SYNDROMES

There is a fundamental point in the analysis of aphasia: aphasia is not one, but two different clinical syndromes, initially described by Broca in 1861 and Wernicke in 1874. These two syndromes have been named in different ways, but roughly corresponding to Wernicke-type aphasia and Broca-type aphasia (e.g., Ardila, 2010; Albert et al., 1981; Alexander & Benson, 1991; Bastian, 1898; Benson & Ardila, 1996; Freud, 1891/1973; Goldstein, 1948; Head, 1926; Hecaen, 1972; Kertesz, 1979; Lichtheim, 1885; Luria, 1976; Pick, 1931; Schuell, Jenkins, & Jimenez-Pabon, 1964; Taylor-Sarno, 1998; Wilson, 1926; see Tesak & Code, 2008, for review).

Aphasia represents a language disturbance and consequently, not only a neurologic/anatomic but also a linguistic understanding is required. Jakobson (1964; Jakobson & Halle, 1956) proposed that these two major aphasic syndromes are related to the two basic linguistic operations: selecting (language as paradigm) and sequencing (language as syntagm). Jakobson (1964) proposed that aphasia tends to involve one of two types of linguistic deficiency. A patient may lose the ability to use language in two rather different ways: the language impairment can be situated on the paradigmatic axis (similarity disorder) or the syntagmatic axis (contiguity disorder). The first one is related with the Wernicke-type aphasia, and the second one with the Broca-type aphasia.

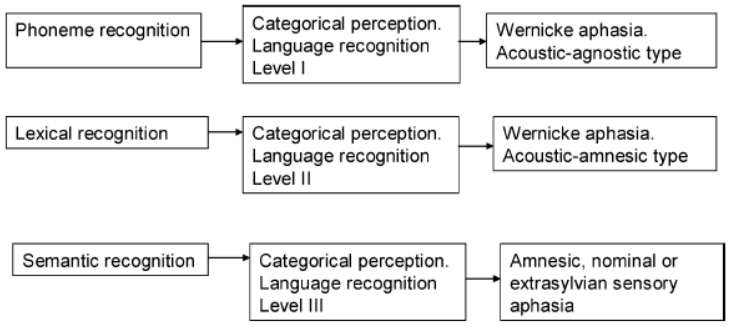
Wernicke-Type Aphasia

In Wernicke-type aphasia the lexical repertoire tends to decrease and language-understanding difficulties are evident. Wernicke's aphasia patients may not fully discriminate the acoustic information contained in speech (Robson, Keidel, Ralph & Sage, 2012). Lexical (words) and semantic

(meanings) associations become deficient. In Wernicke-type aphasia the language defect is situated at the level of meaningful words (nouns). Phoneme and word selection are deficient, but language syntax (contiguity: sequencing elements) is well preserved and even overused (paragrammatism in Wernicke aphasia). Wernicke-type aphasia represents the clinical syndrome characterized by impairments in the selection process (paradigmatic axis defect).

Patients with Wernicke aphasia have problems in recalling the words (memory of the words) and also in associating the words with specific meanings: the semantics of the words can be abnormal. This means that at least three different deficits underlie Wernicke-type aphasia: (1) phoneme discrimination impairments (auditory verbal agnosia); (2) verbal memory impairments; and finally (3) lexical/semantic association deficits. Robson, Sage and Ralph (2012) emphasized that deficits responsible for the comprehension defects in Wernicke aphasia are diverse, including acoustic-phonological defects, and semantic deficits.

Figure 1 presents in a summarized form the model proposed by Ardila (1993) to account for language recognition. It is assumed that there are three different levels of language understanding (phoneme recognition, lexical recognition, and semantic recognition). These three language understanding levels can be impaired in cases of Wernicke-type of aphasia. In consequence, there are three different subtypes of Wernicke aphasia: Acoustic-agnosic type (associated with phoneme recognition defects), acoustic amnesic type (associated with lexical recognition defects), and amnesic, nominal or traditionally called transcortical (extrasylvian) sensory aphasia (associated with semantic recognition defects).



Adapted from Ardila, 1993.

Figure 1. Three levels of language recognition potentially impaired in Wernicke-type aphasia can be distinguished: phonemic (categorical perception level I), lexical

(categorical perception level II), and semantic (categorical perception level III). Three different sub-syndromes can be found: phonemic discrimination defects (acoustic-agnosic or Wernicke's aphasia type I), verbal-acoustic memory defects (acoustic-amnesic or Wernicke's aphasia type II), and semantic association defects (amnesic, nominal or extra-Sylvian sensory aphasia).

Broca-Type Aphasia

In Broca-type of aphasia, language defects are quite different; while the lexical/semantic dimension of the language is preserved, grammar is seriously impaired. Language is scarce, nonfluent, and poorly articulated, but language understanding is relatively well preserved. That means, the selection process (paradigmatic axis) is normal.According to Jakobson (1964) in Broca-type aphasia the syntagmatic axis of language is impaired. There is a defect in language sequencing (morphosyntax). Indeed, in Broca's aphasia two different distinguishing characteristics can be observed, one at the motor level and the other at the purely language level: (1) there is on one hand a motor component (lack of fluency, disintegration of the speech kinetic melodies, verbal-articulatory impairments, etc., that is usually referred as apraxia of speech); and (2) on the other hand, there is a reduction in the grammar, usually referred as agrammatism (e.g., Benson & Ardila, 1996; Berndt & Caramazza, 1980; Goodglass, 1993; Kertesz, 1985; Luria, 1976). Interestingly, a large part of the fronto-parieto-temporal cortex has been observed to be involved with syntactic-morphological functions (Bhatnagar, Mandybur, Buckingham, & Andy, 2000). Apraxia of speech has been observed specifically associated with damage in the left precentral gyrus of the insula (Dronkers, 1996; but see Hillis et al., 2004) It should be noted that not all of apraxia of speech is indeed a contiguity disorder; there are many phonetic-level errors in apraxia of speech that have more to do with segmental distortions. If both impairments (apraxia of speech and agrammatism) are simultaneously observed (i.e., they are very highly correlated), it can be assumed they are just two different manifestations of a single underlying defect. It is not easy to understand which one could be the single factor responsible for these two clinical manifestations, but it may be kind of ''inability to sequence expressive elements''. Broca's area, most likely, is not specialized in producing language, but in certain neural activity that can support not only skilled movements required for speech, but also morphosyntax. It has been observed that indeed language networks

supporting grammar and fluency are overlapped in the brain (Borovsky, Saygin, Bates, & Dronkers, 2007).

HOW TO INTEPRET OTHER APHASIC DISTURBANCES?

Frequently it has been assumed that three major (perisylvian) aphasic syndromes can be distinguished: frontal Broca aphasia, temporal Wernicke aphasia, and parietal conduction aphasia (e.g., Benson, 1979; Goodglass, 1993). These are the three aphasia disorders associated with damage in the so-called ‘‘brain language area’’; a concept introduced by Dejerine (1914), roughly corresponding to the perisylvian area of the left hemisphere, and including partially the frontal, temporal and parietal lobes of the left hemisphere.

In addition to Broca, Wernicke and conduction aphasia, aphasia classifications generally include a diversity of additional language disturbances, such as transcortical (extra-Sylvian) aphasia, and anomic aphasia (e.g., Alexander & Benson, 1991; Benson & Geschwind, 1971; Hecaen & Albert, 1978; Kertesz, 1979; Lecours, Lhermitte, & Bryans, 1983; Luria, 1966). However, some aphasic syndromes can eventually be considered as variants of the Broca and Wernicke aphasias. For instance, as mentioned above, amnesic or anomic or nominal aphasia (usually due to damage in the vicinity of BA 37) (Head, 1926; Hecaen & Albert, 1978; Luria, 1976), as well as transcortical sensory aphasia can be interpreted as subtypes of Wernicke aphasia in which the semantic associations of the words are significantly impaired (see Figure 1).

No question, the major difficulty in interpreting these additional syndromes refers to conduction aphasia, considering that conduction aphasia is frequently regarded as one out the three major aphasia syndromes (in addition to Broca aphasia and Wernicke aphasia).

Conduction Aphasia

A crucial question is, how conduction aphasia—a well recognized and extensively studied aphasic syndrome (e.g., Benson & Ardila, 1994; Damasio & Damasio, 1980; Goldstein, 1948; Kohn, 1992) — can be interpreted?

The most frequent, and classic, explanation of conduction aphasia is as a disconnection syndrome (e.g., Damasio & Damasio 1980; Geschwind 1965;

Wernicke 1874), usually due to a lesion affecting the arcuate fasciculus (Yamada et al., 2007) and sporadically an indirect pathway passing through the inferior parietal cortex (Catani, Jones, & Ffytche, 2005). This is the usually explanation, sometimes referred as the Wernicke-Geschwind disconnection model of conduction aphasia. Alternatively, conduction aphasia has also been interpreted as a segmentary ideomotor apraxia (e.g., Ardila & Rosselli., 1990; Brown, 1972, 1975; Luria 1976, 1980). According to this second interpretation, conduction aphasia could be regarded as a verbal apraxia, an ideomotor apraxia impairing the movements required for speaking, or simply as a kinesthetic apraxia of speech. Luria (1976) suggested that paraphasias in conduction aphasia (Luria's kinesthetic motor or afferent motor aphasia) are indeed articulatory-based deviations (articulatory literal paraphasias), not really phonological disturbances. Paraphasias in conduction aphasia are due mainly to phoneme substitutions and phoneme deletions; they result basically in switches in phoneme manner and place of articulation (Ardila, 1992). Similarities between errors in ideomotor apraxia and conduction aphasia language deficits have been suggested.

According to Benson, Sheretaman, Bouchard, Segarra, Price, and Geschwind (1973) conduction aphasia has three fundamental and five secondary characteristics; so-called secondary characteristics are frequently but not necessarily found in conduction aphasia. The three basic characteristics are: (1) fluent conversational language; (2) comprehension almost normal; 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; and (5) additional neurological impairments. Bartha and Benke (2003) report that conduction aphasia patients present as relatively homogenic 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 so-called *conduit d'approche*. Language comprehension (auditory and reading) is only mildly impaired.

Benson et al.'s. (1973) description of conduction aphasia clearly recognizes that spontaneous language production and language understanding are significantly preserved. In consequence, some mechanisms required for correct language repetition are impaired, but the knowledge of language itself (phonology, lexicon, semantics, and grammar) is not impaired. The critical question is: Should conduction aphasia be interpreted as a primary aphasic

syndrome? Indeed, language repetition impairments can be observed in different aphasia syndromes and language repetition has also been interpreted as a right hemisphere ability (Berthier et al., 1991).

The distinction between ‘‘aphasias with repetition impairments’’ vs ‘‘aphasias without repetition impairments’’ is indeed a general and crude distinction. It has been proposed that different aphasia groups (including the so-called transcortical aphasias) may present language repetition errors; but depending on the specific repetition task, errors may be evident or may be unnoticed in a particular aphasic group (Ardila & Rosselli, 1992). Different mechanisms underlying repetition deficits have been proposed: limitation of auditory-verbal short-term memory, difficulties at the level of phonological production, impairments in phoneme recognition, and semantic and syntactic comprehension defects. Simply speaking, different deficits can be responsible for the repetition defects found in aphasia. Furthermore, difficulties in language repetition depend on the specific repetition task (short words, long sentences, meaningful, meaningless, etc).

Conduction aphasia is, consequently, not a primary form of aphasia, but rather a secondary (or ‘‘peripheral’’) defect in language indirectly affecting a specific language ability (i.e., the ability to repeat). Language itself is not impaired, but rather it represents an impaired ability to reproduce aloud the auditory information that is heard. Of course, this is an important skill used not only to develop language but also to use it correctly. Interpreting conduction aphasia as a secondary (or ‘‘peripheral’’) defect in language (rather than a primary or central form of aphasia) does not in any way decrease the importance of repetition in language.

In brief, it can be argued that conduction aphasia can be interpreted as a ‘‘secondary’’ (or ‘‘peripheral’’) language disturbance, rather than a primary (or ‘‘central’’) form of aphasia. Language knowledge is well preserved in conduction aphasia, but there is a limitation in a particular language function, i.e., repetition. Obviously, if some animals can repeat, that means that language repetition cannot be considered as a primary linguistic ability.

Interestingly, Jakobson (1964) suggested a similar distinction when proposing that in aphasia language could be either ‘‘disintegrated’’ or ‘‘limited’’ (disintegration vs limitation in aphasia). Obviously language is disintegrated only in Wernicke and Broca aphasia. In other forms of aphasia, including conduction aphasia, language is limited, not disintegrated.

Transcortical (Extra-Sylvian) Motor Aphasia

Patients with left convexital prefrontal damage usually present a lack of verbal initiative and a significant limitation in the active use of the language, referred as transcortical (extra-Sylvian) or dynamic aphasia. Extra-Sylvian (transcortical) motor aphasia could be interpreted as an executive function defect specifically affecting language use. The ability to actively and appropriately generate language appears impaired while the phonology, lexicon, semantics, and grammar are preserved.

Should the ability to correctly generate language be regarded as a linguistic ability (i.e., cognitive ability)? Or rather, should it be considered as an executive function ability (i.e., metacognitive ability)? It does not seem difficult to argue that the ability to correctly organize language sequences can be regarded as an executive function and as a metacognitive ability rather than a purely linguistic ability. Some rationales to support this interpretation are: (1) It could be argued that in extra-Sylvian (transcortical) motor aphasia there is a defect in verbal initiative rather than in language knowledge (Kleist, 1934). (2) Different authors (for example, Luria, 1976, 1980) have emphasized that this type of aphasia shares the general characteristics of prefrontal (i.e., dysexecutive) syndrome but specifically with regard to verbal processes. This means, it is the prefrontal (dysexecutive) syndrome affecting the verbal processes (Gold et al., 1997). (3) Further, the impairment in extra-Sylvian (transcortical) motor aphasia does not affect language understanding, and fundamental linguistic processes are preserved (Berthier, 1999). And finally, (4) it could be argued that the prefrontal cortex does not participate in basic cognition, but rather in metacognition (e.g., Ardila & Surloff, 2011).

In consequence, extra-Sylvian (transcortical) motor aphasia does not necessarily have to be interpreted as a primary aphasic syndrome, but rather as a language disturbance due to a more general intellectual impairment (dysexecutive syndrome). Extra-Sylvian (transcortical) motor aphasia could indeed be referred to as ‘‘dysexecutive aphasia’’. Some authors have previously interpreted extra-Sylvian motor aphasia in a similar way (e.g., Luria 1976, 1980). Alexander (2006) suggested that transcortical motor aphasia could be more accurately defined as an executive function disorder rather than aphasia. He proposed that the progression of clinical disorders from aphasia to discourse impairments can be interpreted as a sequence of procedural impairments from basic morpho-syntax to elaborated grammar to narrative language, correlated with a progression of the focus of the damage from posterior frontal to polar and/or lateral frontal to medial frontal.

Transcortical (Extra-Sylvian) Sensory Aphasia

Transcortical (extra-Sylvian) sensory aphasia (TSA) has been a polemic syndrome. Seemingly, the polemic is related to the way TSA is defined.

TSA has been defined in two partially different ways; (1) according to its ''basic'' definition, TSA is a fluent language disorder characterized by impaired auditory comprehension, with preserved repetition (Albert et al., 1981; Berthier, 1999; Goldstein, 1948; Lichtheim, 1885). Consequently, there are only three distinguishing characteristics in TSA (normal fluency, impaired auditory comprehension, and preserved repetition). In such a case, TSA presents similar deficits as in Wernicke's aphasia, but repetition ability is spared and phoneme discrimination impairments are not found. (2) According to its ''extended'' definition, TSA also includes a semantic jargon (Goodglass, 1993; Kertesz, 1982; Lecours, Osborn, Travies, Rouillon, & Lavalle-Huyng, 1981). Kertesz (1985, p. 317) makes a comprehensive definition of TSA: ''TSA is characterized by fluent and often irrelevant speech output, very poor comprehension and well-preserved repetition. Spontaneous speech often consists of semantic jargon that has no relationship to what is being asked of the patient''. This definition clearly recognizes that there are three basic characteristics, and sometimes jargon is found. But jargon is not a required symptom for the diagnosis of TSA. By the same token, other language impairments can also be found, such as poor naming, and preserved oral reading with impaired reading comprehension, but their presence is not essential to establish the diagnosis of TSA (Berthier, 1999).

According to Berthier (1999) the most common pattern of verbal expression is represented by the so-called ''semantic'' or ''verbal'' jargon (e.g., Lecours & Rouillon, 1976). There is an abundant language production, with reduction of meaningful words conferring the impression of emptiness. The content of the sentence is irrelevant. Furthermore, TSA patients appear unaware of their logorrhea (Lebrun, 1987). A second pattern of spontaneous speech described by Berthier (1999) is referred to as ''anomic'' and is associated with an impaired access to content words. This second pattern corresponds to the TSA ''basic'' definition mentioned above.

Because repetition is spared, phonological processing is assumed to be preserved, at least partially, while lexical-semantic information included in the word meaning is impaired (Boatman et al., 2000). Usually, it is accepted that TSA is associated with relatively extensive posterior lesions including the temporo-parieto-occipital junction of the left hemisphere but sparing the areas around the primary auditory cortex (Berthier, 1999). Damasio (1991)

observed that TSA is associated with lesions involving the temporal-occipital area (BA 37), the angular gyrus (BA 39), or the white matter underlying these regions, but sparing the primary auditory cortex (BA 41 and 42), and BA 22. Damasio suggested that the core area for TSA is the temporal-occipital area (BA 37) with variable extension to the occipital lobe and the angular gyrus. Kertesz (1982) analyzed 15 patients with TSA and proposed two different subgroups: one is more medial, inferior, and posterior and is clearly in the posterior cerebral artery territory; and the other is relatively more lateral, superior, and anterior and seems to be in a watershed area between middle cerebral and posterior cerebral arteries.

Benson and Ardila (1996), considering this variability in TSA, also distinguished two subtypes: the first one similar to Luria's amnesic aphasia (BA 37), and the second one corresponding Luria's semantic aphasia (BA 39). This distinction is coincidental with the neuroanatomical correlates of TSA found by Damasio (1991).

Recent reports support the assumption that TSA is usually found associated with extensive lesions of the left hemisphere (e.g., Warabi, Bandoh, Kurisaki, Nishio, & Hayashi, 2006), generally involving large portions of the temporal-parietal-occipital areas. According to Alexander, Hiltbrunner, and Fischer (1989) the critical lesion for transcortical sensory aphasia in these patients involved pathways in the posterior periventricular white matter adjacent to the posterior temporal isthmus, pathways that are most likely converging on the inferolateral temporo-occipital cortex.

TSA represents a disorder in the semantic recognition of language that may or may not be associated during the acute stage with other language impairments, specially logorrhea and jargon, depending on the extension of the lesion. But logorrhea and jargon are not required in the definition of TSA.

Supplementary Motor Area (SMA) Aphasia

This is a type of language disturbance recognized relative late in the aphasia history. Supplementary motor area (SMA) aphasia indeed is not associated with damage in the so-called "language area" of the brain. Penfield and Welch (1951) first observed arrest of speech associated with stimulation of this cortical region. Clinical characteristics of this type of aphasia were described by Rubens (1975, 1976).

Language disturbances in cases of damage of the left SMA have been characterized by, (1) an initial mutism lasting about 2–10 days; (2) later, a

virtually total inability to initiate speech, (3) a nearly normal speech repetition, (4) a normal language understanding, and (5) absence of echolalia. A right leg paresis and right leg sensory loss are observed; a mild right shoulder paresis and Babinski sign are also found. Language recovery is outstanding and it is usually observed during the following few weeks or months (Ardila & Benson, 1996; Rubens, 1975, 1976). The occlusion of the left anterior cerebral artery is the most frequent etiology, but it has also been reported in cases of tumors and traumatic head injury (e.g., Ardila & Lopez, 1984).

SMA is a premotor area (medial extension of BA 6) participating in initiating, maintaining, coordinating, and planning complex sequences of movements; it receives information from the posterior parietal and frontal association areas, and projects to the primary motor cortex (Kandel, Schwartz & Jessell, 1995). SMA damage is also associated with slow reaction time (Alexander, Stuss, Picton, Shallice, & Gillingham, 2007). It has been observed that activation of the SMA precedes voluntary movement (Erdler et al., 2000); a crucial role in the motor expression of speech processing has also been postulated (Fried et al., 1991). Nonetheless, the SMA is located some distance -and indeed far away- from the classic language area postulated by Dejerine (1914) and assumed in most anatomical models of aphasia.

Neuroimaging studies in humans have demonstrated that SMA is active when performing various cognitive tasks, such as spatial working memory (Jonides et al., 1993), verbal working memory (Paulesu, Frith, & Frackowiak, 1993), arithmetic tasks (Dehaene et al., 1996; Hanakawa et al., 2002), spatial mental imagery (Mellet et al., 1996), and spatial attention (Simon et al., 2002).

Evidently, the SMA is a complex motor cortical area, not primarily a language related brain area. Its role in language seemingly refers to the motor ability to initiate and maintain voluntary speech production.

CONCLUSION

From the above analysis it is evident that the term ‘‘aphasia’’ has been used to refer both to primary language disturbances, affecting the language system itself (phonology, lexicon, semantics, grammar), and to other impairments not affecting the language system itself, but affecting some abilities required for using language. Aphasia is usually interpreted and

understood as a disturbance in the language, not in some mechanisms required to produce and use the language.

A major distinction can be established between primary language disturbances (central aphasias, language is disintegrated), and secondary language disturbances resulting from ‘‘peripheral’’ impairments (secondary or ‘‘peripheral’’ aphasias; language is limited). In the primary aphasia the language itself is impaired. In the secondary language disturbances, some mechanism required to produce the language is altered. Sometimes language is not impaired, but the patient cannot use it appropriately because of executive control impairments (dysexecutive aphasia).

Table 2 presents a proposed interpretation and classification of aphasia syndromes. A distinction between primary aphasias (Wernicke-type and Broca-type; language is disintegrated as a paradigm --selection process--; or as a syntagm --sequencing process--) and secondary aphasias (conduction aphasia and aphasia of the supplementary motor area; language is limited in a specific aspect) is introduced; extra-Sylvian (or transcortical) motor aphasia is interpreted as a dysexecutive aphasia (the active use and executive control of the language is limited).

Table 2. A proposed new classification of aphasias. A distinction between primary aphasias (Wernicke-type and Broca-type) and secondary aphasias (conduction aphasia and aphasia of the supplementary motor area) is introduced; extra-Sylvian (or transcortical) motor aphasia is interpreted as a dysexecutive aphasia

Type	**Impairment**
Primary (central) aphasias	*Language system impaired*
Wernicke-type aphasia (fluent aphasia)	Phonological level
	Lexical level
	Semantic level
Broca-type aphasia(non-fluent aphasia)	Sequencing expressive elements at syntactic and phonetic level

Secondary (peripheral) aphasias	*Mechanisms of production impaired*
Conduction aphasia	Disconnection (or segmentary ideamotora verbal apraxia)
SMA aphasia	To initiate and maintain voluntary speech production
Dysexecutive aphasia	*Language executive control impaired*
Extra-Sylvian (transcortical) motor aphasia	Executive control of language

According to Ardila, 2010.

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