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Aphasia revisited: A reply to Buckingham, Kertesz, and Marshall

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Forum

A proposed reinterpretation and reclassification of aphasic syndromes

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Background: Different classifications of aphasic disorders have been proposed over the years. During recent decades new approaches to aphasia study have been developed, suggesting that current aphasia classifications can and should be reconsidered.
Aims: The purpose of this paper is to attempt to integrate contemporary knowledge about brain organisation of language and to propose a new aphasia classification.
Main Contribution: It is emphasised that there are 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). Grammar correlates with the ability to represent actions (verbs) and depends on what is known as Broca's area and its related brain circuits, but it is also related to the ability to quickly carry out the sequencing of articulatory movements required for speaking (speech praxis). Lexical/semantic and grammatical systems not only depend on different brain circuitries, but also on different types of memory and learning (declarative and procedural). Other aphasic syndromes do not really impair language knowledge per se, but rather 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).
Conclusions: A new classification of aphasic syndromes is proposed: primary (or "central") aphasias (Wernicke's aphasia—three subtypes—and Broca aphasia); secondary (or "peripheral") aphasias (conduction aphasia and supplementary motor area aphasia); and dysexecutive aphasia (extra-Sylvian—transcortical—motor aphasia), are distinguished.

Keywords: Aphasia classification; Wernicke's aphasia; Broca aphasia; Central aphasias; Peripheral aphasias; Dysexecutive aphasia.

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APHASIA CLASSIFICATIONS

Over 20 different aphasia classifications have been proposed since Broca's first report (Broca, 1863). Table 1 presents the main aphasia classification proposals presented in recent decades. However, two points should be emphasised: (1) some of these classifications have played a guiding role in clinical practice and language research; and (2) differences are frequently observed in the names used, not in the clinical characterisation of the aphasia syndromes.

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TABLE 1
Some recent classifications of aphasia syndromes

Luria, 1966	*Benson & Geschwind, 1971*	*Hécaen & Albert, 1978*	*Kertesz, 1979*	*Benson, 1979*	*Lecours et al., 1983*
Efferent motor	Broca's	Agrammatic	Broca's	Broca's	Broca's
Sensory	Wernicke's	Sensory	Wernicke's	Wernicke's	Wernicke's type I
Afferent motor	Conduction	Conduction	Conduction	Conduction	Conduction
Dynamic	Transcortical motor	Transcortical motor	Transcortical motor	Transcortical motor	Aspontaneity
—	Transcortical Sensory	Transcortical Sensory	Transcortical Sensory	Transcortical Sensory	Wernicke's type II
—	Isolation language area	—	Isolation	Transcortical mixed	—
Semantic Amnesic	Anomic	Amnesic	Anomic	Anomic	Amnesic
—	Global	—	Global	Global	—
—	Aphemia	Pure motor	—	Aphemia	Pure anarthria

Note. There are two popular aphasia tests that also include an aphasia classification: the Minnesota Test for the Differential Diagnosis of Aphasia (Schuell, 1953, 1973); and Aachen Aphasia Test (Huber, Poeck, Willmes, 1984; Willmes & Ratajczak, 1962).

The two most influential aphasia classifications are the Boston Group classification (by Geschwind, Benson, Alexander, Goodglass, Kaplan, and others); and Luria's aphasia interpretation. These two aphasia classifications have significantly guided the area during the last decades.

Boston Group classification developed from Wernicke's ideas, and two basic distinctions are included: (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) is introduced to account for the language repetition impairments frequently found in left parietal (or insular) damage.

Luria (1966, 1970, 1974, 1976) proposed six (or seven) aphasia subtypes. Until the 1960s he clearly distinguished six aphasia variants (motor efferent or kinetic, motor afferent or kinaesthetic, acoustic-agnosic, acoustic-amnesic, semantic, and dynamic) and suggested a seventh one (amnesic) (Luria, 1966, 1970). Later he overtly referred to seven aphasia subtypes (Luria, 1976, 1980). The reason for this ambiguity is that Luria was not sure if amnesic aphasia should be regarded as an independent aphasia syndrome; or rather, whether the naming impairments associated with semantic paraphasias should be included within semantic and/or acoustic-amnesic aphasia. In Luria's approach, aphasia subtypes and names refer to the specific level of language that is impaired (Table 2).

Benson and Ardila (1996) attempted to integrate the Boston and Lurian aphasia interpretations. They 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 with damage beyond this

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TABLE 2
Factors underlying different aphasic syndromes (according to Luria, 1976)

Type of aphasia	*Impaired factor*
Acoustic-agnosic	Phoneme discrimination
Acoustic-amnesic	Verbal memory
Amnesic	Semantic structure of the words
Semantic	Understanding logic-grammatical quasi-spatial relations
Afferent motor	Articuleme discrimination
Efferent motor	Kinetic melody of speech, inner speech
Dynamic	Verbal initiative

area (extra-Sylvian). Subtypes were introduced for some aphasia syndromes. Aphasias were also regarded as anatomical syndromes (Table 3). This classification is currently used by different authors (e.g., Basso, 2003).

Following contemporary cognitive and psycholinguistic models of acquired dyslexia (alexia)—which usually identify three main "central" dyslexia subtypes (phonological, surface, and deep) and two reading routes (direct and indirect) (e.g., Coltheart, 1980; Friedman, 1988; Friedman & Albert, 1985; Greenwald, 2001; Jefferies, Sage & Ralph, 2007; Marshall & Newcombe, 1966, 1973; Morton & Patterson, 1980; Patterson, 1978; Shallice, & Warrington, 1980)—the terms "phonological aphasia" (dysphasia), "surface aphasia", and "deep aphasia" are sometimes found in contemporary aphasia literature (e.g., Butterworth & Warrington, 1995, Wilshire & Fisher, 2004; McCarthy & Warrington, 1984, 2001; Michel & Andreewsky, 1983). This cognitive approach supposes that there are two routes to language repetition: a phonological route and a semantic route. The phonological representation of words is assumed to be functionally independent of the semantic system (McCarthy & Warrington, 2001). Phonological aphasia would be characterised by poor performance on auditory tasks with a strong phonological component, whereas in spoken word production tasks errors are mainly phonemic

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TABLE 3
Classification of aphasic syndromes (according to Benson & Ardila, 1996)

	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)

and formal paraphasias; single word repetition is poor but semantic errors are not found (Fisher & Wilshire, 2004). In surface aphasia repetition performance is affected by phonological similarity, word frequency, and stimulus lexicality, but is not affected by linguistic complexity, word length, or any semantic variable (McCarthy & Warrington, 2001). Deep aphasia is characterised by semantic errors when repeating speech (Butterworth & Warrington, 1995). This interpretation of language repetition errors assumes that the phonological and semantic spoken language systems have different brain representation and can become dissociated in cases of aphasia. Indeed, this interpretation approaches a specific language ability—language repetition—and, no question, it significantly contributes to further understanding its normal and abnormal brain organisation. But it is not clear if it can be regarded as a comprehensive aphasia classification.

During the last decades, in addition to the clinical descriptions and their anatomical correlates, new approaches to aphasia research have developed: Contemporary neuroimaging techniques, such as fMRI (e.g., Zahn et al., 2000), PET (Cao, George, Ewing, Vikingstad & Johnson, 1998) and tractography (Yamada et al., 2007), have been introduced, significantly extending our understanding of the organisation of language in the brain under normal and abnormal conditions (Lee, Kannan & Hillis, 2006; Small & Burton, 2002); an increased interest in identifying the disrupted cognitive processes underlying different impaired functions has been observed (Hillis, 2007); a significantly better understanding of the brain circuitries supporting language has been developed (e.g., Ullman, 2004); a re-analysis of the classical language areas (Broca's and Wernicke's) has been developed (e.g., Grodzinky & Amunts, 2006); genetic advances have posited new perspectives to the understanding of language organisation and language evolution (e.g., Vargha-Khadem, Watkins, Alcock, Fletcher & Passingham, 1995); and new scientific discoveries, such as the "mirror neurons", have changed our understanding of the functioning of the human brain, including language organisation (e.g., Rizzolati & Arbib, 1998).

In this paper an attempt will be made to integrate these advances to propose a re-classification of the aphasia syndromes.

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TWO MAJOR APHASIC SYNDROMES

Since the 19th century it has been well established that there are two major and fundamental aphasic syndromes, named in different ways, but roughly corresponding to Wernicke-type aphasia and Broca-type aphasia (e.g., Albert et al., 1981; Alexander & Benson, 1991; Bastian, 1898; Benson & Ardila, 1996; Freud, 1891/1973; Goldstein, 1948; Head, 1926; Hécaen, 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). *This is a most basic departure point in aphasia: Aphasia is not a single and unified clinical syndrome, but two rather different (even opposed) clinical syndromes.*

These two major aphasic syndromes have been related to the two basic linguistic operations: selecting (language as paradigm) and sequencing (language as syntagm) (Jakobson, 1971; Jakobson & Halle, 1956; Luria, 1972/1983). 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 similarity disorder restricts the patient's ability to *select* words on the paradigmatic axis. These patients (Wernicke-type aphasia) cannot find words that exist as parts of the system (vocabulary). These aphasic patients have severely limited access to this language repertoire system. Specific nouns tend to be inaccessible. These patients cannot select among alternative names (*apple*, *pear*, *banana*, etc) and may instead fill out their discourse with circumlocutions (a clock may be referred to as "*to know the time*"). Words no longer have a generic (paradigmatic) meaning for these patients, and speech becomes empty. A dog can be referred to as "*fox*", "*it barks*", etc.

Aphasic individuals presenting with what Jakobson referred to as contiguity disorder (Broca-type aphasia) lose the ability to *combine* linguistic elements. Their grammar is restricted or absent, and they can produce and understand only isolated meaningful words. Words with purely grammatical functions (such as articles and prepositions) tend to be omitted. Affixes may be substituted one for another but more likely they are simply not produced. These patients thus tend to use only very short sentences containing mostly meaningful words (nouns). In severe cases, sentences can be as short as a single word ("*dog*") and in general, there is a reduction in resources available for syntactic processing (Caplan, 2006). In the Spanish language with a significant flexibility in the word order, Broca aphasia is associated with difficulties in understanding sentences with a non-canonical word order (Ostrosky-Solis, Marcos-Ortega, Ardila, Rosselli, & Palacios, 1999). Luria further developed Jakobson's ideas in his paper "On the two basic forms of aphasic disturbances" (1972/1983). Luria emphasised that the selection disorder can be observed at different levels of language, corresponding to different aphasia subtypes: phoneme selection (aphasia acoustic agnosic), word selection (aphasia acoustic amnesic), and meaning selection (amnesic aphasia). By the same token, the contiguity disorder can be observed at different levels: sequencing words (kinetic motor aphasia—Broca aphasia) or sequencing sentences (dynamic aphasia—transcortical motor aphasia). Noteworthy, different subtypes of Wernicke's aphasia are frequently distinguished (e.g., Ardila, 2006a). Luria's acoustic agnosic, acoustic amnesic, and amnesic aphasia are indeed subtypes of the language impairment syndrome referred to as a whole as Wernicke's (sensory) aphasia.

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Wernicke-type aphasia

Wernicke-type aphasia represents the clinical syndrome characterised by impairments in the selection process (paradigmatic axis defect). In Wernicke's 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. Lexical (words) and semantic (meanings) associations become deficient. In Wernicke-type aphasia obviously 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's aphasia). Some times these patients present errors that could be interpreted as sequencing errors in phonemic paraphasias (such as "left-to-right" or "right-to-left" transpositions) (Buckingham, 1989), but the percentage of this type of error is very low (less than 1%

of the total number of phonemic errors, at least in Spanish language; Ardila, Montañés, Caro, Delgado, & Buckingham, 1989). Patients have problems in recalling the words (memory of the words) and also in associating the words with specific meanings. This means 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. Figure 1 presents in a summarised form the model proposed by Ardila (1993) to account for the language recognition impairments observed in cases of Wernicke-type of aphasia.

Nouns seem to depend on an organised pattern of brain activity. Contemporary clinical and neuroimaging studies have corroborated that different semantic categories are differentially impaired in cases of brain pathology. For instance, in anomia it has been traditionally recognised that naming body-parts, external objects, and colours depend (and are altered depending) on the activity of different brain areas (e.g., Hécaen & Albert, 1978). It has also been found that finer distinctions can be made with regard to naming impairments, which can be limited to a rather specific semantic category (e.g., people's names, living things, tools, geographical names, etc.) (e.g., Goodglass, Wingfield, Hyde, & Theurkauf, 1986; Harris & Kay, 1995; Lyons, Hanley, & Kay, 2002; Warrington & Shallice, 1984), and even as specific as "medical terms" (Crosson, Moberg, Boone, Rothi & Raymer, 1997). A brain "mapping" of the memory organisation of different semantic categories can be supposed.

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Broca-type aphasia

Broca-type aphasia represents the clinical syndrome characterised by impairments in the sequencing process (syntagmatic axis defect). It is usually recognised that Broca's aphasia has two different distinguishing characteristics: (a) a motor component (lack of fluency, disintegration of the speech kinetic melodies, verbal-articulatory

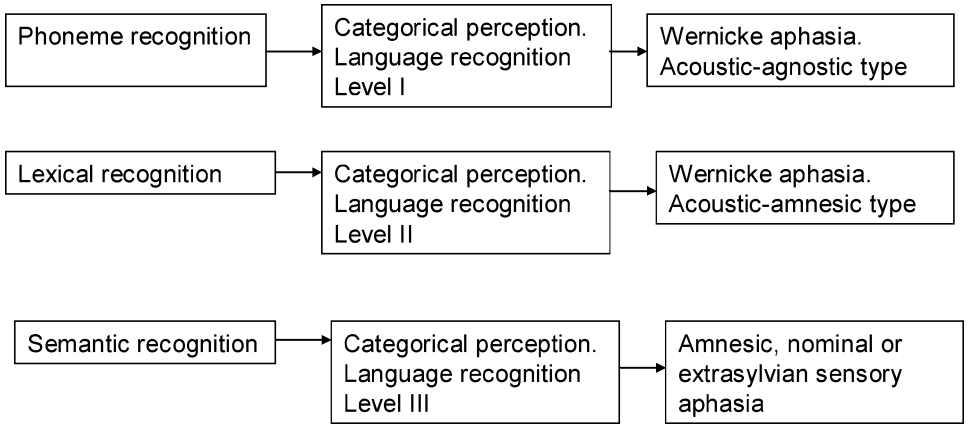

Figure 1. Three levels of language recognition potentially impaired in Wernicke-type aphasia can be distinguished: phonemic (categorical perception level I), lexical (categorical perception level II), and semantic (categorical perception level III). Three different sub-syndromes can be found: phonemic 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). (Adapted from Ardila, 1993.)

impairments, etc., that is usually referred as *apraxia of speech*); and (b) agrammatism (e.g., Benson & Ardila, 1996; Berndt & Caramazza, 1980; Goodglass, 1993; Kertesz, 1985; Luria, 1976). 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 specifically associated with damage in the left precentral gyrus of the insula (Dronkers, 1996; but see Hillis et al., 2004b) 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, including lengthening of fricatives, intercalation of schwas to break up difficult clusters, vocalic elongation due to an evening of syllable stress, and a host of other phonetic aberrations that are not necessarily the result of incorrect sequential ordering (Buckingham, 1998; Code & Ball, 1982).

If both impairments (apraxia of speech and agrammatism) are simultaneously observed (i.e., they are very highly correlated), it simply means 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 an "inability to sequence expressive elements" (Figure 2). A single common factor underlying both impairments may be assumed. Broca's area, most likely, is not specialised 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* (Borovsky, Saygin, Bates, & Dronkers, 2007).

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THE ISSUE OF BROCA'S AREA

In the last decade there has been a significant interest in re-analysing the function of Broca's area (e.g., Grodzinky & Amunts, 2006; Hagoort, 2005; Thompson-Schill, 2005). So-called Broca's area includes the pars triangularis (Brodmann's area—BA—44) and probably the pars opercularis (BA 45) of the inferior frontal gyrus (Foundas, Eure, Luevano, & Weinberger, 1998). BA 45 probably is more "cognitive" than BA 44, which seems to be more motor, more phonetic. From the traditional point of view, Broca's area corresponds to BA 44, but several contemporary authors also include BA 45. In the traditional aphasia literature it was assumed that damage in the Broca's area was responsible for the clinical manifestations observed in Broca's aphasia. Only with the introduction of the CT scan did it become evident that the damage restricted to the Broca's area was not

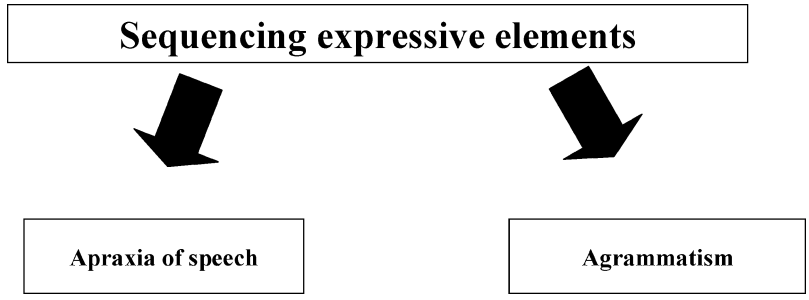

Figure 2. A single factor can account for the two major impairments observed in Broca aphasia. (Adapted from Ardila & Bernal, 2007.)

enough to produce the "classical" Broca's aphasia; extension to the insula, lower motor cortex, and subjacent subcortical and periventricular white matter is required (Alexander, Naeser & Palumbo, 1990). "Broca's area aphasia" ("minor Broca's aphasia") is characterised by mildly non-fluent speech, relatively short sentences and mild agrammatism; phonetic deviations and a few phonological paraphasias can be observed (Mohr et al., 1978); some foreign accent can also be noticed (Ardila, Rosselli, & Ardila, 1988).

Simultaneously including both BA 44 and BA 45 in Broca's area is problematic. BA 44 is a premotor dysgranular area, whereas BA 45 has a granular layer IV and belongs to the heteromodal prefrontal lobe (granular cortex) (Mesulam, 2002). So, from a cytoarchitectonic point of view, BA 44 and BA 45 are quite different. BA 44 is a premotor area whereas BA 45 corresponds to the prefrontal cortex. From the aphasia perspective, some authors have referred to different clinical manifestations associated with damage in BA 44 (Broca-type aphasia) and BA 45 (transcortical motor/dynamic aphasia) (e.g., Luria, 1976). Broca's area is, more than likely, involved in different language and language-related functions (Fink et al., 2006). Some authors have pointed out that indeed Broca's area is a collective term that can be fractionated in different sub-areas (Lindenberg, Fangerau & Seitz, 2007).

Hagoort (2005, 2006) refers to the "Broca's complex", including BA 44 (premotor), and also BA 45 and BA 47 (prefrontal cortex). He argues that Broca's complex is not a language-specific area, and it becomes active during some non-language activities, such as mental imagery of grasping movements (Decety et al., 1994). Functional defined sub-regions could be distinguished in the Broca's complex: BA 47 and BA 45 are involved in semantic processing, BA 44, BA 45, and BA 46 participate in syntactic processing, and BA and BA 44 are involved in phonological processing. Hagoort (2005) proposes that "the common denominator of the Broca's complex is its role in selection and unification operations by which individual pieces of lexical information are bound together into representational structures spanning multiword utterances" (p. 166). Its core function is, consequently, *binding the elements of the language*.

Thompson-Schill (2005) analysed the different deficits observed in cases of damage in the Broca's area: articulation, syntax, selection, and verbal working memory, suggesting that there may be more than a single function of Broca's area. Broca's area is related with several functions, and different disturbances are found in differing cases of pathology. The author proposes a framework for describing the deficits observed in different patients. The proposed framework suggests that Broca's area may be involved in *selecting information among competing sources*. Fadiga, Craighero, and Roy (2006) speculates that the original role played by Broca's area relates to *generating/extracting action meanings*; that is, organising/interpreting the sequence of individual meaningless movements. Ardila and Bernal (2007) conjectured that the central role of Broca's area was related to *sequencing* motor/expressive elements. Novick, Trueswell, and Thompson (2005) consider that the role of Broca's area is related with a general *cognitive control* mechanism for the syntactic processing of sentences.

Grodzinsky (2000, 2006) has presented an extensive analysis of the role of Broca's area. He proposed that most syntax is not located in Broca's area and its vicinity (operculum, insula, and subjacent white matter). This brain area does have a role in syntactic processing, but a highly specific one: *it is the neural home to receptive*

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mechanisms involved in the computation of the relation between transformationally moved phrasal constituents and their extraction sites (syntactic movement). He further assumes that Broca's area is also involved in the construction of higher parts of the syntactic tree in speech production. Interestingly, blood flow in Broca's area increases when participants process complex syntax (Caplan, Alpert, Waters, & Olivieri, 2000). Santi and Grodzinsky (2007) also recognise its role in working memory related with a specific syntactic role in processing filler–gaps dependency relations. Syntax is indeed neurologically segregated, and its components are housed in several distinct cerebral locations, far beyond the traditional ones (Broca's and Wernicke's regions). A new brain map for syntax would also include portions of the right cerebral hemisphere (Grodzinsky & Friederici, 2006).

Haverkort (2005) emphasises that a clear distinction should be established between linguistic knowledge and linguistic use. Patients with Broca's aphasia have a limitation in the use of grammar, but their grammatical knowledge is available. Broca's aphasia patients present a simplified syntax and phrases are short. They select simpler syntactic structures that are less complex because they impose less burden on working memory. In consequence, one major factor in Broca's aphasia relates to an impairment in verbal working memory.

In summary, regardless that expressive language disturbances have been associated for over a century with damage in the left inferior frontal gyrus (later known as "Broca's area"), currently there is incomplete agreement about its limits and its specific functions in language. Different proposals have been presented to explain language disturbances in so-called Broca's aphasia, including: binding the elements of the language (Hagoort, 2005), selecting information among competing sources (Thompson-Schill, 2005), generating/extracting action meanings (Fadiga et al., 2006); sequencing motor/expressive elements (Ardila & Bernal, 2007); cognitive control mechanism for the syntactic processing of sentences (Novick et al., 2005); construction of higher parts of the syntactic tree in speech production (Grodzinsky, 2000, 2006); and verbal working memory (Haverkort, 2005).

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WHAT ABOUT CONDUCTION APHASIA AND OTHER APHASIA SYNDROMES?

Frequently it is assumed that three major aphasic (perisylvian) syndromes can be distinguished: Broca's aphasia, Wernicke aphasia, and 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. Different 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; Hécaen & Albert, 1978; Kertesz, 1979; Lecours, Lhermitte, & Bryans, 1983; Luria, 1966).

Indeed, some aphasic syndromes can eventually be considered as variants of the Broca's and Wernicke aphasias. For instance, amnesic or anomic or nominal aphasia (usually due to damage in the vicinity of BA 37) (Head, 1926; Hécaen & Albert, 1978; Luria, 1976) can be interpreted as a subtype of Wernicke aphasia in which the semantic associations of the words are significantly impaired (see Figure 1).

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 and the elements included in its definition. Some authors have even simply denied the existence of such a syndrome.[1] Currently, two integrative revisions of TSA are available (Berthier, 1999; Boatman et al., 2000).

TSA is defined in two partially different ways; (1) according to its "basic" definition, TSA is a fluent language disorder characterised 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). So, the question is: Should semantic jargon be included in the definition of TSA? Kertesz (1985, p. 317) makes a comprehensive definition of TSA: "TSA is characterised 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" (italics are mine). This definition clearly recognises 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

[1] Once somebody asked Luria: "Why you do not ever refer to transcortical sensory aphasia? Luria simply answered, "because I never have seen a case of transcortical sensory aphasia" (personal observation).

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temporal-occipital area (BA 37) with variable extension to the occipital lobe and the angular gyrus. Crossed TSA has also been reported (e.g., Roebroek, Promes, Korten, Lormans, & van der Laan, 1999). Some similarities between so-called "thalamic aphasia" and TSA have been suggested (e.g., Berthier, 1999; Cappa & Vignolo, 1979).

Kertesz (1982) analysed 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. McCarthy and Warrington (1984) reported two patients with impaired paraphasic performance in repetition tasks but relatively well-preserved spontaneous speech (conduction aphasia); and one patient presenting impaired paraphasic spontaneous speech but intact repetition (transcortical motor aphasia). When active semantic processing was required the conduction aphasics were facilitated and the transcortical motor aphasic impaired; in tasks requiring passive repetition the opposite pattern of dissociation was observed. Departing from these observations, the authors proposed a two-route model of the speech production process. Coslett, Roeltgen, Gonzalez-Rothi, and Heilman (1987) proposed that repetition may be mediated by at least two distinct processes—a lexical process that may involve the recognition and subsequent activation of discrete stored word representations and a non-lexical process that involves phonologic decoding and immediate phonologic encoding from immediate memory. Analysing spontaneous speech, reading, and the tendency to recognise and spontaneously correct syntactic errors in four patients with TSA, Coslett et al. (1987) concluded that there are two subtypes of TSA. In one subtype both the lexical and direct repetition (or speech production) mechanisms are preserved, but in the second subtype the lexical mechanism is disrupted and repetition is mediated by the non-lexical mechanism. 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. However, frequently the lesions responsible for TSA account for the variability observed in its clinical manifestations, suggesting that TSA does not necessarily represent a single aphasic syndrome. When the lesions are restricted to BA 37 or BA 39, specific and well-described language impairments are observed (Benson & Ardila, 1996; Luria, 1976). With more extended lesions additional clinical manifestations, such as jargon, can be found. These additional clinical manifestations *are only observed in the acute stage of the brain pathology*, and progressively disappear (Kertesz, 1979). Dronkers and Larsen (2001) state that "transcortical sensory aphasia always resolves into mild anomic aphasia" (p. 29).

Thus, 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,

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specially logorrhoea and jargon, depending on the extension of the lesion. But logorrhoea and jargon are not required in the definition of TSA.

However, surprisingly TSA is also reported in cases of frontal damage (e.g., Berthier, 1999; Sethi, Burke, Torgovnick, & Arsura, 2007; Maeshima, Osawa, Nakayama, & Miki, 2004; for an extensive review of these cases, see Berthier, 2001). Berthier (2001), departing from the cases reported in the literature and his own patients, concluded that "syndrome of TSA associated with frontal lobe lesions is phenomenologically similar to the 'classical' variant, except for the presence in the former of some deficits in speech articulation, prosody, phonology, and skilled buccofacial movements indicative of frontal lobe involvement" (p. 124). Otsuki et al., 1998) compared 7 patients with TSA following the left frontal lesions (F) and 10 TSA patients following the left posterior lesions (P). The results indicated that the disturbance of the P group was of semantic processing, per se. And the disturbance of F was not only in semantic processing but also in the working memory process.

It sounds quite intriguing that an aphasia syndrome can have two clearly different brain localisations. It seems reasonable to assume that both cortical areas (frontal and posterior) should be included in a common brain circuitry. The similarity in the language disturbances suggests that such a particular circuitry has been impaired at different points. Zahn and colleagues (2000), using fMRI in normal participants, observed that the areas specific to semantic processing were restricted to the left hemisphere: the posterior middle frontal (BA 9) and posterior parietal (BA 7/40) cortex, as well as an inferior temporal area (BA 20/21). They further analysed two cases of TSA with lesions affecting either solely the prefrontal (Patient 1) or both the prefrontal and posterior parietal part of the network activated in normal participants (Patient 2) (Zahn et al., 2002). Both patients showed TSA on acute assessment. Their recovery of language comprehension was associated with activation of a left hemispheric network. Mainly activations of left perilesional prefrontal regions (Patient 2), left Wernicke's area (Patient 2 and Patient 1), or the left posterior middle and inferior temporal cortex (Patient 1) were demonstrated in the TSA patients.

The observation that language can be impaired at the semantic level, with damage located at rather different areas, reinforces the idea that semantics is represented in a complex brain circuit including not only temporal, but also parietal and frontal areas (e.g., Damasio, Tranel, Grabowski, Adolphs, & Damasio, 2004; Scott, 2005; Small, Hart, Nguyen, & Gordon, 1995). Indeed, different brain areas participate in language comprehension, including the posterior middle temporal gyrus and underlying white matter, the anterior superior temporal gyrus, the superior temporal sulcus and angular gyrus, mid-frontal cortex in BA 46, and BA 47 of the inferior frontal gyrus. The middle temporal gyrus may be more important for comprehension at the word level, while the other regions may play a greater role at the level of the sentence (Dronkers, Wilkins, Van Valin, Redfern, & Jaeger, 2004).

Transcortical (extra-Sylvian) motor aphasia as a "dysexecutive aphasia"

Some aphasic syndromes can be interpreted as language disturbances due to a more general underlying disorder. For instance, extra-Sylvian (transcortical) motor aphasia associated with left convexital prefrontal damage 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,

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lexicon, semantics, and grammar are preserved. Simply speaking, the question is: 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 organise language sequences can be interpreted 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) Some authors (Luria 1976, 1980) have emphasised 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, 2006). 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). In this regard, it is a secondary—not primary—aphasia 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). Recently, 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.

It is noteworthy that successful functional communication is significantly associated with executive function in aphasia (Fridriksson., Nettles, Davis, Morrow, & Montgomery, 2006). The ability for successful communication in aphasic patients is apparently dependent on the integrity of executive function skills (Purdy, 2002).

Conduction aphasia as a secondary ("peripheral") aphasia

A crucial question is, how can conduction aphasia—a well recognised and extensively studied aphasic syndrome (e.g., Benson & Ardila, 1994; Damasio & Damasio, 1980; Goldstein, 1948; Kohn, 1992)—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 in an indirect pathway passing through the inferior parietal cortex (Catani, Jones, & Ffytche, 2005). 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

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required for speaking, or simply as a kinaesthetic apraxia of speech. Luria (1976) suggested that paraphasias in conduction aphasia (Luria's kinaesthetic motor or afferent motor aphasia) are articulatory-based (articulatory literal paraphasias). These errors 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 recognises 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. 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 a general and crude distinction. Ardila and Rosselli (1992) analysed 38 Spanish-speaking aphasic patients divided into six aphasia groups (transcortical motor, Broca's, conduction, Wernicke, anomic, and global). Three repetition tests (words, high-probability and low-probability sentences) taken from the Boston Diagnostic Aphasia Examination, Spanish version (Goodglass & Kaplan, 1979) were administered. Repetition errors were generally associated with perisylvian aphasias (Broca's, conduction, and Wernicke's). However, in *all* aphasic groups some repetition errors were observed (Table 4). They were not only quantitative but also qualitatively different. It was concluded that, depending on the specific repetition task, errors may be evident or may be unnoticed in a particular aphasic group. Different mechanisms underlying repetition deficits were proposed: limitation of auditory-verbal short-term memory, difficulties at the level of phonological production, impairments in phoneme recognition, and semantic and syntactic comprehension.

It is interesting to note that in the classification of reading and writing disturbances, a frequent distinction is made between "central" and "peripheral" alexias (dyslexias), and agraphias (dysgraphias) are introduced (e.g., Ellis & Young, 1988). Central alexias and agraphias impair the knowledge of written language (recognition or production), whereas peripheral alexias and agraphias impair the

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TABLE 4
Percentage of correct repetition for each patient group on the three repetition tasks from the Boston Diagnostic Aphasia Examination.

	Words	*High-Probability*	*Low-Probability*
Transc motor ($n = 6$)	98.0	95.0	67.5
Broca ($n = 5$)	46.0	50.0	45.0
Conduction ($n = 6$)	63.0	53.7	21.2
Wernicke's ($n = 13$)	74.0	45.0	22.5
Anomic ($n = 4$)	100.0	71.2	52.5
Global ($n = 4$)	27.0	0.0	0.0

Adapted from Ardila and Rosselli, 1992.

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normal access to written information (in cases of alexias) or the mechanisms required for written language production (in cases of agraphias). Central agraphias are also named "linguistic" or "aphasic" agraphias, whereas peripheral agraphias are referred to as "non-linguistic" or "non-aphasic" agraphias (Benson & Ardila, 1996).

Could a similar distinction be introduced in cases of oral language disturbances? Indeed, a similar rationale could be used in the case of conduction aphasia: conduction aphasia is not a primary form of aphasia, but rather a secondary (or "peripheral") defect in language indirectly affecting specific language abilities (i.e., the ability to repeat). Language itself is not impaired, but rather it is 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. The correct visual-perceptual recognition of the written information is also an important ability required not only in learning to read, but also in correctly accessing written information. 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, I am proposing that conduction aphasia 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 parrots 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, in conduction aphasia language is limited, not disintegrated.

Supplementary motor area (SMA) aphasia

In 1940 Brickner reported that electro-cortical stimulation of SMA (mesial aspect of Brodmann's area 6) resulted in continuous perseveration. Penfield and Welch (1951) observed arrest of speech associated with stimulation of this cortical region. However, language disturbances associated with SMA pathology are reported relatively late in the aphasia literature.

Clinical characteristics of this type of aphasia were described by Rubens (1975, 1976). Jonas (1981) later referred to the participation of the SMA in speech emission.

The occlusion of the left anterior cerebral artery is the most frequent aetiology, but it has also been reported in cases of tumours and traumatic head injury (e.g., Ardila & López, 1984). Speech is characterised 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.

The SMA is a mainly mesial premotor area involved in the ability to sequence multiple movements performed in a particular order (Tanji & Shima, 1994). SMA participates 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.

Recently it has been suggested that SMA has a close connectional relationship with the prefrontal cortex and plays a critical role in the update of verbal representations (Tanaka, Honda, & Sadato, 2005). 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.

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The question of subcortical aphasia

Since Wernicke (1874) it has been assumed that aphasia can represent the consequence of damage to neural networks including both cortical and subcortical structures. Nonetheless, when Dejerine (1914) described the brain's "language area" no specific mention to subcortical structures was made. The idea of "subcortical aphasia" was somehow forgotten during the following decades. Only with the introduction of the CT scan was it observed that aphasia was frequently associated with subcortical pathology, and the discussion and interpretation of subcortical aphasia re-emerged (e.g., Basso, Della Sala, & Farabola, 1987; Cappa, Cavallotti, Guidotti, Papagno, & Vignolo, 1983; Nasser et al., 1982). Contemporary neuroimaging techniques have permitted far better understanding of subcortical pathology involved in aphasia. Nonetheless, whether true aphasia results from isolated subcortical brain damage, or whether it is due to a cortical extension or cortical deactivation, remains unanswered (e.g., Craver & Small, 1997; Démonet, 1997). Subcortical pathology frequently includes altered speech (dysarthria), often beginning with total mutism followed by hypophonic, slow, sparse output, and

poorly differentiated, amelodic speech (Alexander, Naeser, & Palumbo, 1987). In addition to dysarthria, sometimes language impairments are also found. Two neuroanatomical areas are most frequently discussed in subcortical aphasias: the striatocapsular region and the thalamus. Patients with striatocapsular damage show significant articulation impairments. Their language output appears truncated, but it is not agrammatic. Speech mechanisms are generally impaired resulting in impairments in articulation and prosody. Comprehension is intact for casual conversation but breaks down when complex syntax is presented. Word-finding problems may be noted (Alexander et al., 1987). Alexander and colleagues (1987) have proposed six subtypes of verbal output impairment. These subtypes are dependent on the specific neuroanatomical locus of striatocapsular damage, demonstrating that considerable variation in speech and language impairment can follow this type of pathology. Frequently, extension that involves the cortex is present in these cases. Extensive subcortical damage is required to produce a pure striatocapsular aphasia, but prognosis is worse when the posterior limb of the internal capsule is involved (Liang et al., 2001). When no cortical abnormalities are found on MRI, the severity of the left striatocapsular aphasia is associated with the extent and severity of the left cerebral cortical hypoperfusion on brain perfusion SPECT, particularly in the left temporal cortex (Choi et al., 2007).

Mega and Alexander (1994) evaluated 14 cases of striatocapsular aphasia. The clinical profiles of the patients were quite similar, varying in severity in rough proportion to lesion size and varying in quality in proportion to anterior paraventricular extent. Large lesions were associated with impaired "executive" and "generative" language functions. Similar aphasia profiles in patients with deep frontal and paraventricular white matter lesions suggest that damage to a frontal-caudate functional system underlies a "core" aphasia profile in these patients. D'Esposito and Alexander (1995) analysed 13 patients with left putaminal haemorrhage. There was no apparent correlation between lesion site and acute language profiles. During the post-acute epoch there were several distinct correlations between lesion site and specific aphasia dimensions. Nadeau and Crosson (1997), after a critical review of the literature, suggested that linguistic impairments associated with striatocapsular pathology are predominantly related to sustained cortical hypoperfusion and infarction not visible on structural imaging studies.

It has been speculated that this heterogeneity in the aphasia pattern in cases of basal ganglia pathology may reflect variations in cortical hypoperfusion resulting from large vessel stenosis. To test this hypothesis, Hillis et al. (2004a) analysed a consecutive series of 24 patients with left caudate infarct identified with diffusion-weighted imaging who underwent language testing and perfusion-weighted imaging less than 24 hours from onset of symptoms. Specific regions in perisylvian cortex were rated for the percentage of the region that was hypoperfused. Aphasia type was determined on the basis of speech fluency, comprehension, and repetition performance on the language tests. Results demonstrated that in patients with acute left caudate infarct the presence and type of aphasia reflected regions of hypoperfusion and generally followed predictions based on chronic lesion studies regarding anatomical lesions associated with classic aphasia types.

Thalamic pathology associated with aphasia usually produces an acute, catastrophic clinical picture with hemiplegia, hemisensory loss, and alterations in the level of consciousness (Benabdeljlil et al., 2001). The initial language abnormality

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is mutism, which typically improves to a verbose, paraphasic, but hypophonic jargon output. Anomia is often severe. Although thalamic aphasia resembles other fluent paraphasic aphasias, patients with thalamic aphasia show decreased comprehension. When they attempt to repeat a word or phrase, their verbal output is far better than their conversational speech. A similarity to extra-Sylvian sensory aphasia has been noted, even though syntactic impairments have also been reported (De Witte, Wilssens, Engelborghs, De Deyn, & Marien, 2006). Language disorder syndromes noted following thalamic damage can be categorised into three subtypes: (1) medial (left paramedial thalamic area, involving dorsomedial and centromedian nuclei), (2) anterior (left anterolateral nucleus), and (3) lateral (left lateral thalamus). It has been suggested that thalamic nuclei and systems are involved in multiple processes that directly or indirectly support cortical language functions: lexical-semantic functions, working memory, visual processing in reading, and category-specific naming (Crosson, 1999). It has been further proposed that the left thalamus seems to bring online the cortical network involved in language processing (Metz-Lutz et al., 2000).

In brief, aphasia is sometimes associated with subcortical lesions, particularly left striatocapsular and thalamic pathology. To account for subcortical aphasia it has been proposed that aphasia may result from a cortical extension or cortical deactivation. The idea of a cortical deactivation seems to prevail (e.g., Choi et al., 2007; Hillis et al., 2004a). The question of subcortical aphasia suggests the existence of cortical-subcortical circuits in language, as observed in other forms of cognition (e.g., Cummings, 1993; Lichter & Cummings, 2001). The analysis of subcortical aphasia can significantly advance the understanding of the language representation in the brain, but indeed it does not affect the issue of aphasia classification.

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TWO BRAIN LANGUAGE SYSTEMS: PRIMARY APHASIAS

It may be conjectured that language is supported by two different brain systems. Lesions in each will result in Wernicke-type or Broca-type aphasia. These two brain language systems correspond to the lexical/semantic (selecting; language as paradigm) and morphosyntactic (sequencing; language as syntagm). Ontogenetically, they appear at different ages during child language acquisition: the ability to use single words for communication (holophrasic period) is observed around age 12–18 months; whereas the ability to combine several words in a single sentence is found later, around age 24–30 months (Hoff, 2003). Phylogenetically, they probably developed at different stages in evolution: whereas language as a lexical/semantic system may have appeared before the contemporary *Homo sapiens*, language as a grammatical system may have appeared relatively recently in human history (maybe some 10–100 thousand years ago) and seems to be exclusive to *Homo sapiens* (Ardila, 2006b).

Brain representation of nouns and verbs

It has been observed that verbs and nouns clearly depend on different areas of brain activity, and naming objects and actions are disrupted in different types of brain pathology. Increased activity is observed in the temporal lobe while speaking or thinking in nouns, whereas speaking or thinking in verbs activates Broca's frontal area (Raichle, 1994). By the same token, impairments in finding nouns are associated with temporal lobe pathology, whereas impairments in finding verbs are associated

with left frontal damage and Broca's aphasia (Ardila & Rosselli, 1994; Damasio & Tranel, 1993).

Naming actions activates the left frontal operculum roughly corresponding to Broca's area (Damasio et al., 2001). The neural correlates of naming concrete entities such as tools (with nouns) and naming actions (with verbs) are partially distinct: the former are linked to the left inferotemporal region, whereas the latter are linked to the left frontal opercular and left posterior middle temporal regions (Tranel, Martin, Damasio, Grabowski, & Hichwa, 2005). Recently Luzzatti, Aggujaro, and Crepaldi (2006) studied the lesions causing impairments related predominantly to verbs or nouns. Results obtained on these studies indicated that lesions causing impairment predominantly with nouns were located mostly in the middle and inferior left temporal area. Three alternative lesion sites were associated with a verb deficit (left posterior temporo-parietal lesions; large left fronto-temporal perisylvian lesions; deep lesions of the insula and/or the basal ganglia). Arévalo et al. (2007) studied the processing of words and pictures representing actions and objects in aphasic patients and normal controls. Using a manipulability dimension (i.e., items that can/cannot be manipulated and actions that do/do not involve fine hand movements) they found that aphasic patients displayed relative difficulty with the "manipulable" items, while controls displayed the opposite pattern.

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"Mirror neurons" and language evolution

A mirror neuron is a neuron that fires both when an animal performs an action and also when the animal observes the same action performed by another animal. In monkeys, the rostral part of the ventral premotor cortex (area F5) contains neurons that discharge both when the monkey grasps or manipulates objects and when it observes the experimenter making similar actions. These neurons (mirror neurons) appear to represent a system that matches observed events to similar, internally generated actions. In humans, brain activity consistent with mirror neurons has been found in the premotor cortex and the inferior parietal cortex (Rizzolatti & Craighero, 2004; Rizzolatti, Fadiga, Gallese, & Fogassi, 1996).

Transcranial magnetic stimulation and positron emission tomography (PET) experiments suggest that a mirror system for gesture recognition also exists in humans and includes Broca's area (Rizzolati & Arbib, 1998). The discovery of mirror neurons in Broca's area might have immense consequences for understanding brain language organisation and language evolution (Arbib, 2006; Craighero, Metta, Sandini & Fadiga, 2007). An obvious implication of mirror neurons is that they can participate in the internal representation of actions.

Genetics of grammar

Recently a milestone observation was made that significantly enlightened our understanding about the origin of language in general and grammar in particular. In England it was found that about half the members of three generations of a family, usually referred to as the KE family, had presented a significant disturbance in language development. Speech was largely unintelligible; hence they were taught sign language as children to supplement their speech. Affected members presented severe disturbances in articulation and other linguistic skills, and broader intellectual and physical problems. From the genetic point of view the disorder was associated with a

mutation in a single autosomal-dominant gene, FOXP2, located on chromosome 7 (Vargha-Khadem et al., 1995). The disorder was not restricted to speech and also included the following characteristics: impairments in processing words according to grammatical rules; understanding of more complex sentence structure such as sentences with embedded relative clauses; inability to form intelligible speech; impairments in the ability to move the mouth and face not associated with speaking (relative immobility of the lower face and mouth, particularly the upper lip); and significantly reduced IQ in the affected compared with the unaffected in both the verbal and the non-verbal domain.

Further, affected family members presented a pronounced developmental apraxia. The authors refer to the core deficit as one involving sequential articulation and orofacial praxis (Vargha-Khadem et al., 1998; Vernes et al., 2006, 2007). PET study revealed functional abnormalities in both cortical and subcortical motor-related areas of the frontal lobe, while quantitative analyses of MRI revealed structural abnormalities in several of these same areas, particularly the caudate nucleus which was found to be abnormally small bilaterally. An abnormal gene (SPCH1) in the chromosomal band 7q31 was localised. The genetic mutation or deletion in this region was proposed to result in marked disruption of speech and expressive language, including grammar (Fisher, Vargha-Khadem, Watkins, Monaco, & Pembrey, 1998). Corballis (2004) suggests a possible link between FOXP2 and the mirror-neuron system observed in the primate homologue of Broca's area. He proposes that mutation of FOXP2 was the most recent event in the incorporation of vocalisation into the mirror system, and thus the refinement of vocal control to the point that it could carry the primary burden of language. Ardila (2009) has suggested that language developed in two different stages: initially as a lexical/semantic system, and only later as a grammatical system. Recent genetic observations (Enard et al., 2002) have suggested that about 10,000–100,000 years ago there may have occurred a mutation in a single autosomal-dominant gene, FOXP2, located on chromosome 7; this mutation has been proposed to be linked to grammatical language evolution.

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Memory systems for nouns and verbs

Different memory systems for nouns and verbs can be conjectured. As a matter of fact, two major memory systems are frequently distinguished in contemporary memory literature: Declarative memory (divided into semantic and episodic or experiential) and procedural memory (Tulving, Fergus, & Craik, 2004). It has been suggested that the lexical/semantic and grammar aspects of the language are subserved by different neuroanatomic brain circuitries and depend on these two different memory systems (Fabbro, 1999, 2001; Paradis, 2004; Ullman, 2001, 2004). Whereas lexical/semantic aspects of language depend on declarative semantic memory (knowledge about the meaning of the words), grammar depends on procedural memory. Lexical/semantic aspects of the language (language as a paradigm) are explicitly learned, and represent a type of knowledge we are aware of (declarative memory). This depends on the retro-Rolandic cortical structures and the hippocampus. Grammar (language sequences, contiguity) is acquired incidentally. Procedural memory for grammar supposes implicit language knowledge. Procedural grammatical learning is related to the execution of sequences of elements (skilled

articulatory acts and grammar) used for speaking and also for syntax. Procedural memory is related with frontal/subcortical circuitries (Tulving et al., 2004).

Using verbs and using grammar as a single ability

Broca's area damage results in an impairment of grammar and also in an inability to find verbs. In consequence, brain representation of actions and brain representation of grammar is coincidental. *Using verbs and using grammar depends on the very same type of brain activity* and both are simultaneously disrupted in cases of Broca's aphasia. It can be conjectured that verbs and grammar appeared closely or simultaneously in human language evolution; or rather, they are two sides of the same coin. Furthermore, grammar is associated with oral praxis skills (i.e., agrammatism and apraxia of speech appear simultaneously in Broca's aphasia; and language networks supporting both are overlapped; Borovsky et al., 2007), and hence it seems reasonable to suppose that all three emerged simultaneously in the evolution of human language: using verbs, using grammar, and rapidly sequencing movements with the articulatory organs. The observation of the KE family seems to support this inter-relationship.

However, there is a foundation condition for using verbs—the ability to internally represent actions. That is, to internalise ("to think") the actions. One can wonder if "mirror neurons" may be involved in this internal representation of actions. Neuroimaging data have shown that interactions involving Broca's area and other cortical areas are weakest when listening to spoken language accompanied by meaningful speech-associated gestures (hence, reducing semantic ambiguity), and strongest when spoken language is accompanied by self-grooming hand movements or by no hand movements at all, suggesting that Broca's area may be involved in action recognition (Skipper, Goldin-Meadow, Nusbaum, & Small, 2007).

The obvious question at this point is: Can Broca's aphasic patients internally represent actions? Or does Broca's aphasia also imply an inability to internally represent ("think") in actions? Even though specific research on this question is not readily available, the answer seems to be *no*. Some observations point to a deficit in internally representing actions in Broca's aphasia. For example Ardila and Rosselli's (1994) patient had to make the concrete action to retrieve the corresponding verb. Hence, the internal representation of actions and the understanding/using of verbs seem to be closely related abilities. Noteworthy, Broca's area becomes active during the mental imagery of grasping movements (Decety et al., 1994).

Interestingly, Luria (1976) supposed that Broca's aphasia (efferent or kinetic motor aphasia) was the result of two associated impairments: impairment in the kinetic melody of speech (kinetic apraxia), and disturbances in inner speech. Inner speech is condensed and abbreviated internal speech, presumably responsible for self-awareness, problem solving, and thinking. According to Vygotsky (1978, 1986) inner speech follows a specific developmental sequence: first there is social speech (communication with other people), then comes egocentric speech (that disappears around the age of 7 or 8), and finally inner speech is formed. Inner speech represents a major instrument of metacognition and self-control. PET studies have associated the neural correlates of inner speech with activity of Broca's area (McGuire et al., 1996). This is an indirect demonstration that patients with Broca's aphasia may

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present with an impairment in inner speech and thinking in actions. Some investigators (e.g., Levine, Calvanio, & Popovics, 1982) have overtly pointed out that damage of the left inferior frontal area is associated with a loss of inner speech. As an interesting clinical observation, a patient with a paroxysmal Broca's aphasia reported that frequently she had the seizures when she was alone; on these occasions she knew she was having an epileptic seizure because her mind became blank, she had no idea in mind, and was sure that, if she had attempted to speak, she would be unable to produce anything (Ardila & Lopez, 1988).

The question of variability in aphasic syndromes

For some time reports have been published describing unusual and unexpected localisation of aphasia syndromes (e.g., Basso, Lecours, Moraschini, & Vanier, 1985; Vignolo, Boccardi & Caverni, 1986). Vignolo et al. (1986), for instance, studied 37 stroke patients with global aphasia: it was found that while 22 patients presented the expected large lesions including Broca's and Wernicke's areas, 8 had anterior lesions sparing Wernicke's area, 3 had posterior lesions sparing Broca's area, and 4 had deep lesions centred on the insula and lenticular nucleus. It is noteworthy that *all* the patients were evaluated within 2 months after the aphasia onset.

In other aphasic syndromes different localisation of the pathology has been reported. For instance, in TSA two rather different localisations of brain pathology have been described (e.g., Berthier, 2001). The brain lesion responsible for conduction aphasia can be not only left parietal but also insular (Damasio & Damasio, 1980).

These unusual cases are obviously intriguing and deserve special attention. Variability in the clinical manifestations of aphasia is nonetheless limited, and aphasic syndromes are usually observed in cases of relatively specific brain pathology: damage in BA 37 results in anomia associated with semantic paraphasias; damage in or around BA 41 and 42 results in phoneme discrimination impairments, etc. Lesion location is the main determinant of aphasic disorders even at the acute stage. Kreisler et al. (2000) examined 107 stroke patients with aphasia using a standardised aphasia battery and MRI, and concluded that most clinical–anatomical correlations support the classic anatomy of aphasia. How can these unusual cases be explained? We do not have a clear and direct answer to this question, but it can be speculated that at least three different factors might account for this variability:

1. Aphasic symptomatology during the acute stage is more extended and complex due to diaschisis (Von Monakow, 1914). Unusual aphasic syndromes are generally observed during the acute stage of brain pathology (e.g., Vignolo et al., 1986), Jargon and logorrhoea in TSA, for example, are only observed during the acute stage (Dronkers & Larsen, 2001; Kertesz, 1979), suggesting that other brain areas are also involved. The unusual aphasia manifestations may be the result not only of focal brain pathology but also of more extended brain dysfunction due to diaschisis.

2. The brain is a dynamic system and different areas are heavily interconnected. Recent neuroimaging techniques, particularly fMRI, have shown that there are some brain functions (i.e., simple) that can be localised into single brain areas whereas there are others (i.e., complex) that cannot (e.g., Ardila & Bernal, 2007;

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Cabeza & Nyberg, 2000). Complex functions depend on the simultaneous activity of different brain areas, or more exactly, brain circuits. For example, TSA is observed in cases of parietal-temporal-occipital damage, but also in cases of frontal damage (Berthier, 2001), suggesting that both areas participate in a single circuit or brain system involved in naming. Another example: agrammatism and decreased verbal fluency are typically found in cases of Broca's aphasia, but they can also be observed in cerebellar pathology (Akshoomoff, Courchesne, Press, & Iragui, 1992; Daum & Ackermann, 1995; Silveri, Leggio, & Molinari, 1994). A neural cerebellar-frontal loop affecting cognitive, especially linguistic, functions has been suggested. It has been hypothesised that the cerebellum contributes to cognitive processing, particularly the processing of linguistic information, because the cerebellum has anatomical connections to the cerebral cortex, through which it can affect language function (Ackermann, Mathiak, & Riecker, 2007; Hernandez-Muela, Mulas, & Mattos, 2005; Leiner, Leiner, & Dow, 1993). The neodentate cerebellar area projects via the thalamus to the frontal lobe, especially the prefrontal areas and Broca's area.

3. Individual differences in brain organisation of cognition in general, and language in particular, have barely been examined (Telzrow, 1985). It seems to make sense that individual linguistic experiences may affect the brain organisation of cognition in general and language in particular. We know well, for example, that developmental and acquired reading disturbances depend on the idiosyncrasies of the reading system (Karanth, 2003; Paulesu et al., 2001). We also know that over-practising a specific ability may result in changing (enlarging) its cortical representation (e.g., Elbert, Pantev, Wienbruch, Rockstroh, & Taub, 1995). We also know that literacy can change the clinical manifestations of aphasia (e.g., Goldblum, & Matute, 1986; Lecours et al., 1987). Linguistic experiences can be very different in an illiterate person and in a writer; in a monolingual and in a polyglot; in an Inuit and in a New Yorker. There are also very specific individual linguistic experiences, depending on the professional activity, the individual's personal history, etc. We simply do not know well how these differences in linguistic experiences can affect the brain organisation of language (see, for example, Burton, Noll, & Small, 2001).

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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. A major distinction in aphasia can be established between primary language disturbances (central aphasias), and secondary language disturbances resulting from "peripheral" impairments (secondary or "peripheral" aphasias). Sometimes language is not impaired, but the patient cannot use it appropriately because of executive control impairments (dysexecutive aphasia). Table 5 presents a proposed interpretation and classification of aphasia syndromes.

TABLE 5
Proposed interpretation and classification of aphasia syndromes

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 ideomotora 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

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Commentary

Aristotle's functional association psychology. The syntagmatic and the paradigmatic axes in the neurolinguistics of Roman Jakobson and Alexander Luria: An anatomical and functional quagmire

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Alfredo Ardila has written a paper on the reinterpretation and reclassification of aphasic syndromes. The model he uses is an extension of his early work with D. Frank Benson (Benson & Ardila, 1996) in an excellent textbook where they forged an amalgam of A. R. Luria's model of aphasia and the more "classical" model of aphasia, revived in the 1960s by Norman Geschwind and tagged as "The Boston School". My aim is to question the lingering tendency of Lurian theory to insist on an anatomical dissociation between the centuries old association psychology distinction between paradigmatic and syntagmatic linkages set forth by Aristotle. I will assess the relative tractability of assigning the syntagmatic and the paradigmatic to anterior and posterior left cortex in human beings, respectively, by considering recent findings in the neurosciences that demonstrate both the function and the anatomy of these two axes are far to heterogeneous for such a dichotomous division. There are more than two levels at which "frame" and "content" are needed, and the new findings from neuroscience demonstrate a much greater degree of integration of motor and sensory systems in the cerebral cortex. The anatomical division in the Lurian model for syntagmatic and paradigmatic associations will have to be revised.

ARISTOTLE'S LEGACY

Centuries ago, Aristotle laid out many of the initial ideas of association psychology. He claimed that associative links could be established between elements based on their similarities and on their contiguity of occurrence. Items that shared many features of similarity formed associative bondings among themselves and elements that frequently occurred in collocation also became tightly linked. In fact Aristotle

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thought that the strongest associative force was through contiguity. These two principal types of association were carried throughout centuries of natural philosophy, through British empiricism, and on to be two major tenets of behaviourist psychology and are now cornerstones of connectionist modelling (Buckingham, 2002).

ALEXANDER LURIA AND ROMAN JAKOBSON

One of the major theoretical contributions of the co-authored work of Luria and Jakobson was to place these two elements of associationism into a neuropsychological context of linguistic function and neuroanatomy (e.g., Jakobson, 1964; Luria, 1976). The notion of similarity among elements handed down from Aristotle became the cornerstone of the paradigmatic axis, where the primary linguistic process was one of selection from among those elements bonded by similarity. Aristotle's category of contiguity association was co-opted into Jakobson and Luria's scheme as a linear sequential ordering along a syntagmatic axis. The processes of selection and ordering were built into the model and became the major bipartite division in the structure and production of language. They went an important step further, localising as they did the syntagmatic axis in left anterior cortex and the paradigmatic axis in the left posterior cortex. These two Aristotelian associative categories then became paramount for categorising aphasic typologies as well as a metric for analysing error types as syntagmatic in frontal lobe motor aphasias and paradigmatic in temporo-parietal lobe sensory aphasias.

Alfredo Ardila spent many years in Russia studying with Luria, during the cold war years when many Latin American scholars also spent time with Luria or at least became familiar with Luria's teachings through the influence of their colleagues in Latin America. American neuropsychology did not originally benefit nearly as much as did the Latin Americans due to cold war politics. In any event, Luria's silhouette in Ardila's contributions to aphasia is well known. And it was a natural consequence of this that ultimately brought Ardila and Benson together to work out an interactive blending of Moscow and Boston.

In the target paper, this bipartite division still seems to play a role in Ardila's reinterpretations and reclassifications of aphasic symptomatology. For example, when describing Broca's and Wernicke's aphasia, Ardila writes (2010, p. 366):

> These two major aphasic syndromes have been related to the two basic linguistic operations: selecting (language as paradigm) and sequencing (language as syntagm). (…) … 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 overriding impression one forms from Ardila's paper is that Broca's aphasia is a syntagmatic disorder and Wernicke's aphasia is a paradigmatic disorder. By extension, frontal lobe systems are thought to be principally characterised by linear ordering of elements, while posterior temporo-parietal lobe systems are primarily responsible for the selection of elements. However, recent findings from linguistic aphasiology and from a wealth of imaging studies of magnetic resonance of the

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cerebral cortex in vivo have practically rendered this neurolinguistic division of labour into anterior and posterior cerebral systems unattainable.

BROCA'S AREA AS A PARADIGM OF CORTICAL HETEROGENEITY

There are practically insuperable problems with assigning any major role of syntagmatic processing in Broca's area in the left frontal lobe. There are cross-cutting complexities in both anatomical form and linguistic function. Ardila is certainly aware of findings such as Grodzinsky and Amunts (2006), Foundas, Eure, Luevano, and Weinberger (1998), Hagoort (2005), and Thompson-Schill (2005) that demonstrate once again that Korbian Brodmann was correct to distinguish the cellular structures of pars triangularis and pars opercularis, which he numbered, as we are all aware, 45 and 44 respectively. It is now pretty much indisputable that these two areas are distinct neuroanatomically and functionally. And, within reason, these left hemisphere cortical regions are distinguished in both form and function from their non-dominant hemisphere homologues in the human brain, and these areas have distinctions as well in animal frontal lobe cortices, evolutionary underpinnings for man and animal notwithstanding.

In a real sense "Broca's area" has now come to be called "Broca's complex", the region having undergone major "deconstruction". In the first place, neither perimeter coincides with the sulcal boundaries that separate them anatomically: 45 has a granular layer IV, while 44 actually has a dysgranular layer. Each area has a distinct postnatal ontogeny and 44 has a significant cell density asymmetry on the left vs the right; pars triangularis reveals no such asymmetry. Furthermore, both areas receive projections from distinct cortical zones. Within pars opercularis there are some neuropharmacological differences. The dorsal portion reveals a low receptor cell density for serotonin, while the ventral portion has a much greater density of serotonergic receptors. In addition, these studies have included areas 47 and the ventral portion of 6 in the left hemisphere language system of "Broca's complex". Summarising these sorts of recent findings, Hagoort (2006, p. 243) writes:

> In short, from a cytoarchitectonic and receptor-architectonic point of view, Broca's area, comprising BA 44 and BA 45, is a heterogeneous patch of cortex and not a uniform cortical entity. However, the degree of uniformity required for an inference of functional unity is not known.

Ardila should therefore be quite cautious in maintaining Jakobson and Luria's exhilarating and startling bridge from Aristotle to modern functional cortical neuroanatomy.

LINGUISTIC FUNCTIONS IN BROCA'S COMPLEX

To begin with, it is now clear that Broca's region serves perception as well as production, and even more importantly when the perceptions are linguistically communicative. Gandour et al. (2000) and Gandour et al. (2003) have shown a clear lateralisation effect, favouring the left Broca's complex for appreciating distinctive lexical tones for Chinese and Thai speakers, whose languages have tonemes as well as phonemes. Furthermore, frontal cortex comes to be specially tuned in speakers of tone languages, such that they are more amenable to pick up significantly shorter

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pitch contours because such fine-grained acoustic cues are communicatively significant in decoding lexical meanings. Broca's complex does not metabolise in these instances for English and German, since they are not tone languages. The point here is that Broca's region is a perceiving zone as well as a producing zone. Meyer and Jancke (2006) illustrate the complex involvement in frontal opercula of both hemispheres for speech and non-speech perception and production, which extend far beyond expressive syntagmatic chaining.

A closer look at Broca's aphasia and its typical complement of apraxia of speech (AOS) quite often reveals errors that involve substitution. Substitution is a presumed paradigmatic function of selection, where the targets and errors are quite often "similar" in their make-up. All students of AOS have noted substitutions as typical of that pathology. For example, Code (2005, p. 127) and McNeil, Doyle, and Wambaugh (2000) both note "kernel clinical features", one of which is "distorted sound substitutions" (excluding anticipations, perseverations, and exchanges) (Code, 2005, p. 127). These "distorted" sound productions are likely in many instances to be precisely those where the distortion is sufficient to cross phoneme boundaries, and thus give rise to the perception in the mind of the hearer that the substitution was perchance at the level of phoneme selection. Note that the derailment may have occurred at the level of articulation, where the target phoneme had, in reality, been correctly selected but with an apraxic articulation derailed enough to place that sound into another phoneme category, which in turn would be *perceived* by the hearer as belonging to another phoneme. We have noted that, in this instance, the surface utterance would rest at one level of description for the hearer, while the phoneme substitution would in actuality be constructed on the part of the hearer. This has been referred to as "phonemic false evaluation" (Buckingham & Yule, 1987) where paradigmatic errors in effect occur at two levels—one for the speaker and another for those who *listen* to the speech of adults with AOS. Furthermore, this line of reasoning has been extended to very likely subtle motor asynchronies in Wernicke's aphasia with lesions in left superior temporal lobe. Somewhat masked by the fluent output of Wernicke's speech, the subtle gestural offshoot might very well result in the listener's perception of a phoneme not intended by the speaker.

Before leaving this question I would point out that it would seem to be very confusing to describe sound distortions as "substitutive", although this is part and parcel of many descriptions of error types in AOS. It is highly doubtful that an adult with AOS would search through a category of correct sounds and distorted sounds, but select the wrong one, thus producing a substitution of a distorted sound. I have not seen this "lay" use of substitution discussed in the literature of AOS, but in the science of language production and its disorders, the word "substitution" has a more or less accepted technical sense of search and select—either correctly or incorrectly. This cannot be a very felicitous way to describe the phonetic aberrations seen in AOS. The processes of distorting a sound such that it is articulated in some other fashion would not seem best characterised as a substitution but rather more like an aberration of some already selected unit.

Other studies such as Blumstein (1973), who compared phonemic substitutions in Broca's, conduction, and Wernicke's aphasias, found that selection errors far outstripped other types of phoneme errors (deletions and linear transpositions) in all these symptom complexes, and in fact most of those substitutions differed in only one distinctive feature. That being the case, the selection derailments produced errors

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that were similar to the targets; the metric was feature sharing. Accordingly, there were paradigmatic errors secondary to lesions in Broca's area.

In addition, there are studies which indicate that Wernicke's aphasic speakers have more trouble with nouns, while Broca's aphasic speakers have more trouble with verbs. These investigations often use naming tests to demonstrate this kind of dissociation. Broca's aphasic speakers had more difficulty naming verbs, and that is not a syntagmatic breakdown as much as a paradigmatic problem. Therefore, again, we have paradigmatic derailments in patients with frontal lobe damage.

Finally, Michael Arbib (2006) has considered mirror neuron doctrine, and has speculated that area F5 in monkeys, where he finds mirror neurons, may very well be the evolutionary precursor of functions now assigned in general to area 44 in pars opercularis in the human brain. He focuses on the functions of laryngeal and supralaryngeal articulatory limb movements for speech as programmed in 44, and tacitly agrees with Foundas et al. (1998) and Barrett, Foundas, and Heilman (2005). Arbib and these authors argue that it is reasonable that left hemisphere pars triangularis in humans programs *language* functions that are parasitic on the higher-order heteromodal association cortex found there, which in turn serve the complex cross-modal associations of higher-order lexical and semantic linguistic functions. But although Arbib reserves 44 for articulatory and motor speech functions, he insists (2006, p. 1147) that voice and articulation must count on a "… smooth assembly at the level of phonemes …" and that this "… requires a different level of processing, and different inputs and outputs, than does assemblage of words into sentences". And it is this smooth assembly (that others refer to as fluent) of phonemes where we come into both selection and ordering of the abstract units, which are the phonemes. Thus, we cannot avoid further paradigmatic functions in Broca's complex as well as syntagmatic functions and even more so if our model is one of frame and content, since frame and content require paradigmatic selection of content and syntagmatic ordering into the frames.

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FLUENT APHASIA AS A SYNTAGMATIC BREAKDOWN

There are numerous publications that demonstrate sequential ordering malfunctions during the processes of selection and copying of phonemes in Wernicke's and conduction aphasias, whereby often high numbers of anticipations and perseverations of phonemes, groups of phonemes, or paragrammatic sentences are observed. Martin and Dell (2004) have proposed an intriguing model whereby they have charted throughout recovery from severely paraphasic speech a lowering in the number of left-to-right movements of phonemes, and a steady or slightly increasing number of right-to-left movements of phonemes. The result of the interaction of these two syntagmatic operations of phoneme sequential ordering is such that an "anticipation ratio" will increase as the patient moves towards normalcy. Not only does this make an excellent metric to chart recovery patterns, but it also accords with what happens in non-pathologically compromised children and adults. It has been reported that children's phonological ordering errors tend to be more of a perseverative nature at first, and as they further develop their phonologies the later errors have been observed to be much more anticipatory in nature. In addition, it has often been observed that phonemic level slips-of-the-tongue in older children and adults are far more anticipatory in nature. Presumably the "look-ahead" planning mechanism is more engaged in normal processing. This, then, would be why the

aphasic transformations throughout recovery to tend more and more to be of an anticipatory nature. This, then, falls in line with the "continuity thesis" that places disease and health along a continuum in the sense of William James, Hughlings-Jackson, and Sigmund Freud, and accords with connectionist models such as Gary Dell's, where the normal system that undergoes "lesioning"—changes in rates of decay or changes in weight linkages between levels or random changes in connection weights throughout the system ("noise") nicely "simulates" errors in both pathological compromised and non-pathologically compromised speakers. Wijnen and Kolk (2005) summarise a large number of different kinds of study, all of which support one form or another of the continuity thesis. However, the whole issue hinges on the production of large numbers of sequential ordering phoneme errors in the fluent aphasic speakers with lesions in areas where Jakobson and Luria located paradigmatic functions.

We mentioned the cortical heterogeneity in Broca's region that leads to the conflation of the syntagmatic and the paradigmatic in left frontal lobe syndromes; this conflation also is seen in left temporal lobe cortex—especially in and around the region of the left planum temporale. In our study of the "subtle" phonetic deficit in sensory aphasia (Buckingham & Yule, 1987), we alluded to the electro-cortical studies of Ojemann (1983) and the cytoarchitectual measurement investigations of Galaburda (1982, 1984). From his electro-physiological studies, Ojemann (1983, p. 198) wrote that "… the neural representation of motor sequence programs (or the precise timing of these) would be stored in the temporal lobe". Galaburda finds something very special about the temporal lobe region of the planum. He, and Geschwind and Levitsky (1968), had highlighted this temporal lobe cortical area as a major functional component for human language. Left asymmetries were demonstrated for this area, the crucial aspect being that the left planum was significantly larger on the left in humans. Apes have large right plana, but the left is large also, so there is little proportional size difference. Galaburda found large numbers of motor neuron cells (Betz cells) in the third and fourth cortical layers in the planum, directly subjacent to the cortical zone bordering on the supramarginal gyrus. Galaburda also reminds us that the planum works into the arcuate bundle and thus forms the Wernike's area – Broca's area link described in classical aphasiology. But these cells are motor in nature and thus when damaged could lead to the subtle motor deficits in otherwise fluent sensory aphasia. In fact, Galaburda (1982) was so taken aback with the existence of these motor neuron cells in the temporal lobe that he wrote, "In fact, architectonic similarities between anterior or posterior language areas, and the overlap in their connectional organization make it a somewhat surprising finding that lesions in either region produce such different aphasic syndromes" (p. 443).

Further evidence for a motoric component in temporal lobe is the finding of Amaducci, Sorbi, Albanese, and Gianotti (1981) of larger concentrations of the neurotransmitter choline acetyltransferase (ChAT) in the left temporal lobe than in the right; the asymmetries were greatest in the zone of the planum. Not only have depletions of this neurotransmitter been suggested as a contributing factor for recurrent perseveration in fluent aphasias (McNamara & Albert, 2004), but its concentrations are evidence for right-handness, since the larger left concentrations are not only in PT regions—ChAT has also been found to be significantly left hemisphere biased in all regions of the basal ganglia: globus pallidus, caudate, and putamen. Our point, of course, is that this finding would also lend credence to further conflation of the syntagmatic and the paradigmatic productive processing. It

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also and quite importantly serves as further evidence that handedness and language are tightly linked to left peri-Sylvian region.

CONTIGUITY: SERIAL ORDERING OR COLLOCATIONAL SEMANTICS

A serious inconsistency in the descriptor of "contiguity" has often been overlooked and little appreciated in discussions of the Jakobsonian/Lurian dichotomy. The term has often been used to describe serial ordering of elements into either pre-utterance frames or a more motoric level of serial ordering that would include, for example, co-articulatory processing and/or ordered movement dynamics at the speech gesture level. Strangely enough, the speech output of agrammatic patients almost always has typological (canonical) word order consistency for the language of the speaker. Agrammatic speakers may very well have difficulty with non-canonical word orders, but this is not a serial ordering disruption. It is rather something more like a referential, binding, indexing, use of traces, etc., for sentence comprehension. So, here is the "linearisation" interpretation of "contiguity".

"Contiguity", however, is sort of a linear collocational semantics; it is largely metonymic; associations between objects and their containers, e.g., popcorn and bag, beer and bottle. Frequent instrumental arguments of verbs, such as "knife" and "cut", or "pencil" for "write", will come to be linked associatively, as will typical locative arguments for persons, places, or things, e.g., doctor – hospital; stadium – game; bird – nest, etc. These lexical associations and others like them are stored in the lexicon as are other lexical associations based on the similarity metric. Both types of semantic association form part of the lexicon, although the "architecture" of each is different. That architectural difference may very well be described as "syntagmatic" vs "paradigmatic", but both involved processes of search and selection and not serial ordering. When viewed accordingly, there is evidence that aphasia type (e.g., Broca's vs Wernicke's) demonstrates no tendency to correlate with the associative semantics of the similar and the contiguous. Gainotti, Miceli, and Caltagrione (1981) pulled together all lexical level substitutions of both these types from 96 aphasic patients. All paraphasias were recorded, collected, and subsequently given patient by patient to independent judges who did not know the aphasia type of any of them. The judges ended up with a four-way classification of the range of errors. One group of patients had far more similarity errors; another group had far more contiguity errors. One group had only a few more similarity-based semantic paraphasias, while yet a fourth group had only a few more contiguity-based semantic paraphasias. These authors found *no* relationship between type of semantic paraphasia and type of aphasia. In addition they noted that, in general and across all subjects, the semantic substitutions tended to be more of the similarity type than the contiguity type. So, not only is there a relative conflation of contiguity and similarity across aphasia types but, quite obviously, the total set of errors involves paradigmatic selection errors and certainly not syntagmatic serial ordering errors. The clouding, thus, of the paradigmatic–syntagmatic functional brain dichotomy is compounded with the Gainotti et al. (1981) investigation.

CONCLUDING REMARKS

My report on the inconsistencies of the paradigmatic and the syntagmatic are part of my overall feeling concerning Ardila's paper. I do not think it is time to be

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reclassifying the aphasias, and even less so if the reclassification is to engender new kinds of aphasia batteries, assessments, and evaluations in the clinic. At this point in aphasiological research there are too many open questions being generated by recent research—research that is purely functional in nature as well as research that treats anatomy and physiology. Functional magnetic resonance imaging as well as volumetric magnetic resonance imaging is deepening our understanding of the complexities of cerebral lobes and how they function. We are being flooded with new information in the hardware and in the software of aphasiological phenomena, and at least at this point in time we should resist any urge to reclassify; it is simply too early.

The thesis in this paper has been to demonstrate that many of these new findings call into question the half-century-old cortical dissociation between the paradigmatic and the syntagmatic, notions that stretch back to Aristotle's early theories of association psychology. Similarity is the major metric of selection from a paradigm, while contiguity deals with the syntagmatic. Historically, this is essentially Aristotelian theory taken by the structuralist Roman Jakobson and scaffolded across the cerebral cortex. It was quite ingenious of him, and he worked his notion in with the neuropsychological functionalism of Luria. In a sense, it was a major attempt to bridge the mind (functional associationism) with the lobes of the human brain. This anterior–posterior cortical dissociation of the ancient association categories of Aristotle was most certainly heralded as a powerful discovery by Jakobson, the structuralist. It is a shame that it does not work.

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Commentary

Ardila's attempt to alter aphasiology

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Aphasiology has undergone considerable change since Broca and Wernicke, and present day efforts, driven by technology, represent another stage of reinterpretation of theory and revamping classification. As Henry Head (1926, p. 84) remarked about the process 82 years ago: "Round and round like a stage army moves the procession: the clinical appearances are identical, but each fresh group of observers views them with new eyes and with different preconceptions."

Ardila (2010) has created a review and a reformulation, based on the core concept of the Broca–Wernicke aphasia, grammatical/lexicosemantic deficit dichotomy. It is often argued that the classification of aphasias is a sterile exercise, but given the clinical and theoretical importance of the aphasias, this opinion is not justifiable. There is much to be found in this extensively researched and well-argued article by Ardila, and one can agree with most of what he says. The subcortical and transcortical aphasias are particularly well reviewed. He also introduces a great deal of recent neuroimaging and functional activation work, the study of mirror neurons, the dichotomy of verb–noun processing and even the genetics of language capacity, all important discoveries relevant to aphasiology. I shall try to examine and comment on some of the problematic, but interesting, aspects of his review and thesis.

A systematic classification based on the combination of a taxonomic and comparative modification of the Wernicke-Lichtheim-Boston principles appeared in 1976 (Kertesz 1976) and Ardila quotes the book *Aphasia and Associated Disorders* (Kertesz, 1979), which is based on that approach. Ardila's review and simplified table of classification is similar to the comprehensive comparative table of classification that appeared in that article and the book. *The Western Aphasia Battery* (Kertesz & Poole, 1974; Kertesz, 1982, 2006) allows aphasia classification based on scores—in fact the WAB was explicitly designed to do that. Ardila has also approached the classification issue taxonomically in some instances, as evidenced by his table 4 of repetition scores from his article (Ardila & Rosselli, 1992).

Anatomical and phenomenological classifications are often mixed, disregarding the serious conceptual, heuristic, and methodological errors produced. Anterior/grammatical vs posterior/semantic deficits are oversimplifications that are countered

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by a considerable number of factual findings. This conceptual confusion is unfortunately common in the field and leads to sloppy categories that are not only intellectually faulty but can be misleading.

The last sentence of the discussion on the paradigmatic disturbance of Roman Jacobson appears to be confusing, it is inaccurate to say "generic (paradigmatic)" (p. 367). Paradigmatic should refer to the selection of the specific lexical item or example from a category, not the generic. Perhaps Ardila meant to say that and a clarification could resolve the issue.

Interpretation of "deep" and surface aphasia is often questionable. The deep and surface aphasia dichotomy is counterintuitive to most non-linguists, to the extent that it has not been adopted except for written language, where it has been used extensively for a while but its use is also fading among clinicians dealing with classification of written language disturbance. Beyond strictly linguistic interpretation of communication difficulty is the intriguing territory of pragmatics, which is only indirectly alluded to in the discussion of frontal deficits of discourse.

The thesis considers a phonemic discrimination deficit fundamental to Wernicke's aphasia. This remains a debatable issue. There is variable phonemic discrimination deficit in Wernicke's aphasia, as shown by Blumstein, Baker, and Goodglass (1977), and although this may be more universal in the cases of word deafness or what Luria (1966, p. 103) called "acoustic agnostic aphasia". Should that also be reconsidered as a "peripheral" disturbance at the sensory end in Ardila's reinterpretation? The separation of cognitive and metacognitive language abilities to parse language production (and perception) has been attempted before, but it seems to me a dubious exercise fraught with many conceptual traps.

That semantics is processed in widely distributed cortical network is not a surprising finding, but it is worth reiterating in view of recent efforts to shift the locus of its processing from the multimodality dominant parietal-posterotemporal region to the anterior temporal regions on the dominant, left side as evidenced from discussions of semantic dementia. Interestingly, Ardila leaves out the accumulated work on primary progressive aphasia, both the nonfluent and the semantic variety, although there has been much debate on how they should be classified or what can be learned from their linguistic and anatomical differences. A related topic, whether the fluency–nonfluency dichotomy is valid in stroke aphasia, is not discussed, although it remains a major component of many classification systems.

The complex nosology of conduction aphasia is well explored, although one does not have to agree with the conclusions. Ardila's classification of core language disturbance excludes conduction aphasia, although it is considered a central language disturbance by many. Obviously there is a strong linguistic component, as Luria (1966) recognised when he classified the entity to afferent motor and acoustic amnestic varieties. We also found two distinct clusters in conduction aphasia using numerical taxonomy (Kertesz & Phipps, 1977). The reclassification of conduction aphasia as a peripheral disturbance is an innovative oversimplification that is bound to create a controversy. Although it is based on the rationale that repetition is only a "peripheral mechanism", this flies in the face of much work written on the subject. Goldstein (1948), for instance, considered conduction aphasia a central aphasia incorporating a prolific paraphasic output (not only paraphasic repetition, one should add), but also a range of comprehension deficits. Furthermore, impaired short-term verbal or phonological memory was also considered to be an important component. Much depends on the operationalisation

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of the definition of conduction aphasia, and the taxonomic approach, based on actual test scores and boundaries, needs to be considered here.

What is the difference between supplementary motor aphasia and dysexecutive or transcortical motor aphasia except in the name? Dysexecutive syndrome, apart from being an unpleasant neologism, is being applied to aphasia classification, not surprisingly in view of the popularity of the concept of executive function. Ardila marshals an impressive array of supportive literature in favour of the combination of frontal linguistic and motor functions. Particularly extensive is the review of the role of Broca's area. Another aspect of this issue is illuminated in the discussion of the frontal processing of action verbs and grammar and their more than coincident relationship. Furthermore the description and interpretation by Ardila and Lopez (1988) of a case of paroxysmal Broca's aphasia, who reported a blank mind or loss of inner speech when she was not talking during a seizure is illuminating the issue of the locus of inner speech.. Also relevant to this issue is the KE family with articulatory problems and agrammatic, expressive language disorder with the FoxP2 mutation (Marcus & Fisher, 2003)

Transcortical sensory aphasia is perhaps less controversial except for the odd localisation to frontal regions, where one would expect the transcortical motor syndrome to be encountered (Berthier, 1999) The presence of some motor symptoms suggests that these cases represent a mixed transcortical pattern, or an incomplete, partially recovered, or developed isolation of the speech area syndrome. Ardila covers the issue of "false" or atypical localisation in some detail. In addition to the recovery and diaschisis, which is the major factor, there are methodological and taxonomic issues, which are referred to in passing. Often the discrepancies are related to differing definitions and differences in interpretation can be related to the use of the same terminology for variable clinical phenomena.

Finally one is tempted to ask the question: Is any reinterpretation or reclassification in the well-established territory of aphasiology misleading or fanciful or is it a productive, thought-provoking effort leading to further knowledge? One hopes the second premise will turn out to be true. Science is promoted as much by bold deviations from existing truths, while generating efforts to prove them wrong, than the more mundane accumulation of evidence in favour of what is already stated. Ardila managed to do both in his review and reinterpretation.

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APHASIOLOGY, 2010, 24 (3), 408–412

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Commentary

Classification of aphasia: Are there benefits for practice?

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The value of classifying people with aphasia has been a topic of debate for at least 20 years. For example, in 1988 Caramazza and McCloskey wrote (p. 519):

> … the classification of patients into categories such as Wernicke's aphasia, conduction aphasia, expressive aphasia, or more modern categories such as agrammatic aphasia, deep dyslexia, phonological agraphia and so forth, is not only useless but positively harmful for research into the nature of cognitive disturbances and the structure of normal cognitive processing.

Twenty years on, Ardila (2010) is proposing a new system for classifying aphasic syndromes. This response will consider the benefits of his proposal, particularly with respect to clinical practice, and whether the objections of commentators like Caramazza and McCloskey still stand.

Ardila's system draws a distinction between primary aphasias, where language skills are directly affected, and secondary aphasias, where symptoms arise from impairments outside the language system. An example of the latter is transcortical motor aphasia, which is deemed to be an executive problem affecting language use.

The two primary forms of aphasia in Ardila's system are not new at all, but are the highly familiar and long-established Broca's and Wernicke's types. Ardila argues that in these forms of aphasia we see impairments to the fundamental components of language, with Broca's aphasia affecting the grammatical system and Wernicke's aphasia the lexical/semantic system. The neuro-anatomy of aphasia suggests that these systems call upon distinct brain circuits. Ardila adds the interesting suggestion that the grammatical system, impaired in Broca's aphasia, may have evolved from a single genetic mutation crucial for the evolution of language.

Although much of Ardila's argument is familiar, the fact that aphasias fall so pervasively into these broad descriptive categories *is* remarkable. For example, these categories appear across all languages including sign languages (Poizner, Klima, & Belugi, 1987), and show a robust correlation with frontal and posterior lesion sites (Kreisler et al., 2000). The lexical/grammatical dissociation between categories is also

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striking. This is not to say that it is absolute. For example, Edwards (2005) demonstrates that people with fluent aphasia may make a variety of grammatical errors. Nevertheless, it remains true that syntax is typically a *relative* strength in this form of aphasia (see arguments in Marshall, 2006). So nonword and other lexical errors are often appropriately inflected and embedded into phrase structures (e.g., Butterworth, 1985) and people with Wernicke's aphasia reveal surprisingly preserved gap-filling operations on judgement tasks (Swinney & Zurif, 1995; Zurif, Swinney, Prather, Solomon, & Bushell, 1993). In a similar vein, people with Broca's aphasia, who have little or no syntax, may be able to comprehend and produce concrete nouns with relative ease (e.g., see Cairns, Marshall, Cairns, & Dipper, 2007).

Despite these broad patterns, problems remain for the Broca's and Wernicke's classifications, mainly because of the degree of variability observed between category members. To illustrate this point, Table 1 provides assessment data from four individuals with chronic aphasia arising from posterior lesions. In all cases, their speech invited a Wernicke's classification in that it comprised fluent, unintelligible jargon that was composed largely from nonwords. Consistent with this classification, both LT and PK revealed severe deficits in auditory comprehension and lexical decision. Yet this was less evident for CM and particularly RMM, whose auditory input skills were surprisingly intact. CM's repetition, while not preserved, was also a relative strength, which set him apart from the rest of the group. Finally, written comprehension offered a further point of contrast, with PK outperforming his colleagues in this domain.

It is difficult for any classification system to accommodate such variability. Ardila allows for three subtypes of Wernicke's aphasia, yet as all predict comprehension impairments the problems posed by RMM and CM remain. CM may be deemed to have transcortical sensory aphasia, given his relatively preserved repetition. However, this syndrome is typically associated with semantic, rather than neologistic jargon, and anyway is not included in Ardila's final classification (see his table 5). Of course it could be that these individuals are atypical, so cannot easily be assigned to a category, in which case I have to say that it always seems easier to recruit atypical

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TABLE 1
Comprehension, repetition, and naming data from four people with fluent aphasia

	RMM[2]	*PK*[3]	*CM*[2]	*LT*[4]
Lesion	*Left temporo-parietal*	*Left temporo-parietal*	*Left temporo-parietal*	*Left parietal*
Spoken word to picture matching[1]	92.5%	55%	92.5%	75%
Auditory lexical decision[1]	92.5%	Unable	79.3%	Unable
Written word to picture matching[1]	62.5%	95%	80%	77.5%
Repetition[1]	Unable	20%	55%	5%
Naming[1]	Unable	30%	35%	10%

[1]Tests from Kay, Lesser, and Coltheart (1992). [2]See Marshall, Robson, Pring, and Chiat (1998). [3]See Maneta, Marshall, and Lindsay (2001). [4]See Robson, Pring, Marshall, and Chiat (2003).

than typical participants. Alternatively, the differences between them could be dismissed as trivial, and peripheral to the criteria that grant category membership. However, in this case we are still left with the problem of defining the core and essential criteria.

Why does variability matter? One problem is theoretical, in that it becomes difficult to theorise about the nature of the processing impairment, e.g., in Wernicke's aphasia, when such differing symptoms arise. As Caramazza and McCloskey (1988) argue, this in turn makes it difficult to draw any valid inferences about the nature of the normal cognitive system from aphasic group data.

Here, however, I wish to consider another problem—the problem for therapy. A good aphasic classification system should inform clinical practice; indeed clinicians might argue that this is its primary function. For example, such a system should make it possible to develop therapy programmes for each of the aphasic sub-categories, so providing clinicians with "off the shelf" treatment resources and the opportunity to carry out group evaluations of therapy. In line with this, a number of programmes were stimulated by the Boston classification system, such as visual action therapy for people with global aphasia (Helm-Estabrooks, Fitzpatrick, & Barresi, 1982) and the Helm Elicited Language Programme for Syntax Stimulation (HELPSS) for people with Broca's aphasia (Helm-Estabrooks & Ramsberger, 1986).

However, if there is too much variability between members of the same aphasic sub-group we cannot be confident that they will respond positively to the same therapy. Returning to the four people in Table 1, it is not at all clear that their therapy needs are the same. So, PK and LT would probably benefit from treatment focusing on spoken input, while this seems less the case for RMM and CM. Indeed, RMM and PK were involved in positive treatment studies that evaluated completely different interventions, with RMM's therapy targeting writing (Robson, Pring, Marshall, Morrison, & Chiat, 1998) and PK's targeting auditory comprehension (Maneta, Marshall, & Lindsay, 2001).

The observed variability between aphasic people has led to the proposal that therapy should be explored, at least initially, via single-case studies (e.g., Franklin, 1997). Such studies have a number of advantages. The therapy programme is developed in the light of the individual's assessment profile, so has a fair chance of success. It can also be described in sufficient detail to be replicable. The experimental design should enable the researcher to isolate therapy effects from non-specific sources of change, such as spontaneous recovery, and speculate about how and why the therapy has worked.

There is, of course, a limit to what single-case studies can achieve. Most obviously, they do not allow us to generalise the results beyond the person tested. There is also the problem of clinical feasibility, in that therapists working in routine clinical settings cannot be expected to develop novel therapies for each individual client. Rather, they need to be able to turn to recognised therapeutic procedures that are supported by research evidence. In order to get to this point, we need to carry out replications. Here a classification system might help to identify likely candidates for replication, with the assumption that if therapy has worked for one member of the group, it might work for others. However, as we have seen, this may well be an over-assumption. An alternative approach, therefore, uses case series and small group designs to determine which clients are suitable for the therapy and set exclusion criteria. Once this has been achieved large group efficacy and effectiveness studies can be carried out (see arguments in Pring, 2004).

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So far, I have argued that classification systems have very limited prescriptive power and that selecting a treatment on the basis of a syndrome diagnosis is likely to overlook crucial individual differences. Rather, finer-grained decisions need to be made, for instance about the person's priorities for therapy and the processing impairments that contribute to their problems. In an ideal world, this level of assessment should enable the clinician to turn to evidence-based treatment approaches that address their client's particular symptoms.

Should we therefore discard syndrome labels? For me they can be retained providing we are clear about the limit of their contribution. They do, for example, provide a broad descriptive characterisation that is widely understood across the clinical community. So, a clinician who is referred a client with Broca's aphasia can reasonably expect speech to be nonfluent with omissions of verbs and grammatical markers. The second contribution relates to the first. While not prescribing therapy, the syndrome label makes some treatment targets more likely than others. So sentence-level skills are likely to be a priority for clients with Broca's aphasia and lexical/comprehension skills for clients with Wernicke's aphasia. This, however, is as far as it goes. We cannot be sure that the personal priorities of the client will fall in line with Ardila's syndrome classification and even if they do, the specific nature and level of therapy can only be determined by a more thorough investigation. We end up by saying that aphasic clients are all individual. But then, this was probably never in doubt.

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Reply

Aphasia revisited: A reply to Buckingham, Kertesz, and Marshall

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First of all, my sincere gratitude to Hugh W. Buckingham, Andrew Kertesz, and Jane Marshall for their most important and interesting comments to my paper "A proposed reinterpretation and reclassification of aphasic syndromes" (Ardila, 2010). Thank you to them for the time and effort that they devoted to review and comment on my paper. Indeed, in my paper two different questions were approached (interpretation of aphasia and aphasia classification) that are obviously intimately interrelated. Buckingham's comments emphasised interpretation issues, whereas Marshall's emphasised aphasia classification; Kertesz referred to both.

Without doubt, aphasia interpretation has changed significantly during the last four decades, mainly as a consequence of the introduction of new neuroradiological techniques for the study of brain. These techniques were developed in two steps corresponding to two different historical moments: initially during the 1970s and 1980s from the anatomical point of view (e.g., Ardila & Ostrosky, 1989; Damasio & Damasio, 1989; Kertesz, 1983); and later, during the 1990s and 2000s, from the functional perspective (e.g., Cabeza & Kingstone, 2006; Kertesz, 1994).

The CT scan was introduced in the aphasia area during the 1970s (e.g., Mazzocchi & Vignolo, 1979). Its introduction significantly impacted the aphasia interpretation and classification in different ways. In particular (1) a significant interest in distinguishing aphasia subtypes was observed (e.g., Coslett, Roeltgen, Gonzalez-Rothi & Heilman, 1987); as a matter of fact, CT scan allowed a finer analysis of those brain areas involved in different forms of aphasia—the significant interest in pinpointing clinical/anatomical correlations is understandable. (2) A clear interest in analysing the potential role of non-classical language areas in linguistic functions was evident; a special emphasis in subcortical aphasia was noticed (e.g., Alexander, Naeser, & Palumbo, 1987). This interest in approaching subcortical aphasia was obviously related with the general interest in understanding the potential contribution of subcortical structures to cognition, and the broader question of subcortical cognitive disorders (subcortical dementias) (Albert, Feldman, & Willis, 1974; McHugh & Folstein, 1975).

With the introduction of the functional techniques, particularly PET and fMRI, interpretation of brain organisation of language somehow switched from the search

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for those anatomical areas involved in language processing and production to the analysis of the brain "networks" (or "circuits") supporting different linguistic abilities. It became evident that different language tasks (e.g., naming, language understanding, etc.) usually resulted in the simultaneous activation of several brain areas (e.g., Hasson, Nusbaum, & Small, 2007; Newhart, Ken, Kleinman, Heidler-Gary, & Hillis, 2007), and hence it could be more exact to refer to dynamic networks (or circuits) involved in language, than referring to anatomical localisations.

However, attempts to reclassify aphasia during these last four decades have been limited (e.g., Benson & Ardila, 1996; Kertesz, 1979, 2006; Lecours, Lhermitte, & Bryans, 1983), and in general the same fundamental classifications of aphasia (e.g., the Boston Group's, Luria's) continued in use by most researchers and clinicians.

The question of aphasia classification is a complex one. It has been argued that aphasia classification is useless (e.g., Caramazza & McCloskey, 1988), and indeed all aphasic patients are somehow different. However, this—partially true—argument could be used for any clinical syndrome: agnosia classification is useless and all agnosic patients are different; schizophrenia classification is useless and all schizophrenic patients are different, etc., etc. Furthermore, there are always atypical cases, difficult to fit in any existing classification (see Marshall's commentary).

Classifications, nonetheless, are required for systematising purposes and because classifications suggest different underlying mechanisms. In the history of aphasia over 20 different classifications have been proposed, although most of them are at least partially coincidental. Indeed, there are very few really original aphasia classifications (e.g., Head, 1926; Luria, 1970).

Up-to-date aphasia classifications have been proposed by individual researchers (or "schools", or "teams") (including the classification presented in my target paper); however, they have not been the result of panel discussions and analyses. This limitation also holds true for most cognitive brain syndromes (apraxia, agnosia, dysexecutive syndrome, etc); dementia may be the only notable exception.

It can be conjectured that, towards the future, this "individual" approach will be overcome and cognitive brain syndromes will be classified by panels of experts. There are some illustrative examples in clinical areas of this "panel of experts" approach; two excellent examples may be the classification of "mental disorders" (Diagnostic and Statistical Manuals of Mental Disorders, DSMs; APA, 1952, 1970, 1987, 1994), and the classification of epilepsy (Commission on Classification and Terminology of the International League Against Epilepsy, 1981, 1985, 1989). Mental disorders and epilepsy are no longer classified by individual researchers (although the departing point for epilepsy classification was Gastaut's classification; Gastaut, 1969—and the departing point for different syndromes presented in the DSMs were also classifications presented by individuals), but by authoritative panels of experts presenting proposals, inviting colleagues to use the proposed classification for some time, and periodically revisiting the proposed classification. Interestingly, the DSM-IV (APA, 1994) includes a group of cognitive disorders under the title "Delirium, dementia, and amnesic and other cognitive disorders", but indeed only dementia is reviewed in any detail. There is a subgroup of "other cognitive disorders" but no classification is proposed.

It can be anticipated that in the future this approach has to be used with aphasia and other cognitive brain syndromes. It is time to create international panels of experts to classify not only aphasia but also other neuropsychological syndromes. In

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this regard, the classification of aphasia presented in my paper is just a proposal, nothing else.

I will refer next to the specific comments presented by Hugh W. Buckingham, Andrew Kertesz, and Jane Marshall.

BUCKINGHAM

Buckingham (2010) begins by presenting a very interesting history of association psychology, pointing out that Aristotle first proposed that associative links could be established between elements based on their similarities and on their contiguity of occurrence. Further, he explains that Jakobson and Luria were the first to place these two elements in a neuropsychological context, relating similarity with the paradigmatic axis and contiguity with the sequential ordering of elements in a syntagmatic axis. I want to add to that idea that division of disorders into the two major aphasia syndromes is a very strong, well-established, and long-standing idea that was proposed long before Jakobson and Luria, and found since the initial descriptions of the language disturbances associated with brain pathology (see Tesak & Code, 2008).

Buckingham later states that recent findings from linguistic aphasiology and imaging studies of magnetic resonance of the cerebral cortex in vivo have practically rendered this neurolinguistic division of labour into anterior and posterior cerebral systems unattainable. I certainly disagree with Buckingham's conclusion and I am convinced that modern neuroimaging research has not discharged, but confirmed, that frontal lobes are involved in executing (sequencing) and posterior cortex is involved in processing information (for a review of over 2000 fMRI studies see: http://www.fmriconsulting.com/brodmann/)).

Further, Buckingham discusses in some detail the role of Broca's area based in the most recent literature. His analysis indeed supports the discussion presented in my paper, and the general conclusion drawn (p. 371) that,

> … regardless that expressive language disturbances have been associated for over a century with damage in the left inferior frontal gyrus (later known as "Broca's area") currently there is incomplete agreement about its limits and its specific functions in language. Different proposals have been presented to explain language disturbances in so-called Broca's aphasia, including: binding the elements of the language (Hagoort, 2005), selecting information among competing sources (Thompson-Schill, 2005), generating/extracting action meanings (Fadiga et al., 2006); sequencing motor/expressive elements (Ardila & Bernal, 2007); cognitive control mechanism for the syntactic processing of sentences (Novick et al., 2005); construction of higher parts of the syntactic tree in speech production (Grodzinsky, 2000, 2006); and verbal working memory (Haverkort, 2005).

Buckingham moves to review in some detail the potential participation of Broca's area in perception (for an extensive review of functional neuroimaging studies about Broca's area, see http://www.fmriconsulting.com/brodmann/). In particular, he analyses Gandour and colleagues' studies (Gandour et al., 2000, 2003) on the lateralisation effect, favouring the left Broca's complex for appreciating distinctive lexical tones for Chinese and Thai speakers, whose languages have tonemes as well as phonemes. Buckingham states (p. 397) that "frontal cortex comes to be specially

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tuned in speakers of tone languages, such that they are more amenable to pick up significantly shorter pitch contours because such fine-grained acoustic cues are communicatively significant in decoding lexical meanings. Broca's complex does not metabolise in these instances for English and German, since they are not tone languages. The point here is that Broca's region is a perceiving zone as well as a producing zone."

I personally do not find these results surprising at all. Brodmann's area 44 in the right hemisphere is activated in the perception of prosodic information (intonation) in speech (Hesling, Clément, Bordessoules & Allard, 2005; Wildgruber et al., 2005). Wildgruber et al. (2005) point out that comprehension of affective prosody is associated with a specific pattern of right hemisphere activation, including the posterior superior temporal sulcus (Brodmann's area 22), dorsolateral (Brodmann's area 44/45), and orbitobasal (Brodmann's area 47) frontal areas. Activation within left-sided speech areas, in contrast, was observed during the phonetic task. Obviously, for Chinese and Thai speakers recruited in Gandour et al.'s study, tones are language, not prosody. Furthermore, it has to be emphasised that not only Broca's area is activated, but an extended neural network including a diversity of brain areas. As another example, Klein, Zatorre, Milner, and Zhao (2001), using PET, compared tone perception in 12 native Mandarin speakers, who use tonal patterns to distinguish lexical meaning, with that of 12 native speakers of a non-tone language, English. Both groups showed common activation areas, but only Mandarin speakers presented additional activation in frontal, parietal, and parieto-occipital regions of the left hemisphere; not just the Broca's area, it was much more. Furthermore, tones are language in Mandarin but not in English, and left hemisphere language-related areas are activated in Mandarin speakers with tones; not so in English speakers. But stating that "The point here is that Broca's region is a perceiving zone as well as a producing zone" as Buckingham does (p. 398), is a very big leap that I personally do not risk to take.

Further in his commentary, Buckingham reviewed in detail the substitution errors observed in apraxia of speech. Nonetheless, Buckingham himself proposes a potential explanation for the apparently paradigmatic errors observed in Broca's aphasia (p. 398): "derailment may have occurred at the level of articulation, where the target phoneme had, in reality, been correctly selected but with an apraxic articulation derailed enough to place that sound into another phoneme category, which in turn would be perceived by the hearer as belonging to another phoneme."

Buckingham refers to the studies indicating that Wernicke's aphasic speakers have more trouble with nouns, while Broca's aphasic speakers have more trouble with verbs. Broca's aphasic speakers have more difficulty naming verbs and, Buckingham adds, that is not a syntagmatic breakdown as much as a paradigmatic problem. This observation has only an apparent validity, because verbs basically refer to doing, sequencing, etc. This point is analysed in some detail in my target paper and also in Ardila (2009).

Buckingham describes the model proposed by Martin and Dell (2004). According to this model, anticipations and perseverations arise from malfunctions of a mechanism that maintains serial order in speech production. It includes three components: (1) a means to turn off past utterances, (2) a means to activate the present utterance, (3) a means to prime the future utterance. Further, the probabilities of such movement errors are determined by the same variables that influence the production of ordinary word and sound substitutions, such as

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frequency and feature overlap. Buckingham claims (p. 400) that according to this model there is "the production of large numbers of sequential ordering phoneme errors in the fluent aphasic speakers with lesions in areas where Jakobson and Luria located paradigmatic functions". I will not answer this critique in detail, because first I am not familiar with that particular model, and second it has to be kept in mind that a model is "a generalised, hypothetical description, often based on an analogy, used in analysing or explaining something" (Webster's New World Dictionary, 2000). That is, a model is not an explanation, but rather a kind of analogy. Consequently, models are not "true" or "false"; they are "strong" or "weak"; "comprehensive" or "simplified", etc.

In the subsequent paragraph Buckingham refers to the potential evidence for a motoric component in temporal lobe. In the extensive review of over 2000 functional imaging studies referred to above (http://www.fmriconsulting.com/brodmann/), no motor functions were observed in Brodmann's areas 38 (temporal pole), 22 (superior temporal gyrus), 21 (middle temporal gyrus), 20 (Inferior temporal gyrus), 41–42 (primary auditory cortex – Heschl's gyrus), and 37 (posterior inferior temporal gyrus, middle temporal gyrus, and fusiform gyrus). Brodmann's area 37 was activated with "motion after-effect" (Taylor et al., 2000), but of course, an after-effect is a perceptual phenomenon, not a motor one. Indeed, the literature used by Buckingham to support his point of view is mostly from the 1980s, and certainly contemporary neuroimaging studies do not support such a point of view.

Buckingham analyses the meaning of the term "contiguity" emphasising certain inconsistency. He refers to two different uses of this term: serial ordering of elements, and linear collocational semantics (lexical associations); lexical associations are stored in the lexicon and are based on the similarity metric. Contiguity in the Jakobsonian/ Lurian dichotomy clearly refers to the first meaning. He further analyses with some detail Gainotti, Miceli, and Caltagirone's (1981) study distinguishing semantic substitution by contiguity or by similarity. In Gainotti et al.'s study semantic paraphasias produced by 96 Broca's and Wernicke's aphasic speakers were analysed. Three judges classified all responses into one of the following four categories: those having (1) a strong similarity, (2) a strong contiguity, (3) a mild similarity, and (4) a mild contiguity relation to the correct word. No relationship was found between type of semantic paraphasias and clinical forms of aphasia. However, as Buckingham himself mentions, both types of semantic association form part of the lexicon, although the "architecture" of each is different and both involve processes of search and selection and not serial ordering.

In the concluding remarks to his commentary Buckingham expressed disagreement in reclassifying aphasias, arguing that it is too early. At the beginning of this paper I presented my rationale for proposing a reclassification of aphasia, and suggested a potential procedure to move in that direction. I am convinced that aphasia classification proposals (most of them presented decades ago) are outdated, and even the terminology used is sometimes obscure and confusing (e.g., "transcortical" seems to be an obscure and incomprehensible term).

In his final paragraph Buckingham summarises his major concern with my target paper: the distinction between paradigmatic and the syntagmatic can be traced to Aristotle, and he concludes (p. 402) that, "this anterior–posterior cortical dissociation of the ancient association categories of Aristotle was most certainly heralded as a powerful discovery by the structuralist Jakobson. It is a shame that it does not work." I would argue that the distinction between two major (and even opposite)

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types of aphasia was not taken from Aristotle (384–322 BC) but from clinical observations, and it is found in the very first observations about language disturbances associated with brain pathology; Hippocrates (ca. 459–424 BC) referred to *afonos* and *anaudos* as two different subtypes of language impairments (Benton & Joynt, 1960), corresponding to the two major aphasic syndromes: Broca-type aphasia, and Wernicke-type aphasia. It is my impression that this distinction has worked throughout the history and it continues working today, even though certainly some precision may be required.

ANDREW KERTESZ

Kertesz (2010) expressed a fundamental agreement with my paper when stating (p. 404) that "there is much to be found in this extensively researched and well-argued article by Ardila and one can agree with most of what he says". Indeed, my interpretation of aphasia is quite close to Kertesz's aphasia interpretation. It is noteworthy that Kertesz has been significantly involved in the question of aphasia classification for a very long time (Kertesz, 1976, 1979, 1982, 2006; Kertesz & Phipps, 1977).

Kertesz also presents some specific concerns with my paper. He points out that the last sentence of the discussion on the paradigmatic disturbance of Jacobson appears to be confusing, because it is inaccurate to say "generic (paradigmatic)". Kertesz is correct and indeed the whole sentence should be deleted.

I fully agree with Kertesz's comment that the interpretation of "deep" and "surface" aphasia is often questionable. It is mentioned in my paper just in order to have a relatively complete review of aphasia interpretations proposed during the last decades, but certainly I also find that interpretation quite questionable.

The question of the phonemic discrimination defects in Wernicke aphasia is not simple to answer: Should phonemic discrimination defects also be considered as a "peripheral" disturbance at the sensory level? In other words, should word deafness be regarded as a "peripheral" aphasia? I am not sure about the answer, but I think it would make sense to answer "yes".

Kertesz is completely right when pointing out that in my paper the significant amount of work on primary progressive aphasia is not included. Obviously, classification of progressive aphasia represents an important step to understanding the potential variations of aphasic syndromes. Doubtless, the analysis of progressive aphasia represents a significant contribution for the understanding of both dementia and aphasia. I am aware of this important limitation of my paper.

No question, the most potentially controversial question in my aphasia classification proposal refers to conduction aphasia. How, indeed, to explain conduction aphasia? I am afraid there is not a final answer and controversies remain about the nature of the short-memory defects, the phonological output codes, the motor abilities in conduction aphasia, etc. (e.g., Baldo, Klostermann, & Dronkers, 2008; Jacquemot, Dupoux, & Bachoud-Lévi, 2007; Robin, Jacks, Hageman, Clark, & Woodworth, 2008). Kertesz refers to the two distinct clusters in conduction aphasia found when using numerical taxonomy (Kertesz & Phipps, 1977). Nonetheless, in my paper I am only referring to the parietal (or insular) conduction aphasia type. The questions remain: Does conduction aphasia represent a linguistic or a motor (praxic) defect? Or both? Is conduction aphasia just the consequence of damage in the arcuate fasciculus (e.g., Yamada et al., 2007) that obviously is simply

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a connecting pathway between two brain areas? Is repetition a fundamental language ability? Before that question, what should be understood as "fundamental language ability"?

Indeed, the role of repetition in language and the specific function of the arcuate fasciculus remain elusive (Catani & Mesulam, 2008). Recently, using the tractography procedure, it has been reported that the left superior longitudinal fasciculus projection to Broca's areas is absent in most participants and minimal in others; however, its rostral endpoint was reported in the precentral gyrus (not in the Broca's area!) in 100% of cases (Bernal & Altman, 2008), questioning the participation of the arcuate fasciculus in primary language processes.

Certainly, supplementary motor aphasia and dysexecutive or transcortical (extra-Sylvian) motor aphasia share significant similarities, but differences may also be found. I have attempted to illustrate those differences, although it can be argued that in both cases, basic language characteristics are rather similar: good repetition, good understanding, and decreased spontaneous language. Finally Kertesz raises the question (p. 406): "Is any reinterpretation or reclassification in the well-established territory of aphasiology misleading or fanciful or is it a productive, thought-provoking effort leading to further knowledge?" I also certainly hope that the second premise will turn out to be true.

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JANE MARSHALL

Marshall (2010) examines the concern presented by different authors (e.g., Caramazza & McCloskey, 1988) of the value of classifying people with aphasia. She initially refers to the distinction between primary aphasias, where language skills are directly affected, and secondary aphasias, where symptoms arise from impairments outside the language system. Marshall points to the two primary forms of aphasia (Wernicke-type and Broca-type), emphasising that this distinction is not new and indeed it is well established in the realm of aphasia. She further emphasises the fact that aphasias fall pervasively into these broad descriptive categories across all languages, including sign language, and there is a significant association with frontal and posterior lesion sites and grammatical/lexical disturbances.

Marshall argues that despite these general patterns, problems remain for the Broca's and Wernicke's classifications, mainly because of the degree of variability observed between category members. To illustrate this point she presents the assessment data from four individuals with chronic aphasia arising from posterior lesions. These four patients presented a significant variability in the different language tests that were administered: Spoken word to picture matching, Auditory lexical decision, Written word to picture matching, Repetition, and Naming. I certainly agree that it is difficult for any classification system to accommodate such variability. Nonetheless, we have to keep in mind that a significant percentage of aphasic patients cannot be classified into a single aphasia category, but simultaneously correspond to two (or more) subtypes of aphasias, although one subtype may predominate. The same observation is also valid in other clinical syndromes (e.g., agnosia, apraxia, dementia, etc.). The four patients presented by Marshall may potentially correspond to these mixed aphasia groups. By the way, in my proposed classification transcortical (extra-Sylvian) sensory aphasia corresponds

to Wernicke-type aphasia with an impairment at the semantic level (see Figure 1 of the target paper).

The problem for therapy stated by Marshall remains, and certainly if there is too much variability between members of the same aphasic sub-group we cannot be confident that they will respond positively to the same therapy. Although general therapy guidelines can be taken (e.g., melodic intonation therapy is useful in the Broca type of aphasia, but does not really work in fluent aphasias), therapy programmes certainly have to be tailored to each patient.

Finally, I want to state that I am in full agreement with the general conclusions presented by Marshall: (1) Syndrome labels can be retained providing we are clear about the limit of their contribution. (2) While not prescribing therapy, the syndrome label makes some treatment targets more likely than others.

Again, I express my gratitude to Hugh W. Buckingham, Andrew Kertesz, and Jane Marshall for their comments and suggestions. I have found all their observations most important in understanding the nature and characteristics of aphasic disorders.

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