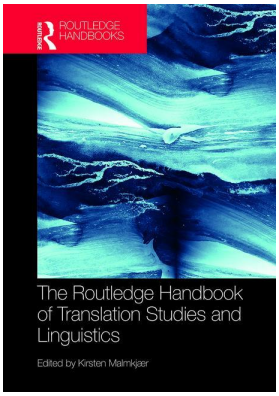

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Language disorders, interpreting, and translation

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Introduction

The study of the language disorders associated with brain pathology (aphasias) represents the real beginning of cognitive and behavioural neurosciences. The analysis of aphasia as a matter of fact is a crucial question not only for neurology and related clinical areas, but also for linguistics, neuropsychology, psychology, and speech therapy. Throughout contemporary history, aphasia has been and continues as one of the most significant and extensively analysed brain syndromes.

Aphasia can be defined as the loss or impairment of language caused by brain damage (Benson and Ardila 1996). The modern conception of aphasia was introduced in 1861 at the Anthropological Society of Paris when Paul Broca presented the case of an individual who suffered a loss of language associated with brain pathology; the patient had a very limited expressive language (virtually limited to a syllable: "tan"), produced with effort; language grammar was absent. Thirteen years later Karl Wernicke described a second type of language disorder characterised by defects in language understanding.

In this chapter three different questions will be approached. Initially, a clinical description of the language disorders associated with brain pathology will be presented. A distinction will be introduced between the fundamental or major aphasic syndromes (Wernicke's and Broca's aphasia) impairing language phonology, lexicon, semantics and, grammar, on the one hand; and other aphasic syndromes affecting the ability to produce language or the executive control of the language, on the other. In the following section, the brain organisation of language in bilinguals will be examined; it will be emphasised that there are some crucial variables (such as the age of acquisition of the second language) that significantly affect the pattern of organisation of the language in the brain. In the last section the question of aphasia in bilinguals will be approached; different patterns of clinical manifestation and recovery will be distinguished. Disturbances in the translation ability observed in bilinguals will be considered.

Language disorders associated with brain pathology

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; Ardila 2014; Benson and Ardila 1996; Head 1926; Hécaen 1972; Kertesz 1979). These two major aphasic syndromes have been related to two basic linguistic operations: selecting (language as paradigm) and sequencing (language as syntagm) (Jakobson 1971; Jakobson and Halle 1956; Luria 1972/1983). In cases of brain pathology it can be observed that an individual may lose the ability to use language in two rather different ways: the language impairment can be situated on the paradigmatic axis (similarity disorder) (found in Wernicke's aphasia), or the syntagmatic axis (contiguity disorder) (found in Broca's aphasia). Thus, in cases of brain pathology language can be impaired in two different ways: at the lexical/semantic level (in Wernicke's aphasia) or at the grammatical level (in Broca's aphasia). Consequently, so-called Wernicke's and Broca's aphasia represent the major (or fundamental) aphasic syndromes

Major aphasic disorders

Wernicke's aphasia

Wernicke's aphasia (also known as sensory or receptive aphasia) results from pathology in Wernicke's area. Wernicke's area corresponds to the auditory association area of the left hemisphere of the brain. The primary auditory area corresponds to Brodmann's area (BA) 41 (Heschel's gyrus, or the transverse temporal gyrus), and some authors also include BA42. It can be assumed that Wernicke's area (the auditory association area of the left hemisphere) corresponds to BA22, BA21, and BA37; frequently BA39 is also included (see Figure 17.1).

In Wernicke's aphasia word-finding difficulties are evident, the patient's vocabulary is decreased, and language-understanding disturbances are significant. Wernicke's aphasia patients may not fully discriminate the acoustic information contained in speech, that is, they may fail to recognise the language phonemes (a defect known as auditory verbal agnosia). Lexical (word) and semantic (meaning) associations may also become deficient. In Wernicke's aphasia the language deficit can consequently be situated at three different levels: at the level of language sound (phonemes) recognition, at the level of vocabulary use and understanding (lexicon), and finally at the level of semantic associations (word meaning comprehension). Phoneme and word selection are deficient, but language syntax is well preserved and even overused.

Speech is produced without effort. No articulatory defects (dysarthria) are observed. Fluency is normal and frequently there is excessive language output. Often extra syllables in words and extra words in sentences are found; this excessive amount of language without a clear meaning is referred to as logorrhoea. Because of the relative absence of meaningful words (so-called "empty speech") and the excessive language output, an overuse of grammatical words (frequently incorrectly selected) is found; this phenomenon is known as paragrammatism (or dyssyntaxis). Dyssyntaxis has been defined as "Pathological linguistic productions in which are observed a fairly large number of sentences that transgress one or more of the normative rules of the community's morphosyntactic convention" (Berube 1991, 62).

Paraphasias (i.e., incorrectly produced words) are abundant. Paraphasias can be both phonological (i.e., the phonological sequence is incorrect because of phoneme substitutions, additions, or omissions) and verbal (a word is replaced by another – usually semantically related – word), even though phonological or verbal paraphasias can predominate in a specific patient. Frequently, neologisms (understandable pseudo-words) are also found. When a patient presents abundant (even excessive) verbal output that is difficult to understand due to

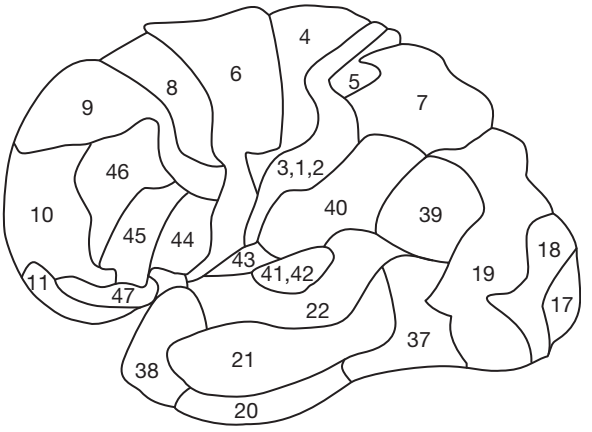

Figure 17.1 Wernicke's area roughly corresponds to Brodmann's areas 22, 21, and 37, although 39 is also frequently included

the significant amount of paraphasias and neologisms and a relative absence of meaningful words (nouns), the term jargonaphasia is frequently used.

These patients present significant difficulties in language understanding. However, language-understanding impairments present significant variations according to different contextual conditions. When short sentences are used, it is notoriously easier for the patient to understand; increasing the number of words in speech results in more severe language-understanding defects. Language understanding requires an increased attention and continuous effort (similar to the attention and effort required to understand a foreign language). Usually at the beginning of the conversation the patient has very remarkable language comprehension defects, but progressively language understanding increases. The language understanding remains relatively high for some short time (maybe 15 or 20 minutes), but later it begins to decrease (this is known as the "fatigue phenomenon"). Furthermore, if changes in the conversational topic are introduced, language understanding immediately decreases. So, language-understanding difficulties are variable according to the specific conversational conditions.

It is assumed that there are two major defects accounting for the language-understanding defects in Wernicke's aphasia. (1) Defects in phoneme discrimination; the extreme situation (i.e., complete inability to discriminate speech phonemes) corresponds to the so-called "pure word-deafness" syndrome. Usually, patients with Wernicke's aphasia have some defects in phoneme discrimination; in severe cases, the patient can suggest that s/he is unable to understand the phonological composition of speech (for instance, s/he may state that other people seem to be speaking using a foreign language, or even that they are not really speaking but making noises). (2) Defects in verbal memory: the patient cannot recall the previously learned verbal information, such as words, sentences, and in general verbal knowledge (i.e., there is a retrograde verbal amnesia); and the patient also has significant difficulties in memorising new verbal information (i.e., there is an anterograde verbal amnesia). For instance, repetition of sentences is limited to sentences consisting of three or four words. However, for understanding conversational language, it is required that an individual is able to keep in operative (working) memory some seven to eight words, and hence, to be able to repeat sentences consisting of seven to eight words. It is consequently obvious that patients with Wernicke's aphasia cannot correctly understand spoken language.

Broca's aphasia

Broca's aphasia – also known as motor aphasia, expressive aphasia, or kinetic motor aphasia – is observed in cases of damage to the so-called Broca's area. Broca's area corresponds to the third frontal gyrus (F3) and is typically defined in terms of the *pars opercularis* and *pars triangularis* of the left inferior frontal gyrus, represented in Brodmann's cytoarchitectonic map as areas BA44 and BA45 (see Figure 17.1).

Speech in Broca's aphasia is not fluent, but language understanding is relatively normal. Repetition is abnormal due to the apraxia of speech; as a matter of fact, during repetition, the same disturbances observed in spontaneous speech are found. Pointing in response to requests like "show me the . . . ." is relatively normal and illustrates relatively normal language understanding.

Motor difficulties are found in the overwhelming majority of patients with Broca's aphasia. A right hemiparesis (weakness in the right hemi-body), more distal (the hand) than proximal (the shoulder) is usually found. Hemiparesis varies in severity but frequently corresponds to a hemiplegia (inability to move the right hemi-body). The hemiparesis is observed in the right arm and face, but it is milder in the right leg. Because of the motor disturbance, dysarthria is almost invariably found; the dysarthria corresponds to a spastic type of dysarthria characterised by an increased muscle tone. Depending on the extent of the damage in the parietal lobe, somatosensory abnormalities can be found, such as right hemi-body hypoesthesia (decreased sensibility in the right hemi-body), two-points discrimination (ability to discern that two nearby objects touching the skin are truly two distinct points, not one) defects, difficulties in localising tactile stimuli in the right hemi-body, etc.

Aphasic individuals presenting Broca-type aphasia (a continuity or syntagmatic disorder according to Jakobson 1971) 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 function (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 (e.g., "dog") and in general, there is a reduction in resources available for syntactic processing (Caplan 2006). This disturbance in the use of grammar is known as agrammatism. Agrammatism is also observed in language understanding; so, these patients have difficulties understanding sentences whose meanings depend on their syntax (e.g., "The dog was bitten by the cat"; who was bitten, the dog or the cat?).

Stereotypes (restricted expression repeatedly used by the patient, as if it were the only language form available) are frequently found (for instance, the initial patient described by Broca in 1863 had a single sterotyped utterance ("tan") that he repeated when attempting to speak). Stereotypes can be short (for instance, a syllable, as in Broca's patient's "tan"), or long (for instance, "/*beintisinko*/"); can be meaningful (e.g., "pencil") or meaningless (e.g., "sood"). Occasionally, the stereotype corresponds to a profanity (that obviously becomes particularly embarrassing not only for the patient but also for other people!). The origin of the specific stereotype is not well understood, but it has been suggested that it corresponds to some language information existing exactly before the onset of the aphasia.

Patients with Broca's aphasia present a defect in making precise articulatory movements; that results in a significant amount of phonetic deviations (inaccurate production of phonemes), occasionally resulting in so-called "foreign accent in aphasia" ("perceived" foreign accent in speech).

In addition to phonetic deviation, patients with Broca's aphasia present a significant amount of phonological paraphasias. Phonological paraphasias in this type of aphasia are

mostly due to phoneme omissions and phoneme substitutions. As a matter of fact, patients can have significant difficulties in producing certain phonemes (e.g., fricative phonemes) and complex syllables (e.g., consonant-consonant-vowel as in "tree"); fricative phonemes are replaced by stop phonemes (e.g., /s/ becomes /t/) and complex syllables become basic syllables (that is, consonant-vowel; for instance, "tree" becomes "tee"). These verbal articulatory defects in Broca's aphasia are known as apraxia of speech. Phonological paraphasias are a result of the apraxia of speech.

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 impairments, etc. that is usually referred as apraxia of speech); and (b) agrammatism (e.g., Benson and Ardila 1996; 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 *et al*. 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*. 2004).

Other aphasic disorders

In addition to the two major aphasic syndromes (Broca's aphasia and Wernicke's aphasia), different aphasia classifications generally include a diversity of language disturbances, such as conduction aphasia, transcortical (extrasylvian) aphasia, anomic aphasia, etc. Indeed, some aphasic syndromes can be considered as variants of Broca's and Wernicke's aphasias. For instance, amnesic or anomic or nominal aphasia (usually due to damage in the vicinity of BA37) can be interpreted as a subtype of Wernicke's aphasia in which the semantic associations of words are significantly impaired. By the same token, extrasylvian (transcortical) sensory aphasia can also be regarded as a subtype of Wernicke's aphasia, and indeed, that is the interpretation proposed by some authors (e.g., Lecours, Lhermitte, and Bryans, 1983).

Conduction aphasia

Conduction aphasia was initially described by Wernicke in 1874, and interpreted as a disconnection between the left superior temporal gyrus (Wernicke's area) and the left inferior frontal gyrus (Broca's area) (Wernicke 1874). Wernicke's interpretation was supported by Geschwind during the 1960s (resulting in the so-called Wernicke–Geschwind model of language), who put it in terms of modern anatomic nomenclature, attributing to the arcuate fasciculus the main role in the speech repetition disturbances. According to Geschwind (1965), disconnection syndromes were higher-function deficits that resulted from white matter lesions or lesions of the association cortices; conduction aphasia was usually presented as the prototypal example of a disconnection syndrome. This remains its most frequent interpretation (e.g., Damasio and Damasio 1980): conduction aphasia is usually due to a lesion affecting the arcuate fasciculus (Yamada *et al*. 2007) and sporadically an indirect pathway passing through the inferior parietal cortex and the insula (Catani, Jones, and Ffytche 2005) (see Figure 17.2).

According to Benson *et al*. (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

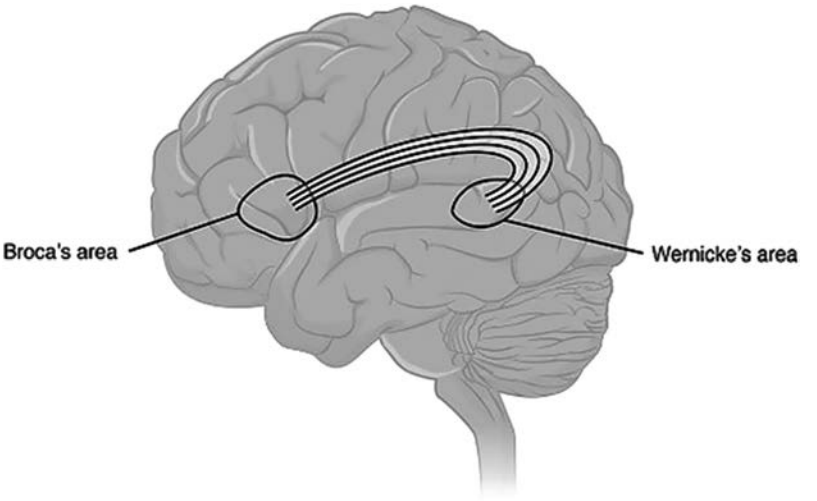

Figure 17.2 Explanation of conduction aphasia as a disconnection between Wernicke's area and Broca's area

(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* (behaviour of approximation). Language comprehension (auditory and reading) is only mildly impaired.

When attempting to repeat (but also on occasion in spontaneous language), a patient with conduction aphasia presents successive approaches to the target word (*conduit d'approche*); every time s/he produces the word, the patient recognises it has been incorrectly produced (because language understanding is preserved), and attempts to correct it. A significant amount of phonological paraphasias are observed and, from time to time, verbal paraphasias are also found. Sometimes it is impossible to produce the word during repetition, but not in spontaneous language.

Transcortical (extrasylvian) sensory aphasia

Transcortical (or extrasylvian) sensory aphasia (TSA) has been a polemic syndrome; frequently it is considered as a subtype of Wernicke's aphasia. 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. Two integrative revisions of TSA are available (Berthier 1999; Boatman *et al*. 2000).

In general, it is considered that TSA includes the following elements: (1) good repetition (the patient repeats words and sentences presented by the examiner, regardless of whether they are incorrect and even in a foreign language); (2) fluent conversational language; (3) significant amount of verbal paraphasias and neologisms; and (4) empty speech. TSA presents similar deficits as in Wernicke's aphasia, but repetition ability is spared and phoneme discrimination impairments are not found. Some authors also include a semantic jargon in the definition of TSA (Goodglass 1993; Kertesz 1982), 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).

Transcortical (extrasylvian) motor aphasia

Transcortical (or extrasylvian) motor aphasia (also known dynamic aphasia; Luria 1966, 1980), is associated with left convexital prefrontal damage. It is characterised by nonfluent language, good comprehension, and good repetition. Prosody, articulation, and grammar are preserved. The patient presents long latencies in language when beginning to speak or when answering questions. Answers to open questions are slow and incomplete, and the patient tends to repeat the words included in the question. Expressive language is limited with some tendency to echolalia and perseveration; occasionally verbal paraphasias are observed. This type of aphasia could be interpreted as a language disturbance at the pragmatic level (use of the language according to the specific social context).

Extrasylvian (transcortical) motor aphasia could be interpreted as an executive function defect specifically affecting language use. The ability to actively and appropriately generate language appears impaired while the phonology, lexicon, semantics, and grammar are preserved. Simply speaking, the question is: should the ability to correctly generate language be regarded as a linguistic ability (i.e., a cognitive ability)? Or rather, should it be considered as an executive function ability (i.e., a metacognitive ability)? Consequently, extrasylvian (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. Extrasylvian (transcortical) motor aphasia could indeed be referred to as "dysexecutive aphasia" (Ardila 2010).

Aphasia of the supplementary motor area

In 1940, Brickner reported that electrocortical stimulation of the supplementary motor area (SMA) (mesial aspect of BA6) resulted in continuous perseveration (Brickner 1940). Penfield and Welch (1951) observed arrest of speech associated with stimulation of this cortical region. However, language disturbances associated with SMA pathology were 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 (that irrigates the SMA) represents its most frequent aetiology, but it has also been reported in cases of tumours and traumatic head injury (e.g., Ardila and 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) nearly normal speech repetition; (4) 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. Spontaneous language is limited, but language understanding and language repetition are normal; there is a significant difficulty in initiating and maintaining speech, even if the patient makes significant effort to speak; reading aloud is defective but reading understanding is nearly normal; writing is slow and painstaking. It is noteworthy that this type of aphasia has sometimes been interpreted as an extrasylvian (transcortical) motor aphasia.

The SMA is a mainly mesial premotor area involved in the ability to sequence multiple movements performed in a particular order (Tanji and 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, and Jessell 1995). SMA damage is also associated with slow reaction time (Alexander *et al*. 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 assumed (Fried *et al*. 1991). 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.

Brain organisation of language in bilinguals

It has been suggested that in bilingual individuals the brain representations of L2 differ in comparison with those of L1; this dissociation in the brain organisation of L1 and L2 is considered to be particularly evident in late bilinguals (people who have learnt L2 after L1 has been acquired; that is, after the age of about 12 years) (Fabbro 2001; Mouthona, Annonia, and Khatebc 2013). Evidently, when L2 is acquired late in life, it is generally mediated by L1; and even the language-learning strategies and mechanisms can be different. For instance, grammatical knowledge and processing is usually associated with procedural memory in L1 (that is, it is a kind of automatic learning), but it is more dependent on declarative memory for L2 (that is, it is more consciously controlled).

So, potentially L1 and L2 can have different brain representation (divergent representation), or coincident brain organisation (convergent representation). Divergent representation means that each language has at least a partially different cerebral organisation than the other. Convergent representation refers to a similar brain organisation of L1 and L2.

The question of language representation in the brain was originally raised on the basis of clinical observations of bilingual aphasic patients. Severity of impairment and recovery characteristics for L1 and L2 were taken into consideration. Several of these reports showed an unequal pattern of impairment in the two languages (selective impairment) (e.g., Fabbro and Paradis 1995), whereas others suggested an unequal pattern of recovery of the two languages (selective recovery) (Paradis 1993), congruent with a different (divergent) brain organisation of L1 and L2.

At a certain point in the history of this question, it was proposed that whereas L1 was organised in the left hemisphere, L2 was more bilaterally represented, indicating an increased involvement of the right hemisphere in L2 (e.g., Wesche and Schneiderman 1982). As for this hypothesis of a more bilateral language representation for L2, clinical studies have shown that the incidence of aphasia with a right hemisphere lesion (in what is commonly called "crossed aphasia") in bilingual subjects is similar to that observed in monolinguals, thereby infirming the assumption of a different representation of languages for L1 and L2.

Clinical observations suggested that different languages might rely on distinct neural substrates (Fabbro, Naatanen, and Kujala 1999). As a matter of fact, electrocortical brain stimulation studies also provided evidence for distinct representation of the different languages in the brain. Various reports indicated that the stimulation of different cerebral locations, during neurosurgery monitoring, interfered unequally with the patients' languages (Giussani *et al*. 2007). These data suggested that some cortical sites might selectively be involved in one of the languages but not the others. By the same token, some functional neuroimaging investigations reported different brain activation between L1 and L2, with different experimental paradigms in regions such as Broca's area, the cerebellum, supramarginal gyri, and others (Dehaene *et al*. 1997).

The alternative possibility, that the different languages converge into the same brain regions and that linguistic knowledge of L1 and L2 are processed by the same neural network, also finds support in both clinical and experimental data. The theory of convergence proposes that the acquisition of L2 even in late learners involves the same brain network used for the L1 as in monolingual speakers, including the processing of semantic, syntactic, phonological, but also grammatical information (Mouthona *et al.* 2013). A common language representation for the different languages in bilinguals had been supported by other clinical studies that reported *similar impairments* in both languages after brain damage (and a *parallel recovery*) (Fabbro 2001).

So, contrary to some functional studies that supported the hypothesis of a differential brain representation for L1 and L2, other studies suggested that the first and second languages use the same neural circuits (e.g., Illes *et al*. 1999). Support for the language representation convergence theory was reported in picture naming, and semantic judgement tasks, where overlapping brain activation was observed for L1 and L2 (Mouthona *et al.* 2013).

Nowadays, the discrepancy between the data provided by the different fields of research or from different studies from the same field (e.g., functional imaging research) are thought to result either from factors inherent to the populations studied (low vs. high proficiency, early vs. late, or balanced vs. unbalanced bilinguals) or from a bad definition of the question addressed.

In conclusion, contemporary research supports both points of view: L1 and L2 have a coincidental brain organisation; and also, L1 and L2 have a partially different brain organisation; L2 uses more extended brain circuits, even involving the right hemisphere. Age of acquisition of L2 and proficiency in L1 and L2 are supposed to represent the two most crucial variables accounting for this difference in brain organisation (Fabbro 2001; Mouthona *et al*. 2013). In early balanced bilinguals both languages have a coincident brain organisation; in late unbalanced bilinguals, brain organisation of L1 and L2 is partially different.

Aphasia in bilinguals

Worldwide, some 6,800 different languages are spoken (http://www.ethnologue.com), and over half of the world's population is bilingual or multilingual. This means that over half of the cases of aphasia are bilingual aphasias.

Parallel and dissociated aphasia

Different clinical observations have demonstrated that bilingual aphasics do not necessarily manifest the same language disorders with the same degree of severity in both languages (Albert and Obler 1978). Aphasia can be parallel (both languages are impaired in a similar way) or dissociated (there is a different aphasia profile for each of the languages). Fabbro (2001) observed, in a sample of 20 bilingual aphasics, parallel aphasia in 65% of the subjects; in the rest (35%) aphasia was dissociated: 20% showed a greater impairment of L2, while 15% of the patients showed a greater impairment of L1. These percentages can be considered relatively typical.

It is assumed that parallel aphasia is usually found in early bilinguals, whereas dissociated aphasia is characteristic of late bilinguals. As a matter of fact, language representation of both languages can be regarded as coincidental in early bilinguals, whereas language representation of L1 and L2 is not completely coincidental in late bilinguals. L2 seems to be acquired

through the same neural structures responsible for L1 acquisition; that means that language understanding is related to the left temporal lobe (Wernicke's area) whereas language production is based on left inferior frontal gyrus activity (Broca's area); however, neural differences may be observed, in terms of more extended activity of the neural system mediating L2 processing (Abutalebi 2008). Indeed many studies have reported that later acquired languages may involve broader activation locations than the first acquired language; largely overlapping, but sometimes distinct cortical areas are involved in the comprehension and production of first and second languages (Obler *et al*. 2007).

In cases of dissociated aphasia, usually the most impaired language is L2, but sometimes, it can be L1. For instance, Ardila (2008) reported the case of a 63-year-old right-handed female native Spanish speaker, who had been living in the USA for 38 years. She never studied English in a formal way, but after years of having been exposed to it, she had learned some English. Suddenly, she presented an extensive left temporal intracerebral haemorrhage. A significant language-understanding defect was found, associated with severe impairments in verbal memory (Wernicke's aphasia), difficulties in language repetition, severe anomia with phonological and semantic paraphasias and neologisms, alexia, and aphasic agraphia. The naming defect was more severe in Spanish than in English; furthermore, there was also a clear tendency to answer in English, to switch to English, and mixing English and Spanish. The patient presented a dissociated aphasia with a better conservation of L2 (English) than L1 (Spanish).

Occasionally, it has been reported that bilinguals can present a different pattern of aphasia in L1 and L2. For example, Silverberg and Gordon (1979) reported two cases of dissociated aphasia; following a left parietotemporal lesion, moderate nonfluent aphasia was found in the native language of the first patient, in contrast to less severe, fluent aphasia in the patient's L2. Conversely, mild anomia was found in L1 of a second patient, while global aphasia was found in L2. His lesion was located in the left posterior frontal area.

Patterns of L1 and L2 recovery

Two opposite points of view were proposed during the 19th century to explain language recovery in bilingual aphasics.

- Ribot's law or Ribot's (1883) rule. This states that the language best recovered by polyglot aphasics is the mother language.
- Pitres' law or Pitres' (1895) rule. Pitres described seven cases of bilingual aphasics presenting differential recovery of the two languages. He suggested that patients tended to better recover the language that was *most familiar* to them prior to the aphasia onset, even if it was not the mother tongue.

Paradis (1977) refers to six different patterns of aphasia recovery in bilinguals.

1. Differential. Each language is impaired separately and recovered at the same or different rate
2. Parallel. Different languages are similarly impaired and restored at the same rate.
3. Antagonistic. Recovery of one language progresses, while the other regresses.
4. Successive. One language does not show any recovery until another has been restored.
5. Selective. One language is not recovered at all.
6. Mixed. Both languages are used in some combinations.

However, most patients present the first (differential) or second (parallel) recovery pattern. The other patterns are indeed unusual. Fabbro *et al.* (1999) report a parallel recovery in about 40% of cases, a better recovery of L1 in 32% of patients, and a better recovery of L2 in about 28% of cases.

How translating ability can be affected

Translation ability represents a crucial ability in bilinguals. It is generally thought that translation is easier from L2 to L1 than in the opposite direction. Translation can be difficult in early bilinguals, because the words in both languages can have different semantic associations; that is, early bilinguals are coordinate bilinguals (Weinreich, 1953). Translation can be included as a specific task in testing bilingual aphasics; and as a matter of fact, it is a subtest in the Bilingual Aphasia Test (Paradis and Libben 2014; www.mcgill.ca/linguistics/research/bat). The preserved ability to translate can be regarded as an index of complex language control.

In bilingual aphasics, mixing of the languages has been frequently observed (Ardila 2008; Perecman 1984). Patients may become unable to separate their languages and to use each one in the appropriate circumstances. Patients can present mixing of languages within a single utterance, or pathological switching, alternating their languages across different utterances; language mixing is most frequently observed in cases of left temporo-parietal pathology. Reports about pathological language switching are not frequently found; Fabbro, Skrap, and Aglioti (2000) described a patient with a lesion to the left anterior cingulate and to the frontal lobe – also marginally involving the right anterior cingulate area – who presented with pathological switching between languages in the absence of any other linguistic impairment.

Idiosyncratic disturbances in the ability to translate have been documented in aphasic patients. Paradis, Goldblum, and Abidi (1982) analysed two patients presenting a pattern of recovery yet unreported. Both patients alternately suffered severe word-finding difficulties in one language while remaining relatively fluent in the other. They retained good comprehension in both of their languages at all times. They were able to translate correctly and without hesitation from the language they could speak well at the time into the language unavailable for spontaneous use, but were unable to translate from their temporarily poor language (which they understood well) into the language they could speak quite well at the time. Aglioti *et al.* (1996) reported a bilingual patient who presented with an uncommon pattern of aphasic deficit consequent to subcortical lesions mainly involving the left basal ganglia. The patient's mother tongue was Venetian, whereas her L2 was standard Italian. The patient had more difficulties when translating into her mother tongue than into her second language; this asymmetrical pattern in translating is not expected; as mentioned above, translating is generally considered to be much more difficult from L1 to L2 than from L2 to L1

Finally, it should be mentioned that the ability to translate between languages represents a complex linguistic ability that potentially could be used in aphasia rehabilitation with bilingual patients. In language therapy it has been documented that recovery of a word in one language usually generalises to the other language for cognate words (e.g., English "fruit", Spanish "*fruta*"), but not for non-cognate terms (e.g., English "pencil", Spanish "*lápiz*") (Roberts and Deslauriers 1999) suggesting that cognate words have a common brain representation in bilinguals. However, this cognate effect is variable across patients (Hughes and Tainturier 2015), probably depending upon the specific patient's bilingualism characteristics.

Further reading

Ardila, A. 2010. A proposed reinterpretation and reclassification of aphasia syndromes. *Aphasiology* 24(3), pp. 363–394.
This journal article is a review of the different aphasia syndromes and a proposed interpretation of aphasia.

Ardila, A. and Ramos, E., eds. 2007. *Speech and Language Disorders in Bilinguals*. New York: Nova Science Publishers.
A collection of articles about the manifestation of speech and language disorders in bilinguals.

Benson, D. F. and Ardila, A. 1996. *Aphasia: A Clinical Perspective*. New York: Oxford University Press.
This is a basic aphasia textbook.

Papathanasiou, I., Coppens, P. and Potagas, C. 2012. *Aphasia and Related Neurogenic Communication Disorders*. Burlington: Jones & Bartlett Learning.
This work presents an integrated review of different speech and language disorders.

Related topics

Bilingualism, translation, and interpreting; Language processing in translation.

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