## Clinical Neurolinguistics of Bilingualism

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#### Introduction

The notion of bilingualism refers to all those people who use or simply know two or more languages, including dialects (Grosjean, 1994). Just to provide some figures, let us consider that something like 7,000 languages are spoken in barely 160 countries. Furthermore, in many of these countries the number of spoken languages is constantly growing due to massive immigration. This means that over 50% of the world population knows at least two languages with different degrees and can be consequently considered not only bilingual but even multilingual (Tucker, 1998).

The issue of language representation and use in multilingual speakers can be approached at different levels of description. Indeed, it constitutes a matter of interest not only for sociolinguistic models of language use and stratification, but also for psycholinguistic theories of language development and functioning, as well as for neurolinguistic models of language representation in the brain, its loss and potential recovery. As such, the issue of bi-/multilingual competence constitutes an important line of research for both clinical and theoretical neurolinguistics (Fabbro, 2001a).

Accordingly, after introducing the issue of the definition of multilingual competence, this chapter will explore the loss and recovery patterns in both adult and child bi-/multilingual speakers with different etiologies (aphasias, specific language impairment, neuropsychiatric and progressive neurodegenerative disorders). In the final section of this chapter, the focus will be shifted to the critical problem of multilingual assessment in the neuropsychological evaluation of linguistic deficits in multilingual patients.

#### A Quick Tour of Linguistic Competence

Language is a complex cognitive function based on the interaction between several levels of processing: a phonological level, crucial for the abstract categorization of the sounds of a language; a word level where lexical processing occurs; a sentence level for syntactic processing; a pragmatic level where words or sentences are contextualized and inferences are drawn; and a text/discourse level where those sentences that make up a written text or a spoken discourse are integrated to get its general meaning or gist (Marini, 2008; Caplan, 1992).

Each aspect of language processing is subserved by neurofunctionally distinct systems that can be separately compromised, inhibited, or preserved after a brain lesion or transient inhibition due to electrical stimulation, paroxystic charge, or pharmacological inhibition (e.g., Ojemann, 1991). According to the declarative/ procedural model (Paradis, 1994, 2004; Ullman, 2001, 2004), language learning and representation are assumed to be subserved by two anatomically and functionally distinct long-term memory systems (declarative and procedural, respectively). Procedural memory is a type of implicit memory implemented in frontal/basal ganglia circuits as well as portions of the parietal cortex, superior temporal cortex, and the cerebellum (Ullman, 2001). Procedural memory underlies implicit linguistic competence. During first-language acquisition, it is involved in the process of learning and consequently executing sensorimotor and cognitive skills such as, for example, those involved in the articulation of the sounds of a language and in syntax. Declarative (or explicit) memory is implemented in bilateral medial and temporoparietal structures, including the hippocampal region and the parahippocampal cortex (Ullman, 2001). It is implicated in conscious learning of facts and events and consists of two subtypes, semantic and episodic memory, respectively. Semantic memory is the system storing one's encyclopedic knowledge of the world (e.g., knowledge about the meaning of words, as well as knowledge about historical events, geographical notions, and social facts). Episodic (or autobiographical) memory refers to one's past experiences that can be consciously recalled. It is assumed that grammar (i.e., syntactic and morphosyntactic implicit competence) is acquired incidentally through procedural memory, whereas lexical semantic explicit knowledge is overtly learned and stored in declarative memory (Paradis, 1994; Ullman, 2001). For example, procedural memory is involved in the acquisition and use of implicit syntactic rules such as principles of government and binding (e.g., Chomsky, 1995) or procedures of syntactic parsing (Frazier & Fodor, 1978). Furthermore, the procedural system is involved in the acquisition and application of word formation rules to morphemes and lexical items stored in declarative memory. Moreover, once access to a lexical item is granted, implicit memory procedures automatically generate the argument structure of that particular word and assign the thematic roles to the required arguments (morphosyntactic processing). As to phonetics, the articulatory programs necessary to produce the target phones of a language become automatized and are transferred to procedural

memory, so that the speaker does not need to think about the articulatory movements while speaking.

#### What about Multilingual Competence?

Our intuitive knowledge of "bilingualism" falls short of a precise definition. Indeed, experts in this field have coined several terms to distinguish among different kinds of bilingualism according to different factors (Edwards, 2004). For example, when the focus is on proficiency, a distinction between balanced and dominant bilinguals has been suggested (Peal & Lambert, 1962): a *balanced bilingual* masters the two languages to the same extent, whereas a *dominant bilingual* is more fluent in one language than in the other. If the interest is on the effect exerted by a second, third or *nth* language (from now on simply L2, L3, L*n*) on the mother tongue (from now on L1 or first language), Lambert (1974) proposed a distinction between additive and subtractive bilingualism, where *additive bilinguals* are those who lower the proficiency in their L1 while mastering a second language. If the factor taken into account is the age of acquisition, then it is possible to distinguish between *early* and *late bilingualism* depending on whether two or more languages have been acquired "early" (i.e., during infancy) or much later than the L1.

In sum, bilingualism has many faces. However, these interpretations of the differential aspects of bilingual competence do not provide a clear definition of what bilingualism is. According to Bloomfield (1933), only those people who have a native-like control of two languages are to be considered bilingual. Therefore, we might extend this definition to identify as multilinguals only those who have nativelike control of more than two languages. However, such a rigid account of bilingualism leaves open the problem of the definition of quite abstract notions such as "balanced," "ideal," or even "perfect" bilingualism. Do perfect bilingual people really exist? It is no doubt hard to find completely balanced bilinguals. When we turn to consider multilingual individuals, the issue becomes even more problematic, as we should exclude from the count of multilingual speakers all those who know more than two languages but simply not as much as they know their L1. Such a view of bi-/multilingual competence has been fiercely challenged by a number of studies. Rather, the focus has shifted toward the degree of proficiency in two or more languages within the same individual. Consequently, several additional definitions have been provided. According to Grosjean (1999), bilinguals are people who know two or more languages, whether they be people who know more than one language but use only one of them in their ordinary communicative interactions (so called "dormant bilinguals") or those who effectively use all of them on a daily basis. Most importantly, bilinguals must not be considered as the sum of two complete or incomplete monolinguals (Grosjean, 1989). Indeed, the interaction of different languages in the same person produces a different but integrated linguistic entity with its own characteristics that cannot be simply reduced to the sum of the parts.

As a matter of fact, bilingual individuals can acquire their languages at different times and in different ways. Furthermore, they can use them with different people and in different situations. This means that bilinguals may be placed at various points of

a situational continuum which induce a particular speech mode. At one end of this continuum, bilinguals are in a totally monolingual speech mode . . . At the other end of the continuum, they are with bilinguals who share their two languages . . . and with whom they normally mix languages (code-switch and borrow): they are here in a bilingual speech mode . . . we should keep in mind that intermediary modes exist between the two. (Grosjean, 1989, p. 8)

A growing number of neuropsychological and neuroimaging studies show that the languages mastered by bilinguals have a complex neurofunctional organization based on the interconnection between shared and distinct neural circuits (Fabbro, 2001b). It seems plausible, thus, to consider the bilingual brain not just as the sum of two or more monolingual language systems but as a "unique and complex neural system which may differ in individual cases" (Abutalebi, Cappa, & Perani, 2001, p. 188).

In what follows, for the direct purposes of this chapter, we will adopt a pragmatic definition of bilingualism. Namely, the term bilingualism (or bilingual competence) will refer to the command and use of two or more linguistic systems, whatever the level of proficiency and the age of acquisition (Marini & Fabbro, 2007).

# The Breakdown of Bilingual Competence: Clinical Features of Bilingual Aphasia

How are multiple languages represented in the bilingual brain? At the microanatomical level, that is, at the level of neuronal networks, it is reasonable to assume that the two languages are represented in completely or partially independent neuronal circuits. On the other hand, the question whether at the macroanatomical level L1 and L2 are represented in common or different cerebral structures is still under debate (Urgesi & Fabbro, 2009). However, investigations assessing language skills in bilingual speakers may provide an insight into the solution to this problem. At a first look, the logic behind the study of aphasias in bilingual individuals is very simple. If, after a brain lesion, a bilingual individual loses all of the languages that he or she had mastered before the insult, it can be assumed that those languages were implemented in the same brain areas. The same holds true if the same patient recovers all languages to the same extent and at the same pace. On the other hand, if, after the lesion, the patient shows selective impairment of one or some of the languages he or she had previously known before and/or shows differential patterns of recovery for the different languages, then we may assume that the cerebral representation of these languages must be implemented in different cerebral circuits.

Unfortunately, the picture stemming from the neuropsychological observations of clinical features of bilingual aphasic patients is not that clear due to inconsistent, often contradictory, findings (Fabbro, 1999b; 2001a). Indeed, some studies report similar impairments in all of the languages mastered by the patients before the insult, whereas others show the opposite pattern, with some languages lost and/or recovered while others remain unaltered or do not recover at all. Furthermore, the linguistic deficits observed in these patients are not necessarily stable but may change over time. The time elapsed since the cerebral lesion is a major factor affecting functional performance in brain-damaged patients. Accordingly, they may be included, sequentially, in one of the following three phases: acute phase, up to 4 weeks after onset; lesion phase, lasting for several weeks up to 4–5 months after onset; late phase, continuing for all of the patient's life.

During the acute phase the patient may present a wide set of linguistic disturbances some of which are destined to be resolved within a brief period. Such disturbances may affect the ability to use one or more of the previously mastered languages for communicative purposes or the ability to process phonological, lexical, and/or grammatical features specific to a given language. Examples include patients with selective dysfunction in the use of one of their languages with preserved linguistic abilities in the other(s) (selective aphasia, e.g., Paradis & Goldblum, 1989), patients with similar impairment in both languages (parallel aphasia, e.g., Fabbro, 1999b), as well as patients with severe word-finding difficulties which may occur sometimes in one language and at other times in another and good comprehension in both (alternating antagonism, e.g., Paradis, Goldblum, & Abidi, 1982; Nilipour & Ashayeri, 1989). Such variability may depend on diaschisis, a process that causes functional alterations in areas that are structurally unaffected but functionally connected to the damaged area. The good news is that this transient phase lasts approximately only 4 weeks after the insult, as the structurally unaffected areas slowly return to normal functioning with the regression of the diaschisis. This means that some of the linguistic disturbances registered immediately after the brain lesion may disappear and give way to more stable linguistic symptoms in the following phase.

In the *lesion phase*, the linguistic disorders within the same language and across the various languages mastered by the patient before the insult are far more stable and strictly related to the functionality of the lesioned tissue. These impairments may affect phonological, lexical, grammatical, or semantic processing in one, some, or even all the languages previously known by the individual with similar or different degrees of symptomatic severity in the languages (Fabbro & Paradis, 1995b). Furthermore, these linguistic symptoms can be used to classify the patient according to traditional aphasiologic classifications (e.g., Broca's aphasia, Wernicke's aphasia, anomic aphasia, global aphasia, etc.). Due to its stability, it is far more useful to carry out a complete assessment of the patients' residual linguistic abilities in this phase including an integrated assessment of all the languages he or she knew before the insult. Notably, in the lesion phase the brain-damaged bilingual individuals begin a recovery that will continue through the late phase. In the *late phase*, the recovery, either spontaneous or following rehabilitation, is generally more moderate than that occurring in the preceding phase. Furthermore, bilingual aphasic individuals present differential patterns of improvement (see the next section).

Before discussing the patterns of recovery in bilingual aphasic individuals, it is time to introduce the reader to the specific patterns of errors typically displayed by these patients. Indeed, apart from typical errors that aphasic patients may commit (i.e., literal, phonological, semantic or verbal paraphasias, paragrammatic errors, omissions, and the like), bilingual aphasic speakers produce additional errors due to a selective difficulty in dealing with specific aspects of their linguistic competence. For example, they may utter a sentence in a language and then produce the next sentence using another language. This phenomenon, called "switching," can be found in healthy bilingual individuals. However, in healthy speakers this ability is used only when there is awareness that the interlocutors share the knowledge of the two (or more) linguistic codes in use and that the communicative purpose can be achieved. In case of bilingual aphasic patients, this behavior is called "pathological switching" as it refers to a compulsive production of sentences in different languages without taking into any account the communicative needs of the interlocutors. In some cases, these phenomena are even more dramatic, as when a patient begins to mix different languages in the same sentence, in some cases even in the same word using morphemes of one language together with morphemes in another. In such cases, a distinction between pathological switching and pathological mixing phenomena has been introduced. Patients with pathological mixing intermingle different languages within a single utterance (a self-contained segment of speech that stands on its own and conveys its own independent meaning). By contrast, patients affected by pathological switching alternate their languages across different utterances.

Additional studies have established that pathological mixing typically occurs in bilingual aphasia and is mainly due to lesions in the parietotemporal structures of the left hemisphere, whereas the nervous structures responsible for switching between languages have not been clearly disentangled so far (Fabbro, 1999b). Fabbro and associates described a 56-year-old bilingual patient (L1 Friulian, L2 Slovenian) with a lesion to the left anterior cingulate and to the prefrontal lobe who presented with compulsive switching between languages in the absence of any other linguistic impairment (Fabbro, Skrap, & Aglioti, 2000). In this case, the lack of aphasic symptoms suggests that the system responsible for switching between languages is independent of language, being part of a more general system underlying the selection among different behaviors.

Bilingual aphasic patients may also present translation disorders. In some cases, a patient is no longer able to translate from one language into another (say, from L1 to L2 or the reverse). In other cases, however, things become much more intriguing and difficult to explain. Let us consider the cases of spontaneous translation, translation without comprehension, and paradoxical translation. *Spontaneous translation* is a condition in which the patients have a compulsive "need" to translate everything which is being said by the patients themselves and/

or by their interlocutors (DeVreese, Motta, & Toschi, 1988). In *translation without comprehension*, the patients do not understand commands that are given to them but can correctly translate the sentences uttered by an interlocutor to express these commands (Veyrac, 1931). Finally, *paradoxical translation* occurs when a patient can translate only into the language that he or she cannot speak spontaneously and not the reverse (Paradis et al., 1982).

#### The Recovery of Bilingual Competence: Patterns of Language Recovery in Bilingual Aphasic Speakers

In more than 60% of cases, patients who have lost two or more languages recover them at the same pace, in what is called *parallel recovery* (Fabbro, 2001a; Paradis, 2004). The high occurrence of this particular form of recovery has led to the formulation of the so-called *extended system hypothesis*, according to which the neural representations of subsequently learned languages are superimposed on the neural representations of the first language acquired (Paradis, 2004).

However, the remaining aphasic bilingual individuals (approximately 40%) display nonparallel patterns of recovery of the languages they knew before the insult. More specifically, five main types of nonparallel recovery have been described so far. The first has been termed *differential recovery*, since it occurs when languages recover differentially relative to their premorbid levels. The second, *selective recovery*, occurs when the patient recovers only one language, while the remaining language(s) are not recovered at all. The third form of nonparallel recovery, *blended* (or *mixed*) *recovery*, occurs when the patient mixes the languages during the process of recovery. So-called *successive recovery* can be observed when the temporal dynamics and the rate of recovery vary between languages. For example, two languages may eventually recover, but retrieval of the second language may only begin after the first has been fully regained. Notably, successive recovery of two languages over different time courses has also been observed in neurological patients undergoing the Wada test procedure before neurosurgery (Berthier, Starkstein, Lylyk, & Leiguarda, 1990).

The fifth form of nonparallel recovery is known as *antagonistic recovery*, and it occurs when one language recovers to a certain extent before it begins to recede as the other language begins to recover. In some cases, an *unusual* recovery of one of the languages known but never used premorbidly for communicative purposes has also been described. Exemplar is the case of a German professor of Latin and Greek described by Adhemar Gelb (1937, in Paradis, 1983) who showed aphasic symptoms after a left-hemisphere lesion. After the insult, he was no longer able to speak in German (his L1) but could still correctly express himself in Latin. On this basis, Gelb concluded that aphasic syndromes tend to affect the most automatized (i.e., unconsciously used) languages more severely, whereas the foreign languages or dead languages are best preserved since they require conscious efforts and reflection.

A second example of unusual recovery has been described in Aglioti and Fabbro (1993) and Aglioti, Beltramello, Girardi, and Fabbro (1996). Patient E. M. was an

Italian bilingual aphasic patient (L1 Veronese dialect, a variant of Venetian; L2 Italian) who strikingly recovered her L2 despite the fact that she had never used it in her daily conversations. As a child, she had attended elementary school for 3 years. In that context, she learnt to read and write Italian (L2). However, at home and with friends, she continued using Veronese on a daily basis. Indeed, her husband confirmed that she had been speaking Italian no more than two or three times a year, and that even in these rare cases she only said a few words in Italian, completing the rest with L1 words. In 1990 E. M. suffered an injury to the left hemisphere with subsequent aphasia. Magnetic resonance imaging revealed a lesion in some left subcortical structures only (mainly caudate nucleus and putamen). After a period of complete mutism lasting 2 weeks, she discovered she could not use her L1 anymore, but that she could express herself in Italian, even though the hospital staff mainly spoke Veronese. A month after the insult, she could understand both her L1 and Italian, but continued expressing herself only in Italian as she could not produce utterances or even words in her L1. Therefore, E. M. had to use Italian to communicate with her family, with adults replying in Veronese dialect and her younger nephews in Italian. In these latter conversations, E. M. noticed that she understood Italian better than Veronese dialect.

Why are there so many different patterns of language impairment and recovery in bilingual patients? As early as 1881, Théodule-Armand Ribot formulated the hypothesis that, in case of memory diseases, the later-acquired language deteriorates earlier than the old one, the so-called Ribot's rule. In 1895 a French neurologist, Albert Pitres, described seven clinical cases of patients exhibiting differential recovery of the two languages they spoke. Based on the frequency of dissociation, Pitres concluded that, if the lesion had not destroyed the language centers, patients tended to recover the language that had been most familiar to them prior to the insult. This hypothesis was subsequently called Pitres' rule. In Pitres' opinion, the patient generally recovered the most familiar language, because the neural elements subserving it were more firmly associated. Subsequently, numerous neurologists compared and contrasted the Pitres' rule (recovery of the most familiar language) with Ribot's law (recovery of the first language). However, neither the native language, nor the most familiar to the patient at the time of the insult, nor the most socially useful, nor the most affectively loaded, nor, still, the language of the environment always recovers first or best. Nor does it seem to be a matter of whether the two languages were acquired and used in the same context as opposed to different contexts or at different times of development (Paradis, 2004). Similarly, neither the type of aphasic syndrome, nor the etiology (tumor, infarction, or cerebral hemorrhage), nor the site of the lesion (cortical vs. subcortical, frontal lobe vs. temporal lobe, etc.) seem to be directly responsible for parallel language recovery versus nonparallel recovery. A possible explanation for the reason why some patients exhibit a better recovery of the second language as opposed to the first language may be found within the framework of the procedural/declarative model of language learning and representation outlined in the second paragraph of this chapter. In order to account for the high inter-individual variability in recovery patterns in bilingual aphasic patients,

Paradis (2004) has proposed a model that may explain both parallel and nonparallel forms of impairment and recovery. According to this model, referred to as the subsystem hypothesis, the representation of the languages known by a bilingual individual is organized in a unified neurofunctional system for language sustained by procedural memory (i.e., the implicit linguistic competence for each language). This system is divided into a number of neurofunctional modules, which subserve phonological, morphosyntactic, and semantic processing; in turn, each module is subdivided into as many subsystems as are the languages spoken by the individual. Each subsystem represents the specific computational procedures of each language and has an internal structure different from that of other subsystems. Linguistic parameters common to two or more languages known by a bilingual are redundantly represented in different subsystems, while language-specific parameters are represented only in the corresponding subsystem. Furthermore, the subsystems are represented in different neural structures, at least at the microanatomical level, and may be differently affected by brain lesions. Reciprocal inhibitory connections between the different language-specific subsystems prevent interference, so that when one subsystem is active, the other is inhibited. Brain damage may affect one module, e.g., phonology, with all the included subsystems, thus inducing a phonologic deficit in all languages. On the other hand, one subsystem may be selectively damaged, inducing language-specific deficits that depend not only on which subsystem has been affected but also on the extent of redundant representations among the different languages. In this context, the variability of the patterns of language impairment and recovery may be explained by considering the interaction between the inter-individual differences in the neurocognitive representation of two or more languages before the insult (determined by factors such as age of acquisition and the way in which those languages had been learned and used), and the microanatomical nature of the lesion.

In sum, no single factor can explain nonparallel impairment and recovery in polyglot aphasic patients. Rather, these might be caused by several interrelated factors independently affecting the neurofunctional organization and the proficiency levels of the languages in a bilingual brain. Several investigations have uncovered a number of such factors such as the context and the modalities of acquisition (e.g., its acquisition in a country where this language is currently spoken by means of its continuous use in real-life contexts, rather than its learning at school by means of conscious and effortful assimilation of rules and words). Similarly, other factors include the age of acquisition (early vs. late acquisition), its usage, and the level of exposure to it (high exposure vs. low exposure; Neville, Mills, & Lawson, 1992; Perani & Abutalebi, 2005; Wartenburger et al., 2003; Weber-Fox & Neville, 1996).

#### The Case of Childhood Bilingual Aphasia

Childhood aphasia is an acquired disorder due to cerebral lesions (traumatic brain injury, tumor, hemorrhage, or cerebral infarction) that occurs in childhood. In

contrast to aphasia in adults, it shows typical characteristics. Independently of the lesional site, immediately after the onset of the disease the child is either mute or tends to speak only a few words. Usually, his/her speech is usually not fluent. These symptoms last for some weeks. A second characteristic of childhood aphasia is the quick recovery of linguistic abilities. However, these children may continue experiencing word-finding difficulties and reduced lexical repertoire for the rest of their life. They often develop a simplified syntax and have writing and calculus disorders. Mutism and articulatory disorders are usually associated with left frontal lesions, whereas comprehension disorders are most of the time linked to left temporal lesions.

Language recovery in childhood is extremely fast (Vargha-Khadem, Isaacs, Watkins, & Mishkin, 2000) and apparently linked to their potential for plasticity allowing for rapid reorganization of linguistic functions. Only a few cases of acquired aphasia in bilingual children have been described so far. Bouquet, Tuvo, and Paci (1981) reported about a right-handed bilingual child whose L1 was Italian and L2 Serbo-Croatian. He used to speak Serbo-Croatian with his grandmother and cousins who did not understand Italian while using Italian with his parents and in the day nursery. When he was four, the child experienced a traumatic brain injury resulting in a lesion in the temporoparietal areas of his left hemisphere. On his awakening, after 13 days of coma, the child was completely mute for a month. During this period, he was still able to understand words in Italian. In the second month, he began to utter some bisyllabic and then trisyllabic words in Italian. The only Serbo-Croatian words he could express were *nos* (nose) and *tresnje* (cherries). Three months after the coma he also began recovering his second language.

When he was dismissed from the hospital, at the end of the third month after the coma, the child had **right**-sided spatial neglect. He used Italian quite appropriately, although his vocabulary included fewer words than expected and his speech was somewhat slow. As for Serbo-Croatian, his L2 sentence construction was labored but correct. Most strikingly, 6 months after the traumatic brain injury, both Italian and Serbo-Croatian were almost completely recovered. The patient used L2 less naturally, as if he translated from Italian to Serbo-Croatian before using the latter.

Fabbro and Paradis (1995a) have described a second case of recovery from childhood aphasia in bilingual children. K. B. acquired Friulian as her first language at home and Italian as a second language at the nursery. When she was 7 years old, she fainted and lost consciousness for a period of approximately 30 minutes. When she woke up she could neither talk nor move her right limbs. The CT scan showed the presence of a left hemispheric ischemic lesion in the frontotemporal areas and the basal ganglia. She was diagnosed with expressive aphasia with preserved comprehension in both languages. Her parents kept on talking to her in Friulian whereas the clinicians talked to her in Italian. After two weeks, K. B. began to utter some bisyllable clusters in Italian. In 1 month, she could begin to produce her first, simple, very slowly uttered sentences only in Italian (her L2). After 2 months she was dismissed from the hospital and, once back home with her Friulian-speaking family, she quickly began to produce also words and sentences in Friulian. However, for more than a year she kept on being telegraphic in both languages, omitting words and having problems with verbal inflections. Two years after the onset of the aphasic symptoms, K. B. had good clinical recovery in both languages. Seven years after the onset of the symptoms, she underwent an accurate evaluation of her linguistic skills in both languages with the Bilingual Aphasia Test (see below). The linguistic evaluation showed a consistent recovery in both languages. However, in spontaneous conversations she kept omitting function words in her L1. These data suggest an almost complete parallel recovery of both languages with the only exception of a persistent, specific problem in the morphosyntactic organization of sentences while speaking in Friulian, her L1.

#### Specific Language Impairment in Bilingual Children

So far, we have described some of the features of linguistic disturbances occurring in bilingual children with a diagnosis of aphasia. Obviously, in such cases a normal linguistic development can be assumed before the accident leading to aphasia. A very different case is that of children exposed to two or more languages that develop a form of specific language impairment (SLI), a developmental disorder characterized by language delay in children with otherwise normal physical, intellectual and cognitive development (for more details on this disorder, see Leonard & Weber-Fox, Volume 2, Chapter 40, and Schwartz & Shafer, Volume 2, Chapter 41). Too often, the condition of bilingualism is regarded in these children as one of the determinants of the impairment. However, in a seminal paper, Lebrun and Hasquin (1971) clearly showed that the linguistic impairment in bilingual children is independent from the condition of bilingualism.

In bilingual children with SLI, both languages can be impaired at the same pace and in the same way. Alternatively, the impairment might affect only one of the languages to which the child has been exposed. To the authors' best knowledge, only a handful of studies have tried to provide an answer to this important question (cf. Fabbro, 2000; Paradis, Crago, Genesee, & Rice, 2003). For example, Paradis et al. (2003) studied the use of grammatical morphemes in linguistic production tasks in three groups of children with diagnosis of SLI: monolingual English speakers, monolingual French speakers, and bilingual French–English speakers. The analysis focused on the use of inflective tense and nontense morphemes. All three groups produced more errors while using morphemes carrying information about the verb's tense compared to other inflective morphemes. It is worth noting that all children showed similar levels of accuracy in the use of these morphemes. Indeed, since the bilingual children with SLI did not differ significantly from the monolingual children with SLI, it can be arguably hypothesized that bilingualism does not hamper the process of language acquisition under conditions of impairment.

Overall, the available data suggest that SLI tends to have a similar impact on all of the languages to which the children are exposed. Here, we provide preliminary

**Table 36.1** Scores obtained from the linguistic assessment of nine bilingual SLI children in their two languages. IQ: Intellective quotient: V = verbal, P = performance; BPVS: British Picture Vocabulary Scale; TCGB: Test di Comprensione Grammaticale nei Bambini; SentR: Sentence repetition; SemFl: Semantic fluency. One asterisk (\*) indicates if the performance in one language goes below 1 standard deviation with respect to standardized scores. Two asterisks (\*\*) indicate if the performance dropped below 2 standard deviations. Table modified from Fabbro and Marini (2010).

Subject	Sex	Age	Languages	IQ	BPVS	Token	TCGB	SentR	SemFl
1	М	6	L1 = Friulian	V = 80	20	17	2	15**	10*
			L2 = Italian	P = 103	20	17.5	4	17*	15
2	F	7	L1 = Friulian	V = 65	21	17*	9**	3.5**	27
			L2 = Italian	P = 77	24	17*	11**	3.5**	17
3	М	8	L1 = Friulian	V = 82	23	16.5*	6**	3**	6**
			L2 = Italian	P = 98	20	18*	6**	9**	$14^{*}$
4	М	10	L1 = Friulian	V = 75	27	20	1.5	10**	31
			L2 = Italian	P = 98	25	20.5	1	11**	28
5	М	11	L1 = Friulian	V = 55	23	$18.5^{*}$	7**	4**	12**
			L2 = Italian	P = 83	21*	18**	10**	7.5**	25*
6	М	11	L1 = Friulian	V = 55	30	20	1	13	37
			L2 = Italian	P = 93	30	19*	3.5	12.5	23*
7	F	12	L1 = Friulian	V = 61	28	17.5**	5**	8**	21*
			L2 = Italian	P = 83	27	17**	4**	9.5**	20*
8	F	13	L1 = Friulian	V = 59	27	19.5	0	7.5**	19*
			L2 = Italian	P = 110	26	20	0.5	10**	19*
9	М	13	L1 = Friulian	V = 101	30	20.5	0.5	10**	30
			L2 = Italian	P = 95	30	20.5	1.5	12.5	27

data about a group of nine bilingual SLI children (L1 Friulian, L2 Italian) aged between 6 and 13 years. The linguistic assessment was performed using a series of tasks assessing semantic comprehension (British Picture Vocabulary Scale, BPVS; DeAgostini et al., 1998), syntactic and morphosyntactic comprehension (Token Test; Test di Comprensione Grammaticale nei Bambini, TCGB; Chilosi & Cipriani, 1995), sentence repetition, semantic fluency (Fabbro, 1999a), and picture description (Marini et al., 2008). Table 36.1 provides the scores of each individual on the tests assessing IQ, comprehension, repetition and semantic fluency. Table 36.2 reports the scores obtained analyzing the connected speech produced in a picturedescription task (for a detailed description of this task refer to Marini, Boewe, Caltagirone, & Carlomagno, 2005; Marini et al., 2008). These data suggest that almost all the bilingual children with SLI included in the study have similar impairments in both languages. This supports the hypothesis of a parallel dysfunction of the languages to which SLI children are exposed and weakens the alternative view of a selective impairment of one of the languages mastered by these children.

Measure	Friulian (L1)	Italian (L2)	p-level
Words	85.1 (32.8)	74.2 (21.2)	n.s.
MLU	9.3 (1.7)	8.7 (1.2)	n.s.
Types	21.9 (5.4)	22.9 (5.8)	n.s.
% Phonological paraphasias	0.5 (0.9)	0.7 (1)	n.s.
% Semantic paraphasias	0.3 (0.6)	0 (0)	n.s.
% Omission of content words	0.6 (0.8)	0.1 (0.3)	n.s.
% Omission of function words	2.7 (2.3)	0.5 (1.7)	<i>p</i> < .03
% Substitution of content words	3.6 (6.2)	1.3 (2.2)	n.s.
% Substitution of function words	3 (2.8)	2.2 (3)	n.s.

**Table 36.2** Means (standard deviations) and significance level of the scores obtained with the analysis of the connected speech produced in a picture-description task by nine bilingual children with SLI in their L1 and L2. MLU = Mean length of utterance. Table modified from Fabbro & Marini (2010).

To sum up, the findings described in the last two sections provide evidence that both SLI and aphasia in bilingual children are associated with impairment in both languages. However, it is important to note that the patterns might differ between languages due to high inter-individual variability that might stem from factors such as age of acquisition and level of exposure to these languages.

#### **Bilingualism and Schizophrenia**

Language disturbance is one of the main clinical and diagnostic features in schizophrenia (Covington et al., 2005; Marini et al., 2008). Indeed, abnormalities in linguistic functions have been consistently reported in schizophrenic patients (e.g., Kuperberg, Deckersbach, Holt, Goff, & West, 2007). Although normal at the levels of segmental phonology (Chaika, 1974) and morphological organization (Chaika, 1990), the speech of persons with schizophrenia is characterized by flattened intonation (Cutting, 1985), word-finding difficulties (McKenna, 1994), and reduced syntactic complexity (Thomas, King, Fraser, & Kendell, 1990). Furthermore, their speech is usually reported as disordered, filled with irrelevant pieces of information and derailments (Andreasen, 1979). Such erratic discourse may be linked to the inability to use pragmatic rules and/or to general cognitive deficits involving factors such as attention (Neuchterlein, Asarnow, & Subotnik, 2002), and/or action planning, ordering, and sequencing (Docherty, Strauss, Dinzeo, & St-Hilaire, 2006), which are crucial for efficient discourse processing. Schizophrenic patients may display positive and/or negative symptoms. Positive symptoms include delusions, conceptual disorganization, and hallucinations, whereas negative symptoms include blunted affect, apathy, and anhedonia.

Polyglot individuals with schizophrenia may display differential performance in their languages. For example, some may become less fluent in one language and keep normal fluency in the other(s) (Hughes, 1981). The selective impairment can affect also grammatical processing, as cases of selective ungrammaticality in one language with spared grammatical competence in the other(s) have also been reported (Javier, 1989; Matulis, 1977). Furthermore, psychotic symptoms may be expressed differentially across languages in bilingual speakers (Schoeman, Chiliza, Emsley, & Southwood, 2008; for a thorough review on this argument see Paradis, 2008).

Perhaps the very first report of communicative behavior in a psychotic bilingual individual was provided by Bruce (1895, reported in Paradis, 2008). Interestingly, the patient showed symptoms that in the aphasia literature would be described in terms of *alternating antagonism*. When he manifested positive symptoms (e.g., he was restless and talkative), he was not able to use Welsh (L1) but could talk and understand English (L2). In contrast, only his L1 was accessible to him when he displayed negative symptoms (e.g., he was apathetic, unresponsive, and suspicious). Most interestingly, when in the English stage, he could clearly remember things he had noticed in previous English periods, but his memory was a blank to anything that occurred during the Welsh stage.

Similar findings have been reported in other studies. For example, in a large study of 30 highly proficient, fluent bilingual schizophrenic individuals, Hemphill (1971) reported that, during the clinical interview, when engaged in L2, the patients looked quite normal and did not show psychotic symptoms that, however, lit up again when they were using their first language. Recently, Schoemann et al. (2008) reported the case of a 27-year-old bilingual who knew Afrikaans (L1) and English (L2). He learned English at 13 years of age. Interestingly, while speaking in L2, he did not show relevant symptoms and his language was described as calm and coherent. However, when using L1 he showed several psychotic symptoms, including production of neologisms, tangential speech, derailments, and auditory hallucinations. As for auditory hallucinations, it makes sense to ask in which language a bilingual individual may "hear voices." Unfortunately, also in this case data are far from clear. Some studies report that auditory hallucinations in these individuals occur only in the L1 irrespective of which language the patients habitually use (e.g., Hemphill, 1971). Other studies show the opposite pattern, with auditory hallucinations only in the patients' L2 (e.g., Malo Ocejo, Medrano Albeniz, & Uriarte Uriarte, 1991) and, in some cases, even in more than one language (e.g., de Zulueta et al., 2001).

Overall, the few studies focusing on language processing and selection in bilingual speakers with schizophrenia seem to support the notion that the languages known by an individual are processed in different neural networks that can be differentially impaired. Furthermore, the available evidence suggests that psychotic symptoms are more severe in L1 compared to L2 and this may be explained in terms of differential emotional valence across the languages mastered by a given individual, with emotionally richer structure for L1 than L2 that makes the first language more prone to express psychiatric symptoms (Schoeman et al., 2008).

#### **Bilingualism and Progressive Neurodegenerative Disorders**

The language deficits displayed by patients with different types of progressive neurodegenerative disorders, e.g., Alzheimer's, Huntington's, or Parkinson's disease, may offer a strong support to the notion that the explicit and implicit memory systems are differentially involved in the different levels of language processing in L1 or L2.

Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by a cognitive deterioration which affects daily-life activities to complete dependency. At the neuropathological level, AD is characterized by neural atrophy due to loss of neurons and synapses in the cerebral cortex and subcortical structures and by the presence of plaques in the brain. In the initial stage of the illness, semantic memory is mostly affected, which is reflected in the language disturbances displayed by AD patients. Indeed, they show mainly semantic difficulties, while phonology and syntax remain relatively unaffected until the advanced stages of deterioration (Bayles, Tomoeda, & Trosset, 1993). For example, AD patients are particularly impaired in retrieving and producing words based on semantic rather than phonemic rules, i.e., in semantic versus phonological verbal fluency tests (Salvatierra, Rosselli, Acevedo, & Duara, 2007).

The semantic deficits may be apparent in all languages mastered by bilingual AD patients (De Picciotto & Friedland, 2001; Salvatierra et al., 2007) or may differentially affect the different languages. For example, Mendez et al. (2004) described two bilingual patients with semantic dementia who were able to understand words in one language but not in the other, suggesting that memory access from L1 and L2 words is segregated and may be differentially affected (see also Paradis, 2008). Pragmatic deficits are also present in AD patients, with difficulties in selecting the language appropriate to the interlocutor and with mixing of L1 and L2 (Frieddland & Miller, 1999). Crucially, inappropriate language selection seems to affect mainly the less proficient language and language mixing seems to consist mostly of intrusion of L1 utterances into L2 conversations (Frieddland & Miller, 1999). This, further, confirms that in bilinguals, L2 is mainly represented by explicit, semantic memory systems, which are more affected by AD (Paradis, 2008).

On the other hand, Huntington's (HD) and Parkinson's diseases (PD) are degenerative disorders mainly affecting the basal ganglia. Cases of patients suffering from Parkinson's disease have been described in which explicit memory or working memory were intact, whereas their ability to learn procedures had been damaged (Saint-Cyr, Taylor, & Lang, 1988). Both HD and PD patients present with a simplification of their language production at the grammatical level as well as deficits in syntactic comprehension (Murray, 2000).

While, to the best of our knowledge, no study has so far compared the deficits in L1 and L2 in bilingual HD patients, a recent study (Zanini et al., 2004) showed that

patients with PD are especially impaired in syntactic processing of L1 as compared to L2. In line with the declarative/procedural model (Paradis, 1994, 2004; Ullman, 2001, 2004), the study showed that patients with PD are especially impaired in accessing implicit, procedural grammatical knowledge of L1. In a similar vein, a single case study (Yazawa, Kawasaki, & Ohi, 2003) reported a bilingual Japanese– English PD patient presenting a more severe micrographia in writing in his native language, which was likely more automatic, than in English writing. This evidence in PD suggest a greater involvement of the basal ganglia in the acquisition and further processing of L1, and in particular of L1 grammatical knowledge, thus further corroborating the view of a major involvement of procedural memory in representing L1 grammar (Paradis, 2004).

#### The Assessment of Linguistic Impairment in Bilingual Individuals

One major problem in dealing with the results from the investigations outlined so far lies in the fact that accurate descriptions of the symptoms have not always been provided and, most of all, the described performance of the patients is not always accurate from an interlinguistic point of view. Indeed, a systematic assessment of all the languages known by an aphasic patient is an essential prerequisite for both clinical procedures (diagnosis, rehabilitation program, assessment of progress in recovery, etc.) and neurolinguistic research on bilingualism (Fabbro, 2001a). For this purpose, a useful tool, developed by Michel Paradis and associates (Paradis & Libben, 1987), is the Bilingual Aphasia Test (BAT). It is an articulated battery of linguistic tests formed by three main parts.

Part A is dedicated to the evaluation of the patient's bilingual history, such as how many (and what) languages did he or she speak in different contexts (at home, at work, with friends, etc.) and how did he or she acquire them (at what age, where, etc.). This part is extremely helpful in that it allows drawing a complete picture of the patient's bilingual history. This helps taking into account at least some of the factors that we have seen playing a major role in the development and functioning of the bilingual competence.

Part B has been designed to provide information about the patient's skills in different linguistic domains (e.g., phonology, morphology, syntax, semantics, discourse, etc.) and across all the languages he or she mastered before the insult. This means that a given patient knowing, say, Italian and English will be administered Part A only once, while Part B will involve a distinct assessment for each of his two languages. This allows getting comparable scores across the languages previously mastered by the patient. The third section of the BAT, Part C, is dedicated to the assessment of translation skills and interference detection in each language pair. The BAT is currently available in 65 languages (Part B) and 160 language pairs (Part C).

#### Conclusion

In this chapter, we have outlined in very general terms some of the main issues regarding the clinical neurolinguistics of bilingualism. As we have shown, this is a widespread phenomenon. Furthermore, disorders affecting bilingual competence may be found in both children and adults with different etiologies.

Neuroimaging studies have shown that the languages mastered by bilingual speakers have a complex neurofunctional organization based on the interconnection between shared and distinct neural circuits. Furthermore, there is consistent evidence suggesting that the exact pattern of such organization depends on several factors such as age of acquisition, proficiency level, modality of acquisition, and level of exposure.

When we turn to bilingual patients, we discover that much must still be done to disentangle the problem of assessment and rehabilitation in bilingual speakers. In this chapter, it has been shown that bilingual patients with different etiologies and ages may present differential patterns of impairment and/or recovery of their languages. This applies to adult aphasia, childhood aphasia, specific language impairment, neuropsychiatric, and neurodegenerative disorders. A reflection on the linguistic characteristics shown by these patients leads to some important clinical implications. First, a systematic assessment of linguistic disorders in all of the patients' languages is essential. This applies to all bilingual patients and becomes particularly important in the case of schizophrenic bilingual patients who may show psychotic symptoms only when using one of their languages and not while using the other(s). This observation leads to the conclusion that clinicians may miss the psychotic symptoms when interviewing in one language only. Furthermore, the possibility of a positive effect of language switching on the regulation of psychotic symptoms in these individuals remains open. Second, the assessment of the patients' linguistic abilities before and after rehabilitation with a test equivalent in both languages may help devise adequate rehabilitation protocols and control for their success or failure. Indeed, one major problem in dealing with the research published on bilingual patients lies in the fact that an accurate description of the symptoms has not always been provided and, most of all, the performance of the patients has not always been accurately described from an interlinguistic point of view. Indeed, a systematic assessment of all the languages known by an aphasic patient is an essential prerequisite for both clinical procedures (diagnosis, rehabilitation program, assessment of progress in recovery, etc.) and neurolinguistic research. For this purpose, a useful tool is the BAT (Paradis & Libben, 1987). A final issue concerns the choice of the language to use in rehabilitation. Should we rehabilitate bilingual individuals in only one language or should we consider also the other language(s)? Does rehabilitation in one language also have beneficial effects on the untreated languages? Unfortunately, research on language rehabilitation in bilingual patients is still at an early stage, with many studies selectively analyzing individual cases and lacking a proper pre- and postrehabilitation assessment of language

disorders. Therefore, to date it is not possible to provide a clear answer to these important questions. However, at present, the identification of the language(s) to rehabilitate may be based on two parameters: (1) a systematic assessment of the patient's linguistic deficits through the BAT in the languages the patient mastered before the insult; and (2) an interview with the patient and his/her relatives in order to gather relevant pieces of information about the languages known by the patient, their contexts of use, and the like. This, of course, should be the target of further studies.

#### References

- Abutalebi, J., Cappa, S. F., & Perani, D. (2001). The bilingual brain as revealed by functional neuroimaging. *Bilingualism, Language and Cognition*, *4*, 179–190.
- Aglioti, S., Beltramello, A., Girardi, F., & Fabbro, F. (1996). Neurolinguistic and follow-up study of an unusual pattern of recovery from bilingual subcortical aphasia. *Brain*, *119*, 1551–1564.
- Aglioti, S., & Fabbro, F. (1993). Paradoxical selective recovery in a bilingual aphasic following subcortical lesions. *NeuroReport*, *4*, 1359–1362.
- Andreasen, N. C. (1979). Thought, language, and communication disorders: Clinical assessment, definition of terms, and assessment of their reliability. *Archives of General Psychiatry*, *36*, 1325–1330.
- Bayles, K. A., Tomoeda, C. K., & Trosset, W. M. (1993). Alzheimer's disease: Effects on language. *Developmental Neuropsychology*, *9*, 131–160.
- Berthier, M. L., Starkstein, S. E., Lylyk, P., & Leiguarda, R. (1990). Differential recovery of languages in a bilingual patient: A case study using selective amytal test. *Brain and Language*, *38*, 449–453.
- Bloomfield, L. (1933). Language. New York: Holt, Rinehart, and Winston.
- Bouquet, F., Tuvo, F., & Paci, M. (1981). Afasia traumatica in un bambino bilingue nel 5° anno di vita. *Neuropsichiatria Infantile*, 236, 159–169.
- Bruce, L. C. (1895). Notes of a case of dual brain action. Brain, 18, 54-65.
- Caplan, D. (1992). Language: Structure, processing, and disorders. Cambridge, MA: MIT Press.
- Cappa, S. F. (1998). Spontaneous recovery from aphasia. In B. Stemmer & H. A. Whitaker (Eds.), *Handbook of neurolinguistics* (pp. 536–547). San Diego: Academic Press.
- Chaika, R. (1974). A linguist look at "schizophrenic" language. Brain and Language, 1, 257–276.
- Chaika, R. (1990). Understanding psychotic speech: Beyond Freud and Chomsky. Springfield, IL: Charles C. Thomas.
- Chilosi, A. M., & Cipriani, P. (1995). *Test di comprensione grammaticale per i bambini*. Pisa, Italy: Edizioni Del Cerro.
- Chomsky, N. (1995). The minimalist program. Cambridge, MA: MIT Press.
- Covington, M. A., He, C., Brown, C., Naçi, L., McClain, J. T., Sirmon Fjordbak, B., et al. (2005). Schizophrenia and the structure of language: The linguist view. *Schizophrenia Research*, *77*, 85–98.
- Cutting, J. (1985). The psychology of schizophrenia. Edinburgh: Churchill Livingstone.
- De Agostini, M., Metz-Lutz, M. N., Van Hout, A., Chavance, M., Deloche, G., & Pavao-Martins, I. (1998). Batterie d'évaluation du langage oral de l'enfant aphasique:

Standardisation française (4–12 ans). Oral language evaluation battery of aphasic children: A French standardization. *Revue de Neuropsychologie*, *8*, 319–367.

- De Picciotto, D., & Friedland, D. (2001). Verbal fluency in elderly bilingual speakers: Normative data and preliminary application to Alzheimer's disease. *Folia Phoniatrica Logopaedica*, 53, 145–152.
- DeVreese, L. P., Motta, M., & Toschi, A. (1988). Compulsive and paradoxical translation behaviour in a case of presenile dementia of the Alzheimer type. *Journal of Neurolinguistics*, *3*, 233–259.
- De Zulueta, F. I. S. (1984). The implication of bilingualism in the study and treatment of psychiatric disorders: A review. *Psychological Medicine*, *14*, 541–557.
- Docherty, N. M., Strauss, M. E., Dinzeo, T. J., & St-Hilaire, A. (2006). The cognitive origins of specific types of schizophrenic speech disturbances. *American Journal of Psychiatry*, *163*, 2111–2118.
- Edwards, J. V. (2004). Foundations of bilingualism. In T. K. Bhatia and W. C. Ritchie (Eds.), *The handbook of bilingualism* (pp. 7–31). Malden, MA: Blackwell.
- Fabbro, F. (1999a). Neurolinguistica e Neuropsicologia dei disturbi specifici del linguaggio nel bambino. *Saggi di Neuropsicologia Infantile, Psicopedagogia, Riabilitazione, 24*, 11–23.
- Fabbro, F. (1999b). The neurolinguistics of bilingualism. Hove, UK: Psychology Press.
- Fabbro, F. (2000), Developmental language disorders in bilingual children. Saggi: Neuropsicologia Infantile, Psicopedagogia, Riabilitazione, 25, 49–55.
- Fabbro, F. (2001a). The bilingual brain: Bilingual aphasia. *Brain and Language*, 79, 201–210.
- Fabbro, F. (2001b). The Bilingual brain: Cerebral representation of languages. *Brain and Language*, 79, 211–222.
- Fabbro, F., & Marini, A. (2010). Diagnosi e valutazione dei disturbi del linguaggio in bambini bilingui. In S. Vicari, & M. C. Caselli (Eds.), *Neuropsicologia dello sviluppo* (pp. 119–132). Bologna: Il Mulino.
- Fabbro, F., & Paradis, M. (1995a). Acquired aphasia in a bilingual child. In M. Paradis (Ed.), *Aspects of bilingual aphasia*. (pp. 67–83). London: Pergamon Press.
- Fabbro, F., & Paradis, M. (1995b). Differential impairments in four multilingual patients with subcortical lesions. In M. Paradis (Ed.), *Aspects of bilingual aphasia* (pp. 139–176). Oxford: Pergamon Press.
- Fabbro, F., Skrap, M., & Aglioti, S. (2000). Pathological switching between languages following frontal lesion in a bilingual patient. *Journal of Neurology, Neurosurgery and Psychiatry*, 68, 650–652.
- Flege, J. E., & Fletcher, K. L. (1992). Talker and listener effects on degree of perceived foreign accent. *Journal of the Acoustical Society of America*, 91, 370–389.
- Frazier, L., & Fodor, J. D. (1978). The sausage machine: A new two-stage parsing model. *Cognition*, 6, 291–325.
- Friedland, D., & Miller, N. (1999). Language mixing in bilingual speakers with Alzheimer's dementia: A conversation analysis approach. *Aphasiology*, *13*, 427–444.
- Grosjean, F. (1989). Neurolinguists beware! The bilingual is not two monolinguals in one person. *Brain and Language*, *36*, 3–15.
- Grosjean, F. (1994). Individual bilingualism. In R. E. Asher (Ed.), *The Encyclopaedia of Language and Linguistics* (pp. 1656–1660). Oxford: Pergamon Press.
- Grosjean, F. (1999). Individual bilingualism. In B. Spolsky (Ed.), *Concise Encyclopaedia of Educational Linguistics* (pp. 284–290). London: Elsevier.

- Hemphill, R. E. (1971). Auditory hallucinations in polyglots. *South African Medical Journal*, 45, 1391–1394.
- Hughes, G. W. (1981). Neuropsychiatric aspects of bilingualism: A brief review. *British Journal of Psychiatry*, 139, 25–28.
- Javier, R. A. (1989). Linguistic considerations in the treatment of bilinguals. *Psychoanalytic Psychology*, *6*, 517–526.
- Johnson, J. S., & Newport, E. L. (1989). Critical period effects in second language learning: The influence of maturational state on the acquisition of English as a second language. *Cognitive Psychology*, *21*, 60–99.
- Kuperberg, G. R., Deckersbach, T., Holt, D. J., Goff, D., & West, W. C. (2007). Increased temporal and prefrontal activity in response to semantic associations in schizophrenia. *Archives of General Psychiatry*, 64, 138–151.
- Lebrun, Y., & Hasquin, J. (1971). Bilinguisme précoce et troubles du langage. In S. De Coster (Ed.), *Aspects sociologiques du plurilinguisme* (pp.60–75). Bruxelles-Paris: Asimav–Didier.
- Malo Ocejo, P., Medrano Albeniz, J., & Uriarte Uriarte, J. J. (1991). Alucinaciones auditivas en sujetos bilingües. *Archivos de Neurobiología*, 54, 15–19.
- Marini, A. (2008). Manuale di neurolinguistica. Rome: Carocci.
- Marini, A., Boewe, A., Caltagirone, C., & Carlomagno, S. (2005). Age-related differences in the production of textual descriptions. *Journal of Psycholinguistic Research*, 34, 439–463.
- Marini A., & Fabbro F. (2007). Psycholinguistic models of speech production in bilingualism and multilingualism. In A. Ardila & E. Ramos (Eds.), *Speech and language disorders in bilinguals*. (pp. 47–67). New York: Nova Science Publishers Inc.
- Marini, A., Spoletini, I., Rubino, I. A., Ciuffa, M., Banfi, G., Siracusano, A., et al. (2008). The language of schizophrenia: An analysis of micro- and macrolinguistic abilities and their neuropsychological correlates. *Schizophrenia Research*, *105*, 144–155.
- Marini, A., Tavano, A., & Fabbro, F. (2008). Assessment of linguistic abilities in Italian children with specific language impairment. *Neuropsychologia*, *46*, 2816–2823.
- Matulis, A. C. (1977). Schizophrenia: Experiment in teaching a new foreign language to inpatients as an analeptic ego aid. *Dynamische Psychiatrie*, 10, 459–472.
- McKenna, P. J. (1994). Schizophrenia and related syndromes. Oxford: Oxford University Press.
- Murray, L. L. (2000). Spoken language production in Huntington and Parkinson's diseases. *Journal of Speech, Language, and Hearing Research, 43*, 1350–1366.
- Neuchterlein, K. H., Asarnow, R. F., & Subotnik, K. L. (2002). The structure of schizotypy: Relationships between neurocognitive and personality disorder features in relatives of schizophrenic patients in the UCLA Family Study. *Schizophrenia Research*, 54, 121–130.
- Neville, H. J., Mills, D. L., & Lawson, D. S. (1992). Fractionating language: Different neural subsystems with different sensitive periods. *Cerebral Cortex*, *2*, 244–258.
- Nilipour, R., & Ashayeri, H. (1989). Alternating antagonism between two languages with successive recovery of a third in a trilingual aphasic patient. *Brain and Language*, *36*, 23–48.
- Ojemann, G. A. (1991). Cortical organization of language. *Journal of Neuroscience*, 11, 2281–2287.
- Paradis, M. (1983). Readings on aphasia in bilinguals and polyglots. Montreal: Didier.
- Paradis, M. (1994). Neurolinguistic aspects of implicit and explicit memory: implications for bilingualism and second language acquisition. In N. Ellis (Ed.), *Implicit and Explicit Language Learning* (pp. 393–419). London: Academic Press.

Paradis, M. (2004). A neurolinguistic theory of bilingualism. Amsterdam: John Benjamins.

- Paradis, M. (2008). Bilingualism and neuropsychiatric disorders. *Journal of Neurolinguistics*, 21, 199–230.
- Paradis, J., Crago, M., Genesee, F., & Rice, M. (2003). French–English bilingual children with SLI: How do they compare with their monolingual peers? *Journal of Speech, Language, and Hearing Research*, 46, 113–127.
- Paradis, M., & Goldblum, M. C. (1989). Selective crossed aphasia in a trilingual aphasic patient followed by reciprocal antagonism. *Brain and Language*, 36, 62–75.
- Paradis, M., Goldblum, M. C., & Abidi, R. (1982). Alternate antagonism with paradoxical translation behavior in two bilingual aphasic patients. *Brain and Language*, *15*, 55–69.
- Paradis, M., & Libben, G. (1987). The assessment of bilingual aphasia. Hillsdale, NJ: Erlbaum.
- Peal E., & Lambert, W. E. (1962). The relation of bilingualism to intelligence. Psychological Monographs, 76, 1–23.
- Perani, D., & Abutalebi, J. (2005). The neural basis of first and second language processing. *Current Opinion in Neurobiology*, 15, 202–206.
- Pitres, A. (1895). Etude sur l'aphasie chez les polyglottes. Revue de médecine, 15, 873-899.
- Ribot, T. (1881). Les maladies de la mémoire. Paris: G. Baillere.
- Saint-Cyr, J. A., Taylor, A. E., & Lang, A. E. (1988). Procedural learning and neostriatal dysfunction in man. *Brain*, *111*, 941–959.
- Salvatierra, J., Rosselli, M., Acevedo, A., & Duara, R. (2007). Verbal fluency in bilingual Spanish/English Alzheimer's disease patients. *American Journal of Alzheimer's Disease and Other Dementias*, 22, 190–201.
- Schoeman, R., Chiliza, B., Emsley, R., & Southwood, F. (2008). Bilingualism and psychosis: A case report. *Schizophrenia Research*, *103*, 333–335.
- Snow, C. (1987). Relevance of the notion of a critical period to language acquisition. In M.H. Bornstein (Ed.), *Sensitive periods in development: Interdisciplinary perspectives*.Hillsdale, NJ: Erlbaum.
- Thomas, P., King, K., Fraser, W. I., & Kendell, R. E. (1990). Linguistic performance in schizophrenia: A comparison of acute and chronic patients. *British Journal of Psychiatry*, 156, 204–210.
- Tucker, G. R. (1998). A global perspective on multilingualism and multilingual education. In J. Cenoz & F. Genesee (Eds.), *Beyond bilingualism: Multilingualism and multilingual education* (pp. 3–15). Clevedon, UK: Multilingual Matters.
- Ullman, M. T. (2001). The declarative/procedural model of lexicon and grammar. *Journal of Psycholinguistic Research*, 30, 37–69.
- Ullman, M. T. (2004). Contributions of memory circuits to language: The declarative/ procedural model. *Cognition*, 92, 231–270.
- Urgesi, C., & Fabbro, F. (2009). Neuropsychology of second language acquisition. In W. C. Ritchie & T. K. Bathia (Eds.), *The new handbook of second language acquisition* (pp. 357–376). Bingley, UK: Emerald Group Publishing.
- Vargha-Khadem, F., Isaacs, E., Watkins, K. E., & Mishkin, M. (2000). Ontogenetic specialization of hemispheric function. In J. M. Oxbury, C. E. Polkey, & M. Duchovny (Eds.), *Intractable focal epilepsy* (pp. 405–418). London: W. B. Saunders.
- Veyrac, G. J. (1931). A study of aphasia in polyglot subjects. In M. Paradis (Ed.), *Readings* on aphasia in bilinguals and polyglots (pp. 320–338). Montreal: Didier.
- Wartenburger, I., Heekeren, H. R., Abutalebi, J., Cappa, S. F., Villringer, A., & Perani, D. (2003). Early settings of grammatical processing in the bilingual brain. *Neuron*, 37, 159–170.

- Weber-Fox, C. M., & Neville, H. J. (1996). Maturational constraints on functional specializations for language processing: ERP and behavioral evidence in bilingual speakers. *Journal of Cognitive Neuroscience*, 8, 231–256.
- Yazawa, S., Kawasaki, S., & Ohi, T. (2003). Is there less micrographia in foreign language in Parkinson's disease? *Neurology*, *61*, 1817.
- Zanini, S., Tavano, A., Vorano, L., Schiavo, F., Gigli, G. L., Aglioti, S. M., et al. (2004). Greater syntactic impairments in native language in bilingual Parkinsonian patients. *Journal of Neurology, Neurosurgery and Psychiatry*, *75*, 1678–1681.