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Bilingualism and the Brain: Myth and Reality

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Abstract

Introduction: The description of bilingual aphasic patients goes back to the 19th century. Since then, the study of the relationship between bilingualism and the brain has questioned whether the neuroanatomical representation of two languages in the same brain is similar or different. The answer to this question has generated erroneous interpretations based on the results of the investigations carried out on this topic.

Development: The present paper will try to distinguish between myth and reality of the following statements: *a)* the neuroanatomical organization of language in bilinguals is different from that of monolinguals, and *b)* language is less lateralized in bilingual speakers.

Results: There is no reason to believe in the existence of qualitative differences in the cerebral organization of language between bilinguals and monolinguals. It is most likely that two languages are represented as different microanatomical subsystems in the same cerebral regions.

Conclusions: The differences are quantitative rather than qualitative, that is, the degree of participation of the different neurofunctional mechanisms involved in the use of language, such as metalinguistic knowledge and implicit linguistic competence.

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PALABRAS CLAVE

Neurolingüística;
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Representación del

Bilingüismo y cerebro: mito y realidad

Resumen

Introducción: La descripción de pacientes bilingües con afasia se remonta al siglo XIX. Desde entonces, el estudio de la relación bilingüismo-cerebro se ha preguntado si la representación neuroanatómica de las lenguas que habla una misma persona es similar o

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lenguaje;
Lateralización del
lenguaje

diferente. La respuesta a esta pregunta ha generado interpretaciones erróneas de los resultados obtenidos en las investigaciones realizadas al respecto.

Material y métodos: En el presente trabajo, se describirá lo que hay de mito y de realidad en las siguientes afirmaciones: a) la organización neuroanatómica del lenguaje en el bilingüe es diferente del monolingüe, y b) la lateralización del lenguaje en el bilingüe es menor.

Resultados: No hay motivos para creer que haya diferencias cualitativas en la organización cerebral del lenguaje en bilingües y monolingües. Lo más probable es que las lenguas que habla una misma persona estén representadas como subsistemas microanatómicos distintos en las mismas regiones cerebrales.

Conclusiones: Las diferencias serían más bien de tipo cuantitativo, es decir, con relación al grado de participación de los diferentes mecanismos neurofuncionales implicados en el uso del lenguaje, entre ellos el conocimiento metalingüístico y la competencia lingüística implícita.

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Introduction

Speaking two or more languages is a common feature for millions of people throughout the world.^{1,2} Bilingualism is an extremely common phenomenon today and has been in existence since humanity developed a spoken language.³ Despite these facts, bilingualism is a topic that still generates significant debate about the way in which two or more languages are represented in the brain. The aim of this study is to distinguish between myth and reality in the relationship between bilingualism and the brain. However, before moving on to the central topic of this work, we will briefly summarise the historical background of the study with aphasic bilingual patients.

Bilingualism and Aphasia: Historical Background

In 1895, Jean-Albert Pïtres published *Étude sur l'aphasie chez les polyglottes* (*Study of Aphasia in Polyglots*), the first monographic work ever written on the alteration of language in bilingual and polyglot aphasic patients.⁴ In his study, Pïtres reviewed the literature published so far on the subject and described 7 of his own clinical cases. He identified 3 patterns of language following a brain lesion: 1) both languages are recovered simultaneously at the same rate, 2) one language is never recovered, and 3) the second language begins to recover only once the first one has been recovered. At present, these patterns are known, respectively, as parallel, selective and successive. Based on the language recovery patterns described, Pïtres concluded that the language recovered first was the one the patient used more often before the lesion, regardless of whether or not it was the patient's mother tongue (this has come to be known as Pïtres' law). The studies published by Paradis^{5,6} on aphasia cases in bilinguals increase the number of ways in which a language can recover after brain injury. Other alterations described in these patients are errors associated with the mechanism that allows a person to choose which

language to use (code switching and code mixing), as well as disorders affecting the ability to translate from one language to another.⁷ These are presented in table 1.

Many factors have been suggested as the causes of non-parallel recovery patterns, including the following: the order of acquisition of the languages,⁸ the frequency of use before the injury,⁴ injury location and severity⁹ the emotional bond established with each language,¹⁰ the type of bilingualism,¹¹ the level of knowledge of each language¹² and the structural distance between the languages spoken.¹³ The problem is that none of these factors on their own can account for all the cases reported to date, or even the majority of them. Therefore, what makes one language recover faster or in a different way to another? This is where the legend begins.

Myth 1. The Brains of Bilinguals Are Different From the Brains of Monolinguals

The most logical explanation for the different degrees of affectation of each language following a cerebral lesion is that the languages spoken by one individual are associated with different cortical areas. It is a myth, therefore, to think that the cerebral representation of language in bilinguals is different from that in monolinguals. The results obtained from cortical electrical stimulation¹²⁻¹⁴ and the reported cases of non-parallel recovery^{15,16} are interpreted as evidence of different neuroanatomical structures for each language within the classic language areas.

In 1978, Ojemann and Whitaker¹⁴ used cortical electrical stimulation to study the performance of 2 bilingual patients in a naming task. The authors found brain areas, always within the left hemisphere (LH), that affected both languages in a similar way (Broca's area and the inferior parietal lobe) when stimulated. They also found regions that affected only one of them (the frontal lobe, Wernicke's area) when stimulated. In both cases, the representation of the second language (L2) was greater than the first (L1). These authors believed that even if two languages share

Table 1 Recovery patterns of language and related disorders**Language recovery patterns*

Parallel recovery pattern: two or more languages are equally affected and recovery occurs at the same time

Non-parallel recovery pattern: each language recovery occurs separately

Differential: involvement of each language differs in degree

Successive: one language does not begin to recover until the other is relatively restored

Selective: the patient does not recover the use of one or more of the languages spoken

Antagonistic: the two languages are not available simultaneously, that is, only one language is available at the beginning and, as the second improves, the first worsens

Alternating antagonistic: during alternating periods, the patient has access to one of the languages spoken

Differential aphasia: each language presents features that correspond to different types of aphasia. This is a controversial recovery pattern, since the difference is not in the nature of aphasic symptoms presented by each language, but in the degree of involvement of each of them.⁶

Selective aphasia: involvement of one of the languages, while the other remains relatively preserved

Disorders related to the use of a given language at will

Pathological code-mixing: improper combination of elements from two or more languages in the same word or sentence.

This interaction can occur in one or more linguistic fields (phonological, morphological, lexical, syntactic, semantic). An example would be the formation of words using the lexical rules of another language (lexeme in one language and suffix in the other). The result can lead to a mixture of incomprehensible words and expressions

Pathological alternation of codes

Pathological fixation on one of the languages that prevents the switch from one language to another

Uncontrollably and frequently alternating complete sentences in two or more languages when speaking

Disorders associated with the ability to translate from one language to another

Inability to translate in any direction of translation, that is, from the first language to the second and vice versa

Spontaneous translation. Compulsive need to translate everything said or told (regardless of the language of the interlocutor)

Translation without understanding. Patients do not understand verbal commands, but can translate to another language

Paradoxical translation. Ability to translate into a language that is inaccessible for spontaneous use, accompanied by an inability to translate the language that can be used at that time

*The different patterns of language recovery, code-switching/ mixing and translation disorders are not mutually exclusive, as they can be combined and/ or follow each other in time.

specific cortical areas, the least automated language depends on additional brain areas around the classic language areas. These results can also be interpreted as resulting from the degree of knowledge of each language.¹⁷ In other words, the cortical area that supports spoken language is inversely proportional to the degree of knowledge of each language. Consequently, the lesser the knowledge of a language, the greater the space it requires in the brain. Later studies have found the same results.^{12,18} The results derived from this research have been criticised for the type of language task used during cortical stimulation as well as for the drawbacks of the actual technique. Although the linguistic paradigm used (naming objects) has provided interesting data, the linguistic system cannot be reduced to this task alone.¹⁹ With regards to the technique itself,²⁰⁻²² firstly, the spatial definition of cortical stimulation lacks accuracy since it is difficult to stimulate the same area at different times and in different subjects. Secondly, the effects of stimulation in the areas where one of the languages is supposedly located are not consistent; in other words, stimulation of the same area sometimes had an effect on a language, while other times it did not. But even if that were the case, how could the alternating, antagonistic recovery pattern be explained?

Some cases of non-parallel recovery have also been used to defend the hypothesis of different neuroanatomical

structures for each language. Berthier et al¹⁵ described the case of a 25-year-old patient with an arteriovenous malformation located in the right hemisphere (RH). For this patient, Spanish was his mother tongue (L1) and English his second language (L2). To assess whether there was a different lateralisation for each language, each cerebral hemisphere was anaesthetised using the intracarotid sodium amytal test (Wada test). The temporary LH inactivation initially caused a general aphasia for both languages. The first language to be recovered after anaesthesia wore off was English (L2), followed by Spanish (L1). According to the study authors, all the languages of a bilingual subject are stored within the left perisylvian region, and while the L2 language is represented within the sylvian fissure, the L1 language is represented in more distal perisylvian areas. This distinction is based on the pattern followed by amytal when leaving the cerebral bloodstream; first the sylvian fissure and then the surrounding regions. However, these findings have also been questioned. According to Paradis,²³ rather than supporting the hypothesis of a different cortical representation for each language, the results show the exact opposite. The study authors attributed the differences observed to the degree of automatization of each language; that is, the less automated a language is, the larger the brain surface needed for its representation. In that case, why is it that the language that the patient used the least

(English) was dependent on the central region of the sylvian fissure and not on the peripheral structures?

In 1995, Gómez-Tortosa et al¹⁶ presented the case of a proficient bilingual woman who, after undergoing an operation to remove an arteriovenous malformation in the perisylvian cortex of the LH, showed a selective deficit of her mother tongue (Spanish) and not of her second language (English). This patient used mostly her L2 in her daily life. According to these authors, the selective affectation of one of the languages following surgery shows that each language has a different neuroanatomical substrate within the left perisylvian region. Gómez-Tortosa et al²⁴ later admitted that the affectation of the native language could also be explained by the physiological inhibition of one of the languages, as Paradis had pointed out in a previous article in which he discussed the case.²¹

Functional neuroimaging techniques can also be used to follow the brain activity of bilinguals. Tables 2 and 3 briefly describe some of the main studies conducted on healthy bilingual controls in production and verbal comprehension tasks, respectively. The main conclusion drawn from these studies is that the differences in the brain representation of two languages are minimal when the level of proficiency in both languages is high, at least on a macroscopic level. However, little knowledge or command of a language is associated with the activation of larger brain areas near classic language areas. The activation of additional brain areas is related to the different computational demands of each language.^{37,38} These differences are more pronounced when the L2 is weak, that is, in the early stages of acquisition or when the level of knowledge is low. According to Abutalebi,³⁸ the anatomical location of these differences may be of two types: 1) increased activity in the same regions or in areas close to the classic language areas, and 2) increased activity in additional brain regions associated with executive functions (e.g., left prefrontal cortex and basal ganglia). These findings are related to Ertl and Schafer's concept of cortical efficiency³⁹: the greater the skill displayed in a specific cognitive task, the more efficient the neural processing; that is, the smaller the amount of energy required to operate is.

This myth, however, has a second part: the existence of brain areas specific to bilinguals. In 1925, Pötzl⁹ described 2 cases of bilingual aphasics who had trouble switching from one language to another. In both cases, the brain lesion was located in the left supramarginal gyrus. From these data, Pötzl suggested that this brain region played a vital role in the choice of language to be used. This hypothesis was soon disproved in patients with lesions of the supramarginal gyrus who had no difficulty switching from one language to another, and also in other patients who did have difficulty switching despite not having posterior lesions.⁴⁰ Another region in the brain that has been linked to the neurological basis of the switching mechanism is the frontal lobe. As a result of a frontal lesion, one of the cases described by Stengel and Zelmanowicz⁴¹ not only had motor aphasia but also mixed languages when speaking and doing the applied naming task. In 2000, Fabbro et al⁴² described the case of a patient with a brain tumour located in the white matter adjacent to the left frontal cortex that later affected the anterior cingulate gyrus of the RH. The first symptom of the

tumour was a compulsive tendency to switch between the two languages spoken (Friulan and Italian), in the absence of other aphasic symptoms and with all the other cognitive functions that were assessed remaining intact. Other cases presenting pathological switching from one language to another have been reported as a result of transcranial magnetic stimulation in the dorsolateral prefrontal cortex of the LH⁴³ and during electrocortical stimulation of the left inferior frontal gyrus.⁴⁴ Damage to subcortical structures can also lead to dysfunction of the mechanisms involved in language selection. For example, after a lesion in LH basal ganglia (specifically, capsular-putaminal), patient EM lost the ability to express himself in his mother tongue but could still do so well in his second language.^{45,46} However, basal ganglia lesions have not only been associated with a pathological fixation on one of the languages, but also with uncontrolled switching between them. This was the case with patient AH of Abutalebi et al,⁴⁷ who due to a lesion in the white matter adjacent to the caudate nucleus, could not maintain a conversation in one particular language, even when specifically asked to. Although the patient was aware of this problem, she was unable to avoid it.

These findings may also be explained by a dysfunction of the dorsolateral prefrontal circuit described by Cummings.⁴⁸ This circuit is involved in the regulation and selection of information to be recovered, as well as in the inhibition of interferences. In the case of bilingualism, this circuit, especially the caudate head, may be related to the selection and inhibition of lexical representations of either language for their subsequent use.⁴⁷ Other neuronal structures involved in this selection/inhibition process are the parietal cortex (activates lexical representation of different possible responses) and the anterior cingulate (detects errors during processing of information).⁴⁹ Both structures are involved in switching from one task to another.

In reality, we do not know whether there is a specific neuroanatomical or neurofunctional structure that allows bilinguals to tune into one language or another. According to Paradis,⁵⁰ the mechanism for switching from one language to another can be either automatic or deliberate. When it is automatic, code-switching or code-mixing is related to the actual linguistic system (e.g., the word spoken is that with a lower activation threshold, and memory is consequently not subject to more conscious control). The neuroanatomical structures involved are the perisylvian cortex, basal ganglia and cerebellum. However, a deliberate switch from one language to another may depend on the same general neuropsychological mechanism of internal choice that enables us to speak or remain silent. We would, therefore, be talking about an ability common to all individuals, which allows us to react according to each situation. In this case, the decision to use a particular language is related to the participation of attention and executive functions, regulated by the prefrontal cortex and anterior cingulate.

Myth 2. Bilinguals Use Their Right Hemisphere More Than Monolinguals

The first author to suggest that the languages spoken by the same person had different lateralisations in the brain was

Table 2 Functional neuroimaging studies conducted in bilinguals while performing verbal production tasks

Study	Technique	Sample	Verbal task	Main result
Klein et al (1995) ²⁵	PET	12 late BL: L1 (Eng)/ L2 (Fr). Acquisition of L2: from age 5. Command of languages: high	Generation of words (rhymes and synonyms) and translation of words	Overlapping of brain areas activated in both languages. Greater activation of left putamen during the repetition of L2 words
Chee et al (1999) ²⁶	fMRI	L1 (Mnd)/ L2 (Eng); 2 groups: a) 15 subjects, L2 acquired before age 6; b) 9 subjects, L2 acquired from age 12	Completing words with a missing part, initial or final	The brain areas activated were the same despite the structural difference between the two languages and age of L2 acquisition
Klein et al (1999) ²⁷	PET	7 late BL: L1 (Ch)/ L2 (Eng). Acquisition of L2: adolescence	Generating verbs and repeating words	Similar brain activity for both languages, with no differences in the activation of subcortical structures
Hernández et al (2000) ²⁸	fMRI	6 early BL: L1 (Spa)/ L2 (Eng) Dominant language: English	Denomination	Similar brain activation for both languages
De Bleser et al (2003) ²⁹	PET	11 late BL: L1 (Du)/ L2 (Fr). L2 studied for 8 years, with performance between "good" and "very good" in a capacity test	Internal generation of the name of pictures presented successively. Types of stimulus: <i>cognates</i> ^a y <i>non-cognates</i> ^b	Few activation differences found between both languages, except for L2 non-cognates, which resulted in increased activation in left inferior frontal area and temporoparietal areas (associated with an increased effort in the recovery of L2 words)
Perani et al (2003) ³⁰	fMRI	11 early BL (Cat/ Spa) with equal command of each language and different degree of exposure to each (L2 exposure was less intense than in BL whose L1 was Catalan)	Verbal fluency with phonetic pattern	a) Reduced extent of brain activation associated with the language acquired in the first place, b) less intense exposure to L2, greater extent of brain regions activated in this language

BL: bilinguals; Cat: Catalan; Ch: Chinese; Du: Dutch; Eng: English; fMRI: functional MRI; Fr: French; L1: first language; L2: second language; Mnd: Mandarin; PET: positron emission tomography; Spa: Spanish.

^a Words with similar form and meaning in both languages.

^b Words with the same meaning, but phonological and orthographic differences.

Görlitzer von Mundy⁵¹ in 1959. This author described the case of a patient whose mother tongue was Slovenian. He started learning his L2, German, at the age of 30 when he enlisted in the Austrian infantry. The patient was illiterate when he joined the army, but he learned to read and write in German there. At the age of 94, he suffered a left-hemisphere stroke and could only speak Slovene (a language which he had not used in decades). According to the author, the language that he had acquired only orally was represented in both hemispheres, while the language he had learned to speak and write was lateralised in the LH. For this reason, the patient described could only speak Slovene. In 1978, Albert and Obler⁵² returned to this hypothesis and, from the bilingual aphasic cases that had been published until then, concluded that cases of non-

parallel recovery could be interpreted as the result of a more symmetrical neuroanatomical representation of the language between both hemispheres.

In 2007, Hull and Vaid⁵³ conducted a comprehensive meta-analysis of the results obtained in healthy bilingual subjects using classic techniques for studying brain lateralisation: tachistoscopia and dichotic listening. The authors concluded that in the studies analysed, brain lateralisation was strongly influenced by the age of acquisition of L2. In bilinguals who had acquired their L2 before the age of 6, the degree of participation of both hemispheres was much higher; for those who acquired L2 at a later age, the dominant hemisphere for both languages was the left one. Another conclusion reached was that in bilinguals who learned their L2 after the age of 6, LH

Table 3 Functional neuroimaging studies in bilinguals while performing verbal comprehension tasks

Study	Technique	Sample	Verbal task	Main result
Perani et al (1998) ³¹	PET	2 groups of BL, high command of L2: 9 late BL (from age 10 years), L1 (It)/ L2 (Eng); 12 early BL (before age 4 years) (Cat/ Spa)	Listening to stories in L1 and L2	Activated areas were the same for both L1 and L2. No differences with regard to age of L2 acquisition
Chee et al (1999) ³²	fMRI	9 early BL, high command of L2: L1 (Mnd)/ L2 (Eng). L2 acquisition: before age 6	Comprehension of sentences	Despite the differences between studied languages (spelling, phonological and structural), cerebral activation was similar
Chee et al (2001) ³³	fMRI	2 groups: 10 early BL (before 5 years), L1 (Eng)/ L2 (Mnd); 9 late BL (after age 12), L1 (Mnd)/ L2 (Eng)	Semantic judgement (select the most similar item to the sample stimulus)	Less cerebral activity in prefrontal and parietal regions of the dominating language or L1 of each group
Pillai et al (2003) ³⁴	fMRI	8 late BL (from 10 years): L1 (Spa)/ L2 (Eng). Average knowledge of L2	Semantic decision (associate verb with name) and phonological (identify words which rhyme)	Specially for the semantic task, L2 activate a greater extension of left prefrontal cortex
Tatsuno et al (2005) ³⁵	fMRI	L1 (Jap)/ L2 (Eng); 2 groups: 14 late BL (age, 13 years), low L2 level; 15 late BL (age, 19 years), high L2 command	Identification of verbs in past tense	Less activity in left inferior frontal gyrus in the group with greater L2 dominance
Yokoama et al (2006) ³⁶	fMRI	36 late BL (from age 8-9 years), L1 (Jap)/ L2 (Eng)	Processing of sentences in passive and active (decide whether they are semantically correct or not)	Similar activation for both languages, except in the processing of complex sentences in L2 (activation in additional cerebral regions)

BL: bilinguals; Cat: Catalan; Eng: English; fMRI: functional MRI; Fr: French; It: Italian; Jap: Japanese; L1: first language; L2: second language; Mnd: Mandarin; PET: positron emission tomography; Spa: Spanish.

involvement was greater when the subject had less L2 knowledge. Functional neuroimaging studies have also found increased activation of some RH regions during verbal comprehension tasks.^{54,55}

But is it really true that the degree of RH involvement in bilinguals is greater? Michel Paradis⁵⁶⁻⁶⁰ widely criticises the results of these studies and suggests that the role of the RH is no different in monolinguals than in bilinguals. His criticism is directed at three areas: the inconsistency of the results obtained, the role assigned to the RH and the conclusions drawn from the results obtained. Firstly, the results are contradictory and inconsistent: whereas for one same studied variable, one team of researchers finds evidence of differential lateralisation, another team does not. Furthermore, the lateralisation differences recorded so far have been attributed to very specific subgroups of bilinguals in very particular conditions (e.g., early or late bilinguals, bilinguals with their eyes closed). Not only is it impossible to apply these results to the entire bilingual population, they cannot be applied to one subgroup either.

Consequently, if the experimental paradigm used can be considered as a variable that can account for the contradictory results, then the validity of these paradigms (dichotic listening, tachistoscopia) should be assessed. The authors of these studies, however, explain this inconsistency in the results in terms of methodological differences or of the nature of the tasks given, and not of their validity.

Secondly, insofar as RH lesions result in disorders of pragmatic or communicative abilities, these do not necessarily have to be different in bilinguals and monolinguals. Greater RH involvement would not be related to a different lateralisation of the language system. Representation and processing of linguistic and grammatical competence cannot be forced in the RH if it does not naturally belong there (except in the case of an early LH lesion). This is why, according to Paradis, greater RH use should not be interpreted as a direct effect of bilingualism, but as the use of compensatory strategies based on pragmatic tracks when the degree of knowledge of one language is lower (a situation that occurs when the L2 is learned or acquired).

Finally, another problem with these studies is the fact that inferences are made from findings based on assumptions that we know to be invalid. There is no use explaining the contradictions found in the various investigations by subtle variations in the method used, if the parameter of each procedure and the conditions under which a different language lateralisation occurs are not specified. It is also necessary to justify the reasons why a language variable has a different cerebral representation in bilinguals and monolinguals or, in any case, how a procedure that uses stimuli presumably related to LH linguistic competence leads to greater RH use. In addition, it is impossible to attribute full language lateralisation to the RH just from the results of a single language task. According to Paradis, the results reported so far could be related to other still undefined aspects or simply to factors unrelated to what is being measured. Furthermore, the tasks used do not serve to indicate cerebral lateralisation; they instead illustrate better hearing in one ear or better vision in one of the visual hemifields, respectively.

Paradis⁵⁷⁻⁵⁹ likens the search for evidence that supports a greater RH involvement to the search for the “Loch Ness monster”. As with the mythical monster, differences in language lateralisation have been observed under certain conditions, but it has proved impossible to replicate them on many other occasions. Even if something had been observed, it is possible that it had nothing to do with what was in fact being sought. Continuing in this line of research is believing that this phenomenon actually exists and that, moreover, it can be proved. However, this is not the case. The fact that while some results do support this hypothesis others do not shows the need to review the validity of the paradigms used, and to justify the reasons why each procedure produces one result or another. If it were true that the RH plays an additional role in bilinguals, cases of crossed aphasia in these subjects would be more common than in monolinguals, but clinical^{61,62} and review⁶³ studies suggest otherwise.

From Myth to Reality...

A neuroanatomical approach is not enough to explain the different patterns of language recovery described. Alternatives to this view are found in the neurophysiological approach and the role that declarative and procedural memory systems play in language representation and processing. However, these hypotheses still do not explain all the cases of non-parallel recovery.

As early as 1895, Pitzes⁴ believed that a neurophysiological approach should be used to study cerebral language organisation in bilinguals and polyglots. According to Pitzes, there were two reasons why the language that was used most before the lesion was the first to recover: firstly, due to stronger neuronal connections, and, secondly, due to the temporary inhibition of the other language. It would, therefore, be related to the functional weakening of one of the language systems, rather than to its physical destruction. This author called this disorder “inertia of the cortical language centres” and it is characterised by the temporary inhibition of the motor or sensory images needed to produce

or understand words. This functional inertia would have a greater impact on the language with the weakest neuronal associations. The arguments supporting this idea were based on the fact that, after some time, the language that had not initially recovered would once again be available. Paradis⁶⁴ took that idea up again and suggested that the weakening of a linguistic system cannot be explained only by an increase in the inhibitory processes, it can also result from an increase in the activation threshold needed for the self-activation of the elements of a language (the threshold activation theory). In aphasic bilinguals, the pathological process raises the activation threshold of one of the languages or parts of it (e.g., items used less frequently), making it less accessible for use. This hypothesis is consistent with that of increased inhibitory mechanisms and, in theory, explains all the recovery patterns described in literature. Nevertheless, the mechanisms behind the activation/deactivation of one language system but not of the other remain unknown.

According to Paradis,⁶⁴⁻⁶⁶ declarative and procedural memory systems play a different role in language learning, representation and use. This model argues that the declarative memory system is associated with lexicon and grammar learning at school (metalinguistic knowledge). The procedural memory system, however, is needed for the automatic application of these grammar rules during speech (implicit linguistic competence) (table 4). In bilinguals, learning and use of L1 grammar depend on procedural memory, while memorization and use of vocabulary depend on declarative memory. With L2, different participation of one system or another would depend on the age of acquisition. While the ability to expand vocabulary is not lost over the years, L2 grammar becomes more dependent on the declarative memory system, since it must be memorized and applied consciously while talking. That is, the weaker a person's L2 language skills, the more he/she will depend on metalinguistic knowledge and on the pragmatic aspects of the language to control producing and interpreting the meaning of a sentence.

From the above and taking into account that implicit linguistic competence and metalinguistic knowledge are independent systems supported by different brain structures, Paradis makes the following working hypotheses: 1) lesions in temporary structures and LH association areas will be linked with greater difficulty using the language that is least used (L2) or was learned at an adult age, and 2) lesions that damage LH frontobasal circuits will mainly affect the use of the language that is used most or was acquired first (L1) and L2 if the speaker learned it at the same time as the mother tongue and has a good command of it. For example, in aphasic patients, a better performance in L2 may be due to the use of a compensatory strategy based on metalinguistic knowledge of the language. In this case, the implicit linguistic competence is affected for both languages, but the metalinguistic knowledge is available for the language that was learned in a more formal context or at a lower level. It is also important to distinguish between the spontaneous recovery of a language (disinhibition of the procedural competence) and the ability to control verbal production through metalinguistic knowledge (replacement of the automatic use of a language for a more controlled

Table 4 Characteristics of metalinguistic knowledge and implicit linguistic competence

	Metalinguistic knowledge	Implicit linguistic competence
Definition	Knowledge of which the individual is aware and can express orally. E.g., vocabulary, knowledge of spelling and grammar rules learned in school	Knowledge deduced from the performance of a subject, that is, their behaviour is compatible with the application of a particular rule of which they ignore the computational processes that generate it. E.g., production in the order and correct inflection of a word sequence
Learning	Conscious	Incidental acquisition.
Storage	Explicit (subject can evoke learned elements at will)	Implicit (information acquired is not available in the conscious sphere)
Use	Under the subject's control	Automatic (improves with practice)
Memory system	Declarative memory (encyclopaedic knowledge)	Procedural memory (learning and maintaining motor and cognitive skills whose execution is automatic)
Neuroanatomical representation	Medial temporal structures and posterior association areas of both hemispheres	Frontobasal and cerebellum structures, inferior parietal region of the left hemisphere
L1	Vocabulary	Grammar
L2: high command	Vocabulary	Grammar
L2: poor command	Vocabulary + Grammar	—
L1: first language; L2: second language.		

use). Reports published in scientific literature show evidence that supports this dissociation. The case described by Ku et al⁶⁷ in 1996 is proof of a worse performance of the second language learned, following a left temporal lesion. In turn, lesions in frontal and basal ganglia structures have shown the contrary, that is, greater impairment of the L1.^{45,68}

Conclusions

The brain of a bilingual or polyglot does not have a separate area for each language, nor unique regions. Similarly, the cognitive functions traditionally associated with each cerebral hemisphere are still the same for all individuals. Believing the contrary would be tantamount to thinking that to learn to type on a keyboard, we would need to grow new fingers on our hands. What actually happens is that to learn a new task, our existing joints and muscles work together in different ways. Obviously, the more a task is repeated, the better the final execution. Something similar happens in the brain when a second language is acquired or learned. In these cases, it is likely that the languages spoken by one person are represented as different microanatomical subsystems within the same cerebral regions.⁶⁴ These linguistic subsystems are independent from each other, both in their structure and function. That is why, after a brain lesion, they may be affected differently. Paradis⁶⁴ argues that there is no reason to believe that there are any qualitative differences in the processing of two or more languages, either between monolinguals and bilinguals, or among different kinds of bilinguals. The differences are more of a quantitative nature, that is, with respect to the degree of use of different brain

mechanisms involved in the processing of verbal communication (metalinguistic knowledge, linguistic and pragmatic competence and motivation).

Finally, according to Fabbro,⁷ the relationship between bilingualism and the brain raises the need to identify and distinguish the different structures involved in the cerebral representation of language, including biochemical, neuroanatomical, neurofunctional, psychological and linguistic aspects. The representation of two or more languages in the brain may differ in some of these structures, but not in all. Once we know each of their functions, we will be in a better position to understand the linguistic problems related to bilinguals and polyglots.⁶⁹ For this reason, an activity as complicated as "language" cannot be explained solely through a neuroanatomical system.

Conflict of interests

The authors declare no conflict of interests.

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References

1. En: Gordon RG, editors. *Ethnologue: Languages of the World*. 15.^a ed. Dallas: SIL International; 2005.

2. Leclerc J. Le multilinguisme: un phénomène universel, 2002 [citado 21 de mayo 2008]. Disponible en: http://www.tlfiq.ulaval.ca/axl/Languages/3cohabitation_phenom-universel.htm.
3. Grosjean F. Life with two languages. An introduction to bilingualism. Cambridge: Harvard University Press; 1982.
4. Pitres JA. Etude sur l'aphasie chez les polyglottes. *Rev Méd.* 1895;15:873-99. Traducido en: Paradis M, editor. Readings on aphasia in bilinguals and polyglots. Montreal: Marcel-Didier; 1983. p. 26-49.
5. Paradis M. Bilingualism and aphasia. *Studies in Neurolinguistics*. 3. New York: Academic Press; 1977. p. 65-121.
6. Paradis M. Bilingual and polyglot aphasia. *Handbook of Neuropsychology*. 2. New York: Elsevier Science Publishers; 1989. P. 117-40.
7. Fabbro F. The neurolinguistics of bilingualism. Hove: Psychology Press; 1999.
8. Ribot T. Les maladies de la mémoire. 2.a ed. Paris: G. Baillière; 1881. Traducido en: Rubio R. Las enfermedades de la memoria. Madrid: Daniel Jorro; 1927.
9. Pötzl O. Über die parietal bedingte Aphasie und ihren Einfluss auf das Sprechen mehrerer Sprachen. *Z Gesamte Neurol Psychiatr.* 1925;96:100-24. Traducido en: Paradis M, editor. Readings on aphasia in bilinguals and polyglots. Montreal: Marcel-Didier; 1983. p. 176-98.
10. Minkowski M. Sur un cas d'aphasie chez un polyglotte. *Rev Neurol (Paris)*. 1928;49:361-6. Traducido en: Paradis M, editor. Readings on aphasia in bilinguals and polyglots. Montreal: Marcel-Didier; 1983. p. 274-9.
11. Lambert WE, Fillenbaum S. A pilot study of aphasia among bilinguals. *Can J Psychol.* 1959;13:28-34.
12. Raport RL, Tan CT, Whitaker HA. Language function and dysfunction among Chinese and English speaking polyglots: Cortical stimulation, Wada testing, and clinical studies. *Brain Lang.* 1983;18:342-66.
13. Ovcharova P, Raichev R, Geleva T. Afaziia u Poliglotti. *Nevrol Psikiatr Nevrokhir.* 1968;7:183-90. Traducido en: Paradis M, editor. Readings on aphasia in bilinguals and polyglots. Montreal: Marcel-Didier; 1983. p. 744-52.
14. Ojemann GA, Whitaker HA. The bilingual brain. *Arch Neurol.* 1978;35:409-12.
15. Berthier ML, Starkstein SE, Lylyk P, Leiguarda R. Differential recovery of languages in a bilingual patient: a case study using selective Amytal test. *Brain Lang.* 1990;38:449-53.
16. Gomez-Tortosa E, Martin EM, Gaviria M, Charbel F, Ausman JI. Selective deficit of one language in a bilingual patient following surgery in the left perisylvian area. *Brain Lang.* 1995;48:320-5.
17. Ojemann GA. Brain organization for language from the perspective of electrical stimulation mapping. *Behav Brain Sci.* 1983;6:189-230.
18. Lucas TH, McKhann GM, Ojemann GA. Functional separation of languages in the bilingual brain. *J Neurosurg.* 2004;101:449-57.
19. Zatorre RJ. On the representation of multiple languages in the brain: old problems and new directions. *Brain Lang.* 1989;36:127-47.
20. Paradis M. Multilingualism and Aphasia. En: Blanken G, Dittmann J, Grimm H, Marshall JC, Wallesch CW, editors. *Linguistic disorders and pathologies: an international handbook*. Berlin: Walter de Gruyter; 1993. P. 278-88.
21. Paradis M. Selective deficit in one language is not a demonstration of different anatomical representation: Comments on Gomez-Tortosa et al (1995). *Brain Lang.* 1996;54:170-3.
22. Fabbro F. The bilingual brain: Cerebral representation of languages. *Brain Lang.* 2001;79:211-22.
23. Paradis M. Differential recovery of languages in a bilingual patient following selective amytal injection: a comment on Berthier et al (1990). *Brain Lang.* 1990;39:469-70.
24. Gomez-Tortosa E, Martin EM, Gaviria M, Charbel F, Ausman JI. Selective deficit of one language in a bilingual patient: replies to Paradis and Hines. *Brain Lang.* 1996;54:174-5.
25. Klein D, Milner B, Zatorre RJ, Meyer E, Evans AC. The neural substrates underlying word generation: a bilingual functional-imaging study. *Proc Natl Acad Sci U S A.* 1995;92:2889-903.
26. Chee MWL, Tan EWL, Thiel T. Mandarin and English single word processing studied with functional magnetic resonance imaging. *J Neurosci.* 1999;19:3050-6.
27. Klein D, Milner B, Zatorre RJ, Zhao V, Nikelski J. Cerebral organization in bilinguals: a PET study of Chinese-English verb generation. *Neuroreport.* 1999;10:2841-6.
28. Hernandez AE, Martinez A, Kohnert K. In search of the language switch: an fMRI study of picture naming in Spanish-English bilinguals. *Brain Lang.* 2000;73:421-31.
29. De Bleser R, Dupont P, Postler J, Bormans G, Speelman D, Mortelmans L, et al. The organisation of the bilingual lexicon: a PET study. *J Neurolinguistics.* 2003;16:439-56.
30. Perani D, Abutalebi J, Paulesu E, Brambati S, Scifo P, Cappa SF, et al. The role of age of acquisition and language usage in early, high-proficient bilinguals: an fMRI study during verbal fluency. *Hum Brain Mapp.* 2003;19:170-82.
31. Perani D, Paulesu E, Galles NS, Dupoux E, Dehaene S, Bettinardi V, et al. The bilingual brain. Proficiency and age of acquisition of the second language. *Brain.* 1998;121:1841-52.
32. Chee MW, Caplan D, Soon CS, Sriram N, Tan EW, Thiel T, et al. Processing of visually presented sentences in Mandarin and English studied with fMRI. *Neuron.* 1999;23:127-37.
33. Chee MW, Hon N, Lee HL, Soon CS. Relative language proficiency modulates BOLD signal change when bilinguals perform semantic judgments. *Blood oxygen level dependent. Neuroimage.* 2001;13:1155-63.
34. Pillai JJ, Araque JM, Allison JD, Sethuraman S, Loring DW, Thiruvaiyaru D, et al. Functional MRI study of semantic and phonological language processing in bilingual subjects: preliminary findings. *Neuroimage.* 2003;19:565-76.
35. Tatsuno Y, Sakai KL. Language-related activations in the left prefrontal regions are differentially modulated by age, proficiency, and task demands. *J Neurosci.* 2005;25:1637-44.
36. Yokoyama S, Okamoto H, Miyamoto T, Yoshimoto K, Kim J, Iwata K, et al. Cortical activation in the processing of passive sentences in L1 and L2: an fMRI study. *Neuroimage.* 2006;30:570-9.
37. Perani D, Abutalebi J. The neural basis of first and second language processing. *Curr Opin Neurobiol.* 2005;15:202-6.
38. Abutalebi J. Neural aspects of second language representation and language control. *Acta Psychol (Amst)*. 2008;128:466-78.
39. Ertl J, Schafer EW. Brain response correlates of psychometric intelligence. *Nature.* 1969;223:421-2.
40. Gloning I, Gloning K. Aphasien bei polyglotten. Beitrag zur dynamik des sprachabbaus sowie zu lokalisationsfrage dieser störungen. *Weiner Zeitschrift für Nervenheilkunde.* 1965;22:362-97. Traducido en: Paradis M, editor. Readings on aphasia in bilinguals and polyglots. Montreal: Marcel Didier; 1983. p. 681-716.
41. Stengel E, Zelmanowicz J. Über polyglotte motorische aphasie. *Z Gesamte Neurol Psychiatr.* 1933;149:292-311. Traducido en: Paradis M, editor. Readings on aphasia in bilinguals and polyglots. Montreal: Marcel Didier; 1983. p. 356-75.
42. Fabbro F, Skrap M, Aglioti S. Pathological switching between languages after frontal lesions in a bilingual patient. *J Neurol Neurosurg Psychiatry.* 2000;68:650-2.
43. Holtzheimer P, Fawaz W, Wilson C, Avery D. Repetitive transcranial magnetic stimulation may induce language switching in bilingual patients. *Brain Lang.* 2005;94:274-7.
44. Kho KH, Duffau H, Gatignol P, Leijten FS, Ramsey NF, Van Rijen PC, et al. Involuntary language switching in two bilingual

- patients during the Wada test and intraoperative electrocortical stimulation. *Brain Lang.* 2007;101:31-7.
45. Aglioti S, Fabbro F. Paradoxical selective recovery in a bilingual aphasic following subcortical lesion. *Neuroreport.* 1993;4:1359-62.
 46. Aglioti S, Beltramello A, Girardi F, Fabbro F. Neurolinguistic and follow-up study of an unusual pattern of recovery from bilingual subcortical aphasia. *Brain.* 1996;119:1551-64.
 47. Abutalebi J, Miozzo A, Cappa SF. Do subcortical structures control "language selection" in polyglots? Evidence from pathological language mixing. *Neurocase.* 2000;6:51-6.
 48. Cummings JL. Frontal-subcortical circuits and human behavior. *Arch Neurol.* 1993;50:873-80.
 49. Abutalebi J, Green D. Bilingual language production: the neurocognition of language representation and control. *J Neurolinguistics.* 2007;20:242-75.
 50. Paradis M. Declarative and procedural determinants of second languages. *Language and communication in multilinguals.* Amsterdam/ Philadelphia: John Benjamins Publishing Company; 2009.
 51. Gorlitz von Mundy V. Ein 94jähriger mit einem deutschen Sprachzentrum und mit wahrscheinlich 2 slowenischen Sprachzentren. *Wien Med Wochenschr.* 1959;109:358. Traducido en: Paradis M, editor. *Readings on aphasia in bilinguals and polyglots.* Montreal: Marcel Didier; 1983. p. 624-5.
 52. Albert ML, Obler K. *The bilingual Brain.* New York: Academic Press; 1978.
 53. Hull R, Vaid J. Bilingual language lateralization: a meta-analytic tale of two hemispheres. *Neuropsychologia.* 2007;45:1987-2008.
 54. Perani D, Dehaene S, Grassi F, Cohen L, Cappa SF, Dupoux E, et al. Brain processing of native and foreign languages. *Neuroreport.* 1996;7:2439-44.
 55. Dehaene S, Dupoux E, Mehler J, Cohen L, Paulesu E, Perani D, et al. Anatomical variability in the cortical representation of first and second language. *Neuroreport.* 1997;8:3809-15.
 56. Paradis M. Language lateralization in bilinguals: Enough already! *Brain Lang.* 1990;39:576-86.
 57. Paradis M. The Loch Ness Monster approach to bilingual language lateralization: a response to Berquier and Ashton. *Brain Lang.* 1992;43:534-7.
 58. Paradis M. Another sighting on differential language laterality in multilinguals, this time in Lok Tok Fling: comments on Wuellemmin, Richardson, and Lynch (1994). *Brain Lang.* 1995;49:173-86.
 59. Paradis M. The bilingual Loch Ness Monster raises its non-asymmetric head again-or, why bother with such cumbersome notions as validity and reliability? Comments on Evans et al (2002). *Brain Lang.* 2003;87:441-8.
 60. Paradis M. Bilingual laterality: unfounded claim of validity. A comment on Hull and Vaid (2007). *Neuropsychologia.* 2008;46:1588-90.
 61. Chary P. Aphasia in a multilingual society: a preliminary study. En: Vaid J., editors. *Language processing in bilinguals: psycholinguistic and neuropsychological perspectives.* Hillsdale: Erlbaum; 1986. 183-97.
 62. Karanth P, Pangamani GN. Crossed aphasia in multilinguals. *Brain Lang.* 1988;34:169-80.
 63. Solin D. The systematic misrepresentation of bilingual crossed aphasia data and its consequences. *Brain Lang.* 1989;36:92-116.
 64. Paradis M. *A neurolinguistic theory of bilingualism.* Amsterdam: John Benjamins; 2004.
 65. Paradis M. Neurolinguistic aspects of implicit and explicit memory: implications for bilingualism and SLA. En: Ellis N.C., editors. *Implicit and explicit learning of languages.* London: Academic Press; 1994. p. 393-418.
 66. Paradis M. Language and communication disorders in multilinguals. En: Stemmer B, Whitaker HA, editors. *Handbook of the neuroscience of language.* Amsterdam: Elsevier Science; 2008. p. 341-9.
 67. Ku A, Lachmann EA, Nagler W. Selective language aphasia from herpes simplex encephalitis. *Pediatr Neurol.* 1996;15:169-71.
 68. García-Caballero A, García-Lado I, González-Hermida J, Area R, Pecimil MJ, Juncos Rabadán O, et al. Paradoxical recovery in a bilingual patient with aphasia after right capsuloputaminar infarction. *J Neurol Neurosurg Psychiatry.* 2007;78:89-91.
 69. Paradis M. *Aspects of bilingual aphasia.* Oxford: Pergamon; 1995.