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## ‘Does Broca’s area exist?’

# Christofredo Jakob’s 1906 response to Pierre Marie’s holistic stance

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### Abstract

In 1906, Pierre Marie triggered a heated controversy and an exchange of articles with Jules Déjerine over the localization of language functions in the human brain. The debate spread internationally. One of the timeliest responses, that appeared in print 1 month after Marie’s paper, came from Christofredo Jakob, a Bavarian-born neuropathologist working in Buenos Aires. The present study comprises an English translation of Jakob’s 1906 paper and a discussion of Jakob’s ideas on the localizationist–holistic approach regarding the role of Broca’s area. This issue is still at the core of scientific debate in the light of current neuropsychological and neuroimaging findings. © 2007 Elsevier Inc. All rights reserved.

**Keywords:** Aphasia; Christfried Jakob (1866–1956); Language areas; Localization of function

### 1. Introduction

Two of the most vivid debates in the history of aphasiology (Benson, 1979) took place when Broca (1861) presented his case to the Anthropological Society of Paris in 1861 and when Marie (1906a) instigated a sensational localizationist–antilocalizationist controversy in 1906 that witnessed one of the most memorable scientific battles of French neurology (Goetz, 2003). Marie’s arguments triggered a cascade of articles comprising two classic papers by Déjerine (1906a, 1906b), a further two articles by Marie (1906b, 1906c), and numerous reports by their assistants (Lecours & Joannette, 1984; Lotmar & de Montet, 1906; Marie & Moutier, 1906a, 1906b, 1906c, 1906d; Moutier, 1906; Souques, 1906) and by authors abroad (Monakow, 1906). The debate culminated with designated discussions at a special joint meeting of the New York and the Phila-

delphia Neurological Societies (McCarthy & Mills, 1906), and a three-session debate (Klippel, 1908) at the Neurological Society in Paris 2 years later (Goetz, 2003; Lecours & Joannette, 1984; Pearce, 2004). The three French 1908 debates, held on June 11th (clinical facts), July 9th (cerebral anatomy), and July 23rd (physiological pathology), involved the most explicit historical quarrel in aphasiology.

The 1906 dispute began when Marie (1906a) provocatively assailed Broca’s claim that the inferior frontal gyrus (‘third frontal convolution’) of the left cerebral hemisphere plays any role in speech and passionately opposed the localization of brain functions, a strong tendency at the dawn of the twentieth century. Marie defended a ‘holistic’, while Déjerine a ‘localizationist’ view in their exchange. Speech disorders had also been dichotomized into motor and sensory varieties (Benson, 1979), an idea also suggested by Wernicke (1874) in his doctoral thesis. ‘Localizationist’ views were expressed by Broca, Wernicke, Lichtheim, Charcot, Bastian, and Déjerine. ‘Holistic’ views of

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language functions were advocated by Hughlings Jackson, Friedrich Goltz, (then) neurologist Sigmund Freud and Marie.

In his 1891 monograph on aphasia, Freud (1983) raised doubt about a mechanistic doctrine of brain centers (Jellinger, 2006) and argued against strict localization, a view not welcomed at the time (Eling, 2006). Challenging Wernicke and even Meynert, Freud argued that one cannot separate aphasias resulting from lesions to language centers or to fasciculi that connect them. He proposed a functional approach to the speech apparatus and distinguished between areas of purely pathologic importance and areas underlying the physiology of language (Jacyna, 2005), following in the footsteps of Jackson; the latter had advocated, 17 years earlier, that ‘to locate the damage which destroys speech, and to locate speech are two different things’ (Jackson, 1874). One should nevertheless mention that Hughlings Jackson did not espouse a totally holistic view of language (Lorch, 2004); his epochal 1864 paper on aphasia and right hemiparesis in patients with embolic stroke supported Broca’s localization, and his analysis does not fit into a rigid localization–holism dichotomy, but rather is part of his neurophysiological principles.

By broaching the subject of Broca’s area in his controversial paper, Marie (1906a) forced the neurological community to re-examine some of its prior conceptions under a harder anatomical look, and re-kindled a healthy skepticism that underlies all scientific advancement (Goetz, 2003). The debate spread internationally (Editorial, 1906; McCarthy and Mills, 1906).

A prompt response (Jakob, 1906a) under the title ‘Does Broca’s area exist or not?’ (Fig. 1) came the month after Marie’s first 1906 article from Christofredo (or Christfried) Jakob (1866–1956), a German-born neuropathologist working in Buenos Aires. Having completed his doctoral thesis under Friedrich Albert von Zenker and served as assistant to Adolf von Strümpell at the Erlangen Medical Clinic, Jakob spent most of his professional life in Argentina, where he is considered the father of Neurobiology and Forensic Histopathology (Meyer, 1981; Orlando, 1966; Pedace, 1949). Jakob held professorships of Neurobiology at the Faculty of Humanities and Educational Sciences of the University of La Plata and of Anatomy and Biology at the University of Buenos Aires, and established one of the most important neuropathological laboratories in South America. He has left an invaluable legacy of 200 articles and 30 monographs (Triarhou and del Cerro, 2006a,b, 2007). *Economio and Koskinas (1925)* consider Jakob one of the three most ingenious neuroanatomists of the early twentieth century, the other two being Santiago Ramón y Cajal (1852–1934) and Theodor Käs (1852–1913).

In attempting to reconstruct the historical foundations of neuropathology, we present—a century later—Jakob’s topical views on aphasia in a modern context; his 1906 paper (Jakob, 1906a) antedates much better known articles by e.g., Déjerine and Marie from that landmark year

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## FISIOPATOLOGIA

### ¿Existe ó no un centro de Broca?

POR EL

**Dr. Chr. JAKOB**

Jefe del Laboratorio de Psiquiatría y Neurología  
 Hospicio de las Mercedes

En uno de los últimos números de *La Semaine Médicale*, P. Marie publica un estudio anatómico-clínico sobre las aphasias, y llega á las siguientes conclusiones:

1.º Que en todas las aphasias existe un fondo considerable de demencia, y más de lo que hasta ahora se ha creído;

2.º Que no existe sino una sola afasia: la sensorial, y su centro sería la primera temporal, los pliegues: supramarginal y curvo (centros de Wernicke y Déjerine);

3.º Que no existe una afasia motriz de Broca, no teniendo nada que ver el pie de la tercera frontal con las funciones del lenguaje. Lo que comúnmente se designa bajo la denominación de afasia motriz, no sería, según él, sino una combinación de la afasia (sensorial) con la anartria, y localiza el centro de ésta en el nucleo lenticular.

Fig. 1. Title page of Christofredo Jakob’s 1906 article (Jakob, 1906a).

(Table 1) regarding an issue still debated in contemporary neuroscience (Brais, 1992; Daffner, Schomer, Cosgrove, Rubin, & Mesulam, 1991; Donnan, Carey, & Saling, 1999; Grodzinsky, 2000; Lecours, Basso, Moraschini, & Nespoulous, 1984). A neuroanatomical sequel published by Jakob on the pre- and postnatal development of white matter tracts related to language based on Weigert-stained specimens (Jakob, 1906b) forms the subject of a separate article (Vivas, Tsapkini, & Triarhou, 2007).

## 2. Does Broca’s area exist?<sup>1</sup>

In one of the last issues of *La Semaine Médicale*, Marie has published an anatomical–clinical study on aphasias, and has reached the following conclusions:

<sup>1</sup> An English translation of *¿Existe ó no un centro de Broca?* by Dr. Chr. Jakob, Chief of the Laboratory of Psychiatry and Neurology, Hospicio de las Mercedes, originally published in Spanish in *La Semana Médica*, Buenos Aires, vol. XIII, no. 26, pp. 677–678, June 28, 1906.

Table 1  
Timeline of publications in the aftermath of Pierre Marie's first 1906 article

Author(s) and reference	Date published	Comment
Marie (1906a)	May 23	First paper of 'trilogy', third frontal gyrus plays no role in language
Jakob (1906a)	June 28	First paper of two, neuropsychological on Broca's area
Marie and Moutier (1906a)	July 6	Examination of the brain in a case of Broca's aphasia
Déjerine (1906a)	July 11	First paper of two, on sensory aphasia
Jakob (1906b)	July 12	Second paper of two, neuroanatomical on myelin development
Déjerine (1906b)	July 18	Second paper of two, on motor aphasia
Marie (1906b)	October 17	Second paper of 'trilogy', on subcortical (pure) aphasia
Souques (1906)	October 24	Defends Marie with motor aphasia without lesion of third frontal gyrus
Moutier (1906)	October 26	Autopsy of a case of Broca's aphasia
von Monakow (1906)	November 15	Article on aphasia and diaschisis
Marie and Moutier (1906b)	November 16	Case of softening of foot of third frontal gyrus without Broca's aphasia
Marie and Moutier (1906c)	November 23	New case of Broca's aphasia without lesion of third frontal gyrus
McCarthy and Mills (1906)	November 24	Discussion at New York & Philadelphia Neurological Societies
Marie (1906c)	November 28	Third paper of 'trilogy', historical critical essay of Broca's doctrine
Lotmar and de Montet (1906)	December 1	Mental function in Broca's aphasia from Déjerine's group
Marie and Moutier (1906d)	December 14	Case of lesion of foot of third frontal without language trouble
Editorial (1906)	December 29	British Medical Journal, Pierre Marie on aphasia

1. There is a considerable element of dementia in all aphasias, more so than what was thought until now.
2. There is only a single type of aphasia, sensory, and its center would be at the first temporal, the supramarginal and the angular gyrus (areas of Wernicke and Déjerine).
3. A motor Broca's aphasia does not exist, and the foot of the third frontal [gyrus] has nothing to do with language functions. What is commonly designated under the denomination of motor aphasia, according to him, is nothing other than a combination of (sensory) aphasia with anarthria, with its center located at the lenticular nucleus.

Such a way of thinking justifiably attracts attention, firstly, because it involves an author with great experience on the subject, and secondly, because it raises the old discussion between Trousseau and Broca, which seemed to have been definitively resolved after the work of Charcot, Wernicke, Déjerine, Monakow and their disciples.

If we investigate the reasons why Marie reached conclusions so different from the ones accepted by science up to this point, we see two. First, the observation in some cases of a lesion of the foot of the third frontal [gyrus] without a co-existing motor aphasia, a point already noticed by other investigators; and second, the opportunity to detect motor aphasia in a small number of cases as well, without the autopsy revealing a lesion in that region.

But these two observations are not totally new. Already in 1881, Exner and Goltz, and in 1883 von Gudden, published cases of lesions of the third frontal [gyrus] without motor aphasia. But from the discussions that took place in scientific congresses, it had been established according to the ideas suggested by Wernicke in 1893, Monakow and others, that such cases, although not rare, demonstrate nothing against the cited localization, since these authors do not take into account the *restitution* of cortical functions. In effect, both clinical and experimental physiology has demonstrated that when a cortical center is destroyed,

in both humans and animals, the vicinity of that area takes over its functions. For example, dogs and apes, experimentally rendered hemiplegic, recover after some months the usual habitual walk; in the same way, a person with hemiplegia, hemianesthesia, etc. may improve to the point of recovery, and talking precisely about motor aphasia, numerous cases in the literature demonstrate the great curability of this affection.

Already in 1898, Monakow used to say that the entire region of Broca could be found destroyed and even more of the cortex in the vicinity, and aphasia would disappear after a certain period of time. For that reason, Exner's and Goltz's cases might have previously been accompanied by an aphasia, which could have passed unnoticed; in the case of Marie, the same critique is possible, because he should have demonstrated that [the patient] never had it.

The second argument, that there are motor aphasias without lesions to Broca's area, was already known and discussed for more than 15 years.

Sachs (school of Wernicke) insists on the possibility that there can be a motor aphasia through an inhibition of the left hemisphere (trauma, vascular disorders), without a focus of softening being formed, possibly leading to the death of the patient before it appears.

In the case of a total lesion of Wernicke's area, motor aphasia is observed indirectly, because the auditory area does not transmit signals to the motor area through the association pathways; moreover, in deaf-mutes, where the function of [Wernicke's] area is inhibited, language is impossible as well. One may cite transient motor aphasia without focal lesions of the third frontal [gyrus] in patients with general paresis, aphasia of uremics, intoxication, non-focal [lesions], etc.<sup>2</sup>

Insofar as Marie affirms his views in such a categorical manner, without entering into the explanations just men-

<sup>2</sup> We exclude the cases of localization in the right hemisphere of the left-handed.

tioned, we can now accept, and therefore conclude, that the fundamentals of his theory—rejecting the existence of Broca's area—are in need of new and more convincing and detailed arguments.

With respect to what he calls a demented state of the aphasics, the fact is that it is observed in the institutionalized, since those patients fall invariably in dementia because of the vegetative and routine they have in the hospitals; on the contrary, it is harder to appear in patients assisted by their families and occupied with their tasks; and then the literature documents many motor aphasic patients who improve much and can even be useful, without showing significant disturbances of intelligence. Marie himself cites the case of a cook with aphasia, who even carried out his role in public, but whose incapacity, after a period of time in an institution, became total.

Therefore, the appearance of the demented state in motor aphasias depends on re-education and hygiene, which in a family setting is associated with the better care, as compared to an institution.

In sensory aphasias with extensive foci a mental decadence is noticed from the outset; Monakow points attention to the fact that the disappearance of an area as important for mental life results in severe disorders in intellectual work. If to these we add the co-existence of a generalized arteriosclerosis, it is clear that dementia will increase by the new alterations, leading to an ever decreasing blood supply.

As to the opinion of Marie of identifying subcortical aphasia with anarthria, we might always accept that, besides the lenticular nucleus, lesions involve equally well the cortico-capsular pathways, in other words, the trajectory from the frontal radiations of the internal capsule; because, both clinical and physiological studies have shown that cortical centers in the opercula participate in articulatory functions, while nothing was established for the lenticular nucleus; cases are even cited of total destruction of the latter without speech being affected.

Finally, that author opposes making subdivisions in the area that he accepts as [the area] of language.

Marie views aphasias as only differing in intensity, and not categorized into verbal blindness and alexia, as it is accepted today. It is true that there are doubts regarding this point, but such doubts are not clarified further by Marie's explanations; moreover, if we analyze the cases of alexia published by Déjerine, we see that they contradict [Marie's] thesis.

In another publication we shall occupy ourselves with the anatomical details of this question.<sup>3</sup>

### 3. Discussion

The present study makes justice to Jakob's 1906 article defending the existence of Broca's area from an anatomo-

functional point of view; Jakob questions Marie's arguments and joins the localizationist tradition—defended by Déjerine among others—that has prevailed in the last half of the twentieth century and in recent years. He was correct in separating aphasia from dementia and in claiming that there could be lesions in Broca's area which do not cause aphasia or which cause it in a transient manner. This might be produced by the compensating action of neighboring areas. On the other hand, lesions in the inferior frontal gyrus accompanied by damage to the neighboring white matter and subcortical regions like the basal ganglia can lead to Broca's aphasia. Thus, Jakob goes in the right direction when speaking of the importance of the lesion in fibers projecting to the internal capsule.

Jakob's article reveals a lucid argumentation, placing special emphasis on the embodiment of the clinico-anatomical method. A practising neuropathologist, Jakob considered morphology in a functional context and formulated ideas on the integrative function of the brain. In general, the Buenos Aires school of neurology was a follower of the anatomo-clinical tradition: What could not be demonstrated in the clinic and the autopsy table, or under the microscope, was out of the realm, with little wish from the medical establishment to venture into *terra incognita* (Manuel del Cerro, Personal Communication, February 10, 2006). Through the initiative of Domingo Cabred, Professor of Psychiatry in Buenos Aires, an attempt had begun in 1898 for ways to promote neuroanatomy in the investigation of nervous and mental disorders (Orlando, 1966). Jakob had been influential in promoting a notion for psychiatry whereby disorders could be traced to the morphology of the nervous system; until the 1930s and even later, psychiatry in Argentina was articulated around such a 'somatic style', i.e., a view of mental illness associated with the body rather than with a 'soul' (Plotkin, 2001).

In some respects (Brais, 1992), Marie's work fell within a wider movement of criticism against associationist neuropsychology (Hécaen and Angelergues, 1965; Ombredane, 1951), which was also backed by philosopher Henri Bergson (1896) in his influential book *Matter and Memory*. Marie (1906a) claimed that 'aphasia is one' and that Broca's aphasia is none other than Wernicke's aphasia complicated by anarthria, the only notable difference between the two being that in Wernicke's aphasia patients speak more or less poorly, whereas in Broca's aphasia they do not speak at all. Marie also propounded the extreme lumping of categories, suggesting that there was only one type of aphasia (i.e., sensory).

The localization debate occupied the best part of the twentieth century. One group that continued to question localization is epitomized by Hildred Schuell and her colleagues in Minnesota (Schuell et al., 1964). An in-depth narrative of the historical controversy on cortical localization with special emphasis on language functions, including cytoarchitectonic and neurophysiological correlates, can be found in standard texts such as Finger (1994).

<sup>3</sup> An English translation of the sequel paper of Jakob can be found in Vivas et al. (2007).

An advocate of localized function, Déjerine is credited with proposing the first disconnectionist account of pure alexia and the existence of a ‘visual verbal center’ in the angular gyrus, functioning to store the visual images of words. Consequently, lesions of the left angular gyrus are associated with ‘alexia with agraphia’, whereas ‘pure alexia’ results from white matter lesions of the left occipital lobe that disrupt the path between the left and right visual areas and such a visual verbal center (Déjerine, 1891, 1892).

A turning point, tremendously influential for the holistic–localizationist controversy, was the proposition of Norman Geschwind that was also based on a disconnectionist framework (Catani and ffytche, 2005; Catani et al., 2005; Geschwind, 1965a,b, 1976). According to such a framework, the disconnection syndromes represented deficits in higher cognitive functions, resulting from lesions of the white matter or of association cortex. For neuroscience research, Geschwind’s ideas led to the formulation of distributed network and connectionist theories of brain function (Catani and ffytche, 2005). The modern neuroimaging techniques offer investigators an unprecedented opportunity to supplement the information previously collected through traditional neuroanatomical tract-tracing methodologies with in vivo data on the connectivity of cortical and subcortical areas in the human brain under normal and pathological conditions.

The classically known pathway that connects Broca’s and Wernicke’s areas is the arcuate fasciculus. An additional pathway connecting Broca’s and Wernicke’s areas indirectly, termed the ‘Geschwind territory’ by Catani et al. (2005), passes through the inferior parietal lobe; it contains a caudal component that connects the temporal with the parietal cortex, and a rostral component that connects the parietal with the frontal cortex. In a confluence of reasoning, the disconnection model of Geschwind, as well as the more recently prediction by Catani et al. (2005) that lesions affecting different territories may cause different types of language deficits, are compatible with the importance attributed by Jakob (1906a) to subcortical projection pathways in Broca’s aphasia.

Marie (1906a,b) further expressed the view that phonological errors arise at the level where phonemes are selected and ordered, whereas phonetic errors arise at the level of articulatory programming (Romani et al., 2002).

In claiming that Broca’s aphasia is a combination of sensory aphasia and anarthria (Brais, 1992), Marie accordingly pointed out that the anatomical lesion of Broca’s aphasia involves Wernicke’s area and the lenticular nucleus. Jakob comments, toward the end of his article, that the associated lesions may not be located in the lenticular nucleus, but rather, in the cortico-capsular pathways, in other words, in the trajectory of the radiations of the internal capsule to cortical areas of the frontal lobe. Along a similar line of reasoning with Jakob, and 2 years later, Augusta Klumpke-Déjerine contested the ‘lenticular zone’ hypothesis during the second of the 1908 Paris debates and gave a different dimension, by convincingly contending

that its anterodorsal part includes association fibers emanating from or projecting to Broca’s area, questioning the remainder of the ‘Marie quadrilateral’ (Klippel, 1908; Lecours, 1999).

In his later *Elements of Neurobiology*, Jakob (1923) wrote: ‘What is of interest here from such a cortical symptomatology is, above all, the complex topic of the aphasias (Fig. 2), the apraxias and the agnosias; they are always of cortical origin and they can never be produced by lesions of subcortical centers or pathways; their effects result from the dynamic workings of the cortex over time and, thus, their alteration disturbs the piling of mental dynamics; therefore, we should take a closer look at the complicated game of the memory deficiencies or the partial dementias.’

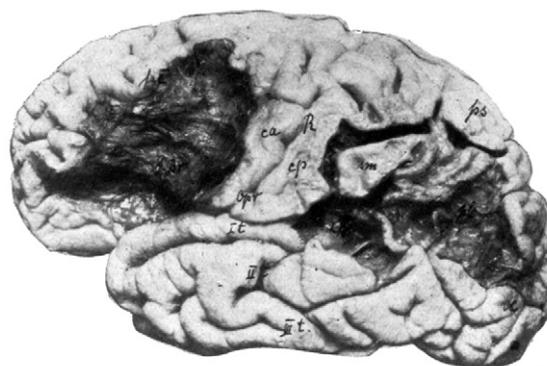


Fig. 49 a. — Caso de afasia sensoriomotora por reblandecimiento izquierdo cerebral (original) : zBr, zona de Broca; zW, zona de Wernicke; opr, opérculo rolándico; cn, cp, central anterior y posterior; R, Rolandica.

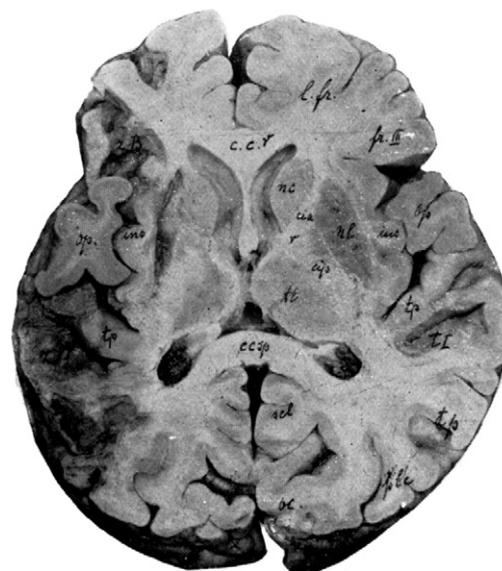


Fig. 49 b. — Corte horizontal de un caso de afasia sensoriomotora por reblandecimiento silviano izquierdo (original) : zBr, zona de Broca; zW, zona de Wernicke; opr, opérculo rolándico; ins, ínsula (compárese fig. 40).

Fig. 2. Original pathological brain specimens of Christofredo Jakob with lesions affecting the speech areas, from his 1923 *Elements of Neurobiology* (Jakob, 1923). Upper field (Fig. 49a): Case of sensory-motor aphasia from left cerebral softening. Lower field (Fig. 49b): Horizontal section of a case of sensory-motor aphasia from softening of the left Sylvian fissure. Abbreviations: zBr, zone of Broca; zW, zone of Wernicke; opr, operculum of Rolando; cn, cp; precentral and postcentral gyrus; R, Rolandic sulcus; ins, insula.

Jakob and Monakow knew each other's work; Constantin von Monakow had in his possession an autographed copy of Jakob's 1899 *Atlas of the Normal and Pathological Nervous System* (Triarhou and del Cerro, 2006a). Jakob refers to von Monakow four times, to earlier works and to the second edition of *Brain Pathology* (Monakow, 1905), as the diaschisis paper (Monakow, 1906) was published in the latter half of 1906 (Table 1). Between 1902 and 1905, studying the localization of brain functions, Monakow developed the neuropsychological concept of 'diaschisis' to account for differences between acute transient severe symptoms after focal lesions in the brain and chronic residual, limited loss of function (Koehler and Jagella, 2002; Monakow, 1905, 1906), with particular relevance to aphasia. Diaschisis is a sudden-onset functional interruption, with points of impact distributed in places where fibers that originate from one area with a focal lesion finally enter a primarily undamaged area of gray matter. A difficult concept at the beginning, diaschisis gained importance with time in relation to recovery theories (Koehler and Jagella, 2002). Perhaps the recovery of function mentioned by Jakob in chronic clinical cases was meant in the context of diaschisis.

Jakob's rebuttal of Marie's view that Broca's area plays no role in language is erudite. Jakob makes it clear that damage to Broca's area without aphasia could be attributed to a restitution of language functions in other cortical areas; motor aphasia without damage to Broca's area can be explained by considering cortical connectivity more broadly. The speech disturbance resulting from lesions of Broca's area is a different condition from Broca's aphasia, which may result from damage far outside Broca's area; the more complex syndrome traditionally referred to as Broca's aphasia, including Broca's original case, is characterized by protracted mutism, verbal stereotypes, and agrammatism (Mohr et al., 1978). Most modern aphasiologists seem to agree that Broca's 1861 patient had a global aphasia that would not justify the diagnosis of 'Broca's aphasia' today (Eling, 1986).

The same issue was revived in the heated discussion that followed the advent of cognitive neuropsychology in the 1980s (Caramazza and Coltheart, 2006). The argument that there is a 'Broca's aphasia' without an underlying lesion of Broca's area and that there are Broca's area lesions without motor aphasia was made by advocates of the cognitive neuropsychology approach against strict neuroanatomical localization (Miceli and Caramazza, 1988), without necessarily advocating the holistic stance for brain function.

Having formulated intricate theories of language functions on the basis of neurological case studies, today's cognitive neuropsychology attempts to bridge such theories with cognitive neuroscience by means of neuroimaging techniques. Investigators use fMRI, PET, and transcranial magnetic stimulation (TMS) to advocate the important role of Broca's area in language functions and to specify such functions in detail. In the technical repertoire, neuro-

imaging (e.g., fMRI) may conceivably demonstrate localized function, and neurodynamical methods (e.g., EEG frequency analyses) are considered as more integrative measurements of summed nervous activity. Of course, using a specific methodology does not necessarily pre-determine the backing of any theoretical view of brain function.

The question of the role of Broca's area remains open, even after a century of rigorous research. Modern imaging techniques allow scientists an unprecedented opportunity to visualize the brain in action. Although the involvement of Broca's area in language seems undisputed, there are multiple views on its exact role(s). Broca's area was found to mediate verbal inflection in both TMS (Shapiro et al., 2001) and fMRI (Tyler et al., 2004) studies. Embick et al. (2000) found that Broca's area is the site of syntactic processing, specifically, of 'syntactic working memory' (Fiebach et al., 2005). Broca's area is also thought of subserving language production, particularly phonological processing (Burton et al., 2001), of priming the motor response to heard speech even without speech production (Watkins and Paus, 2004; Wise et al., 1999), of being involved in semantic lexical processing (Klein et al., 2006), as well as three-dimensional mental rotation—i.e., non-musical visuospatial cognition—in orchestral musicians (Sluming et al., 2007). In considering the origins of human vocal skill, Passingham (1981) views the regulation of sequences of sounds—rather than the production of individual sounds—as the main role of Broca's area. There is a further cytoarchitectonic cortical differentiation, whereby Brodmann area 44 is likely involved in high-level aspects of programming speech production, whereas area 45 seems to be more involved in semantic aspects of language processing (Amunts et al., 2004; Grodzinsky and Amunts, 2006).

Besides the specific language functions that have been attributed to Broca's area through the use of neuroimaging techniques, there are claims that such functions may merely reflect higher-order cognitive processes. Thompson-Schill et al. (2005) claim that Broca's area, or rather the left inferior frontal gyrus, is responsible for lexical selection amongst competing candidates, whereas Rizzolatti and Craighero (2004) argue that the most important role that Broca's area played in an evolutionary sense was in action imitation, due to its location between motor areas and working memory areas. Evidence from primate studies suggests that more general cognitive demands 'prepared' Broca's area during phylogeny for the programming and sequencing of sounds, and eventually of phonemes and words (Petrides et al., 1993).

Another term with a controversial meaning, which has survived for over a century, is 'Broca's aphasia'. Jakob (1906a) writes that 'there are Broca's area lesions without Broca's aphasia and this can be attributed to the restitution of function by other areas of the brain.' The hypothesis that the right hemisphere (RH) takes over after left hemisphere (LH) damage has gained support. Points addressed

in neuroimaging studies include (i) what happens when there is damage to the language areas of the LH and (ii) whether a RH homologous Broca's area exists. In a case with a Wernicke's area lesion, it was found that homologous RH areas seem to take over functionally (Musso et al., 1999; Weiller et al., 1995). After damage to Broca's area in the LH, other areas, such as the homologous RH Broca's and Wernicke's areas, as well as regions in the LH adjacent to Broca's area, seem to be more activated during tasks that normally require a functional involvement of Broca's area (e.g., the detection of syntactic anomalies) after rehabilitation, as Thompson (2000) notes. Such findings lend credence to Jakob's succinct point regarding the restitution of Broca's area functions by adjacent areas.

Regarding the general deterioration of intellectual function in aphasic patients and not merely the breakdown of language functions, Marie had been building such a concept since 1903 in a series of psychological studies co-authored with Vaschide and addressing mental life, immediate memory and ideative association (Brais, 1992). That is another point that Jakob rebuts, attributing the deterioration of cognitive function to the poor hygienic conditions of institutionalized patients as opposed to patients cared for at home. Jakob seems to be ahead of his time, since a current debate, with important implications for the rehabilitation from brain damage, pertains to the effect of enriched environment in compensatory function. Following the work of Schwartz (1964), several other researchers have shown that brain-damaged animals kept in an enriched environment—which offers opportunities for sensory, physical and social interactions—recover better than animals kept in bare cages. The benefit of an enriched environment has been correlated with increased survival of newly generated neurons, dendritic outgrowth and synaptogenesis (Will, Galani, Kelche, & Rosenzweig, 2004). Clearly, a century ago, as Jakob argues, homes might have offered a more stimulating environment compared to hospitals, where patients might have been kept isolated in their wards. Modern rehabilitation centers try, a century later, to instate the beneficial effects of a stimulating environment, like the one that Jakob mentions.

Jakob's rejection of the idea of a dementia–aphasia co-existence seems to have received support since. Severe language deficits in Alzheimer dementia are only found at later stages. Some demented patients show word production difficulties, especially in naming tools; those cases have been studied in the realm of semantic dementia that commences from a deterioration of parieto-temporal regions (Whatmough et al., 2003). The only dementia that has been associated with language disorders and begins as an aphasia without stroke is primary progressive aphasia (PPA); in that condition, patients may manifest problems with verbs, with characteristics of Broca's aphasia, e.g., syntactic difficulties and production problems (Hillis et al., 2002; Kertesz et al., 1994). Those cases quickly evolve into a more general impairment of cognitive functions. In Jakob's era it might have been difficult to make a differential diagnosis—espe-

cially in the absence of neuroimaging technology—in order to exclude a stroke that might confound a dementia of vascular origin with aphasia.

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