In 1861, the French surgeon, Pierre Paul Broca, described two patients who had lost the ability to speak after injury to the posterior inferior frontal gyrus of the brain. Since that time, an infinite number of clinical and functional imaging studies have relied on this brain–behaviour relationship as their anchor for the localization of speech functions. Clinical studies of Broca's aphasia often assume that the deficits in these patients are due entirely to dysfunction in Broca's area, thereby attributing all aspects of the disorder to this one brain region. Moreover, functional imaging studies often rely on activation in Broca's area as verification that tasks have successfully tapped speech centres. Despite these strong assumptions, the range of locations ascribed to Broca's area varies broadly across studies. In addition, recent findings with language-impaired patients have suggested that other regions also play a role in speech production, some of which are medial to the area originally described by Broca on the lateral surface of the brain. Given the historical significance of Broca's original patients and the increasing reliance on Broca's area as a major speech centre, we thought it important to re-inspect these brains to determine the precise location of their lesions as well as other possible areas of damage. Here we describe the results of high resolution magnetic resonance imaging of the preserved brains of Broca's two historic patients. We found that both patients' lesions extended significantly into medial regions of the brain, in addition to the surface lesions observed by Broca. Results also indicate inconsistencies between the area originally identified by Broca and what is now called Broca's area, a finding with significant ramifications for both lesion and functional neuroimaging studies of this well-known brain area.

Keywords: aphasia; Broca; history; language; magnetic resonance imaging

Abbreviations: MRI = magnetic resonance imaging; CT = computerized tomography; S(RH) = sagittal image of the right hemisphere; S(LH) = sagittal image of the left hemisphere; SLF = superior longitudinal fasciculus

He could vary the intonation of the sound but was not able to produce any recognizable words or phrases. Broca saw Leborgne’s lack of speech as a test case for the question of language localization in the frontal lobes, since the patient clearly had no productive language. Leborgne died of his ailments several days later and, at autopsy, a lesion was found on the surface of the left frontal lobe as Broca had suspected. He presented his finding to the Anthropological Society (Broca, 1861b) where some of the earlier discussions had taken place, and to the more established Anatomical Society of Paris (Broca, 1861c) several months later. The finding was met with enthusiasm and taken as support for the premise that cognitive functions could be localized to specific convolutions of the brain.

A few months later, Broca encountered a second patient, Lelong, who also exhibited reduced productive speech as the result of a stroke 1 year before. This 84-year-old patient could say only five words, ‘oui’ (‘yes’), ‘non’ (‘no’), ‘tois’ (a mispronunciation of ‘trois’ (‘three’) which he used to represent any number), ‘toujours’ (‘always’) and ‘Lelo’ (a mispronunciation of his own name). At autopsy, Lelong was also found to have a lesion in approximately the same region of the lateral frontal lobe as the first case, and Broca reported it to the Anatomical Society as an important case, confirming the localization of speech to this area. He wrote,

The integrity of the third frontal convolution (and perhaps of the second) seems indispensable to the exercise of the faculty of articulate language… I found that in my second patient, the lesion occupied exactly the same seat as with the first - immediately behind the middle third, opposite the insula and precisely on the same side (translation ours, Broca, 1861a).

Broca was subsequently presented with other cases of speech disturbance with lesions encompassing the third convolution of the frontal lobe and, within 4 years, had realized that most of the cases were lesioned on the left side of the brain (Broca, 1865). Though Marc Dax may have suggested the same relationship earlier (Joynt and Benton, 1964), Broca’s careful documentation was pivotal in establishing the important connection between speech and the inferior frontal gyrus of the left cerebral hemisphere.

### Broca’s area and Broca’s aphasia

Since Broca’s time, the approximate region he identified has become known as Broca’s area, and the deficit in language production as Broca’s aphasia. Broca’s area is now typically defined in terms of the pars opercularis and pars triangularis of the inferior frontal gyrus, represented in Brodmann’s (1909) cytoarchitectonic map as areas 44 and 45 (Fig. 1). The disorder is considered to be a complex of several symptoms that, together, contribute to the syndrome of Broca’s aphasia (Goodglass, 1993; Benson and Ardila, 1996; Dronkers and Ogar, 2003). These symptoms can include problems with fluency, articulation, word-finding, repetition and producing and comprehending complex grammatical structures, both orally and in writing.

A subset of patients with Broca’s aphasia have a more severe form of the disorder. These patients are not able to produce much in the way of meaningful words or phrases, but typically can only articulate the same recurring sounds, words or phrases that are uttered each time they attempt to speak. It is apparent that Broca’s two patients also suffered from this same severe form of aphasia. The first patient, Leborgne, could only produce the jargon syllable ‘tan’. Broca himself wrote,

He could no longer produce but a single syllable, which he usually repeated twice in succession; regardless of the question asked him, he always responded: tan, tan, combined with varied expressive gestures. This is why, throughout the hospital, he is known only by the name Tan (Broca, 1861c).

Broca’s second patient, Lelong, was able to produce only the same five words. Broca believed that these patients understood what was said to them and that their language was therefore intact. He called this disorder ‘aphemia’ referring to the absence of speech and believed it to be independent of language dysfunction. Later, Trousseau coined the term ‘aphasia’ (Broca, 1864), and over time, the term ‘Broca’s aphasia’ evolved and began to include the many different symptoms of Broca’s aphasia known today.

The relationship between Broca’s aphasia and Broca’s area continued to be studied by numerous researchers and clinicians. Pierre Marie noted early on that the type of disorder described by Broca was not always related to lesions in Broca’s area, but rather to lesions involving more
medial structures, including the insula and basal ganglia (Marie, 1906). Others also questioned Broca’s conclusions regarding the localization of speech functions (e.g. Brown-Sequard, 1877; Bramwell, 1898; Moutier, 1908). Modern lesion studies (e.g. Mohr, 1976; Mohr et al., 1978; Naeser and Hayward, 1978; Kertesz et al., 1979; Schiff et al., 1983; Basso et al., 1985; Murdoch et al., 1986; Alexander et al., 1990) have found that Broca’s aphasia is caused by large lesions encompassing not necessarily Broca’s area, but surrounding frontal cortex, underlying white matter, the insula, basal ganglia and parts of the anterior superior temporal gyrus. This implies that other brain regions also participate in speech production besides Broca’s area. Considering that the syndrome of Broca’s aphasia affects so many different components of language, it is not surprising that other brain areas would also be involved. More specific roles for some of these deeper areas have been discussed for parts of the insula (Dronkers, 1996), basal ganglia (e.g. Crosson, 1985; Radanovic and Scaff, 2003), medial subcallosal fasciculus and periventricular white matter (Naeser et al., 1989) and the arcuate/superior longitudinal fasciculus (Dronkers, 1993).

It is noteworthy that Broca provided minimal discussion of such medial lesions in his original cases. This is because he made the decision not to dissect the brains of his two patients, but to preserve them in alcohol and place them in a Paris museum for posterity. Thus, Broca focused on observable lesions to the cortical surface of the brain. However, Broca did perform a provisional analysis of the brain of Leborgne, and confirmed that the lesion included ‘the small convolutions of the insula, and the corpus striatum’... ‘As to the deep parts... I could half-way examine the inner surface of the anterior horn of the lateral ventricle...’ [translated by Schiller (1992), p. 185]. Thus, he was aware that the lesion included more medial structures but could not determine the precise extent of the damage without dissecting the brain.

Since the advent of neuroimaging technology, the brain of Leborgne has been scanned on two occasions, both in the Radiology Service of E. A. Cabanis and M. T. Iba-Zizen. The first was a computerized tomography (CT) study of the Radiology Service of E. A. Cabanis and M. T. Iba-Zizen. The second patient, Lelong, was never scanned and the extent of the damage in this second case was never determined.

We had the unique opportunity to scan the brains of both Leborgne and Lelong using high-resolution volumetric MRI. This allowed us to view the brains in three dimensions and to examine the extent of both cortical and subcortical lesions in close detail. In particular, we were interested in the exact location of the lesion in the frontal lobe in relation to what is now called Broca’s area and the extent of subcortical involvement. The relation of these lesions to the deficits observed in these two historic cases is discussed later.

**Gross anatomy of the brains**

The brains were removed from the Musee Dupuytren in Paris, France by the museum director at the time, Prof. de Saint Maur. They were transported under his supervision to the Neuroradiology Service of the Centre Hospitalier National d’Ophtalmologie des Quinze-Vingts in Paris where they were photographed and scanned.

Figure 3 (panels A and B) shows lateral views of the brains of the two patients as photographed. A portion of Leborgne’s lesion was clearly visible in the inferior frontal gyrus, most noticeably in its middle third with additional atrophy in the posterior third, just above the sylvian fissure. Furthermore, modest atrophy was observed in the middle frontal gyrus and the anterior superior temporal gyrus. Upon close inspection, deformed and necrotic gyri were also noted in the anterior, inferior parietal lobe. The right hemisphere, not seen in this figure, was intact. These findings are largely consistent with Broca’s original description of the lesion at autopsy (Broca, 1861c).

Importantly, the most significant area of damage in the frontal lobe was in the middle third of the inferior frontal gyrus, not in the posterior third of the gyrus, now typically designated as Broca’s area. The posterior third clearly shows abnormalities but is not the most extensively damaged. Broca considered the posterior half of the inferior frontal gyrus lesion to be related to the articulatory deficit. Thus, the modern view of the location of Broca’s area, restricted to the posterior third of the inferior frontal gyrus, is not entirely consistent with Broca’s own determination that included far more of this gyrus. This has ramifications for both lesion and functional neuroimaging studies ascribing specific functions to Broca’s area, as interpretations of its exact location may have changed over time.

Lateral views of Lelong’s brain are shown in Fig. 3 (panels C and D). The cortex is severely atrophied. The sylvian fissure has become so widened that the insula is, abnormally, almost completely exposed. Broca’s writings indicate that Lelong had resided at the hospital for the 8 years previous to his stroke because of dementia. The report
of a dementing disorder is consistent with the finding of atrophy in this brain of an 84-year-old.

Lelong’s brain also shows evidence of a stroke that affected half of the pars opercularis in the posterior, inferior frontal gyrus, sparing the pars triangularis. Like Leborgne’s lesion, Lelong’s is also inconsistent with the location of Broca’s area as it is defined today. In this case, the lesion actually spares the anterior portion of modern Broca’s area. This is a significant finding as it implies that this second brain on which current theories of localization are based

---

**Fig. 2** Rows 1–4: Samples of the first neuroradiological images of Leborgne’s brain (1978–79) with diagrams. These CT images were the first radiographic scans obtained on this historic brain and, though not of high resolution, gave an indication of the medial extension of the lesion. The CT slices are compared with Dejerine’s diagrams in the three planes. Row 5: MRI sagittal slices (1999) discriminating the cortex and grey and white matter with higher resolution and demonstrating the use of MR imaging in preserved specimens.
does not have a lesion encompassing the entire area we now call Broca’s area. Thus, gross re-examination of these two important brains has revealed that the area defined by Broca as critical for articulation is not necessarily the same as the area currently described.

**MRI findings**

Leborgne and Lelong’s brains were imaged with a 1.5 tesla MRI scanner (GE Signa Echospeed HDX LCC Magnet 8.2.5). Several sequences were conducted, including a fast spin echo series (512 × 512 matrix, zip 1024). This series normally appears T1 weighted, but, in these cases had to be scanned differently from a living brain because of contrast differences caused by the solution in which the brains were preserved.

Representative MRI images of Leborgne’s brain are shown in Fig. 4. Coloured markings illustrate the major sulci of the brain to highlight the gyri and key structures and clarify the extent of the damage. Sulcal locations were determined by two neuroanatomists, independently, and transferred to individual slices by matching coordinates from tracings on the lateral surface of a 3D computerized rendering of the brain. The images demonstrate significant damage throughout the left hemisphere, both cortically and subcortically. The left hemisphere is clearly smaller and distorted due to the destruction of cortex and white matter throughout the hemisphere. Sagittal, axial and coronal slices through the brain reveal lesions in the left inferior frontal gyrus (slices A2, C1, S1), deep inferior parietal lobe (slices A4, C4, S1–3) and anterior superior temporal lobe (slices A2, C1–2, S1). In addition, there is extensive subcortical involvement including the claustrum, putamen, globus pallidus, head of the caudate nucleus and internal and external capsules (slices A2–3, C2–3, S2–3). The insula is completely destroyed (slices A3, C2–3, S2). The entire length of the superior longitudinal fasciculus is also obliterated (slices A4, C2–5, S2–3), along with other frontal-parietal periventricular white matter. The medial subcallosal fasciculus is also affected (slices A2, C2). The right hemisphere is unaffected (in particular, see slices S5–8) and serves as an excellent comparison to the damaged left hemisphere of this preserved brain.

The extent of the damage in the left hemisphere of Leborgne’s brain is most obvious when comparing the
Fig. 4 High-resolution MRI of the preserved brain of Leborgne with representative slices throughout the brain. The first row shows photographs of the lateral and superior surfaces of the brain, with lines indicating the slices shown below. Row A shows axial slices, Row C coronal slices, and Row S sagittal slices through the left and intact right hemisphere for comparison with each other. In the axial and coronal planes, the left hemisphere appears on the left side of the images. The following structures are delineated: interhemispheric/longitudinal fissure (orange), central sulcus/Rolandic fissure (dark blue), sylvian/lateral fissure (aqua), inferior frontal sulcus (red), superior frontal sulcus (yellow), frontomarginal sulcus (pink), superior temporal sulcus (light green) and inferior temporal sulcus (brown). Sagittal slices S3 and S4 show the superior portion of the right hemisphere crossing over the midline due to extensive damage in the left hemisphere.
sizes of the two hemispheres on the MRI images. The left hemisphere, as measured from the midline to the lateral surface, is up to 50% smaller than the right hemisphere. The coronal slices, in particular, exhibit extensive damage in the left frontal lobe when examining the distance from the interhemispheric fissure (in orange) to the Sylvian fissure (in aqua) (slices C2–3). Posterior damage is apparent in the reduced distance from the interhemispheric fissure to the superior temporal sulcus (slices C4–5). Several of the inferior parietal gyri and deep structures are missing. It is difficult to tell from examining the 3D images or even the brain itself whether the supramarginal and angular gyri are affected due to the amount of damage and effacement of identifying landmarks.

MR images of the left hemisphere of Lelong’s brain, as well as 3D reconstructions illustrating the lateral and superior surfaces are shown in Fig. 5. The right hemisphere was not preserved with the left hemisphere specimen. Images were computed from 60 sagittal slices at 512 × 512 resolution, and coloured markings again denote major sulci. Sagittal, axial and coronal slices confirm the severe atrophy noted earlier and the lesion in the posterior part of Broca’s area on the inferior frontal gyrus (slices A6–7, C4, S1). The lesion involves the posterior part of the pars opercularis, while the anterior half of this structure and the entire pars triangularis are completely spared. This can be seen in closer detail in the white square of Fig. 6. In addition, small but distinct lesions are present in the superior longitudinal fasciculus above the insula and lateral to the anterior horn of the left lateral ventricle (slices A7, C4–5, S3). The insula, though severely atrophied, is not specifically lesioned in this case nor are other deep structures, including the medial subcallosal fasciculus. There are also abnormalities in the white matter pathways in the left temporal lobe (slices A3, C7, S2–3) that may have been caused by small strokes.

Commonalities between the cases

Of interest is the fact that both patients had what Broca described as ‘aphemia’, a disruption in the patients’ voluntary speech production ability. A comparison of the anatomy of the two patients’ lesions reveals that both also had lesions in an important white matter pathway, the arcuate/superior longitudinal fasciculus, the long fibre tract that connects posterior and anterior brain areas and includes fibres of the arcuate fasciculus, previously implicated in language processing (Geschwind, 1972; Cabanis et al., 2004). The destruction of this region in the case of Leborgne can be seen most clearly in Fig. 7 where the sagittal image of the right hemisphere [S(RH)] can be compared to the matched sagittal slice through the left hemisphere [S(LH)]. The white matter tracts seen so clearly in the right hemisphere are entirely absent in the left. There is complete destruction of the deep frontal and parietal areas through which the superior longitudinal fasciculus passes. A striking comparison between the two sides of Leborgne’s brain can also be seen in the axial (A) and coronal (C) slice in which both hemispheres are represented. In contrast, the cerebellum, occipital lobes, inferior temporal lobes, superior frontal and parietal lobes and hippocampi are intact in both hemispheres.

Figure 8 highlights the lesions in the superior longitudinal fasciculus in the brain of Lelong. Sagittal, coronal and axial slices through the critical areas are shown, along with a 3D computer reconstruction with crosshairs to demonstrate the positions of the coronal and axial slices. Damage to the superior longitudinal fasciculus is evident on all slices.

Conclusions

Re-examination of the brains of Paul Broca’s two historic cases has yielded several interesting findings. First, high-resolution MRI images showed that the lesions in these two important patients extended far deeper than Broca was able to report and suggests that other areas besides Broca’s area may also have contributed to these patients’ speech deficits. Both cases had lesions extending into the superior longitudinal fasciculus (SLF), a large intrahemispheric fibre tract (Cabanis et al., 2004) that connects posterior and anterior language regions (Geschwind, 1972).

Though lesions to Broca’s area alone may cause temporary speech disruption, they do not result in severe and persisting speech arrest (Penfield and Roberts, 1959; Mohr et al., 1978). Therefore, it is possible that the aphemia characterized by Broca as an absence of productive speech was also influenced by the lesions in the region of the superior longitudinal fasciculus. Damage to Broca’s area in both cases may, in isolation, have resulted in milder speech deficits, but would not likely have caused the complete and persisting disruption of productive speech in these cases.

Though the current findings provide additional anatomical information, they by no means detract from Broca’s phenomenal discovery. Because he elected not to slice the brains, Broca could not have known the extent of underlying damage in his patients and the role it might play in their speech disorders. Broca understood the lesion extended subcortically in Leborgne, but could not determine how medially or posteriorly it extended. Broca wrote that Leborgne’s disease was progressive with the aphemia being the most persistent deficit. Since the most apparent damage was in the inferior frontal convolution, he concluded that this area was the first affected and thus the cause of the articulation deficit. This conclusion was consistent with neurological theory of his time, and Broca had no reason to consider other areas as the source of the speech disorder.

Though it may not play as extensive a role as once thought, Broca’s area is certainly involved in the execution of articulatory movements. Patients with newly acquired lesions restricted to Broca’s area tend to be mute or exhibit speech distortions for a few weeks after the injury (Penfield and
Fig. 5 High-resolution magnetic resonance images of the preserved left hemisphere of the brain of Lelong with representative slices throughout the brain. The first row shows computerized 3D reconstructions of the lateral and superior surfaces of the brain with lines indicating the locations of the slices below. The widened sulci are easily visible and indicate severe atrophy. Row A depicts axial slices, Row C coronal slices and Row S sagittal slices through the left hemisphere. In these images, the colours have been reversed to enhance the contrast; cortex appears white and white matter appears dark. They have been flipped horizontally so that the lateral cortex of the left hemisphere is on the left side of the slice. Coloured lines again show the major sulci of the brain (see Fig. 4 for color codes).
Roberts, 1959). These deficits tend to resolve quickly but nevertheless suggest that the area supports some end-stage articulatory function, probably assisting in control over the muscles of articulation. Functional neuroimaging studies also consistently observe activation in Broca’s area with tasks that involve articulation (Cabeza and Nyberg, 2000).

This current study causes us to re-evaluate the nomenclature used to describe the location of Broca’s area on the inferior frontal gyrus. Inspection of these historic brains indicates that the lesion viewed by Broca and considered by him to be critical for speech is not precisely the same region now termed Broca’s area. Leborgne’s lesion did involve the posterior inferior frontal gyrus, but was even more extensive in the region anterior to it. Lelong’s lesion occupied only the posterior third of what is now called Broca’s area. It is important to note that Broca referred to the ‘posterior inferior frontal gyrus’ as the general area important for speech. The lesions of these two cases do indeed fall within this broad region. It is since his time that Broca’s area was redefined and currently differs from Broca’s original description. Future lesion and functional neuroimaging studies should be advised that the current designation of Broca’s area is not what Broca originally intended and that defining regions of interest that specify...
Broca’s area may not be including the inferior frontal area Broca originally described.

Paul Broca began the study of the localization of function that has matured into the fields of neuropsychology, speech–language pathology, neurolinguistics and cognitive neuroscience. We now know that language and cognition are far more complicated than once thought and involve large networks of brain regions and connecting fibres. During Broca’s time, though some may have proposed networks to support cognition, scientists could not have imagined the complexity of such systems, nor could Broca have supposed that his discovery would launch such remarkable explorations into the functions of the human brain. Fortunately, Broca had great foresight in preserving these historic brains and in some ways, Leborgne and Lelong can speak to us more eloquently now than they could over 140 years ago.

Acknowledgements

We are particularly grateful to the late Professor Philippe Monod-Broca, the great-grandson of Paul Broca, and his wife and daughter for their time and interest in this work. Professor Monod-Broca’s recent book is the most comprehensive biography concerning the contributions of Paul Broca, and his support of new work related to his great-grandfather’s discoveries was inspiring to us. We also thank Professor Paul Prudhomme de St Maur, Director of the Musee Dupuytren, for his generous assistance in obtaining and transporting the brains and in supporting this project. Appreciation is also extended to Benjamin Russell, Michael DeRiesthal, Jelena Jovanovic, David Payet, Bruno Delamain, Darren Husted, Helen Ettlinger, Art Shimamura and the UC Berkeley Institute for Personality and Social Research for their contributions. This work was supported by the Department of Veterans Affairs Medical Research, the National Institute of Neurological Disorders and Stroke (NS040813), the National Institute on Deafness and other Communication Disorders (DC00216) and the Centre Hospitalier National d’Ophtalmologie des Quinze-Vingts, Paris.

References


