



## Was Broca wrong?

Ever since its inception in the 1860s it has been controversial. Broca's proposal that there exists in the left inferior frontal gyrus the faculty that co-ordinates articulated language<sup>1</sup> has always drawn criticism. Some of these concerns have been discussed at length within the pages of this journal. They include the alternative, contemporaneous view of Hughlings Jackson who was reluctant to consider that there might be a faculty of language in the brain. Jackson wondered instead whether lesions that might affect the outputs of the basal ganglia are responsible for compromised expression—verbal or written.<sup>2</sup>

Later in Paris at the turn of the 20th century, Pierre Marie challenged Broca's hypothesis even more strongly, presenting evidence that the region that had come to be known as Broca's area is not the critical one responsible for aphasia. According to Marie, any lesion within a quadrilateral or zona lenticulaire (bounded by the insula and basal ganglia on lateral and medial sides respectively, and including the white matter fibres traversing it) could result in the speech production deficits described by Broca.<sup>3</sup> MRI scans performed on the preserved brains of Broca's initial and two most celebrated cases Leborgne and Lelong, published in *Brain*, also revealed that their lesions extended into the white matter. They involved the region of the arcuate/superior longitudinal fasciculus in both patients, and in Leborgne's case went far deeper to the internal and external capsules, as well as damaging the insula, putamen, globus pallidus, head of the caudate, and the claustrum.<sup>4</sup>

Investigations of a large sample of left frontal stroke patients has provided further evidence to support the view that cortical damage to Broca's area (usually taken to consist of the pars opercularis and pars triangularis, or Brodmann areas 44 and 45) does not lead to long-term speech production deficits.<sup>5</sup> Instead, in that study, the critical left frontal region which when damaged leads to aphasia appeared to be located in the white matter, just above the insula, in the vicinity of the anterior part of the arcuate fasciculus. Neurosurgical reports, dating back to Penfield's seminal work, also reveal that cortical resections of Brodmann areas 44 and 45 do not result in long-lasting aphasia.<sup>6</sup> Other researchers in the modern era have in addition pointed to the involvement of parts of these regions in non-speech production functions, including working memory and executive or cognitive control processes.<sup>7</sup>

Notwithstanding these assaults on its originally proposed functional role, other evidence has documented quite clearly that speech articulation deficits can arise from damage to Broca's area.<sup>8</sup> Importantly, strokes here can lead to apraxia of speech, evident clinically as distortions of consonants, vowels and prosody with sound substitutions and inappropriate assignment of stress during articulation. Indeed, part of this region (pars opercularis) is considered by some authors to be crucial for sequencing syllables.<sup>1</sup>

Over the past three decades, the controversy on the role of Broca's area has been stimulated further by findings in patients


who suffer from the non-fluent variant of primary progressive aphasia (nfvPPA). Typically, these individuals have a slow and effortful non-fluent speech which is hesitant and agrammatic, with relatively intact comprehension. For all intents and purposes, they present as a slowly progressive aphasic patients who have the hallmarks of the syndrome that has classically been labelled as Broca's aphasia. Imaging is consistent with this view demonstrating that there is volume loss centred around the left inferior frontal gyrus, which progresses with time.

While some authors have made a distinction between this aphasia and a related syndrome that they characterize as a primary progressive apraxia of speech (PPAOS),<sup>9</sup> others have wondered whether these might be two extremes of the same disorder. One finding that suggested that the two conditions—nfvPPA and PPAOS—might indeed be different is that the latter appeared to be associated with atrophy of the supplementary motor area (SMA) and dorsal premotor cortex (dPMC),<sup>9</sup> regions that are far away from Broca's area.

Now in this month's issue of *Brain*, Lorca-Puls and colleagues<sup>10</sup> present their results from 104 patients with nfvPPA whose speech and language impairments were characterized using a comprehensive battery. The major finding from the perspective of the debate on Broca's area is that in their analysis, these authors discovered that apraxia of speech was associated with volume loss involving the posterior pars opercularis, deep frontal operculum, anterior insula, putamen and adjacent white matter. Expressive agrammatism, in contrast, was linked to volume loss more anteriorly: in the left pars triangularis, anterior pars opercularis, deep frontal operculum and nearby white matter. Thus these two behavioural features—apraxia of speech and agrammatism—were associated with damage to adjacent, but different parts of the left inferior frontal gyrus, and crucially spanning Broca's area.

These findings in the context of the extant literature, led the authors to propose that nfvPPA is best considered a 'spectrum disorder': a continuum that might cover PPAOS through to pure expressive agrammatism, but associated with both these phenotypes in the vast majority of patients. It is very unlikely that this debate on the nature of nfvPPA will be closed following publications of this important paper. For example, the results do not provide a simple explanation for the previous report of PPAOS being associated with SMA or dPMC atrophy.

Nor is it the case that we now have resolution on the functional role of Broca's area. Slowly progressive neurodegenerative conditions are, by their nature, very different from the sudden effects of strokes—on cortical regions that lose perfusion, as well as their remote connections. Nonetheless, the findings in this new publication on nfvPPA cannot fail to add to the debate on Broca's legacy; the controversies clearly still remain alive today, more than 150 years on from his original proposal. Did Broca get it completely wrong? I'm not sure that he did.

 Masud Husain  
Oxford, UK

E-mail: masud.husain@ndcn.ox.ac.uk

## References

1. Hickok G, Venezia J, Teghipco A. Beyond Broca: Neural architecture and evolution of a dual motor speech coordination system. *Brain*. 2023;146:1775-1790.
2. Lorch MP. The merest logomachy: The 1868 Norwich discussion of aphasia by Hughlings Jackson and Broca. *Brain*. 2008;131(Pt 6):1658-1670.
3. Coutinho L, Caramelli P, Ghizoni Teive HA. Aphasia localization: Was Pierre Marie right? *Brain*. 2021;144:3547-3549.
4. Dronkers NF, Plaisant O, Iba-Zizen MT, Cabanis EA. Paul Broca's historic cases: High resolution MR imaging of the brains of Leborgne and Lelong. *Brain*. 2007;130(Pt 5):1432-1441.
5. Gajardo-Vidal A, Lorca-Puls DL, Warner H, et al. Damage to Broca's area does not contribute to long-term speech production outcome after stroke. *Brain*. 2021;144:817-832.
6. Mandonnet E, Duffau H. Broca's area: Why was neurosurgery neglected for so long when seeking to re-establish the scientific truth? *Brain*. 2021;144:E60.
7. Fedorenko E, Blank IA. Broca's area is not a natural kind. *Trends Cogn Sci*. 2020;24:270-284.
8. Hillis AE, Work M, Barker PB, Jacobs MA, Breese EL, Maurer K. Re-examining the brain regions crucial for orchestrating speech articulation. *Brain*. 2004;127(Pt 7):1479-1487.
9. Josephs KA, Duffy JR, Strand EA, et al. Characterizing a neurodegenerative syndrome: Primary progressive apraxia of speech. *Brain*. 2012;135(Pt 5):1522-1536.
10. Lorca-Puls DL, Gajardo-Vidal A, Mandelli ML, et al. Neural basis of speech and grammar symptoms in non-fluent variant primary progressive aphasia spectrum. *Brain*. 2024;147:607-626.