

Darcey Bella Arnold
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A Narrative Review of Bilingual Aphasia Research

Aphasia, commonly caused by an injury to the brain, is a condition that affects the comprehension or production of language. For bilingual individuals, aphasic impairments—such as the inability to switch between two or more languages, and the involuntary mixing of two or more languages—create unique challenges. For centuries, however, aphasia research focussed on monolingual issues. In the late nineteenth century, several investigators began to address the gap in aphasia research, and their work remains important to formulating research questions about recovery patterns in bilingual aphasia. In more recent times, researchers have studied the brains of bilinguals with aphasia in an attempt to understand which brain regions enable healthy bilinguals to voluntarily switch between languages. This research has provided mixed results however, as one cannot infer that a brain region is directly involved in the switching between languages merely because damage to that brain region disrupts switching between languages.

The first reports of aphasia-like symptoms appeared in the Hippocratic Corpus (c. 400 BCE) associated with the Ancient Greek schools of medicine, yet it was not until 1584 that aphasia was described in detail. In his treatise on language disorders, physician Johannes Schenck von Grafenberg described a condition for which “although the tongue [is] not paralysed, the patient [cannot] speak because, the faculty of memory [is] abolished”. It was more than a century later that the first case of bilingual aphasia was reported by physician Johann Augustin Philipp Gesner. Gesner’s patient was an abbot who had lost the ability to speak. The abbot eventually recovered more Latin than German, and involuntarily mixed the two languages in conversation. Gesner did not impute any importance to the fact that the abbot’s two languages were differentially affected, and like Schenck, he considered aphasia to be a memory deficit. Rather than focal brain damage, Gessner suggested that the abbott’s language impairments were caused by “congestion of nerve ducts” outside the brain, and consequently, he attempted to remedy aphasia with bloodletting and foot baths.

Almost a century later, in 1865, Pierre Paul Broca’s celebrated post-mortem studies of aphasic stroke patients’ brains seemingly established that the ‘language faculty’ was localised in the inferior frontal gyrus (the lowest positioned fold) of the left frontal lobe. Two years later, drawing on Broca’s findings, Robert Edmund Scoresby-Jackson wrote the first paper discussing the neurophysiological implications of bilingual aphasia. Contemplating the case of “a gentleman who, after a blow on the head, lost his knowledge of Greek, and did not appear to have lost anything else”, Scoresby-Jackson speculated that while one’s first language may be stored in the posterior portion of ‘Broca’s area’, subsequently acquired languages may be stored in its anterior portion. Unfortunately, Scoresby-Jackson died shortly after his theory appeared in print. As this theory of multiple languages organised in multiple brain regions went well beyond the available evidence, it was largely rejected, and it took decades before bilingual aphasia again became the topic of research.

Research into bilingual aphasia inadvertently resumed in 1881 when the experimental psychologist Théodule-Armand Ribot published a book on the diseases of memory. In the final section of the book, Ribot mentioned the case of “an ingenious Italian” who spoke only French during his fight with yellow fever, yet “on the day of his death, spoke only the language of his native country”. He interpreted the case in terms of retrograde amnesia (namely that recent memories are more likely to be lost compared to more remote memories), and suggested that in bilingual aphasia, the language learnt earliest in life is the one to recover earliest. This minor point in Ribot’s book was soon challenged by neurologist Jean Albert Pitres, who argued that in bilingual aphasia, the language that was used most routinely is the one to recover earliest, as the most familiar language involves the most “stable cortical associations”. Fierce debate between the ‘Ribot’s law’ and ‘Pitres’ law’ camps ensued, however physicians continued to report cases which did not conform to either theory. What is more, both theories were quickly eclipsed by psychoanalytic theories such as Eugène Minkowski’s theory that the language that recovered earliest in bilingual aphasia is the one “closest to the heart”, and Eduardo Krapf’s theory that whether a patient recovered their mother tongue first depended on whether the patient had a “special need to communicate with the guardian of their vital interests” (i.e., their mother). While they failed to establish a general rule for language recovery in bilingual aphasia, it remains an open question whether the factors identified by these aphasiologists—such as frequency of language use and an individual’s preference for a language—influence language recovery.

As interest in bilingual aphasia grew, researchers began to examine the damaged brains of individuals who involuntarily mixed languages (or involuntarily switched between languages) in an attempt to locate the brain regions responsible for voluntarily switching between languages. In 1925, neurologist Otto Pötzl conducted a post-mortem study of a man who involuntarily switched between German and Czech after suffering a stroke. The study revealed a lesion in the left lower parietal lobe of the man's brain (some distance from the regions thought to be dedicated to language production). Pötzl suggested that a mechanism (i.e., a group of interconnected neurons) may be located in this region of the cortex, and that this mechanism enabled bilinguals to control which language they spoke at any given time. Decades later (with the aid of neuroimaging) many other brain regions have been proposed as candidates for containing such a switching mechanism. In 1993, Salvatore Aglioti and Franco Fabbro reported the first known case of bilingual aphasia after lesioning of the basal ganglia (deep brain structures implicated in voluntary control of motor movements) and suggested that they housed the switching mechanism. Subsequently, in the 2000s, Jubin Abutalebi and colleagues' studies of bilinguals with aphasia have provided evidence that the left basal ganglia and left frontal cortex contain a neural network responsible for controlling the correct language output, while Michael Ullman and colleagues' findings suggest that a switching mechanism relies on the temporal and parietal lobes. In short, the switching mechanism has yet to be found, and given the variability of lesions and impairments in bilingual aphasia, it is possible that such a mechanism relies on different brain regions in different individual brains.

Aphasia often affects individuals' ability to work and meet family responsibilities, which can impact their well-being and the well-being of their families. While it is generally accepted that aphasia can improve with speech and language therapies, the process by which these therapies supposedly remediate aphasia is unclear, and in many cases, it is unclear whether the spontaneous recovery from aphasia is confused with the results of a therapeutic intervention. This lack of clarity is understandable given that there is still little that is understood about the neural mechanisms that support language comprehension and production in healthy individuals, not to mention the little that is understood about the patterns of impairment and recovery in individuals with aphasia. For these reasons, further basic research into the neural mechanisms that support language is required. While this research may not immediately suggest clear-cut ways to lessen the difficulties faced by people with aphasia, it will ultimately aid the development of coherent theories and reliable therapies over time.

Matthew D. Greaves