Broca and the Biology of Language

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This year celebrates the 150th anniversary of Broca's landmark 1861 paper that changed the course of clinical neuroscience by emphasizing more clearly than his predecessors the importance of cerebral localization. This work, in conjunction with his even more comprehensive paper in 1865, set the stage for the modern investigation of brain-behavior relationships. In the intervening one-and-a-half centuries, we have become increasingly sophisticated in the measurement of language as well as in our understanding of the anatomic and physiological mechanisms capable of disrupting function. Whereas precise localization in the past required autopsy verification, radiological tools and physiological probes have allowed us entry into the living human brain, not only to gain better understanding of the nature of behavioral disorders but also to study the impact of treatment.

In this issue of Neuropsychology Review, we first revisit these two remarkable contributions by Broca, followed by commentary, which having the advantage of so many years to fathom progress, brings to our attention his insights not appreciated at the time of their publication. Because focal, ischemic stroke represents the most common etiology of aphasia, there are already extraordinary summaries of that literature, and so we leave to the reader the opportunity to review that work in other sources. Instead, we chose in this issue to explore causes of language disorders in which the lesion is defined less easily and is more chronic in nature, and whose study has required tools only recently available. First, Hamberger and Cole explore language organization

and re-organization in the setting of epilepsy. In contrast to the acute onset stroke and trauma, this chronic condition produces fascinating language syndromes and an idiosyncratic redistribution of cerebral localization first identified by invasive methods such as intracarotid Wada testing and direct cortical stimulation. The identification of the relevance of the basal temporal region to the language network emerged from these studies because naturally-occurring vascular events rarely affect this area. Functional imaging is now beginning to supplant some of these methods, reducing patient risk while yielding new information about language organization in seizure disorders.

Next, we turn to language impairment in autism in which the "lesion" has yet to be defined but whose consequences for communication range from disordered pragmatics to virtually no capacity for understanding or producing language. Stefanatos and Baron show us that it remains uncertain how much of this condition is a language disorder, per se, or a manifestation of a more pervasive abnormality of human interaction. Although uncommon in a scientific review, matters of philosophy and sociology are also raised because of the need to address the survival value of language in the social world. Moreover, autism is not a single condition. As the authors note, language disturbances may be evident in delayed initial acquisition, slow or atypical expansion of abilities, or a loss of previously-acquired language abilities.

Third, we go to the opposite end of the developmental spectrum and present the emerging field of primary progressive aphasia, or PPA, by Harciarek and Kertesz. Also not arising from a single pathological entity, this class of acquired language dysfunction typically arises from neurodegenerative disease, such as frontotemporal dementia/Pick Complex and Alzheimer's disease. In contrast to the aphasias after acute injury in which the severity of

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syndromes are usually at their worst at onset, PPA's can start as subtle retrieval disorders of words or semantic information and evolve over time into more profound disorders of language, followed by worsening of other cognitive domains. PPA's have been classified into nonfluent, semantic, or logopenic variants, each with unique symptomatic features, findings on neuroimaging, pathology, and genetics.

Broca's original observations and these three variations of language dysfunction make clear that such disorders are fundamentally physiological in nature. It therefore stands to reason that in addition to behavioral methods of treatment shown useful to some extent, biologically-based interventions are needed that alter structural brain systems. Two are presented here: First, Schlaug and his colleagues present to us the use of transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (TDCS) as methods to promote excitation in the affected hemisphere and/or to reduce inhibitory influences arising from non-injured regions in contralesional areas. These techniques are appealing because they are noninvasive and relatively safe.

Much has been learned about the post-injury relationship between the cerebral hemispheres and how each in turn plays a role in functional recovery. Second, Berthier et al. review how drug-based intervention holds potential for aphasia recovery, especially after acute injury. Not only is there promise for improvement in function, but the neurotransmitter targets of pharmacological treatment are providing novel insights into physiological systems underlying language and other cortical functions.

Broca would not likely be surprised by these developments in the last 150 years. For him, language was as much a part of brain function as motor and sensory systems. Despite the assertion of others since his time that the lesion method of language study fails to address underlying functional networks, clinicians have the burden of treating disease whose manifestations are frequently highly selective. The development of effective therapies will depend upon our degree of understanding of both the local and remote effects of these biological conditions and their associated underlying, pathophysiological mechanisms.