Language disorder

Problem with any function of language and communication.

In adults, much of what is known about the organization of language functions in the brain has come from the study of patients with focal brain lesions. It has been known for hundreds of years that a left-hemisphere injury to the brain is more likely to cause language disturbance—aphasia—than a right hemisphere injury, especially but not exclusively in right-handed persons. For about a hundred years, certain areas in the adult left hemisphere—Broca’s area in the posterior frontal lobe, and Wernicke’s area in the temporal lobe—have been identified as centrally involved in language functions. However, researchers in the field of adult aphasia are divided over the exact role these brain areas play in language processing and production. Damage to Broca’s area results in marked problems with language fluency; with shortened sentences, impaired flow of speech, poor control of rhythm and intonation (known as prosody); and a telegraphic style, with missing inflections and function words. In contrast, the speech of Wernicke’s aphasics is fluent and often rapid, but with relatively empty content and many neologisms (invented words) and word substitutions. It was initially believed that the two areas were responsible for output (Broca’s) versus input (Wernicke’s), but research does not confirm such a simple split.

Other theories ask whether the two areas might be differentially involved in syntax versus semantics, or phonology versus the lexicon, but the picture is not clear. Some have argued that adult aphasic patients, once they are stable after their injury or stroke, employ many compensatory devices that conceal or disguise the central character of their language difficulties. It then becomes more difficult to assess what is missing or disturbed because the difficulties are overlaid by new strategies, and perhaps new areas of the brain taking over functions for the damaged areas.

Infants and young children who suffer focal brain lesions in advance of acquiring language provide valuable information to neuroscientists who want to know how “plastic” the developing brain is with respect to language functions. For instance, is the left hemisphere uniquely equipped for language, or could the right hemisphere do as well? What if Broca’s or Wernicke’s areas were damaged before language was acquired? Thirty years ago a review of literature on children who had incurred brain lesions suggested that, unlike the case of adults, recovery from language disruption after left-brain damage was rapid and without lasting effect. Researchers concluded that the two hemispheres of the brain were equipotential for language until around puberty, and that this allowed young brain-damaged children to compensate with their undamaged right hemisphere.

However, several studies suggested that left-brain damage caused greater disruption to language than right-sided damage even in the youngest subjects. Children known to be using only their right hemisphere for language (because they had undergone removal of the left hemisphere for congenital abnormalities) demonstrated subtle syntactic deficits on careful linguistic testing, but the deficits failed to show in ordinary conversational analysis. Almost all of these studies were retrospective, that is, they looked at the performance of children at an older age who had suffered an early lesion. Furthermore, the technology for scanning the brain and locating the lesion site, then carefully matching the subjects, was much less developed.

With the invention of new technologies including CT scans and Magnetic Resonance Imaging (MRI), several studies have been conducted to look prospectively at the language development of children with focal, defined lesions specifically in the traditional language areas. There is surprising concordance among the studies in their results: all of them find initial (but variable) delays in the onset of lexical, syntactic, and morphological development followed by remarkably similar progress after about age two to three years. Lasting deficits have not been noticed in these children. Surprisingly, there are also no dramatic effects of laterality: lesions to either side of the brain seem to produce virtually the same effects. However, most of the data comes from conversational analysis or relatively unstructured testing, and these children have not been followed until school age.

Jill De Villiers Ph.D.

Further Reading
Until those detailed studies are extended, it is difficult to reconcile the differing results of the retrospective and prospective studies. Nevertheless, the findings suggest remarkable plasticity and robustness of language in spite of brain lesions that would devastate an adult’s system.

Jill De Villiers Ph.D.

**Further Reading**


---

**Karl Spencer Lashley**

**1890-1958**

American neuropsychologist who demonstrated relationships between animal behavior and the size and location of brain injuries, summarizing his findings in terms of the concepts of equipotentiality and mass action.

Karl Spencer Lashley was born at Davis, West Virginia, on June 7, 1890. Even as a child he was interested in animals, an interest which continued throughout his adult life. His mother, Maggie Lashley, encouraged him in intellectual pursuits. After studying at the University of West Virginia and then taking a master’s degree in bacteriology at the University of Pittsburgh, Lashley did doctoral and postdoctoral research at Johns Hopkins University. While at Hopkins, he was influenced by the zoologist H. S. Jennings, the psychiatrist Adolf Meyer, and the psychologist John B. Watson, the father of behaviorism.

Lashley was at once an experimental researcher and a psychological theoretician. His investigations were published in the leading journals and proceedings of major scientific societies. After several joint studies with Jennings, Lashley published his own thesis, “Inheritance in the Asexual Reproduction of Hydra.” He collaborated with Watson in studying behavior in seabirds, acknowledging Watson’s behavioristic approach the rest of his life.

Collaborating with Shepherd Ivory Franz, Lashley produced several papers on the effects of cerebral destruction upon retention and habit formation in rats. This was the beginning of his preoccupation with one of the persistent problems in psychology, that of cerebral localization. Earlier researchers Gall, Broca, Fritsch and Hitzig, Ferrier, and Munk were all believers in exact cerebral localization, whereas Flourens, Goltz, and Franz doubted it. The culmination of his localization experiments was *Brain Mechanisms and Intelligence: A Quantitative Study of Injuries to the Brain* (1929), his longest, most significant monograph. In it he summarized his concepts of equipotentiality and mass action and marshaled the experimental evidence to support them. Thus he accounted for the absence of precise and persistent localization of function in the cortex. Lashley’s experiments denied the simple similarity and correspondence, previously assumed, between associationistic connectionism and the neuronal theory of the brain as a mass of neurons connected by synapses.

In addition to his researches Lashley taught as professor of psychology at the universities of Minnesota and Chicago and at Harvard University. He held various honorary positions and lectureships, was on the editorial boards of numerous scientific journals, served as member of and adviser to governmental committees, and was elected to many scientific and philosophical societies. He died on August 7, 1958, in Poitiers, France.

**Further Reading**
