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Acquired Language Disorders

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Acquired language disorders are communication impairments that develop after language has been normally established, resulting from neurological damage rather than developmental issues. The term *aphasia* is used to refer to acquired language disorders. Aphasia may impact any combination of spoken language comprehension or production, reading, or writing, depending on the location and severity of the neurological damage. In most individuals, the brain regions that are most essential for language are localized to the left hemisphere of the brain, so acquired language disorders are usually caused by damage to the left hemisphere, with the most common cause being stroke. Understanding acquired language disorders provides crucial insights into the neural organization of language, while these disorders also serve as a model system for investigating fundamental principles of brain organization, neuroplasticity, and recovery that apply broadly across cognitive neuroscience.

History

In 1861, Paul Broca described an individual with a selective deficit in language production (the syndrome now known as *Broca's aphasia*), which he argued was caused by damage to a specific brain region—the left inferior frontal gyrus—revealed at autopsy ([Broca, 1861](#); [Levelt, 2013](#)). This was a seminal finding not just for the neuroscience of language but for cognitive neuroscience in general because it was one of the first compelling demonstrations of the localization of any cognitive function in the human brain (although modern research has refined important details; see [Dronkers et al., 2007](#)). Broca subsequently observed that almost all cases of aphasia followed from damage to the left, and not the right, cerebral hemisphere, leading to the conclusion that the left hemisphere is specialized for language ([Broca, 1865](#)).

A decade later, Carl Wernicke discovered that damage to the left temporal cortex—a different brain region than the one discovered by Broca—caused a comprehension deficit along with fluent but garbled speech production—the syndrome now known as *Wernicke's aphasia* ([Wernicke, 1874](#)). He then described a series of patients with qualitatively different kinds of aphasia, each of which he argued was caused by damage to different brain regions or the connections between them. Wernicke developed a sophisticated model that incorporated multiple brain regions and the pathways between them, which accounted for how different types of aphasia result from damage to the various proposed centers and pathways. Incredibly, this prescient work remains the basis for the classification of aphasia syndromes to the present day ([Goodglass et al., 2001](#)).

Since the 1970s, the application of linguistic concepts to aphasia (e.g., [Grodzinsky, 1984](#)) and the development of structural neuroimaging (e.g., [Mohr, 1976](#)) and functional neuroimaging (e.g., [Crinion & Price, 2005](#)) have dramatically advanced knowledge of the nature and neural basis of acquired language disorders [see [Neuroscience of Language](#); [Neuroscience of Syntax](#)].

Core concepts

Language is distinct from other sensory, motor, and cognitive functions

Aphasia is a disorder of central linguistic processing, not of input or output modalities. A vivid illustration of this concept is that damage to the language areas of the brain in sign language users causes fundamentally similar language disorders to those observed in spoken language speakers, even though the two rely on different modalities of transmission ([Bellugi et al., 1983](#)). It is important to distinguish aphasia from motor speech disorders, which affect neural control of the mechanisms required for speech production, and from hearing disorders, which impact spoken language perception but leave other language functions intact.

Even in individuals whose acquired language disorders are severe, other cognitive, social, and affective functions can be largely or completely spared. For example, individuals with aphasia often have preserved abilities to perform numerical calculations, engage in logical reasoning, utilize theory of mind, make and enjoy music, and navigate their way through the world ([Fedorenko & Varley, 2016](#)) [see [Theory of Mind](#)]. These findings demonstrate that the language system is functionally distinct from other cognitive systems. However, although language can be selectively impaired in principle, concomitant deficits in other systems do often occur too ([Baldo et al., 2005](#)). This observation likely reflects the fact that brain damage often impacts multiple brain networks in parallel.

Language is comprised of multiple modular components, but they are highly interactive

Language is a complex system involving many components, including a *lexicon* (mental dictionary) that links arbitrary word forms to semantic or conceptual knowledge and a grammatical system that allows limitless expression by regulating the structure of sounds (*phonology*), words (*morphology*), and sentences (*syntax*) [see [Language](#); [Phonology](#); [Morphology](#)]. The study of acquired disorders of language has revealed the independence of each of these components of language through the exploration of cases in which particular components are impaired in isolation. For example, individuals with *semantic variant primary progressive aphasia* have profound lexical deficits but strikingly preserved grammar ([Hodges et al., 1992](#)). In contrast, individuals with *agrammatism* have preserved word knowledge but an inability to construct or comprehend sentences ([Caplan & Hildebrandt, 1988](#)). Selective deficits have been documented at even more specific levels. For example, in a careful investigation of two individual patients, one was shown to be selectively impaired in grammatical processing of verbs, whereas the other was selectively impaired in grammatical processing of nouns ([Shapiro & Caramazza, 2003](#)). Through theoretically motivated studies of individuals with diverse manifestations of aphasia, researchers have provided compelling data bearing on the structure of the language system. This is not to imply that such striking dissociations are the norm; indeed, they are not: all the components of the language system interact closely, and most individuals with aphasia present with constellations of deficits across domains ([Casilio et al., 2025](#)).

Acquired language disorders are dynamic

Language disorders are not static; they change and evolve over time. Most individuals with aphasia after stroke experience some recovery of language function over time, although the extent of this recovery varies ([Wilson et al., 2023](#)). This raises the important question of what neural changes underlie this recovery (see below). Conversely, in individuals with neurodegenerative aphasia, language function declines over time as atrophy (cell death) spreads through the language network ([Rogalski et al., 2011](#)).

Questions, controversies, and new developments

The emergence of network concepts

The apparent modularity of the language system has prompted the ongoing development of localizationist models of the organization of language in the brain, in which particular regions and pathways are linked to specific linguistic functions ([Hickok & Poeppel, 2007](#)). In contrast, others have emphasized the functional similarity of different nodes of the language network ([Fedorenko et al., 2024](#)). Bridging these two perspectives, there has been a growing appreciation among aphasia researchers of the importance of functional connectivity between brain regions ([Siegel et al., 2016](#)) and an understanding that brain damage affects not just the region(s) directly impacted but all of the connected regions too ([Fridriksson et al., 2018](#)).

Mechanisms of recovery

The brain mechanisms that support recovery from aphasia are only minimally understood ([Wilson & Schneek, 2021](#)). It appears that neuroplasticity largely takes place within surviving language regions ([Stefaniak et al., 2020](#)) rather than involving the recruitment of new regions ([DeMarco et al., 2022](#)) or drawing on other networks that are not uniquely specialized for language ([De Clercq et al., 2024](#)) [see [Neuroplasticity](#)]. The role of the right hemisphere is hotly debated; although language function remains strongly dependent on the left hemisphere in most individuals with aphasia ([Wilson et al., 2018](#)), there is evidence that the right hemisphere counterparts of left hemisphere language areas may contribute to recovery, especially the right temporal lobe ([Crinion & Price, 2005](#)).

Functional communication and quality of life

From a clinical point of view, aphasia is profoundly debilitating, with one study showing it is associated with the worst health-related quality of life out of 75 diseases and conditions, including cancer and dementia ([Lam & Wodchis, 2010](#)). However, there are many factors beside the core language disorder that contribute to quality of life, including functional communication limitations, mood disorders, and fatigue ([Bullier et al., 2020](#)). The complex relationship between language function and quality of life has been neglected in cognitive science and neuroscience approaches to language disorders.

Broader connections

The current issues in acquired disorders of language—modularity, interactivity, neuroplasticity, and so on—play out in many other areas in cognitive science, such as attention, memory, motor control, and visual processing [see [Attention](#); [Visual Cognitive Neuroscience](#)]. Because language appears to be more strongly localized to a single cerebral hemisphere than any other cognitive process, and therefore more susceptible to unilateral brain damage, it provides an excellent model system for exploring these questions.

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Further reading

- Goodglass, H. (1993). *Understanding aphasia*. Academic Press.
- Levelt, W. J. M. (2013). *A history of psycholinguistics: The pre-Chomskyan era*. Oxford University Press.
- Stefaniak, J. D., Halai, A. D., & Lambon Ralph, M. A. (2020). The neural and neurocomputational bases of recovery from post-stroke aphasia. *Nature Reviews Neurology*, 16(1), 43–55. <https://doi.org/10.1038/s41582-019-0282-1>
- Wilson, S. M., Entrup, J. L., Schneck, S. M., Onuscheck, C. F., Levy, D. F., Rahman, M., Willey, E., Casilio, M., Yen, M., Brito, A. C., Kam, W., Davis, L. T., de Riesthal, M., & Kirshner, H. S. (2023). Recovery from aphasia in the first year after stroke. *Brain*, 146(3), 1021–1039. <https://doi.org/10.1093/brain/awac129>

References

- Baldo, J. V., Dronkers, N. F., Wilkins, D., Ludy, C., Raskin, P., & Kim, J. (2005). Is problem solving dependent on language? *Brain and Language*, 92(3), 240–250. <https://doi.org/10.1016/j.bandl.2004.06.103>

[↩](#)

- Bellugi, U., Poizner, H., & Klima, E. S. (1983). Brain organization for language: clues from sign aphasia. *Human Neurobiology*, 2(3), 155–70.

[↩](#)

- Broca, P. (1861). Remarques sur le siège de la faculté du langage articulé, suivies d'une observation d'aphémie (perte de la parole). *Bulletins de La Société d'anatomie (Paris)*, 2e Serie, 6, 330–357.

[↩](#)

- Broca, P. (1865). Sur le siège de la faculté du langage articulé. *Bulletins de La Société d'anthropologie de Paris*, 6, 377–393. <https://doi.org/10.3406/bmsap.1865.9495>

↩

- Bullier, B., Cassoudeulle, H., Villain, M., Cogné, M., Mollo, C., De Gabory, I., Dehail, P., Joseph, P.-A., Sibon, I., & Glize, B. (2020). New factors that affect quality of life in patients with aphasia. *Annals of Physical and Rehabilitation Medicine*, 63(1), 33–37. <https://doi.org/10.1016/j.rehab.2019.06.015>

↩

- Caplan, D., & Hildebrandt, N. (1988). *Disorders of syntactic comprehension*. MIT Press.

↩

- Casilio, M., Kasdan, A. V., Bryan, K., Shibata, K., Schneck, S. M., Levy, D. F., Entrup, J. L., Onuscheck, C., de Riesthal, M., & Wilson, S. M. (2025). Four dimensions of naturalistic language production in aphasia after stroke. *Brain*, 148(1), 291–312. <https://doi.org/10.1093/brain/awae195>

↩

- Crinion, J., & Price, C. J. (2005). Right anterior superior temporal activation predicts auditory sentence comprehension following aphasic stroke. *Brain*, 128(12), 2858–2871. <https://doi.org/10.1093/brain/awh659>

↩

- De Clercq, P., Gonsalves, A. R., Gerrits, R., & Vandermosten, M. (2024). Individualized functional localization of the language and multiple demand network in chronic post-stroke aphasia. *BioRxiv*. <https://doi.org/10.1101/2024.01.12.575350>

↩

- DeMarco, A. T., van der Stelt, C., Paul, S., Dvorak, E., Lacey, E., Snider, S., & Turkeltaub, P. E. (2022). Absence of perilesional neuroplastic recruitment in chronic poststroke aphasia. *Neurology*, 99(2), e119–e128. <https://doi.org/10.1212/WNL.0000000000200382>

↩

- Dronkers, N. F., Plaisant, O., Iba-Zizen, M. T., & Cabanis, E. A. (2007). Paul Broca's historic cases: High resolution MR imaging of the brains of Leborgne and Lelong. *Brain*, 130(5), 1432–1441. <https://doi.org/10.1093/brain/awm042>

↩

- Fedorenko, E., & Varley, R. (2016). Language and thought are not the same thing: Evidence from neuroimaging and neurological patients. *Annals of the New York Academy of Sciences*, 1369(1), 132–153. <https://doi.org/10.1111/nyas.13046>

↩

- Fedorenko, E., Ivanova, A. A., & Regev, T. I. (2024). The language network as a natural kind within the broader landscape of the human brain. *Nature Reviews Neuroscience*, 25(5), 289–312.

<https://doi.org/10.1038/s41583-024-00802-4>

↵

- Fridriksson, J., den Ouden, D.-B., Hillis, A. E., Hickok, G., Rorden, C., Basilakos, A., Yourganov, G., & Bonilha, L. (2018). Anatomy of aphasia revisited. *Brain*, 141(3), 848–862.

<https://doi.org/10.1093/brain/awx363>

↵

- Goodglass, H., Kaplan, E., & Barresi, B. (2001). *The assessment of aphasia and related disorders* (3rd ed.). Lippincott Williams & Wilkins.

↵

- Grodzinsky, Y. (1984). The syntactic characterization of agrammatism. *Cognition*, 16(2), 99–120.

[https://doi.org/10.1016/0010-0277\(84\)90001-5](https://doi.org/10.1016/0010-0277(84)90001-5)

↵

- Hickok, G., & Poeppel, D. (2007). The cortical organization of speech processing. *Nature Reviews Neuroscience*, 8(5), 393–402. <https://doi.org/10.1038/nrn2113>

↵

- Hodges, J. R., Patterson, K., Oxbury, S., & Funnell, E. (1992). Semantic dementia: Progressive fluent aphasia with temporal lobe atrophy. *Brain*, 115(6), 1783–1806. <https://doi.org/10.1093/brain/115.6.1783>

↵

- Lam, J. M. C., & Wodchis, W. P. (2010). The relationship of 60 disease diagnoses and 15 conditions to preference-based health-related quality of life in Ontario hospital-based long-term care residents. *Medical Care*, 48(4), 380–387. <https://doi.org/10.1097/MLR.0b013e3181ca2647>

↵

- Levelt, W. J. M. (2013). *A history of psycholinguistics: The pre-Chomskyan era*. Oxford University Press. <https://doi.org/10.1093/acprof:oso/9780199653669.001.0001>

↵

- Mohr, J. P. (1976). Broca's area and Broca's aphasia. In H. Whitaker & H. Whitaker (Eds.), *Studies in neurolinguistics* (Vol. 1, pp. 201–233). Academic Press.

↵

- Rogalski, E., Cobia, D., Harrison, T., Wieneke, C., Weintraub, S., & Mesulam, M. M. (2011). Progression of language decline and cortical atrophy in subtypes of primary progressive aphasia. *Neurology*, 76(21), 1804–1810. <https://doi.org/10.1212/WNL.0b013e31821ccd3c>

↩

- Shapiro, K., & Caramazza, A. (2003). Grammatical processing of nouns and verbs in left frontal cortex? *Neuropsychologia*, 41(9), 1189–1198. [https://doi.org/10.1016/S0028-3932\(03\)00037-X](https://doi.org/10.1016/S0028-3932(03)00037-X)

↩

- Siegel, J. S., Ramsey, L. E., Snyder, A. Z., Metcalf, N. V., Chacko, R. V., Weinberger, K., Baldassarre, A., Hacker, C. D., Shulman, G. L., & Corbetta, M. (2016). Disruptions of network connectivity predict impairment in multiple behavioral domains after stroke. *Proceedings of the National Academy of Sciences*, 113(30), E4367–4376. <https://doi.org/10.1073/pnas.1521083113>

↩

- Stefaniak, J. D., Halai, A. D., & Lambon Ralph, M. A. (2020). The neural and neurocomputational bases of recovery from post-stroke aphasia. *Nature Reviews Neurology*, 16(1), 43–55. <https://doi.org/10.1038/s41582-019-0282-1>

↩

- Wernicke, C. (1874). *Der aphasische symptomcomplex*. Cohn and Weigert.

↩

- Wilson, S. M., & Schneck, S. M. (2021). Neuroplasticity in post-stroke aphasia: A systematic review and meta-analysis of functional imaging studies of reorganization of language processing. *Neurobiology of Language*, 2(1), 22–82. https://doi.org/10.1162/nol_a_00025

↩

- Wilson, S. M., Entrup, J. L., Schneck, S. M., Onuscheck, C. F., Levy, D. F., Rahman, M., Willey, E., Casilio, M., Yen, M., Brito, A. C., Kam, W., Davis, L. T., de Riesthal, M., & Kirshner, H. S. (2023). Recovery from aphasia in the first year after stroke. *Brain*, 146(3), 1021–1039. <https://doi.org/10.1093/brain/awac129>

↩

- Wilson, S. M., Yen, M., & Eriksson, D. K. (2018). An adaptive semantic matching paradigm for reliable and valid language mapping in individuals with aphasia. *Human Brain Mapping*, 39(8), 3285–3307. <https://doi.org/10.1002/hbm.24077>

↩