Aphasia is a disorder of communication caused by brain damage. Aphasia constitutes a family of disruptions to comprehension and production of language in both oral and written form.

Much of the history of aphasia has been (and continues to be) concerned with attempts to characterize the natural organization of language in the brain as revealed by the selective manner in which language breaks down under focal brain damage.

The history of the field has precursors in the very earliest recordings of medicine, but largely achieved modern form with the work of Paul Broca (1861) and Carl Wernicke (1874). From this clinical work, two generalizations concerning the brain-language relationship were derived that have become canonical in the field. First, it was documented that lesions to areas in the left, but not right, cerebral hemisphere standardly result in language disruption (leading to the concept of unilateral cerebral dominance for language; e.g., Broca, 1865). Second, within the left hemisphere, lesions to different areas result in reliably different patterns of language loss (e.g., Wernicke, 1874).

Thus, damage to what has become known as Broca's area, in the lower portion of the left frontal lobe (more particularly, the opercular and triangular portions of the inferior frontal gyrus, including the foot of the third frontal convolution and extending into subcortical white matter), produces clinical observations of difficulty in articulation and production of speech with relative (but not complete) sparing of comprehension, resulting in what has come to be called Broca's aphasia. Patients with damage to this area produce little (or at least labored) speech, which is poorly articulated and telegraphic, involving omission of so-called 'function' or 'closed-class' words (articles, auxiliaries, etc.). Their speech relies heavily on nouns, and (to a far
smaller degree) verbs. Their written communication follows this same production-comprehension dissociation, with impaired writing but often less severe disturbance to reading. Because Broca's area lies next to motor areas for muscular control of speech (lips, palate, vocal chords, jaw), early assumptions were that Broca's area was a center for the encoding of articulated speech.

Wernicke's aphasia, by contrast, results from damage to the posterior region of the left hemisphere, specifically in the areas adjacent to the primary auditory cortex on the posterior portion of the superior left temporal gyrus. Patients with Wernicke's aphasia produce speech which is fluent, effortless and rapid (hence the term: 'fluent aphasia'). The content of their productions, however, is remarkable 'empty' and filled with inappropriate word use (verbal paraphasia's). Importantly, patients with Wernicke's aphasia demonstrate a profound comprehension deficit - often even at the single word level. Both writing and (particularly) reading are standardly highly impaired.

The discovery of a link between these two distinct types of language disruption and two distinct brain areas led to neuroanatomical-connectionist models of brain organization for language (Wernicke, 1874, Lichtheim, 1884), which, in one form or another, have been pervasive through to the later-20th century (e.g., Geshwind, 1979). These models attempted to capture and predict the wide variety of aphasic language deficits that had been reported throughout the literature in terms of 'disconnection' syndromes. Thus, for example the early Wernicke-Lichtheim connectionist model easily represented the fact that damage to the arcuate fasciculus (which roughly connects Wernicke's to Broca's area) leads to the inability to repeat language, a syndrome which was termed conduction aphasia. (For a complete review of other, more rare and sometimes less agreed-upon, aphasic syndromes - such as transcortical sensory aphasia, transcortical motor aphasia, subcortical motor aphasia, alexia, agraphia, anomia, etc. - see Goodglass, 1993).
Early versions of such models were modality-based, viewing Broca's and Wernicke's areas as essentially motor and sensory language areas, respectively. Broca's area was considered primarily responsible for the encoding of articulatory-form for production (speaking) and Wernicke's responsible for the organization of language perception (listening/understanding).

However, these connectionist/associationist approaches were criticized nearly from their inception as oversimplifications which did not capture the cognitive and conceptual complexity of the behavioral disruptions found in even the 'classic' (Broca's and Wernicke's) aphasias (e.g., Jackson, 1878; Head, 1926; Pick, 1931; Goldstein, 1948; Luria, 1966). Such criticisms lead to changes in the postulated nature of the 'nodes' underlying anatomical-connectionist models (or to non-connectionist characterizations entirely), with movement toward more linguistically and cognitively relevant characterizations.

Zurif, Caramazza & Myerson (1972) were major modern proponents of this movement, with empirical demonstrations of an 'overarching agrammatism' underlying the deficit in many instances of Broca's aphasia. They demonstrated that not only was production in these patients 'agrammatic', but comprehension, too, suffered from a disruption to the comprehension of structural relationships, particularly when closed-class function words were critical to interpretation or when disambiguating semantic information was unavailable. Similarly, a modality-overarching difficulty in semantical interpretation was claimed for patients with damage to Wernicke's area. In the early versions of this 'linguistic-relevance' approach to aphasia, the loci of damage was described in terms of 'loss of knowledge' (e.g. loss of syntactic rules). However the claim of knowledge-loss proved empirically difficult to sustain, whereas descriptions in terms of disruptions to the processing (access, integration) of linguistically-relevant representations (words, syntax, semantics) was empirically demonstrable. In support of such modality-independent descriptions of aphasia, this same
distribution of deficits has been shown in languages that do not rely on the auditory/oral modality. Studies of sign-language (a visuo-spatial, non-auditory language) in deaf signers have demonstrated that left-hemisphere damage results in marked impairment to sign-language abilities, but right hemisphere damage does not (despite the fact that such damage disrupts non-language spatial and cognitive abilities). Further, syntactic vs. semantic sign language disruptions have been shown to pattern neuroanatomically with the language problems accompanying damage to Broca's and Wernicke's areas, respectively (Bellugi, Poizner & Kilma, 1989).

In all, much work has demonstrated that characterizations of the functional commitment of brain architecture to language as revealed via the aphasias requires explicit consideration of the abstract, modality-neutral functional architecture (syntax, etc.) of language.

The use of behavioral techniques that examine language processing as it takes place in real-time (on-line techniques; e.g., Swinney, Zurif, Prather & Love, 1996; Neville, 1989) have recently served to further detail the brain-language relationships seen in aphasia. This work has demonstrated disruptions to functional systems underlying language at finely detailed levels of linguistic-processing/analysis, even providing a basis for the argument that some disruptions underlying 'classic' syndromes may represent, at least partially, disruptions to elemental processing resources that are recruited by the language system (memory, attention, access, etc.) With the details provided by these temporally fine-grained examinations of aphasias and by modern brain imaging, the apparent lack of homogeneity of the language disruptions found in aphasic syndromes (including the many putative aphasic syndromes not associated with Broca's or Wernicke's areas) appears on course to be better understood. It has led, on the one hand, to increasing examination of individual cases of aphasia for determination of 'new' aspects of the brain-language relationship (and, to more cautious claims
about group/syndrome patterns), and on the other hand, to new models of language, based increasingly on verifiable language behaviors as revealed by 'anomalous' aphasic cases.

References


Zurif, E., Caramazza, A. & Myerson, R. (1972) Grammatical judgments of agrammatic
aphasics. Neuropsychologia, 10, 405-417

Further Readings:


