

Working Memory and Aphasia

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ABSTRACT

Aphasia is traditionally considered to be an acquired language impairment, due to a focal (usually left-lateralized) brain lesion. However, during the last decades, there has been mounting evidence that aphasic patients also demonstrate non-linguistic processing deficits. This paper is focused on the relationship between language and memory deficits. Throughout the history of aphasiology, many scholars have corroborated the idea that there is a strong link between these two fundamental aspects of cognition. Contemporary research further supports this notion, by presenting evidence derived from lesion studies and brain imaging in healthy participants as well. In this context, aphasia is defined as a cognitive disorder with prominent linguistic deficits, rather than a specific language disturbance.

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Key words: Working memory; Aphasia

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REVIEW

Aphasia is traditionally considered to be an acquired language disturbance. That is, an impairment following a focal brain lesion that specifically affects language. However, during the last decades, there has been mounting evidence that aphasic patients also demonstrate non-specific, processing deficits. The crucial question is whether these deficits are associated with language disturbance.

A well-known psychological construct associated with (or, as some would claim, at the core of) the term “cognitive processing” is working memory (henceforth WM). WM was originally coined by Miller, Galanter, and Pribram^[1], and later on refined by Baddeley and Hitch^[2], who developed a model illustrating a cognitive mechanism involved in temporary storing and online manipulation of information. The basic idea included two slave systems for short-term memory of verbal and visuospatial stimuli and a central executive for processing and integrating incoming information. In time, WM became a more generic term, corresponding to online processing of stimuli of several modalities, selective retrieval, manipulation, etc.

The first reference to memory in aphasia literature can be found in the work of phrenologists. Gall^[3] claimed that there are two distinct “organs” for language; he names one of them *Mémoire des Mots*. A few years later, Alexander Hood^[4] reports a case of aphasia which “unfolds to us the nature of that affection by which individuals sometimes suddenly lose the verbal recollection of almost every term in the language, without the ideas being lost, or the judgment impaired” (p. 91). Within the same period, another fellow phrenologist, Bouillaud, made a clear-cut distinction between articulatory and linguistic deficits in aphasia, and attributed the latter to memory impairment^[5]. The publication of the paradigm-shifting studies by Broca^[6] and Wernicke^[7] was followed by several scholars opposing to localizationists’ rationale and stating their holistic views as an alternative to the diagram-making topology. Jackson^[8] examined language within an evolutionary frame and, on the basis of

ontogenetic and phylogenetic principles, he proposed a gradient from lowest, well-organized centers to highest, least organized centers that reflected a transaction from simple to complex cognitive functions. The possibility of a localized organ does not fit in this theoretical framework, since the central idea refers to a network consisting of several interconnecting cortices, as the biological substrate of higher cognitive processes. Head^[9] also opposed to the infamous diagram-makers of his time and corroborated the idea that aphasia is due to impaired ability to form and produce verbal and non-verbal symbols. Goldstein^[10] was also in favor of a holistic approach and claimed that the organism responds to brain lesions via catastrophic reactions. According to his view, aphasia is not due to the lesion, but rather the result of the function of the remaining, reorganized brain, and constitutes a cognitive impairment regarding processing and synthesizing incoming information. Luria^[11] introduced two fundamental psychological constructs: Cognitive function and functional system. Each function is supported by a complex functional system comprised by many different components that are involved in various levels of processing. Hence, language is not a single function, but a functional system. Consequently, aphasia is not a specific linguistic impairment, but the breakdown of a multimodal cognitive system, possibly supported by an underlying neuronal mechanism that corresponds to a network consisting of several cortices interconnected by white matter tracts. The above brief review reveals that the everlasting battle between localizationists and holists throughout the 19th and the 20th century gave birth to a few ideas that are still in the spotlight, generating very interesting debates, as the contemporary aphasia literature clearly indicates. One of the main notions suggested by the seminal works of the aforementioned scholars is that aphasia seems to be a cognitive disorder, which could also include WM deficits.

The aphasiology literature strongly underpins this particular hypothesis, by providing robust evidence for the presence of primary memory impairment in aphasia. For several years, data from lesion studies have been demonstrating in a rather straightforward manner, that individuals with aphasia often exhibit short-term (henceforth STM) and/or WM deficits, which, interestingly, are not restricted to verbal information. During the last decades, several scholars have argued that aphasic patients demonstrate STM/WM impairment on the basis of clinical neuropsychological findings^[12-16]. The reported memory deficits are not always restricted to the verbal modality. In fact, a proportion of the patients demonstrate visuospatial primary memory impairment^[14-18]. Recent findings highlight the existence of modality-dependent and modality-independent memory deficits in aphasia, and further suggest that the modality of STM/WM impairment may be affected by the locus, rather than the extent of the lesion^[19]. Regardless of modality, such deficits have been linked to the presence of aphasia in patients with left hemisphere lesions^[14] (but see also^[20]), and may share a common underlying pathological mechanism with stroke-induced language disturbance^[16]. Data derived from brain imaging studies with healthy participants support the above findings, by showing involvement of left perisylvian areas in verbal and non-verbal STM/WM^[21,22].

Contemporary research reveals possible associations between specific aphasic symptoms and WM. Three examples will be briefly discussed here: comprehension impairment, repetition deficits, and agrammatism. Comprehension is a core linguistic function and its disturbance is a hallmark of well-documented aphasic syndromes, such as Wernicke's aphasia. A recent large-scale study implementing voxel-based morphometry, revealed a strong relationship between auditory short-term memory capacity and speech comprehension^[23].

The authors further suggested that the two aforementioned cognitive abilities may share a common neuroanatomical substrate: the posterior region of the left superior temporal gyrus. According to traditional aphasiology, the main characteristic of conduction aphasia, namely impaired repetition, can be attributed to disruption of communication between Broca's and Wernicke's area. Within the theoretical framework of the Wernicke-Lichtheim model, this type of aphasia is thus a disconnection syndrome and its trademark, i.e. patient's inability to repeat a spoken sentence, is considered to be a language deficit. However, modern studies on conduction aphasia provide robust evidence for a link between repetition impairment and breakdown of a fundamental component of WM, the phonological storage, and further point to specific subcortical and cortical areas as possible lesion correlates of the particular syndrome^[24-26]. Agrammatism, i.e. impaired syntactic processing usually accompanying Broca's aphasia, is a phenomenon that has been thoroughly investigated. Even though it was initially attributed to loss of core linguistic knowledge (an idea put forward by representational accounts), consequently formulated theoretical frameworks aimed to explain agrammatism as a processing deficit. In this context, agrammatic phenomenology stems from reduced WM capacity (for a discussion on the topic, see^[27]).

There are two basic conclusions that can be drawn on the basis of this brief literature review. First, aphasia is not a clear-cut linguistic disorder, since language disturbance is often accompanied by non-linguistic deficits. It should be however noted that WM impairment is not found in all aphasic patients. For example, a closer look at the individual data reported by Potagas *et al.*^[16] reveals that there are patients with no evident WM deficits. Moreover, Kasselimis and colleagues^[19], although arguing in favor of prominent modality-specific and modality-independent WM deficits, report a small proportion of their sample that presented with intact WM capacity. Second, a clinician should thoroughly examine other cognitive domains during an aphasia assessment. Obviously, linguistic behavior could be affected by non-verbal deficits, such as executive impairment. The contemporary clinical neuropsychologist must go back through history and revisit old ideas stated by pioneers like Head, Jackson, Luria, and Goldstein, who highlighted the cognitive component of aphasia, and, last but not least, Pierre-Marie, who, throughout the heated debate with Dejerine in 1908, insisted on his initial argument that aphasia is a cognitive disorder after all^[28].

CONFLICT OF INTERESTS

The authors have no conflicts of interest to declare.

REFERENCES

- 1 Miller, G.A., Galanter, E. and Pribram, K.H. (1960). Plans and the structure of behavior. Holt, Rinehart and Winston, Inc.: New York.
- 2 Baddeley, A. D., & Hitch, G. Working memory. *Psychology of learning and motivation* 1974; 8, 47-89.
- 3 Gall, F. J. (1822–1825) Sur les fonctions du cerveau et sur celles de chacune de ses parties, 6 volumes. Paris: Eigenverlag.
- 4 Hood, A. (1824) "Case 4th - July 28, 1824 (Mr. Hood's Cases of Injuries of the Brain)". *The Phrenological Journal and Miscellany*, Vol. II: 82-94.
- 5 Tesak J., & Code C. (2008). Milestones in the history of aphasia: Theories and protagonists. New York: Psychology Press.
- 6 Broca, P. (1861) Perte de la parole, ramollissement chronique de destruction partielle du lobe antérieur gauche du cerveau. *Bulletins de la Société d'Anthropologie de Paris*, 1861, 235–238.

- 7 Wernicke, C. (1874) *Der Aphasische Symptomencomplex: Eine psychologische Studie auf anatomischer Basis*. Breslau: Cohn & Weigert.
- 8 Hughlings Jackson, J. (1866) Notes on the physiology and pathology of language. In J. Taylor (Ed.), (1958) *Selected writings of John Hughlings Jackson: Vol. 2. Evolution and dissolution of the nervous system, speech, various papers, addresses and lectures* (pp. 121–128). New York: Basic Books.
- 9 Head, H. (1926) *Aphasia and kindred disorders of speech*, 2 volumes. Cambridge: Macmillan.
- 10 Goldstein, K. (1934) *Der Aufbau des Organismus: Einführung in die Biologie unter besonderer Berücksichtigung der Erfahrungen am kranken Menschen*. The Hague: Nijhoff.
- 11 Luria, A. R. (1973) *The working brain: An introduction to neuropsychology*. Harmondsworth: Penguin.
- 12 Beeson, P. M., Bayles, K. A., Rubens, A. W., & Kaszniak, A. W. (1993). Memory impairment and executive control in individuals with stroke-induced aphasia. *Brain and language*, 45, 253-275.
- 13 Caramazza, A., Basili, A. G., Koller, J. J., & Berndt, R. S. (1981). An investigation of repetition and language processing in a case of conduction aphasia. *Brain and Language*, 14, 235–271.
- 14 Kasselimis, D. S., Simos, P. G., Economou, A., Peppas, C., Evdokimidis, I., & Potagas, C. (2013). Are memory deficits dependent on the presence of aphasia in left brain damaged patients? *Neuropsychologia*, 51(9), 1773-1776.
- 15 Martin, N., & Ayala, J. (2004). Measurements of auditory-verbal STM span in aphasia: Effects of item, task, and lexical impairment. *Brain and Language*, 89, 464-483.
- 16 Potagas, C., Kasselimis, D., & Evdokimidis, I. (2011). Short-term and working memory impairments in aphasia. *Neuropsychologia*, 49(10), 2874-2878.
- 17 Albert, M. L. (1976). Short-term memory and aphasia. *Brain and Language*, 3, 28–33.
- 18 De Renzi, E., & Nichelli, P. (1975). Verbal and nonverbal term memory impairment following hemispheric damage. *Cortex*, 11, 341-354.
- 19 Kasselimis, D., Simos, P., Peppas, C., Chatziantoniou, L., Kourtidou, E., Evdokimidis, I., & Potagas, C. (2013). Modality-independent and Modality-specific Memory Deficits in Aphasia: Effects of Left Hemisphere Lesion Extent and Location. *Procedia-Social and Behavioral Sciences*, 94, 120-121.
- 20 Burgio, F., & Basso, A. (1997). Memory and aphasia. *Neuropsychologia*, 35, 759-766.
- 21 Paulesu, E., Frith, C. D., & Frackowiak, R. S. J. (1993). The neural correlates of the verbal component of working memory. *Nature*, 362, 342–345.
- 22 Wager, T. D., Smith, E. E. (2003). Neuroimaging studies of working memory: a meta-analysis. *Cognitive, Affective, & Behavioral Neuroscience*, 3(4), 255-274.
- 23 Leff, A. P., Schofield, T. M., Crinion, J. T., Seghier, M. L., Grogan, A., Green, D. W., & Price, C. J. (2009). The left superior temporal gyrus is a shared substrate for auditory short-term memory and speech comprehension: evidence from 210 patients with stroke. *Brain*, 132(12), 3401-3410.
- 24 Baldo, J. V., Klostermann, E. C., & Dronkers, N. F. (2008). It's either a cook or a baker: Patients with conduction aphasia get the gist but lose the trace. *Brain and Language*, 105(2), 134-140.
- 25 Buchsbaum, B. R., Baldo, J., Okada, K., Berman, K. F., Dronkers, N., D'Esposito, M., & Hickok, G. (2011). Conduction aphasia, sensory-motor integration, and phonological short-term memory—an aggregate analysis of lesion and fMRI data. *Brain and language*, 119(3), 119-128.
- 26 Gvion, A., & Friedmann, N. (2012). Phonological short-term memory in conduction aphasia. *Aphasiology*, 26(3-4), 579-614.
- 27 Kasselimis, D.S., Varkanitsa, M., 2015. Neurological Approaches to Agrammatism. In: James D. Wright (editor-in-chief), *International Encyclopedia of the Social & Behavioral Sciences*, 2nd edition, Vol 16. Oxford: Elsevier. pp. 690–697.
- 28 Lecours A. R., Chain F., Poncet M., Nespoulous J-L., & Joanette Y. (1992). Paris 1908: The hot summer of aphasiology or a season in the life of a chair. *Brain and Language*, 42, 105-152.

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