ABSTRACT

Aphasia is traditionally considered to be an acquired language disturbance, 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-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\(^\text{9}\) 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\(^\text{10}\) 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\(^\text{11}\) 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 19\(^\text{th}\) and the 20\(^\text{th}\) 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\(^\text{12-16}\). The reported memory deficits are not always restricted to the verbal modality. In fact, a proportion of the patients demonstrate visuospatial primary memory impairment\(^\text{14-18}\). 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\(^\text{19}\). Regardless of modality, such deficits have been linked to the presence of aphasia in patients with left hemisphere lesions\(^\text{14}\) (but see also\(^\text{20}\)), and may share a common underlying pathological mechanism with stroke-induced language disturbance\(^\text{16}\). 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\(^\text{21-22}\).

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\(^\text{23}\).

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 conductive 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\(^\text{24-26}\). 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\(^\text{27}\)).

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 Potagás et al\(^\text{12}\) reveals that there are patients with no evident WM deficits. Moreover, Kasselimis and colleagues\(^\text{19}\), 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, throughout the heated debate with Dejerine in 1908, insisted on his initial argument that aphasia is a cognitive disorder after all\(^\text{29}\).

**CONFLICT OF INTERESTS**

The authors have no conflicts of interest to declare.

**REFERENCES**

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