Brain-language relationships

Relações cérebro-linguagem

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Dear Editors,

First of all, we congratulate Devido-Santos et al.[1] by their interesting paper. Until recently, our understanding of how language is organized in the brain depended on analysis of language deficits in patients with fortuitously placed lesions. Language is organized in large-scale, predominantly left-lateralized, temporo-parieto-frontal networks in the human brain[2]. The study of Devido-Santos et al.[1] suggested that there was no direct relationship between lesion topography and stroke aphasia type in their sample, in contrast with the large amount of scientific knowledge accumulated since Paul Broca's descriptions of brain-language relationships in which classic aphasias have been correlated to specific brain regions[3-6].

It is true that a number of traditional concepts regarding mechanisms of aphasia are inconsistent with now abundant data. What has not been directly determined, however, is whether it is the effective connectivity between brain regions – and therefore also the integrity of the white matter tracts that connect them - that underpins successful linguistic analysis.

We should ask if the language changes detected were not actually supported by other cognitive functions associated with basal ganglia or the right hemisphere. In other words, linguistic functions dependent upon, for example, other executive functions, such as attention length, strategic planning and, sure, working memory, a large distributed system involved in many high order language functions.

Another question should be formulated in order to address the lack of relationship between lesion topography and stroke aphasia type: what is the real efficacy of same neuroimaging methods in detecting language circuits involved in subtle aphasic disorders? Conventional magnetic resonance may not be an ideal method to detect such abnormalities, mainly because it can not detect subtle cortical or subcortical dysfunction or even may not grasp diaschisis phenomena (a sudden loss of function in a portion of the brain connected to, but at a distance of, a damaged area). It is important to note that diaschisis is a dysfunction of tissue that is not damaged, but for which the blood supply has been altered because of interruption of "upstream". Besides that, detailed consideration of the vascular events leading to striatocapsular infarction strongly suggests that associated linguistic deficits are predominantly related to sustained cortical hypoperfusion and infarction not visible on structural imaging studies. Maybe tractography or diffusion tensor imaging or functional magnetic resonance imaging (fMRI) should be considered as gold standard neuroimaging methods in this area. In fact, the availability of functional magnetic resonance imaging for in vivo analysis of the normal brain has revolutionized the study of language.

Another unexpected find in their study, i.e., language abnormalities in non-aphasics (G2 patients) who manifested poor performance in verbal fluency tasks and difficulties on tests involving responses of greater complexity, was not sufficiently explained, mainly if we consider they rejected illiterate cases from their sample. Actually, G2 group was defined in methods as non-aphasics, but then were presented as having fluency deficits in the results.

Finally, we would like to congratulate Devido-Santos et al. because they have mentioned many Brazilian authors and for their excellent work in this field, a deserved tribute to Brazilian research in linguistic science.

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