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Review

Aphasia in vascular lesions of the basal ganglia: A comprehensive review

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ABSTRACT

Between 1970 and 1990, the study of aphasia secondary to subcortical lesions (including the basal ganglia – BG) was largely driven by the advent of modern neuroimaging techniques such as MRI and PET. However, attempts to characterize a pattern of language abnormalities in patients with basal ganglia lesions proved unfruitful. We conducted a comprehensive review of language disturbances after vascular lesions in the BG. Literature search in Medline and LILACS (1966–2016) and PsychINFO (last 25 years) was conducted, and returned 145 articles, with 57 eligible for the review yielding data on 303 patients. We report the clinical and neuroimaging features of these cases. Results showed that aphasias caused by BG lesions are heterogeneous with weak clinicoanatomical correlations. Data derived from follow-up and flow/metabolism studies suggest that subcortical aphasia caused by BG lesions involves hypoperfusion in the cortical territories of the middle cerebral/internal carotid arteries (MCA/ICA) and their branches.
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Contents

1. Introduction

Subcortical aphasia is currently defined as aphasia secondary to lesions involving the basal ganglia and thalamus, without

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involvement of cortical structures and minimal compromise of connecting white matter pathways (Alexander & Hillis, 2008).

The advent of modern neuroimaging techniques has allowed a more precise visualization of subcortical structures and consequent identification of neurological syndromes in which subcortical involvement is predominant or exclusive. A great interest in the study of subcortical lesions has emerged in Cognitive Neurology, since the notion that subcortical lesions can impair cognitive functions, although long-held, called for a more accurate confirmatory method of anatomic study *in vivo.* Thus, although interest in language disturbances secondary to subcortical lesions began short after the description by Marie (1906), the widespread clinical use of cranial computed tomography (CT) in the 1970s led to a large number of studies focusing on the role of these structures in language processing.

Pioneering studies on the role of subcortical lesions in language usually centered on extensive brain lesions often involving large areas of periventricular white matter (PVWM) and associated cortical lesion. Following the advent of magnetic resonance imaging (MRI), emphasis was placed on studying smaller and more limited lesions to improve anatomo-clinical correlations, but most studies still addressed lesions affecting both the basal ganglia structures and the PVWM, in varying combinations.

Although attempts to characterize subcortical aphasic "syndromes" in a similar way to classic cortical syndromes have proved unfruitful, it is widely accepted that subcortical lesions share some features, such as a more benign clinical picture and better long-term prognosis. Striatocapsular lesions may lead to articulatory and word-finding deficits, and comprehension may be impaired at more complex syntactic levels, but further anatomo-clinical correlations were found to be much less consistent (Alexander & Hillis, 2008; Ardila, 2014).

Currently, language disturbances secondary to lesions in the basal ganglia are attributed mainly to hypoperfusion and associated cortical ischemia. This notion is justified by the fact that most vascular lesions in this region are caused by occlusion of the middle cerebral artery and its branches or by hemorrhages that can exert a mass effect on adjacent language-related cortex (Hillis et al., 2002, 2004). However, arguments supporting a direct role of the basal ganglia in language processing can be found in functional studies (Crosson et al., 2003; Gill Robles, Gatignol, Capelle, Mitchell, & Duffau, 2005).

Our objective was to present a comprehensive review of studies on language disturbances due to single lesions of vascular etiology (strokes) involving exclusively the basal ganglia (and/or adjacent internal capsule), to identify the pattern of these language disturbances, and also to discuss the main physiopathological hypotheses for their occurrence.

2. Methods

2.1. Search strategy and study selection

A search was conducted of the Medline and LILACS databases for the period spanning from 1966 to 2016, and of the PsychINFO database for the last 25 years, including the following MeSH terms: *subcortical, basal ganglia, putamen, caudate nucleus, lenticular nucleus, striatum associated* through the Boolean terms 'OR' and 'AND' to *aphasia, language*. This preliminary search yielded the following results: *"subcortical aphasia":* 476 papers; *"basal ganglia aphasia":* 426 papers; *"putamen aphasia":* 90 papers; *"caudate aphasia":* 105 papers; *"lenticular nucleus aphasia":* 123 papers; *"striatum aphasia":* 140 papers; *globus pallidus aphasia:* 32 papers; *"basal ganglia language":* 1348 papers; *"putamen language":* 241 papers; *"caudate language":* 375 papers; *"lenticular nucleus language":* 401 papers; *"striatum language:* 535 papers; *globus pallidus language: 135;* performing a total of 4427 papers. Titles and abstracts were screened and those containing the terms *dementia, Alzheimer, Parkinson, Huntington, schizophrenia, alcoholism, cerebellum, epilepsy, obsessive-compulsive, bipolar, corticobasal, PSP, Creutzfeldt-Jacob* were excluded, as were all duplicated papers. The initial search retrieved 1323 articles. Abstracts of all these papers were assessed for content, with a second screening of articles that met the following criteria: studies reporting patients aged older than 18 years, with a single focal vascular lesion (ischemic or hemorrhagic) of subcortical structures (including basal ganglia and internal capsule but not extending to the PVWM) in both hemispheres documented by neuroimaging study and language disturbances secondary to these lesions, published in English, French, Spanish, Italian or Portuguese, and to which authors had access to the full articles. At this point, papers focusing on purely theoretical or experimental aspects of basal ganglia role in language, as well as those focusing on non-vascular lesions or patients up to 18 years of age were excluded. This selection process yielded 145 articles.

The resultant 145 articles were then assessed in full, and a further selection was performed. The primary objective of some studies was not to investigate language disturbances but to provide a general clinical description of "subcortical syndromes", where no specific language tests were applied. In this second stage, most of the excluded articles comprised: (a) description of language disturbances or neurocognitive experiments involving frontostriatal circuits with associated frontal lesions, (b) perisylvian injuries associated with subcortical lesion, (c) multiple cerebral lesions, (d) studies that did not investigate basal ganglia structures separately, (e) studies that did not provide individual descriptions of patients' symptoms, or (f) studies in which patients could not be matched with the corresponding lesions on neuroimaging We also manually searched the reference lists of these articles and selected those that were relevant. This last analysis led to the final inclusion of 57 articles involving a total of 303 patients. In some cases, a few patients in a given study were excluded for failing to meet the above outlined criteria. (Table 1 and Fig. 1).

2.2. Patient selection

Patients aged over 18 years of both sexes with a single (left or right hemisphere) vascular lesion in the basal ganglia and without PVWM or cortical involvement were included. Cases reported to be non-aphasic in the acute stage were included. Out of the total 303 patients included, 6 were subsequently excluded (patients 55, 108, 115, 147, 157, and 269) for not fully satisfying the predefined inclusion criteria, giving a final total of 297 patients (Appendix A). Studies were included independently of how much time had elapsed from the ictus to the actual report. Therefore, we divide our results in: patients with aphasia in the acute stage (up to 30 days following the ictus), subcaute stage (from 31 days up to 6 months after the ictus), and chronic (more than 6 months after the ictus). Patients were not enrolled in this review if there was any mention that they had been submitted to speech therapy in the original study.

2.3. Language assessment

All articles for which data on the parameters fluency, comprehension impairment, repetition deficits, presence of anomia, presence of paraphasias (phonemic and semantic) was available were included. Articles containing a description of results obtained on language tests were included, as were those in which disturbances were described by the authors of the study after clinical examination. Reading and writing abilities were not taken into account for

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