Language Disorders due to Posterior System Strokes - An Ignored Dysfunction

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ABSTRACT

The anterior system is primarily responsible for the clinical picture in a patient that presents with clinical aphasia. However, recent reports have shown that injuries to posterior structures, the cerebellum in particular, may have a role in language processing. Herein, we will look first at the linguistic role of the cerebellum in light of the literature, then of the thalamus and some described clinical syndromes, and finally, specific syndromes resulting from occipital lobe lesions, all of which are supported by the posterior vascular system. The human brain is such a complex organization that in addition to the thalamus and occipital cortex, we can see the involvement of the cerebellum in high cognitive functions. Posterior system strokes may lead to clinical findings of cognitive deficits, including neurolinguistic components. Determining these defects in stroke patients may precipitate changes in current management strategies.

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Stroke and Language Disorders

The term “stroke” refers to a sudden-onset focal neurological situation that specifically results from cerebrovascular disorders (1). Brain tissue basically receives blood through two internal carotid and two vertebral arteries. Multi-level collateral circulations among the anterior and posterior systems preclude against drawing a clear-cut margin between the two. The vertebrobasilar system provides blood to the cerebellum, brain stem, occipital lobes, and posterior portions of the temporal lobes and partially to the internal capsules, thalami, and basal ganglia. Clinicians define vertebrobasilar strokes as more critical processes, since the structures supported by the system, especially the brain stem, cerebellum, and related regions, are responsible for consciousness, vital functions, regulation and coordination of movements, breathing, and swallowing. Could it be that these subcortical structures have an effect on cognition? Would it be erroneous to think that lesions on these structures may produce high-level dysfunction, such as in language processing? In this

current review, we discuss in particular the linguistic disorders caused by lesions in areas supported by the posterior system.

Language Disorders and the Cerebellum

It is traditionally accepted that an aphasic syndrome is brought about by a loss of high cortical functions related to language processing, due to any reason. Thus, the anterior system is primarily held responsible for the picture in a patient that presents with clinical aphasia. However, recent reports have shown that injuries to posterior structures, the cerebellum in particular, may have a role in language processing. As various neuroradiological techniques advance [for instance, functional magnetic resonance imaging (fMRI) and single-photon emission computed tomography (SPECT)], the knowledge gap regarding the neural basis of language is closing, and observations are growing daily. The cerebellum is an important brain structure that regulates motor coordination and has an effect on balance and muscle tone. Cerebellar damage can present with ataxia,

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dysmetria, dysdiadochokinesia, vertigo, hypotonia, nausea, and vomiting to varying degrees. When such a dramatic picture is present in a patient, it is not always possible to demonstrate any linguistic-related problems. However, a detailed observation and neurological examination may expose that at least a fractional number of patients experience some linguistic problems. This implies that the cerebellum, beyond its importance as it relates to motor functions, has a crucial place in cognition, as well. Regarding the posterior system, thalamic aphasias are well described. The thalamus receives blood from both the anterior and posterior systems to varying degrees. While linguistic disorders in which repetition is preserved and speech output is diminished and that anomia and paraphasias accompany are attributed to ventrolateral and ventral anterior nuclei lesions, a fluent aphasia is seen with pulvinar and posterior lateral nucleus lesions. Finally, dorsomedial nucleus damage may lead to verbal memory defects (2). We will look first at the linguistic role of the cerebellum in light of the literature and that of the thalamus and some described clinical syndromes and finally briefly at two specific syndromes resulting from occipital lobe lesions, all of which are supported by the posterior system.

Marien et al. (3) reviewed the functions of the cerebellum in certain cognitive tasks. The cerebellum becomes operational in diverse complex organizations, such as cognitive planning, timing, attention, visuospatial processing, learning, memory production, and visuomotor imagination. Schmahmann and Sherman (4) developed the concept of "cerebellar cognitive affective syndrome," describing it as the loss of executive functions, visuospatial disorganization, impaired visuospatial memory, personality changes, and certain difficulties in language processing. The hypothesis of cross cerebellar-cerebral diaschisis was proposed in 1987, after which growing interest in this subject was seen (5). According to Marien et al. (3), linguistic disorders following cerebellar pathology reflect a diminished input at the cortical level, and the ongoing physiopathological process is the effect of diaschisis. However, the diaschisis hypothesis may not be effectual in all cases; for instance, Gasparini et al. (6) could not show neuroradiological diaschisis in an obvious linguistic disorder that developed immediately after right cerebellar stroke.

De Smet et al. (7) summarized the hypotheses concerning the cerebellar involvement in language processing. The first is the diaschisis, which was proposed appreciably by the aforementioned groups: alterations in cortical blood circulation secondary to reduction of the excitatory impulses may give rise to impairment in high cortical functions. The second is the timing hypothesis, per which the language disorders are the consequences of failure of time-dependent modulations of linguistic functions. The third hypothesis covers the opinions that maintain the cerebellum's direct involvement in language processing. Proofs that support them all are present, and it would be a mistake to exclude the others in favor of any one. For example, Marien et al. (8) demonstrated hypoperfusion of the left medial frontal area in a patient with a right superior cerebellar ar-

tery territory infarction via SPECT analysis. On the other hand, after recapitulating the acoustic, kinematic, and fMRI studies, Ackermann et al. (9) asserted that the cerebellum intervenes in the temporal modulation of language usage at the articular, as well as pre-articular, levels. The proof is that the coordination of orofacial and laryngeal movements is impaired, and the maximum speech rate is reduced. However, there is another aspect, in which the cardinal role of the cerebellum's temporal modulation during covert speech has also been established by fMRI studies. It is in this sense that the authors adduce that cerebellar lesions resulting in impairment of various executive tasks are actually the functions that require covert speech; thus, they emphasize the timing hypothesis. Finally, Leiner et al. (10) established the direct importance of the human cerebrocerebellar system in language functions based on neuroanatomical data.

It is generally accepted that right cerebellar lesions lead to linguistic disorders, while left cerebellar lesions lead to failure in visuospatial function, since the cerebellum is linked with the contralateral cerebral cortex (11). In 1994, Silveri et al. (12) reported a case with dysarthric and agrammatic speech, accompanied by word errors, that developed after right cerebellum infarction; moreover, the mutism in this case at the beginning of the process is noteworthy and constitutes an example of anarthric mutism. Three years later, Zettin et al. (13) presented a case of expressive agrammatism following right cerebellar hemorrhage. In addition, they specified the presence of this clinical situation in the early period of the disease and its reversible tendency in the chronic phase; in this case, left frontotemporal hypoperfusion was also identified with SPECT. More precisely, these cerebellar regions are lateral portions of cerebellar hemispheres and the dentate nucleus, which project to the cerebral cortex much more than any other cerebellar region, and thus, the term "neocerebellum" is used (10). The spectrum of language disorders that are found in cerebellar lesions is extensive and constitutes a heterogeneous group. Ataxic dysarthria is the most frequent problem. Yet, the current opinion is that the pathology is not limited simply to motor coordination of language. Schweizer et al. (14) found statistically significant verbal and semantic fluency defects in cases with right cerebellar lesions as compared to left cerebellar patients and controls. Gasparini et al. (6) reported a dysprosodic and agrammatic linguistic disorder with accompanying word-finding difficulties; moreover, this agrammatism was present in reading and writing functions to a considerable degree. Moretti et al. (15) pointed out an obvious reading difficulty in cerebellar vermian space-occupying lesions; although single-word reading was intact, reading accuracy was reduced when reading long phrases. Silveri et al. (16) described a spatial dysgraphia case secondary to cerebellar atrophy. In their case, letter omissions and repetitions in writing were prominent. The authors reported the asynchrony of motor program and sensorial feedback, and they highlighted the timing hypothesis. Nevertheless, in another research study, cerebellar deficit as a cause of dyslexia was not proven; yet, when

a patient group constituted by children is considered, these results might reflect the temporal difference. Secondly, rather than going methodologically from cerebellar deficit to dyslexia, an etiological investigation was performed in dyslexic patients. Frank et al. showed that cerebellar patients frequently perform within the normal range in standard neuropsychological tests. However, they also stated that abnormalities may be present in more sophisticated testing of language and visuospatial functions. Thus, negative results do not negate the opinion regarding the critical functions of the cerebellum in language processing (17,18). Karacı et al. (19) found a significant difference between cerebellar stroke patients and controls, with patients demonstrating impaired understanding of simple orders and complex questions, reading comprehension, repeating, and picture and color naming.

When one considers linguistic disorders resulting from cerebellar lesions, there is a spectrum ranging from dysarthria and speech apraxia due to diminished motor coordination to impaired verbal fluency, problems in grammatic production, and, rarely, an aphasia syndrome triggered by a cerebellar pathology; the latter is much more complex and is closely associated with cognition (3). A different point of view, relating to the linguistic disorders induced by cerebellar lesions to frontal lobe executive functions, is also attractive, in which insufficient input in cerebellar-frontal projections is thought to lead to a failure in lexical-semantic operations and linguistic strategies (20). Silveri et al. (16) reported yet again the executive errors. The fact that standard aphasia tests are not helpful for disclosing a linguistic disorder and the subclinical course of the findings related to language usage suggest the hypothesis instituting a frontal lobe-associated problem (21).

The cerebellar topography should certainly be considered in linguistic disorders. The flocculonodular lobe, or the vestibulocerebellum, is associated with vestibular and reticular nuclei. Vermis and contiguous hemisphere portions are wholly called the spinocerebellum, which is associated with rubrospinal and corticospinal tracts. Finally, the posterolateral portions of cerebellar hemispheres are the neocerebellum (cerebro-cerebellum); axons originating from the dentate nucleus project to contralateral motor and pre-motor areas via the thalamus. Yet, this gross neuroanatomical differentiation is not adequate for veritable clinical assessments. As we mentioned above, lexical deficiencies have been shown in vermian lesions (15). Dysarthria is generally seen in superior and anterior vermis, as well as paravermis, lesions (3). Like the results of research addressing linguistic disorders, dysarthria was shown to be more frequent in right cerebellar hemispheric lesions (22). In one study, stroke patients with paravermian lesions scored lower with respect to total aphasia (18). Lastly, if the literature is reviewed in detail, it is clear that vermis and paravermis lesions are more closely linked to motor coordination of speech, and the neocerebellum that projects to supratentorial regions is related to

language processing. Tedesco et al. (23) reported that vascular topography and the involvement of deep cerebellar nuclei were the chief factors determining the cognitive profile.

Language Disorders and Thalamus

Another important structure receiving blood from the posterior system is the thalamus. Thalamic aphasias are well described in relation to linguistic disorders that arise from cerebellar lesions and thus are easily perceived by clinicians. The first case of thalamic aphasia was defined by Fisher in 1959 (24). An aphasia syndrome including reduction of speech output, anomia, and paraphasias but without repeating difficulty is attributed to the thalamus. A possible mechanism concerning the aphasias resulting from thalamic lesions is difficulty in accessing verbal memory due to affection of circuits involving the cerebral cortex, thalamus, and brain stem (25,26). A second possible mechanism is that the lesions of the mediotthalamic-frontocortical system give rise to affection in lexical selection processing, thereby causing anomia and semantic paraphasias (27). Word-finding difficulties and paraphasias were frequent in cases presented by Radanovic et al. (2); yet, these patients did not demonstrate a uniform aphasia syndrome. The authors related these variations to the following: the subcortical structures are closely linked to the cortico-striato-thalamo-cortical loops, and differences in lesion localizations alter the consequences on the cerebral cortex. Thus, the diaschisis mechanism in thalamic aphasias appears to be valid once again. De Witte et al. (28) reported an uncommon linguistic disorder due to right thalamic ischemic infarction in which cross-transcortical sensorial aphasia and neologistic jargon agraphia and alexia were present; the SPECT in this patient demonstrated right fronto-temporo-parieto-occipital hypoperfusion. A topographic correlation has also been defined to a considerable degree for thalamic aphasias. While anomia is generally associated with left dorsomedial thalamus lesions, lesions of the ventrolateral and ventral anterior regions lead to paraphasic disorders. Fluency deficits and comprehension defects reflect the thalamo-frontal disconnection caused by pulvinar and paramedian nuclei pathologies (29). Ozeren et al. (30,31) reported global aphasia with left thalamic hemorrhage and right putaminal hemorrhage, with rapid recovery.

Considering the linguistic deficits due to posterior system strokes, we allude to two specific syndromes. Pure alexia was first described by Dejerine in 1892. This clinical finding is seen in strokes that involve the left occipital cortex and extend to the splenium of the corpus callosum. On the other hand, alexia with agraphia points specifically to the posterior parietal lobe. It would not be wrong to mention that in pure alexia, a disconnection is present between the visual cortex and angular gyrus, so that visual-verbal unity is harmed (32). Marsh and Hillis (33) evidenced in a dependable manner with test batteries that such a linguistic deficit is not engendered by right hemineglect. Epelbaum et al. (34) demonstrated the cause of pure alexia neuroradiologically in a patient with surgical dam-

Table 1. Linguistic disorders of posterior system strokes

LOCALIZATION	LINGUISTIC DISORDER
CEREBELLUM	Dysarthria, anarthria
	Verbal fluency deficits
	Reading deficiencies
	Agrammatical speech
	Cerebellar lesion induced aphasia
THALAMUS	Reduction of speech output
	Anomia
	Paraphasia
OCCIPITAL LOBE	Pure alexia

age that cut the interrelation of the “visual word area” located on the left occipitotemporal sulcus and the occipital cortex; the connection between the two is the inferior longitudinal fasciculus, and injury to this pathway leads to a typical disconnection syndrome.

This brings us to an interesting syndrome brought about by occipital cortex damage. As known, the hearing-impaired utilize sign language for communication. A visuospatial modality is essential to transmit the semantic message in sign language. Saito et al. (34) presented sign language aphasia in a deaf signer after a left occipital infarction. In this case, both the production and comprehension of signs were impaired. Thus, the following could be attested: communication material of any sort can be formed by neural plasticity (voice, gesture, images, tactile signs, etc.); this material, in relation with associative regions, can find its consistent route in an appropriate (direct and easy) cortex area; and finally, damage to this and associative areas may give rise to “aphasia” syndromes, as it impairs the semantic and expressive components of the communication material. The lower frequency of linguistic problems in posterior system infarctions may not reflect a specific topography but perhaps the nature of our communication material. Yet, the human brain is such a complex organization that even involvement of the cerebellum in high cognitive functions is seen. Even the most archaic structures, at some point, can become effective on the most complicated functions, as an obvious consequence of neural plasticity.

Posterior system strokes may lead to clinical findings of cognitive deficits, including neurolinguistic components (Table 1). Certainly, determining these defects in stroke patients may precipitate changes in current management strategies. However, let us not forget, as Fisher stated so profoundly, that neurology is learned through stroke, and it evolves and advances with each new case. Reports of the early cases formed the basis of today’s new neuroanatomical-physiological hypotheses. In this sense, forthcoming reports regarding cognitive deficits in posterior strokes will guide our future steps in this ignored area of neurology.

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