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Thalamus and Language: What do we know from vascular and degenerative pathologies

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Abstract

Language is a complex cognitive task that is essential in our daily life. For decades, researchers have tried to understand the different role of cortical and subcortical areas in cerebral language representations and language processing. Language-related cortical zones are richly interconnected with other cortical regions (particularly via myelinated fibre tracts), but they also participate in subcortical feedback loops within the basal ganglia (caudate nucleus and putamen) and thalamus. The most relevant thalamic functions are the control and adaptation of cortico-cortical connectivity and bandwidth for information exchange. Despite having the knowledge of thalamic and basal ganglionic involvement in linguistic operations, the specific functions of these subcortical structures remain rather controversial. The aim of this study is to better understand the role of thalamus in language network, exploring the functional configuration of basal network components. The language specificity of subcortical supporting activity and the associated clinical features in thalamic involvement are also highlighted.

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Full Text

It is widely believed that neuropsychological functions are not simply localized in specific cortical areas but are rather subserved by a mosaic of neuronal structures,[1],[2],[3],[4] richly interconnected with other cortical regions, but participating in subcortical feedback loops within the basal ganglia [BG] (caudate nucleus and putamen) and the thalamus.[5],[6],[7],[8],[9],[10] Recently, an in-vivo tractography study demonstrated wide connections between the Broca's area and subcortical nuclei in human volunteers. In particular, two functionally distinct regions within the Broca's area, the anterior portion implicated in semantic processing and the posterior portion involved in phonology and syntax, have been shown to project to the anterior putamen.[11],[12]

Previous studies described the role of BG as strongly influencing the increase of the signal-to-noise ratio during language processing.[13],[14] This might be a type of enhancing signal for selected items and of suppression for the competing ones.[15],[16]

Cortico-thalamo-cortical circuitry has been extensively demonstrated.[17],[18],[19],[20],[21],[22] The most significant is the intimate relationship between the BG and the thalamus, in five parallel cortical-thalamic loops, namely, two motor loops (skeletomotor and occulomotor) and three non-motor loops. The non-motor loops include a dorsolateral prefrontal-cortex loop (DLPFC including the BG, thalamus and area 9–10), a lateral orbitofrontal loop (LPFC), and an anterior cingulate loop. The DLPFC loop is involved in executive functions, planning, and working memory. The LPFC mediates empathetic and socially appropriate responses. The anterior cingulate loop is involved in communicating reinforcement signals from the ventral tegmental area (VT) and the substantia nigra pars compacta, reinforcing voluntary engagement and volition.

Thalamic relay nuclei are now known to play an important role in changing the dynamics of cortical processing by setting up different oscillation patterns of frequency and synchrony.[23],[24],[25],[26],[27],[28],[29],[30],[31],[32] The "Selective Engagement Model"[33] supports the hypothesis that thalamic nuclei monitor the activity state of distributed cortical areas and control their functional connectivity via connections passing through the inferior thalamic peduncle. In the case of linguistic information, this primarily refers to frontal and temporoparietal cortices between which, for example, phonemic, lexical, and semantic information is exchanged during language perception and production. Corticothalamic language processing is complemented by the BG. However, because there is also a nonreciprocal corticothalamic input to each of these thalamic nuclei, the incoming basal ganglionic information from each circuit is mixed with cortical inputs from a different circuit. The information that the thalamic relay nuclei convey to the cortex is, therefore, not only affected by the parallel pathway through the BG structures but is also modified by the nonreciprocal corticothalamic pathway, resulting in an integrated feed-forward processing similar to that seen in sensory systems.[34]

In the specific context of language processing, the "Response–Release Semantic Feedback Model" claims thalamic and BG functions in language production,[35],[36] controlling the interaction between fronto-opercular and temporocortical cortices for the integration of lexico-syntactic with semantic information. BG are thought to coordinate the release of the provided language plan into speech.

Klostermann [33] referred to thalamus as entering in the "Declarative/Procedural Model," similar to mnemonic operations,[37],[38] and stated that BG provides the requirements to apply grammatical rules to linguistic raw data, the so-called "world knowledge" into lexical input signals.[33],[39]

Borrowed from the classical model of cortico-striato-thalamo-cortical motor processing,[8],[9] the "Lexical Selection Model" views the BG as a machinery to align word-related input to ongoing language plans. This is mainly conceived as a process in which an excess supply of lexical alternatives need to be monitored for unsuited candidate words to be inhibited from further processing. Only the remaining information will be signaled to the thalamus which then initiates frontocortical word release.[40]

What do we know from clinical cases?

Thalamic aphasia should be considered as 'Nessie', the Loch-Ness creature – nobody has seen her, but nobody can deny its existence.

Many different authors described thalamic aphasia as a consequence of thalamus involvement;[41],[42],[43],[44] however, it is not easy at all to relate a defined language disorder, devotedly related to a specific thalamic region of interest. Deficits have often been described in thalamic infarction; however, owing to a relatively complex and sometimes variant vascular supply,[45],[46],[47],[48],[49],[50] aphasic syndromes have been observed with different locations of damage; mostly, they do not occur in isolation but go along with other neuropsychological deficits, and are often accompanied by further extrathalamic lesions. Hence, the allocation of specific symptoms to particular nuclei is genuinely more difficult at this level than it is for cortical regions with their relatively precise functional description [Figure 1].[51]{Figure 1}

The arterial supply of the thalamus and midbrain consists of a complex arterial network involving the anterior and posterior cerebral circulation. The anterior and inferior midbrain and thalami are supplied mostly by the internal carotid artery, whereas the medial, lateral, and posterior territories are irrigated by the vertebrobasilar system. Four territories for thalamic vascularization have been identified [Figure 2]:{Figure 2}

Anterior, tuberothalamic, or polar territory (dorsomedian nucleus is usually spared): This territory, supplied by the

tuberothalamic artery, accounts for approximately 12–18% of all thalamic infarcts.[52],[53],[54],[55],[56],[57] The most frequent etiology is cardioembolismParamedian territory supplied by the paramedian arteries accounts for approximately 22–35% of all thalamic infarcts.[57] The most frequent etiology is cardioembolism [48]Inferolateral or thalamogeniculate territory. This territory, supplied by the thalamogeniculate arteries, accounts for approximately 45–50% of all thalamic infarcts. The most frequent etiology is microangiopathyPosterior choroidal territory (pulvinar nucleus): This territory, supplied by the medial and lateral branches of the posterior choroidal arteries, accounts for approximately 7–9% of all thalamic infarcts. The most frequent etiology is microangiopathy.

Bogousslavsky and Carrera,[47],[48],[49] therefore, divided the retrospective case studies of Lausanne into four patterns of clinical presentation – the anterior, paramedian, inferolateral, and posterior syndromes. Clinical practice, however, can often result in mixed-cases, probably due to the occurrence of border zone infarcts or due to variations in the "classical" distribution of the thalamic arteries.[58],[59]

What is astonishing regarding the definition proposed by Bogousslavsky and Carrera [47],[48],[49] is that it relies not only on the vascular supply but also on the regional interconnection: anterior nuclei are the relay centers of the mamillothalamic tract (MTT) in the thalamus. They are reciprocally connected to the anterior limbic system, cingulate gyrus, hippocampus, parahippocampal formation, entorhinal cortex, retrosplenium, and orbitofrontal cortices.[60] Other connections to the medial prefrontal cortices, posterior area of the neocortex, and ventral pallidum have been described.[45],[46]

To be precise, language is involved in all the thalamic lesions, more precisely in those occurring in the anterior and paramedian regions. On the contrary, the inferolateral or thalamogeniculate and the posterior infarcts result in more complex neuropsychological (but not language or speech) alterations.

Therefore, infarcts in the anterior territory frequently lead to a perseverative pattern of speech with inappropriate maintenance of the category in all memory and executive tasks, with increased sensitivity to interferences.[48] Many patients show a superimposition and "telescoping" of unrelated information with parallel expression of mental activities called palipsychism from Greek palin (again) and psychê (soul).[61] The output speech after an anterior territory infarct is characterized by grammatically correct phrases, but unpredictable topic shifts, which are usually intrusions on previous topics (introduced by the patient or the examiner); correct phrases are often strung together in a nonsensical or illogical fashion and punctuated by paraphasias or neologism.[62],[63] Perseverations were also found [Figure 3].{Figure 3}

Some patients show a spectacular pattern of speech with "impersistance de la pensée," which may be reminiscent of the "psychotic" speech found in schizophrenics. These speech disturbances are mainly found in the unconstrained speech with relative maintenance of the performance of an automatic series or in reciting poems known by heart.

Single-photon emission computed tomography (SPECT) images show a diffuse hypoperfusion of the left hemisphere, especially prominent in the medial and lateral frontal lobes. Bogousslavsky and Carrera [48],[49] have inferred that these behavioral changes with perseverations and frequent topic shifts in output speech result from a disconnection of the ipsilateral cortex, mainly the medial and lateral frontal regions, which seems to be coincident with what we have previously described.[64],[65],[66],[67],[68],[69],[70]

Aphasia was never found to be the main neuropsychological dysfunction after anterior nuclei infarction, and, when present, it is usually consistent with a moderate form of transcortical motor aphasia associated with hypophonia and dysarthria.[61] Aphasia usually occurs with left or bilateral infarcts, although Bogousslavsky et al.,[53] found four patients (two left-handed and two right-handed) with an infarct limited to the right anterior territory, and whose principal clinical manifestation was transcortical motor aphasia. Spontaneous speech is usually reduced, with a lack of speech initiation and the usage of short sentences. Occasional phonemic paraphasias are found ("anassin"/"assassin") as well as semantic paraphasias ("clock"/"watch") or neologisms. Repetition and comprehension are rarely impaired. Phonemic paralexias as well as calculation and writing impairment are rarely found.[71]

Numerous intrusions, perseverations, and false recognition are frequently found. Intrusions during the recall of a word list (provoked confabulations) are more frequent than during spontaneous speech (spontaneous confabulations).[48],[49],[61],[72],[73],[74]

Paramedian nuclei infarcts produced (in physically and emotionally active patients before the occurrence of stroke) a severe apathy; the patients become indifferent as if they have lost motor and affect drive, especially after bilateral lesions,[75] needing constant external programming, which makes them look like robots. The term, "loss of psychic self-activation," has been proposed to describe this peculiar behavioural pattern.[76],[77]

Akinetic mutism is considered when the patients, who appear to be awake and follow the examiner's eyes, fail to respond or become active after a relevant stimuli has been given. The location of the thalamic lesion is quite similar in patients who present with a loss of self-activation and in those with akinetic mutism. However, the involvement of intralaminar nuclei, especially the centromedian and parafascicular nuclei, may be more extensive in patients presenting with akinetic mutism than in those with "psychic loss of self-activation."[78]

To make things even much more complicated in clinical practice, another variant requires a merger with these thalamic disorders of speech: the Percheron' artery supply, an anatomical variant of the paramedian artery supply,[79],[80],[81] that does not have a bilateral supply but is derived from a single thalamic perforating artery, arises from the proximal posterior cerebral artery (PCA), between the basilar artery and the posterior communicating artery. It supplies the rostral mesencephalon and both paramedian thalami. If there is an occlusion of the Percheron's artery, bilateral paramedian thalamic infarction occurs, and the consequences, as mentioned above, are dramatic.[82],[83]

Inferolateral or thalamogeniculate territories damage, involving the major part of the ventral posterior nuclei as well as the ventral and lateral parts of the ventrolateral nucleus more rostrally, are the most common (45%) disorders, and lead to the thalamic pain syndrome, as described by Dejerine and Roussy, or a contralateral ataxia and hypesthesia [Figure 2].[84],[85]

The posterior choroidal arteries (arising from the posterior cerebral artery) supply the subthalamic nucleus and midbrain, the medial half of the medial geniculate nucleus, the posterior parts of the intralaminar nuclei and pulvinar nuclei, and usually produce neglect, apathy, and impaired comprehension.[79],[86],[87],[88]

Thalamic nuclei and the supplementary motor region (SMR)[89],[90] have been proposed to form the fifth functional area, a functional unit in language processing. The impairment of these interconnecitons often leads to a transcortical sensory aphasia. Further, it should be mentioned that in cases with extended lesions confined to the thalamus, permanent global aphasia has been observed [Figure 3].[91],[92]

Apart from the work on language reported on patients who have developed ischemia in various parts of the brain, more recent data has been derived from in-vivo imaging studies on normal subjects and in subjects with other neurological disorders. A left thalamic activation has been found during the differentiation of distinct speech sounds, alongside with activity in the planum temporale, the superior temporal and Heschl's gyri of the dominant hemisphere.[93]

Moreover, the medial geniculate body (thalamic auditory nucleus) has been shown to be active during the recognition of speech sounds. A cortical feedback loop has been found to be involved, which was found impaired in dyslexic persons.[94],[95] Further, thalamus, specifically in the dorsomedial and pulvinar regions, as well as with frontotemporal involvement, has been seen to be activated during lexico-semantic operations and object recall.[96],[97],[98],[99]

Moreover, a chronometrical sequence of activations has been established during the performance of respective tasks. The dorsomedial thalamus and presupplementary motor areas are engaged in the concept formation of perceived vocal signals, whereas the subsequent pulvinar activity reflects the downstream processes for the semantic alignment of this activity with the ongoing input.[100],[101],[102]

Moreover, Anastasia et al., described two functionally distinct regions within the previously imagined unique area, the Broca's area, whose anterior portion was activated during the semantic processing, and the posterior portion could be activated during the phonology and syntax processing; both these areas project to the anterior putamen and are involved in language processing.[18],[103] In addition to studies on the Broca's area, new information has been reported by Ardila et al., with regard to the peripheral zone around the Wernicke's area involved in language associations, particularly the areas BA20, BA37, BA38, BA39, and BA40. These are considered as a part of the "extended Wernicke's area" that projects diffusely to the thalamic nuclei, in particular in the putamen.

[104],[105],[106]

Conclusions

The control and adaptation of cortico-cortical connectivity and providing the bandwidth for information exchange have been shown to be the most relevant thalamic functions. Three properties of thalamocortical neurons are known:[33] first, the presence of local, and remote feed-forward and feedback connections with almost every cortical region are prerequisites for establishing flexible network constellations; second, the ability to convey information in distinct discharge modes is essential for regulating the likelihood with which messages are passed from one cortical region to another; third, a sequential circuitry of thalamocortical information allows the adaptation of the final messages in an iterative process involving various downstream relay nuclei.

Thalamic neurons notify that a specific area has become active so that a functionally related regions can be engaged. In turn, thalamic neurons will receive the output message from the activated downstream cortex. How this iterative process should be adapted to the ongoing demands, particularly in the context of rapidly changing cognitive operations, as required for language processing, is the subject of further research.

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Conflicts of interest

There are no conflicts of interest.

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