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THALAMUS AND LANGUAGE

Interface with attention, memory and executive functions

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ABSTRACT - Subcortical structures are in a strategic functional position within the cognitive networks. Their lesion can interfere with a great number of functions. We studied six patients with thalamic vascular lesions (three left sided, two right sided and one bilateral), to characterize their repercussion in the communicative abilities and the interface between language alterations and other cognitive abilities, as attention, memory and frontal executive. All patients were evaluated through a functional interview (discourse analysis), and the following batteries: Boston Diagnostic Aphasia Examination, Boston Naming Test, Token Test, Benton Visual Retention Test, Trail Making, Wisconsin Card Sorting and frontal scripts. All patients performed MRI and five underwent SPECT. Results show that these patients present impairment in several cognitive domains, especially attention and executive functions (working memory, planning and self-monitoring); those with right lesions have an additional visuospatial impairment. Such alterations interfere with language abilities, and this fact must be considered in the rehabilitation efforts.

KEY WORDS: thalamus, language, attention, memory, prefrontal cortex.

Tálamo e linguagem: interface com atenção, memória e funções executivas

RESUMO - As estruturas subcorticais ocupam posições funcionais estratégicas nas redes cognitivas. Sua lesão pode interferir com grande número de funções. Estudamos seis pacientes com lesão vascular talâmica (três à esqueda, dois à direita e um bilateral), para caracterizar a repercussão da lesão nas suas habilidades comunicativas e a interface entre alterações de linguagem e outras habilidades cognitivas, como atenção, memória e executivas frontais. Os pacientes foram avaliados através de entrevista funcional (análise do discurso), testes de Boston para Diagnóstico da Afasia, Nomeação Boston, Token, Múltipla Escolha de Benton, Trail Making, Wisconsin Card Sorting e *scripts* frontais. Todos os pacientes realizaram ressonância magnética encefálica (RM) e cinco realizaram SPECT. Os resultados mostram que os pacientes apresentam prejuízo em diversos domínios cognitivos, especialmente atenção e funções executivas (memória operacional, planejamento e automonitoração); nos lesados à direita, ocorre prejuízo visuoespacial associado. Tais alterações repercutem nas habilidades de linguagem, o que deve ser levado em conta para reabilitação.

PALAVRAS-CHAVE: tálamo, linguagem, atenção, memória, córtex pré-frontal.

The study of the role of subcortical structures in cognitive processes, largely impelled by neuroimaging development has brought interest in the frontostriatal circuits and the thalamus, and their participation in the linguistic processing. Fisher, in 1959[1], was the first author to describe aphasia in left thalamic hemorrhagic lesions. Aphasia with decreased speech output (although fluent), anomia, paraphasias (that could deteriorate into jargon), comprehension less affected than production, and no repetition problems (or minimum) are described following ventrolateral (VL) and ventral anterior (VA) nuclei lesions; a fluent aphasia, with non-words, may occur especially in lesions of the pulvinar and the posterior lateral (PL) nucleus[2]. Left dorsomedial (DM) lesions are related to anomia, verbal memory deficits and a frontal syndrome[3,4]. Thalamic aphasia is more frequently found in lesions of the dominant tuberothalamic

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(polar) and interpeduncular profunda (paramedian thalamic) arteries, and since they are small vessels, there is a low probability that some associated cortical dysfunction may exist[5]. Thalamic lesions may provoke an association of disorders: disfluency and conditions similar to transcortical aphasia in lesions involving the left VL, pulvinar and paramedian nuclei[6,7] or leading to a disconnection between the frontal area – VA; arousal disturbances (intrusions, contaminations, non-words); comprehension alterations, because of interruption in thalamus - frontal area - superior longitudinal fascicle - Wernicke's area connections; hypophonia, by interruption of pallidonigral afferents to the thalamus[8].

The attempts to explain the mechanisms by which thalamic lesions lead to language disturbances are based upon theories such as cortical activation and integration, modulation and monitoration of the formulated language[9-12]. Some authors hypothesized about the integrative circuits involving the cerebral cortex, the thalamus and the brainstem, especially the ascending reticular activating system, with the participation of verbal memory[13-15]. Cappa and Vignolo[16] attributed the handling of words as semantic units to the thalamus, excluding its participation in phonemic processes, which could explain the few repetition disturbances in subjects with thalamic aphasia. Nadeau and Crosson[17] suggested that the mediothalamic-frontocortical system (including the frontal lobes – inferior thalamic peduncle – nucleus reticularis and centromedian nucleus) could participate in the control of lexical selection processes, its failure leading to a loss of any difference in the activation among a certain lexical item and others semantically related, thus resulting in anomia and semantic paraphasias.

The interaction between language and other cognitive domains is an important factor to be considered, especially in lesions affecting structures that have extensive reciprocal connections with the cerebral cortex, such as the thalamic nuclei. Based on a preliminary study[18], we noted a great interference of factors such as memory and attention in the linguistic performance of patients with thalamic lesions. Damage of short-term memory (phonological loop) can be expressed clinically by sentence comprehension deficits[19], either due to the absence of a phonological memory and/or semantic or syntactic memory. Likewise, the short-term memory deficit may impair sentence planning, leading to verbal production and vocabulary acquisition disturbances[20]. Primary attentional disorders can also disturb linguistic processing. Attention deficits can occur in frontal lesions, and in these, we frequently find verbal fluency alterations, compromising speech initiative in left hemisphere lesions, and of pragmatic aspects of speech in right hemisphere lesions[21]. Neglect dyslexia also was described following thalamic lesions[17].

The aphasias caused by subcortical lesion usually escape the possibility of classification and the use of linguistic tests alone may not be sufficient to identify the language disturbance, which can lead to therapeutic inefficacy. Our purpose in this study was to describe the profile of language disturbances found after right and left thalamic lesions, and to verify the interrelation between these findings and other cognitive impairments, such as attention, memory and executive functions.

METHOD

We studied six patients with vascular thalamic lesions; two patients had right-sided lesions, three had left-sided lesions and in one there were bilateral lesions. All patients were right-handed and Portuguese native speakers. The selection of cases was based on anatomical features (site of lesion in neuroimaging performed when the patient was admitted in the hospital). Patient 6, although presenting bilateral lesion, was included when he suffered a right thalamic stroke, and began to demonstrate the described clinical picture. Exclusion criteria were: delay in language acquisition, antecedents of psychiatric and/or neurological disease, alcoholism, hearing deficiency, associated cortical involvement (based on clinical history and neurological examination). Demographic data are shown in Table 1; neuroimaging features are shown in Table 1 and Figure 1.

All the participants of this study signed an agreement form that was included in the project approved by the Commission of Ethics for Analysis of Research Projects - Hospital das Clínicas – FMUSP.

Instruments

The language evaluation was performed using a functional interview (narrative, argumentative and procedural discourses), the Boston Diagnostic Aphasia Examination (including the Boston Naming Test)[22] and the Token Test[23]. These tests evaluate the phonological, morphological, syntactic and semantic aspects that are present in the processes of language comprehension (word discrimination, commands and text interpretation) and language production (naming, repetition of words and phrases, text production). They take into account the effects related to the modalities of stimuli input and answer output and also those task-specific, in order to control linguistic and non-linguistic variables. The Brazilian reference values to BDAE and Token Test were based on previous studies conducted by the authors[24,25].

An additional test of frontal scripts, translated into Portuguese from Allain's et al. original publication[26], was applied in four patients (two with left lesion and two with

Table 1. Demographic and neuroimaging data.

Patient	Age (yrs) / Gender	Schooling (yrs)	Time interval stroke-evaluation	MRI	Arterial territory	SPECT
1	64 / F	4	6 months	left thalamic infarction	interpeduncular	normal
2	22 / F	11	4 years	left thalamic infarction extending to posterior internal capsule and *globus pallidus*	anterior choroidal / interpeduncular profunda / tuberothalamic	not performed
3	27 / M	8	4 years	left thalamic hemorrhage	total	left thalamus, caudate nucleus and temporo-parieto-occipital hypoperfusion
4	65 / M	4	9 years	right thalamic infarction extending to posterior internal capsule	anterior choroidal	right thalamus and left posterior parietal hypoperfusion
5	49 / M	0	3 months	right thalamic hemorrhage	anterior choroidal / tuberothalamic	right thalamus and diffuse hemispheric hypoperfusion
6	65 / M	5	3 years	right thalamic hemorrhage (with a previous left thalamic lesion)	bilateral geniculothalamic	normal

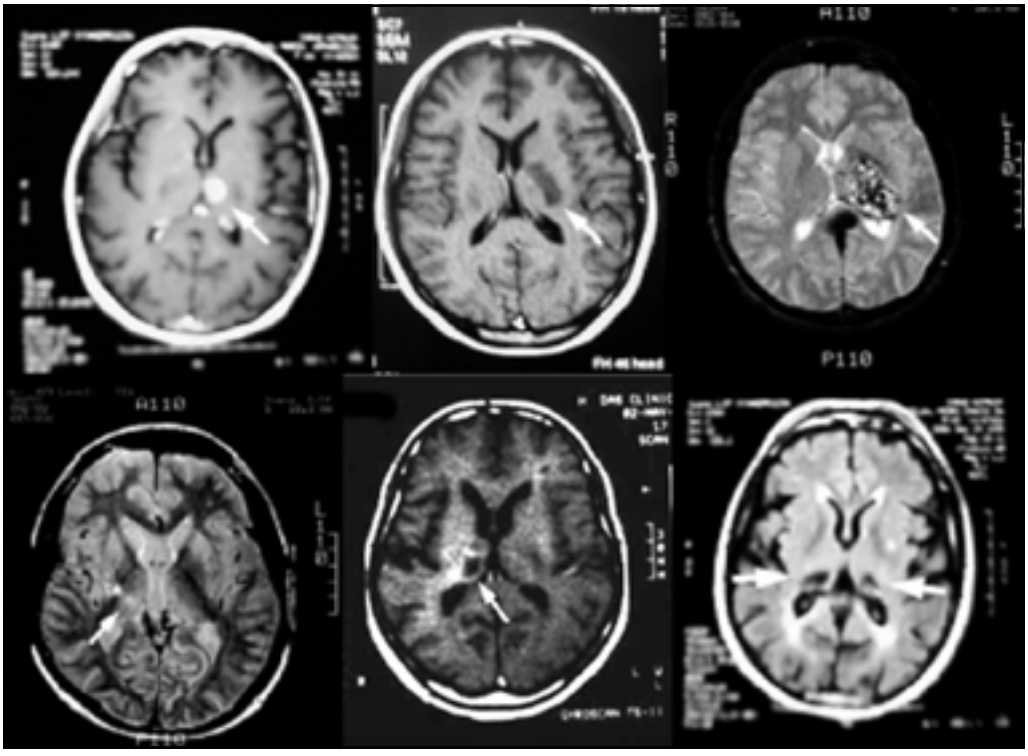

Fig 1. MRI findings in Patients 1 to 6. The arrows indicate the lesions described in Table 1.

Table 2. Performance of patients in the Boston Diagnostic Aphasia Examination.

	Patient					
Subtest	1	2	3	4	5	6
	Left lesion				Right lesion	
Oral comprehension						
Word Discrimination	61	72	65	72	62	62
Commands	9	10	6	11	10	10
Complex Ideational Material	6	10	4	12	10	7
Repetition						
Words	10	10	7	10	10	10
High-Probability Phrases	8	8	1	7	7	6
Low-Probability Phrases	7	8	0	7	6	6
Naming						
Responsive	26	27	22	27	26	25
Visual Confrontation	83	111	87	106	93	95
Animal Fluency	3	13	8	21	8	14
BNT	28	45	14	34	33	26
Reading comprehension						
Word-Picture Matching	10	9	1	10	NA	8
Sentences and Paragraphs	4	10	4	10	NA	8
Writing						
Written Confrontation Naming	8	10	8	10	NA	10
Narrative	4	5	2	5	NA	3
Sentences to Dictation	10	12	9	12	NA	9

NA, not applicable.

Table 3 . Performance of patients in the Token Test.

	Patient					
Part	1	2	3	4	5	6
	Left lesion				Right lesion	
1	10	10	9	10	8	10
2	4	6	6	6	4	6
3	10	10	10	9	9	9
4	2	10	4	9	3	5
5	8	21	8	14	8	10

right lesion) and nine normal individuals, for comparison (normal subjects had an average of 2.6 ordination mistakes and no intrusions). This task investigates the abilities of information recovery using common action sequences and it is sensitive to frontal dysfunction. The subjects are asked to establish the hierarchical organization of six scripts (each one containing from 10 to 17 actions) obeying to the same temporal sequence in which they are usually executed. The actions of each script are presented in mixed cards that must be organized by the subject. The title of each script is presented in a separated card, remaining accessible during the task execution. In the three last scripts some distractors elements are presented, containing actions that do not belong to the general plan and that must be excluded. The ordination and intrusion mistakes are, then, counted. Six scripts were applied in our patients ("Going to the supermarket", "Making a cake", "Going to the movies", "Going to a wedding", "Going to a restaurant", "Taking the subway"), totalizing 76 actions. Patient 3, who had moderate comprehension deficit, and Patient 4, who was illiterate, could not perform this test. The language results are shown in Tables 2 and 3.

Attention, memory and executive functions were evaluated through the Benton Visual Retention (BVRT), Trail Making and Wisconsin Card Sorting (WCST) tests. The BVRT consists of several series of line drawings that must be reproduced by the patient, and it was designed to assess visual perception and short-term visual memory[27]. The Trail Making test measures visual skills (searching and sequencing) and the ability to make alternating conceptual shifts, as it requires the patient to draw a line connecting 25 circles in a certain order, as rapidly and accurately as possible[28]. The WCST assesses abstract ability, conceptual set shifting and the capacity of "learning", as the subject is asked to sort a series of printed cards according to criteria that are changed without previous notice (from colors to forms, numbers, etc)[29]. In the neuropsychological tests, we adopted a performance of 90% as a cut-off score for normality, because there are no standardized values for the Brazilian population, especially low-educated. All patients were able to comprehend the instructions for test accomplishment. The clinical evaluations were performed within a minimum interval of six weeks after symptom installation. All patients performed SPECT and cranial MRI, after a minimum interval of two months from onset of symptoms.

The patients were tested by a speech therapist and a neuropsychologist, both experienced, according to the procedures recommended by Lezak[30,31]. Their language and neurological evaluation were reviewed and analyzed by two authors (respectively LLM and MR).

RESULTS

Language: Five patients persistently complained of memory and word finding difficulties that interfered in their daily activities (exception: Patient 5).

Left lesions

Patient 1: This patient presented moderate comprehension deficit in more complex linguistics tasks (Complex Ideational Material and Reading Sentences and Paragraphs in the BDAE and especially in the fourth and fifth blocks in the Token Test), semantic paraphasias and anomia. This patient had difficulties in changing semantic categories and sequences in formal tests (from letters to numbers, days of the week to months), showing perseverations; her performance in the animal fluency was reduced. In the scripts, she had two ordination mistakes and no intrusions. During the examination, she often complained that she could not remember the instructions or that she forgot the original stimulus while searching for the correct answer. Besides, she was disorganized and inattentive during the search for answers in reading tasks, having difficulties to choose just one answer.

Patient 2 had a normal performance in the formal tests and made no mistakes in the frontal scripts.

Patient 3 showed moderate to severe comprehension, repetition and naming deficits, with phonemic paraphasias. His reading and writing were also defective.

Right lesions

Patient 4 had interpretation difficulties, emphasizing descriptive aspects in the Cookie Theft narrative. In the scripts he had 20 ordination mistakes and no intrusions.

Patient 5 had a slight anomia for visual presented

Table 4. Neuropsychological data.

	Patient					
Test	1	2	3	4	5	6
	Left lesion			Right lesion		
Trail Making						
Part A (degree of impairment)	severe	severe	severe	severe	incapable	severe
Part B (degree of impairment)	severe	severe	incapable	incapable	incapable	severe
Wisconsin Card Sorting						
right answers	41	51	16	30	24	17
categories	1	3	0	1	1	0
perseverative answers	19	4	52	40	54	46
Benton Visual Retention						
memory score	13	27	22	20	10	15
(degree of impairment)	(severe)	(moderate	(moderate)	(moderate)	(severe)	(severe)
visual discrimination score	25	32	28	20	15	20
(degree of impairment)	(moderate)	(normal)	(normal)	(moderate)	(severe)	(moderate)

stimuli, bad performance in all blocks of the Token test and severe difficulties in the integration of the several set elements in the Cookie Theft picture. Although these impairments are difficult to interpret (the patient is illiterate), we consider them as relevant because the patient had subjective complaints in these specific skills, and we could observe anomia in spontaneous speech.

Patient 6: This patient showed scores below normal for repetition, related to sentence extension, and Visual Confrontation Naming, as well as deficits in narrative organization, with digressions and perseverations. In the Token test, he showed bad performance in the fourth and fifth blocks. In the scripts, he had 21 ordination mistakes and 4 intrusions.

Neuropsychological examination: All six patients had performances below normal in the attention and memory tests. All of them, except case two, presented performance below normal in tests of executive functions. The group with right lesions had worse performance in the visual discrimination test. The results are exposed in Table 4.

DISCUSSION

The interaction between language and other cognitive functions is receiving greater attention by researchers in Neuropsychology. Subcortical lesions can interfere with cortical networks in several ways, due to the complexity of cortico-striato-thalamic-cortical loops and also because small changes in the lesion sites can completely change the connections affected and their functional repercussion on the cerebral cortex (through deafferentation mechanisms)[4,12,32,33]. Language alterations in subcortical lesions are often mild, although the patients persistently complain of naming difficulties or that "the thoughts are confused". These deficits are rarely as disabling as those found in cortical lesions (unless the patient has some professional activity which requires a higher level of cognitive/verbal abilities: academic students, lawyers), but they have a significant impact in the subjects' quality of life. We will try to describe the relation between language alterations and the attention, memory and executive function disorders found in our patients.

The comprehension deficits in thalamic aphasias are mild to moderate, and the written comprehension tends to be more preserved than the oral comprehension[16,34]. Verbal memory alterations can contribute to the low scores in these tasks[35]. The concomitant attention and memory deficits found in cases 1 and 3 suggest that these functions can be implied in the comprehension impairment, as we note a worsening of performance related to the stimulus extension and syntactic complexity (both processes are linked to working memory capacity). Patient 1 also presented some findings that suggest an additional deficit of attention, mnesic and planning/monitoring abilities such as impairment in changing categories and sequences (from days of the week to months, for example) with perseverations, reduction in animal fluency, difficulties in scanning strategies and concentration. Furthermore, her complaint that she could not remember the task or the stimuli presented also point to attention and working memory deficits. It is interesting to notice that Patient 2, who did not present any dysfunction in formal tests, had attention and memory alterations, but not of executive dysfunction. Although she performed well, she showed a great delay in answers. This patient complained that she could not talk and write as she did before (especially names), and that "when I read anything long, I do not remember the beginning", being unable to attend her college courses. Her complaint supports the existence of a working memory alteration.

Repetition disturbances were found in Patient 3 and in Patient 6, who had bilateral lesions. It is our impression that attention and working memory dysfunction can also explain these repetition alterations, if we consider that there is a correlation between the decline of repetition capacity and increases in sentence length. Perseverations were found in patients 1 and 6. Their existence in thalamic aphasia is considered as related to attention and vigilance mechanisms[11,15], and may be present in VA lesions[8].

Paraphasias were found in Patients 1 and 3. Patient 1 had semantic paraphasias, especially in naming tests, where she benefited from phonemic cues. Patient 3 showed phonemic paraphasias. These patients also presented anomia. These findings sustain the most consistent theoretical model about the thalamus role as a "floodgate", responsible for the best lexical selection[12,17], with the participation of attention and working memory[36]. A category-specific anomia[37] and proper name anomia[38] were already found after dominant thalamic lesions. The difficulties evidenced in the Token Test also point to a working memory deficit, as they become more evident in those blocks which demand larger mnesic span and the analysis of more extensive sentences.

Patients with right lesions were different from those with left lesions, as a group, as they had impair-

ment in visuospatial, discursive and script tasks. In Patient 5, the visuospatial alteration led to low scores in Visual Confrontation Naming and in the Token Test associated to an inability to adequately distinguish the elements in the Cookie Theft picture. These impairments are difficult to interpret because this patient is illiterate, and there are not reference values for Brazilian illiterates in most neuropsychological tests. In patient 6, the Visual Confrontation Naming task was impaired too. These patients with right thalamic damage were those who had the worst performances in the Benton visual discrimination test.

The ability to elaborate a discourse macrostructure and to integrate information in a context are impaired in patients with right hemisphere lesions, who present difficulties in extracting sentence meaning, discarding irrelevant information and integrating the apprehended meaning within a coherent whole, in order to generate an organized narrative. Contrarily, they have a tendency to fix on individual, isolated and/or less important elements, without performing a connection between them to form a whole[39]. The coherent discourse comprehension, in MRI studies, provokes larger activation in the right frontal lobe than the reading of non-related sentences[40].

The impact of the executive system dysfunction in language is a complex issue. Alexander et al., in 1989[21], tried to correlate language deficits and frontal lesions. They considered four levels in their theoretic model: motor, linguistic-cognitive, activation and formulation, the latter being the most important in our discussion. Thus, in left frontal lesions (especially prefrontal and those in the posterior lateral convexity), the formulation impairment reflects an inability in language structuration and organization (when the subject tries to deal with elements that are more complex or abstract) and of conceptual association. In the right frontal lesions (also prefrontal or of its projections to the posterior convexity), formulation difficulties are more contextual, as goal selection and self-monitoring, leading to tangenciality, inadequate topic changes, improper discourse and confabulation. Frontal symptoms like concrete thinking, deficiency in behavior planning and monitoring, perseveration, apathy and impairment in conceptualization can certainly compromise the discursive ability, generating problems with its macrostructure, organization, cohesion, and inferences as those found in our cases. The attention and working memory deficits can also be inserted in the context of frontal alterations.

An interesting finding was the difference in performance between individuals with left and right lesions in the frontal scripts. According to the cognitive theory, scripts are a class of organized structures that are stored as knowledge nets representing events, stories and frequent activities[41]. Thus, the frontal dysexecutive syndrome also includes impairment in the temporal and sequential processing of this preexisting mental plan[42], although the exact nature of this storage process is still unknown. Studies with functional MRI showed bilateral prefrontal dorsolateral activation in tasks involving frontal scripts, thus evidencing the participation of these areas in the analysis of action sequences[43].

Most errors in our patients occurred in the actions ordination, and not in the choice of which actions belonged to the scripts. This fact puts into evidence a specific inability in performing the temporal-sequential ordination discussed above. An intriguing fact is the preponderance of this dysfunction in patients with right lesion, since most studies point to the participation of bilateral prefrontal cortices in this task. We can speculate that the great demand for spatial movement in most scripts (going to the supermarket, taking the subway) generated an additional disadvantage for these patients. Patient 2, that did not present any mistakes, also did not have executive dysfunction in WCST. Disorders of thought that are independent of primary language disturbances were described in dominant thalamic lesions, and seem to reflect a primary disorganization that can rely on frontal executive mechanisms[44].

Many authors have proposed that the pathophysiological mechanisms underlying subcortical aphasia (and other cognitive symptoms) are associated with cerebral blood flow and metabolism alterations[45-48]. These abnormalities reflect the remote consequences of focal damage (diaschisis of the ipsilesional cortex) and it may explain why these changes are sometimes transient and their improvement follows the pattern of the cortical reperfusion[49,50]. Considering that the anterior and medial thalamus project to the frontal cortex and the lateral thalamus projects to posterior cortex[47], most thalamic lesions will lead to some cortical involvement in associative areas, and thus provoke cognitive symptoms.

The greatest difficulty we found in our study was to correlate the clinical data with the SPECT findings. Hypoperfusion patterns were heterogeneous even when the patients had similar clinical pictures. Compensatory mechanisms may account for these discrepancies[46]. Two patients that showed normal perfu-

sion studies (Patients 1 and 6) were symptomatic. Patient 2, whose results in tests were normal, refused to undergo the procedure. In case 3, there was a relative overlapping between the hypoperfusion pattern (left posterior) and his clinical picture; this patient presented the largest lesion, that affected all the thalamus and extended to the adjacent posterior periventricular white matter, suggesting a more severe deafferentation mechanism. In Patient 5, there was diffuse hypoperfusion in the right hemisphere, making any detailed analysis impossible. In Patient 4, there was an apparent contralateral functional repercussion (left posterior parietal), but of little clinical repercussion. We believe that the small number of cases is the main reason for these inconclusive results.

The study of subcortical-induced language alterations provides very complex and diverse types of clinical syndromes, which helps us to understand how other cognitive processes interact with language and vice-versa. Based on our results, we agree with the interpretation that the causes for a loss of efficiency in the language use caused by thalamic lesions are multifactorial and transcend the boundaries of linguistic processing itself[7,11,13-15]. Lesions in both hemispheres can generate dysfunctions that, although different in nature, become disabling. From an anatomical view, the amnesic syndrome (resembling that caused by the temporal medial lesion) appears more specifically after lesions of the mammillo-thalamic tract (MTT); different memory deficit patterns can occur after lesions of other thalamic structures, even when the MTT is preserved. The dysexecutive syndrome can be found after lesions in DM, midline, intralaminar (IL) or any combination of these nuclei[51]. As our patients had widespread lesions, frequently in more than one arterial territory, and taking into account the complexity and diversity of the thalamic vascularization, is not possible for us to make precise anatomical correlations. We can speculate that MTT, IL and the ventral portion of DM are damaged in Patients 1 and 2, in the latter possibly existing a VA lesion; in Patients 4, 5 and 6, the posterior lesions predominate, probably in pulvinar, PL, posterior portion of DM and lateral geniculate. Patient 3 had a total thalamic lesion.

In many cases, the alterations objectively found in language batteries can be slight and disproportionate to the patient's complaints. Executive, attention, mnesic and visuospatial dysfunctions, caused by the thalamus insertion in the respective cognitive networks, are elements that contribute to language deficits, and must be adequately evaluated and measured to improve the efficiency of therapeutic approaches.

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