

Thalamic aphasia

A conceptual critique

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Summary. Four patients with aphasia due to small circumscribed thalamic lesions are presented. A review of the literature on thalamic aphasia revealed 16 similar cases. While the general consensus that only left-sided thalamic lesions are associated with aphasia is confirmed, analysis of the sites of the thalamic infarctions and the dysphasia elements did not reveal an unequivocal correlation. The explanation of this finding is that (a) disruption of any circuit, whether taking place in the connections or in the nuclei, leads to dysfunction and (b) thalamofrontal connections are not topographically arranged according to the thalamic nuclei, but show a frontal rostro-caudal/thalamic mediolateral interrelationship irrespective of thalamic nuclear masses.

Key word: Thalamic aphasia

Introduction

The use of the CT in clinical practice has established that thalamic lesions of whatever nature may form the structural basis of speech dysfunction [1, 2, 4, 5, 8, 9, 12, 16, 18, 21–30,

33]. This fact has been noted in stereotaxic thalamotomy for parkinsonism [3, 13, 31], as well as in clinical reports [6, 7, 10, 11, 14, 20, 32]. The question whether the observed speech defect is attributable to disruption of the cortico-thalamo-cortical connections or to a lesion in one or more of the thalamic nuclei proper has been the subject of clinical-anatomical correlation studies [1, 12, 24, 29]. Given the intricate composition of the thalamus, in which nuclei and fibre systems are interwoven to such a complex degree that any lesion other than a selective neurotoxic or neurodegenerative one will involve both the fibre systems and the neuronal elements, this question is probably superfluous. Because language implies the activity of a cognitive semantic system, whereas speech is more an instrumental function, the present analysis of four cases with small and well-circumscribed lesions aimed to examine the relationship between speech dysfunction and small thalamic lesions.

The results argue that speech dysfunction cannot be unequivocally tied to involvement of specific thalamic nuclei and underline from a clinical point of view the relevance of the anatomical data provided by Kievit and Kuypers [15, 17], who, by means of horseradish peroxidase injection in different parts of the frontal cortex in the rhesus monkey, showed that the

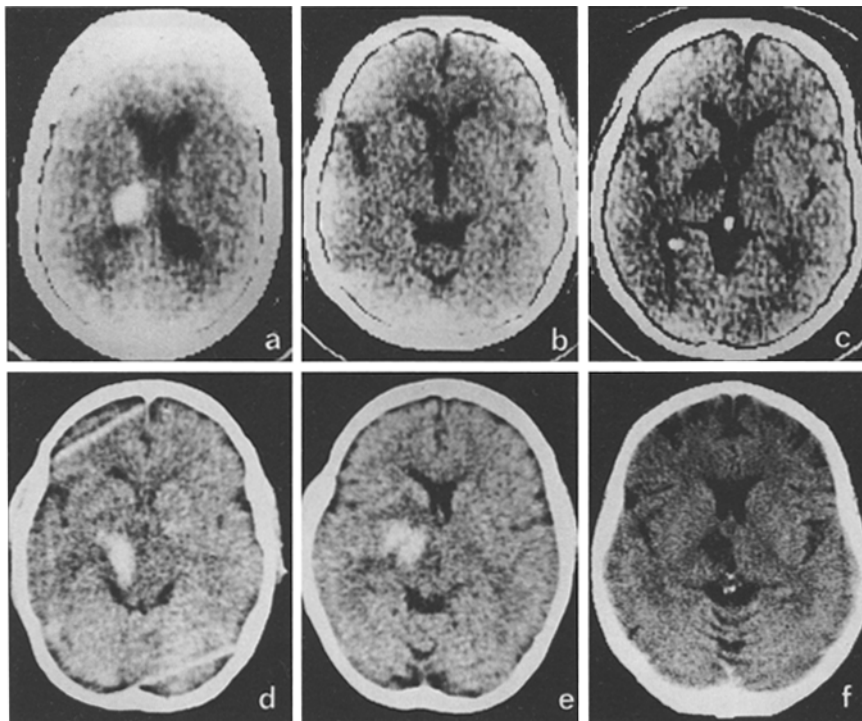


Fig. 1a–f. CT scans showing the extent of the thalamic lesions in the four personal cases. Top line from left to right: **a** case 17, **b** case 18 2.5 months before the thalamic infarct, **c** case 18 at the time of the speech dysfunction. Bottom line from left to right: **d**, **e** case 19 at two levels; **f** case 20. The left side of the picture is the left side of the patient

Table 1. Thalamic aphasia ($n = 20$): clinical signs and CT findings in 16 previously published and 4 personal cases

| Reference, year | Case 1 Samarel et al. [30] 1976 | Case 2 Elghozi et al. [9] 1978, case 3 | Case 3 Reynolds et al. [29] 1979, case 1 | Case 4 Archer et al. [2] 1981 | Case 5 McFarling et al. [23] 1982, case 1 | Case 6 McFarling et al. [23] 1982, case 2 | Case 7 Michel et al. [24] 1982 | Case 8 Papagno and Guidotti [27] 1983 | Case 9 Biller et al. [4] 1984 | Case 10 Graff-Radford et al. [12] 1984, case 1 |
|----------------------------|---|---|---|--|--|--|-----------------------------------|--|----------------------------------|---|
| Reduced spontaneous speech | + | + | NM | NM | + | + | + | + | NM | + |
| Decreased speech volume | + | + | NM | NM | NM | NM | NM | NM | + | - |
| Denomination disturbances | + | + | + | + | + | - | NM | + | - | + |
| Perseveration | + | NM | + | + | + | NM | NM | + | NM | + |
| Neologisms | + | NM | + | NM | NM | NM | NM | + | NM | NM |
| Intact repetition | + | + | NM | + | + | - | + | + | + | + |
| Paraphasias | NM | - | NM | + | - | - | + | + | NM | + |
| Memory disturbances | + | NM | NM | + | - | - | + | NM | + | + |
| Intact comprehension | + | + | + | ↓ | Fluctuating | + | + | + | + | ↓ |
| CT localization | Pulvinar | VL, VPL | VPL | VA, VL | VL, VPL, VA, A | VL, DM | VL, VA, A, DM | Pulvinar | A, VA, VL | VL, DM |
| Sex | M | M | M | M | M | M | M | M | M | M |
| Age | 66 | 53 | 61 | 57 | 52 | 18 | 54 | 78 | 29 | 67 |
| Risk factors | Hypertension | Hypertension | Hypertension | NM | Hypertension, smoking | NM | Hypertension | NM | Venous thrombosis | Hypertension diabetes mellitus |
| Lesion | Haemorrhage | Haemorrhage | Haemorrhage | Infarction | Infarction | Infarction | Infarction | Haemorrhage | Infarction | Infarction |
| Reference, year | Case 11 Graff-Radford et al. [12] 1984, case 2 | Case 12 Graff-Radford et al. [12] 1984, case 3 | Case 13 Lhermitte [18] 1984 | Case 14 Bogoslavsky et al. [5] 1986, case 1 | Case 15 Bogoslavsky et al. [5] 1986, case 2 | Case 16 Mori et al. [16] 1986 | Case 17 | Case 18 | Case 19 | Case 20 |
| Reduced spontaneous speech | + | + | NM | + | + | NM | - | + | + | + |
| Decreased speech volume | - | + | + | NM | NM | NM | - | + | + | + |
| Denomination disturbances | + | + | + | + | + | + | + | + | + | + |
| Perseveration | NM | NM | + | + | + | + | + | + | - | - |
| Neologisms | NM | NM | + | NM | + | + | - | - | - | + |
| Intact repetition | + | + | NM | + | + | + | - | + | + | + |
| Paraphasias | + | - | NM | + | + | + | + | - | - | - |
| Memory disturbances | + | + | NM | + | NM | + | - | - | - | + |
| Intact comprehension | ↓ | ↓ | NM | ↓ | ↓ | + | + | + | + | + |
| CT localization | VL, DM | VL, DM | VL, VPL, VPM, DM | A | A | VL | VL, VPL, VPM, pulvinar | A, VA | VL, VPL, A, VPM, pulvinar | DM |
| Sex | F | M | M | F | M | M | F | M | F | F |
| Age | 66 | 75 | 50 | 45 | 74 | 41 | 64 | 65 | 60 | 59 |
| Risk factors | Hypertension, smoking | Hypertension | Hypertension | Hypercholes terolaemic | Hypertension | NM | Hypertension | Hypertension | Hypertension | Diabetes mellitus |
| Lesion | Infarction | Infarction | Haemorrhage | Infarction | Infarction | Infarction | Haemorrhage | Infarction | Haemorrhage | Infarction |

A = anterior nucleus; VA = ventroanterior nucleus; DM = dorsomedial nucleus; VL = ventrolateral nucleus; VPL = ventroposterolateral nucleus; VPM = ventroposteromedial nucleus; + = present; - = absent; n. m. = not mentioned; ↓ = decreased

fronto-thalamic pathways conspicuously disregard the traditional anatomical concepts of thalamic nuclei.

Materials and methods

Of 9 cases of thalamic (haemorrhagic) infarction seen over a 2-year period (1982–1984), only 4 qualified for this study, their lesion(s) being of a sufficiently restricted size and apparently involving a restricted number of the main thalamic nuclei shown on the CT scan. The reference map based on CT sections of a frozen human head was used [19].

From the literature another 16 patients, meeting the same criterion, were selected from a review of 46 reported cases. The remaining 30 cases were excluded because the lesion of the thalamus was too extensive.

The lesions in the retrospective personal series together with those identically defined in the reported cases were visualized on CT films (Fig. 1). The Boston Diagnostic Aphasia Examination was not used, but the clinical histories of the patients contained ample data to classify the elements of the speech disorder present in the 20 cases as: reduced spontaneous speech, decreased speech volume, anomia, disturbed repetition, perseveration, neologism(s), and paraphasia(s).

Memory disturbances and language comprehension were noted as well. Reading and writing deficits were not investigated because of incomplete documentation in the reports reviewed.

Results

The findings are shown in Table 1 and Fig. 2. There were no gross divergencies in lapses of time between onset and CT or language testing (Table 2). Lesions were restricted to the thalamus in all instances, except for cases 5 and 11, in whom an additional infarction was seen on the CT scan in the occipital lobe, a site which does not seem relevant to the problem under consideration.

Clearly, pulvinar involvement is present when almost any of the seven features of speech dysfunction manifests. Reduced spontaneous speech, anomia, and perseveration are seen in the majority of instances with almost any thalamic lesion. Neologisms and paraphasias are conspicuously the least frequent. With all the reservations that should be kept in mind, given the limited number of observations and the combination of nuclei (disregarded here) as well as the observation that not all dysphasic features mentioned in Table 1 were investigated in the reports reviewed, one notes the following with respect to the dysphasic features: the ventroposteromedial nucleus (VPM) is least and the dorsomedial nucleus (DM) most affected in reduced spontaneous speech, the anterior nucleus (A) least and the VPM most in decreased speech volume, the VPM, the ventroposterolateral nucleus (VPL) and pulvinar most and the ventroanterior nucleus (VA) and A least in anomia, the pulvinar most and dorsomedial nucleus (DM) least in perseveration, pulvinar most and VA not at all in neologisms, and pulvinar and A most and VPL least in paraphasias.

Case 18 is of particular interest in view of the functional inputs and outputs known for almost any of the thalamic nuclei: this patient, who had had a coronary infarction 1 year before, was seen because of an attack of giddiness in September 1982. The CT scan, obtained at the time on the suspicion that the vertigo might be related to a cerebral infarction this time (in

| | VPL n=6 | Pulvinar n=4 | VL n=13 | VA n=6 | DM n=7 | A n=6 | VPM n=3 |
|----------------------------|------------|-----------------|------------|-----------|-----------|----------|------------|
| Reduced spontaneous speech | 3 | 3 | 8 | 4 | 6 | 5 | 1 |
| Decreased speech volume | 3 | 2 | 5 | 3 | 3 | 2 | 2 |
| Denomination disturbances | 6 | 4 | 10 | 4 | 5 | 4 | 3 |
| Intact repetition | 4 | 3 | 10 | 6 | 6 | 6 | 1 |
| Perseveration | 4 | 3 | 7 | 3 | 2 | 4 | 2 |
| Neologisms | 2 | 2 | 2 | 0 | 2 | 1 | 1 |
| Paraphasias | 1 | 2 | 6 | 2 | 3 | 3 | 1 |
| Memory disturbances | 0 | 1 | 7 | 3 | 5 | 3 | 0 |

Fig. 2. Characteristic features of aphasia, summarizing the speech disorders in association with the site of thalamic lesions in the 16 cases previously published in the literature and 4 personal cases. The numbers on top represent the number of patients for which the particular thalamic nucleus was involved. The stippled rectangles represent in absolute numbers how often any particular nucleus was involved if the specific dysphasic feature was present. Of the 20 patients, the columns are headed by the number of instances in which a certain nucleus was involved by infarction. The 8 layers list by numbers and stippled proportional areas the number of times patients exhibited a specific speech defect

Table 2. Time intervals between onset and CT and speech deficit testing

| Case no. | Onset-CT | Onset-Speech deficit testing |
|----------|-------------|------------------------------|
| 1 | NM | Immediately |
| 2 | 1 Month | 2 Weeks |
| 3 | < 2 Weeks | < 2 Weeks |
| 4 | 2 Weeks | 2 Weeks |
| 5 | < 1 Week | < 1 Week |
| 6 | NM | NM |
| 7 | < 3 Weeks | < 3 Weeks |
| 8 | Immediately | 1 Week |
| 9 | NM | NM |
| 10 | Immediately | Immediately |
| 11 | 4 Days | 3 Weeks |
| 12 | < 1 Week | Immediately |
| 13 | NM | NM |
| 14 | 4 Days | NM |
| 15 | 2 Days | NM |
| 16 | Immediately | Immediately |
| 17 | Immediately | < 1 Week |
| 18 | 3 Days | < 4 Days |
| 19 | Immediately | < 1 Week |
| 20 | Immediately | < 1 Week |

NM = Not mentioned

view of the cardiac infarction a year before) was completely normal. Two and a half months later he suddenly developed a right-sided hemiplegia together with aphasia and a CT scan 72 h after onset, revealed infarction of the thalamic anterior and ventroanterior nuclei (Table 1, Fig. 1b, c). His hemiplegia was clearly caused by this infarct as it tangentially involved the internal capsule.

Discussion

Analysis of this admittedly small series of patients confirms that the thalamus is not only a crucial relay centre in subserv-

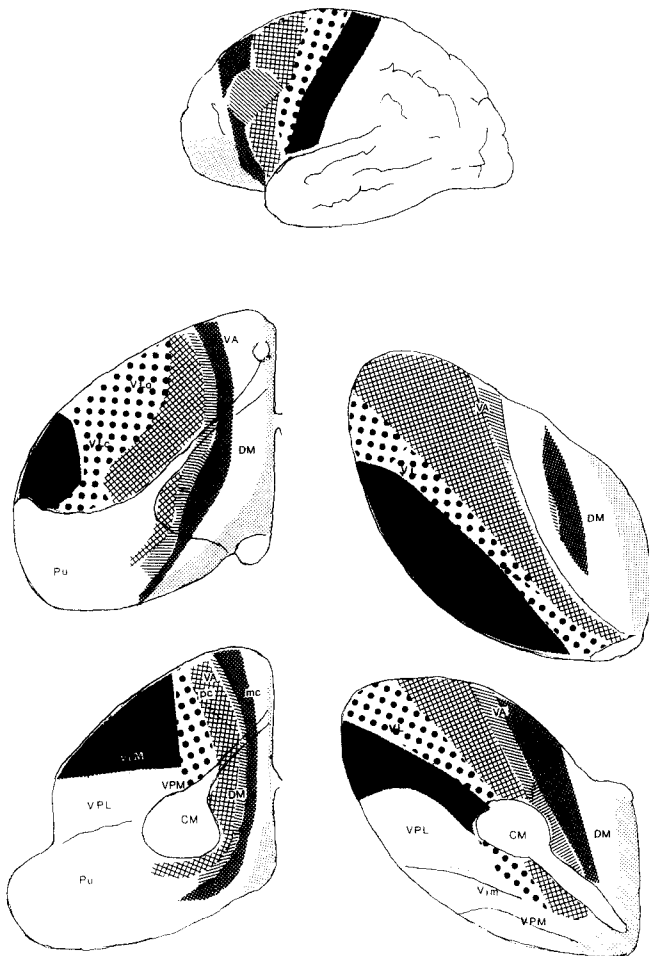


Fig. 3. Diagrammatic corticothalamic projections, modified from Kievit and Kuypers [15]. The *top figure* is a lateral view of the left hemisphere. The two figures on the *left* are horizontal sections through the left thalamus at two different levels in rostrocaudal direction. The two figures on the *right* are cross-sections through the left thalamus in a ventrodorsal direction. The various corresponding cortical-thalamic areas are indicated by stippling and cross-hatching

ing sensory and motor mechanisms, but also subserves “higher” central nervous system activities, such as speech. Of all thalamic nuclei the pulvinar and VPL are most frequently involved [10]. Both nuclei are supplied by the geniculo-thalamic artery, a branch of the posterior cerebral artery. Pulvinar infarcts seem to be associated with all the dysphasic features listed. In addition to the distribution of lesions derived from the literature, the present series adds 2 instances of lesions restricted to the DM (case 20) and to the A/VA (case 18, surprisingly also showing most of the dysphasic features).

Analysis of this series does not provide arguments to answer in the affirmative the question whether there are specific thalamic nuclei that subserves specific elements of speech. This is, however, not very surprising.

Clinical neurologists, traditionally inclined to tie functional deficit to lesions affecting certain tracts or grey matter areas, expect that the sharply outlined thalamic nuclei are specialized in subserving well-defined functions. This is not borne out by the present examination. Kuypers [17] convincingly showed in the primate that this traditional approach is also not warranted by experimental findings; the connections between the

thalamus and frontal lobe completely disregard thalamic nuclear boundaries, showing a pattern of quite a different order instead (Fig. 3). These connections from the most rostral and orbital areas of the frontal lobe to the anterior and posterior lip of the central sulcus show a well-ordered representation in the whole thalamus extending over its entire area from medial to lateral. This implies that the clinician should aim at identifying this rostrofrontal-mediotalamic to caudofrontal-latero-thalamic relationship by distinguishing medially situated from laterally situated thalamic lesions, irrespective of involvement of specific thalamic nuclear areas. In the symptomatology of thalamic infarctions, irrespective of their size and site, symptoms due to interruption of these topologically arranged fronto-cortico-thalamic connections, and, therefore of the frontal cortical areas, are intermingled.

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