Anteromedian, Central, and Posterolateral Infarcts of the Thalamus
Three Variant Types

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Background and Purpose—Thalamic infarcts have traditionally been classified into 4 territories: anterior, paramedian, inferolateral, and posterior. The purpose of this study was to review this classical versus variant distribution in patients with thalamic stroke.

Methods—We reviewed all patients with a first clinical stroke included in the Lausanne Stroke Registry between 1990 and 2002. Among 71 patients with an acute stroke isolated to the thalamus confirmed by MRI, we selected all patients with lesions outside the classical territories and studied their clinical, etiological, and radiological features.

Results—A total of 21 patients (30% of all thalamic stroke patients) showed infarction outside the classical territories, allowing us to delineate 3 variant distributions: (1) Anteromedian territory (9 patients [13%]) involving anterior and paramedian territories, with predominantly cognitive impairment, including executive dysfunction, anterograde amnesia, and aphasia in left-sided or bilateral lesions. The most frequent stroke mechanism was cardiac embolism. (2) Central territory (4 patients [6%]), with lesions on the central part of the thalamus, resulting in a variety of neurological and neuropsychological signs, reflecting the involvement of several adjacent structures. Microangiopathy was the most frequent etiology. (3) Posterolateral territory (8 patients [11%]), involving inferolateral and posterior territories, with hemihypesthesia as the most frequent manifestation, followed by hemiataxia, executive dysfunction, and aphasia in left-sided lesions. Artery-to-artery embolism and microangiopathy were the main stroke mechanisms.

Conclusions—We describe 3 variant topographic patterns of thalamic infarction with distinct manifestations and etiologies. We postulate that these infarcts are the result of a variation in thalamic arterial supply or reflect borderzone ischemia. (Stroke. 2004;35:000-000.)

Key Words: brain infarction ▪ thalamus ▪ stroke

Thalamic infarcts are classically classified into anterior, paramedian, inferolateral, and posterior territories, which are supplied, respectively, by the polar, paramedian, thalamogeniculate, and posterior choroidal arteries. This classification was initially based on neuroanatomical and neuropathological data1 and later confirmed by imaging techniques (computed tomography and MRI).2–6

The aim of the present study was to determine whether thalamic infarcts outside the 4 classical territories exist by using MRI, and if so, to study their clinical, etiological, and radiological features.

Subjects and Methods
Between 1990 and 2002, 3712 patients with first-ever stroke were admitted to the Neurology Department of the Lausanne University Hospital and prospectively entered in the Lausanne Stroke Registry.7 A total of 71 patients with MRI-proven lesions restricted to the thalamus were identified. Patients with hemorrhagic lesions, old infarcts on imaging, or simultaneous acute lesions outside the thalamus were excluded. Patients with lesions outside the 4 classical thalamic territories were included in the present study.

MRI consisted of axial T1- and T2-weighted spin-echo images (5-mm sections) within 7 days after admission. Axial T2-weighted images were used to define lesion size and site. Additional diffusion-weighted imaging was performed in the 13 patients included after 2000 and mainly used to exclude concomitant infarct outside the thalamus. Intracranial and extracranial Doppler ultrasonography with frequency spectral analysis, 12-lead electrocardiography, a minimum of 24 hours of neurological and cardiovascular monitoring, and standard laboratory tests were performed in all patients. Angio-MRI, 2D echocardiography, and 24-hour electrocardiography (Holter) monitoring were performed on selected patients, depending on the suspected etiology.

The following risk factors were recorded: age, sex, hypertension (blood pressure of >160/90 at least twice before the stroke), diabetes mellitus, cigarette smoking, and hypercholesterolemia (cholesterol blood levels >6.5 mmol/L). The etiology of stroke was determined according to criteria described previously7 as artery-to-artery, cardioembolic, or microangiopathy. All patients underwent standardized neuropsychological testing8,9 within 7 days of stroke. Classical thalamic territories were classified according to their arterial supply as reported previously10.
1. the anterior territory, supplied by the polar artery (also known as the tuberothalamic artery, anterior thalamosubthalamic paramedian pedicle, anterior optic artery, or premamillary pedicle). This artery is absent in about one third of cases, and the anterior territory is then supplied by the paramedian arteries;

2. the paramedian territory, supplied by the paramedian arteries (also known as the posterior thalamosubthalamic paramedian, retromamillary, or thalamoperforate pedicle). The left and right paramedian arteries can originate from a single pedicle;

3. the inferolateral territory, supplied by the thalamogeniculate arteries (also known as the inferolateral or inferior external optic arteries); and

4. the posterior territory, supplied by the posterior choroidal arteries.

The variant territories were found and grouped according to their anatomic location within the thalamus and localized in the following regions:

1. Anteromedian territory: defined by infarcts involving the classical anterior and paramedian territories, combining the posterior part of the anterior territory and the anterior part of the paramedian territory.
2. Central territory: defined by infarcts in the central part of the thalamus with involvement of parts of the adjacent 4 classical territories
3. Posterolateral territory: defined by infarcts involving the classical inferolateral and posterior territories, combining the posterior part of the inferolateral territory and the anterior part of the posterior territory.

### Results

Twenty-one patients (30% of all stroke patients with isolated thalamic infarct) had a lesion outside the classical territories and consisted of 13 men (62%) and 8 women (38%). The left thalamus was involved in 10 patients (48%), the right thalamus in 7 (33%), and both thalami in 4 (19%). The mean age was 58 years (range 19 to 86 years).

#### Variant Territories (Table 1; Figure)

**Anteromedian Territory**

Nine patients (13% of all isolated thalamic infarcts) had an infarct in the anteromedian territory (Figure). Mean age was 59 years (range 39 to 86). Cognitive impairment, decreased consciousness, and vertical eye paresis were the most frequent signs. Of the patients with a left lesion, 3 (75%) had memory impairment (anterograde amnesia in 3 cases, retrograde amnesia in 1), 3 (75%) had loss of initiative and executive dysfunction, 1 had aphasia, 1 visual agnosia, and 1 had ideomotor apraxia with orofacial apraxia. Vertical eye paresis was found in 3 (75%) patients with a left lesion. Those with a right lesion had anterograde memory impairment, loss of self-activation, and executive dysfunction, and 1 had aphasia (ideomotor, ideational, constructive, and orofacial), and contralateral hemineglect, and the other had vertical eye paresis and impaired consciousness. Of the 3 patients with a bilateral infarct, all had executive dysfunction and memory disturbances (anterograde amnesia in 3 cases, retrograde amnesia in

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**Table 1. Stroke Mechanisms and Clinical Features of the 3 Variant Thalamic Infarct Types**

<table>
<thead>
<tr>
<th></th>
<th>Anteromedian (n=9)</th>
<th>Central (n=4)</th>
<th>Posterolateral (n=8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>13% of all thalamic infarcts; R=2, L=4, B=3</td>
<td>6% of all thalamic infarcts; R=2, L=1, B=1</td>
<td>11% of all thalamic infarcts; R=3, L=5</td>
</tr>
<tr>
<td>Men</td>
<td>67%</td>
<td>25%</td>
<td>75%</td>
</tr>
<tr>
<td>Stroke mechanism</td>
<td>Cardiac 56%, art-to-art 33%, other 11%</td>
<td>Microangiopathy 50%, cardiac 25%, other 25%</td>
<td>Microangiopathy 38%, art-to-art 38%, cardiac 12%, other 12%</td>
</tr>
<tr>
<td>Decreased vigilance</td>
<td>16% with unilateral and 67% with bilateral lesions.</td>
<td>33% with unilateral and in the patient with bilateral lesions</td>
<td>None</td>
</tr>
<tr>
<td>Vertical eye paresis</td>
<td>67% with unilateral and bilateral lesions</td>
<td>In the patient with bilateral lesions</td>
<td>None</td>
</tr>
<tr>
<td>Hemiparesis</td>
<td>44%</td>
<td>25%</td>
<td>38%</td>
</tr>
<tr>
<td>Hypertension</td>
<td>44%</td>
<td>100%</td>
<td>38%</td>
</tr>
<tr>
<td>Ataxia</td>
<td>44%</td>
<td>50%</td>
<td>63%</td>
</tr>
<tr>
<td>Neuropsychological features</td>
<td>100%</td>
<td>In the patient with bilateral lesions</td>
<td>38% (all with left lesion)</td>
</tr>
<tr>
<td>Memory impairment</td>
<td>89%</td>
<td>In the patient with bilateral lesions</td>
<td>12%</td>
</tr>
<tr>
<td>Aphasia</td>
<td>56% (44% WFD, 44% denomination impairment, 22% paraphasia)</td>
<td>WFD and paraphasia in the patient with bilateral lesions</td>
<td>38% (38% WFD and denomination impairment, 25% repetition impairment)</td>
</tr>
<tr>
<td>Agnosia and neglect</td>
<td>33%</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Apraxia</td>
<td>22% (ideomotor and facio-oral apraxia in 22%, constructive and ideational apraxia in 11%)</td>
<td>None</td>
<td>Facio-oral apraxia in one case</td>
</tr>
<tr>
<td>Executive dysfunction</td>
<td>89%</td>
<td>In the patient with bilateral lesions</td>
<td>38%</td>
</tr>
</tbody>
</table>

R indicates right; L, left; B, bilateral; art-to-art, artery-to-artery mechanism; WFD, word finding difficulties.
2), 2 (67%) had aphasia, and 1 (33%) contralateral hemineglect with visual agnosia. Two (67%) patients had vertical gaze paresis, and 2 had impaired consciousness. One patient with a bilateral infarct displayed confabulations. Motor weakness and ataxia were transient and resolved in all patients within 1 week after admission, whereas decreased consciousness resolved in 3 days in the patient with unilateral infarct. Sensory deficits disappeared in half of the patients and improved in the other. Among the neuropsychological signs, memory deficits usually persisted, whereas executive dysfunction, apraxia, and aphasia improved within the first 7 days. Cardioembolism was the most frequent stroke mechanism (5 patients; 56%). Cigarette smoking was present in 3 (75%) patients, and hypercholesterolemia and hypertension in 2 (50%).

Central Territory
Four patients (6% of all thalamic infarcts) had an infarct in the central territory (Figure). Mean age was 43 years (range 19 to 68). Infarcts of this territory were responsible for a variety of signs and symptoms. Decreased consciousness was found in 2 (50%) patients. The patient with bilateral lesions was the only 1 with cognitive impairment, including aphasia, executive dysfunction, and memory impairment (anterograde and retrograde amnesia). All patients had contralateral hypesthesis. One patient presented with choreoathetotic movements of the contralateral side, and 1 patient had a contralateral homonymous hemianopsia. Vertical eye paresis was found in the patient with bilateral lesions. During the week after admission, consciousness improved in the patient with unilateral infarct, whereas other signs remain unchanged. The most frequent stroke mechanism was hypertensive microangiopathy (2 [50%] patients). Cigarette smoking was present in 3 (75%) patients, and hypercholesterolemia and hypertension in 2 (50%).

Posterolateral Territory
Eight patients (11% of all thalamic infarcts) had an infarct in the posterolateral territory (Figure). Mean age was 64 years (range 52 to 82). Sensory deficit was the main sign in this territory. Seven (88%) patients had moderate to severe contralateral hypesthesia, which was restricted to the acro-oral area in 1 case. Contralateral ataxia and slight motor weakness were found, respectively, in 5 (63%) and 3 (38%) patients. The association of contralateral ataxia and hypesthesia was found in 3 (38%) patients and of contralateral motor weakness, hypesthesia, and ataxia in 2 (25%). Three of the 5
patients with a left lesion had cognitive impairment. Three patients presented with a dystonic posture. Motor signs were transient in all cases and resolved within 1 week after infarct. Phasic troubles improved in 1 case with disappearance of repetition impairment. No change of executive dysfunction was noted. Ataxia and hypesthesia persisted in all patients for >1 week despite improvement in half of the patients. The most frequent etiologies were microangiopathy and artery-to-artery embolism (3 [38%] patients each). Hypertension was found in 5 (63%) patients, cigarette smoking in 4 (50%), and hypercholesterolemia and diabetes in 3 (38%).

Classical Territories
Fifty patients (70% of all thalamic infarcts) had an infarct in 1 of the 4 classical territories (Table 2): 8 (11%) in the anterior territory, 19 (27%) in the paramedian territory, 19 (27%) in the inferolateral territory, and 4 (6%) in the posterior territory. The stroke mechanisms and main clinical features of these infarcts corresponded to patterns described previously.3,4,6,9,16,17 except for paramedian infarcts. After lesions in this territory, memory impairment in our study was less frequent than reported previously.3,4,6

Discussion
This study of 71 patients with thalamic infarcts confirmed by MRI showed that nearly one third of patients did not fall into the classical territories. These variant territories (anteromedian, central, and posterolateral) corresponded to overlapping, combined lesions and still had characteristic clinical and etiological features.

Anteromedian Territory
The main feature of patients with infarcts in anteromedian territory was the severity of the neuropsychological disturbances that occurred in all patients. Anterograde memory impairment was found in all but 1 patient with anteromedian infarct and was more severe after bilateral lesions. Our results suggest that lesions restricted to the anteromedian thalamic structures, such as the anterior parts of the dorsomedian (DM) nucleus, of the intralaminar (IL) nuclei (mainly parafacicular nucleus), and of the internal medullary lamina (IML), may be responsible for severe amnesia. This contrasts with previous reports in which involvement of anterior nuclei and mamillothalamic tract was thought to be necessary9,18–22 to produce severe amnesia. On the other hand, in our study, patients with a paramedian infarct restricted to the posterior part of DM nucleus and IL nuclei (centromedian [CM] and centrolateral [CL] nuclei) or IML presented more rarely with amnesia. Anterior parts of DM nucleus and IL nuclei seem to be strongly involved in anterograde amnesia but not their posterior parts. Except in 1 case with bilateral lesions, we did not find differences between verbal and visuospatial learning according to the side of the lesion, in contrast with previous reports after anterior19,21 or paramedian2,22,23 lesions.

On the contrary of the acute behavioral syndrome of anterior thalamic infarcts,9 the clinical picture of perseverative behavior was not the hallmark of patients with anteromedian infarcts, although executive dysfunction was found in all but 1 patient. The main characteristic of behavior changes was a rather severe loss of self-activation, with a constant need for external stimulation, and several patients resembled robots. Interestingly, we found a lack of initiative not only after bilateral infarcts24,25 but also after unilateral anteromedian infarcts (left and right sides), experienced by more than half of the patients. Disinhibition, distractibility, or hypomnemonic state described after anterior infarcts was not found after anteromedian infarct in the acute phase. The characteristics of linguistic troubles after anteromedian infaracts were word-finding difficulties, reduced fluency, and denomination, as reported previously in thalamic aphasia.26,27 Interestingly, phonemic paraphasia was found in 2 cases. On the contrary of what is described after anterior lesion, aphasia never occurred after right lesion. Apraxia was less frequent than aphasia but occurred not only after left28 but also after right lesions. Decreased consciousness was less frequent after anteromedian infarcts than after paramedian infarcts and never found after anterior infarct. These results underline the role of the posterior parts of DM nucleus, IL nuclei (CM and CL), and IML as relay between the reticular ascending system and the cortex14,29 on the contrary of more anterior nuclei.

Although sagittal or coronal images were not performed, our study confirmed that isolate paramedian or anteromedian infarcts can interrupt supranuclear tracts responsible for vertical gaze control without involvement of mesencephalon.30,31 Overall, infarcts of the anteromedian territory should be suspected with a clinical pattern of cognitive impairment (with mainly loss of self-activation, amnesia, and aphasia), mild decrease level of consciousness, and vertical gaze paresis. With a lesion in this territory, a cardiac source of embolism should actively be sought.

### TABLE 2. Stroke Mechanisms and Clinical Features of the 4 Classic Thalamic Territories

<table>
<thead>
<tr>
<th>Anterior (n=8)</th>
<th>Paramedian (n=19)</th>
<th>Inferolateral (n=19)</th>
<th>Posterior (n=4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>11% of all thalamic infarcts; R=2, L=6</td>
<td>27% of all thalamic infarcts; R=3, L=10, B=6</td>
<td>27% of all thalamic infarcts; R=5, L=14</td>
</tr>
<tr>
<td>Stroke</td>
<td>Cardiac 50%, art-to-art 13%, other 37%</td>
<td>Cardiac 68%, art-to-art 16%, microangiopathy 4%, other 11%</td>
<td>Microangiopathy 58%, cardiac 11%, art-to-art 11%, other 20%</td>
</tr>
<tr>
<td>Main clinical features</td>
<td>100% with cognitive impairment (100% executive dysfunction and memory impairment, 50% aphasia) decreased vigilance in 25%</td>
<td>68% cognitive impairment (63% executive dysfunction, 37% memory impairment and aphasia), 63% impaired consciousness, 63% vertical eye paresis</td>
<td>84% hypesthesia, 74% ataxia and 26% mild motor weakness, 42% cognitive impairment (42% executive dysfunction, 21% aphasia)</td>
</tr>
</tbody>
</table>

R indicates right; L, left; B, bilateral; art-to-art, artery-to-artery mechanism.
Central Territory
Infarcts of this territory were rare, but various neurological and neuropsychological signs were seen, reflecting involvement of adjacent thalamic nuclei. Hypothetasia was found in all patients because of involvement of the median part of the ventroposterolateral (VPL) nucleus. Vertical gaze paresis and neuropsychological signs were found in 1 patient. Decreased consciousness was not as frequent as expected regarding the involvement of IL.14,29 Our results may suggest that DM nucleus, along with IL nuclei, may play an important role in maintaining wakefulness. However, only 4 patients had a lesion restricted to this territory. More patients are needed to determine exactly the clinical features of patients with a lesion in this territory, but we suggest that infarct in the central territory has to be suspected in patients with hypotetasia, ataxia, neuropsychological sign, or vertical eye paresis.

Posterolateral Territory
The most unusual sign after posterolateral lesion was cognitive impairment, which occurred in more than half of the patients with left posterolateral territory lesions. In our study, aphasia was more common than was reported previously in inferolateral or posterior infarcts.2–6,16,17,32 This may be because of the interruption after posterolateral infarcts not only of pulvinar but also of ventrolateral (VL) and VPL nuclei projections to the temporal and parietal cortices.28 However, aphasia after posterolateral territory was different from aphasia after anteromedian or central infarcts, with impaired repetition in 2 cases resembling cortical motor aphasia.

Executive dysfunction was found after left posterolateral lesion, which emphasizes the importance of thalamocortical projections from posterolateral nuclei of thalamus.33 On the contrary, with what is described after inferolateral infarcts,17 we found executive dysfunction to be more frequent after left lesion than right lesion. Further assessment of patients with inferolateral, posterolateral, and posterior lesions are needed to determine a possible lateralization of executive functions.

Sensory deficit was the major sign of patients with posterolateral infarcts, which is explained by the localization of VL and VPL nuclei projections to the temporal and parietal cortices.28 Ataxia was less frequently observed, probably because of the more anterior location of the VL nuclei.34,35

Dystonic posture was found in 3 patients, which confirms the importance of posterolateral nuclei in movement control.36–39 Our results differed from what was described previously38,39 because of the apparition of the dystonic posture within days, rather than weeks or months, after stroke.

In summary, a posterolateral stroke should be suspected in patients with hemihypesthetic ataxic hemiparesis, and with a left lesion, with aphasia and executive dysfunction in a patient experiencing arterial hypertension.

Hypothetical Stroke Mechanisms
A borderzone mechanism between adjacent territories can be suspected in all of the variant territories. A transient occlusion of 1 of the 4 main arteries of the thalamus may result in ischemia of the most distal area supplied by the artery, causing borderzone infarct.40,41 This phenomenon may occur when blood cannot be supplied by adjacent arteries in case of microangiopathy or simultaneous involvement. For example, posterolateral territory infarcts may be the result of transient occlusion of the proximal part of the second segment of the posterior cerebral artery with simultaneous involvement of inferolateral and posterior choroidal arteries. Borderzone infarcts may be suspected especially in patients with small thalamic infarcts.

Second, these variant territories may be explained by variations of the territories supplied by the thalamic arteries. Although we did not perform conventional angiography, several studies suggest that all thalamic arteries can be subject to variation.1,13,15,42,43 This may be illustrated by the anteromedian territory, which lies in a region between the anterior and paramedian territories, a region known for frequent variation of blood supply.15 The variant anteromedian arteries, as paramedian arteries do, probably originate from the first part of the posterior cerebral arteries near the “top of the basilar” artery,44 which explains why cardioembolism is the most frequent etiology of anteromedian infarcts. A sensitivity of the arteries supplying the posterolateral territory to hypertension, as seems to be the case for the thalamogeniculate arteries,3,4,32 may explain the predominant microangiopathic etiology of posterolateral infarcts.

We did not find any case of infarct between posterior choroidal territory and paramedian territories, nor between anterior and inferolateral territories, although such infarcts can certainly occur. We postulate that variation of arteries is less frequent with collaterals, being more important in these thalamic regions.

In summary, our findings emphasize variant topographic patterns of thalamic infarctions, which were poorly identified previously, although being far from uncommon. The described infarct types may be explained by hemodynamic (borderzone) mechanisms and variations in arterial supply.

References
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