The Syndrome of Unilateral Tuberothalamic Artery Territory Infarction

JULIEN BOGOUSSLAVSKY, M.D., FRANCO REGLI, M.D., AND GIL ASSAL, M.D.

SUMMARY The study of 3 personal cases and 5 published cases of unilateral infarct limited to the territory of the tuberothalamic artery suggests that this syndrome should be differentiated from the other thalamic syndromes. The onset is usually sudden, with moderate contralateral weakness. Sensory changes may be present but remain mild. The patients are apathetic, show perseverations and may be disoriented. In left-sided infarcts, transcortical aphasia, verbal and visual memory impairment and sometimes acalculia are found. In right-sided infarcts, hemispatial neglect, visual memory impairment and disturbed visuospatial processing are common. A decreased level of consciousness, disturbed ocular movements, severe motor weakness and delayed abnormal movements do not occur. Involvement of the ventral lateral and dorsomedial nucleus with sparing of the intralaminar nuclei, posterolateral formation and upper midbrain may explain this picture. The fact that the tuberothalamic artery arises from the posterior communicating artery, which often receives its supply from the carotid system, further justifies considering unilateral tuberothalamic infarcts as a syndrome.

Methods and Patients

We diagnosed tuberothalamic artery infarction from the CT scan pictures, according to the topographic description of Percheron.13 All 3 patients were examined by at least 3 neurologists and 1 neuropsychologist. A comprehensive battery of tests was done in patients 1 and 3, and a partial battery was administered to patient 2, because of marked speech disturbances and impaired cooperation. The following evaluation was done: orientation to time and place, spontaneous speech, verbal fluency, naming (Boston naming test14), repetition of isolated, or series (3–5 items) of phonemes or words and sentences (3–15 words), auditory comprehension (Token test15), writing (spontaneous, dictation), reading (words, non-words, text, summary of text meaning), dichotic listening test, oral and written calculation, bucco-linguo-facial praxias, limb (symbolic, imitation) praxias, constructional praxia (spontaneous drawing of a cube, Rey complex Figure16), Benton Facial Recognition test17 and other visual gnosia tests (Ghent,18 Poppelreuter,19 Columbia20), orientation on a map of Switzerland, right-left discrimination, evocation of recent and remote events, verbal learning (Hebb's recurring digits,2122 Rey Auditory Learning23), visual learning (Corsi’s Block-tapping,22 delayed reproduction of Rey Complex Figure16), Wisconsin Card Sorting test,23 Stroop test,24 Luria’s conflicting tasks, sequential rhythms and sequential geometrical figures,25 and a nonverbal intelligence test (Raven progressive matrices26).

Case 1

A 45-year-old right-handed housewife was admitted after she suddenly experienced dizziness, vomiting, numbness in the right side of the face, clumsiness of the right hand and speech disturbances. Her past history revealed episodes of hypoglycemia and migraine, and she was treated with estrogens for primary ovarian insufficiency. She was suffering from familial type IV hypercholesterolaemia, and had a strong family history of coronary heart disease on her father’s side.

On neurological examination, the visual fields, the optic fundi, the pupils and oculomotoricity were normal. A slight light touch hypoaesthesia involving the right side of the face down to the C3 dermatome was present, without involving the mouth and tongue, and with symmetrical corneal reflexes. An “emotional” right facial weakness was present, with normal contraction on command but droop of the lips on the right side when smiling. The taste and the remainder of the cranial nerves were normal. There was a slight downward drift of the right upper limb on the stretched arm test performed with the eyes closed, but no objective weakness could be demonstrated, and the tone, tendon and cutaneous reflexes were normal. The coordination of all four limbs and the gait were normal. However, the patient tended to lose her balance when trying to jump on the right leg. Tactile, temperature, pain, postural and vibratory sensation were preserved, with normal graphesthesia, stereognosis and topoesthesia. Her blood pressure was 110/70 mm Hg with a regular pulse (80/min). No bruit was heard on the neck or precordium, and the general examination was normal.

From the Department of Neurology and the Center of Neuroradiology, Centre Hospitalier Universitaire Vaudois, Lausanne, Switzerland.
Address correspondence to: Dr. Julien Bogousslavsky, Department of Neurology, CHUV, 1011 Lausanne, Switzerland.
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Neuropsychological Examination

The patient was well-oriented in time and space. She complained of concentration difficulty and memory disturbances. Her spontaneous speech was quantitatively reduced, with short sentences (5 words maximum) and slowed output. Occasional phonemic paraphasias were present ("anassin" instead of "assassin") and less frequently, semantic paraphasias ("clock" instead of "watch"). Naming was impaired (46/76, Boston naming test) and verbal fluency was reduced (14 names of animals in 9 minutes). The dichotic listening test did not show any asymmetry. Verbal comprehension (31/36, Token test) was only mildly impaired, despite slowness of answering. The repetition of isolated, or series (up to 5) of phonemes and words and of sentences (up to 15 words) was normal. Words, non-words, and text reading were normal. Spontaneous writing was reduced but without paraphasia or syntactic disturbances. Writing on dictation was unimpaired. Oral calculation was markedly impaired, even for simple arithmetical operation (0/6), with tendency to persevere. Written calculation was similarly impaired, with loss of the arithmetical programs for addition, subtraction, multiplication and division. Spontaneous drawing of a cube was preserved, although slowed. Bucco-linguo-facial praxias were normal, and limb praxias did not show any significant disturbance (4/4 on symbolic gestures, 5/6 on imitation gestures). Visual and visuo-spatial gnosias were normal (Ghent: 35/36, Columbia: 10/12, orientation on a geographic map). Marked difficulties were present on evocation of recent events, and visual as well as verbal learning difficulties were present (Hebb's recurring digits: maximal span of 5, failure of span + 1 learning; Corsi's Block-tapping: maximal span of 5, failure of span + 1 learning). Perseverations were present on execution of Luria's conflicting tasks, sequential rhythms and sequential geometrical figures. On Stroop test, naming was markedly slowed (1'48'', 1 mistake, 7 autoconnections) compared with word reading (1'35'').

Case 2

A 74-year-old right-handed, hypertensive man was admitted after he suddenly experienced speech disturbances, followed a few hours later by mild right-sided weakness. On examination, visual acuity, visual fields, ocular movements and pupils were normal. Optic fundi revealed moderate retinal vascular sclerosis. Facial, buccal and lingual sensation were preserved, with symmetrical corneal reflex. A slight "emotional" right-sided facial paresis was present, with symmetrical contraction on command. The taste and the remainder of the cranial nerves were normal. The strength was normal and symmetrical in the four limbs, but on the stretched arm test, there was a slow drift with pronation tendency of the right arm. Fine motility of the fingers was slowed on the right, and the tone of the right arm and leg was slightly increased, without tendon reflexes asymmetry or abnormality. Abdominal reflexes were present and plantar reflexes were down-going. Coordination was normal in the four limbs, except moderate dysdiadochokinesia in the right upper limb. The gait was normal. Tactile, temperature, pain, postural and vibratory sensations were normal and symmetrical, with normal 2-point discrimination, graphesthesia, stereognosia and absence of sensory extinction. Blood pressure was 160/100, but general examination was normal for his age.

Neuropsychological Examination

The patient was well-oriented in time and place. A detailed neuropsychological assessment was not possible, because of the marked disturbances of language. Spontaneous speech was moderately reduced, with slowed output and shortened sentences (8 words maximum). Phonemic and semantic paraphasias were present, as well as perseverations and occasional neologisms, with preserved syntax and absence of dysarthria. The frequency of the paraphasias rendered the patient's speech very difficult to understand. Naming was impaired (17/76, Boston naming test). Production of automatic series (days of the week, months) was preserved in anterograde order, but paraphasias and perseverations appeared in retrograde order. Verbal fluency was reduced (4 names of animals in three minutes), with perseverations. The dichotic listening
test did not disclose any left-right ear asymmetry. Verbal comprehension was moderately impaired (25/36 Token test), and the repetition of isolated, or series (up to 5) of, phonemes or words and of sentences (up to 15 words) was fully preserved. The reading of a text was normal, although slow, but his account of the story showed appearance of numerous paraphasias, perseverations, and neologisms. Spontaneous writing showed paragraphias and neologisms, with preserved copying. Writing on dictation was impaired, with paragraphias and perseverations of letters and words. Spelling of words was disturbed (2/5). Oral calculation was impaired (1/5). Written calculation showed preservation of addition and subtraction, but multiplication and division were impaired. Spontaneous drawing of a cube was normal. Bucco-linguo-facial praxias were preserved and limb praxias (symbolic and imitation) showed only occasional perseverations with autocorrection. Visual gnosias were preserved (Ghent: 33/36, Poppelreuter: 3/4), with preserved orientation on a geographic map. Learning of series of visual and verbal items was markedly impaired but was not quantified, because the patient did not cooperate well enough. Evocation of recent or remote events was disturbed by the speech disturbances. On execution of Luria's conflicting orders, sequential rhythms and sequential geometrical figures, perseverations appeared, with selfcorrection attempts and refusal to continue the tests.

Standard blood and urine tests were normal. EKG showed moderate ischemic changes with left ventricular hypertrophy. A CT scan performed 2 days after admission showed an isolated lucency in the anterior aspect of the thalamus on the left side, suggesting a recent infarct in the tuberothalamic artery territory (fig. 2).

Case 3

A 68-year-old insulin dependent diabetic right-handed man suddenly experienced transient blurring of vision with persisting left-sided weakness and feeling of intense fatigue. On admission, visual acuity was moderately decreased in both eyes (right: 0.5; left: 0.7) with diabetic retinopathy on optic fundi examination. The visual fields were full, but left-sided extinction appeared on bilateral simultaneous stimulation. The pupils and ocular movements were normal. Light touch and pain sensation were slightly decreased in the left side of the face, with a decreased left corneal reflex. A slight lower facial paresis was present, either on voluntary or emotional contraction. Bilateral hypacusis was present, but the remainder of the cranial nerves showed no abnormality. The strength and tone were normal in the 4 limbs, but the right arm drifted slowly downward and pronated on the stretched arm test. The fine movements of the fingers were slightly slowed on the left side, with dysdiadochokinesia and moderate imprecision on finger-to-nose movements. No hypermetria or rebound phenomenon were found. The tendon reflexes were decreased distally in the 4 limbs. Abdominal reflexes were decreased on the left side. The left plantar reflex was upgoing, but was flexor on the right. Tactile, pain and temperature sensations were slightly decreased on the left side of the body, with a tendency to extinction on simultaneous bilateral stimulation, but with normal graphesthesia.

FIGURE 2. Case 2, CT scan. Infarct of the anterolateral (A) and polar (B) part of the left thalamus.
and stereognosis. Vibratory and postural sensations were decreased distally in the 4 limbs, with some degree of imbalance on Romberg’s manoeuvre and tandem gait. Blood pressure was 160/90 mm Hg, with a pulse of 72/min. The general examination was unremarkable.

Neuropsychological Examination

The patient was well-oriented in time and place, with a normal level of consciousness. His spontaneous speech showed no abnormality, with normal output. Naming was within normal ranges (60/75 Boston naming test). The repetition of isolated, or series of, phonemes or words and of short and long sentences was fully preserved. Auditory comprehension showed very mild difficulties (32/36 Token test). Spontaneous writing and writing on dictation were normal, except for neglect of the left side of the page. The reading of words, non-words and a text was normal, with a good oral summary of the text meaning. On dichotic listening test, left-sided extinction was prominent (right ear: 25/30, left ear: 0/30 with 10/10 on monaural stimulation). Oral calculation was preserved (4/5), but written calculation was markedly impaired (0/4) because of loss of the correct spatial disposition of the operations and right-sided neglect. Bucco-linguo-facial praxies and limb (symbolic, imitation) praxies were normal. Spontaneous drawing of a cube was impaired (loss of perspective). Recognition of faces was preserved (43/54, Benton test). Tests for visual gnosis showed normal results (35/36, Ghent; 10/12, Columbia). The copy of Rey complex figure was impaired (16 points, centile 0). Orientation on a map was preserved, and no right-left confusion was found. Verbal learning was insufficient (Hebb’s recurring digits: span of 5, failure of span + 1 learning; 15 words of Rey: total of 22: 5-3-5-5-4, centile 0). Visual learning was more impaired (Corsi’s block-tapping: span of 4, failure of span + 1 learning; learning of 15 signs: total of 18: 2-3-4-5-4; 11/15 on recognition; delayed reproduction of Rey complex figure: immediate reproduction: 6 points, centile 0, and delayed reproduction: 2 points, centile 0). The Wisconsin card sorting test was much disturbed (0 series realized) and marked slowing was present on the Stroop test (1'39”). The results of the Raven PM 38 test were insufficient (15 points, centile 0).

Standard blood and urine tests were within normal ranges, except elevated cholesterol (8.2 mmole/l). EKG showed a left anterior hemiblock. The CSF was clear, with normal proteins (390 mg/1); 1 white cell/cm³ and negative VDRL and FTA-Abs. Somatosensory evoked potentials (median nerve stimulation) showed an altered morphology and reduced amplitude on the right parietal recording. A CT scan done one week after admission showed an isolated lucency in the anterior part of the right thalamus, suggestive of an infarct in the territory of the right thalamus (fig. 3).

Discussion

The thalamo-subthalamic paramedian artery has also been called the polar artery of the thalamus, the anterior thalamo-subthalamic paramedian artery, the anterointernal optic artery, and the premamillary pedicle.21,22,27 It originates from the middle third of the posterior communicating artery, but may be absent in 30-40% of the population,7,13 in which case its territory is supplied by the posterior thalamo-subthalamic paramedian artery originating from the first segment of the posterior cerebral artery (P, or basilar communicating artery.6,7,13 When the thalamo-subthalamic artery is present, it supplies the reticular nucleus, the mamillo-thalamic tract, part of the ventral lateral nucleus and of the dorsomedial nucleus, and the lateral aspect of the thalamic pole, but not the anterior nucleus.7,13 Three other systems supply the thalamus (fig. 4): 1) The posterior thalamo-subthalamic paramedian artery (or deep interpeduncular profund a artery,27 thalamoperforate or retro-mamillary pedicle2) is often unpaired and originates from P1. It supplies the paramedian part of the upper midbrain and of the thalamus (intralaminar nuclei, dorsomedial nucleus, and the lateral aspect of the thalamic pole, but not the anterior nucleus).7,13 2) The thalamogeniculate pedicle originates from the posterior cerebral artery after the level of the posterior communicating artery (P2) and includes the thalamic infero-lateral arteries and the pulvinar inferior lateral arteries.28 It supplies most of the ventral posterior nucleus, a part of the ventral lateral nucleus, and of the pulvinar.29 3) The posterior choroidal arteries leave the posterior cerebral artery at the same level as the thalamogeniculate branches and separate into a mesencephalo-posteromedial system and a hippocampo-posterolateral system,7,13 which supply the subthalamic region, part of the substantia nigra and

![Figure 3. Case 3, CT scan. Infarct of the anterior part of the right thalamus.](http://stroke.ahajournals.org/DownloadedFrom/index.html)
of the pulvinar, and the medial and lateral geniculate bodies.\textsuperscript{7, 13}

Suggestive clinical syndromes have been reported in unilateral infarcts in these three territories (table 1). The infarcts in the thalamogeniculate pedicle territory are the most common ones, and typically produce a sensory stroke which may or may not evolve towards a Dejerine-Roussy syndrome. The infarcts in the posterior thalamo-subthalamic paramedian artery territory are often bilateral, because this artery is often a single branch coming off the basilar communicating artery (butterfly-shaped infarcts\textsuperscript{13}). When there are 2 arteries, the infarct is unilateral. It typically gives rise to decreased consciousness, neuropsychological impairment, and oculomotor disturbances, sometimes delayed abnormal movements.\textsuperscript{7} Infarcts limited to the posterior choroidal system are very uncommon, and seem to produce partial visual field defects.\textsuperscript{41}

On the other hand, no study has been devoted to infarcts of the tuberothalamic artery territory. We

\begin{table}[h]
\centering
\begin{tabular}{|l|l|l|}
\hline
Territory of thalamic infarction & Origin of artery & Reported clinical features \\
\hline
Posterior thalamo-subthalamic & Initial segment of the PCA = basilar communicating artery (P\textsubscript{1}) & consciousness, confusion, behavioral changes\textsuperscript{7} \\
paramedian artery & & asterixis\textsuperscript{30} \\
& & motor hemineglect\textsuperscript{8, 31} \\
& & memory impairment\textsuperscript{7, 8} \\
& & language disturbances (dominant thalamus)\textsuperscript{8, 32, 33} \\
& & contralateral cerebellar dysfunction\textsuperscript{34} \\
& & faciobrachial tactile hypoesthesia\textsuperscript{8} \\
& & upgaze palsy,\textsuperscript{7, 8} vertical 1\frac{1}{2} syndrome\textsuperscript{59} \\
& & Weber's & Claude's third nerve fascicular syndromes\textsuperscript{7, 36} \\
& & delayed abnormal movements\textsuperscript{7} \\
& & intralaminar nuclei \\
Thalamo-geniculate pedicle & Second segment of the PCA (P\textsubscript{2}) & hemisensory syndromes: Dejerine-Roussy syndrome,\textsuperscript{1, 2} pure sensory stroke,\textsuperscript{4} cheiro (podo) oral/pseudo radicular variants\textsuperscript{3} \\
& & VPL, VPM \\
& & motor hemineglect (non-dominant thalamus)\textsuperscript{37} \\
& & contralateral cerebellar dysfunction\textsuperscript{38} \\
& & ataxic hemiparesis\textsuperscript{59} \\
& & language disturbances (dominant thalamus)\textsuperscript{40} \\
& & VL, pulvinar \\
Posterior choroidal arteries & Second segment of the PCA (P\textsubscript{2}) & homonymous horizontal sectoranopia\textsuperscript{41} \\
& & lateral geniculate body \\
\hline
\end{tabular}
\caption{Reported Syndromes in Unilateral Thalamic Infarction}
\end{table}
could identify only 5 published cases of isolated unilateral infarcts in this territory, with CT scan and neuro-
logical and neuropsychological assessment (table 2). Percheron13 mentioned a personal case with pathological
study, but the clinical picture was unknown. Three
more cases studied by CT scan were reported,11, 42 but
they had an associated infarct in the ipsilateral occipital lobe which obscured the symptomatology. Bilateral
infarcts limited to this territory also seem to be very
rare.43 44 In the cases where the tuberothalamic artery
is absent, its territory is supplied by the posterior
thalamo-subthalamic paramedian artery and infarcts
then involve the whole paramedian area, unilaterally45
or bilaterally.46

The study of our 3 cases and of 5 published cases
with a unilateral infarct limited to the tuberothalamic
artery territory (table 2) shows that delineation of a
suggestive syndrome is possible. The onset is usually
sudden, with mild to moderate contralateral motor
weakness or clumsiness (86%). A contralateral "emo-
tional" facial weakness with preserved voluntary con-
traction is frequently found, as formerly reported.11
Contralateral sensory changes may occur (43%), but
involve only the superficial modalities of sensation and
remain mild. Neuropsychological dysfunction is pres-
ent in 100% of the cases. The patients are withdrawn,
but not drowsy and show apathy, lack of spontaneity
and perseverations, especially in the cases with right-
side infarction. They can be disoriented (28%). In infar-
cents of the dominant thalamus, speech disturbances
are associated with visuo-verbal memory impairment,
and sometimes acalculia. The language disturbances
do not seem to be different from those described in
other types of thalamic infarction involving the lateral
formation40 or the posterior paramedian area,8 32 33 and
suggest transcortical aphasia: speech is reduced, with
anomia and some phonemic/semantic paraphasias and
occasionally neologisms (case 2), with slightly im-
paired or normal comprehension and normal repeti-
tion. The memory disturbances involve both the verbal
and visual items, with learning difficulties. In infarcts
of the non-dominant thalamus, contralateral hemispas-
tial neglect may be prominent, with memory impair-
ment involving mainly the visual learning and altered
visuospatial processing with constructional dyspraxia.

Since no cases with pathological verification are
available, except one with unknown symptomatology,
19 clinical-anatomic correlations are limited to specula-
tions based on other types of thalamic infarcts
or diseases, or on electrocoagulation studies. Lan-
guage disturbances in the setting of a thalamic lesion
have been attributed to involvement of the ventral lat-
eral nucleus,60 47 49 pulvinar,40 47 49 or dorsomedial nu-
cleus.40 50 53 In a case of unilateral left posterior tha-

**Table 2** Unilateral Infarction of the Tuberothalamic Artery Territory: Summary of Clinical Features

<table>
<thead>
<tr>
<th>Authors</th>
<th>Sex</th>
<th>Age</th>
<th>Risks factors</th>
<th>Side</th>
<th>Onset</th>
<th>Sensory changes</th>
<th>Neuropsychological examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cohen et al9 1980</td>
<td>♂</td>
<td>62</td>
<td>DM</td>
<td>L</td>
<td>right-sided weakness and speech disturbances (sudden)</td>
<td>( + ) phonemic paraphasias, anomia, slight of comprehension, normal repetition</td>
<td></td>
</tr>
<tr>
<td>Archer et al10 1981</td>
<td>♂</td>
<td>57</td>
<td>?</td>
<td>L</td>
<td>transient right-sided weakness and persisting speech disturbances (sudden)</td>
<td>- paraphasias; ↓ of comprehension, normal repetition, perseverations, ↓ visual/verbal memory</td>
<td></td>
</tr>
<tr>
<td>Graff-Radford et al11 1984 (case 3)</td>
<td>♂</td>
<td>75</td>
<td>HT</td>
<td>L</td>
<td>subacute change of personality, depression</td>
<td>- disorientation, aspontaneity of anomia, normal repetition, ↓ visual/verbal memory</td>
<td></td>
</tr>
<tr>
<td>Graff-Radford et al11 1984 (case 5)</td>
<td>♀</td>
<td>64</td>
<td>HT multiple aneurysms</td>
<td>R</td>
<td>left-sided weakness and hemineglect (sudden)</td>
<td>- disorientation, normal language, ↓ nonverbal memory, ↓ visuospatial processing</td>
<td></td>
</tr>
<tr>
<td>Biller et al12</td>
<td>♂</td>
<td>29</td>
<td>lupus anticoagulant</td>
<td>L</td>
<td>transient right-sided weakness and numbness, and dysarthria, followed by progressive memory impairment</td>
<td>- disorientation to time, ↓ memory, normal speech but monotone voice, omission of words when reading</td>
<td></td>
</tr>
<tr>
<td>Present study (case 1)</td>
<td>♀</td>
<td>45</td>
<td>hypercholesterolaemia</td>
<td>L</td>
<td>dizziness, vomiting, right-sided facial numbness and hand clumsiness, speech disturbances (sudden)</td>
<td>( + ) paraphasias, aspontaneity of speech, anomia, normal comprehension and repetition, ↓ visual/verbal memory, perseverations, acalculia</td>
<td></td>
</tr>
<tr>
<td>Present study (case 2)</td>
<td>♂</td>
<td>74</td>
<td>HT</td>
<td>L</td>
<td>sudden speech disturbances followed by right-sided weakness</td>
<td>- paraphasias and neologisms, anomia, slight ↓ of comprehension, normal repetition, ↓ visual/verbal memory, perseverations</td>
<td></td>
</tr>
<tr>
<td>Present study (case 3)</td>
<td>♂</td>
<td>68</td>
<td>DM</td>
<td>R</td>
<td>fatigue, transient blurring of vision, persisting left-sided weakness (sudden)</td>
<td>( + ) normal language, multimodal left-sided hemineglect, constructional apraxia, visual more than verbal memory impairment, &quot;frontal&quot; syndrome</td>
<td></td>
</tr>
</tbody>
</table>

DM = diabetes mellitus; HT = hypertension; ( + ) = slight; + = marked; - = absent; R = right; L = left; ↓ = decrease.
lamo-subthalamic paramedian artery territory infarct, which was pathologically studied, we found that the language disturbances and the impairment of verbal memory correlated well with involvement of the dorsomedial nucleus, in the absence of significant involvement of the ventral lateral nucleus, pulvinar and mamillothalamic tract. In the present case, involvement of the dorsomedial nucleus also seems to correlate the best with the dysphasic and memory disturbances, because of the arterial supply topography of the tuberenthalamic artery. The mamillothalamic tract is usually supplied by this artery and may also participate in the memory disturbances. Involvement of the ventral lateral nucleus may also account for at least some of the aphasic deficits, and probably explains the occurrence of "emotional" facial paresis and contralateral neglect in nondominant side infarction. Contralateral neglect may also be produced by a lesion of the intralaminar nuclei, but these nuclei are not supplied by the tuberenthalamic artery. The occasional presence of only mild sensory changes indicates that the ventral posterior nucleus is mostly spared. Initial transient or mild contralateral weakness probably corresponds to continuous involvement of the internal capsule by edema or pressure during the acute ischemic phase, and it is not similar to the severe hemiplegia produced by peduncular involvement in infarcts of the posterior thalamo-subthalamic paramedian or circumferential arteries, or to the hemiplegic syndrome of the posterior cerebral artery.

Many of the neuropsychological disturbances found in this syndrome do not markedly differ from those reported in unilateral infarcts of the posterior thalamo-subthalamic paramedian artery territory, probably because of vascular supply overlap in structures like the dorsomedial nucleus. However, infarcts of the tuberenthalamic artery territory should be differentiated from those of the posterior thalamo-subthalamic paramedian artery territory, because they do not produce altered consciousness, supranuclear upgaze palsy, intra-axial third nerve palsy, severe contralateral hemiplegia or delayed abnormal movements, as they do not involve the intralaminar nuclei and the upper midbrain. Moreover, as the posterior communicating artery — from where the tuberenthalamic artery originates — is filled where the tuberothalamic artery originates, the occasional absence of the posterior cerebral artery territory, because they do not produce altered consciousness, supranuclear upgaze palsy, sustained downgaze, aphasia, amnesia, and motor neglect. J Neurol Neurosurg Psychiatr (in press)"
SYNDROME — UNILATERAL INFARCT TUBEROthalamic ARtery TERRITORY/Bogousslavsky et al 441

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J Bogousslavsky, F Regli and G Assal

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