

The Syndrome of Unilateral Tuberothalamic Artery Territory Infarction

JULIEN BOGOUSLAVSKY, 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 visuo-spatial 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.

Stroke Vol 17, No 3, 1986

THE CLINICAL SYNDROMES of thalamic infarction in the territory of the thalamogeniculate pedicle¹⁻⁴ and in the territory of the posterior thalamo-subthalamic paramedian artery^{2, 3, 5-8} are well recognized. Infarcts in the tuberothalamic artery territory have occasionally been reported,⁹⁻¹² but they have not been specifically studied and no attempt has been made to differentiate their clinical picture from that of the other paramedian thalamic infarcts. We have studied neurologically and neuropsychologically three patients with a unilateral infarct limited to the tuberothalamic artery territory confirmed on CT scan. The presence of certain findings characteristically associated with the absence of other findings suggests that this type of thalamic infarct can be recognized.

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 gnosis 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,^{21, 22} Rey Auditory Learning¹⁶), 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 oculomotricity were normal. A slight light touch hypoesthesia involving the right side of the face down to the C₂ 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 Neurophysiology, Centre Hospitalier Universitaire Vaudois, Lausanne, Switzerland.

Address correspondence to: Dr. Julien Bogousslavsky, Department of Neurology, CHUV, 1011 Lausanne, Switzerland.

Received June 3, 1985; revision #1 accepted August 29, 1985.

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 perseverate. 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 autocorrections) compared with word reading (1'3").

EKG and Standard blood and urine tests were normal. An EEG did not show any abnormality. A CT scan performed 4 days after admission showed an isolated lucency in the anterior paramedian part of the left thalamus, suggesting a recent infarct in the territory of the tuberothalamic artery (fig. 1).

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

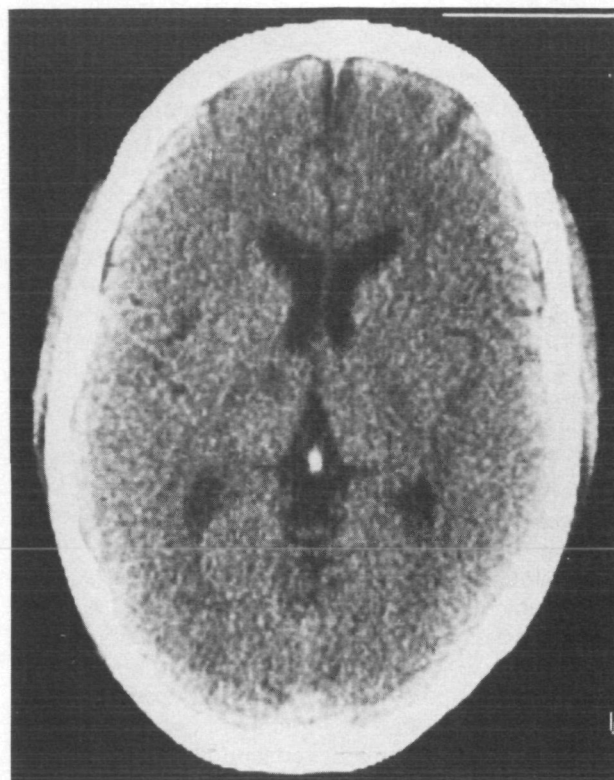


FIGURE 1. Case 1, CT scan. Small infarct in the anterior part of the left thalamus.

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 dysidiadochokinesia 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 preservations 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 paraphasias and neologisms, with preserved copying. Writing on dictation was impaired, with paraphasias 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 hypoacusis 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

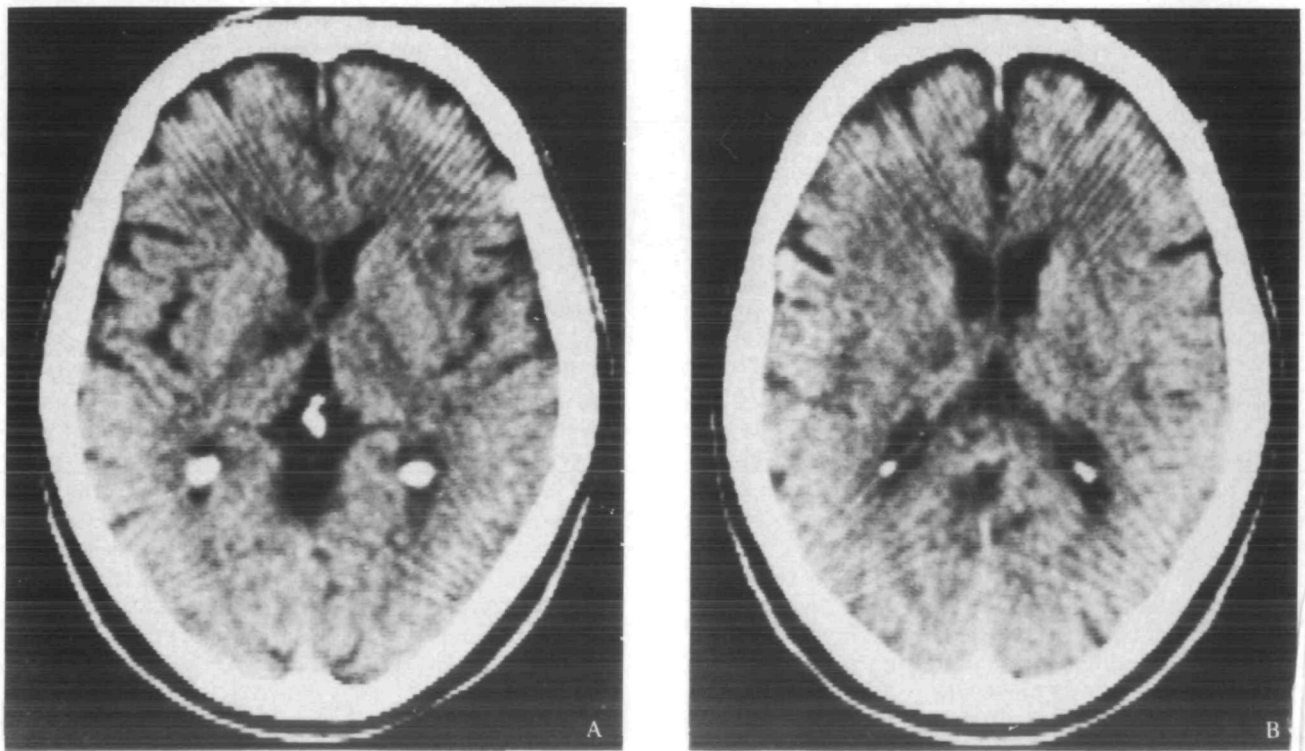


FIGURE 2. Case 2, CT scan. Infarct of the anterolateral (A) and polar (B) part of the left thalamus.

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 praxias and limb (symbolic, imitation) praxias 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/l); 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 tuberothalamic artery (fig. 3).

Discussion

The tuberothalamic 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.^{2, 13, 27, 28} 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 (P₁ or basilar communicating artery.^{6, 7, 13} When the tuberothalamic 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 profunda artery,²⁷ thalamoperforate or retromamillary 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, internal part of the ventral posterior 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 pulvinarian inferolateral arteries.²⁹ It supplies most of the ventral posterior nucleus, a part of the ventral lateral nucleus, and of the pulvinar.²⁹ 3) *The posterior chorioidal 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

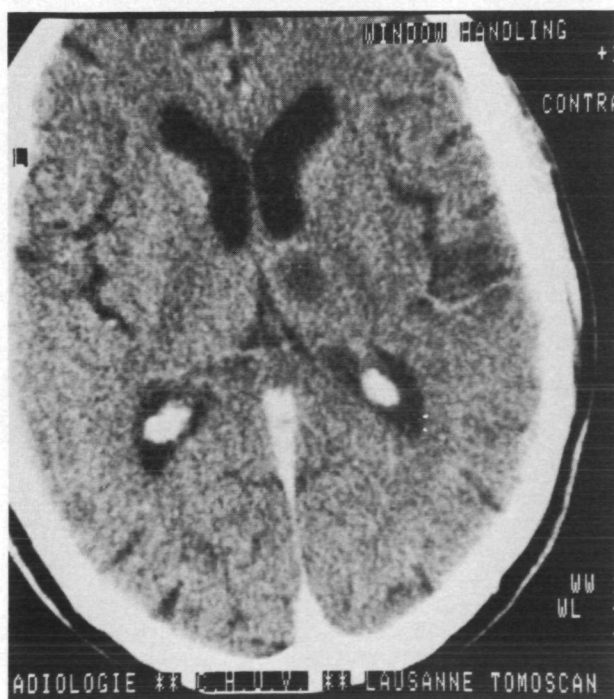


FIGURE 3. Case 3, CT scan. Infarct of the anterior part of the right thalamus.

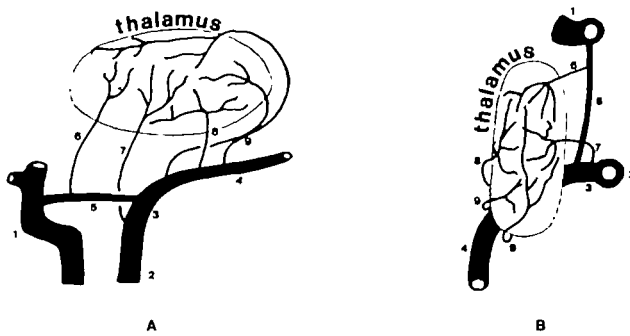


FIGURE 4. Schematic representation of arterial supply to the thalamus after Lazorthes⁵⁵, Schlesinger²⁷ and Castaigne *et al*⁷, A) Lateral view, B) Dorsal view. 1: carotid siphon, 2: basilar artery, 3: basilar communicating artery (P₁), 4: posterior cerebral artery (P₂), 5: posterior communicating artery, 6: tuberothalamic artery, 7: posterior thalamo-subthalamic paramedian artery, 8: thalamo-geniculate pedicle, 9: posterior choroidal arteries.

of the pulvinar, and the medial and lateral geniculate bodies.^{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 infarcts¹³). 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.⁷ Infarcts limited to the posterior choroidal system are very uncommon, and seem to produce partial visual field defects.⁴¹

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

TABLE 1 Reported Syndromes in Unilateral Thalamic Infarction

Territory of thalamic infarction	Origin of artery	Reported clinical features
Posterior thalamo-subthalamic paramedian artery	Initial segment of the PCA = basilar communicating artery (P ₁)	↓ consciousness, confusion, behavioral changes ⁷
		asterixis ³⁰
		motor hemineglect ^{8, 31}
		memory impairment ^{7, 8}
		language disturbances (dominant thalamus) ^{8, 32, 33}
		contralateral cerebellar dysfunction ³⁴
		faciobrachial tactile hypoesthesia ⁸
Thalamo-geniculate pedicle	Second segment of the PCA (P ₂)	upgaze palsy, ^{7, 8} vertical 1½ syndrome ³⁵
		Weber's & Claude's third nerve fascicular syndromes ^{7, 36}
		delayed abnormal movements ⁷
Posterior choroidal arteries	Second segment of the PCA (P ₂)	hemisensory syndromes: Dejerine-Roussy syndrome, ^{1, 2} pure sensory stroke, ⁴ cheiro (podo) oral/pseudo radicular variants ³
		motor hemineglect (non-dominant thalamus) ³⁷
		contralateral cerebellar dysfunction ³⁸
		ataxic hemiparesis ³⁹
		language disturbances (dominant thalamus) ⁴⁰
		homonymous horizontal sectoranopia ⁴¹

↓ = decreased; DM = dorsomedial nucleus; VL = ventral lateral nucleus; VPM = ventral posterior medial nucleus; VPL = ventral posterior lateral nucleus.

could identify only 5 published cases of isolated unilateral infarcts in this territory, with CT scan and neurological 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 "emotional" facial weakness with preserved voluntary contraction 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 present 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 infarcts 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 impaired or normal comprehension and normal repetition. The memory disturbances involve both the verbal and visual items, with learning difficulties. In infarcts of the non-dominant thalamus, contralateral hemispatial neglect may be prominent, with memory impairment 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,¹³ clinical-anatomic correlations are limited to speculations based on other types of thalamic infarcts or diseases, or on electrocoagulation studies. Language disturbances in the setting of a thalamic lesion have been attributed to involvement of the ventral lateral nucleus,^{40, 47-49} pulvinar,^{40, 47, 49} or dorsomedial nucleus.^{40, 50-53} In a case of unilateral left posterior tha-

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

Authors	Sex	Age	Risks factors	Side	Onset	Sensory changes	Neuropsychological examination
Cohen et al ⁹ 1980	♂	62	DM	L	right-sided weakness and speech disturbances (sudden)	(+)	phonemic paraphasias, anomia, slight of comprehension, normal repetition
Archer et al ¹⁰ 1981	♂	57	?	L	transient right-sided weakness and persisting speech disturbances (sudden)	-	paraphasias, ↓ of comprehension, normal repetition, perseverations, ↓ visual/verbal memory
Graff-Radford et al ¹¹ 1984 (case 3)	♂	75	HT	L	subacute change of personality, depression	-	disorientation, aspontaneity of anomia, normal repetition, ↓ visual/verbal memory
Graff-Radford et al 1984 (case 5)	♀	64	HT multiple aneurysms	R	left-sided weakness and hemineglect (sudden)	-	disorientation, normal language, ↓ nonverbal memory, ↓ visuospatial processing
Billier et al ¹²	♂	29	lupus anticoagulant	L	transient right-sided weakness and numbness, and dysarthria, followed by progressive memory impairment	-	disorientation to time, ↓ memory, normal speech but monotone voice, omission of words when reading
Present study (case 1)	♀	45	hypercholesterolaemia	L	dizziness, vomiting, right-sided facial numbness and hand clumsiness, speech disturbances (sudden)	(+)	paraphasias, aspontaneity of speech, anomia, normal comprehension and repetition, ↓ visual/verbal memory, perseverations, acalculia
Present study (case 2)	♂	74	HT	L	sudden speech disturbances followed by right-sided weakness	-	paraphasias and neologisms, anomia, slight ↓ of comprehension, normal repetition, ↓ visual/verbal memory, perseverations
Present study (case 3)	♂	68	DM	R	fatigue, transient blurring of vision, persisting left-sided weakness (sudden)	(+)	normal language, multimodal left-sided hemineglect, constructional apraxia, visual more than verbal memory impairment, "frontal" syndrome

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.⁸ 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 tuberothalamic artery. The mamillothalamic tract is usually supplied by this artery¹³ 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,^{40, 47, 49} 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,⁸ but these nuclei are not supplied by the tuberothalamic 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,³⁹ and it is not similar to the severe hemiplegia produced by peduncular involvement in infarcts of the posterior thalamo-subthalamic paramedian or circumferential arteries,^{7, 36} or to the hemiplegic syndrome of the posterior cerebral artery.⁵⁴

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 tuberothalamic 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 tuberothalamic artery originates — is filled by the internal carotid artery in as many as 40% of the hemispheres,⁵⁵ recognition of this syndrome among the other syndromes of thalamic infarction is important, because it may occasionally be related to carotid system disease. One case of thalamic infarction was reported after carotid artery occlusion,⁵⁶ but the configuration of the posterior cerebral artery was not described. An angiographic study of the pattern of the circle of Willis in thalamic infarction still remains to be done.

In summary, we believe that tuberothalamic artery territory infarcts can be differentiated from the other types of thalamic infarcts by their clinical aspects and CT scan pictures. However, since no cases with clinico-anatomic correlation have yet been reported, we think that the hypotheses advanced for the explanation

of the symptomatology should, for the moment, be accepted with reservation, because they are based on pathologic studies of other thalamic syndromes.

Addendum

Another case of unilateral left infarction in the territory of the tuberothalamic artery has been reported by Gorelick et al (Gorelick PB, Hier DB, Benevento L, Levitt S, Tan W: Aphasia after left thalamic infarction. *Arch Neurol* **41**: 1296–1298, 1984), with a detailed analysis of speech disturbances.

Acknowledgment

We would like to thank Andrew Kertesz, MD, University of Western Ontario, for his helpful comments on the manuscript.

References

- Dejerine J, Roussy G: Le syndrome thalamique. *Rev Neurol* **14**: 521–532, 1906
- Foix C, Hillemand P: Les syndromes de la région thalamique. *Press Méd* **33**: 113–117, 1925
- Lapresle J, Hagueneau M: Anatomico-clinical correlation in focal thalamic lesions. *J Neurol* **205**: 29–46, 1973
- Fisher CM: Thalamic pure sensory stroke: A pathologic study. *Neurology* **28**: 1141–1144, 1978
- Castaigne P, Buge A, Cambier J, Escourrolle R, Brunet P, Degos JD: Démence thalamique d'origine vasculaire par ramollissement bilatéral limité au territoire du pédoncule rétro-mamillaire. A propos de deux observations anatomo-cliniques. *Rev Neurol* **114**: 89–107, 1966
- Segarra JM: Cerebral vascular disease and behavior. I. The syndrome of the mesencephalic artery (basilar artery bifurcation). *Arch Neurol* **22**: 408–418, 1970
- Castaigne P, Lhermitte F, Buge A, Escourrolle R, Hauw JJ, Lyon-Caen O: Paramedian thalamic and midbrain infarcts: clinical and neuropathological study. *Ann Neurol* **10**: 127–148, 1981
- Bogousslavsky J, Miklossy J, Deruaz JP, Regli F, Assal G: Unilateral left paramedian infarction of thalamus and midbrain: clinico-pathologic study of upgaze palsy, sustained downgaze, aphasia, amnesia, and motor neglect. *J Neurol Neurosurg Psychiatr* (in press)
- Cohen JA, Gelfer CE, Sweet RD: Thalamic infarction producing aphasia. *Mt Sinai J Med* **47**: 398–404, 1980
- Archer CR, Ilinsky IA, Goldfader PR, Smith FR: Aphasia in thalamic stroke. CT stereotactic localization. *J Comput Assist Tomogr* **5**: 427–432, 1981
- Graff-Radford NR, Eslinger PJ, Damasio AR, Yamada T: Non-hemorrhagic infarction of the thalamus: Behavioral, anatomic, and physiologic correlates. *Neurology* **34**: 14–23, 1984
- Biller J, Merchut M, Emanuele MA: Nonhemorrhagic infarction of the thalamus. *Neurology* **34**: 1269–1270, 1984
- Percheron G: Les artères du thalamus humain. I. Artère et territoire thalamiques polaires de l'artère communicante postérieure. II. Artères et territoires thalamiques paramédians de l'artère basilaire communicante. *Rev Neurol* **134**: 297–307 & 309–324, 1976
- Kaplan E, Goodglass H, Weintraub S: Boston naming memory test. Boston, Experimental edition 1976
- De Renzi E, Vignolo LA: The Token test: A sensitive test to detect receptive disturbances in aphasia. *Brain* **85**: 665–678, 1962
- Rey A: L'examen clinique en psychologie. Paris, Presses Universitaires de France, 1965
- Benton AL, Van Allen MW: Impairment of facial recognition in patients with central disease. *Cortex* **4**: 344–358, 1968
- Masure MC, Tzavaras A: Perception de figures entrecroisées par des sujets atteints de lésions corticales unilatérales. *Neuropsychologia* **14**: 371–374, 1976
- Poppelreuter W: Die psychischen Schädigungen durch Kopfschuss im Kriege 1914/16. Leipzig, Von Leopold Voss, 1917
- Lezak MD: Neuropsychological assessment. New York, Oxford University Press, 1983
- Milner B: Memory and the medial temporal regions of the brain. In:

- Pribram KH, Broadbent DE: *Biology of Memory*. New York, Academic Press, 1970
22. Milner B: Interhemispheric differences in the localization of psychological processes in man. *Br Med Bull* 27: 272–277, 1971
 23. Berg EA: A simple objective test for measuring flexibility in thinking. *J Gen Psychol* 39: 15–22, 1948
 24. Stroop JR: Studies of interference in serial verbal reactions. *J Exp Psychol* 18: 643–662, 1945
 25. Luria AR: Frontal lobe syndromes. In Vinken PJ, Bruyn GW: *Handbook of clinical neurology*, vol 2 (chapter 23), Amsterdam, Elsevier, 725–757, 1969
 26. Raven JC: *Guide to the Standard Progressive Matrices*. London, HK Lewis, 1960
 27. Schlesinger B: The upper brainstem in the human: its nuclear configuration and vascular supply. New York, Springer, 1976
 28. Duret H: Recherches anatomiques sur la circulation de l'encéphale. *Arch Physiol Norm Pathol*, 1: 60–80, 2: 80–91, 5: 664–693, 6: 919–957, 1874
 29. Percheron G: The anatomy of the arterial supply of the human thalamus and its use for the interpretation of the thalamic vascular pathology. *Rev Neurol* 205: 1–13, 1973
 30. Feldmeyer JJ, Bogousslavsky J, Regli F: Asterixis uni- ou bilatéral en cas de lésion thalamique ou pariétale: un trouble moteur afférentiel? *Schweiz Med Wschr* 114: 167–171, 1984
 31. Watson RT, Valenstein EV, Heilman KM: Thalamic neglect. Possible role of the medial thalamus and nucleus reticularis in behavior. *Arch Neurol* 38: 501–506, 1981
 32. Molnar L: Die lokaldiagnostische Bedeutung der Vertikalen Blicklähmung; Beiträge zur Symptomatologie and Faseranatomie des meso-diencephalen Übergangsbereiches. *Arch Psychiatr Nervenkr* 198: 523–534, 1959
 33. Davous P, Bianco C, Duval-Lota AM, De Recondo J, Vedrenne C, Rondot P: Aphasie par infarctus thalamo-pédonculaire gauche. Observation anatomo-clinique. *Rev Neurol* 140: 711–719, 1984
 34. Garcin R, Lapresle J: Incoordination cérébelleuse du membre inférieur par lésion localisée de la région interne du thalamus contralatéral. *Rev Neurol* 120: 5–13, 1969
 35. Bogousslavsky J, Regli F: Atteinte intra-axiale du nerf moteur oculaire commun dans les infarctus mésencéphaliques. *Rev Neurol* 140: 263–270, 1984
 36. Bogousslavsky J, Regli F: Upgaze palsy and monocular paresis of downward gaze from ipsilateral thalamo-mesencephalic infarction: A vertical "one-and-a-half" syndrome. *J Neurol* 231: 43–45, 1984
 37. Laplane D, Escourolle R, Degos JD, Sauron B, Massiou H: La négligence motrice d'origine thalamique. A propos de 2 cas. *Rev Neurol* 138: 201–211, 1982
 38. Garcin R: Syndrome cérébellothalamique par lésion localisée du thalamus. Avec une digression sur le "signe de la main creuse" et son intérêt sémiologique. *Rev Neurol* 93: 143–149, 1955
 39. Bogousslavsky J, Regli F, Ghika J, Feldmeyer JJ: Painful ataxic hemiparesis. *Arch Neurol* 41: 892–893, 1984
 40. McFarling D, Rothi LJ, Heilman KM: Transcortical aphasia from ischaemic infarcts of the thalamus: a report of two cases. *J Neurol Neurosurg Psychiatr* 45: 107–112, 1982
 41. Frisén L, Holmegaard L, Rosencrantz M: Sectorial optic atrophy and homonymous, horizontal sectoranopia: A lateral choroidal artery syndrome? *J Neurol Neurosurg Psychiatr* 41: 374–380, 1978
 42. Cambier J, Masson M, Graveleau PH, Elghozi D: Sémiologie de négligence lors des lésions ischémiques dans le territoire de l'artère cérébrale postérieure droite. Rôle de la lésion thalamique. *Rev Neurol* 138: 631–648, 1982
 43. Schuster P: Beitrag zur Pathologie des Thalamus opticus. *Arch Psychiatr Nervenkr* 105: 353–432, 1936
 44. Barbizet J, Degos JD, Louarn F, Ngyen JP, Mas JL: Amnésie par lésion ischémique bithalamique. *Rev Neurol* 137: 415–424, 1981
 45. Michel D, Laurmet B, Foyatier N, Blanc A, Portafaix M: Infarctus thalamique paramédian gauche. Etude de la mémoire et du langage. *Rev Neurol* 138: 533–550, 1982
 46. Schott B, Manguière F, Laurent B, Serclerat O, Fischer C: L'amnésie thalamique. *Rev Neurol* 136: 117–130, 1980
 47. Krayenbühl H, Siegfried J, Kohenof M, Yasargil MG: Is there a dominant thalamus? *Confin Neurol* 26: 246–249, 1965
 48. Ojemann GA: Subcortical language mechanisms. In Whitaker H, Whitaker HA: *Neurolinguistics*, vol 1, New York, Academic Press 103–138, 1976
 49. Mazaux JM, Orgogozo JM, Henry P, Loiseau P: Troubles du langage au cours des lésions thalamiques. Etude par le test de Goodglass et Kaplan. *Rev Neurol* 135: 59–64, 1979
 50. Victor M, Adams RD, Collins GH: *The Wernicke-Korsakoff Syndrome*. Philadelphia, FA Davis, 1971
 51. Squire LR, Moore RH: Dorsal thalamic lesion in a noted case of human memory dysfunction. *Ann Neurol* 6: 503–506, 1979
 52. Speedie LJ, Heilman KM: Anterograde memory deficits for visuospatial material after infarction of the right thalamus. *Arch Neurol* 40: 183–186, 1983
 53. Goldenberg GG, Wimmer A, Maly J: Amnesic syndrome with a unilateral thalamic lesion: a case report. *J Neurol* 229: 79–86, 1983
 54. Benson DF, Tomlinson EB: Hemiplegic syndrome of the posterior cerebral artery. *Stroke* 2: 559–564, 1971
 55. Zeal AA, Rhoton AL Jr: Microsurgical anatomy of the posterior cerebral artery. *J Neurosurg* 48: 534–559, 1978
 56. Clinton GL, Fox MJ: Central pain syndrome. A review of the literature and a case report. *Wis Med J* 57: 169–192, 1958