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# Thalamic ataxia

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**Summary.** Seventeen patients with hemiataxia as a manifestation of thalamic infarction were studied. Hemiataxia had the main clinical characteristics of a "cerebellar type" of ataxia, though it never occurred in isolation, being associated with ipsilateral sensory disturbance (hemiataxia-hypaesthesia) in 7 patients, with ipsilateral sensory disturbance and hemiparesis (hypaesthetic ataxic hemiparesis) in 8 patients, and with hemiparesis (ataxic hemiparesis) in 2 patients. Recovery was good, and in all patients the sensory and motor disturbances improved or cleared before the hemiataxia. All patients had an infarct involving the lateral part of the thalamus (thalamogeniculate territory in 16, tuberothalamic territory in 1), also affecting the posterior limb of the internal capsule (PLIC) in 7 patients. Hemiataxia seemed linked to involvement of the caudal part of the ventral lateral nucleus of the thalamus or the immediately adjacent medial part of the PLIC. These structures are near the corticospinal pathways and the ventral posterior nucleus of the thalamus, explaining why hemiataxia is associated with hemiparesis or hypaesthesia in this type of infarct.

**Key words:** Thalamus – Stroke – Cerebral infarct – Ataxia

## Introduction

Ataxia is classically attributed to lesions involving the cerebellar system or the sensory pathways that control movement [1]. Fisher [15, 18] introduced the term "ataxic hemiparesis" and suggested that when associated with hemiparesis, ataxia may be the result of an infarct in the pons, corona radiata or internal capsule.

Ataxia may not be uncommon in thalamic vascular lesions [4, 8, 14, 19, 21, 29, 31, 39, 40], but it has not been studied specifically. We studied a series of 17 patients with ataxia as a minifestation of thalamic infarction.

## Patients and methods

The 17 patients had hemiataxia and acute thalamic infarction and were selected consecutively from all patients with thalamic infarction on CT or MRI admitted to two primary care stroke centres (Lausanne Stroke Programme of the Department of Neurology, Centre Hospitalier Universitaire Vaudois and Department of Neurology, Centre Hospitalier Universitaire Jean Minjoz, Besançon). The Lausanne patients were selected between 1986 and 1990 and were part of the prospective Lausanne Stroke Registry [7]. The Besançon patients were selected between 1985 and 1990, also being part of a prospective stroke registry.

All patients were examined by at least one of the authors. Hemiataxia was defined as limb incoordination with dysmetria, which may also include hypometria, hypermetria, tremor, or dysdiadochokinesia [2].

Records were made of stroke onset and characteristics, previous transient ischaemic attacks (TIAs), heart disease and risk factors (hypertension: blood pressure higher than 160/90 mmHg at least twice before the stroke; diabetes mellitus: known fasting blood glucose > 6 mmol/l before the stroke; hypercholesterolaemia: fasting blood cholesterol higher than 6.5 mmol/l; current cigarette smoking) CT (with and without contrast) or MRI was performed at least once within 1 month of the stroke. The topographic diagnosis of the infarct was made by following the lesion mapping templates developed in the Lausanne Stroke Registry and elsewhere [6, 11, 36]. The maximum diameter was determined independently by two of the authors. All patients had Doppler ultrasonography, ECG and standard blood and urine test. Angiography was performed in 5 patients, echocardiography in 5 patients and cerebrospinal fluid (CSF) analysis in 3 patients.

Large-vessel was considered to be the cause of the infarction when a patient had a stenosis of more than 50% in the appropriate large artery. We considered potential cardiac sources of embolism following criteria defined previously [7]. Small-vessel disease was considered likely in patients with hypertension or diabetes mellitus, in the absence of potential arterial or cardiac sources of embolism.

Follow-up data were obtained from the outpatient clinic between 1 and 12 months after stroke.

## Results

Nine patients were selected by the Besançon centre and 8 by the Lausanne centre. The patients belonged to a group of 66 patients with thalamic infarction on CT or MRI who were seen during the study period (25.8%). There were 10 men and 7 women, with a median age of 76 years (range 38–87). Eight patients had a left-sided infarct and 9 had a right-sided infarct.

Thirteen patients had both CT and MRI, while 4 patients had only CT.

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**Table 1.** Risk factors/presumed aetiology. CS, Cigarette smoking; DM, diabetes mellitus; HC, hypercholesterolaemia; HT, hypertension; AF, atrial fibrillation; PCA, posterior cerebral artery; rICA, right internal carotid artery

| Patient no. | Risk factors | Heart disease | Angiography | Aetiology |
|---|---|---|---|---|
| *Hemiataxia-hypaesthesia* | | | | |
| 1 | CS, HC, DM | – | – | Meningovascular syphilis |
| 2 | HT | – | PCA occlusion | Large-vessel disease |
| 3 | HT, CS, HC | Intra-atrial thrombus | – | Cardioembolism |
| 4 | – | – | – | – |
| 5 | HT, HC | – | – | Small-vessel disease |
| 6 | HT, DM | – | – | Small-vessel disease |
| 7 | HT, HC | AF | – | Cardioembolism |
| *Hypaesthetic ataxia hemiparesis* | | | | |
| 8 | – | AF, left atrial enlargement | – | Cardioembolism |
| 9 | HT, DM | – | – | Small-vessel disease |
| 10 | HT, DM, HC | AF, angor | – | Cardioembolism |
| 11 | HT | – | rICA stenosis > 50% | Small-vessel disease |
| 12 | DM, HC | – | – | Small-vessel disease |
| 13 | HT, DM, CS | – | rICA stenosis > 50% | Small-vessel disease |
| 14 | – | – | – | – |
| 15 | HT | – | Subclavian stenosis > 50% | Large-vessel disease |
| *Ataxic hemiparesis* | | | | |
| 16 | HT, HC | AF | – | Cardioembolism |
| 17 | HT, CS | – | – | Small-vessel disease |

**Table 2.** Clinical findings. P, progressive; S, sudden; sj, purely subjective; &, pain; F, face; U, upper limb; L, lower limb

| Patient no./ age (years)/ sex | Side | TIA | Onset | Head-ache | Ataxia | Hypaes-thesia | Paresis | Follow-up (months) |
|---|---|---|---|---|---|---|---|---|
| 1/38/M | Right | Yes | P (6 h) | + | UL | FU[a] | No | 12 |
| 2/68/M | Left | No | P (7 h) | + | UL | FUL[b] | No | 1 |
| 3/42/M | Left | No | P (2 h) | – | UL | FUL& | No | 0.5 |
| 4/82/F | Left | No | S | + | UL | FUL[a] | No | 4 |
| 5/76/M | Right | No | P (24 h) | – | UL | FUL | No | 0.5 |
| 6/66/F | Left | No | P (12 h) | + | UL | L(sj) | No | 1 |
| 7/80/F | Right | No | S | – | UL | UL | No | 1 |
| *Hypaesthetic ataxic hemiparesis* | | | | | | | | |
| 8/77/M | Right | No | S | – | UL | FUL | FUL | 2 |
| 9/87/F | Right | No | S | – | UL | FUL | FUL | 2 |
| 10/63/M | Left | No | S | – | UL | FUL | FU | 2 |
| 11/68/M | Right | No | P (12 h) | – | UL | FUL[c] | UL | 1 |
| 12/82/F | Left | No | P (4 h) | – | UL | FUL | UL | 0.5 |
| 13/84/M | Right | No | S | – | UL | FUL(sj) | FUL | 1 |
| 14/81/F | Right | Yes | P (12 h) | – | UL | L(sj) | FUL | 2 |
| 15/67/F | Left | No | S | – | UL | FU(sj)& | UL | 6 |
| *Ataxic hemiparesis* | | | | | | | | |
| 16/85/M | Right | Yes | S | – | UL | No | FUL | 1 |
| 17/69/M | Left | Yes | S | + | UL | No | FUL | 1 |

[a] Vibration and position sense spared
[b] Vibration and position sense disturbed only in lower limb
[c] Purely subjective in lower limb

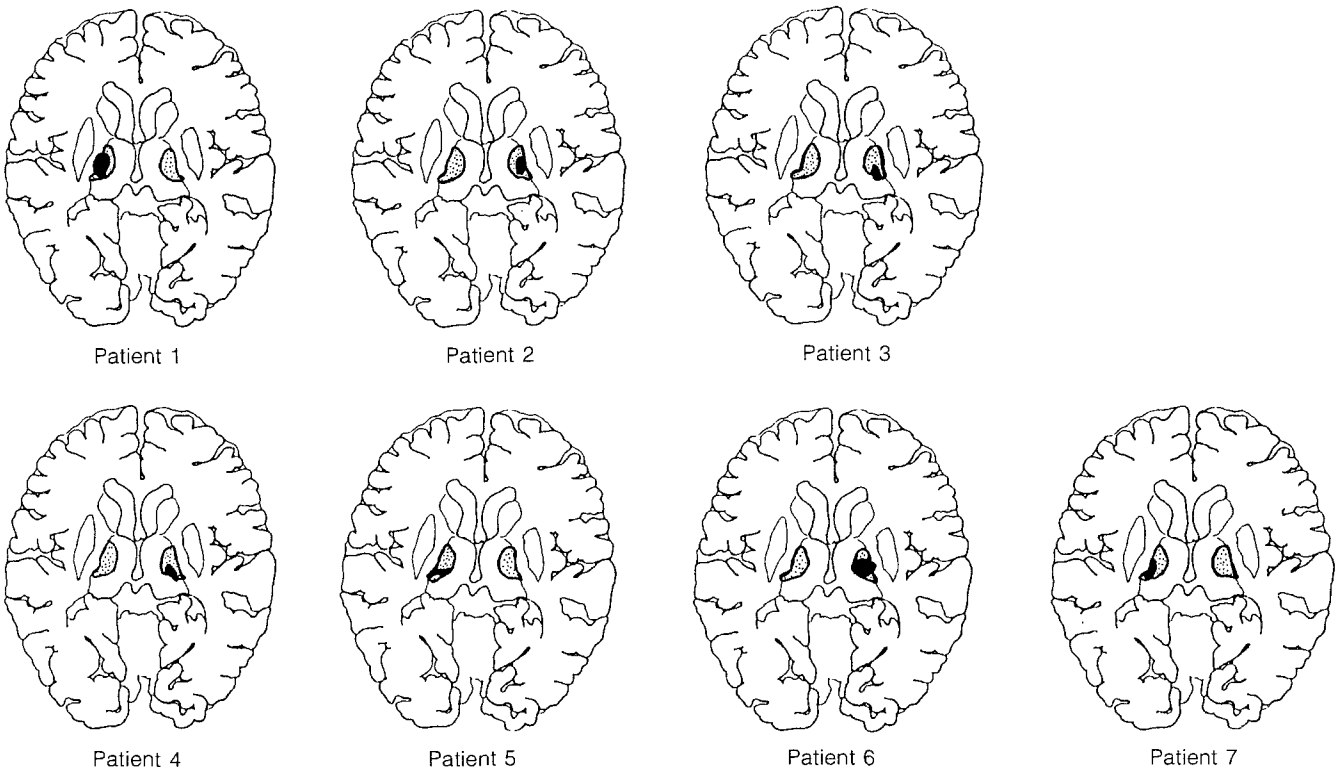


**Fig. 1.** Hemiataxia-hypaesthesia. Topography of infarcts on CT or MRI. ▨ Thalamogeniculate arteries territory; ▨ tuberothalamic arteries territory infarct

All patients had hemiataxia contralateral to the thalamic infarct, with incoordination on finger-to-nose, finger-to-finger and heel-to-knee tests, with normal initiation and velocity of movements, but with irregular accelerations and decelerations producing oscillations when near to the target, with a series of secondary corrective movements. This incoordination seemed unchanged with the eyes open or closed, and without improvement by visual clues [34]. Dysdiadochokinesia and Holmes rebound phenomenon were also present in all patients. Hemiataxia never occurred in isolation: 7 patients had hemiataxia with ipsilateral sensory disturbance [hemiataxia-hypaestesia (HH)], 8 had hemiataxia with ipsilateral sensory disturbance and hemiparesis [hypaesthetic ataxic hemiparesis (HAH)] and 2 had hemiataxia with hemiparesis [ataxic hemiparesis (AH)].

Risk factors and associated disease are summarized in Table 1. The most common presumed aetiology was small-vessel disease (in 7), followed by cardioembolism (in 5) (Table 1).

### *Hemiataxia-hypaesthesia*

Stroke progressed over 2–24 h in 5 patients and was sudden in 2 patients. Only one patient (no. 1) had a TIA (with symptoms identical to those of the infarct) a few hours before the infarct. At the time of infarction, 4 patients reported headache. Sensory disturbances were the first symptom in all patients with progressive onset, usually starting with prickling in the face and distally in one or both limbs. The hemisensory defect involved only touch, temperature, and pain sensation in 2 patients (nos.

**Table 3.** Location of thalamic infarcts. +, Showed the infarct; −+, only the 2nd CT showed the infarct; −, did not show the infarct; ND, not done; PLIC, posterior limb of the internal capsule; VL, ventral lateral nucleus; VP, ventral posterior nucleus; VA, ventral anterior nucleus

| Patient no. | CT/MRI | Thalamic location | PLIC extension | Size (mm) |
|---|---|---|---|---|
| *Hemiataxia-hypaesthesia* | | | | |
| 1 | +/ND | VL/VP | Yes | 12.5 |
| 2 | +/ND | VL/VP | No | 10 |
| 3 | +/+ | VL/VP | No | 10 |
| 4 | +/ND | VL/VP | No | 10 |
| 5 | −/+ | VL/VP | No | 7.5 |
| 6 | −+/ND | VL/VP | Yes | 9 |
| 7 | −+/+ | VL/VP | No | 10 |
| *Hypaesthetic ataxic hemiparesis* | | | | |
| 8 | −+/ND | VL/VP | Yes | 5 |
| 9 | −+/ND | VL/VP | No | 9 |
| 10 | −+/ND | VP | Yes | 9 |
| 11 | −+/ND | VL/VP | Yes | 8 |
| 12 | ND/+ | VL/VP | Yes | 12.5 |
| 13 | +/ND | VL/VP | Yes | 5 |
| 14 | −+/+ | VA/VL | No | 9 |
| 15 | −+/+ | VL/VP | No | 10 |
| *Ataxic hemiparesis* | | | | |
| 16 | −+/ND | VL/VP | No | 8 |
| 17 | +/ND | VL/VP | No | 9 |

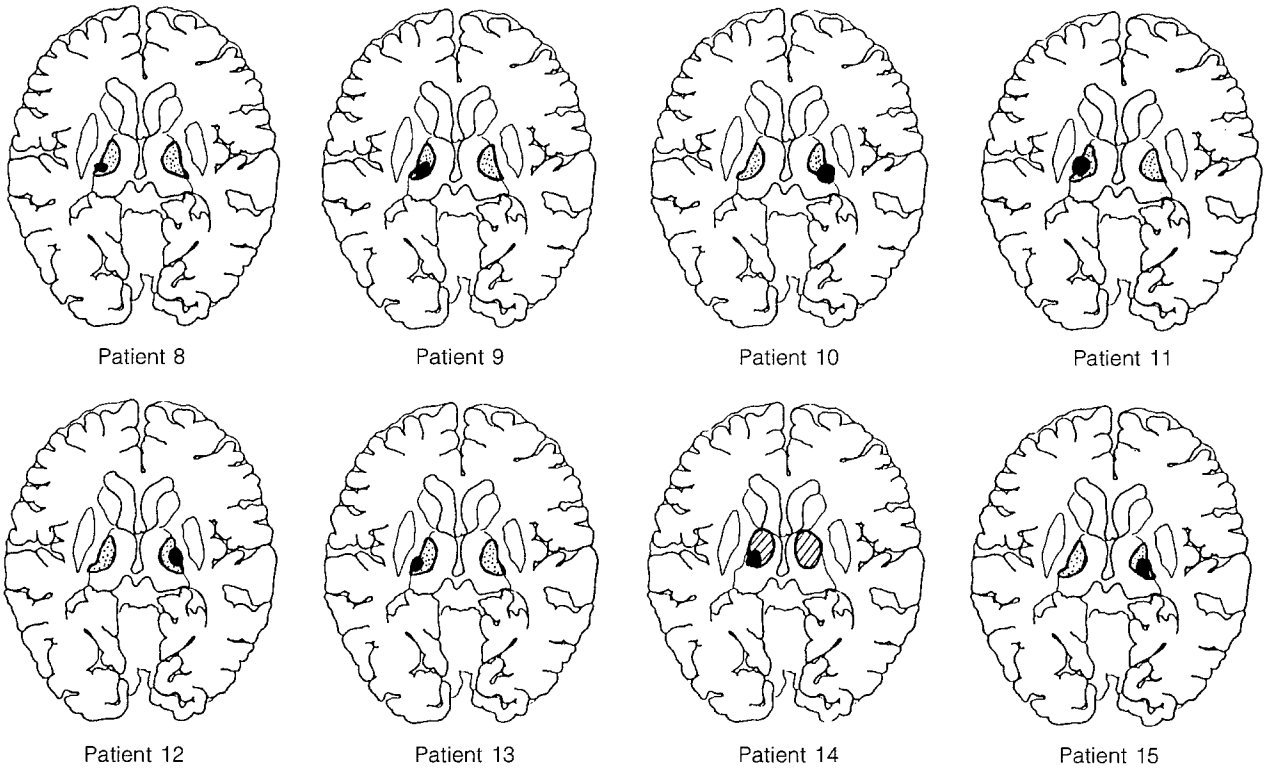


**Fig. 2.** Hypaesthetic ataxic hemiparesis. Topography of infarcts on CT or MRI. ▨ Thalamogeniculate arteries territory; ▨ tuberothalamic arteries territory infarct

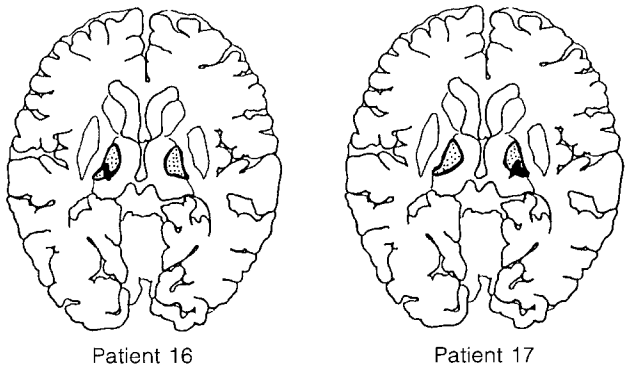


**Fig. 3.** Ataxic hemiparesis. Topography of infarcts on CT. ▨ Thalamogeniculate arteries territory; ▨ tuberothalamic arteries territory infarct

1, 4), while in 4 other patients (nos. 2, 3, 5, 7) position and vibration sensation was also involved. One patient (no. 6) had a purely subjective sensory disturbance (lower limb paraesthesias). With the exception of patient 2, who had a transient asterixis involving the hand contralateral to the infarct, the remaining neurological examination was normal in all patients, including tendon reflexes, muscular strength and plantar response (Table 2).

All patients had CT performed once or twice during the 1st week which showed the infarct in all but 1 patient. In that patient (no. 5) the lesion was visible on MRI performed 36 days later. On lesion mapping on CT or MRI, all infarcts involved the ventral posterior nucleus and the ventral lateral nucleus (caudal part) of the thalamus (thalamogeniculate territory [6]) (Fig. 1). The internal capsule was affected in 2 patients (nos. 1 and 6). The maximum diameter of infarct varied between 7.5 and 12.5 mm (mean 9.9) (Table 3).

*Hypaesthetic ataxic hemiparesis*

Stroke was immediately complete in 5 patients and progressed over 4–12 h in 3 patients. Sensory disturbances were the first symptom in these 3 patients. One patient (no. 14) had a TIA 24 h before the stroke. No patient complained of headache. Five patients had sensory loss involving light touch, pain, temperature, position and vibration sense. These sensory disturbances involved the entire hemibody in all patients but one (no. 11) in whom the lower limb sensory disturbance was only subjective. Three patients (nos. 13–15) had a purely subjective sensory disturbance, involving the entire hemibody in 1 patient (no. 13), and limited to the face and arm in 1 patient (no. 15) and to the leg in 1 patient (no. 14).

All patients had a slight hemiparesis ipsilateral to the ataxic and sensory disturbance. The paresis spared the face in 3 patients (nos. 11, 12, 15) and the leg in 1 patient (no. 10). On Mingazzini and Barré tests the paretic limb fell slowly. When the patients tried to maintain the arms against gravity, a pronator drift and spooning of the hand developed. All patients had brisk tendon reflexes. Plantar response was extensor on the hemiparetic side. The remaining neurological examination was normal in all patients (Table 2).

**Table 4.** Reported cases with ataxia and thalamic stroke. VPL, Ventral posterior lateral nucleus; IL, intralaminar nuclei; DM, dorsomedial nucleus; CM, centromedian nucleus; AU, autopsy; DP, diagnostic procedure. See Table 3 for other abbreviations

| Reference | No. of cases | DP | Ataxia | Hypaesthesia | Paresis | Thalamic location | PLIC |
|---|---|---|---|---|---|---|---|
| Azouvi et al. [3] | 1 | CT/MRI | + | + | − | VL/VP (haemorrhage) | No |
| Bogousslavsky et al. [7] | 6 | CT | + | + | − | VL/VP | ? |
| Caplan et al. [10] | 1 | CT | + | + | − | VL | No |
| Garcin and Lapresle [20] | 1 | AU | + | − | + | VPL/DM/CM/IL | No |
| Bogousslavsky et al. [7] | 2 | CT | + | − | + | DM/IL/VP | ? |
| Trouillas et al. [39] | 1 | CT/MRI | + | − | + | VPL/CM | Yes |
| Boiten and Lodder [8] | 1 | CT/MRI | + | − | + | VL | No |
| Dejérine and Roussy [12] | 3 | AU | + | + | + | VPL/DM | Yes |
| Garcin [19] | 1 | AU | + | + | + | VPL/VL | No |
| Bogousslavsky et al. [4] | 1 | CT | + | Pain | + | VL | Yes |
| Verma and Maheshwari [40] | 2 | CT | + | + | + | Lateral thalamus | No |
| Hommel et al. [29] | 1 | CT/MRI | + | + | + | VPL/CM | No |
| Caplan et al. [10] | 1 | CT | + | + | + | VL | No |
| Bogousslavsky et al. [6] | 2 | CT | + | + | + | VP/VL | ? |
| Lee et al. [31] | 1 | CT/MRI | + | + | + | Lateral thalamus | Yes |
| Helgason and Wilbur [28] | 7 | CT/MRI | + | + | + | Lateral thalamus | Yes |

CT, performed between days 2 and 30, showed the infarct in all patients. Patient no. 12 had only MRI, which was performed at day 2, also showing the infarct. Within the thalamus, the infarct involved the ventral posterior nucleus and the caudal part of the ventral lateral nucleus (thalamogeniculate territory [6]) in 6 patients. In 1 patient only the ventral posterior nucleus seemed to be involved, and in another patient, more rostral areas including the ventral anterior nucleus (tuberothalamic territory [6]) were affected (Fig. 2). The adjacent part of the internal capsule was involved in 5 patients. The mean maximum diameter of the infarcts was 8.4 mm (5–12.5 mm) (Table 3).

*Ataxic hemiparesis*

This picture was found in only 2 patients. The onset was sudden. Both patients had had TIAs (2 and 5 episodes) during the preceding week. One patient had headache. A detailed examination showed no sensory disturbance. The paresis involved the entire hemibody. The tendon reflexes were brisk and the plantar response was in extension on the hemiparetic side (Table 2).

CT performed on day 12 (no. 16) and day 2 (no. 17) showed involvement of the most caudal part of the ventral lateral nucleus and ventral posterior nucleus impinging on the most medial part of the internal capsule (Fig. 3). The maximum diameter of the infarct was 8 mm (no. 16) and 9 mm (no. 17) (Table 3).

*Evolution*

In 4 patients (nos. 3, 13, 15, 16), the recovery was complete at 0.5, 1 and 6 months, the ataxia being the last disturbance to disappear. In patients 4 and 12 moderate ataxia with mild sensory loss persisted at the time of follow-up. In the other patients, the hemiataxia persisted (at 0.5, 1, 2 and 12 months) despite complete sensory and motor recovery.

## Discussion

Our findings show that hemiataxia may be present in 25% of patients with thalamic infarction. Table 4 summarizes previously reported cases with ataxia from thalamic stroke.

In our patients, hemiataxia was the main neurological disturbance. While it was always associated with either hemiparesis or hemihypaesthesia, it also had the characteristics of a cerebellar type of ataxia. That associated hemiparesis or hemihypaesthesia may not explain most of the ataxia is also suggested by our finding that in the majority of patients, the ataxia persisted despite recovery of sensory and motor disturbance. We delineated three clinical groups, the first of which was highly suggestive of the lesion being in the thalamus.

1. *Hemiataxia-hypaesthesia.* This association has not been studied in detail previously. We found only 8 patients in the literature who had a similar picture [3, 6, 10]. Six of them belonged to a group of 18 patients with thalamogeniculate territory infarction studied previously in the Lausanne Stroke Registry [6]. HH was not found in the 1075 patients without thalamic infarction from the Lausanne Stroke Registry who were admitted during the same period. To our knowledge, this clinical picture has been reported only once without thalamic involvement, in relation to anterior choroidal artery (AChA) infarction involving the posterior limb of the internal capsule immediately adjacent to the thalamus, which probably disrupted part of the thalamic radiations. [5].

2. *Hypaesthetic ataxic hemiparesis.* HAH was the most frequent picture in our patients with hemiataxia from

thalamic infarction. The term HAH was first coined in 2 patients with thalamic haemorrhage [40]. Helgason and Wilbur [28] reported 23 patients with HAH and concluded that it was most commonly the result of infarction in the posterior limb of the internal capsule (PLIC). Only 7 of these patients had lateral thalamic infarction adjacent to the PLIC. These authors suggested that the lateral thalamus was probably more frequently affected, but that the insensitivity of CT for diagnosing small thalamic infarcts may explain this rather low proportion. They concluded that AChA territory infarction was the main contributor to this syndrome, but that thalamogeniculate territory infarction may not be uncommon. It may be mentioned that in the "thalamic stroke syndrome" reported by Dejérine and Roussy in 1906, the triad of HAH was found in all patients, although additional features were also present [12].

3. *Ataxic hemiparesis.* Only two of our patients had AH. Clearly, AH from thalamic infarction is rare. AH is most commonly the result of infarction in the pons, corona radiata or internal capsule [15, 18]. In some instances, stroke location in the thalamus may have been erroneous, because the infarct was not localized to the thalamus on the CT published [35, 38].

The lateral part of the thalamus was involved in all patients at the level of the caudal part of the ventral lateral nucleus and the ventral posterior nucleus (thalamogeniculate territory [6]). More rostral areas (anterior part of the ventral lateral nucleus, tuberothalamic territory [6]) seemed affected in only 1 patient. The internal capsule was more frequently involved when hemiparesis was present.

Involvement of the ventral lateral nucleus, which was present in all but 1 patient, may be responsible for hemiataxia by interrupting dentato-rubro-thalamic fibres. Previous findings of a crossed cerebellar diaschisis in patients with thalamic stroke may support this hypothesis, although clinical and cerebral blood flow correlation remains controversial [37]. To some extent, ataxia may have also been due to associated hemiparesis or proprioceptive loss [12, 13, 30]. However, in our patients ataxia persisted despite recovery of sensory and motor disturbances. Also, the incoordination, which was not affected by visual clues, was not suggestive of paretic or proprioceptive ataxia. While sensory loss our patients may be explained by involvement of the ventral posterior nucleus on the appropriate side [16], hemiparesis and other pyramidal signs may be related to direct or indirect (oedema, compression) involvement of the adjacent internal capsule [20, 21].

Cardiac or artery-to-artery embolism was the likely stroke aetiology in more than one-third of the patients. Although hypertension or diabetic arteriolopathy has been considered the most common cause of thalamic infarcts [9, 16, 22–25, 27, 33], large-artery disease or cardioembolism may not uncommonly be responsible. Indeed, 17% of the patients with distal basilar artery embolism reported by Fisher [16] had an isolated thalamic infarction. Goto et al. [26] found that 2 of 20 patients with posterior cerebral artery occlusion had an infarct limited to the thalamus and Caplan et al. [10] reported a case of thalamic infarction related to posterior cerebral artery occlusion.

Why small-vessel disease may be a less common contributor here than in lenticulostriate infarcts may be related in part to the fact that unlike the lenticulostriate penetrators, the deep perforators to the thalamus may show important reciprocal collateralization [32]; thus, single penetrator occlusion from small-artery disease may not always be responsible for a corresponding infarct (lacunar infarct), while multiple embolic occlusions impairing collateral supply may more often be responsible.

Our findings highlight the common occurrence of ataxia in thalamic infarction, usually from involvement of the thalamogeniculate territory.

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