Brain (2001), **124,** 299–309

Delayed onset mixed involuntary movements after thalamic stroke

Clinical, radiological and pathophysiological findings

Jong S. Kim

Department of Neurology, University of Ulsan, Asan Medical Center, Seoul, South Korea

Correspondence to: Jong S. Kim, MD, Department of Neurology, Asan Medical Center, Song-pa, PO Box 145, Seoul 138-600, South Korea
E-mail: jongskim@www.amc.seoul.kr

Summary

Although occurrence of involuntary movements after thalamic stroke has occasionally been reported, studies using a sufficiently large number of patients and a control population are not available. Between 1995 and 1999, the author prospectively identified 35 patients with post-thalamic stroke delayed-onset involuntary movements, which included all or some degree of dystonia–athetosis–chorea–action tremor, occasionally associated with jerky, myoclonic components. A control group included 58 patients examined by the author during the same period who had lateral thalamic stroke but no involuntary movements. Demography, clinical features and imaging study results were compared. There were no differences in gender, age, risk factors, side of the lesion and follow-up periods. During the acute stage of stroke, the patients who had involuntary movements significantly more often had severe (≤III/V) hemiparesis (50 versus 20%, $P < 0.05$) and severe sensory loss (in all modalities, $P < 0.01$) than the control group. At the time of assessment of involuntary movements, the patients with involuntary movements significantly more often had severe sensory deficit (in all modalities, $P < 0.01$) and severe limb ataxia (60 versus 5%, $P < 0.01$) than the control patients, but neither more severe motor dysfunction (7 versus 0%) nor more painful sensory symptoms (57 versus 57%). The patients with involuntary movements had a higher frequency of haemorrhagic (versus ischaemic) stroke (63 versus 31%, $P < 0.05$). Further analysis showed that dystonia–athetosis–chorea was closely associated with position sensory loss, whereas the tremor/myoclonic movements were related to cerebellar ataxia. Recovery of severe limb weakness seemed to augment the instability of the involuntary movements. Persistent failure of the proprioceptive sensory and cerebellar inputs in addition to successful, but unbalanced, recovery of the motor dysfunction seemed to result in a pathological motor integrative system and consequent involuntary movements in patients with relatively severe lateral-posterior thalamic strokes simultaneously damaging the lemniscal sensory pathway, the cerebellar-rubrothalamic tract and, relatively less severely, the pyramidal tract.

Keywords: cerebrovascular disorder; chorea; dystonia; thalamus; tremor

Introduction

In 1906, Dejerine and Roussy described three patients with thalamic stroke who developed delayed-onset choreoathetosis and hemiataxia. The involuntary movements worsened on eye closure and were closely related to concurrent hemisensory loss. Foix and Hillemand (1925) attempted to explain the phenomenon by an involvement of the thalamic relay nuclei. They emphasized frequent coexistence of choreoathetosis and incoordination, and attributed the choreoathetotic movements to involvement of the cerebellar–thalamic rather than the striatothalamic pathway. Garcin (1955) later described dystonic hands caused by thalamic strokes and attributed the symptom to associated hypertonicity due to concurrent involvement of the pyramidal–extrapyramidal tract.

More recently, with the advent of imaging techniques, post-thalamic stroke involuntary movements have increasingly been described that include chorea (Sharp, 1994), dystonia (Sunohara *et al.*, 1984; Karsidag *et al.*, 1998), tremor (Kim, 1992; Ferbert and Gerwig, 1993), myoclonus (Gatto *et al.*, 1998) and complex movements encompassing several components of all of these (Ghika *et al.*, 1994). Nevertheless, understanding of these post-thalamic stroke involuntary movements is still incomplete, due mainly to the paucity of well described literature. Previous studies were

© *Oxford University Press 2001*

usually done retrospectively, with a small number of patients and without clear description of the nature and location of stroke or concomitant neurological deficits. Moreover, although these involuntary movements frequently manifest as complex symptoms, the authors often emphasized a single component of the involuntary movements and described it as tremor (Kim, 1992; Ferbert and Gerwig, 1993), dystonia (Sunohara *et al.*, 1984; Karsidag *et al.*, 1998), delayed-onset cerebellar syndrome (Louis *et al.*, 1996), pseudoathetosis (Sharp *et al.*, 1994) or pseudochoreoathetosis (Kim *et al.*, 1999). Although the complexity of involuntary movements was correctly identified by several authors, the symptoms were often described using unclear terms such as 'jerky dystonic unsteady hand' (Ghika *et al.*, 1994). Finally, to the author's knowledge, there have been no studies of post-thalamic stroke movement disorders with the use of a control population, and which neurological deficits are related to the pathogenesis of involuntary movements remains unknown. Therefore, 35 consecutive patients who developed delayed-onset involuntary movements after thalamic stroke were studied. Because all of the patients had lateral thalamic lesions, the control group comprised patients with lateral thalamic stroke who did not develop involuntary movements.

Subjects and methods

Between April 1995 and December 1999, the author prospectively identified 35 consecutive patients attending the outpatient clinic of the Asan Medical Center who developed delayed-onset post-thalamic stroke involuntary movements. In the present study, involuntary movements that occur at the onset of stroke, such as hemichorea–ballism (Martin, 1927; Kase *et al.*, 1981), asterixis (Massey *et al.*, 1979) or acute hemidystonia (Marsden *et al.*, 1985) were not considered. The patients who were followed-up for <3 months were excluded. The involuntary movements were defined according to the modification of Fahn (Fahn, 1997): dystonia as sustained, inappropriate, twisted finger (or rarely wrist) posture (flexion, extension, rotation) at rest or during action; choreoathetosis as rapid (chorea) or slow (athetosis) involuntary movements of the fingers or toes (flexion–extension, adduction–abduction, writhing, sometimes piano-playing movements) which are irregular, non-rhythmic and purposeless; tremor as rhythmic, oscillatory involuntary movements; action tremor as oscillatory movements present on action, especially during the finger-to-nose manoeuvre; and myoclonic jerks as sudden, brief, shock-like involuntary hand movements.

All the patients were examined and followed by the author. Selected patients ($n = 10$) were videotaped and assessed repeatedly. Of the 35 patients, 18 had been examined by the author from the acute stage (<1 week, mostly <2 days after the onset) and followed-up while others ($n = 17$) were transferred to the author at the subacute or chronic stage, and detailed initial neurological findings were therefore unknown. Thorough neurological examination was performed. Muscle strength was graded as I–V using the MRC (Medical Research Council) scale. Clumsiness without definitive weakness was designated as V–. Pinprick, temperature and vibration senses were examined and graded as mild, moderate and severe when the sensory perception was >70, 30–70 and $<30\%$, respectively, of the normal side. Position sense was graded as 'mild' when a patient correctly identified the up and down movements but with a feeling of 'unclear' sensation; 'moderate' when there was an occasional error; and 'severe' when most of the movements could not be identified (Kim and Choi-Kwon, 1999). The presence of painful sensory symptoms was defined when the patients required pain-relieving medications such as amitriptyline. Ataxia, if present, was graded as mild (slight dysmetria observed when target was closely approached), moderate (the grade between mild and severe) or severe (gross oscillation of the arm observed from the start of the manoeuvre with severe overshooting of the desired point) on the finger-to-nose manoeuvre. In this study, the motor, sensory and cerebellar symptoms were generally less severe in the legs, and grading of the severity was based on the deficits detected in the upper limbs.

Stroke subtypes were divided into brain infarction and intracerebral haemorrhage according to the imaging findings. Subtypes of ischaemic stroke were further divided as follows.
Small vessel infarction: (i) presence of hypertension and/or diabetes mellitus; (ii) CT and/or MRI performed 72 h after the onset of stroke showing a single thalamic infarction $\leqslant 1.5$ cm in diameter; (iii) arterial imaging (conventional angiogram or MR angiogram) showing no evidence of significant ($>50\%$) large vessel disease; and (iv) history and EKG do not show evidence of emboligenic heart disease (atrial fibrillation, valvular disease, sick sinus syndrome, cardiomyopathy, acute myocardiac infarction).
Probable small vessel infarction: if arterial imaging was not performed.
Large vessel infarction: (i) CT and/or MRI performed 72 h after the onset of stroke showing thalamic infarction >1.5 cm in diameter or two concomitant infarcts in the territory of posterior cerebral artery (thalamus + occipital lobe); (ii) no evidence of emboligenic cardiac disease; and (iii) arterial imaging showing evidence of occlusive or stenotic ($>50\%$) disease of the posterior cerebral artery that explains the patient's infarction.
Probable large vessel infarction: if arterial imaging was not performed or showed large vessel disease unrelated to the present stroke.
Cardiogenic embolic infarction: (i) infarction that does not fit with small or large vessel infarction; (ii) concurrent presence of emboligenc cardiac disease; and (iii) arterial imaging showing no evidence of significant arterial disease.
Probable cardiogenic embolic infarction: if arterial imaging was not performed.
Unknown: infarcts that do not fit any of the above criteria, e.g. imaging results showing infarcts affecting the thalamus

and the occipital lobe while angiogram and ECG findings were normal.

Because all of the patients had lateral-posterior thalamic lesions, the author selected patients for the control (i) who were examined at the author's out-patients clinic during the same period as mentioned above, (ii) who were followed-up at least 3 months after the onset, (iii) who had lateral thalamic stroke confirmed by imaging (CT or MRI) results and (iv) who did not develop delayed-onset involuntary movements. Patients with lesions selectively involving the ventral anterior or dorsomedian thalamic nucleus (Bogousslavsky *et al.*, 1998) were excluded. The demographic, clinical features and pathogenic mechanisms were compared between the patients with involuntary movements and control subjects. For statistical analyses, we used Student's *t*-test (for numerical variables) and χ^2 test (for nominal scale variables). Fisher's exact test was used when the number of the subjects was small and ANOVA (analysis of variance) was used when there were more than two variables. All statistical tests were two-tailed and performed with the use of PC-SAS package (version 6.10); P values <0.05 were regarded as indicating significance.

Results

Table 1 summarizes the characteristics of the patients.

Demography and risk factors of the patients

The study comprised 22 men and 13 women with ages ranging from 28 to 74 years (mean 57.5 years). The risk factors for stroke were hypertension in 30 patients, diabetes mellitus in five patients and current cigarette smoking in six patients. One had a history of coronary heart disease. One patient (Patient 10) had mitral valve disease and atrial fibrillation, and one patient (Patient 3) had head trauma just before the occurrence of stroke. Follow-up periods ranged from 4 to 240 (mean 40.4) months.

Neurological deficits

Motor dysfunction

Among the patients who were examined by the author at onset ($n = 18$), initial hemiparesis was seen in all the patients; severe (≥III) in nine and mild in nine patients. For those who were referred to the author ($n = 17$), history taking illustrated that the majority of the patients had significant hemiparesis. At the time of assessment of involuntary movements, the motor function had improved and all the patients ($n = 35$) had mild (≥IV) motor dysfunction. Spasticity was noted in 15 patients.

Sensory dysfunction

Among the patients who were initially examined by the author ($n = 18$), all the patients had sensory deficits. The degree of deficit was not accurately tested in some of the patients who were drowsy and uncooperative. In others, the degree of the sensory deficit was usually severe. Position sense, in particular, was impaired to a moderate to severe degree in all the patients except for one (Patient 6).

At the time when involuntary movements were examined, the majority ($n = 29$) of the patients still had significant position sensory deficits that were moderate in four patients and severe in 25 patients. On the other hand, sensations of pinprick, temperature and vibration had improved significantly and were decreased to a moderate to severe degree in 13, eight and six patients, respectively. Hypersensitivity to pinprick ($n = 7$) or temperature ($n = 11$) and vibration ($n = 3$) were noted in some patients. All the patients except for two (Patients 15 and 30) had subjective sensory symptoms (paraesthesiae). The symptoms were described as numb in 26 patients, cold in seven, aching in four, burning in three, squeezing in three and heavy in three, in various combinations. Twenty patients required pain-relieving medications (amitriptyline and/or carbamazepine) and are designated as having painful sensory symptoms. The onset of painful paraesthesiae varied from immediately after the onset to 6 months after the onset (usually at 1–3 months).

Limb ataxia

In the acute phase, most of the patients had significant hemiparesis and two patients with mild hemiparesis showed limb ataxia. At the time of analysis of involuntary movements, as muscle strength improved, dysmetria on the finger-to-nose manoeuvre became apparent in as many as 32 patients (mild in 11, moderate in seven and severe in 14). However, dysmetria of the leg on the heel-to-shin test was much less severe. Only two patients were wheelchair bound and five needed assistance in walking.

Others

Other signs included visual field defects in 10 patients. At the acute stage, drowsiness, aphasia and memory impairment were found in five, two and three patients, respectively, all of which improved at the time of assessment of involuntary movements.

Nature and location of the lesion

Twenty-two patients had intracerebral haemorrhages (three had concomitant intraventricular haemorrhage) and 13 had infarcts. The location of the lesion is illustrated in Fig. 1. The lesions were located in the posterolateral thalamus in all the patients, which involved the posterior limb of the internal capsule to a variable degree. The occipital lobe was concomitantly involved in eight patients. Among 13 patients with infarction, the lesions were located mainly in the thalamogeniculate artery territory in nine patients and

Table 1 *Demography and clinical features of all patients*

Pt no.	Sex	Age (years)	Side	Nature of the lesion	Initial deficits					Deficits at the time of study					Reflex	Par-aesthesia	Ataxia	Onset of IM*	Nature of IM	F-up
					Motor sensory					Motor sensory										
					MSG	PP	T	V	PO	MSG	PP	T	V	PO						
1	M	57	R	ICH	IV	M	M	M	S	V–	M	M	N	S	N	M	MO	12 mo.	DAC	60
2	M	61	R	ICH	I	MO	MO	M	S	IV	MO	N	N	S	inc	M	MO	4 m.	DACaT (jerky)	46
3	M	29	L	Inf (UK)	II	S	S	S	S	V–	S	S	M	S	inc	S	None	1–2 mo.	DAC	5
4	M	49	R	ICH	I	S	S	S	S	V–	MO	MO	MO	S	inc	M	M	3–4 mo.	DACaT	6
5	M	70	L	Inf (pLVI)	II	UC	UC	UC	UC	V–	M	M	M	M	N	S	M	UK	DACaT	25
6	M	74	R	ICH	III	M	M	M	M	IV	M	N	N	M	N	S	MO	12 mo.	DACAT (jerky)	25
7	M	58	L	ICH	III	S	S	S	S	IV	S	S	S	S	inc	S	M	UK	AC-aT	6
8	M	76	L	Inf (LVI)	IV	S	S	S	S	V–	S	S	S	S	N	M	M	1 mo.	DACaT	17
9	M	63	L	ICH	II	S	S	S	S	V–	S	S	S	S	inc	M	M	UK	DAC	19
10	F	28	L	Inf (CE)	IV	MO	MO	MO	S	V–	M	M	M	MO	N	M	M	imm.–2 mo.	Mild DAC	15
11	F	48	L	Inf (LVI)	IV	M	M	MO	MO	V–	inc	inc	M	M	N	S	None	UK	Mild DAC	9
12	M	64	R	ICH	III	S	S	S	S	V–	S	N	N	S	N	M	M	2 weeks	Mild DAC	30
13	F	63	R	Inf (pLVI)	IV	UC	UC	UC	UC	V–	inc	N	inc	N	N	M	M	UK	D-T	4
14	M	73	R	Inf (pSVI)	V–	MO	MO	MO	MO	V–	N	N	N	M	N	M	S	UK	AC-aT	7
15	M	47	L	ICH	I	S	S	S	S	IV	MO	MO	N	S	inc	None	S	UK	DACaT (with GO)	19
16	F	71	R	Inf (LVI)	IV	S	S	S	S	IV	M	inc	M	S	N	S	S	UK	DACaT (with GO)	13
17	M	53	R	Inf (LVI)	IV	S	S	S	S	V–	N	inc	N	MO	N	M	M	2 mo.	Mild DACaT	5
18	M	53	L	Inf (LVI)	IV	S	S	S	S	V–	M	inc	N	S	inc	S	S	imm.–2 mo.	DACaT	17
19	F	62	R	ICH	UK	UK	UK	UK	UK	IV	M	N	inc	S	N	S	MO	12 mo.	DACaT (jerky)	60
20	M	58	L	Inf (pLVI)	UK	UK	UK	UK	UK	V–	MO	N	MO	S	N	S	M	UK	DACaT	84
21	F	49	R	ICH	UK	UK	UK	UK	UK	V–	M	inc	N	S	inc	S	S	UK	C-aT	22
22	M	55	R	ICH	UK	UK	UK	UK	UK	IV/IV	inc	inc	N	S	N	S	S	UK	DACaT	12
23	F	51	R	ICH	UK	UK	UK	UK	UK	IV	inc	inc	N	S	N	S	MO	24 mo.	DA	70
24	F	57	R	ICH	UK	UK	UK	UK	UK	IV/IV	MO	N	N	MO	inc	S	M	UK	DA (dystonia dominant)	48
25	F	58	L	ICH	UK	UK	UK	UK	UK	V–	S	S	M	S	inc	S	S	UK	DACaT(with GO)	24
26	M	62	R	ICH	UK	UK	UK	UK	UK	V–	inc	inc	inc	S	inc	S	S	7 mo.	DA-aT (with GO)	7
27	M	63	R	ICH	UK	UK	UK	UK	UK	V–	MO	MO	N	S	inc	S	S	UK	DACaT (jerky)	52
28	F	59	R	ICH	UK	UK	UK	UK	UK	V–	inc	inc	N	S	N	S	MO	UK	DACaT(with GO)	23
29	M	66	L	ICH	UK	UK	UK	UK	UK	V–	M	N	N	S	N	M	S	UK	DACaT (jerky, with GO)	144
30	M	58	L	ICH	UK	UK	UK	UK	UK	V–	S	S	S	S	inc	None	S	UK	DACaT	46
31	F	65	R	Inf (LVI)	UK	UK	UK	UK	UK	IV	M	inc	N	MO	N	S	MO	UK	DACaT (jerky)	12
32	F	60	R	ICH	UK	UK	UK	UK	UK	IV/IV	inc	inc	M	S	inc	S	S	12 mo.	DACaT	60
33	F	51	L	ICH	UK	UK	UK	UK	UK	V–	M	M	M	M	N	M	None	UK	Mild C	39
34	M	66	L	ICH	UK	UK	UK	UK	UK	V–	M	M	M	S	N	M	S	UK	DACaT (jerky)	240
35	M	37	L	Inf (pLVI)	UK	UK	UK	UK	UK	IV	MO	M	MO	S	inc	S	S	2 mo.	DACaT (jerky)	144

Pt = patient; F = female; M = male; R = right; L = left; ICH = intracerebral haemorrhage; Inf = infarction; UK = unknown; p = probable; LVI = large vessel infarction; CE = cardiogenic embolism; SVI = small vessel infarction; PP = pinprick; T = temperature; V = vibration; PO = position; M = mild; MO = moderate; MSG = muscle strength grade; S = severe; N = normal; UC = uncheckable; inc = increased; IM = involuntary movment; mo. = month; imm. = immediate; D = dystonia; A = athetosis; C = chorea; aT = action tremor; GO = gross oscillation of arm; F-up = follow up (months from the onset). *From the onset of stroke.

posterior choroidal artery territory in four (Patients 3, 18, 31 and 35) (Bogousslavsky *et al.*, 1988; Tatu *et al.*, 1998) (Fig. 2).

Vascular study results and pathogenesis of the stroke

All the patients with haemorrhagic stroke had hypertension, and hypertensive intracerebral haemorrhage was considered as an aetiopathogenesis. In 13 patients with infarction, seven had angiograms (conventional angiogram in one and MR angiogram in six). Five patients showed posterior cerebral artery disease (stenosis in two and occlusion in three), explaining the patients' infarction. One had significant vertebral artery stenosis and slightly narrowed posterior cerebral artery, suggesting artery to artery embolism as a pathogenesis. Judging from imaging findings and vascular study results of 13 patients with infarction, we considered 10 patients as having large vessel disease (including four with probable large vessel disease), one (Patient 14) with small vessel infarction, one (Patient 10) with cardiogenic embolic infarction and one (Patient 3) with an unknown cause.

Description of involuntary movements

The onset time of involuntary movements was not certain in some patients, partly because they did not clearly remember it and partly because the involuntary movements developed so gradually. Among 13 patients in whom the onset of involuntary movements was relatively clear, the movements started 2 weeks to 24 months (mean 6.9 months) after the onset of stroke. The involuntary movements usually developed later than the occurrence of paraesthesiae. In two patients (Patients 10 and 18), dystonia–athetosis started immediately after the onset, but the full spectrum of involuntary movements appeared at ~2 months post-stroke. The involuntary movements always developed in the fingers/hand (dystonia, choreoathetosis, tremor), and the elbow/shoulder (tremor) was involved in severe cases. Dystonia–

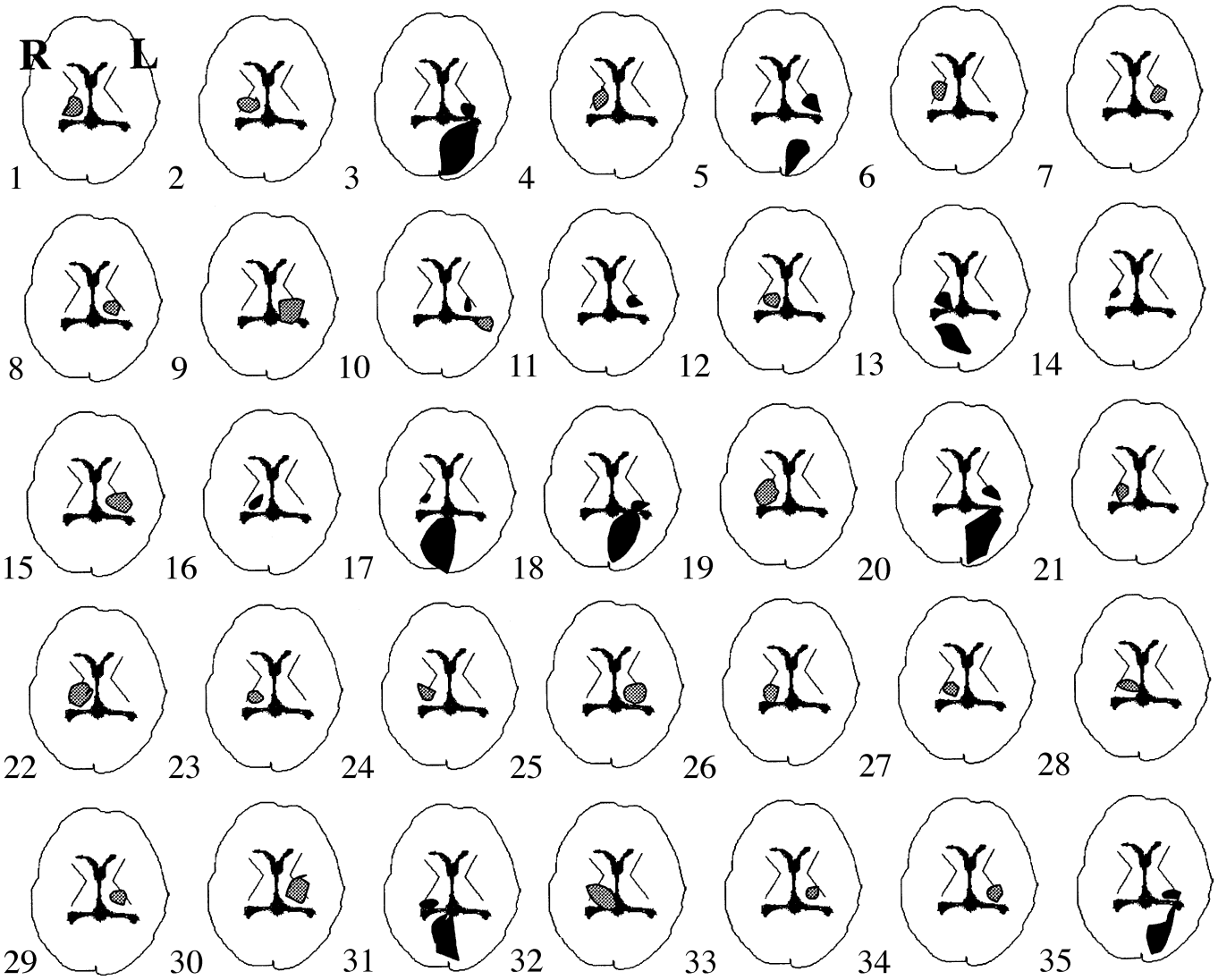

Fig. 1 Location of the lesions. Number indicates patient's number. Dark areas indicate infarction and dotted areas indicate haemorrhage.

athetosis–chorea was also noted in toes in five patients (Patients 8, 16, 29, 30 and 32) and jaw tremor was observed in one patient (Patient 28). A trunk or proximal leg was never involved. Generally, the involuntary movements were aggravated when the patients were anxious or fatigued, and disappeared while they were sleeping.

The involuntary movements were divided into two groups.

(1) *Dystonia–athetosis–chorea.* Nine patients (Patients 1, 3, 9–12, 23, 24 and 33) showed dystonia–athetosis–chorea but not tremor. Dystonia usually manifested as flexion of the metacarpophalangeal joints, extension of the interphalangeal joints and adduction–abduction of individual fingers resulting in misalignment of the fingers. The dystonic fingers moved slowly (athetosis), often associated with rapid, irregular abduction–adduction or piano-playing finger movements (chorea). The amplitude and rhythmicity were variable even in the same patients. Some patients had dystonia–athetosis (Patients 23 and 24) or choreic finger movements only (Patient 33). Patient 12 had more severe sensory deficit in the ulnar-sided fingers than in the radial-sided fingers and dystonia-choreic movements were also more severe in the former. The dystonia–athetosis–chorea tended to be aggravated on eye closure to a variable degree.

(2) *Dystonia–athetosis–chorea–action tremor.* Patient 26 showed dystonia–athetosis plus action tremor and Patient 13 showed dystonia plus action tremor. Neither had chorea. In the majority of patients, dystonia–athetosis–chorea movements were intermixed with action tremor of the fingers. In severe patients (Patients 15, 16, 25, 26, 28 and 29), gross oscillation of the proximal arms was noted. The tremor consisted of rhythmic 2–4 Hz flexion–extension movements of the fingers. Unlike the Parkinsonian tremor, the amplitude and rhythm of the tremor fluctuated, and when the intensity of the tremor was not severe, it was often blended with choreic movements. The tremor was always aggravated on stretching the involved arm or during inten-

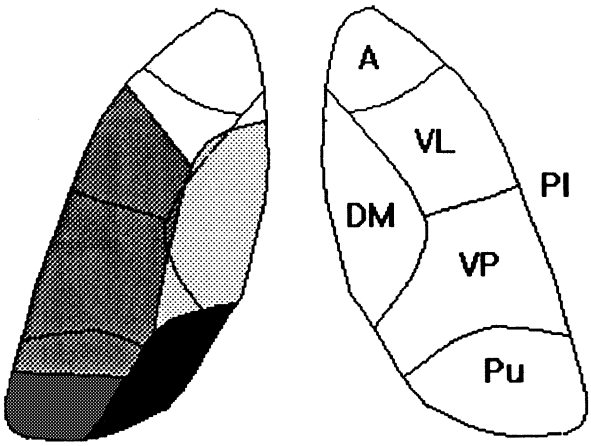

Fig. 2 Vascular territory of the thalamus at midthalamic level (modified from Pullicino, 1993 and Tatu *et al*., 1998). Black area = posterolateral choroidal artery; dark grey area = posteromedial choroidal artery; medium grey area = thalamogeniculate artery; light grey area = paramedian thalamic artery; white area = thalamotuberal artery. A = anterior thalamic nucleus; DM = dorsomedian thalamic nucleus; VL = ventral lateral thalamic nucleus; VP = ventral posterior thalamic nucleus; Pu = pulvinar; PI = posterior limb of internal capsule.

tional movements such as performing the finger-to-nose manoeuvre. During these manoeuvres, tremors were blended with jerky, myoclonic hand movements in patients with severe symptoms (Patients 2, 6, 19, 27, 29, 31, 34 and 35).

Course of the involuntary movements

The involuntary movements usually started gradually, and progressively worsened for weeks or months and then stabilized. During the follow-up period of an average 40.4 months, the involuntary movements remained persistent in 31 patients and worsened progressively in three patients (Patients 6, 8 and 20). Only one patient (Patient 10), who developed mild dystonia–athetosis–chorea in the hand, showed gradual improvement of her involuntary movements, which was associated with the improvements of the position sense.

Differences between the patients with involuntary movements and those without (controls)

Control subjects included 58 patients who met the criteria described above. Among them 37 patients were examined during the acute stage of stroke, while others were seen in the subacute or chronic stage. The proportion of the patients examined from the onset was not different between the two groups. Table 2 summarizes the differences between the patients with involuntary movements and control subjects. There were no differences in age, gender and risk factor profiles. The patients with involuntary movements had haemorrhagic stroke (versus infarction) significantly more often ($P < 0.01$) than the controls. Among the patients with infarction, large vessel infarction (including probable large vessel infarction) was significantly ($P < 0.01$) more common in patients with involuntary movements, while small vessel infarction (including probable small vessel infarction) was more common ($P < 0.01$) in the control subjects. Among the patients who were examined from the acute stage, the presentations of pure sensory stroke including restricted acral sensory involvement (Kim, 1994) and hypaesthetic ataxic hemiparesis were significantly greater in the control population ($P < 0.01$ for each).

For comparison of the neurological deficits, we converted the following data dichotomously: the motor dysfunction as mild (no deficit + IV/V) and severe (⩾III/V); sensory as mild (none + mild) and severe (moderate + severe); ataxia as mild (none + mild) and severe (moderate + severe). In the patients who were examined in the acute phase (patient group, $n = 18$; control, $n = 37$), the patients with involuntary movements had initially severe motor dysfunction ($P < 0.05$) and severe sensory deficit (in all modalities, $P < 0.01$) significantly more often than the control group of patients. At the day of assessment of involuntary movements, there was no difference in the severity of motor dysfunction, the presence of paraesthesiae and painful sensory symptoms between the two groups. However, patients with involuntary movements significantly more often had severe sensory deficit ($P < 0.01$ in all modalities) and severe ataxia on the finger-to-nose test ($P < 0.01$) than the controls.

Discussion

Terminology of the involuntary movements and literature review

The involuntary movements described here had three components: dystonia, choreoathetosis and tremor. Although the patients usually had complex movements encompassing several components, the symptoms may be divided into two subgroups. The first is the dystonia–athetosis–chorea group. Nine patients showed dystonia–athetosis–chorea but not tremor. In the literature, Dejerine and Roussy (1906), Sharp *et al.* (1994) and Kim *et al.* (1999) described similar symptoms, which were frequently, though not always, of delayed onset. As in our series (see below) the dystonia–athetosis–chorea in previously reported patients was closely related to positional sensory loss and has been described as pseudochoreoathetosis (Sharp *et al.*, 1994; Kim *et al.*, 1999). In our study, dystonia–athetosis was seen from the beginning of stroke in Patients 10 and 18, in whom the motor dysfunction was not severe. However, choreic movements became apparent some time after the onset. The second group is the dystonia–athetosis–chorea–action tremor group. The author has never observed a patient with isolated tremor following thalamic infarction. The tremor was usually associated with dystonia–athetosis–chorea, although in some patients (Patients 13 and 26), choreic components were

Table 2 *Differences between the patients with and without involuntary movements (IMs)*

	Patients with IMs (n = 35)	Controls (n = 58)	
Age (year, mean ± SD)	57.5 ± 11.0	60.1 ± 8.4	
Female (%)	37.1	50	
Left-sided lesion (%)	45.7	43.1	
Haemorrhagic stroke (%)	62.9	31	$P < 0.05$
Subtypes (LVI + pLVI, %)*	77	20	$P < 0.01$
Initial neurological presentation (n = 18 for patients with IMs; n = 37 for controls)			
Pure sensory stroke (%)	0	35.1	$P < 0.01$
Ataxic hemiparesis (%)	5.6	18.9	$P < 0.01$
Motor deficit (severe, %)	50	20	$P < 0.05$
Sensory deficit (severe, %)			
Pinprick	81.3	29.7	$P < 0.01$
Temperature	81.3	29.7	$P < 0.01$
Vibration	75	32.4	$P < 0.01$
Position	93.8	32.4	$P < 0.01$
Neurological deficits at the time of assessment			
Motor deficit (severe, %)	0	7.4	
Sensory deficit (severe, %)			
Pinprick	40	1.8	$P < 0.01$
Temperature	27.7	1.8	$P < 0.01$
Vibration	20	0	$P < 0.01$
Position	85.7	0	$P < 0.01$
Ataxia (severe, %)	60	5.4	$P < 0.01$
Paraesthesiae (%)	94	93.1	
Painful paraesthesiae (%)	57.1	57.1	
Follow-up (months; mean ± SD)	40.4 ± 48.9	26.9 ± 29.7	

LVI = large vessel infarction. *Among ischaemic stroke.

absent. The tremor of our patients was almost always aggravated on arm stretching or intentional voluntary movements (in particular the finger-to-nose test), and observed during rest only when it was very severe; therefore, the tremor may be called an 'action tremor'. The presence of action or intention tremor due to thalamic stroke has been recognized previously (Schlitt *et al.*, 1986; Mano *et al.*, 1993; Mossuto-Agatiello *et al.*, 1993; Miwa *et al.*, 1996; Qureshi *et al.*, 1996; Soler *et al.*, 1999). In addition, Sunohara and colleagues described a patient showing dystonia and irregular oscillation (1–2.5 Hz) of the arm and leg, which was always induced by voluntary action (Sunohara *et al.*, 1984). Case 3 in Kim (1992), the patients described by Ferbert and Gerwig (1993) and Mossuto-Agatiel (1993), and case 4 of Louis *et al.* (1996) had a mixture of dystonia-choreoathetosis–tremor. As expected, many patients described as having hand dystonia (Karsidag *et al.*, 1998) or pseudochoreoathetosis (Kim *et al.*, 1999) actually had additional intention tremor.

Furthermore, in our series, the patients' involuntary movements usually fluctuated in amplitude and rhythmicity according to patients' conditions. Therefore, it would be impossible to differentiate clearly dystonia from athetosis, athetosis from chorea (Adams *et al.*, 1997; Fahn, 1997), and even chorea from tremor. As will be discussed, the tremoric component of the involuntary movements was almost always associated with ataxia on the finger-to-nose manoeuvre, and the jerky, myoclonic components of the involuntary movements appeared to be the most severe form of the ataxic tremor (Manto, 1996). In the literature, Lehéricy *et al.* (1996) described four patients who had 'myoclonic dystonia', indicating hand dystonia plus postural, irregular myoclonic jerks with variable amplitude and duration, while Ghika *et al.* (1994) described complex movement disorders characterized by jerky, ballistic, unsteady hands. Therefore, although there indeed were variations in the manifestation, the symptoms we observed may be collectively described as delayed-onset mixed (dystonia–athetosis–chorea–action tremor) involuntary movements, which encompass the extremely variable spectrum of the complex movement disorders.

Presumed pathogenesis of mixed involuntary movements

Role of sensory deficit

We found that the patients with involuntary movements had severe sensory deficit more often than control subjects in both the acute and chronic stages. Among sensory modalities, however, only the proprioceptive sensation remained impaired while the spinothalamic (pinprick and temperature) sensations frequently improved or were occasionally perceived in an

exaggerated manner at the time of assessment of involuntary movements. The only patient who retained normal position sense (Patient 13) had mild dystonia plus intention tremor without athetoid-choreic movements. During follow-up, Patient 10 had resolution of dystonia–athetosis–chorea along with the improvement of the proprioceptive sensory deficit. Patient 12 had more severe sensory deficit in the ulnar-sided fingers than radial-sided ones, and dystonia–athetosis–chorea was also more severe in the former.

Thus, the dystonia–athetosis–chorea components of mixed involuntary movements seem to be a pseudochoreoathetosis due to the loss of position sense as addressed previously by other authors (Sharp *et al.*, 1994; Ghika and Bogouslavsky, 1997). The loss of proprioceptive inputs to multiple joint movements may produce impaired synergic stabilization, resulting in spontaneously moving fingers (choreoathetosis). In addition, it has also been shown that development of dystonia is related to proprioceptive sensory dysfunction (Ghika *et al.*, 1993; Hallett, 1995; Byl *et al.*, 1996). In patients with hand dystonia, Bara-Jimenez *et al.* (1998) found that the area representing D1 in relation to D5 was reorganized in their primary somatosensory cortex. Moreover, Tinazzi and colleagues recently reported enhanced cortical somatosensory-evoked potentials in patients with dystonia (Tinazzi *et al.,* 1999). These pieces of evidence suggest that decreased proprioceptive sensory input may result in excessive cortical activation that leads to an abnormal co-contraction of antagonistic muscles, resulting in dystonic postures (Cohen and Hallett, 1988).

Although severe spinothalamic sensory deficit was also present in some of our patients with involuntary movements, this does not appear to play a role in producing involuntary movements considering that many patients with improved spinothalamic sensory function developed involuntary movements as well. In our study, the frequency of paraesthesiae or painful sensory symptoms did not differ between the patients with involuntary movements and the controls. It has been shown that post-stroke painful sensory symptoms are usually, although not always, associated with spinothalamic sensory dysfunction (Tasker *et al.*, 1991; Vestergaard *et al.*, 1995; Bowsher, 1996). Hypersensitivity to spinothalamic stimulation, especially cold sensation, is fairly common in these patients, which was observed in 12 of our patients. Thus, the paraesthesiae and spinothalamic sensory disturbances, commonly found in patients with involuntary movements, are a simple coexistence in the setting of severe sensory dysfunction, which is not related to the development of involuntary movements.

Role of cerebellar dysfunction

The proprioceptive sensory deficit alone does not appear to explain the full spectrum of the mixed involuntary movements. For example, Patient 6 had mild positional sensory deficit, but also a severe, jerky hand tremor. Patient 13 did not have position sensory deficit, but did have a dystonia-intention tremor. Previous literature also showed patients with intact sensation who developed tremor (Mossuto-Agatiollo *et al.*, 1993). In our study, severe cerebellar ataxia was significantly more common in patients with involuntary movements than in controls. Considering the imaging findings, the lesions seem to have involved the cerebellorubrothalamic-cortical tract at or near the ventral lateral nucleus, which revealed cerebellar ataxia as the patients' initial hemiparesis improved. In the present study, only three patients (Patient 3, 11 and 33) did not have ataxia on the finger-to-nose manoeuvre. All of them had mild dystonia–athetosis–chorea without tremoric components. During the follow-up of the patients, the author occasionally observed patients whose performance of the finger-to-nose manoeuvre became progressively disorganized, jerky, and accompanied by severe action tremor. The jerky hand movements or gross oscillatory arm movements were always associated with moderate to severe limb ataxia (Table 1). Therefore, the jerky, myoclonic hand movements are likely to be an exaggerated manifestation of cerebellar tremor, indicating severe loss of synergic stabilization of agonist/antagonist muscles (Manto, 1996).

The dysmetria of our patients may, at least in part, be related to proprioceptive loss. The so called proprioceptive ataxia is often indistinguishable from cerebellar ataxia (Critchley, 1953). With significant deafferentiation, the patient is unable to maintain a constant motor output and fails to sustain long sequences of motor programs and to maintain a constant level of contraction to hold a specified position or force (Rothwell *et al.*, 1982). In an experimental study with monkeys subjected to cerebellar injury, Mackel (1987) found that compensation of cerebellar deficits was considerably impaired if the sensory cortex was concomitantly removed. Moreover, modulation of peripheral sensory input was shown to affect the severity of cerebellar tremor (Dash, 1995). Thus, persistent failure of the proprioceptive system in our patients may have augmented or perpetuated the cerebellar tremor. Delayed-onset or progressively worsening cerebellar syndrome has been recognized previously mostly in patients with strokes occurring in the thalamomesencephalic region (Louis *et al.*, 1996).

The role of unbalanced recovery of motor dysfunction or spasticity

In the present study, the patients with involuntary movements had initially severe hemiparesis significantly more often than control subjects. However, the motor deficit had invariably improved at the time of assessment of involuntary movements, when the severity was not different between the patients with involuntary movements and controls. Therefore, the initial limb weakness and its satisfactory recovery may play a role in the development or aggravation of involuntary movements. There are several pieces of evidence for this argument. First, control subjects presented more often with syndromes of

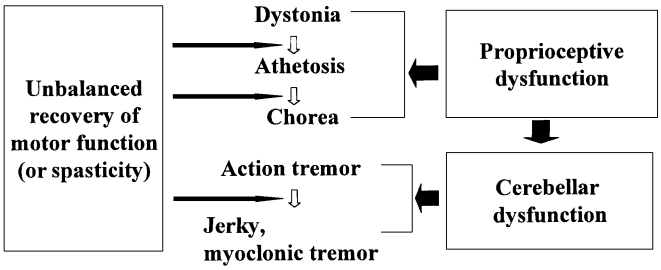

Fig. 3 Schematic diagram showing the pathogenesis of involuntary movements.

pure sensory stroke or hemihypaesthetic ataxic hemiparesis than patients with involuntary movements (Table 2) that require normal or only mildly impaired muscle strength. Despite the presence of sensory and/or cerebellar dysfunction, these patients did not develop delayed-onset involuntary movements. Secondly, among the patients with involuntary movements, nine (Patients 1, 8, 10, 11, 13, 14 and 16–18) had relatively mild (⩾IV) initial hemiparesis. Although they did develop delayed onset involuntary movements, their symptoms were usually mild and without jerky, myoclonic hand movements (Table 1). Thirdly, in Patients 10 and 18, initial dystonia–athetosis gradually changed into more unstable movements, chorea and chorea–action tremor, respectively, as their motor dysfunction improved. Fourthly, the involuntary movements in our patients were not present during the acute stage. The delayed onset (see below) or progressively worsening symptoms for several weeks or months (see Results) appears to be related to coincidental improvement of muscle strength.

It appears that relatively successful recovery of muscle strength as opposed to persistent failure in the proprioceptive and cerebellar system results in worsening of the unsteadiness of the involuntary movements in the direction of dystonia→athetosis→chorea, and action tremor→gross oscillation or jerky, myoclonic movements (Fig. 3). It has been shown that the functional recovery of motor dysfunction is related to a plastic reorganization of the motor cortex or activation of the uncrossed pyramidal pathways from the opposite hemisphere (Chollet *et al.*, 1991; Lee and van Donkelaar, 1995). In the presence of persistent failure of original proprioceptive and cerebellar inputs, the newly organized proprioceptive/cerebellar motor integrative system should be unstable or even misdirected. However, whether the cause of the instability is the unbalanced recovery of the limb strength itself or consequent spasticity cannot be clearly differentiated in our study.

Involved body sites

In our patients, fingers/hands were predominantly involved, whereas other body parts were generally spared. In several patients in whom toes were involved, the position sensation was also lost in toes, and the heel-to-shin test revealed dysmetria. The predominant involvement of hands in our study is related to the fact that sensory motor dysfunction in the feet was milder than in the hands. Previous studies also reported that sensory symptoms are relatively more frequent in the hands than in the feet in patients with thalamic stroke (Kim, 1994), and motor impairment is more severe and frequent in the upper than lower extremity in patients with strokes involving the internal capsule (Donnan *et al.*, 1991). In addition, Bastian and Thach (1995) found that fine pinching movements using fingers were more often disorganized than reaching movements using the shoulder/elbow in patients with ventrolateral thalamic nucleus lesions, whereas both movements were similarly impaired in patients with cerebellar lesions. This observation may also explain the relatively predominant involvement of the distal rather than proximal limb in our patients with thalamic lesions.

Location, nature and pathogenic mechanism of the stroke

As shown in Fig. 1, all the patients had lesions in the lateral-posterior part of the thalamus, the majority in the thalamogeniculate artery territory and some in the posterior choroidal artery territory. The internal capsule was also involved to a variable degree. We found that haemorrhagic (versus ischaemic) stroke was significantly more frequent in patients with involuntary movements than in controls. Among the patients with ischaemic stroke, large vessel stenosis/occlusion is more commonly associated with patients with involuntary movements, whereas small vessel disease is more often associated with controls. These findings, as well as the neurological deficits discussed above, imply that relatively large, destructive lesions simultaneously involving the ventral posterior nucleus, the ventral lateral nucleus and the internal capsule produce mixed involuntary movements, whereas small lacunar infarcts usually do not. Previously, Lehéricy *et al.* (1996) and Krystkowiak *et al.* (1998) attempted to identify by MRI the lesions responsible for involuntary movements and suggested that centromedian nucleus lesions are important in producing dystonia. However, their patients were small in number, and the possibility that lesions occurring at other areas produce similar symptoms cannot be excluded. In the presence of multiple components of involuntary movements and large lesions in most of our cases, it was impossible to localize precisely the specific nuclei responsible for the individual component of involuntary movements.

Delayed onset

The reason for the delayed onset of involuntary movements remains speculative. The delay may indicate the time required for the unbalanced, but successful recovery of the motor function (or the development of spasticity) and consequent development of pathological neuronal circuitry. It also may reflect the time required for the possible changes in neuronal

synaptic activities (Ohye *et al.*, 1985; Ghika *et al.*, 1994; Louis *et al.*, 1996; Scott and Jankovic, 1996).

Are delayed onset mixed involuntary movements specific for thalamic lesion?

Although the purpose of this paper was to describe delayed onset, mixed involuntary movements caused by thalamic strokes, during the study period the author observed similar delayed-onset symptoms in patients with strokes occurring in the lenticulocapsular area ($n = 4$), the frontoparietal area ($n = 2$), the dorsal pons ($n = 5$) and the medial medullary area ($n = 2$). All the patients had prominent position sensory loss, cerebellar ataxia and relatively well recovered muscle strength. Therefore, although thalamic strokes occupy the majority of the lesions producing delayed onset mixed involuntary movements, these symptoms can also be produced by non-thalamic strokes as long as they damage the lemniscal sensory system, cerebellar output fibres and, less severely, the pyramidal tract.

Acknowledgement

I wish to thank Dr D. G. Jung for his interpretation of some of the French articles.

References

Adams RD, Victor M, Ropper AH. Abnormalities of movement and posture due to disease of the basal ganglia. In: Adams RD, Victor M, Ropper AH. Principles of neurology. 6th ed. New York: McGraw-Hill; 1997. p. 64–83.

Bara-Jimenez W, Catalan MJ, Hallett M, Gerloff C. Abnormal somatosensory homunculus in dystonia of the hand. Ann Neurol 1998; 44: 828–31.

Bastian AJ, Thach WT. Cerebellar outflow lesions: a comparison of movement deficits resulting from lesions at the levels of the cerebellum and thalamus. Ann Neurol 1995; 38: 881–92.

Bogousslavsky J, Regli F, Uske A. Thalamic infarcts: clinical syndromes, etiology, and prognosis. Neurology 1988; 38: 837–48.

Bowsher D. Central pain: clinical and physiological characteristics. J Neurol Neurosurg Psychiatry 1996; 61: 62–9.

Byl NN, Merzenich MM, Jenkins WM. A primate genesis model of focal dystonia and repetitive strain injury: I. Learning-induced dedifferentiation of the representation of the hand in the primary somatosensory cortex in adult monkeys. Neurology 1996; 47: 508–20.

Chollet F, DiPiero V, Wise RJ, Brooks DJ, Dolan RJ, Frackowiak RS. The functional anatomy of motor recovery after stroke in humans: a study with positron emission tomography. Ann Neurol 1991; 29: 63–71.

Cohen LG, Hallett M. Hand cramps: clinical features and electromyographic patterns in a focal dystonia. Neurology 1988; 38: 1005–12.

Critchley M. The parietal lobes. London: Edward Arnold; 1953. p. 160–1.

Dash BM. Role of peripheral inputs in cerebellar tremor. Mov Disord 1995; 10: 622–9.

Dejerine JJ, Roussy G. Le syndrome thalamique. Rev Neurol (Paris) 1906; 14: 521–32.

Donnan GA, Bladin PF, Berkovic SF, Longley WA, Saling MM. The stroke syndrome of striatocapsular infarction. Brain 1991; 114: 51–70.

Fahn S. Phenomenology of movement disorders. In: Fahn S, Marsden CD, Jankovic J, editors. 7th annual course: a comprehensive review of movement disorders for the clinical practitioner. New York: Columbia University; 1997. p. 3–61.

Ferbert A, Gerwig M. Tremor due to stroke. Mov Disord 1993; 8: 179–82.

Foix C, Hillemand P. Les syndromes de la région thalamique. Presse Med 1925; 33: 113–7.

Garcin R. Syndrome cérébello-thalamique par lésion localisée du thalamus avec une digression sur le 'signe de la main creuse' et son intérêt sémiologique. Rev Neurol (Paris) 1955; 93: 143–9.

Gatto EM, Zurru MC, Rugilo C, Mitre S, Pardal AM, Martinez M, et al. Focal myoclonus associated with posterior thalamic hematoma. Mov Disord 1998; 13: 182–4.

Ghika J, Bogousslavsky J. Spinal pseudoathetosis: a rare, forgotten syndrome, with a review of old and recent descriptions. Neurology 1997; 49: 432–7.

Ghika J, Regli F, Growdon JH. Sensory symptoms in cranial dystonia: a potential role in the etiology? J Neurol Sci 1993; 116: 142–7.

Ghika J, Bogousslavsky J, Henderson J, Maeder P, Regli F. The 'jerky dystonic unsteady hand': a delayed motor syndrome in posterior thalamic infarctions. J Neurol 1994; 241: 537–42.

Hallett M. Is dystonia a sensory disorder? [editorial]. Ann Neurol 1995; 38: 139–40.

Karsidag S, Ízer F, Sen A, Arpaci B. Lesion localization in developing poststroke hand dystonia. Eur Neurol 1998; 40: 99–104.

Kase CS, Maulsby GO, deJuan E, Mohr JP. Hemichorea-hemiballism and lacunar infarction in the basal ganglia. Neurology 1981; 31: 452–5.

Kim JS. Delayed onset hand tremor caused by cerebral infarction. Stroke 1992; 23: 292–4.

Kim JS. Restricted acral sensory syndrome following minor stroke: further observation with special reference to differential severity of symptoms among individual digits. Stroke 1994; 25: 2497–502.

Kim JS, Choi-Kwon S. Sensory sequelae of medullary infarction: differences between lateral and medial medullary syndrome. Stroke 1999; 30: 2697–703.

Kim JW, Kim SH, Cha JK. Pseudochoreoathetosis in four patients with hypesthetic ataxic hemiparesis in a thalamic lesion. J Neurol 1999; 246: 1075–9.

Krystkowiak P, Martinat P, Defebvre L, Pruvo JP, Leys D, Destée A. Dystonia after striatopallidal and thalamic stroke: clinicoradiological correlations and pathophysiological mechanisms. J Neurol Neurosurg Psychiatry 1998; 65: 703–8.

Lee RG, van Donkelaar P. Mechanisms underlying functional recovery following stroke. Can J Neurol Sci 1995; 22: 257–63.

Léhéricy S, Vidailhet M, Dormont D, Piérot L, Chiras J, Mazetti P, et al. Striatopallidal and thalamic dystonia: a magnetic resonance imaging anatomoclinical study. Arch Neurol 1996; 53: 241–50.

Louis ED, Lynch T, Ford B, Greene P, Bressman SB, Fahn S. Delayed-onset cerebellar syndrome. Arch Neurol 1996; 53: 450–4.

Mackel R. The role of the monkey sensory cortex in the recovery from cerebellar injury. Exp Brain Res 1987; 66: 638–52.

Mano Y, Nakamuro T, Takayanagi T, Mayer RF. Ceruletide therapy in action tremor following thalamic hemorrhage. J Neurol 1993; 240: 144–8.

Manto M. Pathophysiology of cerebellar dysmetria: the imbalance between the agonist and antagonist electromyographic activities. [Review]. Eur Neurol 1996; 36: 333–7.

Marsden CD, Obeso JA, Zarranz JJ, Lang AE. The anatomical basis of symptomatic hemidystonia. Brain 1985; 108: 463–83.

Martin JP. Hemichorea resulting from a local lesion of the brain (syndrome of the body of Luys). Brain 1927; 50: 637–51.

Massey EW, Goodman JC, Stewart T, Brannon WL. Unilateral asterixis: motor integrative dysfunction in focal vascular disease. Neurology 1979; 29: 1180–2.

Miwa H, Hatori K, Kondo T, Imai H, Mizuno Y. Thalamic tremor: case reports and implications of the tremor-generating mechanism. [Review]. Neurology 1996; 46: 75–9.

Mossuto-Agatiello L, Puccetti G, Castellano AE. 'Rubral' tremor after thalamic haemorrhage. J Neurol 1993; 241: 27–30.

Ohye C, Shibazaki T, Hirai T, Kawashima Y, Hirato M, Matsumura M. Plastic change of thalamic organization in patients with tremor after stroke. Appl Neurophysiol 1985; 48: 288–92.

Pullicino PM. Diagrams of perforating artery territories in axial, coronal, and sagittal planes. Adv Neurol 1993; 62: 40–61.

Qureshi F, Morales A, Elble RJ. Tremor due to infarction in the ventrolateral thalamus. Mov Disord 1996; 11: 440–4.

Rothwell JC, Traub MM, Day BL, Obeso JA, Thomas PK, Marsden CD. Manual motor performance in a deafferented man. Brain 1982; 105: 515–42.

Scott BL, Jankovic J. Delayed-onset progressive movement disorders after static brain lesions. Neurology 1996; 46: 68–74.

Schlitt M, Brown JW, Zeiger HE, Galbraith JG. Appendicular tremor as a late complication of intracerebral hemorrhage. Surg Neurol 1986; 25: 181–4.

Sharp FR, Rando TA, Greenberg SA, Brown L, Sagar SM. Pseudochoreoathetosis: movements associated with loss of proprioception. Arch Neurol 1994; 51: 1103–9.

Soler R, Vivancos F, Munoz-Torrero JJ, Arpa J, Barreiro P. Postural tremor after thalamic infarction. Eur Neurol 1999; 42: 180–1.

Sunohara N, Mukoyama M, Mano Y, Satoyoshi E. Action-induced rhythmic dystonia: an autopsy case. Neurology 1984; 34: 321–7.

Tasker RR, de Carvalho G, Dostrovsky JO. The history of central pain syndromes, with observations concerning pathophysiology and treatment. In: Casey KL, editor. Pain and central nervous system disease: the central pain syndromes. New York: Raven Press; 1991. p. 31–58.

Tatu L, Moulin T, Bogousslavsky J, Duvernoy H. Arterial territories of the human brain: cerebral hemispheres. Neurology 1998; 50: 1699–708.

Tinazzi M, Frasson E, Polo A, Tezzon F, Bovi P, Deotto L, et al. Evidence for an abnormal cortical sensory processing in dystonia: selective enhancement of lower limb P37-N50 somatosensory evoked potential. Mov Disord 1999; 14: 473–80.

Vestergaard K, Nielsen J, Andersen G, Ingeman-Nielsen M, Arendt-Nielsen L, Jensen TS. Sensory abnormalities in consecutive, unselected patients with central post-stroke pain. Pain 1995; 61: 177–86.

Received July 10, 2000. Revised October 19, 2000.
Accepted October 18, 2000