J Neurol (1997) 244 : 579–585

ORIGINAL COMMUNICATION

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Central motor conduction studies in internal capsule and corona radiata infarction

Received: 13 January 1997
Received in revised form: 5 June 1997
Accepted: 27 June 1997

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Abstract Clinical and evoked-potential studies in internal capsule and corona radiata infarction are lacking. We report the results of a clinical and central motor conduction time (CMCT) study in 16 patients with internal capsule and 17 with computed tomography (CT)-proven corona radiata infarction. Patient's outcome was defined at the end of 3 months on the basis of the Barthel Index score. Four patients with type A capsular infarction (middle third of posterior limb of internal capsule) all had severe weakness, while 2 also had persistently unrecordable CMCT and poor outcome. Twelve patients with type B internal capsular infarction (genu, anterior limb, anterior or posterior third of posterior limb) had a milder degree of weakness, and CMCT was recordable in 9. At 3 months' follow-up, however, CMCT was recordable in all 12 patients. All of these patients had a partial (n = 4) or complete (n = 5) recovery. Thirteen patients with type A corona radiata infarction (middle third of corona radiata) had more pronounced weakness, and CMCT was unrecordable in all of these patients except 1 on initial examination. Follow-up after 3 months was possible in 8 patients, and CMCT became recordable in 3. One of these patients had complete, 3 partial, and 4 poor recovery. In type B corona radiata infarction (anterior or posterior third of corona radiata), the clinical signs and CMCT did not follow a regular pattern. Clinical and CMCT abnormalities in internal capsular infarction followed a more predictable pattern compared with those in corona radiata infarction. A less predictable pattern of weakness and CMCT change in corona radiata infarction may be attributed to a less definite organisation of motor pathways compared with the internal capsule.

Key words Motor evoked potential · Stroke · Infarction · Corona radiata · Internal capsule · Prognosis

Introduction

Internal capsule and corona radiata infarctions are among the most common lacunar strokes [19], which result in a varying degree of hemiplegia. The clinical picture and recovery pattern of capsular stroke have been reported [9]. The location of capsular infarction does not always result in a predictable pattern of weakness [10, 19, 21], which raises doubts about a homunculus organisation of the motor pathways in the internal capsule. The motor pathways can be studied objectively employing transcranial electrical or magnetic stimulation. There has been no comprehensive report correlating clinical and central motor conduction time (CMCT) changes in different locations of internal capsule and corona radiata infarctions. In a study on lacunar infarction, seven out of eight patients with internal capsular infarction had abnormal CMCT, but the location of infarctions in the internal capsule was not correlated with CMCT and these patients were not followed up

[1]. The study of clinical and CMCT changes in patients with capsular and corona radiata infarction may not only provide information about their outcome but also about the organisation of motor pathways. The present study reports the clinical and CMCT changes in different locations of capsular and corona radiata infarctions.

Patients and methods

In the present study, the patients with corona radiata and internal capsule infarctions were from a group of consecutive stroke patients managed by us during the period 1991–1996. The patients were examined within 2 weeks of the ictus, and the diagnosis was confirmed by computed tomography (CT) using a third-generation scanner, W400 Hitachi Japan. On CT the infarction appeared as hypodense lesions in the internal capsule or corona radiata. The infarctions were classified into type A if involving the middle third of the posterior limb of the internal capsule or corona radiata and type B if involving the anterior limb, genu or anterior or posterior third of the posterior limb of the corona radiata. The patients with a history of previous stroke or those with any other lesion on CT were excluded. A detailed neurological examination was carried out in each patient. Motor functions were assessed using the motoricity index (MI), which is a modification of the MRC (Medical Research Council) scale [7]. Muscle tone was classified into normal, hypotonia and spasticity. Pinprick, joint position, vibration and cortical sensations were also tested. The activities of daily life were assessed using the Barthel Index (BI). The patients were reevaluated at 1 and 3 months. The recovery was classified into complete, partial and poor on the basis of the Barthel index score at the end of 3 months. A score of 20 was defined as complete, 12–19 as partial and below 12 as poor recovery [17].

Patients' details

Our results are based on 16 patients with capsular and 17 with corona radiata infarction. All the patients were right handed, their mean age was 54 years (range 22–80) and 7 of them were female. They were examined after a mean duration of 5.8 days (range 1–14). The anatomical distribution of infarction is shown in Fig. 1. Four capsular and 13 corona radiata infarctions were of type A. On CT, there was no other abnormality to account for the clinical signs. Of the type A patients, all 4 patients with capsular (mean MI 0) and 12 patients with corona radiata (mean MI 16.0) infarction had pronounced motor deficits. Muscle tone was significantly reduced in 3 of 4 patients with capsular and 6 of 13 patients with corona radiata infarction. Tendon reflexes were diminished in 3 patients with capsular and 2 with corona radiata infarction. There were 12 patients with type B capsular and 4 with type B corona radiata infarction. Type B patients had milder weakness; the mean MI in capsular infarction was 54 (range 0–74) and in corona radiata infarction 34.3 (range 0–100). Muscle tone was reduced in 3 patients with capsular and in none with corona radiata infarction. Similarly, hyporeflexia was present in 2 patients with capsular and in none with corona radiata infarction.

Central motor conduction time

CMCT was recorded by stimulating the motor cortex using a high-voltage electrical stimulator Digitimer D-180, delivering a shock up to 150 V with a time constant of 50–100 μs. The stimulating electrode was a saline-soaked felt pad 1 cm in diameter mounted on a plastic handle. To activate the abductor digiti minimi (ADM), the cathode was placed at the vertex and the anode 7 cm laterally and 1 cm anterior to a line drawn from the vertex to the tragus. For cervical stimulation, the cathode was placed below the spinous process of the seventh cervical vertebra (7) and the anode proximal. During cortical stimulation, the patient was asked to contract the ADM slightly (10% of the maximum force irrespective of the degree of weakness). The patient was asked to relax during spinal stimulation. For cortical stimulation, the stimulus strength was progressively increased to get the maximum response. If the motor-evoked potential (MEP) was unrecordable, the stimulus strength was increased to 100% of the stimulator output. For spinal stimulation 50%–60% of the maximum output was employed. Three responses were obtained at 10-s intervals, and the one with the shortest latency was measured. The MEP was recorded from the ADM using a surface electrode in a belly tendon montage. The electromyogram (EMG) signals were amplified and filtered through 20–2000 Hz at a gain of 0.5–2.0 mV/division. Minimum onset latency and the amplitude of the negative phase were measured. Central motor conduction time was calculated by subtracting the latency of the MEP on C7 stimulation from that on cortical stimulation [18]. The results were compared with the normal values of

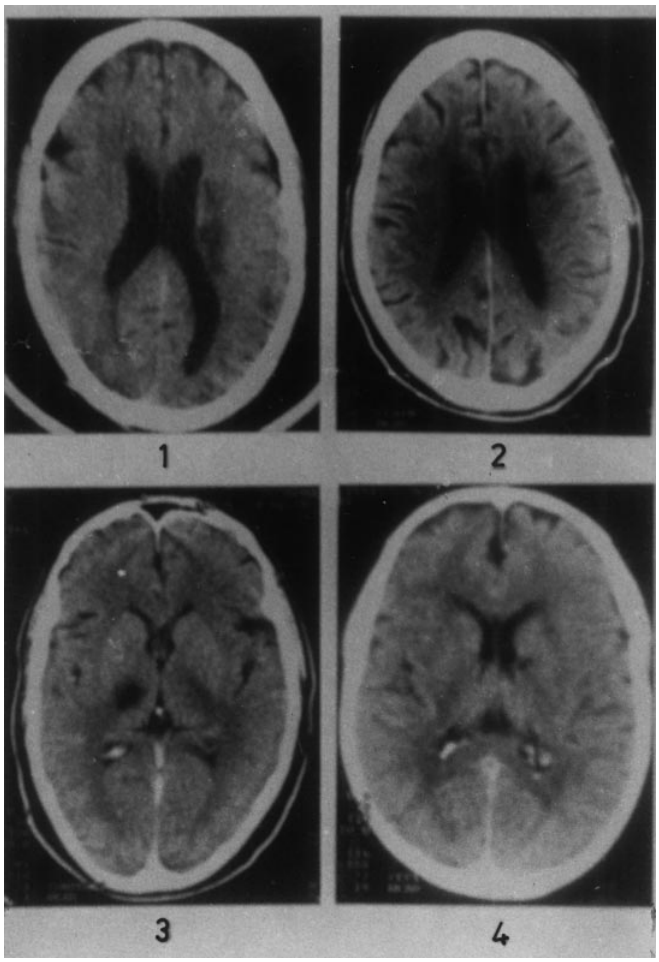

Fig. 1 Pattern of infarction: *(1)* type A corona radiata involving the middle third with anterior extension on the right side, *(2)* type B corona radiata infarction involving the anterior third, *(3)* type A internal capsular infarction involving the middle third of the posterior limb of the internal capsule on the left side, *(4)* type B internal capsular infarction involving genu of the internal capsule on the right side

our laboratory, which were obtained from 32 healthy adult volunteers whose age ranged between 18 and 50 years. The abnormality was defined as mean ± 2.5 SD of controls. The latency of MEP on cortical stimulation was 19.2 SD 1.2 ms, and that on spinal stimulation 13.9 SD 1.0 ms. CMCT-ADM was 5.1 SD 1.2 ms; the amplitude of MEP on cortical stimulation was 3.5 SD 1.8 mV and on spinal stimulation 6.1 SD 1.9 mV [17]. The CMCT was classified into (1) unrecordable, (2) prolonged or (3) normal. The amplitudes, although measured, have not been used for defining abnormality because of their wide variation even in normal subjects. The relationship between CMCT, clinical signs and outcome were evaluated using the chi-square test. For statistical analysis, the CMCT was categorised into recordable and unrecordable, muscle power into MI ≥ 50 or MI < 50 and tone into increased, reduced or normal.

Results

Initial CMCT abnormalities

The CMCT was unrecordable in all 4 patients with type A capsular and 12 out of 13 patients with type A corona radiata infarction. In the patients with type B infarction there was a mixed pattern of CMCT changes. In type B capsular infarction, CMCT was unrecordable in 3, prolonged in 3 and normal in 6 patients. In type B corona radiata infarction, CMCT was unrecordable in 2, and prolonged and normal in 1 patient each respectively. On the nonhemiplegic side, CMCT-ADM was normal in all patients. The clinical and CMCT findings in capsular infarction are shown in Table 1 and those in corona radiata infarction in Table 2.

Table 1 Clinical and central motor conduction-time (CMCT) findings in capsular infarction (*MI of UL* motoricity index of upper limbs, *CMCT-ADM* central motor conduction time to abductor digiti minimi in milliseconds, *ampl* amplitude of cortical motor-evoked potential in millivolts, *LIC* limb of internal capsule, *NR* not recordable, *post* posterior, *ant* anterior, *N* normal)

Patient	Age/sex (years)	Day	Location of infarction	MI of UL	Reflex	Tone	CMCT-ADM (ampl)		Outcome
							Hemiplegic	Nonhemiplegic	
Type A									
1	65 M	6	Post LIC	0	↓	↓	NR (NR)	4.4 (2.2)	Poor
		90		0	↑	↑	NR (NR)	4.4 (2.0)	
2	70 M	2	Post LIC	0	↓	↓	NR (NR)	4.6 (0.9)	Poor
		90		0	↑	↑	NR (NR)	3.4 (3.3)	
3	58 F	5	Middle 2/3	0	↑	↑	NR (NR)	8.0 (1.5)	Partial
		90	post LIC	71	↑	↑	4.2 (1.0)	7.2 (0.5)	
4	35 M	3	Middle 2/3 post LIC	0	↓	↓	NR (NR)	4.4 (1.8)	–
Type B									
5	52 M	3	Genu + ant	75	↑	↑	NR (NR)	7.2 (2.1)	Partial
		90	1/3 post LIC	84	↑	↑	7.8 (1.8)	5.0 (2.7)	
6	55 M	12	Genu + ant	75	↑	N	4.4 (0.6)	3.4 (2.0)	Partial
		90	1/3 post LIC	100	↑	N	4.4 (0.8)	3.4 (2.0)	
7	26 M	14	Genu + ant	28	↑	↑	NR (NR)	5.0 (1.4)	Complete
		90	1/3 post LIC	69	↑	↑	11.6 (1.2)	5.4 (3.5)	
8	57 M	8	Genu + ant	76	↓	N	7.6 (0.3)	7.0 (3.8)	Complete
		90	1/3 post LIC	100	↓	N	7.3 (0.3)	7.0 (3.8)	
9	62 M	3	Genu + ant	75	↑	↓	6.4 (2.0)	5.2 (2.6)	Partial
		90	1/3 post LIC	82	↑	↓	4.2 (2.8)	4.6 (2.5)	
10	70 F	3	Ant 1/3 post	48	↑	↑	8.4 (0.3)	5.4 (1.3)	Partial
		90	LIC	74	↑	↑	6.0 (1.2)	5.4 (1.3)	
11	43 M	2	Genu	74	↑	N	4.0 (2.1)	4.0 (3.5)	Complete
		90		100	↑	↑	4.0 (2.1)	4.0 (3.5)	
12	53 M	12	Genu	93	N	↓	NR (NR)	5.2 (2.4)	–
13	54 M	4	Genu	48	N	N	8.8 (0.8)	4.2 (4.5)	Complete
		90		100	N	N	4.2 (1.5)	4.2 (3.6)	
14	56 M	2	Genu	0	↑	↑	8.8 (1.2)	7.2 (3.7)	–
15	65 M	14	Post 1/3 post LIC	0	↑	N	7.4 (1.3)	6.0 (1.7) 6.3 (1.0)	–
16	45 M	1	Ant LIC	56	↓	↓	6.5 (0.8)	6.4 (1.0)	Complete
		90		100	N	N	6.5 (0.8)		

Table 2 Clinical and CMCT findings in corona radiata infarction

Patient	Age/sex (years)	Day	Location of infarct	Reflex	Tone	MI of UL	CMCT-ADM (ampl)		Outcome
							Hemiplegic	Nonhemiplegic	
Type A									
1	50 M	1	Middle 1/3	↑	↓	0	NR (NR)	6.2 (2.7)	Poor
		90		↑	↓	18.6	NR (NR)	6.8 (2.5)	
2	57 M	5	Middle 1/3	↓	↓	47	NR (NR)	6.4 (1.2)	Partial
		90		↑	↓	74	5.4 (1.2)	6.4 (1.1)	
3	80 M	2	Middle 1/3	↑	↑	37	NR (NR)	6.0 (3.5)	Partial
		90		↑	↑	65	4.2 (0.8)	5.4 (3.0)	
4	22 F	1	Middle 1/3	↑	↓	30	NR (NR)	4.3 (0.9)	–
5	42 M	2	Middle 1/3	↑	↓	39	NR (NR)	4.8 (0.8)	–
6	60 M	4	Ant 2/3	↑	↑	33	NR (NR)	4.8 (0.8)	Poor
		90		↑	↑	71	NR (NR)	4.8 (0.8)	
7	78 M	4	Ant 2/3	N	↓	0	NR (NR)	7.2 (2.5)	Poor
		90		↑	↑	0	NR (NR)	– –	
8	70 M	9	Ant 2/3	↑	↑	0	NR (NR)	5.2 (4.3)	–
9	63 M	14	Complete	↓	↓	0	NR (NR)	8.0 (2.4)	–
10	35 M	7	Ant 2/3	↑	↑	0	NR (NR)	8.0 (1.7)	Poor
		90		↑	↑	48	NR (NR)	8.0 (1.5)	
11	55 M	4	Complete	↑	N	0	NR (NR)	6.8 (1.1)	Partial
		90		↑	↑	74	8.2 (3.7)	6.0 (1.0)	
12	40 M	14	Ant 2/3	N	N	7	NR (NR)	7.2 (1.1)	–
13	45 F	5	Middle 1/3	↑	N	100	3.8 (1.2)	4.8 (3.5)	Complete
		90		↑	N	100	3.4 (1.6)	4.8 (3.2)	
Type B									
14	48 M	8	Post 1/3	↑	↑	37	11.6 (0.3)	4.8 (1.0)	Complete
		90		↑	↑	74	3.2 (1.2)	4.4 (1.3)	
15	52 F	3	Ant 1/3	↑	N	100	5.2 (1.5)	4.8 (1.0)	Complete
		90		↑	N	100	5.2 (1.7)	4.8 (1.1)	
16	62 F	7	Ant 1/3	↑	↑	0	NR (NR)	7.0 (1.3)	–
17	60 M	5	Ant 1/3	↑	↑	0	NR (NR)	4.5 (1.3)	–

Follow-up and clinical correlation

The clinical examination and CMCT studies were repeated in 22 patients, of whom 12 had capsular and 10 corona radiata infarction. In 2 out of 3 patients with type A capsular infarction the CMCT remained unrecordable and there was no improvement in muscle power (MI = 0). Both of these patients, however, developed spasticity, the reflexes became exaggerated and they had a poor outcome. One patient with type A capsular infarction had partial recovery and CMCT became normal; 8 patients with type A corona radiata infarction were followed up. Their mean MI improved from 30.9 to 56.3; hypotonia was replaced by spasticity in 1 and reflexes became exaggerated in 2 patients in whom the reflexes were initially reduced ($n = 1$) and normal ($n = 1$). Initially unrecordable CMCT became normal in 2, prolonged in 1, and remained unrecordable in the remaining 5 patients. One of these patients had complete, 3 partial and 4 poor recovery.

Nine patients with type B capsular infarction were followed up. These patients had less severe weakness compared with that of type A capsular infarction, which further improved after 3 months. The mean MI improved from 61.7 to 89.9. Two patients with initially flaccid ($n = 1$) and normal tone ($n = 1$) developed spasticity. In one patient the tendon reflexes which had been normal on initial examination, became exaggerated. The CMCT improved in both patients in whom it was initially unrecordable, although it was prolonged in 1 (Fig. 2). Five patients had complete and 4 had partial recovery. Out of 4 patients with type B corona radiata infarction, 2 were followed up and both had complete recovery. One patient (patient 15) had transient weakness for 2 days and no impairment of motor functions thereafter, and his CMCT was also normal.

The CMCT correlated with muscle weakness ($\chi^2 = 9.19$, $df = 1$, $P < 0.01$), and muscle tone ($\chi^2 = 9.35$, $df = 2$, $P < 0.01$). The CMCT abnormalities were more frequent in type A infarction compared with type B ($\chi^2 = 14.40$, $df = 2$, $P < 0.01$). The degree of weakness ($\chi^2 = 9.58$, $df = 2$, $P < 0.01$) and type of infarction ($\chi^2 = 10.50$, $df = 2$, $P < 0.01$) significantly correlated with the recovery of patients. One patient with type A corona radiata infarction did not follow an expected pattern of CMCT abnormality

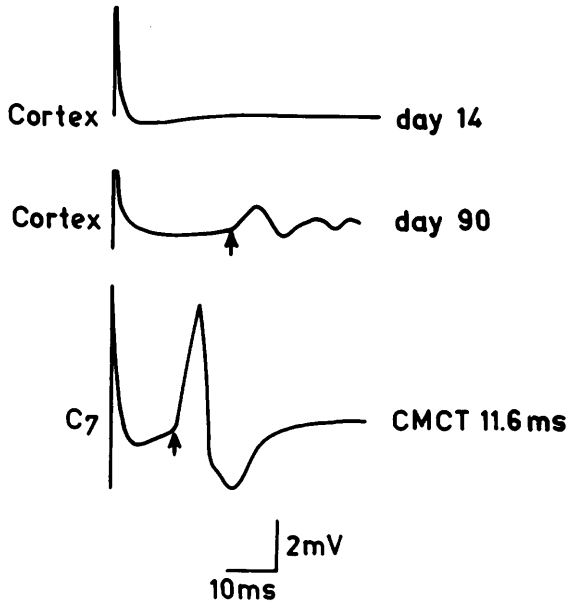

Fig. 2 Serial central motor conduction time (CMCT) recording in a patient with type B internal capsule infarction (patient 5), showing initially unrecordable CMCT becoming recordable although prolonged on day 90

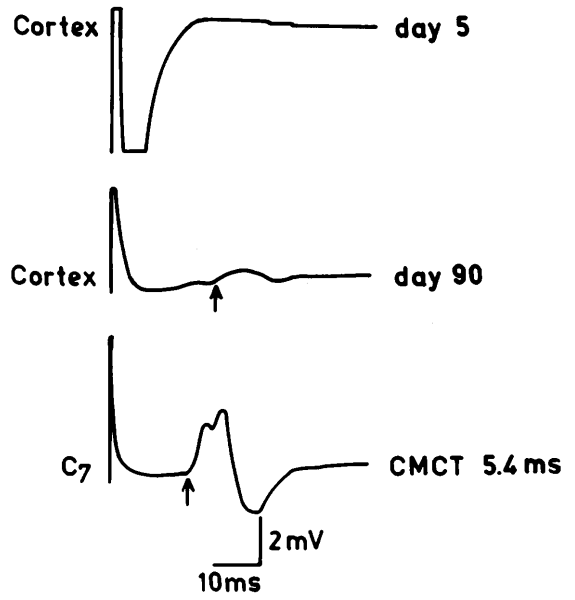

Fig. 3 Sequential CMCT studies in a patient with type A corona radiata infarction (patient 12). Initially unrecordable CMCT became normal on day 90

and recovery (patient 13). This patient had normal initial CMCT and recovered completely (Fig. 3).

Eleven patients could not be followed up. Their mean age was 51.6 years (range 22–70) and 9 of them were male. Four of these patients had capsular infarction (3 cases were of type B) and 7 corona radiata infarction (2 cases of type B). The mean MI of the patients with type A infarction was 12.6 and that of type B 18.6. The CMCT was not recordable in 9 patients (6 of type A) and was prolonged in 1 (type B). The age, sex and other clinical and radiological variables of this group were comparable with the group that was followed up.

Discussion

The CMCT was unrecordable in all except 1 patient with type A capsular and all except 1 with corona radiata infarctions, whereas in type B infarctions, it was unrecordable in 5, prolonged in 4 and normal in 7 patients in the acute stage. The classification of corona radiata and capsular infarctions into types A and B is based on the presumed involvement of the fibres from the primary motor area and its connections [9].

The reduction of MEP amplitude without dispersion may be consistent with a reduced number of functioning large diameter pyramidal axons [6, 23]. The amplitude of MEP on cortical stimulation, however, has been reported to correlate with neurological deficit [12], though wide variability of cortical MEP amplitude in normal subjects has also been reported [22]. In our control group the MEP amplitude also had a wide range. A number of patients with stroke may not be able to cooperate for required preactivation owing to pronounced weakness, aphasia or cognitive impairment rendering the MEP amplitudes less reliable. Many investigators therefore have not depended on the amplitude of cortical MEP for defining abnormality [3, 16]. We have recorded the amplitudes but have not correlated them with clinical variables for the above-mentioned reasons. CMCT on the nonhemiplegic side was normal in all of our patients, including those with unrecordable CMCT on the hemiplegic side. Therefore, cognitive impairment, aphasia and lack of motivation is unlikely to influence our results. In a study on acute stroke, the unrecordable CMCT was regarded as a feature of cortical lesions and prolongation of subcortical involvement [16]. Our results, however, differ from that study because in 21 of 33 patients with capsular or corona radiata infarction, the CMCT was not recordable. We used 100% stimulus strength before declaring the cortical MEP unrecordable. The disadvantage of such a high strength of stimulation is the spread of current. The spread of current to the other hemisphere does not influence an unrecordable MEP on the hemiplegic side, which was reported in our earlier study [18]. On increasing the stimulus strength the latency shortens and amplitude increases. This phenomenon is more prominent with magnetic than with electrical stimulation, especially at high stimulus strength [6].

In type A infarction, unrecordable MEP may be due to extensive damage to the motor pathways. In type B infarction, the primary motor pathways are likely to escape severe ischaemic injury. In these patients, the unrecordable CMCT may be due to desynchronisation of descend-

ing volleys as a result of partial damage to motor pathways, oedema or both. In some of these patients (patients 16 and 17) with profound weakness, the lack of preactivation may also contribute to unrecordable CMCT. In our study on initial examination, 3 patients with type B capsular infarction had prolonged CMCT (patients 10, 13 and 14). The CMCT prolongation in such patients may be due to partial interruption of motor pathways resulting in temporal dispersion of descending volleys [23]. The prolongation of CMCT may also result from a shift in the pyramidal fibre spectrum. The corticospinal fibres have variable diameters ranging between 1 and 20 μm. The majority of fibres (92%) are of small diameter (1–4 μm) [25]. The fast conducting fibres are more vulnerable to ischaemia. Following ischaemic lesions, therefore, only the slow conducting fibres may survive, and conduction through these may result in prolongation of CMCT [12].

In the follow-up studies 3 months after stroke, CMCT in type A capsular and corona radiata infarction remained unrecordable in 6 and was normal in 4 patients, whereas in type B infarction it was recordable in all patients with capsular and corona radiata infarctions. In 3 patients with type A corona radiata and 1 patient with type A capsular infarctions, however, the CMCT was normal. This unexpected finding may be because of more diffuse organisation of primary motor pathways in the corona radiata. The motor pathways generally maintain a well-defined organisation descending down the internal capsule. The primary motor cortex is represented in a single homunculus in the internal capsule with the hand and eyes in the anterior limb; the mouth, larynx and pharynx in the genu; the upper limb in the anterior and the lower limb in the posterior part of the posterior limb of the internal capsule [8]. A recent study on the topographic organisation of the motor pathways has revealed that the fibres from medial cortical structures such as the supplementary motor area and the limbic motor fields pass through the anterior limb of the internal capsule; those from the premotor cortex through the anterior-most part of the posterior limb; and from the primary motor area through the middle third of the posterior limb of the internal capsule [9]. The latter fibres may be concentrated in the middle third of the corona radiata, which is consistent with unrecordable CMCT in type A corona radiata infarction in our study.

In 1 patient with type A corona radiata infarction, CMCT was normal even in the initial stage (patient 13) and CMCT returned to normal at 3 months in 2 more patients (patients 2 and 3). These findings may result from partial damage to motor pathways, resolution of oedema and reorganisation of motor pathways [5, 13]. The pyramidal neurons establish synapses amongst themselves and adjacent cortical neurons [15]. The corticospinal neurons also establish synapses in the midbrain tegmentum and medullary reticular formation [5, 14]. It is possible that conduction through these pathways may be responsible for the improvement in CMCT. Transcranial electrical stimuli propagate longitudinally and the motor fibres are activated at the axon hillock or the first internode [23]. On increasing the strength of stimulation: motor pathways in the subcortical region, i.e. in the internal capsule and cerebral peduncle, may be stimulated, which may result in shortening of CMCT [4]. In our patients, however, CMCT values were within the normal range.

In our study, CMCT correlated with muscle weakness ($P < 0.01$); however, CMCT was recordable in patients with an MI of 0 (patients 14 and 15), who had a genu infarct. In CMCT studies the fastest conducting fibres are evaluated, which are responsible for phasic movements. Muscle-power testing relies on tonic muscle contraction, which is a function of slow-conducting motor pathways [2]. This can account for the occasional discrepancy between muscle power and CMCT.

A recordable CMCT in stroke is associated with a good outcome and unrecordable with a poor outcome [11, 17, 18]. The results of the present study are in agreement with this observation. In our study, the capsular infarction followed a more uniform pattern of clinical and CMCT changes. The infarctions involving the middle third of the posterior limb of the internal capsule had a profound motor deficit and poorer outcome compared with those involving the anterior limb, genu and anterior third of the posterior limb. In the literature, a wide variation in the clinical picture of capsular infarction has been reported. In a patient with severe hemiplegia, the infarction was confined to the third quarter of the posterior limb of the internal capsule [21], whereas another patient with a similar lesion had less severe clinical deficit [10]. A spectrum of symptoms of hemiparesis varying from complete hemiplegia to partial syndromes of faciobrachial or crural weakness has been reported [20, 24]. The discrepancy between the clinical and anatomical studies on the organisation of capsular motor pathways has been highlighted in a clinicopathological report [19], but all except 1 of our capsular infarctions had a predictable pattern of recovery. In our study, all patients with type B capsular infarction improved and only 1 with type A capsular infarction recovered partially. In corona radiata infarction, however, of 10 patients followed up, 4 did not have a predictable pattern (patients 2, 3, 11 and 13). This could be attributed to diffuse distribution of motor pathways in the corona radiata. Further studies are needed to evaluate the organisation of motor pathways from different motor areas and their clinical implications.

Acknowledgement We gratefully acknowledge the secretarial help of Mr. Shekhar in preparing the manuscript.

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