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Motor recovery following capsular stroke

Role of descending pathways from multiple motor areas

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SUMMARY

The functional anatomy of motor recovery was studied by assessing motor function quantitatively in 23 patients following capsular or striatocapsular stroke. While selective basal ganglia lesions (caudate and/or putamen exclusively) did not affect voluntary movements of the extremities, lesions of the anterior (plus caudate/putamen) or posterior limb of the internal capsule led to an initially severe motor impairment followed by excellent recovery, hand function included. In contrast, lesions of the posterior limb of the internal capsule in combination with damage to lateral thalamus compromised motor outcome. In experimental tracing of the topography of the internal capsule in macaque monkeys, we found axons of primary motor cortex passing through the middle third of the posterior limb of the internal capsule. Axons of premotor cortex (dorsolateral and post-arcuate area 6) passed through the capsular genu, and those of supplementary motor area (mesial area 6) through the anterior limb. Small capsular lesion can therefore disrupt the output of functionally and anatomically distinct motor areas selectively. The clinically similar motor deficits with a similar course of functional restitution following disruption of these different descending motor pathways indicate a parallel operation of cortical motor areas. They may have the further capability of substituting each other functionally in the process of recovery from hemiparesis.

INTRODUCTION

The central motor system is, in clinical practice, commonly considered to be hierarchically organized, with the primary motor cortex (area 4) in control of limb movements, executing its influence via the pyramidal tract (*see* Davidoff, 1990). Premotor cortex (dorsolateral area 6) and supplementary motor area (mesial area 6) are thought to contribute to 'planning' and 'preparation' of voluntary movements (Laplane *et al.*, 1977; Wise, 1985; Freund, 1987; Verfaellie and Heilman, 1987). It follows from this view that lesion of the primary motor cortex or pyramidal tract on one side should lead to persisting hemiparesis.

The clinical literature, however, contains three cases with autopsy proven complete pyramidal tract degeneration, yet excellent recovery of willed movements (Foerster, 1934; Aguilar, 1969; Ropper *et al.*, 1979). Further, surgical sections of the pyramidal tract

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at the peduncular or medullary level in patients with dyskinesias have produced surprisingly few lasting motor deficits (Bucy and Keplinger, 1961; *see* Bucy *et al.*, 1964; Jane *et al.*, 1968). Even unilateral surgical section of the pyramidal tract at spinal cord level allows almost complete motor recovery (Nathan and Smith, 1973).

Recently, we demonstrated Wallerian degeneration of the pyramidal tract *in vivo* by magnetic resonance imaging in patients with lacunar strokes located in the posterior limb of the internal capsule (Danek *et al.*, 1990). After severe initial hemiparesis, these patients experienced a remarkable motor recovery that included the capacity for independent finger movements (Fries *et al.*, 1990). Transcranial electrical stimulation of the damaged hemisphere could evoke motor responses not only in their contralateral, but also ipsilateral distal limb musculature (Fries *et al.*, 1991). Measurements of cortical metabolic activity in capsular stroke patients during use of the formerly paretic hand indicated substantial co-activation of ipsilateral motor cortex and contralateral non-primary motor areas (Fries *et al.*, 1990; Chollet *et al.*, 1991; Weiller *et al.*, 1992). Thus, motor recovery in these cases suggests a substantial functional reorganization using descending motor pathways which are unaffected by the lesion and bilaterally organized.

Experimental studies in monkeys led to the notion that corticospinal motor control does not rest solely on the integrity of primary motor cortex (*see* Strick, 1988). Corticospinal axons have been found to originate beyond primary motor cortex both from premotor cortical areas (dorsolateral premotor cortex and supplementary motor area) as well as from post-central areas (Murray and Coulter, 1981; Toyoshima and Sakai, 1982; Martino and Strick, 1987; Hutchins *et al.*, 1988). Their course through the internal capsule and the topographical arrangement within it, however, have not been reported.

Little evidence is available as to the origin and white matter course of the pyramidal tract in man. In the absence of positive findings, the notion is perpetuated that the origin of the pyramidal tract in man is more restricted than in monkey (Schoen, 1964; *see* Davidoff, 1990). Descending corticospinal fibres from the primary motor cortex have been demonstrated by gross dissection (Ross, 1980) to be located in the middle third of the posterior third quarter of the posterior limb of the internal capsule. Ischaemic lesions in this location have been found to cause almost complete degeneration of the ipsilateral pyramid (Englander *et al.*, 1975). Electric stimulation in this location during stereotaxic operations yielded low threshold excitation of distal limb musculature (*see* Hardy *et al.*, 1979). However, the primary motor cortex output contributes only 60% of the total of fibres in the medullary pyramid (Jane *et al.*, 1967). Degenerating fibres have been traced through the pyramid following surgical ablation of premotor cortex, using silver techniques (*see* Minckler *et al.*, 1944). Their position in the posterior limb of the internal capsule seems slightly anterior to that of primary motor cortex fibres when compared with the data of Ross (1980), Englander *et al.* (1975) and Hardy *et al.* (1979), yet their spatial relationship to primary motor cortex fibres has not been properly assessed.

In the present study we pursued the question of the functional anatomy underlying motor recovery by a twofold approach. First, we examined patients with partial capsular lesions at different locations due to ischaemic infarctions, asking whether they show differential motor deficits with a different course of recovery. Quantitative assessments of functional motor skills using the Rivermead Stroke Assessment (Lincoln and Leadbitter,

1979) were performed in the stable chronic stage after stroke. Results were correlated with site of lesion as reconstructed from computerized tomography (CT).

In parallel, we studied experimentally the topographical arrangement of descending fibre systems in the internal capsule. For that purpose, we injected, in macaque monkeys, the enzyme wheatgerm agglutinin coupled with horseradish peroxidase (WGA−HRP) into primary motor cortex, dorsolateral premotor cortex and supplementary motor area and reconstructed the course of labelled descending fibres.

PATIENTS AND METHODS

Twenty-three stroke patients were chosen according to the anatomical criterion of a single small pseudocystic lesion in the capsular region, as visualized by CT scans. Time since stroke ranged from 5 to 96 months (mean 31 ± 18 SD). Motor functions were assessed quantitatively using the Rivermead Stroke Assessment (Lincoln and Leadbitter, 1979), which probes functional motor skills rather than parameters such as force, speed and accuracy of movement. In this test, patients are required to perform a number of 'daily life' movements of increasing difficulty, assessing the function of axial, and proximal and distal limb musculature separately. Performance is rated by assessing the patient's ability to do the task, scoring '0' or '1', respectively. On 38 separate items (gross function, 13; leg and trunk, 10; arm and hand, 15) patients could score a maximum of 38 points. For better comparison, scores were converted into percentages of the maximum. Motor performance immediately after stroke was rated retrospectively from the patient's history and from the medical records, and was also quantified with the criteria of the Toronto Stroke Scale (Norris, 1976) which uses an ordinal scale for rating the degree of hemiparesis between 0 (none) and 4 (severe).

Sites of lesion were reconstructed from CT scans by projection onto appropriate stereotaxic atlas planes (Talairach *et al.*, 1967). According to lesion location, four patient groups were formed: (i) group BG had lesions confined to caudate or putamen; (ii) group ANT/BG had lesions of the anterior limb of the internal capsule also affecting caudate and/or putamen; (iii) group POST had lesions of the posterior limb of the internal capsule exclusively; (iv) group POST/THAL showed dorsolateral thalamus lesions in addition to lesions in the posterior limb of the internal capsule. All patients had suffered ischaemic infarction, except for patient 20 with a thalamic bleeding.

For experimental tracing studies, four macaque monkeys (*Macaca fascicularis*) received cortical injections of the tracer WGA−HRP in deep general anaesthesia (15 mg/kg body weight pentobarbital i.v., supplemented by 30 mg/kg ketamine plus 0.5 mg/kg xylazine). The tracer was delivered by a small Hamilton syringe (needle diameter 0.3 mm). In two of the four animals (PT_2 and PT_4), two neighbouring injections of 0.1 μl of a 1% solution of WGA−HRP in saline, spaced by 1 mm in the anterior−posterior direction, were placed into the mesial cortex and upper bank of cingulate sulcus [rostral part of the supplementary motor area representing hand and face (Woolsey *et al.*, 1952; Mitz and Wise, 1987; Hutchins *et al.*, 1988)]. One animal (PT_1) received three small injections (0.1 μl) 1 mm apart in the dorsoventral direction in the anterior bank of central sulcus in the hand and the foot representation, respectively. In one animal (PT_3) two small injections (0.1 μl), 1 mm apart, were made into the posterior bank of arcuate sulcus [supposed hand representation of post-arcuate premotor cortex (*see* Gentilucci *et al.*, 1988; Strick, 1988)]. Animals PT_1 and PT_3 received additional single injections of tritiated leucine (50 μCi) into post-arcuate premotor cortex, and hand representation of the primary motor cortex, respectively. After survival of 30−40 h, the animals were perfused transcardially in deep barbiturate anaesthesia with fixative (3% formaldehyde in phosphate buffer). Brains were removed from the skull and sectioned horizontally on a freezing microtome at 80 μm slice thickness. Standard histochemistry of HRP enzyme activity was performed on every fifth of the serial sections using tetramethylbenzidine (Mesulam, 1978) in order to visualize labelled axons in white matter and to trace their course through the internal capsule. A set of immediately adjacent sections was processed for standard autoradiography (Cowan *et al.*, 1972) using NTB3 (Kodak) emulsion (exposure time 4 weeks) and counterstained routinely with cresyl violet. Findings were documented both by the drawing of successive serial sections and by photomicrography at lower power dark field combined with polarized optics. Complete reconstructions of the injection sites will be documented elsewhere (W. Fries and A. Danek, unpublished results).

RESULTS

Clinical findings

The main clinical findings are listed in Table 1. The degree of initial paresis was rated using the criteria of the Toronto Stroke Scale (Norris, 1976). With patients grouped

TABLE 1. CLINICAL SIGNS DURING THE ACUTE STAGE OF CAPSULAR STROKE

Patient/age/sex	*Hemiparesis (side/face, arm, leg)*	*Hyperreflexia*	*Pyramidal signs*	*Spasticity*	*Sensory deficit*	*Dysarthria*
BG						
1/59/M	0	0	0	0	0	0
2/58/F	0	0	0	0	0	0
3/55/F	0	0	0	0	0	0
4/46/M	0	0	0	0	0	0
5/49/M	0	0	0	0	0	0
6/61/F	0	0	0	0	0	0
7/45/M	0	0	0	0	0	+
8/47/F	0	0	0	0	0	+
9/28/F	0	0	0	0	0	0
ANT/BG						
10/54/F	L/3, 4, 4	+	+	+	0	0
11/58/M	R/0, 4, 2	+	0	+	0	0
12/35/F	L/1, 4, 4	+	+	+	+	+
POST						
13/56/M	R/1, 4, 4	+	+	0	0	0
14/42/M	R/0, 4, 4	+	+	0	0	0
15/57/M	L/3, 3, 2	+	0	0	0	(+)
16/53/M	R/0, 2, 2	+	+	0	(+)	0
17/22/M	R/3, 4, 4	+	+	0	0	(+)
18/59/M	R/0, 2, 1	+	+	0	0	0
19/28/M	R/3, 4, 4	+	+	0	0	+
POST/THAL						
20/60/F	L/3, 4, 4	+	+	+	+	0
21/42/F	L/2, 4, 4	+	0	0	+	0
22/53/M	L/0, 3, 2	0	0	0	+	+
23/66/M	R/3, 4, 4	+	0	+	+	0

Clinical signs during the acute stage of capsular stroke in 23 patients, grouped according to the site of lesion. The degree of hemiparesis was rated according to the Toronto Stroke Scale (Norris, 1976) (grade 0 = none to grade 4 = plegia) for face, arm and leg separately, in that order; L and R refer to left and right side, respectively.

according to the site of lesion, a fairly homogeneous pattern of the initial motor deficit and its recovery emerged. Size and location of the lesions are shown at their maximal extent in semi-schematic horizontal sections of the capsular region [~8 mm above AC−PC level (Talairach *et al.*, 1967)]. It should be noted that none of the lesions extended into the cerebral peduncles, nor into the most ventral part of the internal capsule.

Lesions restricted exclusively to the basal ganglia did not cause a motor impairment of the extremities. In these patients (BG; $n = 9$), lacunar ischaemic lesions in the lenticulo-striate territory were confined to caudate or putamen (Fig. 1A). In most of

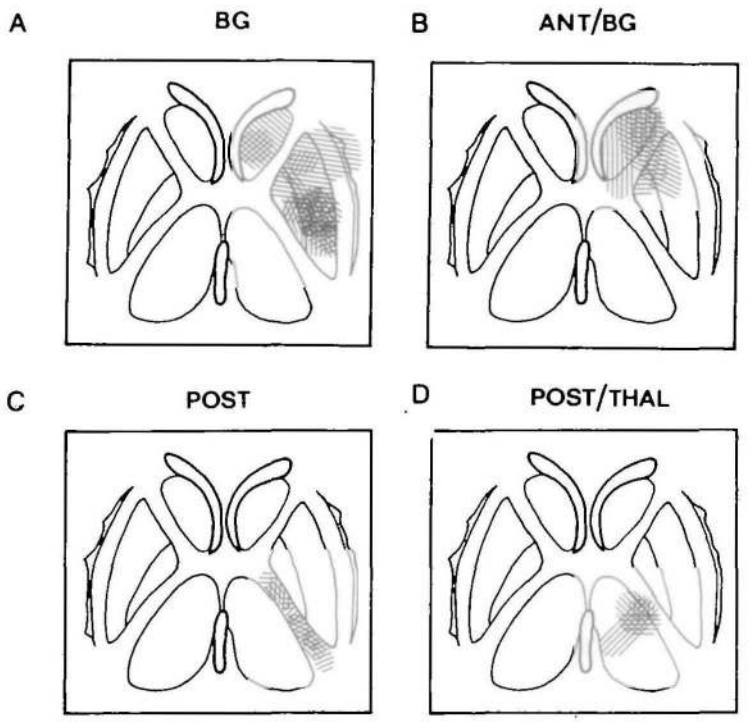


FIG. 1. Localization of 23 ischaemic lesions superimposed onto a representative drawing of the capsular region (~8 mm above AC–PC line; from Talairach *et al.*, 1967); the individual lesions as seen on CT are indicated by shading at different orientation. BG, lesions of the basal ganglia leaving both limbs of the internal capsule unaffected; ANT/BG, lesions of the anterior limb of the internal capsule including damage to the basal ganglia; POST, lesions confined to the posterior limb of the internal capsule; POST/THAL, lesions of the posterior limb of the internal capsule combined with damage to the dorsolateral thalamus.

the cases, the lesions were detected incidentally in CT scans performed for headache or other unrelated reasons; none of the patients reported motor symptoms of the limbs. There was a spell of dysarthria in two patients, none was hospitalized.

Damage to the anterior limb of the internal capsule (ANT/BG; $n = 3$), in the territory of the medial lenticulo-striate arteries and of Heubner's artery, led to an initially severe motor impairment. These lesions always included neighbouring basal ganglia, i.e. the head of the caudate nucleus and the putamen (Fig. 1B). Two of the three patients were hemiplegic. Mean global score of the Rivermead Stroke Assessment was $11.4 \pm 16.1\%$ SD; all three scored with their affected hand at 0%. In spite of this severe deficit, they recovered remarkably well, reaching a global score of $87 \pm 11.8\%$ SD and a score for hand function of $82.2 \pm 25.1\%$ SD (Fig. 2B).

A similar pattern emerged for the patients with lesions confined to the posterior limb of the internal capsule (POST; $n = 7$). Caused by infarction in the territory of the anterior choroidal artery, lesions were restricted to white matter without affecting neighbouring thalamic or basal ganglia tissue (Fig. 1C). Four of the seven patients presented with hemiplegia equally affecting arm and leg, and the other three showed hemiparesis of

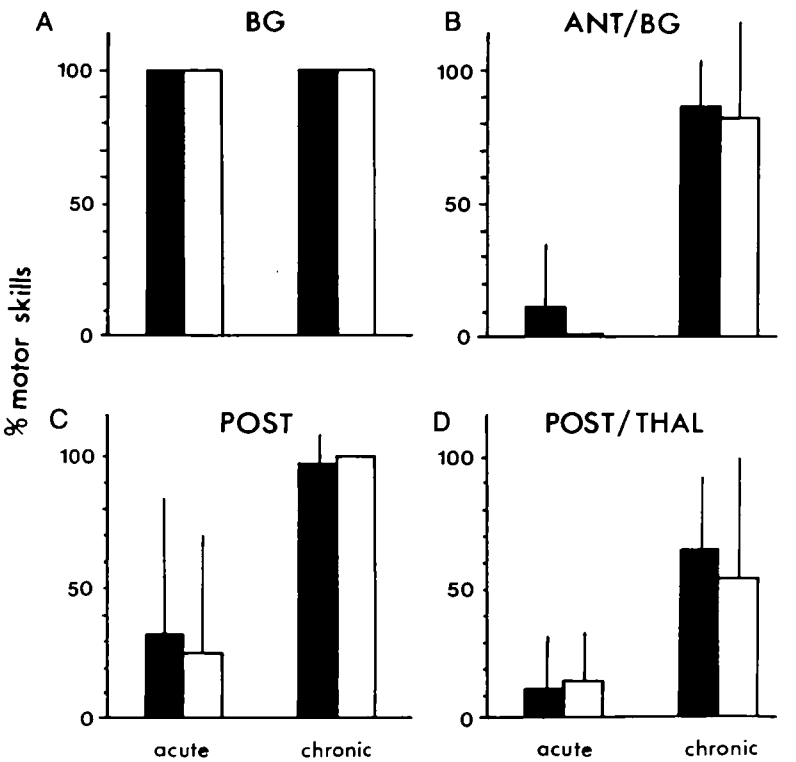


FIG. 2. Quantitative assessment of functional motor skills (Lincoln and Leadbitter, 1979) performed in the chronic stage ('chronic') following stroke (mean interval 31 ± 18 months SD), and, retrospectively, for the acute stage ('acute'). Filled columns indicate the global motor score, open columns indicate the score for hand motor function. Column height gives the percentage of the maximally obtainable score and represents the degree of functional motor skill. Patient groups are the same as in Fig. 1.

brachiofacial or crural type (*see* Table 1). Therefore, the score of the initial deficit (global motor function 31.2 ± 36.7% SD; hand function 26.7 ± 31.7% SD) showed substantial variability. All patients experienced excellent motor recovery (global score of 98.1 ± 3.6% SD). Hand function recovered to 100% without exception (Fig. 2C). This included independent finger movements as well as precision grip. Only dexterity on the affected side was inferior to that of the unaffected hand.

Patients with lesions affecting both the posterior limb of the internal capsule *and* lateral thalamus (POST/THAL; $n = 4$) experienced a less satisfactory motor recovery. In this group, one patient had suffered a small thalamic bleeding invading the internal capsule, the other three showed ischaemic infarctions in the territory of the thalamogeniculate arteries and possibly the anterior choroidal artery (Fig. 1D). The initial motor impairment of these POST/THAL patients (global score 12.5 ± 14% SD; hand score 15.5 ± 13.7% SD) was comparable to that after capsular lesions (ANT/BG and POST groups). Yet, the amount of motor recovery (global score 63.1 ± 19.8% SD; hand score 55.5 ± 32% SD) ranged clearly below ANT/BG and POST patients both for general and hand motor

performance (Fig. 2D, *see also* Table 1), although there was less structural damage in the internal capsule than in most POST patients. Particularly, the patient with the thalamic bleeding developed severe spasticity in the affected arm, rendering it functionally useless; another patient developed moderate to severe spasticity in his leg (Table 1).

Experimental tracing of descending motor pathways

Primary motor cortex, dorsolateral premotor cortex and supplementary motor area were injected in isolation, respectively, in each of the four macaque monkeys with the axonal tracer WGA–HRP, in order to elucidate course and topographic arrangement of their descending fibre tracts in the internal capsule. Tracer injections were confined to cortical grey matter with only marginal diffusion into underlying white matter (Fig. 3). White

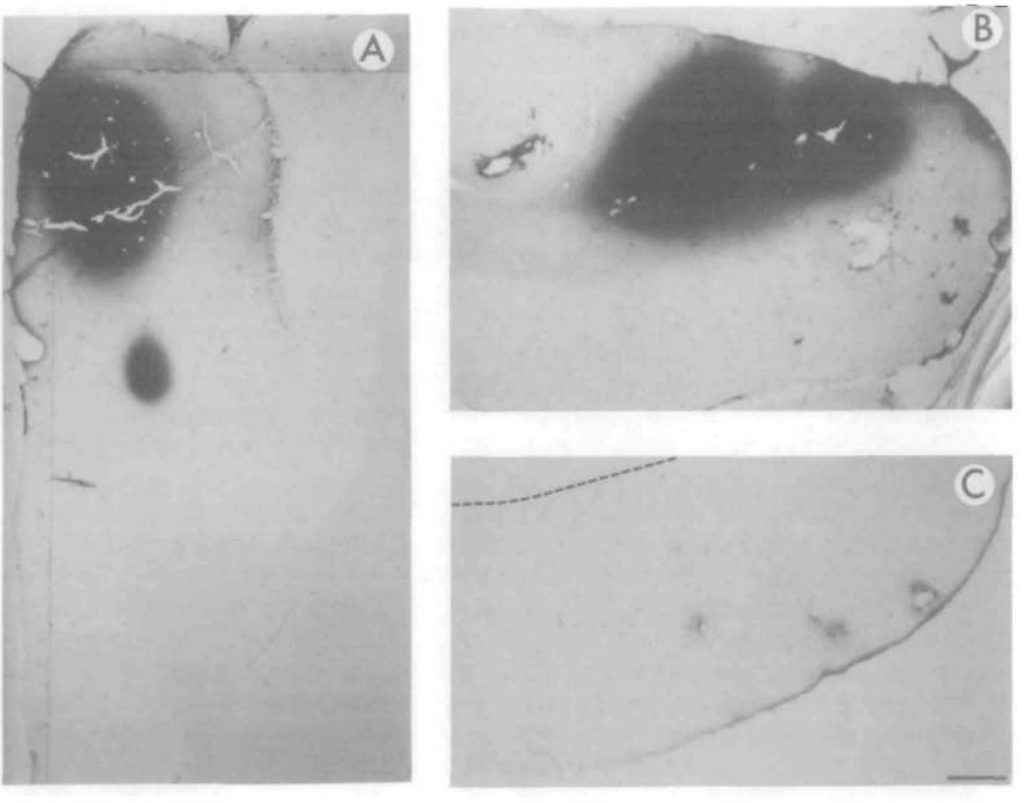
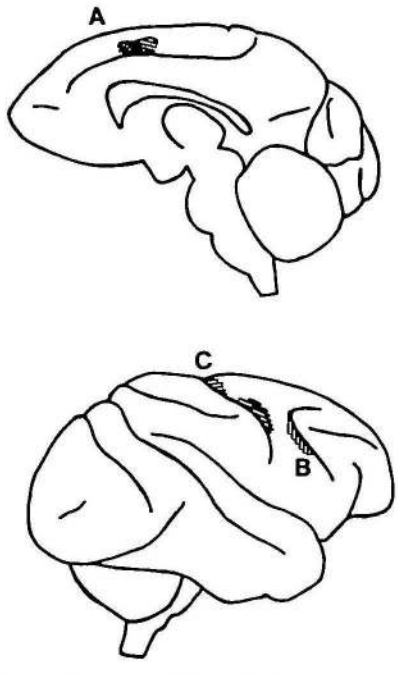


FIG. 3. Photomicrographs showing the injection sites and extent of injected label within the different motor fields following diaminobenzidine histochemistry of horizontal sections of monkey hemispheres. The respective injection sites are also indicated in the lateral view of the monkey hemisphere in Fig. 4. A, injections into mesial cortex corresponding to anterior supplementary motor area (Hutchins *et al.*, 1988); B, injections into the posterior bank of arcuate sulcus corresponding to premotor cortex (Gentilucci *et al.*, 1988); C, injections into the anterior bank of central sulcus corresponding to primary motor cortex (area 4); the label is partly faded, since the injected volume corresponded to that shown in A and B. The dashed line in C marks the approximate grey matter/white matter boundary. Anterior is top, in all three cases a right hemisphere is shown. The calibration bar = 1 mm. The material was not counterstained.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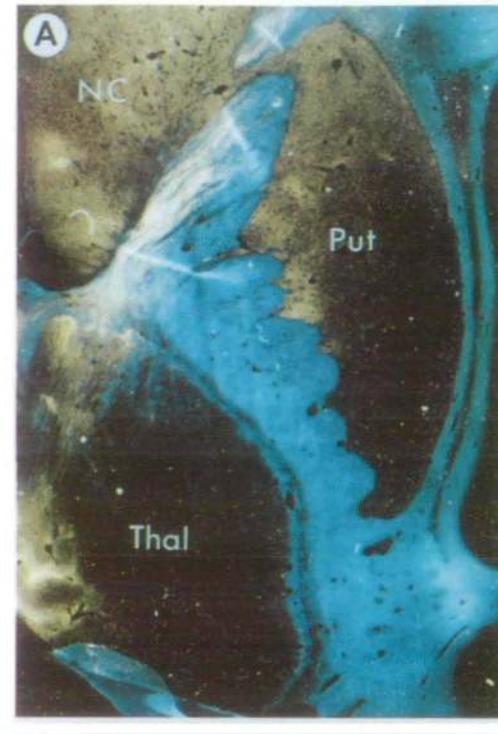
C
B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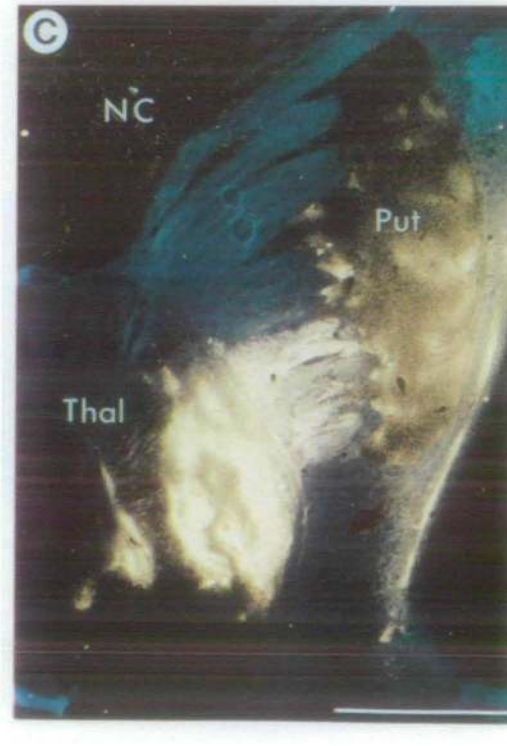
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matter was never hit by the needle tracks. The attribution of injection sites to cyto-architectonic areas and somatotopic subregions was made by comparison with standard maps of monkey motor areas (Brodmann, 1905; Woolsey *et al.*, 1952; *see* Strick, 1988). In the hand region of the primary motor cortex, retrogradely labelled cells were found in the anterior bank of central sulcus both after supplementary motor area and dorsolateral premotor cortex injections. Because of the well-known somatotopic arrangement of reciprocal connections of primary motor cortex with supplementary motor area and dorsolateral premotor cortex (*see* Muakkassa and Strick, 1979; Matelli *et al.*, 1986; Strick, 1988) this finding confirmed the proper location of the injection site.

The topographical distribution of labelled axons in the internal capsule showed an orderly arrangement (Fig. 4). Fibres descending from the supplementary motor area course first in a slightly anterior direction, then around the head of the caudate nucleus before entering the anterior limb of the internal capsule (Fig. 4A). There, they run almost horizontally until they reach and join, in the genu of the internal capsule, the descending fibre bundles coursing in a dorsoventral direction. Fibres from the dorsolateral premotor cortex occupy the anteriormost part of the posterior limb of the internal capsule close to its 'knee' (Fig. 4B). Fibres from primary motor cortex, representing both hand and foot, occupy the middle third of the posterior limb of the internal capsule (Fig. 4C). In each case, labelled fibres could be followed in their course through the internal capsule down to the cerebral peduncles. Labelled fibres from primary motor cortex were seen to shift position moderately in the posterior limb of the internal capsule similar to findings in man (Ross, 1980). Yet, there was no overlap in the position of labelled fibres when different animals with injections in different motor areas were compared. Findings of double labelling experiments with WGA–HRP and radioactively labelled amino acids (W. Fries and A. Danek, unpublished results) confirmed this view as there was little or no overlap of the spatial arrangement of primary motor cortex and dorsolateral premotor cortex fibres in their course through the internal capsule, until they have reached the cerebral peduncles. It follows that the different parts of the internal capsule are occupied by descending fibre tracts originating in distinct cortical motor fields. Since in each case, hand representation was injected, the separate course of labelled fibres suggests further that each set of fibres maintains its own somatotopical arrangement during its course through the internal capsule.

DISCUSSION

Clinical descriptions of ischaemic infarction in the region of the internal capsule vary substantially with respect to course and prognosis of motor deficits. This seems to depend

FIG. 4. Topographic arrangement of labelled descending fibre tracts in the internal capsule of macaque monkey following WGA–HRP injections into (A) supplementary motor area, (B) dorsolateral premotor cortex and (C) primary motor cortex. The site of injection is marked by shading on the lateral and medial view of a right hemisphere. Fibre tract labelling in the internal capsule is indicated by the pale yellowish colour. Note also the differential terminal labelling in caudate nucleus (NC), putamen (Put) and thalamus (Thal) (low power photomicrograph of a 80 μm horizontal section at upper thalamic level showing anterior and posterior limb of the internal capsule of the right hemisphere; dark field combined with polarization optics). Calibration bar = 5 mm.

on selection criteria focusing on vascular territories and their syndromes or on underlying pathophysiological mechanisms rather than on the precise lesion topography and its anatomical relationship to structures of the pyramidal motor system. Thus, lacunar infarcts in general are considered to have an excellent short-term prognosis (Fisher, 1982; Mohr, 1982; Arboix *et al.*, 1990). Similarly, the syndrome of capsular pure motor hemiplegia seems to have a good motor outcome, including complete recovery from initially complete hemiplegia; there are, however, some exceptions (Fisher and Curry, 1965; Rascol *et al.*, 1982). Both cases of excellent and of poor motor recovery have been reported for capsular (Fisher, 1979) and striatocapsular infarcts (Bladin and Berkovic, 1984; Caplan *et al.*, 1990; Weiller *et al.*, 1990; Donnan *et al.*, 1991).

This variability in outcome after capsular stroke seems hard to reconcile with the traditional view, according to which the somatotopic representation of primary motor cortex (*see* Foerster, 1934; Penfield and Boldrey, 1937) is represented as a single homunculus at the internal capsule level, with head and eye in its anterior limb, mouth and larynx/pharynx in the genu, upper extremities in the anterior and lower extremities in the posterior part of its posterior limb (Déjerine, 1901; Foerster, 1936). Following this concept, a 'capsular genu syndrome' was described only recently, with predominantly masticatory–palatal–pharyngeal weakness (Bogousslavsky and Regli, 1990). Anatomical as well as experimental stereotactic data in humans are in agreement in restricting the pyramidal tract, defined as output fibres from primary motor cortex or fibres of low threshold excitability to electrical stimulation evoking repetitive distal limb movements, to the middle third of the posterior limb of the internal capsule (Bertrand *et al.*, 1965; Englander *et al.*, 1975; Hardy *et al.*, 1979; Ross, 1980). The central question therefore remains, why anterior limb lesions can lead to hemiplegia affecting both face, arm and leg (Rascol *et al.*, 1982; Caplan *et al.*, 1990; Weiller *et al.*, 1990) as much as posterior limb lesions, and why in both instances such good motor recovery as in our patients can occur.

Experiments in monkeys have convincingly established that the central motor apparatus consists of multiple cortical motor fields each containing a 'simusculus', i.e. a somatotopic representation of muscles/movements (*see* Strick, 1988). Although the functional significance of neuronal activity within these different motor areas is not yet fully understood, it seems justified to assume that there is division of labour by functional specialization in the cortical motor system, similar to that in sensory systems, such as the visual or the somatosensory cortex (Zeki, 1978; Kaas, 1983).

Our own experimental data suggest that the descending pathways from these multiple cortical motor maps run through the internal capsule at distinct locations, yet in an orderly, topographically organized manner. Hence, descending fibres from mesial cortical structures such as the supplementary motor area and the limbic motor fields pass through the anterior limb of the internal capsule, those from premotor cortex through the anteriormost part of the posterior limb and those from primary motor cortex through the middle third of the posterior limb. If we assume that the topographical arrangemement of descending motor pathways in man is widely homologous to that in monkey, it follows that small ischaemic lesions in the internal capsule can de-efferent the different motor areas selectively. Evidence from somatosensory evoked potential analysis showing selective de-afferentation of either prerolandic or parietal cortex, depending on a more anterior or posterior location of small ischaemic capsular lesions, supports this notion

(Mauguière and Desmedt, 1991). Thus, the precise anatomical reconstruction of lesion site in lacunar stroke offers an interesting model for the study of output functions of different motor areas.

Clinical reports of motor function following selective pre-central gyrectomy, though quite anecdotal (Foerster, 1936; Bucy, 1942; Jane *et al.*, 1967), and following surgical ablation of the supplementary motor area (Rostomily *et al.*, 1991) fit the picture of excellent outcome after selective damage of the anterior or posterior limb of the internal capsule.

Hence, time-course and degree of motor recovery, comparable in both instances, further confirms the view that the different motor areas operate in parallel rather than in a hierarchical fashion, and this suggests they are able to substitute for each other functionally. Contributions of the undamaged hemisphere to the process of recovery as suggested both by electrophysiological and metabolic studies (Fries *et al.*, 1990, 1991; Chollet *et al.*, 1991; Weiller *et al.*, 1992), mediated either by bilateral pathways or uncrossed or recrossing pyramidal tract fibres, may also play an important role. We consider, however, the reorganization of parallel-acting multiple motor areas ipsilateral to the lesion site as the central mechanism in motor recovery. Uncrossed pyramidal tract fibres, or pyramidal tract fibres recrossing at segmental level, from the undamaged hemisphere seem not to be able to generate much of functional recovery in patients with large or complete infarctions of the middle cerebral artery territory, or after subcortical infarcts undercutting most of the descending fibres in the corona radiata (W. Fries, unpublished observations), i.e. in cases where no cortical motor field or descending fibre system is left to take over the task of functional substitution. Electrophysiological evidence for such (ipsilateral) functional reorganization has been obtained recently at single neuron level in the supplementary motor area after experimental primary motor cortex lesions (Aizawa *et al.*, 1991). If somatosensory feedback is disturbed, the functional deficit recovers to a lesser degree than after a pure capsular lesion, as the cases of thalamic lesions extending into the posterior limb of the internal capsule have shown. Such a role of thalamic afferents in the mechanism of motor recovery is emphasized in an interesting animal model (Bornschlegl and Asanuma, 1987).

Implications for motor prognosis after stroke seem obvious. Partial and selective damage to one motor area has a good prognosis for functional recovery. The more motor areas or their descending pathways are affected, the lesser becomes the potential for neural functional substitution, and the poorer the outcome will be in terms of motor deficit. A concomitant lesion of post-central structures, common in subtotal middle cerebral artery infarction, further reduces the prospect of recovery.

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