Hand coordination following capsular stroke

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Summary
Motor outcome following stroke of the internal capsule is variable and its determinants are poorly understood. While many patients fully regain their abilities, recovery of motor functions remains incomplete in others. We analysed functional motor tasks of the upper limb to determine the pattern of focal disability after a small infarct of the internal capsule (‘pure motor stroke’) in the chronic stage (mean 2.4 years after stroke) with kinematic recordings of a reaching-to-grasp movement, with a quantitative analysis of the precision grip, and with clinical rating scales. The location of the lesions within the posterior limb of the internal capsule (PLIC) in 18 patients was determined from neuroimages obtained in the acute stage (5–20 days after the insult). Involvement of the PLIC was assessed at the level of the basal ganglia, approximately 8 mm above the anterior commissure–posterior commissure level. The distance between the posterior edge of the internal capsule and the centre of gravity of the lesion was determined. Chronic disabilities affected dextrous movements, while paresis was mild and sensitivity for light touch or passive finger flexion was almost normal. For both the reaching-to-grasp movement and the precision grip paradigm, the slowness of movement or force development was confined to the phases when grip formation and stabilization occur, while the onset of hand transport and of the vertical lifting force were not delayed. Grip forces were increased. We observed a close correlation between posterior location within the PLIC and the altered measures of timing and precision grip force. The more posterior the acute lesion was located within the PLIC, the more pronounced were the chronic motor deficits, as seen both in the quantitative measures and in the rating scales. The present study demonstrates for the first time that the amount and quality of chronic motor deficits of dextrous movements are related to a simple measure drawn from routine neuroimaging in the acute stage in patients with capsular stroke. The poor motor outcome in lesions involving the most posterior parts of the PLIC could be due to the condensed organization of corticofugal projections and the density of pyramidal fibres from the primary motor cortex in this subsector. Even small infarcts of this strategic area can disrupt many of the projections from the motor cortices and could thereby limit recovery strategies between homolateral motor representations.

Keywords: stroke; motor recovery; hand function; kinematics; precision grip

Abbreviations: CUE = Capabilities of Upper Extremity Instrument; FIM = Functional Independence Measure; IC = internal capsule; PLIC = posterior limb of the internal capsule; RMA = Rivermead Motor Assessment

Introduction
Many stroke victims regain their abilities for activities of daily living but nevertheless have quantitative limitations, such as reduced speed and accuracy despite neurorehabilitation (Platz et al., 1999; Cirstea and Levin, 2000; Fisher et al., 2000). Clumsy and slow performance may be a considerable handicap to these patients, especially if fine manipulations are required. Individual finger movements are a prerequisite for dextrous motor acts and these recover the least following stroke-induced hemiparesis of the upper limb (Fugl-Meyer et al., 1975; Shelton and Reding, 2001; Kwakkel et al., 2002). Research on the factors involved in chronic functional disability of upper limb movements and dexterity would help to enhance treatments and to elucidate the pathophysiology of motor recovery after stroke.
How can we quantify the abnormalities underlying functional disability of the hand? Abilities such as grasping and lifting small objects in the precision grip require a complex pattern of motor acts. Therefore, impairments of dexterity require an analysis of motion and force production by object-pattern of motor acts. Therefore, impairments of dexterity lifting small objects in the precision grip require a complex attendance of control of hand functions to intact cortical areas (Chollet et al., 1997; Liepert et al., 2002). The time spent in the distinct movement phases, the path of movement and the precision of grip formation of forces in natural grasping is lacking.

The impact of neuroplasticity depends on the integrity of the corticofugal fibres. Converging evidence has been presented that lesions of the corticospinal tract can induce a shift of control of hand functions to intact cortical areas (Chollet et al., 1997; Weililer, 1995; Dettmers et al., 1997; Honda et al., 1997; Liepert et al., 1998; Marshall et al., 2000; Calautti and Baron, 2003). The corticofugal fibres from several motor cortices travel through the internal capsule (IC) to the motor neurons (Hardy et al., 1979; Fries et al., 1993). It was commonly assumed that hemiparesis following an isolated lesion of the IC (‘pure motor stroke’) has a good prognosis (Ferrand, 1902; Marie, 1981; Rascol et al., 1982), but this view has been challenged by later studies. Fries and his group observed poor recovery if the lesion extended both into the posterior limb of the internal capsule (PLIC) and into the lateral thalamus, while an excellent outcome was seen in patients with other locations within the IC or the basal ganglia (Fries et al., 1993). Shelton and colleagues found that motor recovery following strokes of different size and locations depended crucially on an intact PLIC (Shelton and Reding, 2001). Posterior lesions of the IC in primates may affect many of the efferent tracts from the primary motor cortex which reside posteriorly within the PLIC, as has been shown in primates (Fries et al., 1993; Morecraft et al., 2002). Also, pyramidal fibre density is greatest in the posterior subsector of the PLIC. It was suggested recently that the severity of motor deficits may increase as a lesion occupies more posterior regions of the IC (Morecraft et al., 2002). However, detailed quantitative analyses of the relation of functional motor performance with the location of the stroke within the PLIC are lacking. It was the aim of the present study to explore the relationship between structural damage of the PLIC and functional recovery using quantitative measures.

Subjects and experimental conditions

Eighteen adults (five women, 13 men, mean age 60.9 years, range 40–81 years) who matched all the following criteria were selected from the stroke database of the Department of Neurology, Kiel: (i) paresis of at least the upper limb that lasted a minimum of 2 days; (ii) a first-ever stroke, consisting of a single small ischaemic infarct with an axial diameter ≤2 cm that involved the internal capsule as visualized by imaging; (iii) no additional cerebrovascular accident since the index event; (iv) age between 20 and 80 years, no cortical or bilateral lesions, no evidence of generalized small-vessel disease, no bilateral paresis, no other neurological or musculoskeletal diseases affecting arm mobility, and no cognitive dysfunction of a severity that was incompatible with participation in movement analysis (i.e. severe behavioural disturbances or speech comprehension deficits).

Initially (time = T0), nine patients suffered from paresis of the right hand and nine of the left hand (Table 1). All were right-handed. In the chronic stage (T1 at a mean of 2.4 years after stroke) the patients had regained a high level of upper limb function, including the ability to grasp and lift light objects. Written informed consent was given by each participant for assessment in this study, which was approved by the Ethics Committee of the Kiel University Hospital. Eighteen healthy right-handed volunteers, matched for age and sex, served as control subjects in the neurophysiological

Table 1 Clinical data of the patients initially after stroke (T0) and at the time of neurophysiological assessment (T1)

| Age (years) | 60.9 ± 10.7 |
| Sex (M, F) | 5, 13 |
| Years after stroke | 2.4 ± 1.9 |
| Affected limb (R, L) | 9, 9 |
| Paresis at T0 (0–5) | 2.9 ± 1.1 |
| Paresis at T1 (0–5) | 4.4 ± 0.8 |
| Spasticity T1 (0–5) | 1.4 ± 1.5 |
| Hypo-aesthesia at T1 (0–2) | 0.39 ± 0.5 |
| Proprioception (0–2) | 0.06 ± 0.24 |
| RMA arm at T1 (0–15) | 10.6 ± 2.9 |
| CUE hand at T1 (0–28) | 19.2 ± 8.4 |
| FIM at T1 (0–91) | 84.6 ± 12 |

Scores of the affected side are shown for paresis (MRC grades 1–5/5), spasticity (range 0–5), hypo-aesthesia and proprioception (range 0–2). FIM is the Functional Independence Measure™; RMA arm depicts arm functions of the Rivermead Motor Assessment; CUE hand depicts the hand items of the Capabilities of Upper Extremity Instrument. Score ranges are in parentheses. Results are mean ± SD.
tests (mean age of the patients, 60.9 years versus 62 years for the controls; not significant, t test).

Imaging was performed at the subacute stage (T0), within the first 5 days after the onset of symptoms. Neuroimaging studies were reviewed by an investigator who was unaware of individuals' clinical data. Involvement of the PLIC was assessed at the level of the basal ganglia, ±8 mm above the anterior commissure-posterior commissure level (Talairach et al., 1967).

The location of the lesion was determined by the distance of its centre of gravity from the posterior border of the internal capsule, as shown in Fig. 1. The borders of the lesion and surrounding tissue were drawn from T2-weighted MRIs in 16 and from high-resolution CT images in two patients, as depicted in Fig. 2. Planimetry was performed on a graphic tablet (Summa Sketch II+; CTCO CalComp, Columbia, USA) using enlarged images at 1 : 1 scale.

All patients received a full neurological examination at the time of acute stroke (T0) and at the chronic stage before the neurophysiological assessment (T1). The MRC score (Medical Research Council, 1976) of paresis at T0 was taken from the medical records, while paresis of extension of the wrist at T1 was assessed prospectively on the same scale (0 = plegia to 5 = no paresis). Cutaneous and proprioceptive sensitivity at T1 was studied by touching the tips of index fingers with a cotton swab and by passive flexion of the distal index joint by the experimenter, respectively. It was graded as follows: 0 = normal without side difference; 1 = not abolished, but side difference; 2 = abolished. Spasticity due to passive extension of the wrist was scored at T1 on the five-point modified Ashworth scale (Bohannon and Smith, 1987). The motor subscore of the Functional Independence Measure (FIM™) (Heinemann et al., 1994) was used as a measure of independence from care. Functional disability of the upper extremity was assessed with the Rivermead Motor Assessment (RMA) (Lincoln and Leadbitter, 1979) performed by one of the researchers (R. W. or H. S.) and by a German translation of the Capabilities of Upper Extremity Instrument (CUE) (Marino et al., 1999) at T1. The validity of the RMA arm score for recovery of motor functions has been shown extensively (Adams et al., 1997; Jones, 1998; Scheidtmann et al., 2001). It scores a wide range of 15 exercises, from simple to complex and dexterous tasks, such as placing a string around the head and tying a bow at the back. The CUE consists of 17 items for assessment of self-perceived focal abilities of the upper extremity that had been validated for motor restitution after spinal cord injury (Marino et al., 1999). The sum of four items of the CUE related to dexterous hand functions was analysed in the present study: picking up a paper clip (item 13), using a key (item 14), turning a coin with thumb and fingers (item 16), and dialling a touch-tone telephone (item 17).

**Kinematic analysis of the reach-to-grasp movement**

The methods have been published in detail before (Deuschl et al., 2000; Wenzelburger et al., 2000) and are therefore described only briefly here. Subjects were seated on a comfortable chair with their back supported and the active hand lying on a table in front of them. The target consisted of a plug with a diameter of 1 cm that was fixed to a heavy support, and the subject was instructed to reach out and precisely grasp the plug with their thumb and index finger. The target was located 34 cm above the table in a parasagittal plane, at a comfortable distance of approximately 50 cm from the body to prevent substantial movements of the trunk.

The subjects placed their hands on the resting platform, then grasped the target after a beep signal, released it and went back to the resting position. The subjects were instructed to move at a comfortable velocity. All subjects performed at least five training trials until they were familiar with the task, followed by at least 10 test trials for off-line analysis.

Reflective markers with a diameter of 5 mm were placed on the radial surface of the following landmarks: tip of the thumb, tip of the index finger and at the epicondylus of the
A passive infrared movement analysis system (Mac Reflex 3.2; Qualisys, Sweden) was used to sample the movement paths as 3D Cartesian coordinates. Tangential movement velocity was calculated as the change in wrist position over time (dp/dt). A minimal grip aperture (i.e. distance between thumb and index markers) indicated the end of the target period (see below).

The movement time during acceleration (MT\textsubscript{ACC}) lasted from the time-point when the velocity of the wrist exceeded 0.05 m/s until peak velocity (Fig 3A). The subsequent time for deceleration (MT\textsubscript{DEC}) lasted until wrist velocity dropped below 0.05 m/s for the first time. Movement time in the subsequent target period, MT\textsubscript{TARG}, ended at the definite end of the grasping movement, which was determined at minimal grip aperture. During the target period, some short additional accelerations and decelerations of the hand were observed even in most of the healthy control subjects. They were considered to represent terminal corrective movements. The total movement time was the sum of MT\textsubscript{ACC}, MT\textsubscript{DEC} and MT\textsubscript{TARG}.

We defined the vertical overshoot as the difference between the highest positions of the wrist marker during the grasping movement minus the vertical marker position at the end of the target period. Mean values were calculated from all test trials.

**Measurement of the precision grip**

The experimental procedure for the analysis of grip force coordination was similar to that described previously (Johansson and Westling, 1984; Wenzelburger et al., 2002) and therefore is summarized here only briefly. All subjects washed their hands before the experiment and the table was positioned such that the forearm was parallel to the floor when the object was grasped between the thumb and index finger (Fig. 3B). Its weight was 220 g and the grip surfaces were covered with sandpaper. Horizontal grip forces and vertical load forces were measured from the thumb and index finger using 3D sensors (Assurance F/T, USA), and were digitized at 400 Hz using SC/ZOOM software (Umea, Sweden). Subjects were instructed to perform the task at a normal pace, i.e. no instructions were given regarding speed, accuracy or force. The hand was held open at the level of the object and the subject grasped and lifted the object at a beep. After a holding phase of 5 s, the subject was told to replace it at the table. Fifteen repetitions were recorded with a 5–10 s pause in between. The first five trials were regarded as practice trials and were not considered for data analysis.

We focused on temporal parameters indicating the times needed to establish a stable grip until lifting-off of the object. Temporal parameters were derived from the two early latencies, related to grasping (duration of grip preparation = DUR\textsubscript{GPREP} and duration of preload phase = DUR\textsubscript{LOAD}), and from the late phase, when the vertical lifting force increases (duration of load phase = DUR\textsubscript{LOAD}). These were the latencies between four discrete events: first contact of a finger, definite grip by thumb and index, onset of positive load force, and movement of the object (for details see Fig. 3B). The total duration (from first contact until object movement = DUR\textsubscript{TOTAL}) was computed additionally. Peak and static precision grip forces were also measured as described previously.

**Fig. 2** Location of the ischaemic lesion in the 18 patients. Each template was drawn schematically at the individual axial slice representing the internal capsule (IC) ~8 mm above the intercommissural line. Drawings of the lesion (black) and the internal capsule (grey) show their maximal visible extent. The distance between the centre of gravity of the infarction and the posterior border of the IC (D) is depicted for each patient. Patients with a lesion involving the most posterior aspects of the posterior limb of the internal capsule (PLIC) are shown first, followed by those with a lesion located more anteriorly within the PLIC. All infarcts are drawn on the right, although nine patients had a left-sided infarct. R indicates the side reversal in these patients. Gp = globus pallidus; Th = thalamus.
Spearman correlation coefficients were computed as:

\[ r_s = \frac{\sum (x_i - \bar{x})(y_i - \bar{y})}{\sqrt{\sum (x_i - \bar{x})^2 \sum (y_i - \bar{y})^2}} \]

Differences between groups were interpreted only after appropriate corrections for multiple comparisons. Nominal \( P \) values are shown in this exploratory study to show also subtle differences or relations.

Statistics
The affected side of the upper extremity was studied in the patients and analysis of the control subject was matched accordingly, each group having nine right sides and nine left sides analysed. Multivariate analysis of variance was used to compare patients and controls (GLM procedure of SPSS 11; SPSS, Chicago, IL, USA). \( P < 0.05 \) was assumed as significant in these multiple comparisons only if the multivariate \( F \) statistic also indicated a significant (\( P < 0.05 \)) difference between patients and controls over the total set of variables from each test (reach-to-grasp and grasp-to-lift). Differences between groups were interpreted only after verifying the homogeneity of variances by Levene’s test. Spearman correlation coefficients \( (r_s) \) were computed as appropriate. Nominal \( P \) values are shown in this exploratory study to show also subtle differences or relations.

Results
Clinical measures
Initial paresis at T0 ranged from severe to mild (MRC grades 1–4, mean 2.9/5) (Table 1). A substantial recovery of force was seen at the chronic stage (T1). Ten patients had regained full strength at the time of movement analysis. Five patients suffered from mild paresis (grade 4) and moderate paresis (grade 3) was seen in three patients. Mild to moderate spasticity was seen in 10 of the 18 patients (Ashworth grades 1–4), including all those with persistent paresis. Eight patients reported hypo-aesthesia for light touch at the index finger. All patients had reached almost complete independence from care, as shown by the FIM score (Table 1). The RMA arm score indicated residual disability of upper limb functions in all but two patients (patients 10 and 11, overall mean 10.6 out of a possible 15 points), although eight of the 18 patients reported residual focal disabilities (CUE hand mean, 19.2 of a possible 28 points) (Table 1).

Comparison between patients and controls
The total movement time when reaching to grasp the object was increased in the stroke patients compared with the healthy controls (MTTOTAL, +54%, \( P < 0.01 \)) (Table 2). Detailed analysis of the different phases of the movement, however, revealed distinct patterns (Figs 4 and 5). The time needed for acceleration was only minimally increased (MTACC, +17%, not significant), while the movement was slowed during deceleration and even longer in the target phase (MTDEC, +33%, \( P < 0.05 \); MTTARG, +132%, \( P < 0.01 \)). The time for acceleration and deceleration was longer than the target

### Table 2 Comparisons of variables concerning kinematics and force between stroke patients (at T1) and healthy controls (mean ± SD)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Stroke</th>
<th>Control</th>
<th>( F )</th>
</tr>
</thead>
<tbody>
<tr>
<td>MT_TOTAL</td>
<td>1681 ± 800**</td>
<td>1090 ± 77</td>
<td>10.2</td>
</tr>
<tr>
<td>MT_ACC</td>
<td>419 ± 144</td>
<td>359 ± 31</td>
<td>3.2</td>
</tr>
<tr>
<td>% MT_ACC</td>
<td>27 ± 7**</td>
<td>33 ± 4</td>
<td>11.4</td>
</tr>
<tr>
<td>MT_DEC</td>
<td>635 ± 283*</td>
<td>476 ± 66</td>
<td>5.7</td>
</tr>
<tr>
<td>% MT_DEC</td>
<td>38 ± 5**</td>
<td>44 ± 5</td>
<td>10.9</td>
</tr>
<tr>
<td>MT_TARG</td>
<td>634 ± 431**</td>
<td>273 ± 66</td>
<td>13</td>
</tr>
<tr>
<td>% MT_TARG</td>
<td>36 ± 8***</td>
<td>25 ± 5</td>
<td>24.6</td>
</tr>
<tr>
<td>Overshoot</td>
<td>9.06 ± 6.66**</td>
<td>4.29 ± 2.12</td>
<td>8.8</td>
</tr>
<tr>
<td>DUR_TOTAL</td>
<td>711 ± 437*</td>
<td>232 ± 76</td>
<td>5.4</td>
</tr>
<tr>
<td>DUR_GPREP</td>
<td>275 ± 172**</td>
<td>49 ± 31</td>
<td>7.9</td>
</tr>
<tr>
<td>% DUR_GPREP</td>
<td>35 ± 11**</td>
<td>20 ± 9</td>
<td>10.1</td>
</tr>
<tr>
<td>DUR_LOAD</td>
<td>284 ± 169***</td>
<td>64 ± 27</td>
<td>12.2</td>
</tr>
<tr>
<td>% DUR_LOAD</td>
<td>45 ± 13**</td>
<td>27 ± 8</td>
<td>10.7</td>
</tr>
<tr>
<td>DUR_TOTAL</td>
<td>152 ± 185</td>
<td>119 ± 34</td>
<td>0.2</td>
</tr>
<tr>
<td>DUR_TARG</td>
<td>21 ± 8***</td>
<td>54 ± 12</td>
<td>68.1</td>
</tr>
<tr>
<td>GF_PEAK</td>
<td>7.35 ± 2.89*</td>
<td>5.05 ± 0.24</td>
<td>5.1</td>
</tr>
<tr>
<td>GF_STATIC</td>
<td>5.85 ± 2.1**</td>
<td>3.72 ± 0.93</td>
<td>7.4</td>
</tr>
</tbody>
</table>

All parameters except acceleration time (MT\_ACC) and load phase duration (DUR\_LOAD) differed significantly between groups. The percentage values refer to the respective fractions of MTTOTAL and DUR\_TOTAL. Movement times (MT) and durations of phases (DUR) are shown in milliseconds. Vertical overshooting of the wrist was measured in millimetres. Grip force (GF) is given in newtons. *** \( P < 0.001 \); ** \( P < 0.01 \); * \( P < 0.05 \) compared with control. The \( F \) statistics indicate the strength of differences between groups.
phase in the controls but this pattern was reversed in the patients (Fig. 6). A pathological overshooting in the sagittal plane of the wrist approaching the target was also seen in the patients (+111%, \(P < 0.01\)). Therefore, the abnormalities that were most prominent in the late periods of reaching to grasp were grip formation and terminal corrections.

The analysis of grasping to lift exhibited a similar abnormality. The total time from grasping the object in the precision grip until lift-off was markedly slowed (DUR\(_{\text{TOTAL}}\), +206%, \(P < 0.05\) compared with controls). The first two phases were clearly slowed (DUR\(_{\text{GPREP}}\), +461%, \(P < 0.01\); DUR\(_{\text{PLoad}}\), +343%, \(P < 0.001\) compared with controls) (Figs 4 and 5), whereas the duration of the last phase was only mildly prolonged (DUR\(_{\text{LOAD}}\), +28%, not significant). This indicated a slowness of grasping until the stable grasp was achieved, while the development of vertical lifting force seemed not to be disturbed. The time spent for the different phases shifted from DUR\(_{\text{LOAD}}\) in controls to DUR\(_{\text{GPREP}}\) and DUR\(_{\text{PLoad}}\) in the patients (Fig. 6).

A moderate increase in precision grip force was also seen in patients, both at peak grip force and in the static holding phase (GF\(_{\text{PEAK}}\), +46%, \(P < 0.05\); GF\(_{\text{STATIC}}\), +57%, \(P < 0.01\) compared with controls).

**Correlations between clinical and kinematic measures and morphology**

A posterior location of the lesion within the IC had a strong impact on performance in both analyses, as exemplified in two patients in Fig. 6. Patients with a posterior location of the lesion within the PLIC showed a terminal slowing of the terminal phases of reaching to grasp, with a segmentation of the movement, although the acceleration time was not slowed consistently. The coordination of forces in the grasping-to-lift paradigm was irregular in these patients, with a large variance between trials. Patients with lesions extending into the region of the genu while sparing the posterior subsector of the PLIC behaved almost like the controls. The correlation analysis showed that initial and chronic paresis depended strongly on the posterior location (\(r_S = 0.79, P < 0.0001\) at T0; \(r_S = 0.86\) at T1, \(P < 0.00001\)) (Fig. 7 and Table 3). A posterior location was also related (\(r_S = 0.62, P < 0.01\)) to a minor reduction in paresis (\(\Delta\) paresis, T1 – T0) (Table 3). Chronic spasticity (T1) was related moderately to posterior location (\(r_S = 0.62, P < 0.01\)). Hypo-aesthesia at T1 was not correlated significantly with posterior location or measures from the movement analysis. This was not due only to the three-step score because mild disturbances of cutaneous sensitivity were seen equally in patients with more posterior or anterior lesions of the IC (patients 4, 6 or 9, 15 and 16). Mild proprioceptive deficits were seen only in one patient (patient 4), which precluded correlation analysis. Focal disability at the chronic stage (T1) was closely related to a posterior location. This was true for the ratings of both expert and patient [RMA arm, \(r_S = 0.76, P < 0.01\) (Fig. 7); CUE hand,
Ten patients had reached full functional independence at the chronic stage (FIM motor score), unlike those with a lesion extending into the posterior border of the PLIC. We observed a moderate correlation between FIM and posterior location \( (r_S = 0.6, P < 0.01) \).

The correlations between timing of the reach-to-grasp movement and posterior location indicated a distinct pattern. The final phases of the movement were strongly correlated with posterior location, while the acceleration phase was not \( [MT_{DEC}, r_S = -0.71, P < 0.01; MT_{TARG}, r_S = -0.83, P < 0.0001 \text{ (Fig. 7)}; MT_{ACC}, r_S = -0.36, \text{ not significant}] \) (Table 3). This indicated that chronic slowness of the terminal grasping period was mainly seen in the patients with posterior IC lesions, while these patients did not exhibit a slowness of hand acceleration.

A similar pattern was seen for the timing of forces in the precision grip paradigm. The duration of the first two phases in which the grasp was completed were correlated very closely with the index of posterior location, while the latest phase in which the vertical loading force develops was correlated moderately with posterior location \( [DUR_{GPREP}, r_S = -0.91, P < 0.000001 \text{ (Fig. 7)}; DUR_{LOAD}, r_S = -0.77, P < 0.001] \). Posterior location was correlated with precision grip force, both at its peak and in the static holding phase \( (GF_{PEAK}, r_S = -0.84; GF_{STATIC}, r_S = -0.74; P < 0.001 \text{ in both}) \).

**Discussion**

**Pattern of chronic motor deficits in capsular stroke**

Recovery of functional motor independence was almost complete at the chronic stage, whereas focal disabilities were still remarkable. Disabilities involved movements with rapid changes of direction or dextrous movements, such as...
pronation/supination of the wrist, tying a knot or turning a coin with the fingers. Such abilities were disturbed in many of the patients despite minor residual paresis. The quantitative analysis revealed distinct patterns for the timing of reach-to-grasp movements and for the calibration and timing of forces during precise grasping to lift. Acceleration of the reaching hand was smooth and not prolonged, whereas deceleration and, moreover, the terminal grasping phases were clearly altered. Those phases were slowed selectively and the trajectories were often hypermetric, the hand approaching the target from above. The early phases of grasping to lift were slowed, whereas the timing of lifting force was regular. Therefore, slowness was confined to the phases in which grip formation and stabilization occur, while the onsets of hand transport and

**Fig. 7** Regression of lesion location with pareses of the contralesional hand in the acute and chronic states, the arm section of the Rivermead Motor Assessment (RMA arm) and with quantitative measures of the contralesional hand. These were movement time in target phase ($MT_{TARG}$), duration of grip preparation ($DUR_{GPREP}$) and peak grip force ($GF_{PEAK}$). The distance of the centre of gravity of the lesion from the posterior border of the PLIC is depicted in millimetres. Note that patients with posterior lesions had incomplete recovery of paresis, were more disabled (lower RMA arm), needed longer for grasping ($MT_{TARG}$, $DUR_{GPREP}$), and applied higher precision grip force ($GF_{PEAK}$). The Spearman correlation coefficients $r_s$ and corresponding $P$ values are shown.
the vertical lifting force were not delayed. Grip forces were increased in the patients.

This is the first study on the relation between circumscribed stroke-related damage of the IC and upper limb function. Fries and colleagues reported that lesions of various sizes involving the posterior capsule induce severe hemiplegia initially, followed by almost complete functional restitution at the chronic stage (Fries et al., 1993). Kunesch and colleagues, however, described poor recovery of force and dextrous functions 6 weeks after reaching (MT), phase durations during grasping (DUR) and grip forces (GF) were all related to a posterior location of the capsular lesion (distance between the centre of gravity of the lesion and posterior border of PLIC), whereas hypo-aesthesis and vertical overshoot were not correlated with location. Neurophysiological assessment was performed at T1 only.

Most of the quantitative studies in stroke have assessed aiming or reaching in heterogeneous groups of patients with lesions of various sizes and locations within the territory of the middle cerebral artery. Overall, these studies have found a decreased velocity of the arm (Wing et al., 1990; Trombly, 1993; Roby-Brami et al., 1997; Archambault et al., 1999), increased movement times for acceleration and deceleration (Platz et al., 2001), increased path length (Levin, 1996; Cirstea et al., 2003) as well as segmentation of the movement (Trombly, 1993; Roby-Brami et al., 1997; Archambault et al., 1999; Krebs et al., 1999). The present results demonstrate that small infarcts of the PLIC do not cause general slowness at the chronic stage, but disturb the terminal phases of the reach-to-grasp movement and the initial phases of the grip-to-lift task almost selectively. These are the phases in which formation and stabilization of the grip occur (Jeannerod, 1984, 1986; Johansson and Westling, 1984). Timing of hand transport and onset of the lifting force was almost normal, suggesting a minor disturbance of hand transport. A chronic disturbance of precision grip functions by capsular lesions was further underlined by the results from grip force analysis.

Forces produced by the thumb and index finger during the lifting of an object in the precision grip show a pathological exaggeration and slowed onset in some types of cerebellar lesions (Muller and Dichgans, 1994; Fellows et al., 2001). Recently, Hermdsorfer and colleagues studied the coordination of precision grip forces in a group of patients at the chronic stage with partly extensive and multifocal cerebral stroke (Hermdsorfer et al., 2003). Our findings are consistent with this study and additionally show that similar abnormalities also occur in small infarcts of the PLIC.

**Relation between abnormalities of movement and the location of the lesion**

The present study shows that the amount and quality of chronic fine motor deficits are related to a simple measure drawn from routine neuroimaging of small capsular infarction in the acute state. The internal capsule containing the corticospinal tract is visible on MRI and CT scans (Rascol et al., 1982; Fries et al., 1993; Kunesch et al., 1995; Binkofski et al., 1996; Miyai et al., 1997; Shelton and Reding, 2001). The location of the lesions was determined with reference to the midsagittal plane, the thalamus, the basal ganglia and the visible borders of the IC. The more posterior the lesion was located within the PLIC the more severe were the residual chronic impairments and disabilities. This relation was seen both for clinical scores and for instrumented measures of functional motor tasks. Chronic paresis was only seen after lesions extending into the posterior 6 mm of the PLIC. Strong correlations were observed between a posterior location of the lesion within the IC and the time until a stable grasp was achieved, whereas posterior location was not related to the transport of the reaching hand and to the preparation for lifting the object. Moreover, a posterior location was correlated with exaggeration of the precision grip force. Such

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### Table 3 Correlation of posterior location of the lesions with the variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Correlation coefficient ($r_s$)</th>
<th>Nominal $P$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paresis at T0</td>
<td>0.791</td>
<td>0.00009</td>
</tr>
<tr>
<td>Paresis at T1</td>
<td>0.864</td>
<td>0.000004</td>
</tr>
<tr>
<td>Δ paresis T1 – T0</td>
<td>−0.624</td>
<td>0.006</td>
</tr>
<tr>
<td>Spasticity at T1</td>
<td>0.621</td>
<td>0.006</td>
</tr>
<tr>
<td>Hypo-aesthesis at T1</td>
<td>−0.09</td>
<td>0.73</td>
</tr>
<tr>
<td>RMA arm at T1</td>
<td>0.758</td>
<td>0.0003</td>
</tr>
<tr>
<td>CUE hand T1</td>
<td>0.681</td>
<td>0.002</td>
</tr>
<tr>
<td>FIM</td>
<td>0.595</td>
<td>0.009</td>
</tr>
<tr>
<td>MTACC</td>
<td>−0.36</td>
<td>0.14</td>
</tr>
<tr>
<td>MTDEC</td>
<td>−0.71</td>
<td>0.001</td>
</tr>
<tr>
<td>MTARG</td>
<td>−0.83</td>
<td>0.000002</td>
</tr>
<tr>
<td>Overshoot</td>
<td>0.06</td>
<td>0.98</td>
</tr>
<tr>
<td>DUR(grip)</td>
<td>−0.91</td>
<td>0.000001</td>
</tr>
<tr>
<td>DUR(LOAD)</td>
<td>−0.875</td>
<td>0.000002</td>
</tr>
<tr>
<td>DURgrip</td>
<td>−0.771</td>
<td>0.0002</td>
</tr>
<tr>
<td>GFpeak</td>
<td>−0.84</td>
<td>0.00001</td>
</tr>
<tr>
<td>GFstatic</td>
<td>−0.735</td>
<td>0.0005</td>
</tr>
</tbody>
</table>

Clinical scores (initial paresis at T0, chronic paresis at T1, Δ paresis T1 – T0, RMA arm at T1, CUE hand at T1, FIM at T1), movement times for reaching (MT), phase durations during grasping (DUR) and grip forces (GF) were all related to a posterior location of the capsular lesion (distance between the centre of gravity of the lesion and posterior border of PLIC), whereas hypo-aesthesis and vertical overshoot were not correlated with location. Neurophysiological assessment was performed at T1 only.
posterior location within the PLIC would correlate with the location of the M1 pathway, as identified in the human (Ross, 1980) and monkey (Fries et al., 1993; Morecraft et al., 2002). The crucial role of the posterior subsector of the PLIC for dextrous functions holds true even if we suppose limited precision of the imaging in the subacute stage and the heterogeneous vertical extent of the lesions, because an almost linear relation was seen between location and dysfunction.

The present data fully support the hypothesis of Morecraft and colleagues that ‘the severity of motor deficit is likely to increase as a lesion occupies progressively more posterior regions of the internal capsule’ (Morecraft et al., 2002). Posterior lesions of the PLIC affect the strongest and densest projections of the primary motor cortex, which gives rise to 35–49% of the corticospinal projections (Dum and Strick, 1991; Galea and Darian-Smith, 1994). In contrast, the projection from the rostral cingulate motor cortex, which gives rise to about 5% of the corticospinal projection, resides in the anterior part of the PLIC. Our findings underline the assumption that integrity of the pyramidal tract is vital for recovery of fine motor functions of the upper limb, particularly its strongest projection arising from the primary motor cortex, which resides in the posterior subsector of the PLIC (Fries et al., 1993; Morecraft et al., 2002). A crucial role of the PLIC could be due to the condensed organization of segregated projections from six cortical motor representations of the contralateral upper limb, which has recently been shown in detail in primates. These are corticofugal tracts (posterior locations first) from the primary motor cortex, caudal cingulate motor cortex, dorsal lateral prefrontal cortex, ventral lateral premotor cortex, supplementary motor area and rostral cingulate motor cortex, which all converge in the PLIC (Morecraft et al., 2002). A lesion with a diameter of less than 1 cm could destroy several of these fibre systems and thereby limit the potential for motor recovery of the contralateral upper limb.

Conclusion

The present study shows that the more posterior a lesion is located in the PLIC, the more severe are the residual motor deficits measured with quantitative behavioural methods. As has been predicted from experiments in animals and humans, the crucial factor for motor recovery was a sparing of the most posterior parts of the PLIC, which contains dense corticofugal projections from the primary motor cortex but also from the cingulate motor cortex and the lateral prefrontal cortices. An extension of the lesion into this structure, as detectable on routine imaging in the acute state, may indicate prolonged or limited recovery of fine motor functions. This study underlines that patients with even small lesions involving the PLIC may suffer from prolonged disability of dextrous upper limb functions, which is masked by an overall satisfactory recovery of the activities of daily living. Further therapeutic studies with specific training for dextrous movements are required to determine the potential for recovery in such patients.

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