Chapter 2

Cerebral reorganisation of human hand movement following dynamic immobilisation

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Abstract

Surgical treatment of a flexor tendon lesion of the hand is followed by a 6-week period of dynamic immobilisation. This is achieved by the elastic strings of a Kleinert splint, enabling only passive and no active flexor movements. After such immobilisation, the appearance of a temporary clumsy hand indicates decreased efficiency of cerebral motor control. Using PET we identified the recruitment of contralateral parietal and cingulate activations specifically related to the suboptimal character of these hand movements. After 6–8 weeks, normalised movement was related with contralateral putamen activation. Activations of the sensorimotor cortex and cerebellum were present during both scanning sessions. Changes in the pattern of cerebral activations reflect functional reorganisation. The shift from cortical to striatal involvement, observed in the group of four patients, generates the concept of unlearned movements being relearned.
Introduction

Accidental injury from a knife often affects the volar side of the non-dominant hand. Surgical treatment of an associated digital flexor tendon lesion is followed by a 6-week period of dynamic immobilisation. The latter is achieved by elastic strings that connect the tips of all fingers with the volar side of the wrist (Kleinert splint), thus enabling active extension movements followed by passive flexor movements. All digits are included in order to avoid dehiscence of the sutured tendon by traction due to synergistic flexor movement. Passive movement is encouraged in order to avoid synovial adhesion and joint fixation. After splint removal, however, patients report initial clumsiness in task performance which is not explained by stiffness of the fingers or adhesions. This may last from days to weeks, and suggests the loss of efficient cerebral control of flexor movements due to selective disuse. In order to test this hypothesis, we used PET to detect changes in the cerebral organisation of hand movement induced by a period of functional immobilisation.

Subjects and methods

Four right-handed patients treated for a left-hand flexor tendon lesion were studied with PET. They gave informed consent and the studies were approved by the Medical Ethics Committee of the University Hospital Groningen. Each patient underwent two series of PET scans (Siemens ECAT HR+ scanner operated in 3D mode, 15.2 cm axial field of view). Task-related increases of regional cerebral bloodflow (rCBF) were used as indicators for local neuronal activations and measured with H$_2^{15}$O-labelled water. The first series of scans (study 1) was performed immediately after removal of the wrist-band used for dynamic immobilisation. A subsequent study followed 6–8 weeks later. In this interval, all movements were allowed, although lifting weight was initially restricted. An increase of using muscle strength was gradually allowed with physiotherapeutic guidance. In both studies, movement-related activations were identified by comparing six scans acquired during a left-hand movement condition with three scans made in rest. Each scan lasted 90 s, during which the patients listened to randomly presented beeps (20/min). In the movement condition they responded to each beep by making two flexion movements with the fingers of the treated hand (digits 2–4, thumb excluded). The wrist was neutrally positioned and supported by an extension of the scanner table. The volar side of the hand faced the floor. In the control condition, patients listened to the beeps only. Within each study, intervals between the nine scans were 10 min. One of two conditions was assigned to each scan, the overall sequence being ordered rest, three times movement, rest, three times movement and finally rest again. Statistical parametric mapping (SPM99) was used for image
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realignment, transformation into standard stereotactic space (template of the Montreal Neurological Institute), smoothing (10 mm FWHM) and statistical analysis\textsuperscript{5,6}. For each of the two studies, significant activations that resulted from contrasting the movement and control conditions are reported (thresholded at $p=0.05$, false detection rate corrected for whole brain volume).

Surface electromyography (EMG) was performed on a fifth patient who did not participate in PET. In two studies with a 6-week interval, the same stimulus protocol was applied as for PET. Recordings were made from both the digital flexor and the extensor muscles of the lesioned hand's forearm. The hand was positioned similar to the position in the PET protocol.

**Results**

The four patients who participated in the PET study indeed reported increased clumsiness when released from the wrist band. During scanning in study 1, they valued the feeling of decreased skillfulness in performing the instructed movements as, respectively, 40, 25, 50 and 50 (on a scale of 0–100, representing insufficient to normal). In study 2, their scores were 95, 90, 80 and 80. Visual monitoring of performance during scanning revealed that all patients were able to accomplish the task as instructed, although the two separate movements made in response to each beep were generally less brisk in study 1.

Surface EMG performed on the fifth patient, who did not participate in PET, showed a normal pattern of digital extensor muscle contractions of the lesioned hand's forearm in both studies. Surface recording during contraction of the flexor muscles, however, particularly showed increased extensor co-contraction in study 1. No full relaxation was seen in between the two movement responses made to each beep. This pattern had normalised in study 2. Group analysis of rCBF changes showed that the left-hand movement condition in the first as well as the second study was related with activation of the contralateral sensorimotor cortex and ipsilateral cerebellum (Fig. 2.1, see Appendix, regions 6 and 7). In the initial study, additional activations (Table 2.1) were present in the posterior parietal cortex (Fig. 2.1, see Appendix, region 1) and deep in the caudal part of the cingulate sulcus (Fig. 2.1, see Appendix, region 2), both in the right hemisphere. In the second study, performed 6–8 weeks later, additional activations related to the left hand movement condition (Table 2.1) were present in the contralateral putamen (Fig. 2.1, see Appendix, region 3) and posterior insula (Fig. 2.1, see Appendix, region 4). The movement-related activation at a more lateral position along the lateral fissure (Fig. 2.1, see Appendix, region 5) was not regarded to be specific for study 2. The plotted contrasts (Fig. 2.1,
see Appendix, diagram 5) illustrate that a similar effect was indeed present in study 1, but failed to reach statistical significance.

Table 2.1 Study-specific activations related to left hand flexor movement

<table>
<thead>
<tr>
<th>Brain region</th>
<th>Stereotactic coordinates</th>
<th>Z-score</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>x,y,z</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right parietal cortex</td>
<td>30,-56,66</td>
<td>4.43</td>
<td>0.002</td>
</tr>
<tr>
<td>Right cingulated sulcus</td>
<td>14,2,40</td>
<td>4.19</td>
<td>0.003</td>
</tr>
<tr>
<td>Study 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right putamen</td>
<td>26,0,0</td>
<td>3.98</td>
<td>0.004</td>
</tr>
<tr>
<td>Right posterior insula</td>
<td>44,-12,6</td>
<td>3.76</td>
<td>0.008</td>
</tr>
</tbody>
</table>

Localisation of rCBF increases by SPM (group of four subjects; p < 0.05, FDR corrected for the whole brain volume), comparing the six movement conditions with the three control condition in each study. These study-specific activations were in addition to the activation of the right sensorimotor cortex and left cerebellum, present in both sessions. Activation along the lateral fissure (58,-18,16) is not presented because it was not regarded study-specific (see Fig. 2.1b, see Appendix, diagram 5). Coordinates are given in mm. Positive x, y and z coordinates indicate locations respectively right, anterior and superior of the middle of the anterior commissure.

**Discussion**

The four patients who underwent PET were able to make the instructed flexor movements in the two studies. This performance, however, was suboptimal in study 1, although the hand was intact again. We explain this phenomenon by a change in cerebral motor control, induced by a period in which the patients do not actively command flexor movements. EMG demonstrated that, indeed, this temporary inefficiency concerned only the flexor movements. More co-contractions were made; extensor movements were not affected, which illustrates that joint movement had remained intact. Moreover, it indicates that the execution of active extensor movements during dynamic immobilisation prevented the underlying cerebral control from deteriorating.

The parietal activation, that was related to flexor movement in study 1 suggests an increased demand on a body scheme representation needed for instructing the appropriate parts of the hand to move\textsuperscript{7,8}. In this respect, one may further consider that task-related hand movement such as grasping is effectuated by the tuning of particularly flexor movements to the shape of a target (after opening the hand). This may suggest an intimate relation between parietal motor function
and particularly flexor movement\(^9,10\). The cingulate activation in study 1 was deep in the cingulate sulcus, around the vertical traversing the anterior commissure. This location has been labelled the caudal cingulate zone\(^11\), and points at the recruitment of a secondary motor function for the execution of simple hand movement\(^11,12\).

Only in study 2 was the series of double flexor responses related with activation of the contralateral putamen. Such an effect was fully absent in study 1 and suggests that simple movements have been relearned in comparison to the first study\(^13\). The association of reduced co-contraction and putamen activation in study 2 is consistent with the previously described role of the basal ganglia to switch off maintained motor activities that would otherwise interfere with voluntary movement commands\(^14\). The execution of relearned movement thus implies the improved selection of specific muscles to be used. Movement rate was identical in the two scanning sessions. Although reaction times and movement amplitudes were not quantified, we do not regard possible changes in these parameters crucial for explaining the differences in activations.

Given the representations of somatosensory and auditory modalities in the posterior insula\(^15\), increased activation related to the movement responses in study 2 may reflect enhanced efficiency of the related stimulus response associations\(^16\)]. A role of the insula in commanding hand movement\(^12,17\) is consistent with this explanation. At the more lateral position along the lateral fissure, possibly including the second somatosensory cortex SII of the parietal operculum\(^16\), increased activation in study 2 was not movement specific compared with study 1. The rCBF response in the control condition with only listening to the auditory signals was also larger. Whether this indicates that the cues gained a meaning associated with movement remains speculative.

**Conclusion**

A 6-week period of functional flexor immobilisation appears to induce a temporary loss of efficient cerebral control of hand movement, characterised by an increased cortical demand and reduced striatal involvement. The present observation demonstrates the impact of a relatively short period of immobilisation on the functional organisation of the brain.
References


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