Does ERD correlate with the strength of impairment after stroke?

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Introduction

Motor impairment after stroke is related to altered brain activation, like reduced activation of ipsilesional motor cortical areas [1]. A change of these alterations back to normal is related to recovery of motor function [2]. Aim of this study was to investigate if this relationship between motor impairment and brain activation is reflected in the event-related desynchronization (ERD) of motor cortical areas.

Methods

EEG was recorded from 21 positions covering motor cortex (see Figure 1) in 7 subacute subcortical stroke patients with left hand hemiparesis (see Table 1).

Table 1: Description of the sample with scores of European Stroke Scale (ESS), Medical Research Council for upper limb (MRC) and Modified Ashworth Scale for Spasticity for upper limb (MAS).

<table>
<thead>
<tr>
<th>Code</th>
<th>Year of birth</th>
<th>Lesion site</th>
<th>Months since event</th>
<th>ESS</th>
<th>MRC</th>
<th>MAS</th>
</tr>
</thead>
<tbody>
<tr>
<td>P01</td>
<td>1964</td>
<td>MCA occlusion</td>
<td>3</td>
<td>63</td>
<td>54</td>
<td>5</td>
</tr>
<tr>
<td>P02</td>
<td>1946</td>
<td>Temporal &amp; basal ganglia hemorrhage</td>
<td>3</td>
<td>63</td>
<td>54</td>
<td>5</td>
</tr>
<tr>
<td>P03</td>
<td>1930</td>
<td>Nucleo-capsular ischemia</td>
<td>6</td>
<td>83</td>
<td>77</td>
<td>0</td>
</tr>
<tr>
<td>P04</td>
<td>1950</td>
<td>Nucleo-capsular, temporal lobe ischemia</td>
<td>16</td>
<td>74</td>
<td>46</td>
<td>5</td>
</tr>
<tr>
<td>P05</td>
<td>1956</td>
<td>MCA thrombosis with ischemia</td>
<td>1.5</td>
<td>98</td>
<td>78</td>
<td>0</td>
</tr>
<tr>
<td>P06</td>
<td>1940</td>
<td>MCA thrombosis with ischemia</td>
<td>1.3</td>
<td>96</td>
<td>78</td>
<td>0</td>
</tr>
<tr>
<td>P07</td>
<td>1923</td>
<td>Centrum semiovale and corona radiata</td>
<td>2</td>
<td>87</td>
<td>72</td>
<td>1</td>
</tr>
</tbody>
</table>

Figure 1: Recorded electrode positions and regions of interest (ROI) used for correlation.

The participant’s task was executing cue-guided grasping and extension of the affected/unaffected hand (ME) for 4s (60 trials each hand). Figure 2 shows the timeline of a trial.

ERD was calculated referenced to the intertrial interval (ITI 1.5s, where patients stayed at rest) and averaged over 6 regions of interest (ROI, see Figure 1). Stroke impairment was measured by European Stroke Scale (ESS), Medical Research Council Scale for muscle strength (upper limb, MRC) and Modified Ashworth Scale for spasticity (upper limb, MAS). These measures were then correlated with ERD values of the ROI.

Results

Figure 4 shows the exemplary time-frequency maps of a less and a more impaired patient. ERD is represented in orange coloured dots. For the less impaired patient a clearly stronger ERD can be found in the region around Cz and C4 compared to the more impaired patient.

Figure 3: Exemplary time frequency maps for motor execution (ME) of the affected hand of one less (see Table 1 P06) and one more impaired patient (see Table 1 P01). Orange coloured dots represent event-related desynchronization (ERD), blue coloured dots represent even-related synchronization (ERS).

For ME of the affected hand significant correlations between MRC, MAS and right centroparietal ERD were found between 8 and 20Hz. The correlation between ERD and MRC was highly negative ($r = -0.76 - -0.90$; higher MRC values/less motor impairment $= $ lower ERD values/stronger ERD). The correlation between ERD and MAS was highly positive ($r = 0.77 - 0.95$; higher MAS values/stronger spastic $= $ higher ERD values/weaker or no ERD).

Discussion

The results are in line with previous fMRI findings and show that higher motor impairment after stroke correlates with reduced ipsilesional brain activation.

References


Acknowledgments

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