High-Resolution EEG in Poststroke Hemiparesis Can Identify Ipsilateral Generators During Motor Tasks

Joseph B. Green, MD; Yolanda Bialy; Elena Sora, MS; Anthony Ricamato, MSEE

Background and Purpose—Multimodal neuroimaging with positron emission tomography (PET) scanning or functional MRI can detect and display functional reorganization of the brain’s motor control in poststroke hemiplegia. We undertook a study to determine whether the new modality of 128-electrode high-resolution EEG, coregistered with MRI, could detect changes in cortical motor control in patients after hemiplegic stroke.

Methods—We recorded movement-related cortical potentials with left and right finger movements in 10 patients with varying degrees of recovery after hemiplegic stroke. All patients were male, and time since stroke varied from 6 to 144 months. All patients were right-handed. There was also a comparison group of 20 normal control subjects.

Results—Five of 8 patients with left hemiparesis had evidence of ipsilateral motor control of finger movements. There were only 2 cases of right hemiparesis; in addition, 1 patient had a posteriorly displaced motor potential originating behind a large left frontal infarct (rim).

Conclusions—Reorganization of motor control takes place after stroke and may involve the ipsilateral or contralateral cortex, depending on the size and site of the brain lesion and theoretically, the somatotopic organization of the residual pyramidal tracts. Our results are in good agreement with PET and functional MRI studies in the current literature. High-resolution EEG coregistered with MRI is a noninvasive imaging technique capable of displaying cortical motor reorganization. (Stroke. 1999;30:2659-2665.)

Key Words: electroencephalography ■ hemiplegia ■ image processing, computer assisted ■ rehabilitation

Recovery that continues beyond 3 or 4 weeks after a stroke has been attributed to neuroplasticity, a reorganization of the brain in which functions previously performed by the ischemic area appear to be assumed by other ipsilateral or contralateral brain areas. Neuroplasticity has been variously attributed to redundancy (parallel distributed pathways), changes in synaptic strength, axonal sprouting with formation of new synapses, assumption of function by contralateral homologous cortex, and substitution of uncrossed pathways.1,2 Transcranial magnetic stimulation,3 positron emission tomography (PET),4 and functional MRI (fMRI)5 have been successfully applied to demonstrate cortical reorganization after hemiplegia. We applied the new modality of 128-electrode high-resolution electroencephalography (hr-EEG)6 to detect reorganization of cortical motor control after spinal cord injury.7,8 We now report that hr-EEG recording of movement-related cortical potentials (MRCPs) can also reveal cortical motor reorganization after recovery from hemiplegic stroke. We evaluated the motor potential (MP) component of the MRCPs in the present study. Direct subdural recordings in humans have shown that the MP has maximal amplitude over the contralateral somatosensory cortex.9 The MP has been interpreted as the cortical activation of the common pathway, the pyramidal tract.10 The recording of the MP may provide a useful tool for the localization of cortical motor control.

Subjects and Methods
We studied 10 outpatients and 20 control subjects. All were male, and the average age was 59.4 years (range 50 to 71 years) (see Table). None were acutely ill. Eight had a right hemiparesis and 2 a left hemiparesis. Average time after stroke when studied was 43.4 months (range 6 to 144 months). Seven patients had diabetes mellitus, and 3 were hypertensive. One patient had a capsular lesion, 2 had infarcts confined to the right or left cerebral cortex, 1 had bilateral brain stem and cortical lesions, 1 had frontal and pontine infarcts, and 5 had involvement of cortex and basal ganglia. All patients received rehabilitation therapy, and all showed some degree of recovery (Table). None were noted to have mirror movements. The Institutional Human Use Committees approved the study, and all subjects gave informed consent.

Data Acquisition and Analysis
MRCPs were recorded in the EEG with movements of the index or middle fingers, and the MP component was selected for mapping and dipole source localization studies. There is agreement that MRCPs11 consist of (1) the Bereitschaft potential, with onset 1 to 1.5 seconds before self-paced movement, followed by (2) a steep negative slope ~500 ms before movement, then (3) a brief premotor positivity at 50 ms before movement onset, and finally (4) a sharply rising MP, which may begin shortly before movement. MP latencies are measured from movement onset to peak negativity. The MP peak is

Received July 23, 1999; final revision received September 20, 1999; accepted September 20, 1999.

From the Edward Hines Jr Veterans Affairs Hospital (J.B.G., Y.B., E.S., A.R.); the Memphis Veterans Affairs Medical Center (J.B.G.); and the Department of Neurology, Loyola University-Stritch School of Medicine, Chicago, Ill (J.B.G.).

Correspondence to Joseph B. Green, MD, Veterans Affairs Medical Center, 1030 Jefferson Ave, Memphis, TN 38104.

© 1999 American Heart Association, Inc.

Stroke is available at http://www.strokeaha.org

2659
the highest negativity reached in the scalp-recording motor cortex. The MP component was selected for mapping and dipole source localization studies because it is recorded over the hemisphere contralateral to the finger movement and generated mainly in the primary motor cortex (M1).11,12

An electromagnetic digitizer (Polhemus) was used to sample the surface of the head and the electrode positions on the scalp to establish the accurate location of electrode coordinates in 3-dimensional space. Five thousand to 7000 points were obtained or separation of the skin, skull, and brain surfaces from the MR image. We modeled these compartments using the boundary element method by assigning different appropriate conductivity values for each surface (eg, skin and skull). This allowed accurate localization of cortical activity by restricting the model to neurophysiologically accurate volume conductor geometry to the individual anatomy. The shape of the optimum volume-conductor model was determined by segmentation algorithms (eg, single dipole, multiple dipole, and current density distribution) with subject-specific MR images to restrict the volume-conductor model to the correct dipole source analysis with a current reconstruction and imaging software package known as CURRY Multi-modal Neuroimaging.13 This package used several reconstruction algorithms (eg, single dipole, multiple dipole, and current density distribution) with subject-specific MR images to restrict the volume-conductor geometry to the individual anatomy. The shape of the optimum volume-conductor model was determined by segmentation or separation of the skin, skull, and brain surfaces from the MR image. We modeled these compartments using the boundary element method by assigning different appropriate conductivity values for each surface (eg, skin and skull). This allowed accurate localization of cortical activity by restricting the model to neurophysiologically accurate volume conductor geometry to the individual anatomy. The shape of the optimum volume-conductor model was determined by segmentation.

Dipole Source Analysis

Dipole source analysis was accomplished with a current reconstruction and imaging software package known as CURRY Multi-modal Neuroimaging.13 This package used several reconstruction algorithms (eg, single dipole, multiple dipole, and current density distribution) with subject-specific MR images to restrict the volume-conductor geometry to the individual anatomy. The shape of the optimum volume-conductor model was determined by segmentation or separation of the skin, skull, and brain surfaces from the MR image. We modeled these compartments using the boundary element method by assigning different appropriate conductivity values for each surface (eg, skin and skull). This allowed accurate localization of cortical activity by restricting the model to neurophysiologically accurate volume conductor geometry to the individual anatomy. The shape of the optimum volume-conductor model was determined by segmentation.

### Dipole Source Analysis

Dipole source analysis was accomplished with a current reconstruction and imaging software package known as CURRY Multi-modal Neuroimaging.13 This package used several reconstruction algorithms (eg, single dipole, multiple dipole, and current density distribution) with subject-specific MR images to restrict the volume-conductor geometry to the individual anatomy. The shape of the optimum volume-conductor model was determined by segmentation or separation of the skin, skull, and brain surfaces from the MR image. We modeled these compartments using the boundary element method by assigning different appropriate conductivity values for each surface (eg, skin and skull). This allowed accurate localization of cortical activity by restricting the model to neurophysiologically accurate volume conductor geometry to the individual anatomy. The shape of the optimum volume-conductor model was determined by segmentation.

#### MP Locations With Right or Left Finger Movement

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, y</th>
<th>Time Since Stroke, mo</th>
<th>MRI Infarct Locations</th>
<th>MP With RF Movement</th>
<th>MP With LF Movement</th>
<th>Remarks</th>
<th>Rehabilitation Results*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>53</td>
<td>6</td>
<td>Bilateral brain stem and cortical lesions</td>
<td>L Post</td>
<td>R Post</td>
<td>L hemiparesis</td>
<td>+++++</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>LAT=104 ms</td>
<td>LAT=188 ms</td>
<td>Diabetes</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>50</td>
<td>24</td>
<td>R internal capsule</td>
<td>L</td>
<td>C</td>
<td>L hemiparesis</td>
<td>+++++</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>LAT=206 ms</td>
<td>LAT=154 ms</td>
<td>Diabetes</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>50</td>
<td>14</td>
<td>R frontotemporal</td>
<td>L</td>
<td>C</td>
<td>L hemiparesis</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>LAT=170 ms</td>
<td>LAT=184 ms</td>
<td>Diabetes</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>56</td>
<td>18</td>
<td>R frontal, basal ganglia</td>
<td>L LAT=170 ms</td>
<td>Post LAT=159 ms</td>
<td>L hemiparesis</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Hypertension</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>61</td>
<td>22</td>
<td>R frontal, basal ganglia</td>
<td>L LAT=104 ms</td>
<td>LAT=170 ms</td>
<td>L hemiparesis</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Diabetes</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>60</td>
<td>48</td>
<td>L hemisphere white matter</td>
<td>C LAT=184 ms</td>
<td>L LAT=535 ms</td>
<td>L hemiparesis</td>
<td>+++++</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>R post inferior cerebellar artery occlusion</td>
<td></td>
<td></td>
<td>Diabetes</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>50</td>
<td>26</td>
<td>R basal ganglia, temporoparietal</td>
<td>L LAT=243 ms</td>
<td>R LAT=312 ms</td>
<td>L hemiparesis</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Seizures</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>71</td>
<td>144</td>
<td>R frontal</td>
<td>L LAT=272 ms</td>
<td>R LAT=367 ms</td>
<td>R hemiparesis</td>
<td>+++++</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>R ataxia</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>76</td>
<td>72</td>
<td>R temporoparietal, basal ganglia</td>
<td>L LAT=89 ms</td>
<td>LAT=120 ms</td>
<td>L hemiparesis</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Diabetes</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>67</td>
<td>60</td>
<td>L frontotemporoparietal, basal ganglia</td>
<td>L LAT=257 ms</td>
<td>R LAT=352 ms</td>
<td>R hemiparesis</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Post LAT=352 ms</td>
<td></td>
<td>Hypertension</td>
<td></td>
</tr>
</tbody>
</table>

RF indicates right finger; LF, left finger; Post, posterior location of MP; C, central location of MP; and LAT, latency of MP.

*Rehabilitation results are shown on a scale of 0 to +++++. 
appropriate source locations, such as the cortex. Calculations were based on a window of 50 ms before and after the MP peak for dipole analysis. We limited our use of dipole source analysis to the comparison of the MP distribution fields with their putative sources in individual subjects. The spatial localization of dipole sources of MRCPs has been shown to be accurate, and with self-paced movements, a single dipole can be found with low variance, ie, 5% to 10%.

Figure 1. A, Case 5: right finger movement. Left central and parietal electrodes are recording the MP negativity. Patient had a left hemiparesis. B, Case 5: left finger movement. Left central and parietal electrodes are recording the MP negativity. An MP is recorded at some right central electrodes, but amplitude is lower than on the left. Patient had a left hemiparesis.
Results
Cases 1 through 7 and case 9 had infarctions of the right hemisphere and left hemiparesis. Case 8 had right frontal, pontine, and cerebellar infarctions causing right hemiparesis and ataxia. Case 10 had left frontal and basal ganglia infarctions with right hemiparesis and aphasia. MPs were recorded in all patients. (See Table.)

Right Hemisphere Infarction
In cases 5 and 9, each with left hemiparesis, the MPs were recorded in a left frontal location with movements of either left or right fingers (contralateral to the lesioned hemisphere). The cortical infarct was right frontal in case 5 and right temporoparietal in case 9. There was basal ganglia involvement in both cases. Figures 1A and 1B (case 5) display the MPs averaged at each of 120 electrodes with movements of the right and left index fingers. With right finger movement, there was a normal distribution of the MPs, with higher amplitude on the left. Movements of the left finger were also associated with higher amplitude on the left, a paradoxical result. The grand averages of all the electrodes produced the summations shown in Figure 2, where left-sided MPs were associated with both left and right finger movements. This result was not present in any of the normal control subjects. Figure 3 shows the MP averages in case 9, with the addition of dipole source localization. The dipole generators of both left and right finger movements originated in the left hemisphere. An extensive infarction can be seen on the right (in this Figure, the image of the right hemisphere is on the viewer’s left).

In cases 2 and 3, movements of the affected left fingers were associated with centrally placed MPs, whereas the MPs were contralateral (left frontal) with right finger movements. Figure 4 (case 2) combines MPs, current densities, MRI, and source localization. With left and right finger movements, the current densities were more intense over the left hemisphere. The dipole generators originated in the left hemisphere. In case 6, the patient had a recent white-matter infarct adjacent to the left lateral ventricle and a prior occlusion of the left posterior inferior cerebellar artery. He had a left hemiparesis. Bilateral calcification of the carotid and vertebral arteries was present. There was a central MP with left finger movement and a left-sided MP with right finger movement (not illustrated). The latency of 535 ms with left finger movement was exceptionally long.

Figure 2. Cases 9 (A) and 5 (B). Both patients had left hemiparesis; maps of the averaged MPs with left (LF) and right (RF) finger movement are shown for both patients. MPs are the left-of-center areas of negativity (dark end of the gray scale) in the maps.
Left Hemisphere Infarction
In case 10 (Figure 5), the MP with left finger movement was in a normal location. In contrast, the MP with right finger movement was in a posterior position. There was an extensive infarction of the left hemisphere (Figure 5), with encephalomalacia involving the frontal lobe and extending into the basal ganglia. Figure 6 shows the dipole source originated from behind the infarct (the rim) in the involved hemisphere. The patient had a nonfluent aphasia with a right hemiparesis.

Figure 3. Case 9. MPs with either left or right finger movement are on the left side of the maps and were best recorded in the left hemisphere. Bottom, The dipole sources (arrows) within the left hemisphere were oriented inferi orly and toward the right. The beginning of the dipole arrow identifies the source, the size demonstrates the strength, and the point identifies the direction. Note the large right tempora l lobe lesion in the MRI.

Figure 4. Case 2. Juxtaposition of MPs, current densities, and dipole sources. The MP with left finger movement is in a central rather than a normal right (contralateral) position. The current density field shows activity bilaterally (yellow, green, and red, with yellow indicating peak density, red the least density, and blue, no density). The dipole source originates in the left hemisphere, close to the midline (red arrows). Right finger movement is associated with a left (contralateral) MP, as expected. The electrical field differs from the right hemisphere field in that it is more active (yellow and green) and localized close to the left sensorimotor cortex. The dipole source originates in the left hemisphere. Patient had an infarction of the right internal capsule and a left hemiparesis.
Cases 1, 4, 7, 8, and 10 had contralateral dominant MPDs with movements of the affected side. The mean latencies in the contralateral group (n=5) were longer than in the ipsilateral group (cases 2, 3, 5, 6, and 9), but only with movements of the right finger. Otherwise, there were no differences with respect to lesion location, recovery, age, or duration of stroke. Latencies were not different between intact and affected side movements, but the numbers were small.
In summary, normal individuals had MP field distributions at 120 electrodes that, when averaged, localized to the hemisphere contralateral to the finger movements. Patients recovering or recovered from right hemisphere infarcts had MPs, which mapped either to the left hemisphere or a central location with finger movements of the left hand. A patient with right hemiparesis and aphasia had a dipole source at the posterior rim of a large frontal infarct when moving his right finger.

Discussion

We have demonstrated that hr-EEG coregistered with MRI is capable of identifying reorganization of motor control in stroke patients with recovered hemiplegia. This may involve a total or partial shift to the ipsilateral hemisphere, or there may be relocation in the same hemisphere. Honda et al reported MRCFs, regional cerebral blood flow measurements, and PET activation studies in 2 patients with hemiparesis. Their multimodal studies suggested an important role of the ipsilateral hemisphere in the process of motor recovery. Recently, Cao et al reported in an fMRI study that sensorimotor cortex in the intact hemisphere was activated in 6 of 8 patients during movements of the affected hand. In addition to observing such ipsilateral activation, we noted a contralateral posterior displacement of the MP to the rim of the peri-infarct area in our case 10. Although the patient had a left internal carotid artery occlusion with right hemiparesis, the right sensorimotor cortex was not activated, perhaps because the right internal carotid artery was stenosed. There has been much speculation about the identity and role of compensatory processes in stroke recovery. Cramer et al in their fMRI study of patients recovering from stroke suggested 3 processes related to motor recovery, namely, “activation of a motor network in the unaffected hemisphere greater than seen in controls, increased degree of SMA [supplementary motor area] activation, and activation of focus along the rim of a cortical infarct.” Our methodology did not allow us to record the degree of supplementary motor area activation, but we can confirm their other 2 conclusions. Despite completely different methodologies, our EEG studies have also shown both activation of a motor network in the unaffected hemisphere much greater than that seen in controls and activation of foci along the rim of a cortical infarct. The dipole source in our case 10, originating behind the rim of a left frontal infarct, suggests a reorganization occurring in the area of the former ischemic penumbra. There was no evidence of ipsilateral activation, probably because of right hemisphere ischemia due to an internal carotid occlusion.

Further understanding of how manifestations of neuroplasticity relate to stroke recovery in this and other cases should come from the continued application of multimodal techniques in brain imaging. The combination of fMRI and hr-EEG may be complementary, because 1 modality (fMRI) has greater spatial and the other (hr-EEG) better temporal resolution.

Acknowledgments

This study was supported by the Office of Research and Developmental and Rehabilitation Research and Development Service, Department of Veterans Affairs, Washington, DC. The authors wish to express their gratitude to the patients who volunteered to participate in this research and to their family members who accompanied them. Thanks are also due to the medical staff of Hines Veterans Affairs Hospital for their referrals.

References

High-Resolution EEG in Poststroke Hemiparesis Can Identify Ipsilateral Generators During Motor Tasks
Joseph B. Green, Yolanda Bialy, Elena Sora and Anthony Ricamato

Stroke. 1999;30:2659-2665
doi: 10.1161/01.STR.30.12.2659

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/30/12/2659

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/