The influence of skull defects and reperfusion after extra-intracranial arterial bypass surgery on the sensorimotor EEG rhythm*

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SUMMARY Twelve patients with small burr hole skull defects after extra-intracranial arterial bypass surgery were studied. The amplitude and frequency of the sensorimotor rhythm were measured 7 days and 1, 2, 4, and 6 months after surgery in follow-up EEGs from the central region. Seven patients showed a frequency decrease (compared with preoperative measurement) on the operated side 7 days and/or 1 month after surgery. There was no case of frequency decrease 6 months after surgery; four patients displayed a late frequency increase. Ipsilateral amplitude enhancement was never found 7 days postoperatively, but after 6 months in nine patients. Such physical factors as burr holes and bone replacement can only partially explain the amplitude enhancement, and cannot explain the frequency decrease. It may be assumed that temporary clamping of the middle cerebral artery and/or reperfusion of an ischaemic area result in a brief deterioration of brain function, as indicated by frequency slowing and delayed amplitude enhancement; related observations were made in patients with cerebrovascular disorders and mild to moderate neurological deficit about 20 days after the onset and correlated with clinical recovery.

The sensorimotor rhythm is defined as rhythmic EEG activity located in the sensorimotor cortex; it may be found within the alpha band (8–12 Hz) or even below, or within the beta band. In the normal brain the sensorimotor rhythm is reactive to voluntary movement and somatosensory stimuli. It is variously called the mu rhythm, rythme en arceaux, rolandic wicket rhythm,1,2 or central beta rhythm.3,4 The sensorimotor rhythm is generated within the territory supplied by the middle cerebral artery and is affected by cerebrovascular disorders in that region.5,6 and can therefore serve as an indicator of central brain function before and after extra-intracranial arterial bypass surgery, in which a branch of the middle cerebral artery is used for end-to-side anastomosis. EEG changes after extra-intracranial arterial bypass surgery in the form of amplitude enhancement were reported by Holbach et al,7 De Weerd et al8 and Yamakami et al.9 Before the sensorimotor rhythm can be used to indicate intracerebral metabolic changes as a result of revascularisation surgery, however, the effect of burr holes and bone replacement on the sensorimotor rhythm recorded from the scalp must be thoroughly considered, as skull defects can result in amplitude enhancement of the mu or mu-like rhythm.10–12 Here, we are particularly concerned with the results of a follow-up EEG study of 12 extra-intracranial arterial bypass patients with respect to the effect of skull defects and reperfusion on sensorimotor rhythm within the alpha range or below.

Material and methods

Patients Twelve patients underwent extra-intracranial arterial bypass surgery and computed EEG follow-up studies. The clinical, angiographic, EEG and CT data are summarised in

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It should be noted that nine patients had cortical infarctions (hypodense area in the CT scan), whilst two had infarction of deep structures and one had mild atrophy. Ten patients remained neurologically unchanged after the operation. Two patients (nos 1 and 2) reported improvement of vision and facial palsy, respectively, within 24 hours after surgery. On day 16, patient no 1 had a speech arrest; patient no 2 suffered another stroke in the operated hemisphere 7 months after surgery.

**EEG recording and data processing**

EEG recordings were made using six bipolar channels, three from each hemisphere, with electrodes in an equidistant transverse configuration in positions C3, C4, and Cz of the international 10–20 system. The other electrodes were fixed between the standard positions. Because of the operation scar on the temporal region, all electrodes were moved forward by about 2 cm from the standard and interspaced positions (fig 1). The EEGs were recorded during rest and during voluntary hand movements. Under both conditions the patient sat with his eyes closed, if possible, in a comfortable chair in a soundproof room. Sixty trials of 6 s each with intervals of about 10 s between trials were sampled during rest and during movements. Under movement conditions, the patient held a rubber ball, diameter 6 cm, in one hand and squeezed it at self-paced intervals longer than 10 s. In seven patients, the hand contralateral to the operated hemisphere performed the movement task; otherwise the ipsilateral hand was used.

After anti-aliasing filtering (cutoff frequency 30 Hz) and sampling with 64 Hz, the 6 s epochs were analysed with a PDP 11/03 computer by calculating the power spectra using

![Figure 1](https://example.com/fig1.png)

Electrodes affixed to the scalp of a patient who had undergone extra-intracranial arterial bypass surgery. The electrode near the hairline is the ground.

**Table 1 Clinical data**

<table>
<thead>
<tr>
<th>No</th>
<th>Age</th>
<th>Sex</th>
<th>Clinical symptoms</th>
<th>Neurological signs</th>
<th>Angiographic findings</th>
<th>CT findings</th>
<th>EEG abnormalities</th>
<th>Interval CS (TIA)–EIAB (months)</th>
<th>Side of EIAB</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>56</td>
<td>M</td>
<td>CS</td>
<td>right hemiparesis</td>
<td>ocel ICA left</td>
<td>severe atr left old inf oecip right</td>
<td>diffuse</td>
<td>5</td>
<td>left</td>
</tr>
<tr>
<td>2</td>
<td>59</td>
<td>M</td>
<td>CS</td>
<td>severe right hemiparesis</td>
<td>ocel ICA left</td>
<td>ocel ICA left</td>
<td>focal</td>
<td>2</td>
<td>left</td>
</tr>
<tr>
<td>3</td>
<td>35</td>
<td>F</td>
<td>CS</td>
<td>hemiparesis left</td>
<td>ocel MCA right</td>
<td>ocel ICA left</td>
<td>focal</td>
<td>4 yrs</td>
<td>right</td>
</tr>
<tr>
<td>4</td>
<td>29</td>
<td>F</td>
<td>CS</td>
<td>hemiplegia right</td>
<td>ocel ICA left</td>
<td>ocel ICA left</td>
<td>focal</td>
<td>2</td>
<td>left</td>
</tr>
<tr>
<td>5</td>
<td>49</td>
<td>M</td>
<td>CS</td>
<td>hemiparesis right</td>
<td>ocel ICA left</td>
<td>ocel ICA left sten ICA right, VER right</td>
<td>focal</td>
<td>3–5</td>
<td>left</td>
</tr>
<tr>
<td>6</td>
<td>57</td>
<td>M</td>
<td>CS</td>
<td>hemiparesis left</td>
<td>ocel ICA left</td>
<td>ocel ICA right</td>
<td>focal</td>
<td>1–5</td>
<td>right</td>
</tr>
<tr>
<td>7</td>
<td>44</td>
<td>F</td>
<td>CS</td>
<td>hemihyposthesia left</td>
<td>ocel ICA right</td>
<td>ocel ICA right</td>
<td>no</td>
<td>3</td>
<td>right</td>
</tr>
<tr>
<td>8</td>
<td>68</td>
<td>M</td>
<td>CS</td>
<td>none</td>
<td>ocel ICA left</td>
<td>ocel ICA right</td>
<td>mild atrophy</td>
<td>1</td>
<td>left</td>
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<tr>
<td>9</td>
<td>69</td>
<td>M</td>
<td>TIA</td>
<td>mild hemiparesis left</td>
<td>ocel ICA left</td>
<td>ocel ICA left</td>
<td>no</td>
<td>7</td>
<td>right</td>
</tr>
<tr>
<td>10</td>
<td>56</td>
<td>M</td>
<td>CS</td>
<td>hemiplegia right</td>
<td>ocel ICA left</td>
<td>ocel ICA left</td>
<td>focal</td>
<td>2–5</td>
<td>left</td>
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<tr>
<td>11</td>
<td>54</td>
<td>M</td>
<td>CS</td>
<td>mild hemiparesis right</td>
<td>ocel VER bilat</td>
<td>ocel ICA right</td>
<td>focal</td>
<td>2</td>
<td>left</td>
</tr>
</tbody>
</table>

ICA = internal carotid artery, MCA = middle cerebral artery, VER = vertebral artery, OCCL = occlusion, STEN = stenosis, CS = completed stroke, TIA = transient ischaemic attack, INF = infarction.
were used with rhythm measurements. The magnitude squared Fast Fourier Transform (FFT) was greater bilateral frequency values out Ten Results channel sensorimotor 6-12 Hz band was chosen to determine the reliability of the peak measurements. The magnitude of the highest spectral peak (power) within 6-12 Hz was used as a measurement of the sensorimotor rhythm amplitude. The power value of the middle channel on the operated side was referred to the intact (non-operated) side; a positive percentage indicates unilateral amplitude enhancement and a negative percentage unilateral attenuation. In normal subjects both frequency and amplitude (power) show a high degree of bilateral symmetry. Significant (p < 0.05) amplitude enhancement was assumed when the percentage increase was greater than 135% (this limit was obtained from a group of 38 neurologically normal subjects—part of the data published). Analysing the EEG data during voluntary hand movement, we found in 10 of the 12 patients a blocking of sensorimotor rhythm on at least one occasion during the follow-up measurements. Close relationship between the sensorimotor rhythm and sensorimotor function could therefore be assumed.

Results

SENSORIMOTOR RHYTHM AMPLITUDE ASYMMETRY

Ten out of 12 patients displayed unilateral amplitude enhancement (whereby the peak power was at least 135% higher on the operated than on the non-operated side) at least once during the follow-up period. One patient (no 10) always showed amplitude symmetry and another (no 9), amplitude attenuation. Table 2 presents a summary of the results for all the patients. It should be noted that three patients already showed enhanced sensorimotor rhythm amplitude in the preoperative measurement, while no patient showed enhanced 7 days after extra-intracranial arterial bypass surgery.

SENSORIMOTOR RHYTHM FREQUENCY CHANGE

Sensorimotor rhythm frequencies were measured on the operated and non-operated sides and compared to preoperative values. Table 3 shows the frequency increases (+) and decreases (−) found 7 days, 1 month and 6 months after surgery. At 7 days and/or 1 month after surgery, seven of the 12 patients showed a frequency decrease and only one, an increase. Six months after surgery, no patient showed a decrease, and four an increase.

CHANGES IN THE AMPLITUDE AND FREQUENCY OF THE SENSORIMOTOR RHYTHM IN PATIENTS WITH PREOPERATIVE CORTICAL INFARCTION

Amplitude asymmetry and frequency changes (referred to the preoperative value) from eight patients with cortical infarction shown by the preoperative CT scan were averaged and displayed for discrete measurements after extra-intracranial anastomosis (fig 2). One additional patient with infarction of the total territory of the middle cerebral artery and severe neurological deficit was not included in these data. The other three patients had no sign of cortical infarction on the CT scan. Statistical calculation was omitted owing to the small number of patients. Six of these eight cortical infarction patients displayed frequency decrease 7 days and/or 1 month after extra-intracranial arterial bypass surgery. Six of the eight also showed reduced amplitude enhancement or amplitude attenuation 7 days after extra-intracranial arterial bypass. All eight patients displayed an amplitude enhancement between 7 days and 1 month after surgery. Decreased frequency and unilateral amplitude enhancement

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**Table 2** Amplitude asymmetry before and at different time intervals after extra-intracranial arterial bypass. The reference side is always the non-operated hemisphere

<table>
<thead>
<tr>
<th>Patient’s no</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
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</thead>
<tbody>
<tr>
<td>Pre-EIAB</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>0</td>
<td>+</td>
<td>0</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>7 days</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1 month</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>0</td>
</tr>
<tr>
<td>6 months</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>+</td>
<td>0</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>post-EIAB</td>
<td>(1)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>

0 amplitude symmetry.

+ amplitude enhancement on ischaemic (operated) side of 135–270% (levels arbitrary; significant enhancement > 135%).

+ + > 270%.

+ + > 135%.

− amplitude decrease on the ischaemic side by 56–73%.

= > 73%.

(1) only 4 months after EIAB.

EIAB = extra-intracranial arterial bypass.

**Table 3** Frequency increase or decrease on the operated side at different intervals after extra-intracranial arterial bypass surgery, referred to the preoperative control measurement

<table>
<thead>
<tr>
<th>Patient’s no</th>
<th>1</th>
<th>2</th>
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<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
</tr>
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<tbody>
<tr>
<td>7 days</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>−</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1 month</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>−</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>6 months</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>post-EIAB</td>
<td>(2)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

0 frequency unchanged.

+ frequency increased by 0.5–0.9 Hz (arbitrary levels).

+ + > 0.9 Hz.

− frequency decreased by < 0.5–0.9 Hz.

= > 0.9 Hz.

(1) frequency already very slow before EIAB (< 7.5 Hz).

(2) only 4 months after extra-intracranial arterial bypass.
after the 7th day were therefore characteristic changes in the sensorimotor rhythm after extra-intracranial arterial bypass surgery in patients with cortical infarction. There was a tendency to amplitude attenuation at 7 days, before the enhancement develops.

**Absolute and Relative Mu Power After Extra-Intracranial Arterial Bypass**

Mu power is defined as power within the mu range of 6–10 or 8–12 Hz (depending on the peak frequency) and can be expressed absolutely in μV² or relatively in percent referred to the total power from 0–32 Hz. The total power is equivalent to the variance of the EEG signal. Amplitude enhancement may be either localised in the mu range alone, or present in all frequency bands. Physical factors, such as burr holes, can only be taken into consideration as the reason for the enhancement when all frequency bands are enhanced by the same factor. These two cases may be differentiated by calculating the absolute and relative power within the 6–10 Hz (or 8–12 Hz) band. An amplitude increase in sensorimotor rhythm alone may be assumed when both power values are increased by about the same factor. When the absolute power alone is increased and the relative power is unchanged, a general increase in all EEG frequencies is probable, and physical factors may be taken into consideration. We found both types of absolute/relative power behaviour in our data. Fig 3 shows examples.

**Discussion**

Three factors seem to be causally related to the sensorimotor rhythm changes resulting from revascularisation surgery. Defects in the skull very
often result in an enhanced mu rhythm or mu-like rhythm, sometimes with a frequency below the mu range. This rhythm has also been called a "breach rhythm." (1) Defects in the skull caused by incomplete bone replacement, and the small hole for the extra-intracranial anastomosis. (2) Clamping of the middle cerebral artery branch during the microsurgical anastomosis. Clamping a middle cerebral artery branch possibly produces temporary ischaemia, depending on the amount of collateral circulation. (3) Reperfusion of an area of low perfusion with the extra-intracranial bypass. Sudden increase of the perfusion pressure in an ischaemic, that is abnormally autoregulating area, could result in oedema after establishment of extra-intracranial perfusion. This phenomenon has been called "normal perfusion-pressure breakthrough" under similar pathophysiological circumstances of arteriovenous malformations. In this situation the function/flow correlation is lost as in the "luxury-perfusion syndrome".

Tables 2 and 3, as well as fig 1 show how the amplitude and frequency of the sensorimotor rhythm varied at different intervals after surgery. Seven days after revascularisation surgery the sensorimotor rhythm was significantly slowed, with a tendency to amplitude attenuation. One month after surgery the decrease in frequency persisted, but with a marked enhancement in amplitude on the operated side. Several months after surgery about half the patients showed increased frequency parallel to an enhanced amplitude. The sensorimotor rhythm thus displays different patterns 7 days, 1 month, and several months after surgery.

Frequency decrease, found in seven out of 12 patients 7 days and/or 1 month after surgery indicates a form of brain dysfunction. EEG frequency slowing is observed in parallel with decreased oxygen uptake and decreased cerebral blood flow. Frequency decrease is also typical in senescence, after hypoxia and in patients with cerebrovascular disease. A direct relationship between frequency slowing and the skull defect is improbable, since this decrease cannot be explained by physical factors, that is by a reduction in resistance resulting from the burr hole and the replaced bone. The frequency decrease (deterioration of brain function) thus can only be explained as result of clamping of the middle cerebral artery branch and/or reperfusion of an ischaemic area. Four patients showed frequency increase 6 months after surgery; in only one patient (no 9) this improvement appeared immediately after surgery. Such frequency increase, seen for example parallel to clinical recovery after stroke, can be viewed as a sign of increased cerebral metabolism and improved brain function. Fig. 4 shows typical examples of frequency increase accompanying clinical recovery in three patients with cerebrovascular accidents treated medically. Two of these patients had occlusions of the middle cerebral artery and internal carotid artery, respectively, and are comparable to patients in our study. In fig 4 also the frequency change sequence of two patients (nos 2 and 4) of our bypass study group is illustrated.

In contrast to the frequency changes, there is no such simple interpretation for the changes in amplitude, as other than physical factors must be considered. Bone replacement and burr holes can only be partially responsible for unilateral amplitude enhancement (see fig 3). Other factors must also be involved, as mentioned in Cobb et al because: (1) two patients showed no enhancement during all follow-up measurements (nos 9 and 10); (2) three other patients (nos 4, 6, 8) showed unilateral enhancement even before surgery; and (3) amplitude enhancement was not found in any patient 7 days after surgery. This is in agreement with the observation of Fischgold et al and Cobb et al that breach rhythms are not present shortly after surgery and take some time to develop.

Two general observations of patients with cerebrovascular disease are important in this respect: (1) capsular haemorrhage, small lesions in the posterior part of the thalamus and infarction in the basal ganglia can result in an ipsilaterally enhanced mu or alpha rhythm. (2) Cortical infarction is often accompanied in the first days or weeks after the onset
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by an ipsilaterally attenuated mu rhythm which later changes to an enhanced mu rhythm. Patients with this delayed ipsilateral enhancement usually had a mild-to-moderate neurological deficit and showed good clinical recovery (fig 5). The transition from unilateral amplitude attenuation to an enhancement is thus a typical sign of brain recovery after a cerebrovascular accident. Conversely, a growing unilateral amplitude attenuation can be interpreted as a sign of progressive deterioration of brain function. Fig 2 shows the characteristic change in amplitude asymmetry—a trend to attenuation immediately after surgery followed later on by a distinct enhancement—which suggests that the amplitude asymmetry, like the frequency decrease, reflects to an as yet not precisely defined extent a deterioration in brain function in the first days after bypass surgery. This is probably due to the clamping of the middle cerebral artery branch and/or to the reperfusion of an ischaemic area. An improvement then follows. It should be emphasised that these characteristic amplitude and frequency changes were found mainly in patients with cortical infarction in the preoperative CT scan (patients nos 1–8). None of the three patients without cortical infarction (nos 9, 11 and 12) displayed a frequency decrease, but two showed amplitude enhancement (nos 11 and 12). No 12 had a parallel enhancement of absolute and relative mu power (compare fig 3), which can be explained by physical factors.

The delayed amplitude enhancement starting 7 days after surgery can be interpreted as a sign of recovery from temporary brain dysfunction after reperfusion in parallel with a lessened frequency decrease. In this context, the sooner this amplitude enhancement occurs, probably the more favourable the outlook. The two patients with speech arrest and a second stroke after revascularisation showed maximal enhancement late, at 5 and 6 months after surgery, respectively. Of the four patients with improved brain function (defined by increased frequency) 6 months after surgery, three had maximal enhancement in the 1st month, and one in the 3rd month after surgery. The other patients without frequency increase as a long term effect showed maximal enhancement 4 to 8 months after revascularisation.

We may say, in summary, that unilateral amplitude enhancement is only partly the result of the burr hole and bone replacement; the clamping of the middle cerebral artery and/or the reperfusion of an ischaemic area seem to be more significant factors. The delayed amplitude enhancement (on the order of 7 days to 1 month) is similar to that also found in patients with cerebrovascular disorders and mild-to-moderate neurological deficit about 20 days after the onset in parallel with clinical recovery. Amplitude enhancement is therefore, like frequency decrease, another sign of brain dysfunction and cannot be explained by physical factors alone. Early development of enhancement (for example after 1 month) is a favourable sign, indicating prompt recovery from temporary brain dysfunction induced by surgery.

References


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