Significance of EEG Changes at Carotid Endarterectomy

WARREN T. BLUME, M.D., F.R.C.P. (C), GARY G. FERGUSON, M.D., F.R.C.S.(C), D. KENT MCNEILL, R.E.T.

SUMMARY Visually apparent EEG changes associated with clamping the internal carotid artery appeared in 55 of 176 consecutive patients (31%) undergoing carotid endarterectomy without shunt. Attenuation of higher frequency activity was the most common change. Changes were moderate in 33 patients (19%) and major in 22 (12.5%). Major changes usually commenced earlier than less severe alterations. EEG changes resolved within 10 minutes of clamp release in 36 of 55 patients (65%) after an average clamp time of 36.25 minutes.

Changes occurred more commonly when pre-operative EEGs were abnormal contralateral to clamping and when the contralateral carotid artery was more than 90% stenosed.

Of the inhalational anesthetics employed with nitrous oxide and oxygen, isoflurane was associated with the lowest incidence of clamp-associated EEG change.

Post-operative strokes occurred in 2 of 22 patients (9%) with major clamp-associated EEG changes, none of 33 patients with moderate changes and none of 121 without changes. However, the mechanism of this relationship remains in doubt.

Stroke Vol 17, No 5, 1986

SOME ADVOCATES OF SELECTIVE SHUNTING at endarterectomy monitor the procedure with EEG to make their decision, assuming that an EEG change during internal carotid artery clamping indicates the need for a shunt.1-2 Although three patients reported in the literature had tolerated clamp-associated EEG changes for 10-24 minutes3,4 and although several series of endarterectomies without shunts have been reported,5-8 this assumption had never been tested in a series of patients.

Our surgical policy not to use shunts at carotid endarterectomy has afforded us an opportunity to study EEG changes during more prolonged internal carotid artery clamping and to examine their significance.

Methodology

One hour prior to surgery, 20 scalp electrodes were applied with collodion according to the International Ten-Twenty System of electrode placement. Electrode impedances were always less than 2000 Ohms. A bipolar anterior-posterior 16 channel montage covering the parasagittal and temporal regions recorded potentials while awake and during anesthesia. The high frequency filter was set at 70 and the low frequency filter set at 0.3. The sensitivity was usually 5 uV/mm. Paper speed was 3 cm/sec for the wake recording and usually 1.5 cm/sec during anesthesia.

An awake recording of 15 minutes duration was obtained immediately prior to the surgery. Recording while anesthetized was carried out for 10 minutes immediately prior to internal carotid artery clamping, throughout the period of carotid clamping, and for 10 to 15 minutes post clamping.

The recordings were interpreted by a single electroencephalographer who was unaware which side was clamped but was aware of the time of clamping and unclamping of the carotid artery.

We defined a major EEG change as an attenuation of 8–15 Hertz activity to minimal or nil and/or a two-fold or more increase of delta activity at one Hertz or less. The changes could be ipsilateral to clamping or bilateral (figs. 1, 3, 5).

A moderate change consisted of clearly persisting 8–15 Hertz activity whose amplitude was attenuated by at least 50% and/or an obvious and persistent increase of delta activity at greater than 1 Hertz. The changes could be ipsilateral or bilateral (figs. 2, 4).

Except where indicated, p values were calculated using chi square with Yates’ continuity correction.

Results

Types and Degree of Change

A visually apparent EEG change associated with clamping of the internal carotid artery occurred in 55 of 176 patients (31%). The most common change was an attenuation of ongoing higher frequency activity and this constituted the only change in 19 patients (figs 1, 2). Delta activity appeared or was augmented as the only change in 6 patients (figs 3, 4). Both attenuation of higher frequencies and increased delta activity constituted the EEG change in 30 patients, but the attenuation component almost always was the more prominent (fig 5).

In 39 instances, the change was confined to the hemisphere ipsilateral to the clamped internal carotid artery while the changes involved both sides in 16 instances. This laterality of change did not correlate with its nature (attenuation and/or delta).

Although the quantity of change was moderate in 33 patients (19%), major changes appeared in 22 cases (12.5%). A complete or almost complete loss of higher frequency activity constituted the major change in 12 patients, a marked increase in delta activity appeared in 3 patients and both conditions prevailed in an additional 7 patients. However, the distribution of such marked changes resembled that of the entire group: 14 were ipsilateral and 8 were bilateral.
CAROTID ENDARTERECTOMY
EFFECT OF CLAMPING

BEFORE
DURING

FIGURE 1. Major change. Marked diminution of background activity in left hemisphere (temporal regions illustrated) during left internal carotid artery clamping. Note slight preclamp asymmetry, left lower. Periodic sharp waves in second, third and eighth channels are ECG. EEG annotation for Ten-Twenty System of electrode placement; odd numbers left, even right.

Latency to Change

Of the 51 patients in whom the latency between clamping the internal carotid artery and the EEG change could be determined with confidence, the change occurred within the first minute in 42 patients (82%) and within the first 20 seconds in 29 of these 42 (69%). 18 of 22 major changes (82%) occurred within 20 seconds as compared to only 11 of 29 moderate changes (38%) \(p < .005\).

Attenuation and increased regional delta occurred simultaneously in 22 of the 28 instances in this group in which both were present; delta appeared earlier in 4 and attenuation earlier in 2 patients. There was little difference in the latencies for attenuation (median: 20 seconds) and delta (median: 25 seconds).

Clamp Time

When no EEG change occurred, the clamp time averaged 37.9 minutes (range: 22–60 minutes), but when an EEG change occurred, the clamp time averaged 32.6 minutes (range: 16–56 minutes). Clamp time for major EEG changes averaged 32 minutes (range: 22–50 minutes). The difference in clamp times may relate to GGF’s policy of performing the endarterectomy himself when an EEG change is observed instead of supervising the resident’s performance.

There was no correlation between clamp time and reversibility of EEG change. For example, the EEG changes in 7 of 12 patients whose clamp times ranged from 20–29 minutes reversed in the 10 minutes recording time after clamp release; those in 4 of 6 patients whose clamp times ranged from 50–59 minutes reversed.

Resolution

The abnormalities fully resolved either during clamping or more commonly within 10 minutes of clamp release in 36 of the 55 patients (65%). Incidence of resolution was unaffected by the type of EEG change or by its spatial extent. Attenuation resolved in 35 of 49 instances (71%) and delta in 24 of 36 cases (67%). Ipsilateral changes resolved in 27 of 39 patients (69%) and bilateral changes resolved in 9 of 16 instances (56%).

Twenty-four of 33 (73%) moderate changes resolved completely while 11 of 22 (50%) major changes fully resolved \(p = N,S\).

Clinical Implications of EEG Changes

New neurological deficits appeared in 5 of the 55 patients (9%) with EEG changes, 3 of which cleared within 24 hours of the endarterectomy. None developed among the 121 patients without EEG change.

Two of the 176 EEG monitored patients (1.1%) developed immediate intra-operative strokes. These two had had major EEG changes. One had a marked delta and marked attenuation beginning 20 seconds after clamping while the other had a marked attenuation and mild delta beginning 12 seconds after clamping. The delta resolved in the former patient but neither phenomenon resolved in the latter.

Thus, 2 of 22 patients with major changes (9%) developed intra-operative stroke as compared to none of 154 patients with no or moderate change \(p = 0.015\, \text{Fisher’s exact text, one-tailed}\). (A one tailed test is appropriate here as stroke would expectedly be associated with a larger EEG change than would no stroke).

Factors Associated with EEG Change During Clamping

Pre-Operative EEG

Nine of 16 patients (56%) whose pre-operative EEGs showed exclusively or primarily contralateral abnormalities (table 1) had EEG changes during clamping as compared to only 45 of 158 patients (28%) whose recordings were normal, had ipsilateral or diffuse abnormalities, or whose background was asymmetrical (table 1) \(p < .025\). Two patients had no preoperative EEG.

Nine of 20 patients (45%) whose pre-operative EEGs showed delta and/or attenuation on either side had EEG changes during clamping as compared to 46 of 150 patients (31%) whose preoperative EEGs did not contain either of these features, but the differences

CLAMPING REDUCES AMPLITUDE

FIGURE 2. Moderate change. Reduced, but clearly persistent higher frequency activity in left hemisphere (parasagittal derivations shown) during left internal carotid artery clamping.
EEG at Endarterectomy/Blume et al

A page from a document containing text about EEG changes during carotid endarterectomy. The text discusses the effects of clamping the right internal carotid artery on EEG recordings, including changes in delta and theta activities. It also mentions the use of general anesthesia and the correlation between EEG changes and cerebral blood flow measurements.

Contralateral Carotid Artery

Patency of the contralateral carotid artery as estimated at angiography varied inversely with the incidence of either a major change or moderate change as compared to no EEG change (Table 2). For example, an EEG change appeared in 17 of 25 patients (68%) whose contralateral carotid artery was either occluded or more than 90% stenosed as compared to 24 of 107 patients (22%) in whom that artery was normal or less than 50% stenosed.

In contradistinction, the patency of the artery to be operated upon did not correlate with the incidence of EEG change during clamping.

General Anesthesia

General anesthesia was used in all cases; the type depended upon preference of the anesthetist and not on clinical features. It was maintained with nitrous oxide and oxygen in combination with Halothane, Enflurane, Isoflurane or a narcotic-relaxant. EEG changes occurred in only 12 of 58 patients (21%) with Isoflurane and this is less than with Halothane (15 of 38 patients, 39%), (p < .10) or with Enflurane (10 of 25 patients (40%) (p = N.S). Only 5 of 22 patients (23%) in whom narcotic-relaxant technique was used had EEG changes, but the differences failed to attain statistical significance in comparison with Halothane or Enflurane. Anesthetic type was not available for some early cases.

Cerebral Blood Flow

Cerebral blood flow (CBF) was measured in 29 patients using the intracarotid injection of 133-xenon. Most of the measurements were made in the early part of this series; no aspect of any case determined the use of CBF measurements.

Values ranged from 12 ml/100 gm/min to 62 ml/100 gm/min. All 4 patients with a CBF below 15 ml/100 gm/min had a major EEG change during internal carotid artery clamping. Six of the 10 patients with flows between 16 and 29 ml/100 gm/min had EEG changes with no distinct trend within this range. Only one of 15 patients with a cerebral blood flow greater than 30 ml/100 gm/min had an EEG change and this was major. None of the 2 patients with complicating stroke or the patients with transient post-operative ischemia had cerebral blood flow studies.

Factors Not Influencing Incidence of EEG Change

Several clinical factors failed to correlate with EEG change during internal carotid artery clamping. These included: age, sex, side of operation and indication for}

Figure 3. Major change. Most prominent change, best seen 70 seconds after onset of clamping right internal carotid artery, is right hemisphere delta activity (temporal regions illustrated) with some decrease in higher frequency activity in right hemisphere; this is preceded by an increase in right hemisphere theta at 5 seconds. 5, 70 sec after = time after onset of clamping in seconds.

Figure 4. Moderate change. Increase in right hemisphere 2 Hertz delta activity (temporal derivations shown) with right internal carotid artery clamping. Slight increase in delta at Fpl.

Figure 5. Major change. Both diminution of higher frequency background activity and an increase in <.5 Hertz delta activity are seen on the right in the segment taken 3 minutes after onset of clamping the right internal carotid artery.
Table 1  Pre-operative EEG and EEG Change during Clamping

<table>
<thead>
<tr>
<th>Pre-operative EEG</th>
<th>Change (patients)</th>
<th>No change (patients)</th>
<th>Percent change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>23</td>
<td>50</td>
<td>32</td>
</tr>
<tr>
<td>Abnormalities ipsilateral to clamping</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ipsilateral only</td>
<td>11</td>
<td>37</td>
<td>23</td>
</tr>
<tr>
<td>bilateral, chiefly ipsilateral</td>
<td>3</td>
<td>8</td>
<td>27</td>
</tr>
<tr>
<td>total ipsilateral</td>
<td>14</td>
<td>45</td>
<td>24</td>
</tr>
<tr>
<td>Abnormalities contralateral to clamping</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>contralateral only</td>
<td>5</td>
<td>2</td>
<td>71</td>
</tr>
<tr>
<td>bilateral, chiefly contralateral</td>
<td>4</td>
<td>5</td>
<td>44</td>
</tr>
<tr>
<td>total contralateral</td>
<td>9</td>
<td>7</td>
<td>56</td>
</tr>
<tr>
<td>Diffuse abnormalities only</td>
<td>5</td>
<td>11</td>
<td>31</td>
</tr>
<tr>
<td>Asymmetry of background potentials only</td>
<td>3</td>
<td>7</td>
<td>30</td>
</tr>
</tbody>
</table>

As seen clinically in acute stroke, loss of higher frequency background activity and the appearance or increase of regional delta activity constitute the principal changes which may be associated with clamping the internal carotid artery. Although regional delta activity is the major finding in acute stroke, diminution of higher frequency components occurred more commonly as the principal finding in earlier endarterectomy series and the present one. Trojaborg and Boyesen found attenuation of EEG activity to be the most prominent change at very low cerebral blood flow (CBF) values (11–19 ml/100 gm/min) and a regional increase in delta activity to be the principal finding when CBF reductions were less marked (16–23 ml/100 mg/min). However, the wide overlap indicates that other factors play a role.

Such EEG changes can result from hemodynamic alterations, emboli dislodged by the clamping, or both. It is likely that embolism created the EEG change in only a small minority of our cases because: 1) such changes were associated with post-operative neurological deficits in only 2 of 55 patients with any EEG change and within this 2 of 22 patients with major change, 2) EEG changes usually reversed upon clamp release, and 3) the preoperative angiographic appearance of the operated vessel did not correlate with the incidence of EEG change. If hemodynamic alterations induced clamp-associated EEG changes in most of the patients of earlier reports, factors predisposing to such alterations must exist in about 10–30% of endarterectomy patients judging from the incidence of changes in other series and ours.

Features of the pre-operative clinical picture suggesting a reduced capacity for collateral flow correlated with the incidence of EEG change in this and earlier studies. Patency of the contralateral carotid artery correlated inversely with the incidence of EEG change in our study and others. Moreover, clamp-associated EEG changes in our study occurred more commonly among those whose pre-operative EEGs contained contralateral abnormalities than among those with normal, ipsilateral, or diffuse changes. We are not aware of any similar comparison in the literature although Sharbrough et al and Sundt et al found that clamp-associated EEG changes were more likely to develop when pre-operative EEGs were abnormal.

Table 2  Effect of Contra-lateral Internal Carotid Artery Patency on Incidence and Type of EEG Change

<table>
<thead>
<tr>
<th>Patency</th>
<th>Minor change</th>
<th>Major change</th>
<th>Major + minor change</th>
<th>No change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Occluded</td>
<td>7</td>
<td>6</td>
<td>13</td>
<td>7</td>
</tr>
<tr>
<td>&gt; 90% stenosed</td>
<td>11</td>
<td>6</td>
<td>17</td>
<td>8</td>
</tr>
<tr>
<td>50–90% stenosed</td>
<td>7</td>
<td>7</td>
<td>14</td>
<td>30</td>
</tr>
<tr>
<td>0–49% stenosed</td>
<td>15</td>
<td>9</td>
<td>24</td>
<td>83</td>
</tr>
<tr>
<td>stenosed, &lt; 50%</td>
<td>6</td>
<td>4</td>
<td>10</td>
<td>33</td>
</tr>
<tr>
<td>ulcer only</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>previous endarterectomy</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>13</td>
</tr>
<tr>
<td>normal</td>
<td>7</td>
<td>4</td>
<td>11</td>
<td>32</td>
</tr>
</tbody>
</table>

Comparing occluded and > 90%, 50–90%, and < 50% stenosis: any change vs no change: p < 0.005; major change vs no change: p < 0.025; minor change vs no change: p < 0.025; minor change vs major change: NS.
lular space which may further impair oxygen delivery to neurones.

As mentioned above, there is some correlation between regional cerebral blood flow (rCBF) and clamp-associated EEG changes. All of Sundt et al.'s patients with rCBF less than 10 ml/100 gm/min had clamp-associated EEG changes, but no patient with rCBF exceeding 25 ml/100 gm/min had an EEG change. Earlier, the same group had found major EEG changes at levels below 18 ml/100 gm/min, minor changes from 18-30 ml/100 gm/min and no change above 30 ml/100 gm/min. However, more than CBF must influence the EEG as Trojanbog and Boysen found that attenuation was associated with rCBF values from 11 to 19 ml/100 gm/min and increased delta at 16-23 ml/100 gm/min. These ranges indicate that other factors such as PaO2 and PaCO2 are likely involved. If such changes reflect acute alteration of cerebral function, why are they reversible and why did over 90% of our patients have no neurological deficit despite major unilateral and bilateral EEG change?

First, there appear to be differences between blood flow producing EEG change, that leading to ion changes described above, and that producing neuronal damage. Thus, studies in the baboon showed that extracellular potassium concentration in the cortex remained normal or only slightly elevated at a point when EEG activity ceased. There exists a range of perfusion values associated with loss of electrical activity which are sufficient to maintain a close to normal tissue concentration of ATP. Unfortunately, the CBF range between these levels is only moderate: the threshold for major EEG changes in man is about 17 ml/100 gm/min that for massive ionic changes in the tissue concentration of ATP. However, more than CBF must influence the EEG as Trojaborg and Boysen found that attenuation was associated with rCBF values from 11 to 19 ml/100 gm/min and increased delta at 16-23 ml/100 gm/min. These ranges indicate that other factors such as PaO2 and PaCO2 are likely involved. If such changes reflect acute alteration of cerebral function, why are they reversible and why did over 90% of our patients have no neurological deficit despite major unilateral and bilateral EEG change?

As would be expected in regional ischemia, the EEG changes appear before ion alterations. When the monkey is subjected to unilateral cerebral ischemia by complete occlusion of the blood supply to one hemisphere the ipsilateral EEG activity ceases within 20 seconds but potassium efflux appears at about 5 minutes and sodium influx occurs by 20 minutes. [In man, the rapidity of this sequence may also reflect the degree of ischemia. Sharbrough et al. found that major EEG changes would occur within 20 seconds when the regional CBF was less than 17 ml/100 gm/min but developed at 40-100 sec with flows of 17-18 ml/100 gm/min. Similarly, we found that major EEG changes were more likely to appear within 20 seconds of clamping than were moderate changes]. As such ion changes reflect ion pump (ATP) failure, such time differences provide another indication of the gap between electrical failure (EEG changes) on the one hand and ion pump and energy failure on the other.

However, as CBF values of a small percentage of patients would fall below both thresholds, this margin cannot provide an entire answer to our original question.

An additional factor is the reversibility of such changes. Hossmann et al. subjected cat cortex to ischemia for one hour and found prompt resolution of the aforementioned ion changes after restoration of blood circulation. Several authors have documented full clinical recovery in half of animals after temporarily occluding the middle cerebral arteries (MCAs) of awake monkeys for periods ranging from 15 minutes to 3 hours. Jones et al. occluded the MCA for 15-30 minutes in 9 awake monkeys and noted complete clinical recovery in 6. After 2 hours, Sundt and Michenfelder found no deficit in 3 of 6 monkeys and a mild hemiparesis in an additional 2. Six of Crowell et al.'s eleven monkeys had neurological deficits after 1-2 hours of MCA clamping and 5 fully recovered after clamp release. Unfortunately, such studies differ from the clinical situation in several significant respects: 1) collateral potential for the internal carotid artery, clamped at endarterectomy surgery, exceeds that of the middle cerebral artery, 2) longer experimental occlusion time than clamp time at endarterectomy in most studies, 3) no arteriopathy in these monkeys and therefore probably greater collateral flow, and 4) lack of general anesthesia in most experiments. Sundt et al. used Pentobarbital and found no deficit in 65% of squirrel monkeys whose MCAs were occluded for 3 hours.

The quantity of collateral flow, and therefore residual local blood flow after clamping, strongly affects reversibility. Symon and Brierley found that in chronic ischemic infarction, the area in which infarction developed corresponded to zones which had flow rates of less than 10 ml/100 gm/min immediately following acute occlusion. Conversely, recovery without histological signs of infarction occurred only when local CBF was sustained above 12 ml/100 gm/min. Marcox et al. reported that tissue damage in the MCA-occluded awake monkey occurred at higher CBF values for gray than for white matter. Median regional CBF values for histologically normal gray or white matter was 12.5 ml/100 gm/min with a range of 5-27 ml/100 gm/min while that for infarcted tissue was 6 ml/100 gm/min with a range of 3-16 ml/100 gm/min (their table 3). Degree of infarction correlated somewhat with regional CBF. In their study, implanted electrodes measured CBF by hydrogen clearance.

During MCA occlusion in squirrel monkeys, regional CBF in the core areas of ischemia ranged from 12 to 90 ml/100 gm/min, values that would generally be greater than the infarct threshold (see above) and which would explain the reversibility of neurological signs after reopening the MCA in aforementioned experimental works. However, Sundt et al. found blood flows of less than 10 ml/100 gm/min in 99 of 1104 patients (9%), probably reflecting decreased collateral flow as compared to the experimental condition. From Symon and Brierley's and Morawetz et al.'s studies one would expect that about 1 in 11 non-shunted patients would awake with a permanent neurological deficit. Why this does not happen (see below) is not clear, but some theories can be offered.
First, the blood flow measurements of Sundt et al\textsuperscript{2} were done within a few minutes of clamping the internal carotid artery; Sundt and Waltz\textsuperscript{28} found that some recovery of local CBF occurred after 15 minutes of clamping the MCA of squirrel monkeys. Such recovery in humans might elevate the CBF above the infarction threshold before irreversible cellular changes occur. This effect would be compatible with our finding that reversibility of EEG changes bore no relationship to duration of ICA occlusion.

General anesthesia itself is a second possible factor. The lowered cerebral metabolic rate by inhalation anesthetics such as Halothane, Enflurane and Isoflurane, and possibly nitrous oxide, and by narcotics\textsuperscript{29} alters carbohydrate metabolism such that brain glucose and glycogen levels rise and lactic acid concentrations fall. Such alterations would reduce the damaging effects of ischemia. Moreover, it is possible that cellular lactic acidosis is one of the critical factors causing cerebral death.\textsuperscript{23} Additional factors must be involved, however, as Halothane lowers CMRO\textsubscript{2}, but fails to reduce infarction from MCA occlusion.\textsuperscript{30,31} Moreover, agents acting as vasoconstrictors (narcotics)\textsuperscript{32} or as weak vasodilators (Isoflurane) were associated with a lower incidence of EEG change during internal carotid artery clamping in this study than cerebral vasodilators (Enflurane and especially Halothane).\textsuperscript{37} Although Halothane and Enflurane increase CBF, this effect may "steal" blood from less vaso-responsive ischemic areas and thereby lower CBF in such regions.\textsuperscript{31,33,34}

Thirdly, moderate ischemic levels may be tolerated for limited periods. Using microelectrode recordings of single cell activity, Heiss and Rosner\textsuperscript{35} studied the functional recovery of cortical neurones after MCA occlusion in anaesthetised cats. They found that post ischemic viability was determined not only by residual flow but also by duration of ischemia. For example, their data suggests that for an occlusion time of 30 minutes the threshold blood flow for functional and histological damage would be about 5 ml/100 gm/min. (their fig. 5). Sundt et al\textsuperscript{2} found values below this threshold in only 25 of their 1104 patients (2.26%) with CBF measurements.

Finally, it must be considered that small infarcts which escape clinical and laboratory detection could occur and that such may be due to insufficient blood flow or to embolism. Blair and Waltz\textsuperscript{26} reported the significant variability of regional CBF following MCA occlusion; such heterogeneity may result in patchy or variable degrees of cortical damage. For example, Tamura et al\textsuperscript{37} found that ischemic changes from MCA occlusion were most marked at the depths of sulci. Marcoux et al\textsuperscript{27} recorded the lowest level of regional cerebral blood flow in the putamen. Blair and Waltz\textsuperscript{26} found that the degree and extent of ischemia in deeper regions of the brain failed to correlate with more superficial alterations. These data suggest that the EEG may not always accurately portray the cerebral condition, particularly if significant alterations are confined to depths of sulci or to subcortical grey matter. This variability may partially explain the similarity of clamp-associated EEG changes of the two patients with post-operative deficits to those of many others without any apparent complication.

Conceivably, small infarcts could occur from any of the currently used methods of endarterectomy. Our data suggest that a clamp-associated major change indicates about a 10% chance of a post-operative deficit occurring, but that a moderate EEG change carries no greater risk of post-operative deficit than no change. Unfortunately, no aspect of such major changes determined which patients would awake with a deficit.

If EEG changes indicate which patients (if any) need a shunt, then series of patients using EEG in this manner should have lower complication rates than those in which a shunt is never used, with or without monitoring. Such is not the case.\textsuperscript{38} The complication rate for most series in which no shunts were used\textsuperscript{27} was less than in those where they were selectively employed on the basis of EEG changes.\textsuperscript{1,2} As such EEG changes almost always recede when a shunt is placed,\textsuperscript{2} they must usually be related to lowered regional cerebral blood flow and rarely to embolism whereas embolism is thought to be the cause of all intra-operative strokes.\textsuperscript{39} The incidence of such clamp-related EEG changes ranges from about 10–30% (1,2, this series); therefore, a substantial number of patients in series done without shunts must have tolerated regional CBF reductions sufficient to give EEG changes had monitoring been done. Ours is not the only series reporting such tolerance. Chiappa et al,\textsuperscript{1} advocates of EEG-related selective shunting, documented two patients who tolerated clamp-associated major EEG changes for 13 and 24 minutes without post-operative deficit. Sharbrough et al,\textsuperscript{1} using a similar approach, reported a patient who sustained a major EEG change for 10 minutes without a post-operative deficit. Such patients may be exceptions only because of the procedures these centres follow. Finally, of nine post-operative deficits reported by Chiappa et al,\textsuperscript{1} only two had clamp-associated EEG changes.

The foregoing therefore suggests that although the majority of clamp-associated EEG changes are related to lowered regional CBF, the post-operative deficits are usually caused by embolism. It would be expected that the incidence of post-operative complications would always be slightly higher among patients with clamp-associated EEG changes, as in the present series, as these will inevitably include a few with embolism, not helped by a shunt.

If only major clamp-associated EEG changes, as defined, are acted upon; the incidence of shunt use, with its attendant risks, would drop from about one-third to one-eighth of cases. Using EEG in this revised manner still does not establish its value: further experience may show that a major change indicates some incidence of post-operative deficit whether or not a shunt is placed. Pre-operative data, as suggested in this paper, might then be more valuable in predicting post-operative deficit than intra-operative EEG monitoring.
Acknowledgments

The authors wish to thank Dr. J. K. Farrar for performing the Cerebral Blood Flow Studies and Mrs. Maria Raffa and Mrs. Frances Dyson for careful preparation of the manuscript.

References

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W T Blume, G G Ferguson and D K McNeill

Stroke. 1986;17:891-897
doi: 10.1161/01.STR.17.5.891

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1986 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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