RELATION BETWEEN EEG, REGIONAL CEREBRAL BLOOD FLOW AND INTERNAL CAROTID ARTERY PRESSURE DURING CAROTID ENDARTERECTOMY

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Continuous EEG recording has been used to estimate the adequacy of cerebral perfusion during a test occlusion of the internal carotid artery, and to determine whether or not temporary by-pass shunt is necessary during carotid endarterectomy (Wells et al. 1963; Perez-Borja and Meyer 1965; Spunda 1966; Harris et al. 1967; Radvanyi and Garnier 1967; Galbraith 1968; Marshall and Longhead 1969). Slowing of the EEG during clamping of the carotid artery was taken to indicate cerebral ischaemia and occurred when the mean jugular venous oxygen tension fell to less than 26 mm Hg (Meyer et al. 1965). This is only a rough estimate of regional blood flow since about one-third of the venous jugular blood is derived from the opposite half of the brain. Pressure in the stump of the internal carotid artery (ICA) is a more reliable indication of blood flow (Moore and Hall 1969; Ehrenfeld et al. 1970; Fourcade et al. 1970) and has been related to measurements of regional cerebral blood flow (rCBF) by intra-arterial injection of $^{133}$Xe (Boysen et al. 1971, 1972).

It was the purpose of the study presented here to relate manual EEG frequency analysis in patients with stenotic lesions of the ICAs to changes in rCBF and ICA blood pressure before and during a test occlusion of the carotid artery. These procedures were performed during reconstructive surgery under general anaesthesia to establish the value of each of them as an indicator of cerebral ischaemia.

MATERIAL AND METHODS

There were 42 men and 10 women, 51–72 years old. The 52 patients were selected for carotid endarterectomy because of stenosis or occlusion of both of the ICAs. Cerebral ischaemia had been transient in 19 patients and had left neurological sequelae in 33 patients (Table I). Stenosis was unilateral in 35 patients (in 13 of these angiography was performed on one side only), and bilateral in 10 patients; 7 had stenosis on one side and occlusion on the other side. One patient was operated on both sides in two stages. Operation was not performed when the artery was totally occluded.

The operations were performed under general anaesthesia. After induction with a short-acting barbiturate, anaesthesia was maintained by Halothane 1% or less in oxygen 50%–nitrous oxide 50%. The patients were ventilated by a

<p>| TABLE I |
|-----------------|-----------------|-----------------|
| Symptoms and signs of cerebral involvement in 52 patients with stenosis or occlusion or both of the internal carotid arteries. |</p>
<table>
<thead>
<tr>
<th>n</th>
<th>Transitory</th>
<th>Manifest</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unilateral stenosis</td>
<td>35</td>
<td>10</td>
</tr>
<tr>
<td>Bilateral stenosis</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>Unilateral stenosis with contralateral occlusion</td>
<td>7</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>52</td>
<td>19</td>
</tr>
</tbody>
</table>
non-rebreathing respirator. The systemic blood pressure was recorded continuously by a Statham pressure transducer attached to a catheter in the radial artery. Similarly the pressure was measured in the ICA above the site of the stenosis. The arterial blood pressure was maintained at the pre-operative level by infusion of angiotensin amide (2 μg/ml) unless otherwise stated.

The EEGs were recorded on an 8-channel Kaiser electroencephalograph and were performed about one week pre-operatively, during surgery, and about one week post-operatively. Fourteen platinum needle electrodes were placed symmetrically over both frontal, central, anterior temporal, mid-temporal, posterior temporal, parietal and occipital regions and one electrode was placed over the vertex in the midline. Recordings were bipolar, to an electrode over the vertex and to an average from all electrodes. One channel was used to monitor the electrocardiogram.

Manual frequency analysis was by the method described by Sulg (1969). It consisted of determining, by a special ruler, the frequency of all waves with amplitudes over 5 μV between 1 and 20 c/sec. The percentage of time occupied by each frequency class was determined and the mean frequency (m.f.) was calculated from the frequency distribution graph. Since EEG abnormalities, when they occurred, were mainly localized to the fronto-central or temporo-central regions, these montages were measured for 20–30 sec. The analysis included the waking record before operation, the record during general anaesthesia immediately before and at the end of a 2 min test occlusion. The m.f. was calculated for each of the epochs analysed and was compared with the rCBF and ICA pressure obtained simultaneously.

The rCBF was determined according to the method of Høedt-Rasmussen et al. (1966) using intra-arterial injection of $^{133}$Xe with external recording of the clearance by either one uncollimated or 14–16 collimated scintillation detectors. The rCBF was calculated from the initial slope of the efflux on a semilogarithmic plot (Boysen 1971; Boysen et al. 1971). A mean value for the hemisphere was calculated from the regional values and was compared with the m.f.

The ICA pressure and the rCBF were determined simultaneously before and during a 2 min test occlusion of the common and external carotid arteries. During the test occlusion, clamping of the arteries was done as soon as the clearance curve had reached its peak, thus ensuring that no tracer got trapped in the ICA. Since the clearance curve in 6 patients showed a plateau 20–60 sec after the test occlusion, the second minute was used to establish the slope.

Simultaneously with the flow measurements, arterial blood samples were drawn to determine the arterial PCO₂ (PaCO₂) by radiometer equipment. Since both hypo- and hypercapnia cause slowing of the EEG and changes in rCBF (Kety and Schmidt 1948; Clowes et al. 1953; Alexander et al. 1964; Gotoh et al. 1965) these parameters were compared when the PaCO₂ was constant, except when otherwise stated.

RESULTS

1. Pre- and post-operative EEG findings

Nearly two-thirds of the 52 patients had a normal EEG. Of the 21 patients with an abnormal EEG, 19 were examined less than 2 months after the onset of symptoms. The abnormalities were: (i) focal slowing in 16 patients, localized mainly to the frontal, central and temporal regions and consisting of delta or theta activity or both; (ii) an increased admixture of theta activity in 5, occurring bilaterally, either over the fronto-temporal or temporo-occipital leads. Focal abnormalities were more frequent in patients with focal cerebral signs than in those without and correlated well with clinical findings. Diffuse EEG changes occurred equally often in both groups (Table II).

There were only minor differences between pre- and post-operative EEG findings. Three patients who had a normal EEG developed abnormalities; 2 showed slight bilateral slowing and one had marked focal slowing. In the latter patient focal abnormalities developed during operation before clamping of the carotid arteries. He woke up after the operation with a hemiparesis corresponding to the side of slowing, probably due to an embolus loosened from the arteriosclerotic plaques by manipulation of the carotid artery. Five patients with abnormalities before had normal EEGs after the operation.
TABLE II

EEG findings in 52 patients before and after carotid endarterectomy.

<table>
<thead>
<tr>
<th>Signs and symptoms of cerebral involvement</th>
<th>n</th>
<th>EEG findings</th>
<th></th>
<th>Abnormal</th>
<th></th>
<th>Abnormal</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Pre-operative</td>
<td>Normal</td>
<td>Focal</td>
<td>Diffuse</td>
<td>Normal</td>
</tr>
<tr>
<td>Transitory 19</td>
<td>13</td>
<td>4</td>
<td>2</td>
<td>11</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>Manifest 33</td>
<td>18</td>
<td>12</td>
<td>3</td>
<td>17</td>
<td>12</td>
<td>4</td>
</tr>
<tr>
<td>Total 52</td>
<td>31</td>
<td>16</td>
<td>5</td>
<td>28</td>
<td>17</td>
<td>7</td>
</tr>
</tbody>
</table>

2. EEG findings during general anaesthesia

An increase of PaCO₂ causes slowing in the EEG (Fig. 1, Clowes et al. 1953). As long as the PaCO₂ was kept at a normal level neither direct observation nor m.f. showed any difference in the EEG during general anaesthesia and the waking state (Fig. 2 and 4). One patient was an exception: he had focal slowing over the fronto- centro-temporal regions, an m.f. of 6.8 ± 0.9 c/

Fig. 1. Illustrates the effect of carbon dioxide accumulation on the EEG (above) during general anaesthesia in a 63-year-old man with stenosis of the left internal carotid artery. Below, graphs of EEG frequencies. Arrows indicate the m.f. (1) PaCO₂ = 35 mm Hg, m.f. 10.2 ± 1.1 c/sec; (2) PaCO₂ = 55 mm Hg, m.f. 6.0 ± 1.3 c/sec. In this and following figures: F = frontal; C = central; T = temporal; P = parietal; O = occipital; L = left; R = right; ECG = electrocardiogram.

sec¹ in the waking record and 3.7 ± 0.7 c/sec during general anaesthesia. This difference was probably due to a deeper level of anaesthesia.

3. EEG findings during a 2 min test occlusion of the carotid arteries.

Changes were observed in 14 patients 20–40

Fig. 2. EEGs in the same patient as in Fig. 1 in whom the rCBF fell from 30 to 16 ml/100 g/min during the test occlusion. (1) Waking record, m.f. 11.3 ± 1.0 c/sec; (2) during general anaesthesia, m.f. 10.2 ± 1.0 c/sec, PaCO₂ = 35 mm Hg; (3) 1 min after test occlusion of the left carotid arteries, m.f. 4.6 ± 1.2 c/sec, PaCO₂ = 32 mm Hg; (4) 30 sec after removal of the clamp.

¹ Here and elsewhere ± indicates the mean error of the S.D.
sec after the clamping and consisted of flattening or slowing of cortical activity or both (Fig. 2 and 3). In one patient marked flattening was present only 8 sec after clamping (Fig. 3) but in another changes did not occur until after 160 sec. The EEG changes occurred suddenly and were not accompanied by changes in heart rate or systemic blood pressure. After removal of the clamp, cortical activity reappeared within 5–30 sec. EEG changes during the test occlusion were equally frequent among patients with clinical sequels of cerebrovascular attacks as among those without. They occurred in 5 of 23 patients with a one-sided stenosis of the carotid arteries (22%) and in 5 of 17 patients with a bilateral affection (29%); in 4 patients angiography was performed on one side only.

The EEG changes were confined to the hemisphere ipsilateral to the clamping and were distinguished from changes due to variation in PaCO_{2}, since the change with the depth of anaesthesia was bilateral.

4. Relation between EEG and haemodynamic changes

The patients were divided into three groups according to the changes in rCBF during the test occlusion. The PaCO_{2} was the same before and during the test occlusion and averaged 35 mm Hg in groups I and III, and 41 mm Hg in group II.

Group I comprised 17 patients in whom the average rCBF was diminished from 36 to 16 ml/100 g/min ($P < 0.001$, Wilcoxon’s test for pair differences), and the average ICA pressure from 96 to 36 mm Hg ($P < 0.001$). There were marked changes in the EEG in 14 patients, in 10 consisting of flattening to a degree that frequency analysis was not possible, and in 4 consisting of slowing. In the patients with flattening of cortical activity during the test occlusion the rCBF averaged 14 ml/100 g/min and in those with slowing it averaged 18 ml/100 g/min (Table III). In 6 of the patients with EEG flattening the isotope clearance curve showed a plateau for 20–60 sec after the test occlusion before it declined, indicating poor collateral circulation. The EEG was unaffected by the haemodynamic changes in 3 patients in whom the rCBF averaged 20 ml/100 g/min (Table III, Fig. 6).
Fig. 5. Relation between rCBF and internal carotid artery pressure above the stenosis before and during a test occlusion in 52 patients in whom 53 endarterectomies were performed. (I) 17 patients in whom the average rCBF fell from 36 to 16 ml/100 g/min (56%); (II) 16 patients in whom the average rCBF fell from 49 to 35 ml/100 g/min (29%); (III) 20 patients without changes in rCBF before and during the test occlusion. ○ flattening of the EEG; ▼ slowing of the EEG.

TABLE III
EEG findings in 17 patients with pronounced haemodynamic changes during clamping of the carotid artery.

<table>
<thead>
<tr>
<th>EEG findings</th>
<th>Mainly flattening (n = 10)</th>
<th>Mainly slowing (n = 4)</th>
<th>No changes (n = 3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>rCBF (ml/100 g/min)</td>
<td>mean 14</td>
<td>range 11-19</td>
<td>mean 18</td>
</tr>
<tr>
<td>ICA stump pressure (mm Hg)</td>
<td>mean 32</td>
<td>range 15-36</td>
<td>mean 38</td>
</tr>
<tr>
<td>PaCO₂ (mm Hg)</td>
<td>mean 36</td>
<td>range 28-54</td>
<td>mean 33</td>
</tr>
</tbody>
</table>

Group II comprised 16 patients (including one patient from group III who was operated on both sides) in whom the rCBF was diminished from 49 to 35 ml/100 g/min (P < 0.001). The average ICA pressure fell from 98 to 67 mm Hg (P < 0.001). Although the reduction in the rCBF was more than 30% in 6 patients, the EEG remained unchanged. One patient, as mentioned above, developed focal slowing on the side of the operation before the test occlusion when the rCBF was 78 ml/100 g/min. During the test occlusion no further EEG changes occurred although the rCBF fell to 38 ml/100 g/min and the ICA pressure dropped from 117 to 56 mm Hg.

Group III comprised 20 patients in whom the average rCBF was the same before and during the test occlusion (31 ml/100 g/min). The average ICA pressure fell from 99 to 78 mm Hg (P < 0.001). The m.f. before and during clamping was the same (Fig. 4 and 5).

5. Effect of induced hypercapnia and hypertension on EEG, rCBF and ICA pressure
In 7 of the 14 patients with EEG changes during the test occlusion a second test occlusion
TABLE IV
Effect of induced hypercapnia and hypertension on rCBF, ICA stump pressure and EEG during a second test occlusion in 7 patients (a–g).

<table>
<thead>
<tr>
<th>Subject</th>
<th>First test occlusion</th>
<th>Second test occlusion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PaCO₂* rCBF** ICA*** EEG</td>
<td>PaCO₂ rCBF ICA EEG</td>
</tr>
<tr>
<td>Hypercapnia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>a</td>
<td>35 16 29 slow</td>
<td>55 8 21 flat</td>
</tr>
<tr>
<td>b</td>
<td>28 18 35 flat</td>
<td>53 25 30 —</td>
</tr>
<tr>
<td>c</td>
<td>MABP† rCBF ICA EEG</td>
<td>MABP rCBF ICA EEG</td>
</tr>
<tr>
<td>d</td>
<td>125 20 46 slow</td>
<td>147 22 50 flat</td>
</tr>
<tr>
<td>e</td>
<td>109 13 27 flat</td>
<td>145 13 30 flat</td>
</tr>
<tr>
<td>f</td>
<td>119 11 28 flat</td>
<td>131 15 32 flat</td>
</tr>
<tr>
<td>g</td>
<td>115 20 46 slow</td>
<td>147 21 55 —</td>
</tr>
<tr>
<td></td>
<td>99 16 40 slow</td>
<td>111 17 48 —</td>
</tr>
</tbody>
</table>

* In mm Hg.
** In ml/100 g/min.
*** Stump pressure in mm Hg.
† Mean systemic arterial blood pressure in mm Hg.
— = No change.

was performed, in 2 during hypercapnia (PaCO₂ 53–55 mm Hg) and in 5 during hypertension (mean arterial blood pressure increased from an average of 113 to 136 mm Hg). The values of rCBF and ICA stump pressure thus obtained were compared with those obtained during the first test occlusion (Table IV).

During hypercapnia the second test occlusion further reduced the rCBF from 16 to 8 ml/100 g/min in one patient. The slowing of the EEG present during the first test occlusion was more marked and was accompanied by flattening during the second test occlusion. In the other patient with flattening during the first test occlusion hypercapnia had the opposite effect. The rCBF increased from 18 to 25 ml/100 g/min and there was no flattening. The EEG showed diffuse bilateral slowing due to accumulation of carbon dioxide.

Induced hypertensive had little or no effect on rCBF, ICA stump pressure or EEG during a second test occlusion in 3 patients, whereas the ICA stump pressure rose from 46 to 55 mm Hg in one, and from 40 to 48 mm Hg in another. The rCBF remained unaltered but the slowing of the EEG seen during the first test occlusion disappeared during the second.

In summary, EEG abnormalities appeared when the rCBF fell below 23 ml/100 g/min and the ICA stump pressure to 50 mm Hg. Slowing was seen when the rCBF was 16–22 ml/100 g/min and the ICA stump pressure was 29–50 mm Hg. Flattening occurred when the rCBF was 11–19 ml/100 g/min and the ICA stump pressure was 15–46 mm Hg.

DISCUSSION

According to previous reports the EEG during light Halothane anaesthesia combined with nitrous oxide consists of low voltage 15–20 c/sec activity with some admixture of
5–6 c/sec rhythms with an amplitude of 50 μV and is clearly distinguishable from the waking record (Gain and Paletz 1957; Sadove et al. 1967). In our patients the EEG recorded during light anaesthesia did not differ from that in the waking state provided the PaCO₂ was kept at a normal level. An increased admixture of theta or delta activity or both was observed during hypercapnia, as described by Clowes et al. (1953), probably due to a cumulative effect of CO₂ on the anaesthetic agents.

Before clamping of the carotid artery during general anaesthesia the rCBF averaged 36 ml/100 g/min (PaCO₂ = 36 mm Hg) similar to that found by Meyer et al. (1967) in conscious patients with cerebrovascular disorders. During a 2 min test occlusion the rCBF fell significantly in 33 patients but EEG changes occurred in only 14. When the EEG is unaltered in spite of a fall in rCBF one might assume that the reduction in flow is counteracted by an increased oxygen extraction to maintain a constant metabolic rate for oxygen (CMRO₂). On the other hand, when the rCBF fell below 23 ml/100 g/min oxygen extraction was probably insufficient, resulting in a lowering of CMRO₂ and slowing or flattening of cortical activity. However, the critical level of CMRO₂ for maintenance of cortical activity in man is not known. When the rCBF was diminished to 16–22 ml/100 g/min during the test occlusion there was slowing of the EEG and when it was diminished to 11–19 ml/100 g/min flattening of cortical activity occurred. It is well known that hypoxia causes flattening of the EEG and as anoxia progresses it is followed by flattening of cortical activity (for references see Bokonjic 1963). Consistently we found the lowest value of rCBF in patients with marked flattening of cortical activity. The determination of rCBF at the same time showed a plateau of the isotope clearance curve immediately after the carotid artery was occluded. This initial plateau probably reflects an almost complete arrest of tissue perfusion due to delayed autoregulatory vasodilatation.

The ICA pressure above the clamp is a measure of the collateral circulation (Moore and Hall 1969; Boysen et al. 1971, 1972). When the ICA stump pressure fell to 29–50 mm Hg, slowing of the EEG occurred; and when it fell to 15–46 mm Hg flattening of the cortical activity was seen. The relatively high arterial pressure needed to maintain a sufficient cerebral perfusion is probably due to a moderate elevation of intracranial pressure during the Halothane anaesthesia. During this anaesthesia the cerebral venous pressure was 15–25 mm Hg, indicating that cerebral perfusion pressure was only one-third to one-half the stump pressure (Fourcade et al. 1970).

The EEG, the rCBF and the ICA stump pressure all give information as to collateral circulation during a test occlusion of the carotid artery. Measurement of the stump pressure is by far the simplest and most easily applied of the tests and can be used to indicate whether or not a by-pass shunt is needed to avoid cerebral ischaemia.

The interindividual relationship between rCBF and EEG mean frequency (m.f.) found by Ingvar et al. 1965, Baldy-Moulinier and Ingvar 1968 and Ingvar and Sulg 1969 was not confirmed in this study (Fig. 7). Moreover, in 8 patients the rCBF was reduced more than 30% during the test occlusion without significant changes in m.f. (Fig. 5). The lack of correlation in this study was not due to differences in PaCO₂ level before and during clamping, nor was it an effect of the anaesthesia since the m.f. was the same before and during anaesthesia with un-

![Fig. 7. Relation between rCBF and mean frequency (m.f.) in 26 patients: 9 from group I, 6 from group II and 11 from group III. Patients studied in a hypercapnic state were not included. • before (N = 26, mean PaCO₂ 33 ± 1.2 mm Hg), and ○ during a 2 min test occlusion (N = 23, mean PaCO₂ 34 ± 1.3 mm Hg). The dashed line is the calculated linear regression (r = 0.20, P > 0.1). It was the same whether considering the values before clamping separately or those during clamping alone.](image-url)
clamped arteries (Fig. 4). However, about two thirds of the patients had neurological symptoms and signs of focal cerebral damage, which is known to abolish the EEG–rCBF relation (Ingvar and Sulg 1969). The lack of correlation in the individual patients (closed circles, Fig. 7) is probably due to constancy of cerebral metabolic rate in spite of an even considerable reduction of rCBF induced by clamping. Thus, changes in rCBF were not reflected in the EEG unless the cerebral blood flow was reduced below a critical level.

SUMMARY

The EEG was recorded in 52 patients with cerebrovascular disorders selected for reconstructive surgery due to stenosis of the carotid arteries. Changes in cortical activity during 2 min test occlusion of the carotid arteries were related to regional cerebral blood flow and internal carotid artery pressure.

The test occlusion induced flattening or slowing of the EEG or both in 14 patients. Flattening occurred when the cerebral blood flow fell to 11–19 ml/100 g/min and when the pressure in the internal carotid artery above the stenosis fell to 15–46 mm Hg. Slowing of the EEG was seen when the regional cerebral blood flow fell to 16–22 ml/100 g/min and the internal carotid artery pressure to 29–50 mm Hg.

Measurement of the pressure in the stump of the internal carotid artery is an easy method and is a reliable measure of collateral circulation. It serves as a useful guide to whether or not a temporary by-pass shunt is needed to avoid cerebral ischaemia.

Manual frequency analysis of the EEG failed to demonstrate any inter- or intra-individual relation between cerebral blood flow and the mean frequency, probably because of focal cerebral anoxia induced either by the stroke or by the test occlusion.

RESUME

RELATION ENTRE L’EEG, LE DEBIT SANGUIN CEREBRAL REGIONAL ET LA PRESSION ARTERIELLE DE LA CAROTIDE INTERNE AU COURS DE L’ENDARTECTOMIE CAROTIDienne

L’EEG a été enregistré chez 52 malades présen
tant des désordres vasculaires cérébraux dus à une sténose des artères carotides et sélectionnés pour chirurgie réparatrice. Des modifications de l’activité corticale au cours d’un test de 2 min d’occlusion des artères carotidiennes ont été mises en relation avec le débit sanguin cérébral régional et la pression artérielle carotidienne interne.

Chez 14 malades, le test d’occlusion induit soit un aplatissement, soit un ralentissement de l’EEG, soit les deux. L’aplatissement survient quand le débit sanguin cérébral tombe à 11–19 ml/100 g/min et quand la pression dans l’artère carotidienne interne au-dessus de la sténose tombe à 16–46 mm Hg. Le ralentissement de l’EEG s’observe quand le débit sanguin cérébral régional tombe à 16–22 ml/100 g/min et quand la pression de l’artère carotidienne interne tombe à 29–50 mm Hg.

La mesure de la pression dans le moignon de l’artère carotidienne interne est facile et réalise une mesure fidèle de la circulation collatérale. Elle constitue un guide utile pour savoir si un court-circuit temporaire par shunt est nécessaire pour éviter l’ischémie cérébrale.

L’analyse de fréquence manuelle de l’EEG n’a pas montré de relation inter- ou intra-individuelle entre le débit sanguin cérébral et la fréquence moyenne périodique.

REFERENCES


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