EEG and Arteriographical Findings in Carotid Artery Transient Ischemic Attacks (TIAs)

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Abstract:
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Thirty patients over 35 years of age with one or more episodes of carotid TIA lasting less than one hour were studied. No patient had a history of brain disease and each had an EEG followed by an arteriogram. Arteriographical study showed 15 patients to have occlusive disease of the internal carotid artery, and four of these had intracranial occlusive arterial disease as well. An additional three patients had only intracranial occlusive arterial disease. Twelve patients had normal arteriographical studies. The EEGs of nine patients were diffusely or focally slow. Twenty-one patients had normal records. Six of the nine patients with abnormal EEGs had arteriographical evidence of intracranial occlusive arterial disease. Only one patient with extracranial carotid stenosis alone showed an EEG abnormality. In two patients with an abnormal EEG the arteriogram was completely normal or showed insignificant change. These data indicate that the EEG has the capacity to reflect changes in cerebral function not clinically evident in patients with carotid TIAs. Such changes were present in almost one-third of the patients in this study. Most commonly, generalized or focal EEG slowing in patients with the clinical syndrome of carotid TIA is associated with the presence of intracranial occlusive arterial disease. Alternatively, the finding of a normal EEG in such patients would suggest the absence of intracranial occlusive arterial disease, but occurs with equal frequency in patients with stenosis of neck vessels and in patients with no arteriographically demonstrable lesion.

**Additional Key Words**
carotid artery occlusion  
carotid arteriogram  
intracranial arterial occlusion  
carotid artery stenosis

**Introduction**

The purpose of this investigation is to evaluate the usefulness of the EEG in the study of patients with transient ischemic attacks (TIAs) within the distribution of the internal carotid artery. The specific objectives of the study were: (1) to determine whether the EEG could demonstrate cerebral dysfunction in patients with carotid TIAs, which was not apparent from analysis of neurological symptoms and findings, and to consider the pathophysiological mechanisms by which such EEG changes might be produced, (2) to ascertain whether EEG findings were of positive or negative value in predicting the presence of significant angiographically demonstrable extracranial carotid stenosis, and (3) to compare the results of this study with those previously reported.

**Methods**

The records of 200 patients discharged from various services of the St. Louis University Hospitals during the years 1969 through 1971 with a diagnosis of transient ischemic attacks or cerebral vascular insufficiency were examined. Patients were selected for inclusion in the study using five criteria: (1) age over 35 years, (2) no history of brain disease, (3) one or more bouts of neurological symptoms which could be interpreted as an effect of ischemia within the distribution of the internal carotid artery including unilateral sensory or motor symptoms, aphasia, and amaurosis fugax singly or in any combination, (4) attacks of less than one hour in duration without residual symptoms or neurological deficit, and (5) the patients must have had an electroencephalogram followed by an arteriogram as part of their evaluation. Thirty of the 200 patients satisfied these criteria. All of the patients ultimately included in the study were from the neurological or neurosurgical service and had been examined by a neurologist or a neurosurgeon, or both.

EEGs were made on an eight-channel Model 6 Grass machine using needle or disk electrodes placed...
bilaterally in a frontal, central, occipital, anterior temporal, and midtemporal location. Some records were taken with electrodes placed according to the International 10 to 20 system. Monopolar and bipolar recording techniques were employed. Patients were maintained awake during the recording, and at least 18 minutes of recording time was studied on each patient. Hyperventilation for three minutes was the only activating technique employed. All patients were free of symptoms during the EEG. Records were classified as normal, generally slow, and focally slow. A record was classified as generally slow if more than 20% of the EEG activity from both hemispheres was less than 8 hertz (Hz) in the bipolar record. Records were classified as focally slow if they showed an asymmetry from the homologous contralateral area with activity of less than 6 Hz.

Angiograms were done by injection of the ascending aorta by brachial or femoral catheter. Films were taken at one-half to two-second intervals over five to ten seconds. Films of the extracranial and intracranial portions of the carotid artery were studied. Carotid patency was evaluated in an AP and a lateral view, and the maximal degree of stenosis in either view was noted. Carotid patency was classified as normal, less than 50% stenosis, 50% to 75% stenosis, greater than 75% stenosis, and total occlusion. Intracranial vessels were evaluated in AP and lateral views. The criteria for significant intracranial occlusive disease were absence of vessels within a major arterial distribution and/or reversal of flow within intracranial vessels.

Results

Four of the 30 patients were between the ages of 35 to 44, eight were between ages 45 to 54, nine between ages 55 to 64, and nine between ages 65 to 75. Eight patients had experienced a single attack, 12 had two to four attacks and ten had more than four attacks. Sixteen patients had been symptomatic longer than three months.

Angiographical studies, as listed in table 1, revealed three patients to have total internal carotid artery occlusion. Seven patients had greater than 75% stenosis of one internal carotid, two patients had 50% to 75% stenosis of one internal carotid artery, and three patients had less than 50% stenosis of one internal carotid artery. All of these carotid lesions were appropriate in laterality to clinical symptoms. Two of these patients had less than 25% stenosis of the opposite internal carotid artery. Seven patients showed evidence of intracranial arterial occlusion with or without an associated carotid stenosis in the neck. Twelve patients had no angiographical abnormality.

A total of 21 patients had normal EEGs. Seven patients showed some degree of generalized EEG slowing. Three of these were mild in degree as indicated by the broken lines in figure 1. In four cases the slowing was moderate to severe in degree as indicated by the broken lines in figure 2. Two patients showed focal slowing of their EEG contralateral to the side of clinical symptoms. One 73-year-old patient showed single and serial 3.3 to 1.25 Hz activity in the anterior and midtemporal linkages with an average frequency of 1.1 times per ten seconds. One 58-year-old patient showed single 2.5 to 1.5 Hz activity in the anterior and midtemporal linkages with an average frequency of 0.9 times every ten seconds.

Ten of the 15 patients with extracranial carotid occlusive disease had neck bruits. All of the patients with abnormal EEGs had a brain scan which was normal in each instance. Twenty patients had brachial diastolic blood pressures between 90 to 100 mm Hg, three between 101 and 110 mm Hg, and one of 140 mm Hg. All four of the patients with diastolic blood pressures in excess of 100 had normal EEGs. No patient was anemic or polycythemic. Five patients had EKG evidence of old myocardial infarction, but no patient had clinical or EKG evidence of acute heart disease. Aside from one patient who showed ventricular premature

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<thead>
<tr>
<th>Arteriographical findings</th>
<th>Intracranial arteries</th>
<th>EEG findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Internal carotid artery</td>
<td>Focal slow</td>
<td>Generalized slow</td>
</tr>
<tr>
<td>Total occlusion</td>
<td>Normal (3)</td>
<td>1</td>
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<td></td>
<td>Occlusion (0)</td>
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<td>75% + stenosis</td>
<td>Normal (3)</td>
<td>2</td>
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<td>Occlusion (4)</td>
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<td>50 to 75% stenosis</td>
<td>Normal (2)</td>
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EEG AND ARTERIOGRAPHICAL FINDINGS IN TIAs

broken lines are a manual EEG frequency analysis of a representative ten-second epoch of right occipital to central linkage in three patients with mild generalized slowing. Solid line is a control group from Obrist and Henry (see text).

contractions, no patient had abnormalities of cardiac rhythm. None of the patients with abnormal EEGs had evidence of a metabolic abnormality, except for two patients who were mild diabetics controlled by diet. No patient was on drugs having an effect on the EEG.

Discussion

Millikan and Siekert defined the syndrome of “carotid insufficiency” as consisting of “intermittent attacks of unilateral impairment of sensory or motor function, or both, in certain instances associated with a disorder of speech, involvement of vision homolateral to the artery affected, or both (and) the patient returns to normal following each episode.” The NINDB ad hoc Committee on Cerebrovascular Diseases suggested a time limit of one hour in duration for such attacks. All of the 30 patients in this study fulfill these diagnostic criteria. Marshall extended the time limit for transient ischemic attacks to 24 hours, though only two of his 158 patients had attacks “anywhere approaching this duration.” Subsequent series are about equally divided between those using the one-hour and 24-hour time limit for TIAs. The one-hour time limit was chosen for this study since it corresponds closely with the clinical course of most patients, and since it seemed less likely to reflect cerebral infarction.

The clinical features of the patients being reported upon from the standpoint of patient age, length of neurological history, frequency of TIAs and incidence of hypertension and EKG abnormalities are comparable to the series reported by Marshall, Drake and Drake and Friedman et al. The incidence of neck bruits in these patients is similar to the series reported by Shapiro et al. Angiographical findings in patients with carotid TIAs vary greatly among reported series. Burrows and Marshall first reported on the angiographical findings in patients having exclusively carotid TIAs and found one-fifth of their patients to have occlusive extracranial carotid disease and almost one-half of the patients to have normal neck vessels. Conversely, Janeway and Toole, in a recent report, found more than one-half of patients with carotid TIAs to have greater than 50% stenosis of the carotid in the neck and only one-fifth of patients to have normal neck vessels. Only two of the 35 patients reported by Burrows and Marshall had intracranial occlusive arterial disease; however, Acheson et al. found five instances of intracranial occlusive arterial disease in 30 patients with carotid TIAs. One-fifth of the patients reported here had evidence of intracranial arterial occlusive disease.

The EEG findings in patients with TIAs in the carotid distribution have been the subject of four prior reports. The series of Meyer et al., Paddison and Ferriss, Kreindler et al., and Mizuno and Hughes include a total of 180 such patients. The findings include 44 patients with normal records, 86 patients with focal slowing, 15 patients with generalized slowing and 35 patients upon whom data are not given. Twenty-four patients in the series of Mizuno and Hughes showed “epileptiform discharges” in one or both temporal areas, usually during sleep. Angiographical correlation with EEG findings was not undertaken in these series except in
five of the 13 patients reported by Paddison and Ferriss. All of these reports contain an undeterminable number of patients with prior strokes and residual neurological deficit, frequently severe, or include patients with attacks followed by residual neurological symptoms or findings lasting for days or weeks. In view of differing criteria for what constitutes a carotid TIA, our patients cannot be considered completely comparable to those previously reported.

As can be seen in table 1, a third of our patients with carotid TIAs had an abnormal EEG. In two the abnormality was focal slowing in one temporal area, and in five the slowing was generalized. Six of the nine patients with an abnormal EEG showed evidence on angiography of intracranial arterial occlusion. Only one patient with extracranial occlusive disease alone, a patient with total carotid occlusion, had an abnormal EEG. Two-thirds of the patients with carotid TIAs had a normal EEG, and these patients were equally divided between those showing internal carotid artery stenosis in the neck and those with normal arteriograms.

As can be seen in figures 1 and 2, the degree of generalized EEG slowing in the seven patients being reported upon is clearly beyond the lower frequency limits found by Obrist and Henry in psychiatric patients over age 65 without evidence of brain disease. One of these seven patients had total occlusion of the internal carotid artery. Generalized EEG slowing was noted in some patients with carotid occlusion reported by Hass and Goldensohn and by McDowell et al. Meyer et al. suggested that this may represent a “steal” phenomenon in which one patent carotid is required to supply a contralateral hemisphere in addition to an ipsilateral one to the detriment of both. Four of the remaining six patients with generalized EEG slowing showed angiographical evidence of intracranial arterial occlusion. These patients almost certainly had cerebral infarction since Mooshy has shown that, while infarction without evidence of arterial occlusion is common, intracranial arterial occlusion without resultant cerebral infarction is exceedingly rare. Hjöd-Rasmussen and Skinhoj have reported that cerebrovascular lesions which appear clinically and radiologically strictly focal within one hemisphere may show significant reduction of blood flow also within the contralateral “healthy hemisphere” which they attribute to a neurally mediated generalized depression of cerebral metabolism. Such a series of events might produce a parallel generalized slowing of the EEG. On the other hand, Marquardsen and Harvald correlated EEG and postmortem brain findings and reported that cases showing diffuse slow wave abnormalities had multiple hemispherical softenings.

Kooi et al. reported that almost 30% of neurologically normal adults over age 60 have 40 to 60 microvolt single delta waves as often as one every 20 seconds focally in the temporal area. Similar findings were reported by Busse and Obrist. Both of our patients had single and serial delta waves in the temporal area more frequently than the limits established by Kooi. The postmortem studies of Cohn et al. and others have shown focal EEG slowing in patients with cerebral vascular disease to reflect the presence of cerebral infarction. This correlation is further supported by the regional cerebral blood flow studies of Sulg and Ingvar which have demonstrated focal alterations of cerebral blood flow corresponding to focal areas of EEG slowing. Blood flow is usually decreased in a focus of EEG slowing, though it is occasionally increased in acute lesions. Millikan has reviewed a variety of mechanisms by which the TIA syndrome may be produced. Any of these processes, if sufficiently long in duration, could produce cerebral infarction. Since both patients in this series with focal EEG slowing had angiographical evidence of intracranial arterial occlusion, it is highly likely that they had cerebral infarction, though not necessarily involving the temporal lobe, since the tendency for slow wave “displacement” from all other areas to the temporal area is a well-known though poorly understood phenomenon.

Lacking long-term follow-up data on these patients, the prognostic implications of an abnormal EEG remain unknown. The Joint Study of Extracranial Arterial Occlusion reported a 46% mortality in 44 patients with carotid stenosis having “transient attacks with incomplete recovery” in a 42-month follow-up period compared to a 27% mortality rate in 62 patients having “transient attacks with full recovery” during a similar follow-up period. If a persistent EEG abnormality constitutes an “incomplete recovery,” patients with such a finding would be at greater risk than patients with a normal EEG.

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