Current Role of Electroencephalography in Cerebral Ischemia

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Background: The electroencephalogram has been largely supplanted by neuroimaging techniques for the diagnosis and localization of ischemic stroke. However, because of its close correlation with cerebral metabolism and its ability to detect brief transient alterations in cortical function, the electroencephalogram may still be useful for certain diagnostic applications in stroke.

Summary of Review: The relation of electroencephalographic phenomena to cerebral blood flow and metabolism is reviewed. Ten clinical questions that can be addressed by the electroencephalogram in patients with stroke are posed. The presence of seizures, confirmation of diagnosis, intraoperative monitoring, and level of consciousness are areas of proven usefulness. The electroencephalogram provides less worthwhile information about the time course, prognosis, and localization of strokes. Computerized techniques are of potential but unproven value.

Conclusions: The electroencephalogram retains a worthwhile place in the evaluation of patients with cerebral ischemia when it is used to answer specific questions. All patients with strokes do not need electroencephalograms, but the test can provide uniquely useful data in some. (Stroke 1993;24:609–613)

Key Words • cerebral ischemia • cerebral ischemia, transient • cerebrovascular disorders • electroencephalography

I s an electroencephalogram (EEG) useful in treating a patient with a stroke? For 50 years, electroencephalographers have made energetic attempts to answer this question. If it is to have a role, the EEG must be shown to provide more or different information from radiographic studies or the clinical neurological examination. EEG phenomena are a rather direct reflection of cerebral metabolism. The test has a time resolution of seconds, is noninvasive, and is very widely available. Despite these advantages, an EEG should not be ordered routinely in all patients with stroke. It can answer certain clinical questions; for others it is a useless expense. This review critically examines applications of the EEG in patients with cerebral ischemia.

Relation of EEG Changes to Cerebral Blood Flow, Oxygenation, and Glucose Metabolism

The basic repertoire of EEG changes in ischemia was delineated long ago: 1) decreased beta-range fast activities; 2) increased slowing in theta and delta ranges; 3) loss of normal background rhythms such as the alpha rhythm; and 4) decreased overall amplitude.1–3

Quantitative studies of experimental stroke in animals4,5 and in humans during carotid endarterectomies6–8 indicate that faster frequencies—beta rhythms in the waking state and anesthetic-induced beta and alpha activities—are the most sensitive to minor degrees of ischemia. The appearance of polymorphic delta waves indicates a greater degree of ischemia, and ultimately the amplitude of all frequencies decreases. In appropriate areas, evoked potential changes may precede EEG changes.9

Normally, a tight correlation exists between regional cerebral blood flow (CBF) and oxygen uptake and, therefore, neuronal metabolism. During recovery from stroke10 or under the influence of some anesthetics such as isoflurane,11 cerebral metabolism may be partially dissociated from CBF. Under these circumstances, the EEG more closely reflects cerebral metabolism than does the CBF, making the EEG potentially more valuable as a measure of neuronal function.

However, under most conditions an orderly relation exists between CBF and electrical changes. Thresholds of CBF can be defined at which progressive neuronal dysfunction is reflected by specific electrical changes.9,12–14 While these thresholds vary depending on species and type of anesthesia, a representative sequence of functional ischemic thresholds (expressed in milliliters CBF per 100 grams per minute) is as follows: 20: decreased amplitude of somatosensory evoked potentials; 18: abnormal EEG, loss of fast frequencies; 15: increases in slow frequencies, loss of postsynaptic evoked responses; 12: EEG flat, loss of presynaptic evoked responses.

The EEG may indicate a more specific end point in severe hypoglycemia: at the point that the EEG becomes totally flat, necrosis of neurons apparently occurs rapidly.15 Ischemia or hypoxemia of a degree sufficient to produce a flat EEG may be associated with shifts in cationic distributions. An increase in cytosolic free calcium has been suspected of triggering irreversible
cell injury, and it is associated with severe EEG changes in a cat model of ischemia. The physiological reasons for differential effects of ischemia on fast and slow EEG rhythms are unclear. Loss of fast rhythms may reflect gray matter ischemia, whereas an increase in slow rhythms may reflect functional deafferentation of cortex caused by white matter ischemia, which may be considered analogous to the experimental lesions produced by Gloor et al.17 Strokes in humans usually involve both gray and white structures, and both loss of fast activities and polymorphic slowing are commonly observed.18 In humans, a summary EEG measure, mean EEG frequency, correlated well with oxygen uptake ($r=0.78$) and with gray matter CBF ($r=0.68-0.76$). Correlation with white matter CBF was lower ($r=0.3$).19

An important question in EEG studies of ischemia is whether there is an EEG change that indicates irreversible neuronal damage. Studies from series of endarterectomies suggest that critical EEG changes, if allowed to continue for certain times, do indicate an increased risk for infarction: a major loss of amplitude and the appearance of delta waves are EEG changes that, if they last longer than 30 minutes, have been associated with postoperative deficits.20 This is the basis for EEG surveillance techniques in cerebral ischemia.

### Clinical Questions for the EEG in Strokes

Are there clinical questions that the EEG may answer that cannot be answered as well by the history, physical examination, or imaging studies? I have considered 10 questions about cerebrovascular disease that physicians sometimes expect the EEG to answer. They may be sorted into three categories based on a review of the literature: those that the EEG often can answer, those for which EEG data are of questionable value, and those that the EEG either cannot answer or for which the EEG provides only redundant information.

#### Questions That an EEG Often Can Answer

1. **Is there a seizure focus?** Ten to twenty percent of thrombotic and embolic infarctions are associated with seizures. In such instances, the focus is usually demonstrable on EEG.21 Clinical seizures may be hard to differentiate from other involuntary movements seen in acutely ill patients. Decerebrate spasms, or myoclonus from anoxia or uremia, may resemble seizures. The EEG can be helpful in deciding whether to start antiepileptic drugs. The most common epileptiform pattern after a stroke is periodic lateralized epileptiform discharges (PLEDs).22,23 There is controversy about whether PLEDs represent an ictal or an interictal phenomenon. They are often associated with altered consciousness, but this may be because of the underlying lesion. If they are associated with continual contra-lateral focal motor seizures (epilepsia partialis continua), they are clearly ictal. After severe cerebral insults, which could include ischemia, as well as during late stages of status epilepticus, some patients may develop the syndrome of subtle generalized convulsive status—altered consciousness and subtle motor manifestations such as facial twitching or nystagmus.24 These patients have generalized epileptiform discharges on EEG, but the discharges may be quite asymmetrical, consisting of PLEDs of high amplitude on one side and much lower amplitude on the other. There is, in fact, a continuum between strictly unilateral PLEDs and bilaterally symmetrical periodic epileptiform discharges (PEDs).25 Because many patients at both ends of this spectrum have alterations of consciousness and not a few have subtle or obvious ictal motor phenomena, I advocate treating all patients with PLEDs, PEDs, or other more continuous and rhythmic ictal EEG discharges with antiepileptic drugs. I would not treat sporadic interictal spikes or sharp waves in the absence of observed behavioral seizures. Not all neurologists agree with these practices, but at the very least the detection of epileptiform activity in the EEG after a stroke should alert the clinician to the possibility of seizures. Poststroke seizures are often temporary; it is reasonable to taper patients off antiepileptic drugs when the EEG no longer shows epileptiform features.

2. **The imaging studies are normal. Has this patient had a stroke?** Computerized tomographic (CT) scans done within 24 hours after an ischemic stroke are abnormal only about half the time. Although CT abnormalities eventually appear in most such patients, it is useful to have a test verifying the clinical impression of stroke and its location immediately. Although magnetic resonance imaging (MRI) studies are more sensitive,26 they may miss early strokes before the development of edema.27 An MRI is often impractical on an emergency basis, and it requires a degree of patient cooperation. EEG sensitivity for cortical ischemic stroke is good; a representative figure is 76%.28 Although focal EEG slowing is not specific for ischemic stroke, it does document the presence and lateralization of a lesion affecting cortical function, which can confirm the clinical impression of stroke. Even if imaging studies are normal, an EEG is unnecessary in syndromes that can be localized precisely on clinical grounds. As examples, patients with aphasia and hemiparesis or with Wallenberg’s syndrome do not need EEGs. However, patients with ambiguous localizations, such as hemiparesis with no other cortical or brain stem dysfunctions, monopareses, etc., may have the site of their strokes clarified by an EEG.

3. **Is cerebral ischemia developing during surgery?** On a daily basis in our hospital, the most common use of the EEG in cerebrovascular disease is to monitor cortical function during carotid endarterectomy. Many large series assessing its usefulness have been reported.7,8,29,30 These studies may be summarized as follows: 1) The EEG is a sensitive means of detecting neuronal dysfunction caused by ischemia during the procedure. 2) The most common change is attenuation of the anesthetic-induced fast activity.8 This phenomenon occurs in 14–30% of occlusions.8,31 If slight (perhaps less than 50% decrease in amplitude), this should be considered a relatively minor change that does not correlate well with postoperative clinical deficit. 3) Severe attenuation of the EEG amplitude, with or without the appearance of delta-range polymorphic slow waves, is a more serious pattern after occlusion. It does correlate with an increased incidence of clinical deficits, at least in patients with long occlusion times.20 Many surgeons use the appearance of EEG changes as a cue to place a shunt. However, other options are open to the surgeon, such as raising the blood pressure, checking the artery for an acute thrombus, assessing
oxygenation, altering the anesthetic depth, or simply making haste to complete the procedure. Dissenting views are heard: some authors think that the value of the EEG in this situation, compared with its trouble and expense, is marginal.\textsuperscript{22,23} Color-coded topographic displays may be easier to interpret in this setting,\textsuperscript{34} but there is no reason to suppose that they contain more information than the raw EEG, and their practical superiority has not been established.

A preoperative EEG may be useful to identify patients with poor collateral circulation to aid in the assessment of the indications for endarterectomy.\textsuperscript{31} An EEG can also be used to assess collateral circulation when carotid ligation for treatment of aneurysms, carotid body tumors, or during radical neck surgery is contemplated. Rarely, an unfavorable preoperative EEG may predict a higher likelihood of perioperative stroke and so tip the scales away from a decision for surgery.

4. Is this paralyzed patient conscious? Ischemia of the ventral pons may render the patient unable to move any extremity, to speak, or to move the facial muscles. This situation—the "locked-in" syndrome—may go unrecognized, with the patient being treated as if comatose. Vertical eye movements are usually preserved, and patients can communicate this way. They may also be able to open and close their eyes, and some have preserved lateral eye movements. The EEG in the locked-in syndrome is normal.\textsuperscript{35} The technologist must passively open and close the patient's eyes to demonstrate alpha attenuation, since the patient may not be able to do this voluntarily. On the other hand, strokes affecting the tegmentum of the midbrain or upper pons will produce either diffuse bilateral slowing or an invariant coma pattern unresponsive to external stimulation, such as an alpha coma or theta coma. Patients with such patterns are stuporous or comatose. Photic stimulation produces normal driving responses in locked-in patients but not in comatose patients.

Although a careful neurological examination should identify most locked-in patients, in the real world a few such patients are not identified until an electroencephalographer raises this question.

Questions for Which an EEG Is of Questionable Value

5. Is this stroke getting better or worse? Efforts to use the EEG as a surveillance tool for stroke progression or therapeutic guidance in specialized stroke or intensive care units have been rather disappointing. There is a practical problem: a skilled interpreter cannot be available to watch a paper tracing continuously. Identification of quantitative EEG changes on a type of display that can be understood easily by nonelectroencephalographers may expand this use. As effective stroke therapies develop, it can be expected that the EEG, as a rapid monitoring technique, will become more attractive.

The ability to monitor EEG changes in the hours or days after a stroke does not prove that it is of practical use. Research should demonstrate that EEG changes precede irreversible worsening, that a therapeutic intervention at the time of the EEG change is effective, and that the clinical picture cannot be assessed more easily or cheaply by some other method. These criteria have not yet been met.

6. Is this stroke cortical or subcortical? A common problem on neurological wards is to distinguish hemiparetic strokes caused by cortical infarction from those caused by insular or brain stem lacunes. This is an important question for selection of treatment. Cortical infarctions are often due to artery-to-artery emboli, in which case the clinician may wish to consider anticoagulation or carotid surgery. Subcortical infarctions are often due to small-vessel arterial disease caused by hypertension or diabetes, which may not warrant such interventions. In one study, cortical infarction produced EEG changes in 76\% of patients and cerebral lacunar in only 9\%.\textsuperscript{26} However, EEG changes were reported to be present in many lacunar syndromes in a small series of patients.\textsuperscript{36} Small cortical infarctions can still escape detection by the EEG. Because the EEG is not absolutely reliable in making the distinction between cortical and subcortical strokes, it can only provide supportive data. A final decision on therapy could not be made on the EEG findings alone. However, this is the nature of many clinical data.

7. Does this patient have a new stroke or an old stroke? Clinical histories are often vague, and it may be important to know whether an observed hemiparesis or dysphasia is due to a recent or remote lesion. Many patients with atherosclerotic disease have multiple lesions of varying vintage on CT and MRI scans. Imaging studies are not always accurate at assessing the age of an infarction unless edema is clearly visible, and they may be normal soon after the event. A normal EEG in the presence of either a major clinical deficit of cortical function or a major lesion on the scan involving cortex is presumptive evidence that the infarction is at least several weeks old. Simply put, this is because abnormal EEG waves are produced by sick neurons: when these neurons either die or recover, many EEG abnormalities subside. Longitudinal studies\textsuperscript{10} show that most EEG improvement occurs within the first 3 months. On the other hand, the presence of PLEDs almost always indicates a recent event.\textsuperscript{33} The common EEG changes of ischemia; slowing, or loss of background activities are not helpful indicators of the age of a stroke, because these changes may persist to some degree for months or years.

8. Can computerized EEG analysis or brain topographic mapping improve the sensitivity of the EEG in cerebral ischemia? Frequency analysis of the EEG in vascular disease has been done for many years, usually with, at best, modest improvements in sensitivity over visual inspection.\textsuperscript{37,38} An increase in sensitivity of 34\% over the routine EEG has been reported for a method comparing multiple parameters to a normative database in a group of 94 patients with stroke or transient ischemic attack (TIA).\textsuperscript{39} The important question is whether these techniques can improve on the routine EEG in a clinically important way. This has not been proven, despite many publications and aggressive commercial promotions.

One area in which computerized analysis may improve on visual analysis is in the diagnosis of transient cerebral ischemia, although more research is required. It is sometimes hard to tell from the history whether a patient has had a recent TIA. Routine EEG is usually normal after TIA. However, in monkeys it can be shown by quantitative EEG analysis that fast activities remain...
suppressed for at least 24 hours after an experimental TIA, even though the neurological exam has returned to normal. In humans, brain topographic mapping may delineate a similar subtle residual effect. Human studies so far have been rather descriptive and unconvincing. Among 25 patients with TIA, Nagata et al found 68% to have abnormal brain topographic maps when 58% of the studies were done within 2 weeks after the TIA. Among 43 patients with TIA in another series, 63% had abnormal topographic maps and only 26% abnormal routine EEGs. Detection of minor strokes is another area of research to which computerized analysis has been applied. Nuwer et al compared 20 patients with mild strokes with 20 normal subjects. The maps were read blindly, without knowledge of the clinical deficit. In 17 of 20, abnormal increases in delta power and decreases in normal. It is common with large areas of ischemia anywhere in the hemisphere and can even be the only residual sign of anterior circulation stroke. Strictly anterior cerebral artery area infarctions may produce bifrontal slowing or even frontal intermittent rhythmic delta activity. Posterior cerebral artery territory ischemia causes parieto-occipital slowing and an asymmetry of the alpha rhythm and photic driving responses. Another clinical problem in EEG localization of stroke is the common presence of slowing contralateral to the presumed side of the stroke. All series of EEGs in stroke patients include increased numbers of bilaterally slow records compared with normal subjects. The contralateral slowing is probably caused by remote functional or metabolic effects on the normal hemisphere produced by the ischemic hemisphere. It may sometimes be caused by brain swelling with downward pressure on the upper brain stem, which can affect EEG rhythms bilaterally. Bilateral slowing does indicate bihemispheric dysfunction and often correlates with altered levels of consciousness after a severe stroke, but it further diminishes the localizing usefulness of an EEG.

10. What is the prognosis? The relevant question here is not whether the EEG correlates with the prognosis—obviously, it does—but whether it can outperform the neurological exam in predicting outcome. The early clinical picture is not definitive: some patients with hemiplegia eventually walk, others do not. Certain EEG features, such as loss of the alpha rhythm or overall suppression of amplitude, are said to be predictive of poor outcome. Patients with PLEDs also fare poorly. A report of a detailed frequency analysis for prediction of prognosis was discouraging, indicating that the "neurometric parameters obtained from the initial EEG had no value in this respect." There are as yet no data to indicate that the EEG can outperform clinical or imaging data in prognostic assessment of strokes. Future Directions The EEG relates more closely to cerebral metabolism, and therefore to neuronal function, than do secondary measures such as CBF or radiographic images. Direct measures of cerebral metabolism, such as 2-deoxyglucose positron emission tomography, are cumbersome, expensive, and have limited time resolution. The EEG therefore has considerable appeal, especially if it can be made more quantitative and more convenient. At this time, the most likely areas for future research in the application of frequency analysis and topographic mapping techniques are 1) retrospective diagnosis of TIA and minor strokes, 2) differentiation of diffuse from focal or multifocal cortical dysfunction, 3) improved surveillance of carotid and other vascular surgery, and 4) early warning techniques for stroke progression in the intensive care setting. Conventional EEG retains a useful place in the evaluation of patients with cerebral ischemia. Its usefulness, however, is limited to answering specific questions. Is the EEG useful in stroke? The answer depends on the stroke and on the information needed.

Acknowledgment Joanne Cage provided expert editorial assistance. References


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*Stroke*. 1993;24:609-613
doi: 10.1161/01.STR.24.4.609

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1993 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:
http://stroke.ahajournals.org/content/24/4/609

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