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Somatosensory Evoked Potentials Sensitivity Relative to Electroencephalography for Cerebral Ischemia During Carotid Endarterectomy

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Background and Purpose: **The relation between electroencephalographic pattern changes and cerebral ischemia during carotid endarterectomy under general anesthesia is well established. Pattern changes seen on somatosensory evoked potentials under the same conditions are reported to be more sensitive indicators of cerebral ischemia. We estimated the sensitivity and specificity of somatosensory evoked potentials relative to electroencephalography for detecting cerebral ischemia during carotid endarterectomy under general anesthesia.**

Methods: **We simultaneously monitored electroencephalographs and somatosensory evoked potentials in 53 carotid endarterectomies performed on 51 patients under general anesthesia, and we determined the extent to which somatosensory evoked potentials detected cerebral ischemia defined by electroencephalographic pattern changes at the time of carotid cross-clamp.**

Results: **Twenty-three of the 53 cases studied had electroencephalographic evidence of ischemia following carotid cross-clamp. Ten of these 23 cases had an increased somatosensory evoked potential latency of 0.1 msec or greater (sensitivity 0.43). One of these 23 patients had a decrease in somatosensory evoked potential amplitude of 50% or greater (sensitivity 0.04). Of the 30 subjects who had no electroencephalographic evidence of ischemia, 13 had either no change or a decrease in somatosensory evoked potential latency (specificity 0.45). None of these 30 cases had a significant decrease in somatosensory evoked potential amplitude (specificity 1.0). If somatosensory evoked potential latencies were a sensitive method for detecting cerebral ischemia (true sensitivity of 0.95 or higher), the probability of only 10 subjects having somatosensory evoked potential latency increases would be less than 0.001. Therefore, our observed sensitivity cannot be attributed to chance.**

Conclusions: **We conclude that measuring somatosensory evoked potentials is not a sensitive method for detecting cerebral ischemia during carotid endarterectomy. (*Stroke* 1992;23:498–505)**

KEY WORDS • **cerebral ischemia • electroencephalography • endarterectomy • evoked potentials, somatosensory**

The close correlation between cerebral electrical activity and the level of cerebral blood flow required to maintain neuronal metabolic function is the reason electroencephalographic (EEG) activity is monitored continuously during carotid endarterectomy performed under general anesthesia.[1–5] Characteristic EEG pattern changes suggestive of cerebral ischemia have been well documented and are used by surgical and anesthesia teams to detect and manage intraoperative cerebral ischemia.[1,3,5] Several investigators have identified comparable pattern changes in somatosensory evoked potentials (SEPs) and have argued that these are more sensitive than the recognized EEG pattern changes as markers of cerebral ischemia.[6–12]

Only EEG recording is used at our institution for monitoring during carotid endarterectomy. Particular attention is paid to the EEG pattern after carotid cross-clamp to evaluate the need for intracarotid shunt placement and to determine the adequacy of cerebral perfusion once a shunt has been placed. Because EEG recording is the accepted standard of monitoring in our hospital, this study estimates the sensitivity and specificity of intraoperative SEP monitoring relative to EEG monitoring for detecting cerebral ischemia in patients having carotid endarterectomy and general anesthesia.

Subjects and Methods

The protocol was approved by the Subcommittee on Human Studies at the Massachusetts General Hospital. Informed consent was obtained from each patient for surgery with neurophysiological monitoring. Between July 1, 1988, and February 28, 1989, we monitored 97 carotid endarterectomies with EEG recording. Five patients who underwent carotid endarterectomy followed by coronary artery revascularization were excluded. Thirty-three patients could not be studied simultaneously with EEG and SEP recording owing to the lack of monitoring personnel, and six additional pa-

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tients had to be omitted because of incomplete data collection. Fifty-three carotid endarterectomies performed on 51 patients (two patients underwent bilateral procedures) constitute the set of cases analyzed in this study. Patients were 30 men and 21 women, with a mean age of 64.8 (range 31–85) years.

Preoperative assessment included an electrocardiogram, cerebral angiogram, and a physical examination with a detailed neurological evaluation. Forty-four patients were considered symptomatic, defined as having had a transient ischemic attack, a mild, acute stroke, or worsening of an old stroke within 6 months before surgery. In the remaining seven asymptomatic patients, severe internal carotid stenoses were determined by radiographic criteria. Immediately after surgery, each patient had an abbreviated neurological examination to screen for significant alterations in mental status, visual fields, and motor and sensory function. A complete neurological evaluation was repeated within 24 hours after surgery.

Anesthesia was induced with thiopental in 52 cases and with alfentanil and oxygen in one. Anesthesia was maintained with a narcotic/nitrous oxide/muscle relaxant technique. Low concentrations of isoflurane or enflurane (0.2–0.6%) and phenylephrine infusions were used as needed to regulate systolic blood pressure within 20% of average preoperative values. The delivered concentration of the inhalational agents or infusion of narcotics was held constant throughout the cross-clamp period. Each patient was monitored intraoperatively with a radial artery catheter, an electrocardiograph, a pulse oximeter, an oral temperature probe, and a capnograph.

In 52 cases, the surgery performed was angioplasty of the carotid bifurcation and the initial segment of the internal carotid artery. Forty-six of these patients had primary closures, and six were closed with vein-patch grafts. In one patient a trauma-induced carotid aneurysm was resected and repaired with a vein-patch graft. Twenty-seven patients underwent surgery on the right carotid artery, and 22 underwent surgery on the left; two had bilateral procedures, staged 7 days apart.

Twenty-three Grass E5GH gold cup electrodes were placed on each patient's scalp according to the International 10-20 System. These electrodes were applied with collodion, filled, and maintained with a conduction gel. Electrode impedances were measured at less than 2,000 Ω. A Nihon Khoden Model 4321 electroencephalograph was used to record 16 bipolar channels of EEG with an anteroposterior montage and four channels of EEG referenced either to the second cervical vertebra or average reference. The remaining channel was used for recording the electrocardiograph. A high-frequency filter was set at 70 Hz with the time constant of 0.3 seconds, and a 60-Hz notch filter was used when necessary. Three minutes of baseline, awake EEG recordings were made before induction of anesthesia but after administration of preoperative medication.

Our criteria for EEG pattern changes consistent with ischemia were those established by Sharbrough et al[1] and expanded by Chiappa et al.[3] These changes are a loss or diminution of fast frequencies, primarily beta and alpha, an increase in theta and delta slowing, accompanied by an augmentation or attenuation of amplitude. Such changes are often located ipsilateral to the cross-clamp but are sometimes present bilaterally or in a specific region.

Additional electrodes for SEP recordings were placed at Erb's point and over the second cervical vertebra. One reference electrode was placed on the contralateral ear or shoulder and a second on the forehead at frontal polar zero. Grass E2 subdermal platinum electrodes were placed over the median nerves at the wrists and secured with tape. A Nicolet Pathfinder I was used to record SEPs with cortical representation at C3′ and C4′ located at 50% of the distance between electrodes C3 and P3 and between electrodes C4 and P4, respectively. The ground electrode was placed at the acromion. The bandpass filter was set at 30–1,500 Hz, and the time base was 50 msec. A 60-Hz filter was not used. Somatosensory evoked potentials were obtained by stimulating with a 0.2-msec constant current square wave impulse at 7.1 Hz for 210 averages for an update approximately every 35 seconds. The stimulus range of 5–20 mA was adequate to produce visible twitch of the thenar muscle before the induction of anesthesia and administration of muscle relaxants.

Several traces were superimposed, stored, and copied for on-line as well as future off-line analysis. Latencies were measured with a cursor, ascertained by the first upward (negative) deflection. Amplitude was measured baseline to peak. The peaks of particular importance included N9, N13-14, and N19-P23 complex, generated by ascending volleys through the brachial plexus, the dorsal column nuclei and medial lemniscus, and the thalamocortical projections, respectively.[13] Central conduction time was measured from N13-14 to N19. During carotid occlusion, only the median nerve contralateral to the side of surgery was stimulated for SEP assessments.

We define SEP pattern changes consistent with cerebral ischemia as either 1) a difference between the last central conduction time immediately preceding and the first one immediately following carotid cross-clamp greater than a specified threshold criterion or 2) an amplitude reduction of 50% or greater in the thalamocortical response (N19-P23 complex). Because of the inherent variability in the estimate of central conduction time,[14,15] our latency criteria for ischemia were made liberal to avoid bias against SEP. The four threshold criteria we studied were 0.1, 0.2, 0.3, and 0.4 msec.

From induction of anesthesia to the point at which the patient had sufficiently recovered to leave the operating room, one EEG technologist monitored the EEG and a second the SEP recording. An electroencephalographer was present during all studies to supervise the monitoring and to ensure the quality of the SEP tracings. The anesthesiologist and surgeons were kept apprised of important EEG pattern changes, and, if the EEG pattern changes at cross-clamp were suggestive of ischemia, the surgeon made the decision either to place an intracarotid shunt or to have systolic blood pressure raised with phenylephrine. The SEP changes were not used to make any intraoperative decisions.

Sensitivity was estimated as the fraction of patients who had ischemic SEP changes among the group of patients who had ischemic EEG changes after carotid cross-clamp. Specificity was estimated as the fraction

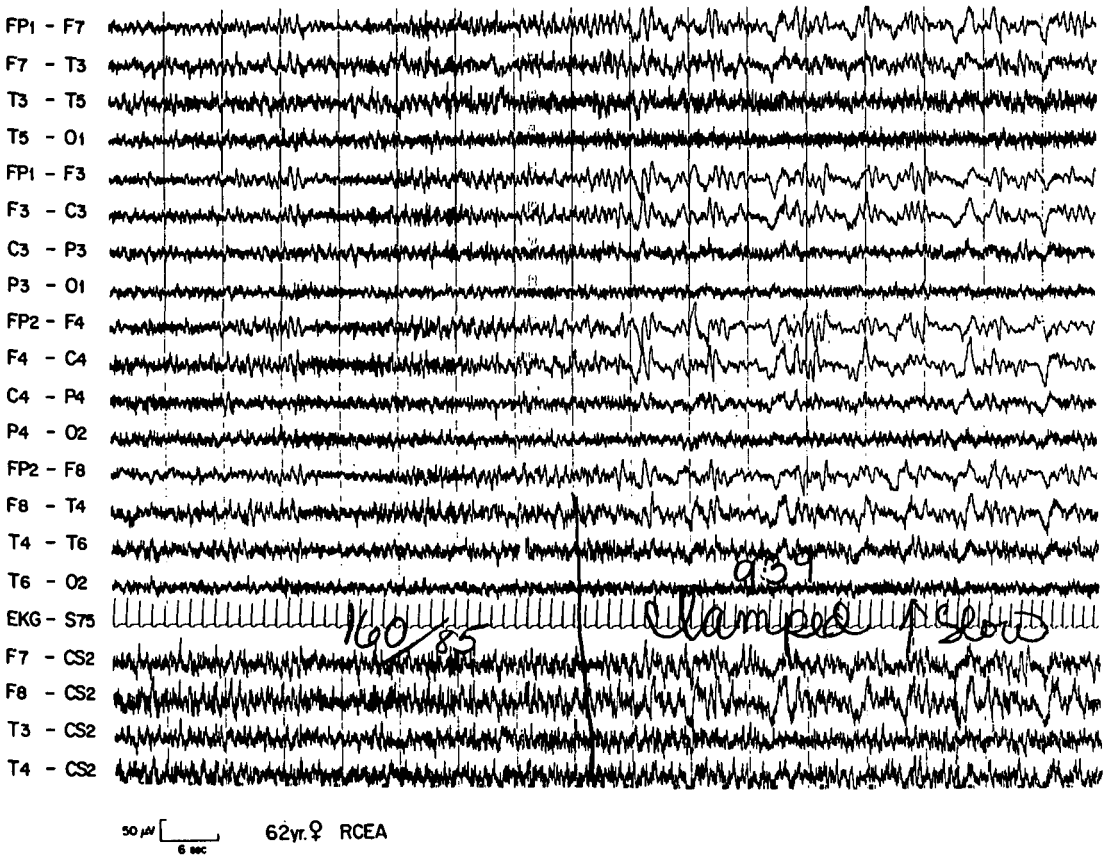


FIGURE 1. Top panel: *Electroencephalographic tracing showing abrupt onset of high amplitude slowing in right hemisphere, with some spread to left, seconds after right carotid artery cross-clamp in patient undergoing right carotid endarterectomy.* Bottom panel: *Right thalamocortical response (N19-P23 complex) of somatosensory evoked potentials of same patient. Waveforms are shown as acquired in sequence according to time, from bottom to top of vertical axis. Above each column of responses are data labeling waveforms (A1–A8 on left and B1–B8 on right), time at which they were recorded, latencies measured in milliseconds, and amplitudes. Note that latency diminishes from 19.3 to 18.6 msec and amplitude increases from 2.14 µV to 2.27 µV after carotid artery cross-clamp (A8 and B1).*

of patients without ischemic SEP changes among the group of patients who had no ischemic EEG changes after carotid cross-clamp. The predictive value positive is the probability that a patient has ischemic EEG changes given that he has ischemic SEP pattern changes, and we estimated this probability as the fraction of patients who had ischemic EEG changes among the group with ischemic SEP changes. The negative predictive value is the probability that a patient has no ischemic EEG changes given that he has no ischemic SEP pattern changes. This probability was estimated as the fraction of patients who had no ischemic EEG changes among the group without ischemic SEP pattern changes. Sensitivity, specificity, negative predictive value, and positive predictive value were computed for each of the four latency criteria and the amplitude criterion for SEP ischemic pattern changes. Confidence statements were computed from

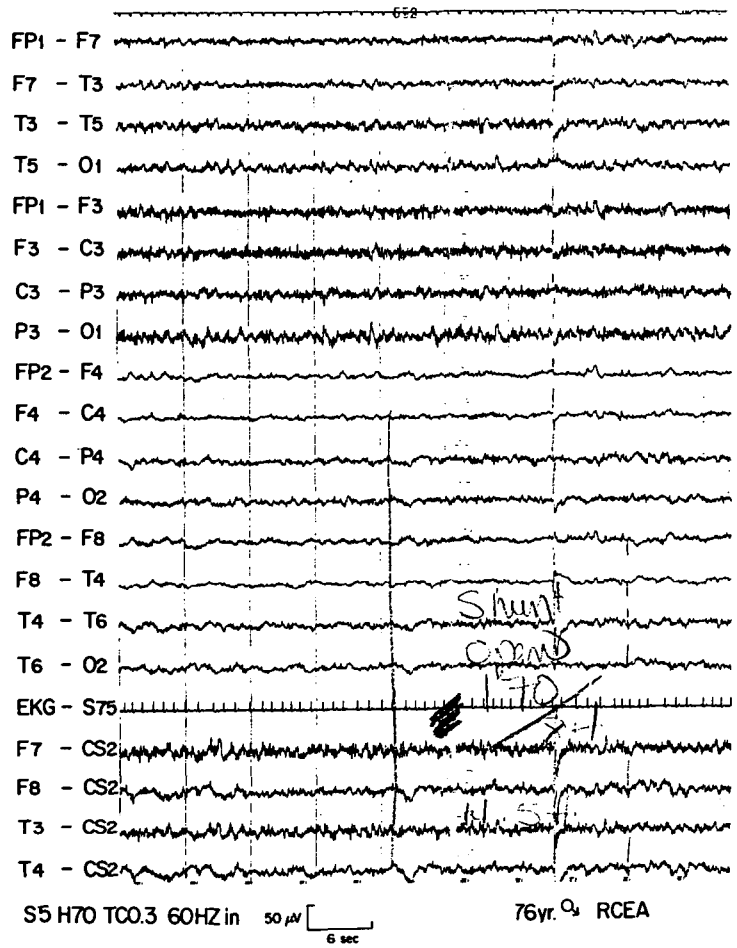
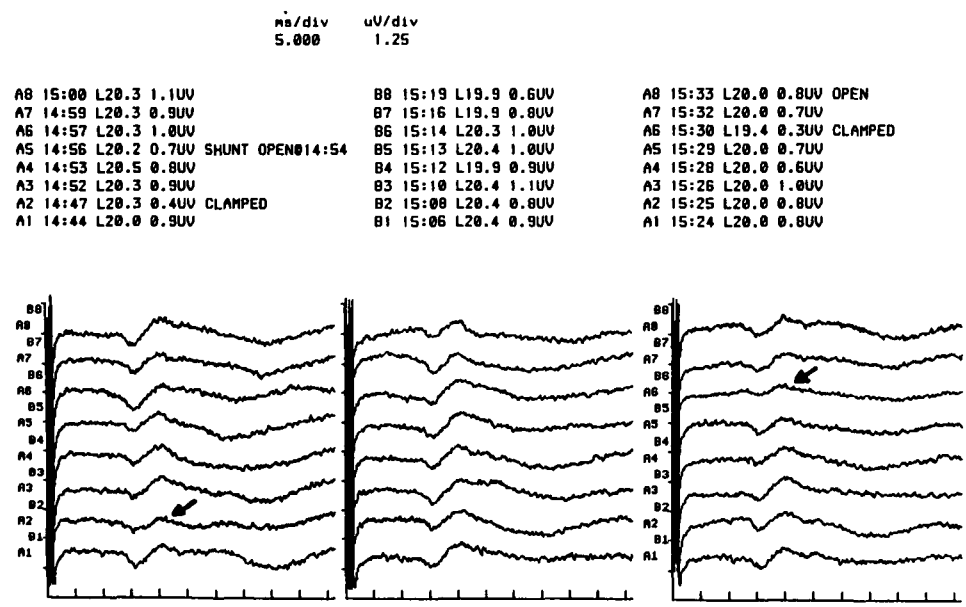


FIGURE 2. Top panel: *Electroencephalographic tracing, 7 minutes after right carotid artery cross-clamp in patient, revealing marked decreased amplitude and diminution of fast activity on right.* Bottom panel: *Right thalamocortical response of SEP of same patient revealing loss of amplitude at carotid artery cross-clamp* (arrows) *from 0.9 to 0.4 μV (55.6%) and 0.7 to 0.3 μV (57.1%), respectively. Note return of amplitude while cross-clamp is still in place and before insertion of intracarotid shunt.*



the Gaussian approximation to the binomial distribution,[16] and power calculations were performed using the tables of the binomial distribution.[17] We considered an SEP criterion equally as sensitive as the EEG criteria for detecting cerebral ischemia if the upper bound on the 95% confidence interval of the former exceeded 0.95.

Results

At induction of anesthesia, EEG pattern changes included a combination of a generalized admixture of delta and theta slow waves with superimposed diffuse alpha and beta (fast) frequencies. At carotid artery cross-clamp, 23 patients (44%) had EEG changes

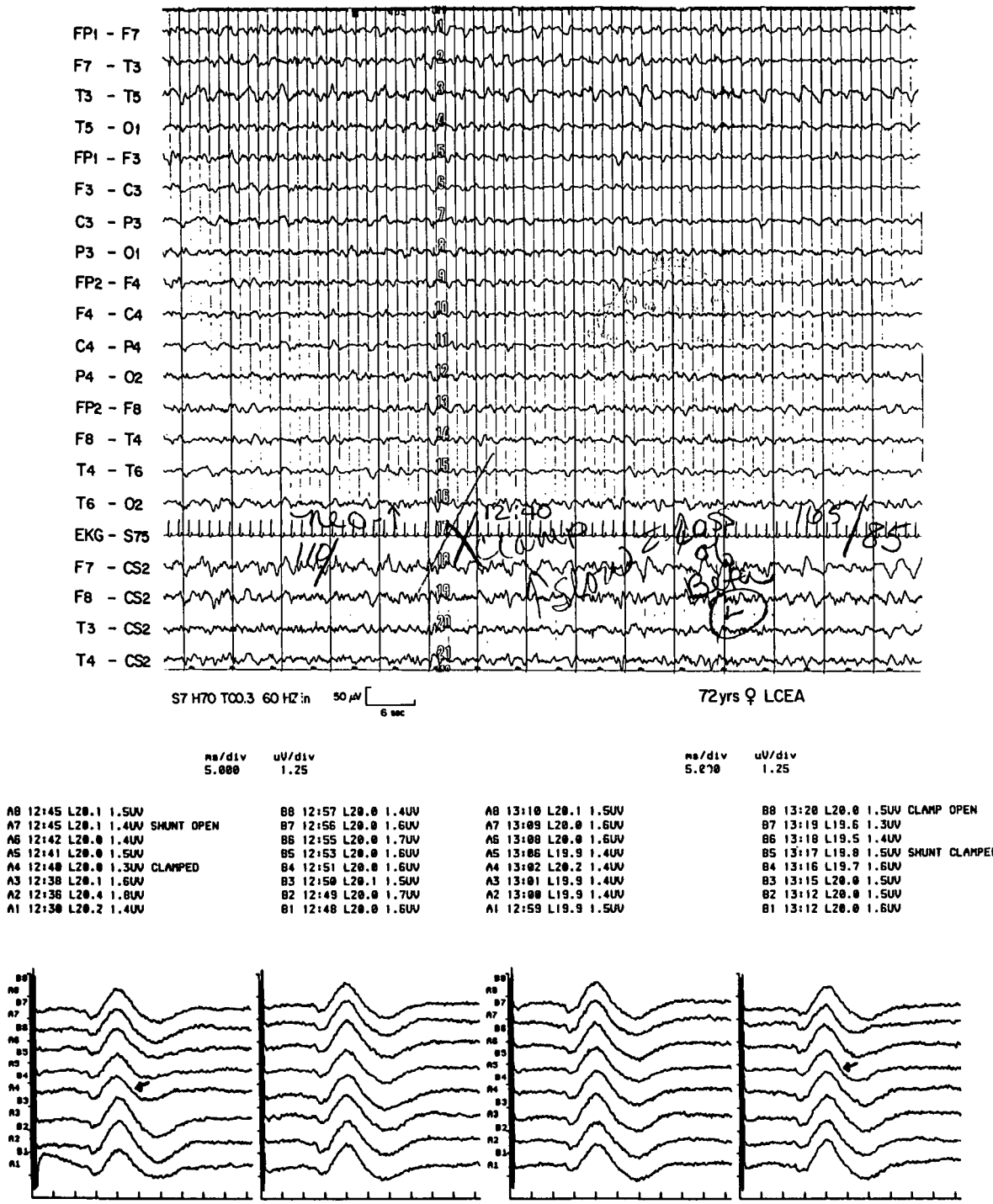


FIGURE 3. Top panel: *Electroencephalographic tracing of patient undergoing left carotid endarterectomy showing loss of fast frequencies and increased amplitude on left after carotid artery cross-clamp.* Bottom panel: *Left thalamocortical SEP of same patient showing little detectable change at carotid artery cross-clamp* (arrows).

suggestive of ischemia. The ischemic changes were characterized as follows: 12 cases of a mild-to-moderate shift to slower frequencies with increased amplitude on the scalp ipsilateral to the clamp; four cases of a similar shift to slower frequencies accompanied by an ipsilateral decrease in amplitude; three other cases of a generalized change to slower frequencies, with the greater change occurring on the ipsilateral side; three recordings of slower frequencies ipsilaterally with spread into the contralateral scalp; and one with mild, intermittent, ipsilateral slowing. In all cases, EEG changes showed some degree of ipsilateral diminution of beta (fast) activity. Ischemic EEG pattern changes took place within 20 seconds of carotid cross-clamp in all but one operation, in which these changes were delayed for 10 minutes. Examples of these EEG pattern changes are illustrated in the top panels of Figures 1, 2, and 3.

TABLE 1. Accuracy of Latency Thresholds for Detecting Ischemia

Central conduction time latency threshold (msec)	Sensitivity	Specificity	Positive predictive value	Negative predictive value
0.1	0.43	0.43	0.37	0.5
0.2	0.35	0.5	0.35	0.5
0.3	0.3	0.57	0.35	0.53
0.4	0.22	0.83	0.5	0.58

Seventeen of the 43 cases (40%) with normal preoperative EEGs and six of 10 cases (60%) with abnormal preoperative EEGs had ischemic changes at carotid cross-clamp. Four of the latter six cases (67%) had had a previous stroke. Ischemic EEG pattern changes occurred in 20 of 46 (43%) symptomatic and in three of seven (43%) asymptomatic patients. Intracarotid bypass shunts were placed in 20 of the 23 procedures with ischemic EEG pattern changes. In each patient receiving a shunt, ischemic changes resolved. In two of the three surgeries with ischemic changes in which shunts were not placed, systolic blood pressure was elevated with phenylephrine and the EEG returned to baseline. In the third, the surgeon elected not to use a shunt because of a high carotid artery bifurcation. This situation was also managed with phenylephrine-induced hypertension that resulted in an increase in faster frequencies without a complete return to the EEG pattern before carotid cross-clamp. In one operation without ischemic EEG changes, the surgeon placed a shunt to facilitate closure of the arteriotomy.

The sensitivity of the latency criteria for ischemia increased from 0.22 to 0.43 as the latency threshold decreased from 0.4 to 0.1 msec (Table 1). The 95% confidence intervals for the true sensitivities based on these two thresholds were 0.05,0.39 and 0.23,0.64, respectively. The specificity of the latency criteria increased from 0.43 to 0.83 as the latency threshold increased from 0.1 to 0.4 msec. The 95% confidence intervals for the true specificity based on these criteria were 0.26,0.61 and 0.77,0.90, respectively. The maximum positive predictive value of 0.5 and the maximum negative predictive value of 0.58 occurred for the latency criterion of 0.4 msec. Their respective 95% confidence intervals were 0.19,0.81 and 0.51,0.66. Both before and after carotid artery cross-clamp, the typical range of central conduction time latencies extended from 0.1 to 1 msec.

In only one of the 23 cases with ischemic EEG changes did the SEP amplitude at the time of carotid cross-clamp decrease by more than 50% compared with the period immediately preceding its placement. The sensitivity of the amplitude criterion is 0.04, with a 95% confidence interval of 0.001,0.086. This subject's amplitude returned to its pre–cross-clamp magnitude before shunt placement (Figure 2, bottom panel). None of the 30 patients without ischemic EEG changes satisfied the SEP amplitude criterion for ischemia, making its estimated specificity 1. The bottom panels of Figures 1 and 3 also illustrate our general observation that the variability in SEP signal amplitude did not differ between the periods before and after cross-clamping.

Within 24 hours all patients had returned to clinical preoperative neurological baseline status. Two patients experienced prolonged recoveries from anesthesia. Four patients with documented motor deficits before surgery required up to 4 hours to regain their preoperative levels of motor function.

Discussion

We selected cross-clamp of the carotid artery as the event for comparison of SEP and EEG because this maneuver is frequently associated with EEG evidence of cerebral ischemia. We modified the latency criterion of Russ and coworkers[10] and assessed its validity for ischemia over a range of 0.1–0.4 msec, the lower limit representing the smallest gradation on our scale and the upper a fourfold increase. As expected, liberalizing the criterion by lowering the threshold improves its sensitivity at the expense of decreasing its specificity. Our amplitude criterion was based on studies reporting that a 50% reduction in SEP amplitude is a criterion for ischemia.[6,8–10,12] The data from our study show that none of the SEP criteria reliably identified those instances of compromised cerebral perfusion that were readily discernible by EEG.

We computed the statistical power of our study for a sample of 23 patients and assumed a true sensitivity of SEP relative to EEG of 0.95. That is, given 23 patients with ischemic EEG changes, how likely would it be to see only 10 cases of SEP ischemia if the true sensitivity of the SEP latency criterion were 0.95 or higher? This probability is less than 0.001, suggesting that our findings cannot be attributed to chance.

There are two reasons that the results from our investigation differ from those reported in previous studies assessing SEP reliability in detecting cerebral ischemia during carotid endarterectomy. One reason is that our definition of cerebral ischemia is based on the documented correlation between EEG pattern changes and regional cerebral blood flow during carotid endarterectomy. Neither an SEP amplitude reduction of 50% nor an increase in central conduction time of greater than 20% has been established as a physiological marker of impaired cerebral perfusion under operative conditions. Studies that have examined SEP use in carotid endarterectomy argue the validity of the criteria for cerebral ischemia based on postoperative neurological outcome. Investigators have demonstrated that during carotid endarterectomy, patients may have cerebral ischemia (defined as either diminution in cerebral blood flow or the appearance of EEG patterns considered indicative of ischemia) without developing postoperative neurological deficits.[18,19] Therefore, the appearance of neurological deficits may not be used as an acceptable definition of

intraoperative cerebral ischemia. By analogy, the imperfect correlation between cardiac ischemia and the occurrence of myocardial infarction prevents the latter from being a surrogate definition of the former.

The second reason is that SEP criteria create complex problems of interpretation because the SEP is an averaged electrical response generated by stimulating mixed peripheral nerves consisting of different sensory fiber sizes and conducting capacities and modified by several neuronal synapses. Multiple subcortical and cortical generators contribute to the scalp-recorded SEP N19 waveform, the morphology, latency, and amplitude of which are influenced by different stimulating and recording techniques.[20-22] A functional definition of SEP, therefore, must incorporate intraindividual variability.[15] The magnitude of this variability or noise in the SEP signal (identifiable in subjects in the clinic as normal or abnormal based on normative data) in patients undergoing carotid endarterectomy and general anesthesia becomes problematic because it is so prominent. Our findings agree with those of others who have described in another surgical setting marked intraoperative variability of the SEP latency and amplitude unassociated with any simultaneous surgical or anesthetic manipulation.[23] Although there is variability in the EEG signal as well, pattern changes suggestive of cerebral ischemia are reproduced more reliably, and information from the matrix of EEG scalp electrodes allows simultaneous analysis of qualitative and regional differences.

The probable reason that SEP responds less consistently to cerebral ischemia than does EEG is that the subcortical generators contributing to the SEP thalamocortical response are less affected by cerebral ischemia and hypoxia than are the cortical generators of EEG.[24] Although some investigators have shown abrupt loss of the cortical SEP in experimental regional ischemia,[25,26] others have reported robust, though delayed, cortical SEP signals in the presence of frank cerebral infarction.[27] In the present study there were marked EEG pattern changes of ischemia at carotid artery cross-clamp without reliable correlates of SEP change.

Our data indicate that a clearer explanation of the relations between cerebral ischemia and SEP amplitude and latency alterations is necessary before SEP may be considered an alternative to EEG as a method of monitoring cerebral ischemia during carotid endarterectomy. Further studies using both EEG and SEP monitoring simultaneously with regional cerebral blood flow analysis may provide a better understanding of the relation between these electrophysiological modalities and cerebral ischemia.

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