

# Monitoring Electrophysiologic Function during Carotid Endarterectomy: A Comparison of Somatosensory Evoked Potentials and Conventional Electroencephalogram

Arthur M. Lam, M.D., F.R.C.P.C.,\* Pirjo H. Manninen, M.D., F.R.C.P.C.,†  
Gary G. Ferguson, M.D., F.R.C.S.C.,‡ William Nantau, B.Sc.§

There is no consensus as to the most appropriate monitor for detecting ischemia during carotid endarterectomy. Accordingly, simultaneous 16-channel continuous electroencephalogram (EEG) and somatosensory evoked potential (SSEP) monitoring were performed in 64 normocapnic patients undergoing carotid endarterectomy and anesthetized with isoflurane or halothane-nitrous oxide (supplemented with fentanyl). Recordings were obtained before, during, and for 15 min after cross-clamping of the internal carotid artery. Internal shunt was not used in any patient, regardless of EEG and SSEP changes. Significant amplitude reduction in the cortical component of the primary negative peak (>50%) in SSEP occurred in 6 patients, and an increase in central conduction time (CCT) (>1 ms) occurred in 5 patients. Major EEG changes occurred in 6 patients, 4 of whom also had SSEP changes. Two patients had transient neurologic deficits postoperatively, with both having SSEP changes (amplitude reduction > 50%), whereas one had EEG changes. Based on these observations, the relative sensitivity and specificity for EEG and SSEP (amplitude reduction > 50%) in detecting postoperative neurologic deficits were 50% and 92% for EEG and 100% and 94% for SSEP, respectively, differences that were not statistically significant. Regarding SSEP, the use of latency change (CCT) as a criterion was associated with a sensitivity of 0% ( $P = 0.046$  from sensitivity of amplitude) and a specificity of 87% ( $P = 0.17$  from specificity of amplitude). The authors concluded the following, regarding the use of EEG and SSEP as diagnostic tests of postoperative neurologic deficit: 1) both forms of electrophysiologic monitoring are associated with a considerable false-positive rate; 2) compared with conventional EEG, SSEP monitoring during carotid endarterectomy has a similar sensitivity and specificity; and 3) amplitude reduction greater than 50% is a better indicator than latency increase during SSEP monitoring. (Key words: Brain; electroencephalography; evoked potentials. Monitoring; electroencephalography; evoked potentials; stump pressure. Surgery: carotid endarterectomy.)

CAROTID ENDARTERECTOMY is associated with a significant risk of perioperative stroke.<sup>1,2</sup> Although there is some controversy regarding the need for monitoring of central nervous system function during cross-clamping of

the carotid artery,<sup>3-5</sup> consideration of physiologic principles indicates that intraoperative detection of cerebral ischemia during carotid cross-clamping allows the insertion of an internal shunt and therefore may reduce this risk. However, there is no consensus on the most appropriate monitor for such purposes.

The conventional 16-channel EEG has been used extensively for this purpose, and many people consider it to be the gold standard.¶ More recently, somatosensory evoked potentials (SSEP) have been introduced for such purposes.<sup>6-15</sup> SSEP monitoring offers the potential advantage that it is technically easier to perform and interpret and provides information specific to the sensory cortex, an area supplied by the middle cerebral artery that is placed at risk during cross-clamping of the carotid artery. Despite these theoretic advantages and the publication of several reports<sup>6-15</sup> on such use, no definitive criteria for interpretation have emerged and, with the exception of a preliminary study by McPherson *et al.*,\*\* no comparison of SSEP and EEG in regard to their relative sensitivity and specificity for the detection of cerebral ischemia has been reported. Because it is the standard surgical practice in our institution that internal shunts not be used regardless of changes of monitored parameters, this situation presents us with a unique opportunity to address this issue and evaluate the potential clinical utility of such monitoring.

## Materials and Methods

### PATIENTS STUDIED

The study was approved by the Ethics Committee on Human Research of the University of Western Ontario. Sixty-seven consecutive patients undergoing carotid endarterectomy were studied. The indications for the surgical procedure included transient ischemic attack, minor stroke, and asymptomatic high-grade stenosis of the in-

\* Associate Professor of Anesthesiology and Neurosurgery, University of Washington School of Medicine.

† Associate Professor of Anaesthesia, University of Western Ontario.

‡ Professor of Neurosurgery, University of Western Ontario.

§ Research technician, University of Western Ontario.

Received from the Department of Anesthesiology and the Department of Neurosurgery, Harborview Medical Center, University of Washington School of Medicine, Seattle, Washington and the Department of Anaesthesia and the Department of Clinical Neurological Sciences, University of Western Ontario, London, Ontario, Canada. Accepted for publication February 28, 1991.

Address reprint requests to Dr. Lam: Department of Anesthesiology, Harborview Medical Center, 325 9th Avenue, Seattle, Washington 98104.

¶ Lanier WL: Cerebral function monitoring during carotid endarterectomy. *J Neuro Anesth* 1:207-210, 1989.

\*\* McPherson RW, Johnson RM, Graf M: Intraoperative neurological monitoring during carotid endarterectomy: Electroencephalogram versus somatosensory evoked potential (abstract). *ANESTHESIOLOGY* 59:A368, 1983.

ternal carotid artery. Because of technical difficulties, SSEP could not be recorded in three patients, and they were excluded from data analysis. Of the 64 patients studied, the mean age was  $63 \pm 6$  yr. (SD), and mean weight was  $72 \pm 13$  kg. There were 24 women and 40 men.

#### ANESTHETIC TECHNIQUE

Patients received no preanesthetic medications. Anesthesia was induced with sodium thiopental  $3\text{--}4 \text{ mg} \cdot \text{kg}^{-1}$ , fentanyl  $2\text{--}5 \text{ } \mu\text{g} \cdot \text{kg}^{-1}$ , and lidocaine  $1 \text{ mg} \cdot \text{kg}^{-1}$ . Tracheal intubation was facilitated with succinylcholine  $1 \text{ mg} \cdot \text{kg}^{-1}$  or a nondepolarizing muscle relaxant. Anesthesia was maintained with a nitrous oxide-oxygen mixture (50%/50%) and 0.25–1.0% inspired isoflurane (62 patients) or 0.2–0.5% halothane (2 patients). A nondepolarizing muscle relaxant was used to provide muscle relaxation, and fentanyl was administered intravenously in 50- $\mu\text{g}$  increments as needed. All patients' lungs were mechanically ventilated to maintain normocapnia with  $\text{PaCO}_2$  of 38–42 mmHg. Intraoperative monitoring included an electrocardiogram, intraarterial catheter for direct blood pressure measurement, end-tidal capnometry, and nasopharyngeal temperature. At least one arterial blood gas sample was obtained before cross-clamping of the carotid artery and again during cross-clamping. To minimize the influence of inhaled anesthetics on EEG and SSEP, the inspired concentration was held constant from at least 15 min before cross-clamping to 15 min after clamping. No attempt was made to increase blood pressure to greater than preclamp values during cross-clamping, but a phenylephrine bolus or infusion was used whenever necessary to maintain normotension.

#### EEG AND SSEP MONITORING

Electroencephalogram and SSEP were recorded for 10 min before carotid clamping, continuously throughout clamping, and for 10–15 min after clamping.

##### *Electroencephalogram*

A standard 16-channel EEG was monitored using 20 scalp electrodes applied with collodion, according to the International Ten-Twenty System of electrode placement. The 16-channel montage, with 8 channels for each hemisphere, covered the parasagittal and temporal regions. The impedances were less than 2,000 ohms. The high-band pass and low-band pass filters were set at 0.3 and 70 Hz, and the sensitivity was  $5 \text{ } \mu\text{V} \cdot \text{mm}^{-1}$ . The paper speed was 1.5 cm/s. A significant change was defined as the appearance or increase of theta or delta activity, suppression of alpha and beta activity of greater than 50%, or both in the raw EEG data.<sup>16</sup> The changes could be ipsilateral to clamping or bilateral.

#### *Somatosensory Evoked Potential*

Scalp electrodes were attached with collodion over the somatosensory cortex bilaterally (C3' and C4'), as well as over the C7 spine. The reference electrode was placed over the forehead (Fpz). The impedances were less than 3,000 ohms. With the use of a Nicolet CA1000 averager, SSEP was recorded in response to a square wave current of 200  $\mu\text{s}$  duration applied to the contralateral median nerve by needle electrodes. The stimulus was set at 15 mA and a visible twitch confirmed before the administration of muscle relaxants. The high-band pass and low-band pass filters were set at 30 and 3,000 Hz, and the evoked potential was averaged from 256 repetitions recorded with the use of a 50-ms time base.

Before cross-clamping, baseline bilateral SSEPs were recorded sequentially by stimulation of each median nerve alternately. During cross-clamping of the carotid artery, ipsilateral recordings were obtained continually, and contralateral recordings were performed whenever visible changes in ipsilateral recordings were detected. SSEPs were analyzed according to the peak-to-trough amplitude of the primary negative peak ( $\text{N}_{20}$ ) and the central conduction time (CCT), which is the latency difference between  $\text{N}_{20}$  and the negative peak recorded over C7 spine ( $\text{N}_{13}$ ). Because no definitive criteria have been established, the changes in SSEP were classified into four categories: 1) grade 1: unchanged in amplitude and CCT; 2) grade 2: a decrease in amplitude greater than 50%; 3) grade 3: an increase in CCT greater than 1.0 ms; 4) grade 4: a decrease in amplitude greater than 50% and an increase in CCT greater than 1 ms.

#### ANALYSIS

Both SSEP and EEG changes that occurred after cross-clamping of the carotid artery were further characterized as follows: 1) transient—changes that resolved spontaneously within 10 min without intervention; 2) reversible—changes that reversed with release of cross-clamp; 3) persistent—changes that persisted to the end of the monitoring period. The analysis was performed in a blinded manner; the raw EEG interpreted by an electroencephalographer off-line without knowledge of SSEP changes as part of his ongoing evaluation of the reliability of intraoperative monitoring of EEG. SSEP was interpreted by two of the authors (AML and PHM), and, although some changes were observed intraoperatively, all final interpretations were performed off-line without the examiner's knowledge of intraoperative EEG changes. A thorough neurologic examination was performed immediately after the patient awakened, 24 h after, and at the time of discharge (1 week postoperatively). Neurologic deficits also were defined as transient if the deficit resolved within 24 h and fixed if the deficit was present at the time

TABLE 1. Variables during Cross-clamping of the Carotid Artery

|                          |                       |
|--------------------------|-----------------------|
| Duration                 | 36 ± 10 min           |
| PaCO <sub>2</sub>        | 40 ± 2 mmHg           |
| Systemic pressure        | 90 ± 13 mmHg          |
| Isoflurane concentration | 0.5 ± 0.2% (inspired) |

All values are mean ± SD.

of discharge. Both SSEP and EEG changes were then compared with the occurrence of the neurologic deficits to assess their relative sensitivity and specificity. Chi-squared analysis was used to assess statistical significance.

**Results**

Satisfactory EEG and SSEP recordings were obtained in 64 patients. The duration of carotid artery occlusion, PaCO<sub>2</sub>, the mean systemic blood pressure, and the mean inspired isoflurane concentration during carotid cross-clamping are listed in table 1. Before cross-clamping, all patients had normal SSEPs and 13 patients had some abnormality on their EEG. Of these 13 patients, in 3 patients additional EEG changes developed during cross-clamping, which were reversible in 2 and transient in the other. A transient postoperative neurologic deficit developed in one patient with a reversible EEG change. Regardless of EEG or SSEP changes, internal shunts were not inserted in any of the patients.

Results regarding EEG and SSEP data are shown in table 2. Fifty-three patients, 2 with EEG changes but none having neurologic deficits, had unchanged SSEPs (grade 1) throughout the recording periods. Three patients, 2 of whom with EEG changes, had grade 2 SSEP changes that were reversible. Transient neurologic deficits developed in 2 of these 3 patients (hemiparesis of less than 24

TABLE 2. Distribution of Intraoperative Changes in SSEP and EEG, and Postoperative Neurologic Deficits

| Grade | SSEP Changes |              | EEG Changes |              | Neurologic Deficits |              |
|-------|--------------|--------------|-------------|--------------|---------------------|--------------|
|       | Duration     | Patients (n) | Duration    | Patients (n) | Duration            | Patients (n) |
| I     |              | 53           | T           | 0            |                     |              |
|       |              |              | R           | 2            | T                   | 0            |
|       |              |              | P           | 0            | F                   | 0            |
| II    | T            | 0            | T           | 1            |                     |              |
|       | R            | 3            | R           | 1            | T                   | 2            |
|       | P            | 0            | P           | 0            | F                   | 0            |
| III   | T            | 2            | T           | 1            |                     |              |
|       | R            | 2            | R           | 1            | T                   | 0            |
|       | P            | 1            | P           | 0            | F                   | 0            |
| IV    | T            | 1            | T           | 0            |                     |              |
|       | R            | 2            | R           | 0            | T                   | 0            |
|       | P            | 0            | P           | 0            | F                   | 0            |

T = transient (changes that resolved spontaneously within 10 minutes); R = reversible (changes that reversed with release of cross-clamp, range = 14–35 min); P = persistent (changes that persisted to the end of the monitoring period, duration = 41 min in the only patient with SSEP grade III changes); F = fixed.

h duration), but only 1 had EEG changes. (The other patient had an abnormal preclamp EEG with ipsilateral slowing but no intraoperative changes during cross-clamping). Grade 3 SSEP changes developed in 5 patients, 2 of whom with EEG changes, but neurologic deficits did not develop in any of them. Three patients had grade 4 changes, of whom none had EEG changes or a neurologic deficit postoperatively. The overall rate of new neurologic deficit was 3.1%, whereas the comparative rate for EEG changes was 9.3% and SSEP changes (grade 2–4) 17%. The cross-clamp times for the 2 patients with postoperative deficits were 29 and 37 min and the durations of SSEP changes were 14 and 32 min, respectively, with the EEG changes in the first patient lasting more than 10 min and no EEG changes occurring in the other.

Calculations of the relative sensitivity and specificity<sup>††</sup> of SSEP and EEG in their ability to detect postoperative neurologic deficits are shown in table 3. The difference in sensitivity and specificity between the two modalities was not statistically significant (*P* = 0.5 for both). Power analysis<sup>18</sup> indicates that, to achieve statistical significance with the observed difference at an *α* error of 0.05 and a *β* error of 0.2, it is necessary to have 12 patients with neurologic deficits and 1,440 patients without neurologic deficits. To compare amplitude *versus* latency as useful criteria for interpretation of SSEP changes, we also computed the incidence of change based solely on amplitude (*i.e.*, grades 2 and 4 combined [both groups fulfill the amplitude criterion]) and the incidence based on latency alone (grades 3 and 4 combined). The incidence was 9.3% for the former and 13% for the latter. The relative sensitivity and specificity for detection of postoperative neurologic deficit were 100% and 94% for the amplitude criterion and 0% and 87% for the latency criterion, respectively. The difference in sensitivity was marginally significant (*P* = 0.046, chi-square analysis), and the difference in specificity was not significant (*P* = 0.17).

**Discussion**

In this study we have attempted to demonstrate the potential utility of SSEP monitoring during carotid endarterectomy; no new neurologic deficits developed in any patient who had unchanged SSEPs, and both patients who developed postoperative deficits had SSEP changes.

†† Sensitivity and specificity are standard analytical methods used to assess the validity of any diagnostic test in comparison with a gold standard.

$$\text{sensitivity} = \frac{\text{true positives by gold standard}}{\text{true positives} + \text{false negatives}} \quad (1)$$

$$\text{specificity} = \frac{\text{true negatives by gold standard}}{\text{true negatives} + \text{false positives}} \quad (2)$$

TABLE 3. Relative Sensitivity and Specificity of SSEP and EEG

|                             | True Positive<br>(Neurologic Deficit) | True Negative<br>(No Deficit) |  |
|-----------------------------|---------------------------------------|-------------------------------|--|
| SSEP +<br>(amplitude < 50%) | 2                                     | 4                             | Sensitivity = $\frac{2}{2+0} = 100\%$  |
| SSEP -                      | 0                                     | 58                            | Specificity = $\frac{58}{4+48} = 94\%$ |
| EEG +                       | 1                                     | 5                             | Sensitivity = $\frac{1}{1+1} = 50\%$   |
| EEG -                       | 1                                     | 57                            | Specificity = $\frac{57}{5+57} = 92\%$ |
|                             | True Positive<br>(EEG +)              | True Negative<br>(EEG -)      |  |
| SSEP +<br>(amplitude < 50%) | 2                                     | 4                             | Sensitivity = $\frac{2}{2+4} = 33\%$   |
| SSEP -                      | 4                                     | 56                            | Specificity = $\frac{56}{4+56} = 93\%$ |

Comparison of SSEP monitoring with simultaneous 16-channel EEG monitoring suggests that SSEP monitoring may be slightly more sensitive in its efficacy in detecting cerebral ischemia of a magnitude that may cause a neurologic deficit. At the same time, amplitude reduction of  $N_{20}$  greater than 50% appears to have a specificity similar to that of EEG. However, because of the small number of patients with neurologic deficits in the series, the observed difference clearly was not statistically significant. Although the lack of difference may have arisen from a type II error, the power analysis has put this into perspective; the large number of patients required to demonstrate any significant difference would render it meaningless clinically, because the number of patients who potentially could benefit from this improvement in sensitivity and specificity of monitoring would be exceedingly small.

The rationale for monitoring electrophysiologic function during carotid endarterectomy is based on the need to prevent intraoperative stroke. Monitoring of SSEP is a logical alternative to conventional EEG because it specifically monitors the function of the sensory cortex, an area chiefly supplied by the middle cerebral artery, which is the major territory placed at risk during clamping of the carotid artery. The basic physiologic characteristics of SSEP also have been well elucidated in animal models. The amplitude of the primary sensory cortical component decreases and the latency increases as ischemia develops, and in primate studies it is abolished when regional cerebral blood flow (rCBF) decreases to less than  $12 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$ .<sup>19</sup> This contrasts with the critical rCBF associated with EEG changes of  $10\text{--}18 \text{ ml} \cdot 100 \text{ g}^{-1} \cdot \text{min}^{-1}$  during carotid endarterectomy.<sup>20,21</sup>

Several earlier studies investigated the use of SSEP monitoring during carotid endarterectomy<sup>6-15</sup>; however,

there was no agreement on what criteria should be used for interpretation or, indeed, on what component of the SSEP should be used for analysis. Both short-latency and long-latency cortical components have been used. Early studies used the long-latency components; Markand *et al.*<sup>8</sup> used them to determine the need for shunting, and Jacobs *et al.*<sup>7</sup> demonstrated a correlation between changes in these long-latency potentials and the development of postoperative neuropsychologic disturbances. The use of the primary short-latency component ( $N_{20}$ ) in recent studies is more appropriate because the late cortical components are more susceptible to anesthetic influence, which may interfere with interpretation.<sup>22</sup> We therefore chose to evaluate the  $N_{20}$  component for the current study.

Isoflurane was used as our basal anesthetic because we observed previously that the incidence of major EEG changes was less during isoflurane than during halothane,<sup>16</sup> an observation since confirmed by Michenfelder *et al.*<sup>23</sup> The anesthetic depth was held constant so that any potential anesthetic-induced deterioration of SSEP would be eliminated. Blood pressure also was maintained relatively unchanged from the preclamping period to eliminate any potential influence on SSEP. Body temperature also can affect SSEP significantly; therefore, we kept the temperature near normal levels.

The previous reports did not systematically evaluate the usefulness of SSEP monitoring in the prevention of stroke or its efficacy relative to conventional EEG monitoring. The mere demonstration that insertion of a shunt based on SSEP changes is associated with intact postoperative neurologic function does not prove that such monitoring is useful. Therefore, the sensitivity and specificity of this monitoring modality must be validated. (A

highly sensitive test would have a low false-negative rate, whereas a highly specific test would have a low false-positive rate. The calculations are shown in table 3.) Our standard surgical practice does not use shunting and therefore provides a unique opportunity to accurately assess the potential utility of SSEP monitoring as well as its efficacy relative to conventional EEG monitoring during carotid endarterectomy. Although the number of patients in this series is relatively small, to our knowledge this is the only study other than the preliminary study of McPherson *et al.*\*\* in which SSEP and conventional EEG were monitored simultaneously and shunting was not used irrespective of changes.

Because no established criteria for SSEP changes exist, and both amplitude and CCT<sup>24</sup> have been used as indices of ischemia, we arbitrarily classified the changes into four categories: 1) unchanged N<sub>20</sub>; 2) a decrease in amplitude greater than 50% of baseline; 3) CCT increase greater than 1 ms; and 4) a decrease in amplitude greater than 50% and an increase in CCT greater than 1 ms. We did not find the use of CCT beneficial, and the best combination of sensitivity (100%) and specificity (94%) was achieved with the use of the criterion of amplitude alone (grade 2 + grade 4, because the latter also met the criterion). In contrast, using all SSEP changes (grades 2-4) as a diagnostic test for postoperative deficit would yield a sensitivity of 100% and a specificity of 87%, but the corresponding values for the latency criterion (grades 3 and 4) are 0% and 87%, respectively. Consistent with some experimental observations made on evoked potential and cerebral ischemia,<sup>25</sup> this confirms that amplitude change may be a better criterion to use than latency change as an index of cerebral ischemia.

An alternative view is that latency changes occur early, and therefore may be more sensitive indicators of ischemia but may be less specific. Indeed, grade 3 patients included two patients with EEG changes. SSEP generally is considered to be more robust than EEG,<sup>25</sup> but our series indicates that including both latency and amplitude criteria would lead to a higher incidence of changes compared with conventional EEG. With regard to sensitivity and specificity, although SSEP monitoring (amplitude criterion) seems to compare favorably with EEG monitoring, such comparison probably is not meaningful because of the small number of patients with postoperative neurologic deficits. Excluding patients with transient EEG changes would increase the specificity to 95% without changing the sensitivity.

Although the number of patients with changes is small, we have demonstrated a correlation between SSEP changes during cross-clamping and the subsequent development of neurologic deficit. Despite the reversal of the SSEP changes, transient neurologic deficits developed

in two patients. This apparent discrepancy can be explained by the fact that SSEPs are specific to the sensory cortex or its subcortical pathway, whereas the deficits observed were primarily motor deficits. Their proximity nevertheless allowed the SSEP changes to act as predictors. The lack of EEG changes in one of these two patients may be attributed to the ability of SSEP to detect subcortical structure ischemia, whereas EEG pertains largely to cortical structures, although there was no confirmation that this was indeed the case. Of note is that both patients with postoperative deficits had some preexisting abnormalities on their preclamp EEG, a fact that may have contributed to the lack of sensitivity for EEG and may be suggestive of a potential advantage of SSEP monitoring in these cases.

Our results are consistent with those of Russ *et al.*<sup>10</sup> and Amantini *et al.*,<sup>12</sup> who reported similar sensitivity and specificity. Gigli *et al.*,<sup>11</sup> in their study of 40 patients, also found that amplitude reduction is a better correlate with postoperative neurologic deficits than an increase in CCT. In contrast, De Vleeschauwer *et al.*<sup>13</sup> described a low sensitivity (high false-negative rate), reporting three patients with postoperative neurologic deficits who had unchanged SSEPs intraoperatively. This is the only series that reported a relatively high false-negative rate. A summary of previous investigations, complete with their sensitivity and specificity in detection of postoperative neurologic deficits (transient and permanent), is listed in table 4. None of these studies, however, truly assesses the usefulness of SSEP monitoring because either routine shunting or selective shunting according to SSEP changes was used before the validity of SSEP as an intraoperative monitor of cerebral ischemia was ascertained. Moreover, there was no comparison with conventional EEG. Our results suggest that amplitude reduction is a useful criterion for SSEP monitoring during carotid endarterectomy. Because the interpretation of SSEP using the 50% amplitude reduction is relatively straightforward, this would simplify its usage.

It should be mentioned that SSEP monitoring, *per se*,

TABLE 4. Comparative Sensitivity and Specificity of SSEP Monitoring during Carotid Endarterectomy

| Author(s)                                  | Patients (n) | Sensitivity (%) | Specificity (%) | Year |
|--|--------------|-----------------|-----------------|------|
| Lam <i>et al.</i>                          | 64           | 100*            | 94              | 1991 |
| Russ <i>et al.</i> <sup>9</sup>            | 149          | 86*             | 96              | 1988 |
| DeVleeschauwer <i>et al.</i> <sup>13</sup> | 156          | 40              | 83              | 1988 |
| Amantini <i>et al.</i> <sup>12</sup>       | 58           | 100             | 98              | 1987 |
| Gigli <i>et al.</i> <sup>11</sup>          | 40           | 100             | 83              | 1987 |
| Russ <i>et al.</i> <sup>10</sup>           | 106          | 83              | 99              | 1985 |
| Markand <i>et al.</i> <sup>8</sup>         | 36           | ?               | ?               | 1984 |
| Jacobs <i>et al.</i> <sup>7</sup>          | 25           | ?               | ?               | 1983 |

\* Amplitude < 50% is used as the criterion.

does not distinguish between the embolic cause *versus* the hemodynamic cause, although Amantini *et al.*<sup>12</sup> suggested that the former usually is associated with an abrupt disappearance of N<sub>20</sub>, whereas the latter is associated with a gradual decline of the amplitude. Because the insertion of an internal shunt usually will ameliorate ischemia resulting from inadequate collateral flow during cross-clamping, but would be of little benefit for embolic causes and may even aggravate an embolic stroke because shunting itself carries an embolic risk, the ability to distinguish between these two etiologic factors would allow the most appropriate therapeutic treatment. However, because we had only a relatively small number of patients with changes, we could not address this issue.

Although the objective of the study was to compare conventional EEG with SSEP monitoring, our results also illustrate the difficulty in establishing the role of electrophysiologic monitoring during carotid endarterectomy. The incidence of EEG and SSEP changes is small in our series, and, despite the withholding of surgical or anesthetic intervention, no patient had a fixed neurologic deficit develop. These results, however, are entirely consistent with Ferguson's previous observation that EEG changes often do not result in postoperative neurologic deficit.<sup>26</sup>

It is not surprising that false-positive results occur, because an ischemic penumbra can exist where blood flow is inadequate for generation of electrical activity and yet sufficient to maintain neuronal viability.<sup>25</sup> Whether this ischemic zone will return to normal function or progress to infarction probably depends on the duration of ischemia as well as the quantity of residual flow. Thus, transient changes result in no deficits, whereas persistent changes (none occurred in our current series) will be expected to result in neurologic deficits. The fact that on an experimental basis the ischemic penumbra appears to be smaller with SSEP compared with EEG<sup>19,25</sup> suggests that the false-positive rate may be lower with SSEP than with EEG, a contention supported but not confirmed by our findings. It is unlikely, however, that refinement of criteria can eliminate false-positive results. One also can argue that major changes in EEG during cross-clamping always represent warning signals and should not be interpreted as false-positive results, even if neurologic deficits do not occur, and the insertion of an internal shunt with its attendant risks is not necessarily justified. If we accept this premise and use EEG as the gold standard, then SSEP monitoring using amplitude as the only criterion has a sensitivity of 33% and specificity of 93% (table 3).

Nevertheless, the thrust of our study is the comparison between SSEP and conventional EEG in prediction of postoperative neurologic deficits, and, based on our find-

ings, we conclude the following: 1) SSEP monitoring is feasible and simple to use during carotid endarterectomy; 2) transient changes in SSEP are of no consequence; 3) no new neurologic deficits occur with an unchanged SSEP (N<sub>20</sub>); 4) an amplitude reduction greater than 50% during cross-clamping may indicate the onset of cerebral ischemia of important magnitude; and 5) compared with conventional EEG, SSEP monitoring appears to have similar efficacy in the prediction of postoperative neurologic deficits.

The authors thank Dr. W. T. Blume for providing the EEG interpretations.

### References

1. Fode NC, Sundt TM Jr, Robertson JT, Peerless SJ, Shields CB: Multicenter retrospective review of results and complications of carotid endarterectomy in 1981. *Stroke* 17:370-376, 1986
2. Toronto Cerebrovascular Study Group: Risks of carotid endarterectomy. *Stroke* 17:848-852, 1986
3. Ferguson GG: Intra-operative monitoring and internal shunts: Are they necessary in carotid endarterectomy? *Stroke* 13:287-289, 1982
4. Sundt TM Jr: The ischemic tolerance of neural tissue and the need for monitoring and selective shunting during carotid endarterectomy. *Stroke* 14:93-98, 1983
5. Ferguson GG: Carotid endarterectomy—To shunt or not to shunt? *Arch Neurol* 43:615-617, 1986
6. Lam AM, Teturswamy G: Monitoring of evoked responses during carotid endarterectomy and extracranial-intracranial anastomosis. *Int Anesthesiol Clin* 22:107-112, 1984
7. Jacobs LA, Brinkman SD, Morrell RM, Shirley JG, Ganji S: Long-latency somatosensory evoked potentials during carotid endarterectomy. *Am Surg* 49:338-344, 1983
8. Markand ON, Dilley RS, Moorthy SS, Warren C Jr: Monitoring of somatosensory evoked responses during carotid endarterectomy. *Arch Neurol* 41:375-378, 1984
9. Russ W, Thiel A, Moosdorf R, Hempelmann G: Somatosensory evoked potentials bei desobliterierenden Eingriffen an der Carotisgabel. *Klin Wochenschr* 66(Suppl 14):35-40, 1988
10. Russ W, Fraedrich G, Hehrlein FW, Hempelmann G: Intraoperative somatosensory evoked potentials as a prognostic factor of neurologic state after carotid endarterectomy. *Thorac Cardiovasc Surg* 33:392-396, 1985
11. Gigli GL, Caramia M, Marciani MG, Zarola F, Lavaroni F, Rossini PM: Monitoring of subcortical and cortical somatosensory evoked potentials during carotid endarterectomy: Comparison with stump pressure levels. *Electroencephalogr Clin Neurophysiol* 68:424-432, 1987
12. Amantini A, De Scisciolo G, Bartelli M, Lori S, Ronchi O, Pratesi C, Bertini D, Pinto F: Selective shunting based on somatosensory evoked potential monitoring during carotid endarterectomy. *Int Angiol* 6:387-390, 1987
13. De Vleeschauwer P, Horsch S, Matamoros R: Monitoring of somatosensory evoked potentials in carotid surgery: Results, usefulness and limitations of the method. *Ann Vasc Surg* 2:63-68, 1988
14. De Vleeschauwer P, Horsch S, Haupt WF, Huber P: The use of

- somatosensory evoked responses in carotid surgery for monitoring brain function. *Acta Chir Belg* 85:293-298, 1985
15. Thiel A, Russ W, Nestle HW, Hempelmann G: Early detection of cerebral ischemia during carotid endarterectomy using transcranial doppler sonography and somatosensory evoked potentials. *Thorac Cardiovasc Surg* 37:115-118, 1989
  16. Blume WT, Ferguson GG, McNeill DK: Significance of EEG changes at carotid endarterectomy. *Stroke* 17:891-897, 1986
  17. Haynes RB: How to read clinical journals: II. To learn about a diagnostic test. *Can Med Assoc J* 124:703-710, 1981
  18. Zar JH: *Biostatistical Analysis*. Englewood Cliffs, Prentice-Hall, 1984, pp 398
  19. Branston NM, Symon L, Crockard HA, Pasztor E: Relationship between the cortical evoked potential and local cortical blood flow following acute middle cerebral artery occlusion in the baboon. *Exp Neurol* 45:195-208, 1974
  20. Sharbrough FW, Messick JM Jr, Sundt TM Jr, O'Fallon WM: Correlation of continuous electroencephalograms with cerebral blood flow measurements during carotid endarterectomy. *Stroke* 4:674-683, 1973
  21. Messick JM Jr, Casement B, Sharbrough F, Milde LN, Michenfelder JD, Sundt TM Jr: Correlation of regional cerebral blood flow (rCBF) with EEG changes during isoflurane anesthesia for carotid endarterectomy: Critical rCBF. *ANESTHESIOLOGY* 66:344-349, 1987
  22. Verma NP, Peters GM, Jacobs LA, Dahn MS, King SD: An assessment of the variability of early scalp-components of the somatosensory evoked response in uncomplicated, unshunted carotid endarterectomy. *Clin Electroencephalogr* 16:157-160, 1985
  23. Michenfelder JD, Sundt TM, Jr, Fode N, Sharbrough FW: Isoflurane when compared to enflurane and halothane decreases the frequency of cerebral ischemia during carotid endarterectomy. *ANESTHESIOLOGY* 67:336-340, 1987
  24. Symon L, Hargardine J, Zawirski M, Branston NM: Central conduction time as an index of ischaemia in subarachnoid hemorrhage. *J Neurol Sci* 44:95-103, 1979
  25. Ropper AH: Evoked potentials in cerebral ischemia. *Stroke* 17:3-5, 1986
  26. Ferguson GG, Gamache FW Jr: *Cerebral protection during carotid endarterectomy: Intraoperative monitoring, anesthetic techniques, and temporary shunts*, *Stroke and the Extracranial Vessels*. Edited by Smith RR. New York, Raven Press, 1984, pp 187-201