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Somatosensory Evoked Potentials and Electroencephalography During Carotid Endarterectomy Predict Late Stroke But Not Death

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Abstract

Objective—Late stroke and death rates are anticipated to be higher in patients undergoing carotid endarterectomy (CEA) compared to healthy counterparts. However, little is known regarding predictors, other than the baseline comorbidities. We have recently shown that dual intraoperative Somatosensory Evoked Potentials (SSEP) and electroencephalogram (EEG) monitoring improves the ability to predict perioperative strokes. We seek to determine if dual intraoperative monitoring (IOM) can further predict long-term strokes and death.

Methods—Consecutive patients who underwent CEA under dual SSEP and EEG intraoperative monitoring between 1/1/2000 and 12/31/2010 were analyzed. Patients were divided in two groups, those with and those without IOM changes. IOM changes were classified as either occurring during carotid cross clamp placement or at any time during the operation. Endpoints were time to stroke and death. Log rank tests and Cox regression analysis were used to identify predictors of postoperative stroke and death.

Results—A total of 853 CEAs (mean age 70.6±9.5 years, 58.7% male, 38.9% symptomatic) were performed during the study period with a mean clinical follow up of 48±38 months. 107 patients (13.6%) had significant SSEP or EEG changes at the time of clamping, while considerably more patients (217, 25.4%) had SSEP and/or EEG changes recorded at any point during the procedure, including during clamping. Baseline characteristics including rates of bilateral disease, statin use, and antiplatelet use, were similar between groups. Female gender, symptomatic disease, and significant contralateral carotid stenosis were more frequent in the group with IOM changes. The overall stroke-free survival rate at 5 years was significantly higher in patients without IOM changes (94.7% vs. 88.2%, $p<0.05$) and at 10 years (86.1% vs. 78.0%, $p<0.05$). Despite differences in stroke-free survival, overall survival at 10 years was not different

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between groups (44.0% in patients with IOM changes vs. 42.8% in patients without, $p=0.7$). Renal insufficiency (HR = 2.13, $p=0.03$), diabetes (HR 1.84, $p=0.005$), and age greater than 80 at the time of operation (HR=3.24, $p=0.001$) were significant predictors of late stroke, while statins were significantly protective (HR = 0.55, $p=0.05$). Controlling for these factors, IOM changes (HR = 2.5, $p=0.004$) were a strong predictor of long-term risk of stroke after CEA.

Conclusion—Intraoperative SSEP and/or EEG changes are predictive of late stroke but not death following CEA indicating a need for further elucidation and management of the underlying risk factors driving the elevated stroke risk in this subset of CEA patients.

Introduction

The efficacy of carotid endarterectomy (CEA) for stroke risk reduction has been established through landmark randomized trials on both symptomatic and asymptomatic patients. While the perioperative morbidity and mortality of modern carotid endarterectomy is quite low[1–3], the long-term risks for patients with this disease process remain elevated compared to healthy counterparts. Patients remain at above average risk of future strokes and death even after carotid revascularization[4]. Data from the major symptomatic trials showed a 5-year risk of any stroke after CEA was 17–20%. The 5-year stroke rate was 12.4% in the Asymptomatic Carotid Atherosclerosis Study (ACAS) and 6.9% at 5 years in the Asymptomatic Carotid Surgery Trial (ACST).[5–7] Several recent reviews of both symptomatic and asymptomatic patients have reported long term stroke rates after CEA of 13–16% up to 10 years after surgery [4, 8]. Carotid revascularization helps prevent stroke attributed to large artery territory, however many patients have other risk factors for stroke making it difficult to predict patients' overall benefit from surgery. Several baseline factors such as age and gender and multiple medical comorbidities have all been associated with an increased risk of late stroke after CEA[8]. In addition, long-term survival is substantially reduced in patients undergoing carotid surgery compared to healthy counterparts [2].

Efforts to reduce perioperative morbidity and mortality include intraoperative monitoring (IOM) techniques to determine the need for selective intraluminal shunting. [9] One such method of detecting cerebral hypoperfusion during CEA is neurophysiologic monitoring with electroencephalography (EEG) and/or somatosensory evoked potentials (SSEP) [10]. Dual intraoperative monitoring, using both SSEP and EEG, has increased sensitivity to detect cerebral hypoperfusion and predict perioperative stroke[10].

Our hypothesis is that IOM changes may suggest an overall decreased cerebrovascular reserve and elevated risk for long-term stroke and death. Cerebrovascular reserve is the ability of the cerebral circulation to increase blood flow in response to various physiological and pharmacological stimuli[11]. It has been shown that patients with poor cerebrovascular reserve as measured by transcranial doppler ultrasound have increased risk of long term strokes[12]. However, the predictive value of the intraoperative monitoring changes on long-term stroke and mortality remains unknown. Our primary aim is to determine if IOM changes noted during the time of CEA might further predict long-term stroke and death.

Methods

Study Design

Consecutive individuals who underwent CEA by the Division of Vascular Surgery at the University of Pittsburgh between 1/1/2000 and 12/31/2010 were identified by the Current Procedural Terminology (CPT) code 35301. The records were reviewed for demographics, baseline risk factors, carotid duplex studies, operative indications, intraoperative data, perioperative complications, and follow-up events. The social security death index was used to record death dates. Patients were included if they had undergone carotid endarterectomy under dual modality neurophysiologic monitoring. Patients who underwent concomitant cerebrovascular procedures (e.g., carotid-to-subclavian bypass) or concomitant coronary artery bypass grafting were excluded from the study. Endpoints were stroke and death occurring any time after the operation.

Definitions

All baseline risk factors were recorded based on established diagnoses identified in the medical records. Preoperative parameters of interest included age, gender, and a history of any of the following: hypertension, diabetes mellitus, chronic renal insufficiency (CRI), Hypertension, smoking, coronary artery disease (CAD), and congestive heart failure (CHF). CRI was defined as serum creatinine consistently greater than 1.5 mg/dL. Preoperative and discharge medications were also documented. Symptomatic carotid stenosis was defined as a preprocedural ipsilateral ischemic stroke or transient ischemic attack occurring in the 6 months prior to operation[13]. Post-operative stroke was defined as stroke occurring within 30 days of the operation. After 30 days only the first ipsilateral and/or contralateral stroke were recorded and these were defined as follow up strokes.

Carotid Endarterectomy

Carotid endarterectomy was performed under general anesthesia in all cases. Closure technique (primary, patch, or eversion) and shunt use (selective vs routine) were variable and based on physician preference.

Neurophysiologic Monitoring

All patients underwent both EEG and SSEP monitoring. EEG was recorded using electrodes placed on the scalp according to the International 10–20 system. EEG was recorded from these eight channels F3-P3, P3-O1, F3-T3, T3-O1, F4-P4, P4-O2, F4-T4, and T4-O2. The 60 Hz notch filter was utilized and band pass filtering set from 1–70 Hz. Sensitivity varied between 5–7 microvolts/div and a time base of 30 mm/sec was utilized. For generation of SSEPs, the left and right median or ulnar nerves were independently stimulated at the wrist using subdermal needle electrode pairs. For SSEP recording, scalp electrodes were placed at P4/Fz and P3/Fz (according to the international 10–20 system) to record the thalamocortical potentials (N20/P30), an electrode was localized on the mastoid and referenced to Fz to record the brainstem potential (dorsal column nucleus) and electrodes were placed at the bilateral erbs point referenced to one another to record the peripheral potential generated in the brachial plexus. Constant current stimulation at intensities sufficient to evoke a

consistent and supra-maximal response was used for generation of SSEPs. Stimulation frequency was set at 3.43 Hz with duration of 0.2 ms. Band pass filters were set at 10–300 Hz with a gain of 20k for cortical recordings and 30-1K Hz with a gain of 50k for brainstem recordings and erbs point recordings. Averages were computed for at least 128 trials.

Significant SSEP changes were defined as a persistent and consistent prolongation of latency of 10% or 50% decrease in the amplitude in greater than 2 averaged trials [14]. Significant EEG changes were defined as a decrease in the amplitude of fast frequency by more than 50% or increase in theta or delta activity by more than 50% [15]. IOM changes were classified as occurring anytime during the operation or as clamp-induced changes, which were defined as changes occurring during the first 5 minutes of carotid cross clamp.

Statistical Analysis

Patients were divided in two groups, those with and those without IOM changes. Those with IOM changes were further divided into those with clamp-induced changes and those with changes at other time points during the operation (Figure 1). Descriptive characteristics are reported as means $\pm$ standard deviations or as number of cases and percentages. Baseline characteristics were compared by chi-squared tests for categorical data and students t-test for continuous data. Survival functions were estimated by the Kaplan Meier method and evaluated using a log-rank test. Cox regression models were used to assess the effect of baseline predictors. Time zero was defined as the date of the initial procedure. For stroke-free survival analyses, cases were censored on the date of the last recorded clinical follow-up that allowed accurate information collection, unless the studied event occurred at an earlier time point. For the mortality end point, events were recorded on the date of death according to the social security death index. Patients were censored on the date of the last available clinical follow-up. Results were considered statistically significant when p value was less than 0.05. Data analysis was performed using Stata Statistical Software: Release 14 (College Station, TX: StataCorp LP.).

Results

Over the 11-year study period, 805 patients underwent 853 carotid endarterectomies under dual SSEP and EEG monitoring. Mean clinical follow up was 48.5 ± 38.0 months. 38.9% of patients underwent CEA for symptoms experienced within 6 months of the procedure (49% of those for documented stroke), 58.7% were men, and the mean age of the cohort was 70.6 ± 9.5 years. Baseline characteristics, including rates of statin and antiplatelet use and comorbidities such as diabetes and coronary artery disease were similar between groups. Female gender, symptomatic disease, and significant (70%) contralateral stenosis were more common in the group with intraoperative monitoring changes (Table 1). 217 patients (25.4%) had SSEP and/or EEG changes recorded at any point during the procedure, but only 107 patients (13.6%) had changes at the time of clamping.

The post-operative stroke rate was higher in patients with any IOM changes during the operation (2.76% v. 0.79%, $p=0.02$). The difference was greater for patients with clamp-induced IOM changes (4.67% vs. 0.88%, $p=0.002$). In patients with IOM changes that occurred before cross-clamp or after the fifth minute of carotid clamping, no excess rate of

perioperative stroke was seen compared to those with no changes (0.90% vs. 0.85%, $p=0.96$). Table 2.

The overall stroke-free survival in the cohort was 93% at 5 years and 83.9% at 10 years. The stroke-free survival rate was significantly better in patients without IOM changes at both 5 years (94.7% vs. 88.2%) and 10 years (86.1% vs. 78.0%), $p=0.002$ (Figure 2). Stroke-free survival in patients with clamp-related IOM changes was 88.6% at 5 years and 75.0% at 10 years compared to 95.7% at 5 years and 89.2% at 10 years in patients without changes, $p=0.004$ (Figure 3). Finally, while the perioperative stroke rate was not affected, the long-term stroke-free survival rate was similarly reduced in patients that had IOM changes *unrelated* to clamping compared to patients without any IOM changes at 5 years (90.4 vs. 96.6%) and at 10 years (85.0% vs. 90.4%), $p=0.04$ (Figure 4).

Renal insufficiency was a predictor of late stroke ($HR = 2.13$, $p=0.03$), as was diabetes ($HR=1.84$, $p=0.05$) and age greater than 80 ($HR=3.24$, $p=0.001$) while statins were protective ($HR=0.55$, $p=0.05$). Controlling for these factors as well as the potential confounders of contralateral occlusion and symptomatic status, IOM changes were a strong predictor of long-term risk of stroke after CEA ($HR=2.5$, $p=0.004$). Both clamp-induced IOM changes ($HR = 2.46$, $p=0.008$) and non-clamp induced changes ($HR=2.17$, $p=0.04$) alone were independent predictors of late stroke as well in the risk-adjusted model (Table 3).

Overall survival in our cohort was 72.3% and 43.7% at 5 years and 10 years respectively. Overall survival at 10 years was not different between groups: 42.8% in patients with IOM changes vs. 44.0% in patients without, $p=0.5$ (Figure 5). For the asymptomatic cohort alone, the overall survival at 10 years was 47.1% in patients without IOM changes vs. 47.0% in patients with IOM changes, $p=0.7$. Overall survival was also not predicted by clamp related IOM changes (Figure 6) or non clamp-related changes (Figure 7). Predictors of decreased overall survival in this cohort were age > 80 ($HR = 3.22$, $p<0.001$), diabetes ($HR=1.29$, $p=0.07$), COPD ($HR=1.40$, $p=0.04$), a symptomatic indication for surgery ($HR=1.75$, $p<0.001$), CHF ($HR=2.65$, $p<0.001$), and renal insufficiency ($HR=2.5$, $p<0.001$). Statins were protective ($HR = 0.79$, $p=0.05$). Postoperative stroke was not a predictor of mortality ($p=0.8$).

Discussion

Patients with clamp or non-clamp related IOM changes are not only at risk for perioperative stroke but remain at elevated risk of late stroke compared to patients who do not have EEG or SSEP changes in the operating room. We have also shown that patients who experience non-clamp-induced SSEP and EEG changes may not be at increased risk of stroke in the 30-day postoperative period, but are at increased risk of stroke in the long term. The risk of late stroke is further increased in patients older than 80, with diabetes, or with renal insufficiency. Late stroke risk is reduced with statins. Despite these differences in stroke rates, we did not demonstrate a difference in overall survival at 5 and 10 years for patients who experienced clamp-induced IOM changes or changes at any time during the operation.

It has been shown that the mere presence of single vessel occlusive disease alone is an inadequate indicator of the risk of stroke and treatment with carotid endarterectomy decreases the long-term risk of stroke rather than bringing it down to baseline risk[13]. Changes in intraoperative monitoring with SSEP and EEG can predict an elevated long-term risk of stroke but not death following CEA. The pathophysiology of this phenomenon cannot be fully explained from retrospective data, but may implicate reduced cerebrovascular reserve in these patients who experience either clamp-induced or subtler IOM changes during CEA (ie those changes that occur at other time during the operation). There is an ongoing need for further elucidation and management of the underlying risk factors driving the elevated stroke risk in this subset of CEA patients.

Follow up from the early carotid surgery trials first demonstrated the elevated long-term stroke risk in patients with carotid stenosis even after CEA. Cunningham et al, analyzed the long-term stroke risk in the European Carotid Study Trial (ECST) cohort and found that patients undergoing CEA remained at approximately double the risk for ipsilateral ischemic stroke compared to the background risk patients with less than 30% carotid stenosis [16]. They identified presentation with cerebral symptoms, diabetes, elevated systolic blood pressure, smoking, male sex, increasing age, and a lesser severity of preoperative stenosis as risk factors for late stroke after CEA[16]. Even with modern medical therapy, the long-term rates of stroke after CEA remain substantially above baseline rates.

A number of comorbidities have been identified in the literature as predictors of late stroke which were also identified in our cohort. Renal insufficiency has been repeatedly identified as an independent risk factor for stroke[4, 18, 19]. Statins, conversely, have repeatedly been associated with decreased long-term stroke risk[20]. Symptomatic disease has been shown to have long-term ramifications for stroke risk as well [21]. Kang et al retrospectively analyzed 3,014 carotid endarterectomies performed between 1989 and 2004 (33.5% symptomatic) and found an overall 93.8% stroke-free survival at 4 years and an 86.9% stroke-free survival at 10 years which is comparable to our cohort drawn from a similar time period (93.0% at 5 years an 83.9% at 10 years)[8]. Kang et. al. identified several risk factors of long-term stroke including diabetes, symptomatic disease, male gender, and increasing age[8]. We similarly found a trend towards increased late stroke risk in older patients, patients with diabetes and renal insufficiency and re-demonstrated the protective effect of statins. However, this is the first study to identify changes during intraoperative monitoring with SSEP or EEG as highly predictive of long-term stroke risk. This marks a previously unidentified group of patients that are at increased risk of long-term stroke.

As mentioned, patients with carotid disease undergoing CEA remain at elevated risk of death even after the perioperative period[2, 22]. Overall survival in our cohort is on par with multiple recent series[2, 8]. In a post hoc analysis of the Carotid Revascularization Endarterectomy versus Stenting Trial (CREST) data, Hill et al found that patients that suffered perioperative stroke were at a nearly 3-fold increased risk of future mortality compared to those without stroke. The 4-year mortality was 21.1% in the periprocedural stroke group compared to 11.6% in those without a stroke[23]. A number of predictors of later mortality after CEA have been previously elucidated. Ballotta et al found diabetes and coronary disease to be independent predictors of late death following carotid

revascularization surgery [24]. Renal insufficiency has been shown to predict mortality, and it has been suggested that for patients on dialysis, the risks of CEA outweigh the benefits in patients with asymptomatic disease [4, 25]. Female gender and statin use have been demonstrated to be protective [8]. In our cohort, age greater than 80, diabetes, COPD, symptomatic disease, CHF, and renal insufficiency were all predictors of late mortality. Though we found IOM changes were predictive of long-term stroke after carotid endarterectomy, we were unable to demonstrate an increased risk of death in this subset of patients.

Contralateral carotid occlusion, vertebral artery disease, an incomplete circle of Willis, and an increased burden of intracranial vascular disease are believed to increase the likelihood of IOM changes during clamp placement[26, 27]. In addition, many patients experience changes in intraoperative monitoring during CEA with fluctuations in anesthesia, blood pressure, or heart rate with or without a clamp on the carotid artery[28]. Studies in awake patients show a smaller number of patients require shunts than have recorded SSEP or EEG changes[28–30]. This points to the sensitivity of neurophysiological monitoring. It is clear that cross clamping and hemodynamic changes during surgery result in decreased cerebral blood flow in a number of patients but this decrease is not always functionally important (ie, requiring a shunt). However, our study showed that these changes as well as clamp-induced changes are a significant predictor of increased risk of stroke up to ten years post-operatively.

The results of our study need to be interpreted with caution due to several limitations. This is a retrospective study. Risk factors, postoperative and long term events were determined from patient electronic medical record reviews and chart diagnoses. Medication use is subject to patient adherence which was not captured in this data. Moreover, it is possible that patients with limited follow-up developed neurologic events and were treated elsewhere so that symptomatic patients would be under-represented in our analysis. Transient ischemic attacks during the follow cannot be accurately captured so our analysis accounted for strokes only. Nonetheless, we identified comorbidities placing patients at increased risk of late stroke or death consistent with contemporary literature adding strength to our identification of IOM changes as predictors of late events.

Conclusion

Changes in intraoperative SSEP and EEG monitoring are predictive of long-term stroke risk in patients undergoing carotid endarterectomy. Patients are at elevated risk of long-term stroke whether these changes occur during carotid cross-clamping or at another time during the procedure. Further study is necessary to understand the anatomical and pathophysiological mechanisms underlying this phenomenon with the ultimate goal of risk factor reduction.

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Figure 1.

Figure 2.

Figure 3.

Figure 4.

Figure 5.

Figure 6.

Figure 7.

Table 1

Baseline Characteristics

	IOM changes	No IOM changes	p value
Male Gender	53%	60.7%	0.05 *
Age (years)	70.8±9.6	70.6±9.5	0.8
Symptomatic	49.8%	35.3%	<0.001 *
Diabetes	34.7%	29.9%	0.2
CAD **	54.0%	50.5%	0.4
Hypertension	87.9%	86.2%	0.5
CHF **	11.4%	7.3%	0.06
GFR **	67.3±20.8	68.5±23.2	0.5
Smoking	21.0%	23.0%	0.6
Statin use	68.8%	67.4%	0.7
Antiplatelet use	89.6%	88.5%	0.7
Contralateral disease > 70%	34.56%	26.37%	0.02 *

* denotes statistically significant difference

** CAD = Coronary Artery Disease, CHF = Congestive Heart Failure, GFR = Glomerular Filtration rate

Table 2

Postoperative stroke rates

Grouped by:	Changes recorded	No changes recorded
All IOM changes	6/217 2.76%	5/636 0.79%
Clamp-induced IOM changes	5/107 4.67%	6/681 0.88%
Only changes not associated with clamping	1/111 0.90%	5/586 0.085%

Table 3

Multivariate Cox model for late postoperative stroke

Predictor	Hazards Ratio	p value
Renal insufficiency	2.13	0.03
Statins	0.55	0.05
Age > 80	3.24	0.001
Diabetes	1.84	0.05
Contralateral stenosis	1.23	0.4
Symptomatic	1.04	0.9
IOM changes	2.50	0.004