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Risk factors for cranial nerve deficits during carotid endarterectomy: A retrospective study

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ABSTRACT

Background: Cranial nerve deficits during CEA are a known complication. The purpose of this study is to evaluate if significant changes in somatosensory evoked potentials and electroencephalography increase cranial nerve deficits during CEA.
Procedures: This is an observational retrospective case–control study analyzed with data collected from patients who underwent CEA at the University of Pittsburgh Medical Center. Five hundred and eighty-seven patients were included in the final analysis. Due to the small number of cranial nerve deficits and the comparatively large number of potential covariates, we used a regression analysis with Bayesian shrinkage.
Findings: Analysis was performed on 587 patients, of which a total of 11 (1.8%) cases of cranial nerve deficits were recorded. The marginal mandibular branch of the facial nerve was injured in nine (81%) patients and hypoglossal nerve was injured in two (19%) patients. Of the 11 patients, 9 cases resolved by the time of discharge, the 2 cases that persisted both were injuries to the facial nerve. Multivariate analysis using Bayesian shrinkage showed that after adjusting for all risk factors only IOM changes increased the risk of cranial nerve deficits (OR 38.47, 95% CI 7.73, 191.42).
Conclusions: Cranial nerve injury is 38 times more likely in patients who experienced a change in IOM during CEA shunt. Future studies examining the effect of stretch and the degree of retraction on the CN might be more helpful in reducing cranial nerve deficits.

Carotid endarterectomy (CEA) is the procedure of choice to prevent stroke in symptomatic and asymptomatic patients with carotid artery stenosis [1–3]. Cranial nerve deficits during CEA is a known complication and commonly involves injury to the marginal mandibular nerve [4] resulting in inability to express emotions [5]; recurrent laryngeal nerve, resulting in vocal cord paralysis [6]; the hypoglossal nerve resulting in tongue weakness [7]; and the greater auricular nerve [4,8] resulting in numbness around the ear. Reports of the injury to glossopharyngeal nerve resulting in pharyngeal weakness, superior laryngeal nerve resulting in change in pitch of the voice [7], spinal accessory nerve resulting in difficulty with moving the head, and cervical sympathetic chain resulting in Horner's syndrome have also been documented [4,8]. The rate of cranial nerve deficits reported varies widely from 3% to 23% [8,9,4,10,11,6,12–14] in the literature with variations secondary to the type of research study [4], surgical technique [15], the year the procedure was performed [8] and increased awareness of the cranial nerve damage [8]. Though most of these cranial nerve deficits are transient it can lead to decreased quality of life [16] in addition to increased length of stay (LOS) after the CEA [17]. Decreased LOS can have a significant impact on the cost-effectiveness of CEA [18]. Clinical pathways for patients undergoing CEA can significantly reduce the LOS and its cost [18]. Identifying risk factors and strategies to prevent cranial nerve deficits can potentially decrease LOS and the overall cost of CEA.

Transient cranial nerve deficits after CEA could be secondary to inadvertent stretch and retraction of the cranial nerve (CN) [4],

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whereas permanent cranial nerve deficits could be secondary to prolonged stretch or complete transection. Very few studies have identified specific etiologies that can increase the risk of cranial nerve deficits during CEA. Increased stretching of cranial nerve could be secondary to increased length of surgery [4], the need to place a carotid shunt or the presence of a high carotid bifurcation. Intraoperative neurophysiological monitoring (IOM) using electroencephalography [19–21] (EEG) and somatosensory evoked potentials [22,23] (SSEPs) has been used as an aid for the use of shunting [24] during CEA to evaluate cerebral perfusion. The purpose of this study is to evaluate if significant IOM changes indicating ischemia and leading to carotid shunting increase cranial nerve deficits during CEA.

1. Clinical material and methods

1.1. Patient population and materials

This is an observational retrospective case–control study analyzed with data collected from patients who underwent CEA at the University of Pittsburgh Medical Center. Data from 911 patients who underwent CEA for carotid stenosis with intraoperative neurophysiological monitoring between 2007 and 2012 were reviewed for this study. Five hundred and eighty-seven patients were included in the final analysis. This was based on patients who had documented preoperative and postoperative neurological status evaluations done by the surgeon and the neurologist. Neurological status of the cranial nerves before and after CEA and co-morbid conditions were obtained from medical records. Preoperative documentation including complete history and physical examination was searched to account for any preexisting cranial nerve deficits. Specifically, the medical records were searched for the following terms: facial droop, swallowing difficulty, hoarseness of voice shoulder movement, and tongue movement difficulty. Risk factors collected included age, gender, ethnicity, significant ipsilateral and contralateral carotid stenosis (>70%) [20], vertebral disease (unilateral/bilateral and percentage of occlusion), repeat CEA, diabetes mellitus, body mass index (BMI), hypertension, coronary artery disease, hyperlipidemia, smoking status, symptomatic carotid stenosis [25] and length of hospital stay [17]. The study was approved by the IRB for retrospective review of data on human subjects at the University of Pittsburgh (MOD08120394-04/PRO08120394).

1.2. Intraoperative neurophysiological monitoring

Routine neurophysiological monitoring data recorded during CEA included electroencephalography (EEG) and somatosensory evoked potentials (SSEPs). Significant changes in either EEG or SSEP were an indication for shunting during CEA and were defined as follows. The EEG was recorded using 10-20 International system [26]. EEG amplitude attenuation of fast frequency (>12 Hz) by more than 50%, or an increase in the theta or delta amplitude by more than 50% was considered significant [20,27]. SSEPs elicited from the median nerve stimulation were collected as described previously [28]. We considered a 50% reduction in somatosensory cortical amplitude or a prolongation of response latency by >10% from baseline to be significant. Significant changes were changes in amplitude or latency of the SSEPs in >2 averaged trials. The primary reason for using >2 trials is to eliminate technical issues like noise. Data concerning any significant change from baseline EEG or SSEP, and type of change (temporary/permanent) were collected from neurophysiology records. EEG or SSEP changes that did or did not return to the baseline values at the end of the procedure were designated as temporary and permanent respectively. Significant changes in the EEG and/or SSEP resulted in placement of an intraluminal shunt.

1.3. Cranial nerve deficits

Cranial nerve complications were recorded in the perioperative period and also upon discharge. Any motor deficits involving the seventh, tenth, eleventh, twelfth cranial nerves and sympathetic chain (Horner's syndrome) and sensory deficits in the cervical dermatomes (3–4) in the post-operative period were documented and included in the study as complications. Cranial nerve deficits which resolved at discharge were labeled as transient, and those which did not resolve by discharge were designated as permanent. Patients with no documented cranial nerve injuries in the post-operative period were assumed to be free of the complication.

1.4. Statistical analysis

We performed exploratory analysis (box plots, stem and leaf plots, and outlier estimation) using cranial nerve deficits along with the risk factors to identify extreme values. The variables were reviewed for missing data before the analysis was performed. Missing data were assumed to be missing at random. Age, duration of surgery and length of stay were converted into categorical variables. We compared the risk factors to the cranial nerve deficits (yes/no) using chi-square statistic, and Fisher exact test if the expected counts were small. Odds ratios (OR) and confidence intervals (CI) were calculated for significant variables using standard methods. To analyze the effect of neurophysiology changes on cranial nerve deficits, multivariate analysis [4] was done to examine the effects of more than one risk factor at a time on cranial nerve deficits. Due to the small number of cranial nerve deficits and the comparatively large number of potential covariates, we used a regression analysis with Bayesian shrinkage. Bayesian shrinkage allows adjustment for all risk factors, thus reducing bias, while stabilizing the model by setting a prior distribution centered at zero with a large variance on the potentially confounding variables. If the potential confounder added to the regression analysis, then an effect would be estimated. If the potential confounder did not add to the regression analysis, then the estimates would remain at zero, the value of the prior mean. This technique is recommended so that all potential confounders can be incorporated while allowing the model to converge while providing valid estimates [29–31]. We used SPSS (version 22, IBM SPSS, Inc.) for statistical analysis of the data in this study.

2. Results

2.1. Clinical characteristics and cranial nerve injuries

Analysis was performed on 587 patients, of which a total of 11 (1.8%) cases of cranial nerve deficits were recorded (Table 1). The marginal mandibular branch of the facial nerve was injured in nine (81%) patients and hypoglossal nerve was injured in two (19%) patients. Of the 11 patients, 9 cases resolved by the time of discharge, the 2 cases that persisted both were injuries to the facial nerve. No patient had more than one cranial nerve deficits. No other sensory cranial nerve injuries were recorded. Baseline data on the patients with and without cranial nerve deficits are shown in Table 1. Patients with cranial nerve deficits had significantly higher IOM changes (81%) and neurological deficits (54.5%).