Limitations of median nerve somatosensory evoked potential monitoring during carotid endarterectomy

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OBJECTIVE Hypoperfusion during carotid artery cross-clamping (CC) for carotid endarterectomy (CEA) may result in the major complication of perioperative stroke. Median nerve somatosensory evoked potential (MNSSEP) monitoring, which is an established method for the prediction of cerebral ischemia, has low sensitivity in detecting such hypoperfusion. In this study the authors sought to explore the limitations of MNSSEP monitoring compared to tibial nerve somatosensory evoked potential (TNSSEP) monitoring for the detection of CC-related hypoperfusion.

METHODS The authors retrospectively analyzed data from patients who underwent unilateral CEA with routine shunt use. All patients underwent preoperative magnetic resonance angiography and were monitored for intraoperative cerebral ischemia by using MNSSEP, TNSSEP, and carotid stump pressure during CC. First, the frequency of MNSSEP and TNSSEP changes during CC were analyzed. Subsequently, variables related to stump pressure were determined by using linear analysis and those related to each of the somatosensory evoked potential (SSEP) changes were determined by using logistic regression analysis.

RESULTS A total of 94 patients (mean age 74 years) were included in the study. TNSSEP identified a greater number of SSEP changes during CC than MNSSEP (20.2% vs 11.7%; $p < 0.05$). Linear regression analysis demonstrated that hypoplasia of the contralateral proximal segment of the anterior cerebral artery (A_1 hypoplasia) ($p < 0.01$) and hypoplasia of the ipsilateral precommunicating segment of the posterior cerebral artery (P_1 hypoplasia) ($p = 0.02$) independently and negatively correlated with stump pressure. Both contralateral A_1 hypoplasia (OR 26.25, 95% CI 4.52–152.51) and ipsilateral P_1 hypoplasia (OR 8.75, 95% CI 1.83–41.94) were independently related to the TNSSEP changes. However, only ipsilateral P_1 hypoplasia (OR 8.76, 95% CI 1.61–47.67) was independently related to MNSSEP changes.

CONCLUSIONS TNSSEP monitoring appears to be superior to MNSSEP in detecting CC-related hypoperfusion. Correlation with stump pressure and SSEP changes indicates that TNSSEP, and not MNSSEP monitoring, is a reliable indicator of cerebral ischemia in the territory of the anterior cerebral artery.

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KEYWORDS carotid endarterectomy; hypoperfusion; somatosensory evoked potential; circle of Willis; collateral circulation; diagnostic technique

CAROTID endarterectomy (CEA) is a promising procedure for symptomatic and asymptomatic patients with severe carotid artery stenosis.^{2,5,16,27,28} Median nerve somatosensory evoked potential (MNSSEP) monitoring during CEA is useful in identifying intraoperative cerebral hypoperfusion, which is related to the risk of perioperative stroke and deficits in cognitive performance.^{4,8,19,22,24}

Reported studies have investigated the limitations of MNSSEP monitoring during CEA. MNSSEP is less sensitive than electroencephalography in detecting carotid artery cross-clamping (CC)-related ischemia, and furthermore, there are inconsistencies between MNSSEP and near-infrared spectroscopy changes.^{3,9,17} There are also case reports of perioperative cerebral infarctions in the absence of MNSSEP changes.^{6,11} A recent meta-analysis

ABBREVIATIONS ACA = anterior cerebral artery; ACoA = anterior communicating artery; CC = carotid artery cross-clamping; CEA = carotid endarterectomy; MNSSEP = median nerve somatosensory evoked potential; MRA = magnetic resonance angiography; PCoA = posterior communicating artery; SSEP = somatosensory evoked potential; TIA = transient ischemic attack; TNSSEP = tibial nerve somatosensory evoked potential.

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described MNSSEP monitoring as having high specificity (91%) but low sensitivity (58%) in the prediction of perioperative stroke.¹⁸ However, the reasons for its lower sensitivity are yet to be explained.

MNSSEP is thought to be derived from the territory supplied by the middle cerebral artery and may not detect hypoperfusion in the territory of the anterior cerebral artery (ACA). On the other hand, tibial nerve somatosensory evoked potential (TNSSEP) monitoring may, owing to the territory involved, detect hypoperfusion in the ACA territory.²¹ However, only a few studies have explored the role of TNSSEP monitoring during CEA.^{12,14}

In this study, we evaluated hypoperfusion by measuring stump pressure²³ and defined the factors that correlated with stump pressure reductions. Subsequently, we explored whether MNSSEP and TNSSEP could detect these hypoperfusion-related factors.

Our study involved three sequential steps. First, we explored the relationship between MNSSEP and TNSSEP changes during CC-related hypoperfusion. Second, we determined the factors associated with stump pressure reduction. Third, we analyzed the factors associated with MNSSEP and TNSSEP changes. We expected that these steps would explain the limitations of MNSSEP.

Methods

Patients

After institutional review board approval, we retrospectively reviewed the records of consecutive patients undergoing CEA for symptomatic or asymptomatic carotid stenosis at our institution from July 2011 to December 2015. The inclusion criteria were as follows: 1) Absence of significant carotid artery stenosis on the nonoperated side; 2) completion of preoperative magnetic resonance angiography (MRA); and 3) availability of assessable intraoperative data (MNSSEP, TNSSEP, and stump pressure). Data pertaining to preoperative demographic factors (age and sex), comorbidity (smoking, hypertension, hyperlipidemia, diabetes mellitus, symptomatic stroke, and transient ischemic attack), and perioperative factors (side of lesion and occlusion time [total time of shunt placement and removal]) were collected.

Imaging Evaluation

All patients underwent preoperative MRA performed with a 1.5-T MR imaging unit (Signa, GE Healthcare) with 3D time-of-flight pulse sequences. The absence of the anterior communicating artery (ACoA), proximal segment of the anterior cerebral artery (A_1), posterior communicating artery (PCoA), and precommunicating segment of the posterior cerebral artery (P_1) were evaluated retrospectively by two authors (S.O. and Y.T.) who were blinded to the patients' clinical and perioperative information. The paired collateral vessels of the circle of Willis (A_1 , PCoA, and P_1) were analyzed as ipsilateral and contralateral to the CEA side. The ACoA, which was located in the midline, was analyzed as a single collateral vessel. In the event of a lack of consensus between the two authors, the opinion of the senior author (S.N.) was considered as final. Cohen κ was used to guarantee the interobserver concordance for MRA

evaluation. There was a good agreement with κ values of 0.78 for ACoA, 0.91 for A_1 , 0.86 for PCoA, and 0.97 for P_1 .

Carotid Endarterectomy and SSEP

All CEA procedures were performed with the patient under general anesthesia and with MNSSEP and TNSSEP monitoring. A T-shaped flexible shunt tube (Mizuho Co., Ltd.) that was placed for all cases provided stump pressure measurements. During the carotid occlusion, if the amplitude of somatosensory evoked potential (SSEP; N20/P25 for MNSSEP and P40/N50 for TNSSEP) decreased by more than 50%, it was defined as an SSEP change. If SSEP changes were observed, we made efforts to declamp as fast as possible, and then we increased blood pressure and administered edaravone.

Real-time monitoring of SSEP was performed by using an intraoperative monitoring device (Nicolet Endeavor CR, Nicolet Biomedical, Inc.). Bilateral median and posterior tibial nerves were stimulated at the wrists and medial part of the ankles, respectively. Stimuli were repeated 100–200 times, every 300 msec, at amplitudes of 20–25 mA (350 μ sec square pulse) for MNSSEP and 30 mA (350 μ sec square pulse) for TNSSEP. MNSSEP and TNSSEP were recorded from the lateral (C3/C4'-Fpz) and medial (CPz-Fpz) parietal regions, respectively. Baseline waveforms were recorded following the induction of general anesthesia and before CC, while MNSSEP and TNSSEP were recorded during CC.

Statistical Analysis

The frequencies of MNSSEP and TNSSEP changes and their overlaps were determined to establish the relationship between the two methods of monitoring. Paired categorical data were analyzed with the binomial exact version of the McNemar test. Confidence intervals were estimated by using the nonparametric bootstrap method.

Univariate analysis was performed for demographic, radiographic, and perioperative factors and patient comorbidity by using a Student t-test to determine the contribution of these variables to stump pressure reduction. All variables with a univariate p value < 0.2 were included in stepwise multivariable linear models.

Factors related to the contribution of each SSEP change were identified by univariate and multivariable analysis. In univariate analysis, patients with and without SSEP changes were compared by using a Student t-test for continuous variables and a chi-square test for dichotomous variables. In the multivariable analysis, logistic regression was used while adjusting for the clinical factors. We adjusted for potential confounders by applying a stepwise method and included variables with a univariate p value < 0.2 in multivariable logistic models.

All statistical analyses were performed with SAS version 9.4 (SAS Institute Inc.) and Matlab (The Mathworks). In all cases, 2-sided tests were used, with a value of $p < 0.05$ considered statistically significant.

Results

Patient Characteristics

A total of 109 patients underwent CEA at our insti-

TABLE 1. Clinical characteristics in 94 patients

	Value
Demographics	
Age, yrs	74 ± 6
Female	13 (14)
Comorbidity	
Hypertension	69 (73)
Hyperlipidemia	56 (60)
Diabetes mellitus	38 (40)
Smoking	29 (35)
Symptomatic lesion	51 (54)
TIA	18 (19)
MRA data*	
ACoA, absent	18 (19)
Ipsilat A ₁ , absent	13 (14)
Contralat A ₁ , absent	10 (11)
Ipsilat PCoA, absent	81 (86)
Contralat PCoA, absent	81 (86)
Ipsilat P ₁ , absent	8 (9)
Contralat P ₁ , absent	9 (10)
Periop data	
Rt side	57 (61)
Occlusion time, secs	992 ± 234

Values are presented as the number of patients (%) or mean ± SD.

* Ipsilateral and contralateral correspond to the side of operations.

tution between July 2011 and December 2015. Of these, 15 patients were excluded for reasons that included bilateral operations (11 cases), unavailability of MNSSEP (1 case) and TNSSEP (1 case) recordings, and lack of stump pressure data when intraluminal shunts were not used (2 cases). Finally, 94 patients were included in the analysis (mean age 74 ± 6 years; women, 14%; symptomatic, 54%). Baseline characteristics of the patients are shown in Table 1. Postoperative neurological deficits were observed in 6 patients (6%). Two patients developed transient ischemic attack (TIA) without postoperative stroke. Among the 4 cases in which patients developed postoperative stroke, 2 resolved completely within 3 months, and the remaining 2 had permanent disability.

Distribution of MNSSEP and TNSSEP Changes

Carotid cross-clamping was associated with SSEP changes in 22.3% of patients (21/94). TNSSEP monitoring detected more SSEP changes than MNSSEP monitoring (20.2% vs 11.7%, $p < 0.05$; Fig. 1). The proportion of the study population that showed TNSSEP changes without MNSSEP changes (isolated TNSSEP changes) was 10.6% (10/94), and this proportion was significantly more frequent than zero (95% CI 5.3–17.2). However, the proportion of patients with MNSSEP changes but without TNSSEP changes (isolated MNSSEP) (2.1%, 2/94) was not significantly more frequent than zero (95% CI 0–5.3). These findings statistically confirm the presence of isolated TNSSEP changes. Detailed characteristics of

FIG. 1. Venn diagram illustrating the distributions of the patients who showed changes with either MNSSEP or TNSSEP monitoring or with both methods. *Significantly different from zero ($p < 0.05$).

patients who showed postoperative stroke symptoms or SSEP changes are shown in Table 2. In this study, we did not find an association between CC time and postoperative deficits (univariate regression, $p = 0.47$).

Predictors of Stump Pressure Decline

To determine the factors related to hypoperfusion, we analyzed the relationship between stump pressure decline and clinical variables. Hypertension, diabetes, absent contralateral A₁, absent ipsilateral PCoA, and absent ipsilateral P₁ were found to be significant variables on univariate analysis. On multivariate analysis, only absent contralateral A₁ (β [SE] −21.44 [7.16], $p < 0.01$) and absent ipsilateral P₁ (β [SE] −16.17 [7.02], $p = 0.02$) were found to be independently associated with stump pressure decline (Table 3).

Predictors of MNSSEP and TNSSEP Changes

A logistic regression model was designed to identify whether MNSSEP and TNSSEP monitoring could detect the absence of contralateral A₁ and ipsilateral P₁, which were identified as predictive factors for hypoperfusion as measured by stump pressure. The model indicated that MNSSEP was significantly associated with absent ipsilateral P₁ (OR 6.77, 95% CI 1.33–34.45, $p = 0.02$) but not with absent contralateral A₁ (OR 3.25, 95% CI 0.56–18.86, $p = 0.19$). TNSSEP was significantly associated with both absent ipsilateral P₁ (OR 5.14, 95% CI 1.15–23.04, $p = 0.03$) and absent contralateral A₁ (OR 18.50, 95% CI 3.34–102.55, $p < 0.001$). After adjustment for potential confounders, the association between MNSSEP changes and CC was significant in cases with absent ipsilateral P₁ (OR 8.76, 95% CI 1.61–47.67, $p = 0.01$), but not with absent contralateral A₁ (OR 4.87, 95% CI 0.77–30.61, $p = 0.09$). The association between TNSSEP changes and CC was significant both in cases with absent ipsilateral P₁ (OR 8.75, 95% CI 1.83–41.94, $p < 0.01$) and in cases with absent contralateral A₁ (OR 26.25, 95% CI 4.52–152.51, $p < 0.001$) (Fig. 2).

TABLE 2. Clinical features of patients with postoperative symptoms or SSEP changes

Age (yrs), Sex	Op Side	MNSSEP Change*	TNSSEP Change*	Absence of A _i †	Absence of P _i †	Occlusion Time (secs)	Symptomatic Stroke
80, M	Lt	No	Bilat	Contralat	Contralat	1039	TIA
65, M	Rt	No	Bilat	Ipsilat	No	954	Transient lt hemiparesis; rt watershed infarctions
78, M	Rt	Lt	Lt	No	No	914	Transient lt hemiparesis; rt watershed infarctions
78, M	Rt	No	Rt	No	Ipsilat	1085	TIA
69, M	Lt	Rt	Rt	No	Bilat	719	Rt hemiparesis; lt MCA territory infarctions
81, M	Lt	No	No	No	No	1635	Rt hemiparesis; lt watershed infarctions
71, M	Lt	No	Bilat	Contralat	No	930	No
83, M	Rt	Lt	Lt	No	No	1110	No
80, M	Lt	No	Rt	No	No	1149	No
72, F	Rt	Lt	Lt	Ipsilat	Ipsilat	1563	No
74, M	Rt	Lt	No	No	No	693	No
72, M	Lt	Rt	Rt	Contralat	No	1053	No
79, F	Lt	No	Bilat	Contralat	No	755	No
75, M	Lt	Rt	No	Contralat	No	879	No
74, M	Rt	No	Bilat	No	No	1232	No
67, M	Rt	Lt	Lt	No	No	973	No
77, M	Rt	Lt	Bilat	No	No	1373	No
69, M	Rt	No	Rt	Contralat	No	980	No
78, M	Rt	Lt	Lt	Ipsilat	Ipsilat	730	No
78, M	Rt	No	Lt	Contralat	No	1044	No
78, M	Rt	Lt	Lt	No	No	902	No
70, M	Rt	No	Rt	No	No	936	No

* SSEP changes observed by stimulating the right or left extremity.

† Ipsilateral and contralateral correspond to the side of operations.

TABLE 3. Correlation between stump pressure and clinical variables: univariate analysis

Variable	Stump Pressure Correlation, mm Hg (SE)
Age	-0.38 (0.33)
Female	0.48 (6.03)
Hypertension	-7.03 (4.65)
Hyperlipidemia	-0.50 (4.24)
Diabetes	5.58 (4.20)
Smoking	4.16 (4.48)
Symptomatic lesion	-1.90 (4.17)
TIA	0.35 (5.29)
Rt side	-1.20 (4.26)
ACoA, absent	2.26 (4.99)
Ipsilat A _i , absent	-3.99 (6.01)
Contralat A _i , absent	-16.87 (7.25)*
Ipsilat PCoA, absent	-7.15 (5.35)
Contralat PCoA, absent	-3.53 (5.67)
Ipsilat P _i , absent	-15.10 (7.29)*
Contralat P _i , absent	-7.36 (7.03)
Occlusion time	0.002 (0.01)

* p < 0.05.

Association With Perioperative Symptoms

Univariate logistic regression identified that TNSSEP change was significantly related to perioperative stroke or symptoms (OR 27.14, 95% CI 2.94–250.27, $p < 0.01$), but MNSSEP change was not (OR 4.50, 95% CI 0.72–28.10, $p = 0.11$).

Discussion

In our study, we compared and analyzed the differences in the changes between TNSSEP and MNSSEP monitoring for the detection of CC-related hypoperfusion during CEA for carotid artery stenosis. We found that cerebral hypoperfusion was more frequently detected by TNSSEP than by MNSSEP monitoring. Contralateral A_i hypoplasia and ipsilateral P_i hypoplasia were found to be significant factors that predicted cerebral hypoperfusion as measured by stump pressure. The changes in TNSSEP reflected both of these predictive factors while the changes in MNSSEP reflected only ipsilateral P_i hypoplasia, suggesting that MNSSEP monitoring may not detect hypoperfusion in the ACA territory. Therefore, this study supports the well-known fact that MNSSEP monitoring identifies hypoperfusion in the MCA but not the ACA territory.^{12,15}

Previous studies have demonstrated that MNSSEP monitoring during CEA has high specificity but low sensitivity for the prediction of perioperative stroke. However,

FIG. 2. Forest plot demonstrating the association of SSEP changes and clinical variables.

the reasons for this lower sensitivity of MNSSEP have not been explained. The main component of MNSSEP, N20, reflects the sensation of the arm corresponding to the lateral part of the primary sensory cortex and is located in the posterior part of the inverted omega convexity in the central sulcus.¹ On the other hand, P40 of TNSSEP reflects the sensation of the ankle corresponding to the medial surface of the primary motor cortex.²⁶ Despite the neurophysiological evidence that CC-related hypoperfusion affects both the lateral and medial parts of the cerebral cortex, most centers use MNSSEP alone for monitoring cerebral hypoperfusion during CEA.

We determined absent contralateral A₁ as one of the predictors of cerebral hypoperfusion as measured by stump pressure. However, according to our data, this does not affect MNSSEP, suggesting that changes in MNSSEP are not associated with ACA-related collateral function. This implies that MNSSEP monitoring may not detect hypoperfusion in the ACA territory during CC, explaining the 10.6% rate of isolated TNSSEP changes in our study.

In the present study, we explored SSEP changes caused by CC and correlated them with the incidence of major complications. On analyzing data from 6 patients who developed perioperative stroke or symptoms, significant SSEP changes were observed in 2 cases during MNSSEP monitoring and in 5 cases during TNSSEP monitoring.

In 3 cases isolated TNSSEP changes occurred without MNSSEP changes; in 2 of these cases the A₁ segment was absent. These findings further reinforce the conclusion that MNSSEP monitoring may fail to detect ACA-related hypoperfusion. The association of TNSSEP with perioperative stroke and lack of association between MNSSEP and stroke further suggest the superiority of TNSSEP.

We used intraluminal shunts in all cases, but short-term CC during shunt insertion can cause cerebral hypoperfusion, depending on variations in collateral circulation. Weigand et al.²⁹ reported that 6 ± 1 minutes of occlusion during shunt insertion caused jugular venous-arterial malondialdehyde concentration differences. Inoue et al.⁸ demonstrated that MNSSEP changes caused by 17 ± 5 minutes of CC for shunt insertion were associated with lower postoperative cognitive functions. We found no association between CC time and postoperative deficit; however, to reduce subclinical complications, we detected cerebral ischemia using MNSSEP and TNSSEP monitoring. SSEP changes were addressed by declamping as fast as possible, increasing blood pressure, and administering edaravone. MNSSEP and TNSSEP would be valuable for detection of a short term of hypoperfusion and their alarms should be attended to, to increase the safety of carotid endarterectomy.

Our study found isolated TNSSEP changes in 10.6% of

patients (10/94), demonstrating the superiority of monitoring with TNSSEP over MNSSEP in the detection of CC-related hypoperfusion. However, Manninen et al.¹⁴ found no isolated TNSSEP changes (0/153), while Malcharek et al.¹² found isolated TNSSEP changes in 1.1% (3/264) of their patients. This discrepancy may be explained by differences in the stimulus techniques employed by different investigators. Good-quality SSEPs require a stimulus high enough to obtain supramaximal SSEP amplitude.⁷ We used 350 μ sec of square pulse, compared to the impulse duration in their study of 250 μ sec, a value greater by 1.4 times. In addition, Malcharek et al. also reported a higher failure rate with TNSSEP monitoring (7.9%–12%) than ours (1%; 1/96), suggesting significant procedural differences between our studies.

Recently, multimodal monitoring during CEA has more commonly involved TNSSEP recording than previously; this method helps to prevent ischemic complications or to inform the selection of intraluminal shunts. It is worth mentioning that there is a difference in laterality of SSEP change between MNSSEP and TNSSEP. In our patients there were 19 cases of TNSSEP changes, for which changes in the ipsilateral lower extremity were observed in 9 cases (47%; 9/19). On the other hand, no MNSSEP changes were observed in the ipsilateral upper extremity (0%; 0/11). Thus, SSEP changes of the ipsilateral extremity were more common in TNSSEP monitoring. We believe these ipsilateral TNSSEP changes should not be regarded as false positives but should be used to inform clinical decision-making.

We detected two types of abnormal collaterals of the circle of Willis that were predictive of a higher risk of CC-related hypoperfusion. Both of these variants result in a greater perfusion demand on the internal carotid artery (ICA). Tanaka et al. analyzed volume flow rates of the ICA and basilar artery in healthy volunteers and found an increase in the relative flow rates of the contralateral ICA (by 25%) and the ipsilateral ICA (by 18%) in subjects with A_1 and P_1 hypoplasia compared to flow rates in subjects with normal cerebral vasculature.²⁵ Similarly, increased flow in the ICA, as seen on MRA, is related to ischemic change during CC, supporting the view that lesions resulting in such an increased flow may be reliable predictors of CC-related hypoperfusion.²⁰

The present study has several limitations. First, this was a retrospective study and subject to the inherent limitations of such a study design. Second, the actual number of SSEP changes in our study was small, thus affecting the generalizability of our findings. Third, evaluating collateral function of the circle of Willis by using MRA has its own limitations. Reduced inflow in the anterior segment of the circle of Willis caused by carotid artery stenosis may affect the evaluation of the patency of the ACoA and ipsilateral A_1 . High-resolution MRA may overcome such a limitation. Fourth, in the current study, we did not employ electroencephalography, which covers a broader range of the cortex. Further research is warranted to compare the effectiveness of electroencephalography and TNSSEP. Finally, there is another limitation of MNSSEP and TNSSEP monitoring. MNSSEP and TNSSEP reflect the function of the thalamocortical pathway that projects to the primary

somatosensory cortex and the primary motor cortex but do not reflect the corticospinal tract that is supplied by the lenticulostriate artery.¹⁰ To monitor the corticospinal tract, motor evoked potential (MEP) monitoring is a more reliable modality.¹³

Conclusions

Our study demonstrated that ACA-related hypoperfusion was associated with TNSSEP changes but not with MNSSEP changes during CEA. Routine use of MNSSEP monitoring with TNSSEP monitoring may reduce cerebral hypoperfusion and its attendant complications during CEA.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Okuyama, Nishimura. Acquisition of data: Okuyama, Nishimura, Takahashi, Kubota, Hirano, Kazama, Tomii, Matsuyama, Sato. Analysis and interpretation of data: Okuyama, Nishimura. Drafting the article: Okuyama. Critically revising the article: Nishimura. Reviewed submitted version of manuscript: Nishimura. Approved the final version of the manuscript on behalf of all authors: Okuyama. Statistical analysis: Okuyama, Takahashi. Administrative/technical/material support: Mizuno, Matsushima, Watanabe. Study supervision: Nishimura.

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