Predictive Value of Somatosensory Evoked Potential Monitoring during Resection of Intraparenchymal and Intraventricular Tumors Using an Endoscopic Port

Parthasarathy Thirumala,[a] Daniel Lai,[b] Jonathan Engh,[a] Miguel Habeych,[a] Donald Crammond,[a] Jeffrey Balzer[a]

[a]Center for Clinical Neurophysiology, Department of Neurologic Surgery, University of Pittsburgh Medical Center, Pittsburgh, PA, USA
[b]Department of Neurology, Stanford University, Palo Alto, CA, USA

Background and Purpose Intraoperative neurophysiological monitoring (IONM) using upper and lower somatosensory evoked potentials (SSEPs) is an established technique used to predict and prevent neurologic injury during intracranial tumor resections. Endoscopic port surgery (EPS) is a minimally-invasive approach to deep intraparenchymal and intraventricular brain tumors. The authors intended to evaluate the predictive value of SSEP monitoring during resection of intracranial brain tumors using a parallel endoscopic technique.

Methods A retrospective review was conducted of patients operated on from 2007-2010 utilizing IONM in whom endoscopic ports were used to remove either intraparenchymal or intraventricular tumors. Cases were eligible for review if an endoscopic port was used to resect an intracranial tumor and the electronic chart included all intraoperative monitoring data as well as pre- and post-operative neurologic exams.

Results 139 EPS cases met criteria for inclusion. Eighty five patients (61%) had intraparenchymal and fifty four (39%) had intraventricular tumors or colloid cysts. SSEP changes were seen in eleven cases (7.9%), being irreversible in three (2.2%) and reversible in eight cases (5.8%). Seven patients (5.0%) with intraparenchymal tumors had SSEP changes which met our criterea for significant changes while there were four (2.9%) with intraventricular (p-value=0.25). Five patients suffered post operative deficits, two reversible and two irreversible SSEP changes. Only one case exhibited post operative hemiparesis with no SSEP changes. The positive predictive value of SSEP was 45.4% and the negative predictive value was 99.2%.

Conclusions Based on the high negative and low positive predictive values, the utility of SSEP monitoring for cylindrical port resections may be limited. However, the use of SSEP monitoring can be helpful in reducing the impact of endoscopic port manipulation when the tumor is closer to the somatosensory pathway.　　　　　**J Clin Neurol 2013;9:244-251**

Key Words endoscopic port, somatosensory, evoked potentials, somatosensory, monitoring.

Correspondence
Parthasarathy Thirumala, MD, MS
Center for Clinical Neurophysiology,
Department of Neurologic Surgery,
University of Pittsburgh
Medical Center,
UPMC Presbyterian-Suite B-400,
200 Lothrop Street, Pittsburgh,
PA 15213, USA
Tel　+1-412-648-2228
Fax　+1-412-383-9899
E-mail　thirumalapd@upmc.edu

Introduction

Somatosensory evoked potentials (SSEPs) have been used during skull base,[1,2] spine[3-6] and brain tumor[2,7] operations to predict and reduce post operative neurologic deficits.[1,8] Intraoperative monitoring of SSEPs allows for the dynamic assessment of the spinal cord dorsal columns, the brainstem medial lemnsical pathways to the thalamus, and thalamic connections to the primary sensory cortex. Persistent changes in the SSEPs during these procedures can predict post operative neurologic deficits.[1,9] Intraoperative restoration of SSEP changes may prevent post operative neurologic complications.[1] Regional and hemispheric changes of cerebral blood flow (CBF) could potential-

ly be identified with SSEP monitoring and thereby prevent neurovascular injury[9-13]. Additionally, SSEP monitoring can evaluate potential peripheral injury related to neck position,[14-16] brachial plexus[17] or peripheral nerve injury[14-16] during surgery.

Endoscopic port surgery (EPS) presents an alternative approach for resection of deep tumors. The port itself is a transparent cylindrical retractor measuring 4.00 mm in diameter; the length of the port is tailored to the depth of the resection. The port is introduced into the brain over a bullet shaped dilator. In contrast to traditional microsurgical deep tumor resection, this technique attempts to limit retraction and dissection injury while improving the field of view at deep sites.[18-20] The endoscopic port has been used to resect tumors within deep white matter and the ventricles. Our study will be the first to evaluate the utility of intraoperative SSEP monitoring to prevent postoperative neurologic deficits after endoscopic resection of brain tumors.

Methods

Between 2007 and 2010, we monitored 139 patients who underwent resection of either intraventricular or intraparenchymal tumors using EPS. We retrospectively reviewed all cases of endoscopic tumor resections; inclusion criteria for the study included patients who underwent endoscopic tumor resections with intraoperative SSEP monitoring. Patients were excluded if the data was unavailable for analysis due to technical reasons or because of missing data. This study was approved by the local institutional review board for retrospective review of clinical outcomes.

Neurophysiologic monitoring
Intraoperative SSEP monitoring is standard at our institution for EPS cases.[1] SSEP baseline values were obtained prior after anesthesia induction, but before the patients were positioned.

Upper and lower extremity SSEP responses were continuously obtained throughout the procedure. Physician oversight and interpretation was performed using a combined on-site and remote model utilized at the University of Pittsburgh Medical Center.[21] All patients underwent the procedure in a stereotactic frame for tumor localization. General inhalational anesthesia was given to all patients with additional intravenous medications in some cases. In our institution we typically used inhalational anesthestics including sevoflurane, desflurane, isoflurane in concentrations with a Minimum Alveolar Concentration between 0.5-0.75.

Upper extremity SSEPs
Median or ulnar nerve stimulation was performed bilaterally in an alternating fashion at the wrist with subdermal needle electrode pairs. Recordings were obtained from the scalp and cervical region with subdermal electrodes. P4/Fz and P3/Fz scalp electrodes were used (per the international 10-20 system). A cervical electrode was located at the C2 spinous process or mastoid and referenced to Fz. Stimulation frequency was 2.33-2.45 Hz with duration of 0.2-0.3 milliseconds. Band pass filters were set at 10 to 300 Hz for cortical recordings and 30 to 1000 Hz for subcortical recordings. Averages were computed for either 128 or 256 trials, depending on the signal quality.

Lower extremity SSEPs
For the lower extremities, bilateral alternating tibial nerve stimulation, interleaved after the upper stimulation was used. Peroneal nerve stimulation was used when reliable tibial nerve responses could not be elicited. Tibial nerve stimulation was performed at the medial malleolus of the ankle with subdermal needle electrodes. The peroneal nerve was stimulated using pairs of subdermal needles located at the head of the fibula and medially in the popliteal fossa. Recordings were obtained from the scalp and cervical region with subdermal electrodes. Pz/Fz and P4/P3 scalp electrodes were used (per the international 10-20 system). A cervical electrode was located at the C2 spinous process or at the level of the mastoid and referenced to Fz. Stimulation frequency was 2.33-2.45 Hz with duration of 0.2-0.3 milliseconds. Band pass filters were set at 10 to 300 Hz for cortical recordings and 30 to 1000 Hz for cervical recordings. Averages were computed for either 128 or 256 trials depending on the signal quality.[21]

Alarm criteria
A SSEP epoch average was collected approximately every 40 seconds and compared against the baseline. We defined as 'significant' a signal change persisting over at least two consecutive epochs characterized by either a 50% reduction in amplitude or a prolongation of response latency by >10% from baseline. This criterion is widely used in the literature, particularly in spine surgery cases.[22-24]

Medical record review
Medical records for all 139 patients were reviewed to determine whether any new neurologic deficit was identified postoperatively. New motor/sensory deficits were defined as being present if there was a new focal deficit noted in the chart during the immediate post operative period as compared to pre-operative neurologic exam. Similarly, the intraoperative records for all 139 patients were reviewed to identify any significant SSEP changes.

Data analysis
To determine the sensitivity and specificity of intraoperative

SSEP monitoring for detecting impending or resultant iatrogenic neurologic injury, we defined and then classified each of the 139 operative cases as one of the following.

True positive (TP): significant SSEP signal changes accompanied by a new postoperative neurologic deficit; a case in which significant SSEP signal deterioration occurred as the result of a recognized intraoperative cause, event, or complication; or a case in which a significant SSEP signal deterioration improved to the baseline value after a specific intraoperative intervention.

True negative (TN): normal intraoperative SSEP signals in the absence of a new postoperative neurologic deficit.

False positive (FP): persistent significant SSEP signal deterioration that did not improve with intraoperative interventions, or in which no intraoperative adjustments were made in cases of reversible changes and the patient woke up neurologically intact.

False negative (FN): normal intraoperative SSEP signals with a new postoperative neurologic deficit.

Patients that were either TPs, FP of FNs were catagorized as 'group 1'. TN patients were catagorized in 'group 2'.

Quantitative analysis

The quantitative analysis has been described in detail previously.[21] The first part of analysis involved interpreting intraoperative SSEP data from patients who demonstrated significant changes. It was scrutinized to determine whether such a change occurred in temporal proximity to specific intraoperative events such as changes in mean arterial pressure (MAP), manipulation of the endoscopic port, or a pharmacologic change in anesthesia administration (Table 1). In such cases, intraoperative records were reviewed to determine whether any subsequent intraoperative countermeasures such as increasing the MAP, repositioning the port, or decreasing the anesthesic dose were undertaken and whether the SSEP signals improved after these actions. The second part of the analysis details with measuring changes in latency and amplitude at the point of maximum change and at closure compared with baseline values at incision. Labels were used to refer to the SSEP responses, e.g., right cortical upper extremity amplitude (RCUA)-right (R), cortical (C), upper-limb response (U), amplitude (A)-and LBLL-left (L), brainstem (B), lower-limb response (L), latency (L)-refers to SSEP responses from the left upper-limb response in the brainstem and the contralateral cortex. The latency and amplitude values were recorded at incision, maximum change, and closure. A percentage change in latency and amplitude from incision was calculated at 2 instances during surgery: at the maximum change and at closure (Table 2). The percentage of change from incision (I) to maximum change (M) was calculated by the following formula: $100\ [(I-M)/I]$; similar change was calculated from incision to closure. The purpose of the calculation is to follow the trend of the responses and to correlate them with postoperative outcomes.

Table 1. Neurophysiological changes, intraoperative interventions and neurologic status in patients who underwent endoscopic port surgery with SSEP monitoring

Patient	Diagnosis	Cannulation route	Neurophysiologic change	Clinical sequelae	Intraoperative adjustment	Statistics
1	Glioblastoma	IP	Reversible, LCUA	Yes, weaker left lower extremity	None	FP
2	Glioblastoma	IP	Reversible, LCUA	No	None, resecting near thalamus	TP
3	Glioblastoma	IP	Reversible, RCUA	No	None	FP
4	Metastatic small cell carcinoma	IP	Irreversible, RCUA, RCUL, RCLA, LCUA, LCUL	Yes, death	Adjusted port trajectory	TP
5	Anaplastic astrocytoma	IP	Irreversible RCUA, LCUA, LCLA	No	Adjusted port trajectory	TP
6	Glioblastoma	IP	Irreversible, RCUA, LCUA	Yes, weaker right upper extremity	Gave lorazepam	TP
7	Glioblastoma	IP	Reversible, RCUA, LCLA	No	None	FP
8	Epidermoid tumor	IV	Reversible, RCUA, LCUA	Yes, right hemiparesis	Retractor traction	TP
9	Cavernous malformation	IV	Reversible, RCUA, LCUA	No	Gave blood products	FP
10	Colloid cyst	IV	Reversible, RCUA, LCUA	No	None-increased stimulation	FP
11	Central neurocytoma	IV	Reversible, LCUA	No	None	FP
12	Metastatic adenocarcinoma	IP	None	Yes, right hemiparesis	None	FN

FN: false negative, FP: false positive, IP: intraparenchymal, IV: intraventricular, LCLA: left cortical lower limb amplitude, LCUA: left cortical upper limb amplitude, LCUL: left cortical upper limb latency, RCLA: right cortical lower limb amplitude, RCUA: right cortical amplitude upper limb amplitude, RCUL: left cortical upper limb latency, SSEP: somatosensory evoked potential, TP: true positive.

Table 2. Quantitative analysis of cortical somatosensory evoked potential monitoring response amplitude and latency as a percentage of baseline and at the point of maximum change and closure

Patient	Statistic	Maximum change in cortical amplitudes, % of baseline				Cortical amplitudes at closure, % of baseline			
		RCUA	LCUA	RCLA	LCLA	RCUA	LCUA	RCLA	LCLA
1	FP		-92.3				-82.0		
2	TP		-63.2				+31.6		
3	FP	-50.0				-3.6			
4	TP	-100	-100	-61.5		-100	-100	-61.5	
5	TP	-50.0	-83.3		-52.6	+33.3	+117		-52.6
6	TP	-66.7	-80.0			-66.7	-70		
7	FP	-75.0			-76.5	+75.0			+35.3
8	TP	-50.0	-65.5			-29.7	-29.3		
9	FP	-84.6	-73.3			+83.8	-50.0		
10	FP	-76.9	-64.3			-7.7	+14.3		
11	FP		-58.3				-33.3		
12	FN	-35.7	-40.0	+28.6	0	-21.4	-25.0	+28.6	0

FP: false positive, FN: false negative, LCLA: left cortical lower limb amplitude, LCUA: left cortical upper limb amplitude, RCLA: right cortical lower limb amplitude, RCUA: right cortical amplitude upper limb amplitude, TP: true positive.

Table 3. Demographics information of patients with and without changes in somatosensory evoked potentials

	Group 1 (TP, FP, FN)	Group 2 (TN)
Total	12	127
Age, mean	52.7	49.6
<50 years	6	61
≥50 years	6	67
Sex ratio (M : F)	1 : 1.4	1.1 : 1
Most common diagnosis (%)	5 glioblastoma (41.7)	28 glioblastoma (22.0)
Cannulation route (%)	8 intraparenchymal (5.8)	77 intraparenchymal (55.4)
	4 intraventricular (2.9)	50 intraventricular (36.0)
Neurologic deficits (%)	4 intraparenchymal (2.9)	
	1 intraventricular (0.7)	

FN: false negative, FP: false positive, TN: true negative, TP: true positive.

Statistical analysis

The Fischer's exact test analysis was used to compare the incidence of SSEP changes between intraparenchymal and intraventricular tumors. Analysis comparing demographic differences between groups one and two was complete with a two sample *t* test.

Results

We analyzed 139 port cases: 85 patients (61%) had intraparenchymal tumors and 54 (39%) had intraventricular or colloid cysts. The average age of patients with SSEP changes was 52.7 years as compared to 49.6 without changes (*p*=0.60). Demographics are reported in Table 3. Glioblastoma was the most common intracranial lesion in groups 1 and 2, though the incidence was somewhat higher in group 1. Each of the patients included in group 1 is listed in Table 2 which describes the changes observed during the surgery, the intraoperative management and post operative sequelae. The resultant statistical analysis is listed in Table 4.

Intraoperative SSEP changes

In total, SSEP changes were seen in eleven (7.9%) patients, eight cases (5.8%) had reversible waveform changes which were either at baseline or improving towards baseline at the conclusion of the surgery. Three (2.2%) had irreversible waveform changes. Of the intraparenchymal tumors, seven patients (5.0%) had significant changes in the SSEPs, four were reversible and three were irreversible. Among the intraventricular tumors, four patients (2.9%) had significant SSEP changes, all of which were reversible.

Post operative neurologic deficits

There were a total of five cases (3.6%) with post operative neurologic consequences. In two of the cases there were reversible SSEP changes, where permanent neurologic morbidity occurred. Another two cases had irreversible SSEP changes, with permanent neurologic morbidity. One patient had no

Table 4. Intraoperative somatosensory evoked potential monitoring results and postoperative neurologic status in a 2x2 table

	New sensory or motor deficit	
SSEP changes	Present	Absent
Present	5 (TP)	6 (FP)
Absent	1 (FN)	127 (TN)

Sensitivity=83.3%, specificity=95.5%, positive predictive value: 45.4%, negative predictive value: 99.2%.
FN: false negative, FP: false positive, SSEP: somatosensory evoked potential, TN: true negative, TP: true positive.

significant intraoperative SSEP change, but did show a new hemiparesis following the operation.

True positives

Patient 2 had reversible changes to the left cortical upper extremity amplitude (LCUA) SSEP. At the time of the evoked potential change, the surgeons were operating close to the thalamus. The location of the ongoing surgical manipulation was thought to be the cause of the waveform change. After the procedure, the SSEP improved and the patient did not have any post operative neurologic deficit.

Patient 4 had irreversible changes to RCUA, the LCUA, and the right cortical lower extremity amplitude (RCLA). Latencies in the right and left upper extremities became prolonged until the waveforms were completely lost and could not be measured. The left cortical lower extremity amplitude (LCLA) was also diminished but was not significant. Cerebral cortex swelling was noted during the procedure. Post operatively, a CT scan demonstrated intraparenchymal and subarachnoid bleeding. The patient died postoperatively.

Patient 5 had irreversible changes to the LCLA, but had reversible changes to the LCUA and RCUA. The cortical latencies for the left arm, left leg and right arm did not change. The SSEPs in the RCUA and LCUA improved with intraoperative adjustment of the port. Moreover, by the end of the surgery, the RCUA and LCUA exceeded baseline values. However, the LCLA did not improve with adjustment. Notably, the patient's post operative exam was remarkable for improved weakness of the left lower extremity except for dorsiflexion of the tibialis anterior.

Patient 6 had irreversible SSEP waveform changes to the RCUA, but slight recovery of the LCUA at the conclusion of the operation. Intraoperative EEG was used during the procedure in addition to SSEP monitoring in this case. There was concern that the EEG demonstrated seizures. He was given lorazepam before the conclusion of the surgery. There was new weakness in the right arm post operatively.

Patient 8 had reversible changes to the RCUA and LCUA with subsequent improvement of both cortical amplitudes, but did not return to baseline. Latencies were not significantly affected. The trajectory of the endoscopic port was adjusted,

as it was believed that the changes were secondary to retraction. The patient had a new right hemiparesis after the operation.

False positives

Patient 1 was noted to have a reversible change to the LCUA, without changes to the subcortical waveforms or left lower extremity waveforms. The waveforms most significantly decreased after the withdrawal of the port from the brain. The patient was found to have left lower extremity weakness after the surgery.

Patients 3 and 7 both had a reversible changes to the RCUA. The LCLA in patient 7 also demonstrated a reversible change. Patient 11 had a reversible change to the LCUA. For these cases, no cause was determined to have precipitated the waveform change in any patient. No intraoperative adjustments were made. At the conclusion of the surgery, the RCUA for patient 3 returned baseline as did both the RCUA and LCLA for patient 7. The LCUA for patient 11 did improve, but was still 33% lower than baseline. Patients 3, 7 and 11 had no new post operative deficits.

Patients 9 and 10 both had significant changes to the RCUA and LCUA. In both cases there was no discernable cause for the change. Latencies were unaffected. Patient 9 did have significant blood loss requiring intraoperative transfusion, though blood pressure remained relatively constant. However, there was no temporal correlation between the neurophysiological changes and blood transfusion. The changes were noted earlier during the surgery and transfusions were done late in the surgery. In addition, the blood transfusion was not an intraoperative measure suggested by the neurophysiology team. Otherwise, no intraoperative adjustments were made. At the end of the procedure, the RCUA of patient 9 exceeded baseline while the LCUA improved, but was still 50% lower than baseline. The RCUA of patient 10 improved nearly to baseline and the LCUA slightly exceeded baseline. There were no post operative deficits noted for either patient.

False negative

Patient 12 did not have significant intraoperative waveform changes; however changes were noted in the LCUA and RCUA. The RCLA improved throughout the course of the surgery and had higher amplitude at the time of the procedure's conclusion. The patient was noted to have new right hemiparesis in the immediate post operative period consistent with a left supplementary motor area syndrome. Over several weeks, the patient gradually regained strength in the affected side.

True negatives

The remaining 127 patients did not have intraoperative chang-

es that met alarm criteria and did not have new post operative sensory or motor deficits.

Discussion

Our study aims to elucidate the utility of intraoperative SSEP monitoring during endoscopic brain tumor resection. In particular, we are concerned with potential injury which may be prevented due to regional parenchymal compression secondary to port manipulation and hemispheric changes in blood flow related to the procedure. The use of intraoperative SSEP monitoring has been shown to be effective in predicting post operative deficits under a number of circumstances including spine,[3-6] carotid endarterectomies,[10,13] skull based,[1,2] and more recently minimally invasive skull based surgeries.[21] This form of intraoperative monitoring can predict and prevent post operative morbidity by alerting the surgeon to intraoperative changes which may compromise the somatosensory pathway. The introduction of an endoscopic port may cause injury due to mechanical strain during cannulation or withdrawal, localized ischemia due to regional compression, or injury due to intraoperative adjustments. Since the sensory and motor strip share a common vascular supply, motor deficits may also be detected.[23] In our study there was no significant difference in the incidence of deficits between intraparenchymal and intraventricular tumor removal. The FN case in this study occurred with resection of an intraparenchymal tumor in the supplementary motor area. Hence in our study, we performed a combined analysis of intraventricular and intraparenchymal endoscopic approaches for tumor removal.

In our study we had 5 cases with SSEP changes corresponding to potential or realized post operative deficits (TPs). Amongst these TPs, irreversible changes were found in three cases (patients 4, 5, and 6). Irreversible SSEP changes have been associated with new post operative clinical deficits and were seen in patients 4 and 6 after the procedure.[21,25] The waveform changes with patient 4 were severe, resulting complete loss of RCUA and LCUA. During the operation, the brain parenchyma was noted to become edematous. Post operatively, there was a significant intracranial hemorrhage which resulted in death. Patient 5 did have irreversible waveform loss, but with adjustment of the port, a post operative deficit was potentially avoided. There were also 2 TP cases with reversible changes, one of which (patient 8) demonstrated a new clinical deficit. Patient 2 was categorized as TP due to surgical manipulation near the thalamus at the time of the SSEP change. The majority of TP cases (patients 1, 4, 5, and 8) in our study exhibited SSEP changes related to manipulation of the port. It is possible that such manipulation can result in significant regional parenchymal compression, leading to ischemia or even infarction.

Reversible and irreversible changes[1,10,21,25] in SSEP waveforms significantly increase the likelihood of post operative deficits and allow for possible intraoperative adjustments to prevent post operative complications. Animal models indicate that a drop in CBF below 16 to 20 mL/100g/minute causes a reversible decrease in the amplitude of cortical SSEP responses.[26,27] Additionally, animal studies have shown that an increase in the MAP, and consequently the CBF, after a 50% reduction in SSEP amplitude can result in SSEP recovery to control values.[28] In humans, persistent reduction of SSEP amplitude by 50% is observed when CBF decreases below 14 mL/100 g/minute.[29] Further animal studies have shown that there is a narrow hemodynamic window where a loss of cortical SSEP response does not imply loss of neuronal viability and that reversing the changes in CBF in a certain time frame reverses injurious or iatrogenic cellular ionic changes.[30] Hence an early warning using alarm criteria might be helpful to readjust and reperfuse the brain parenchyma after port manipulations. This might prevent post operative neurologic complications in the somatosensory or motor pathway which otherwise could not have been easily identified during port manipulation.

Significantly, there were six cases we recorded as being FPs. Our observed positive predictive value of 45% has been observed in prior studies during scoliosis operations.[31] The cause of FPs can be due to instrumentation error, anesthetic effects, transient alterations in MAP,[28] or ineffective documentation of surgical or physiological changes. Most of the FPs with no documentation occurred during cases performed shortly after EPS was initially developed. The learning curve of the surgical, anesthesia and neurophysiology team might have contributed to this phenomenon as previously reported.[21] A single case (patient 12) was recorded during which the SSEP waveforms did not reach alarm criteria, yet there was a new postoperative hemiparesis. Causes of FNs are of paramount importance as they represent lost opportunities to potentially intervene prior to development of the neurologic deficit. 'Delayed onset' neurologic deficits have been recognized in spinal surgeries[32] as well as skull base procedures.[2] These delayed onset cases may be attributed to latent vascular or mechanical compression.[32] More likely in our case, the affected parenchyma was the supplementary motor area which was distant from the pathway which SSEPs monitor. Hence the ischemia in the region had some, but not significant, effect on the somatosensory area resulting in changes that did not reach alarm criteria. A previous case series by Wiedemayer et al.[2] notes that one of the major reasons for FNs was the location of the lesion being outside the monitored pathway.

Taken together, SSEP monitoring during EPS may be able

to help prevent post operative deficits secondary to port manipulation. During our procedure the patient is in a stereotactic frame for localization and has an endoport and a light source fitted for visualization. This instrumentation is never removed, which is necessary for us to perform a transcranial motor evoked potential study in our institution. Without the removal, any patient movement during the procedure might increase the chances of iatrogenic injury secondary to movement.[33]

In line with previous studies, we observed a robust negative predictive value with expected specificity and sensitivity.[2,13,21,31,32] However, our positive predictive value was lower than expected. A low positive predictive value potentially results in both needless interruptions to the surgical procedure and an inability to intervene on patients in whom there is an impending neurologic complication. This study is limited by its relatively small sample size, its retrospective nature and inadequate documentation, making broader generalizations difficult to make. The retrospective nature hindered assessment of patients' post operative physical exams. As noted, delayed onset of neurologic deficits may remain latent and may only be realized until well after the surgery.[2] The lack of long term follow up is a further limitation to this study.

Conclusions

Based on the high negative predictive and low positive predictive value, the utility of SSEP monitoring during EPS for deep seated tumors appears genuine but limited. The use of SSEP monitoring can be helpful in reducing the impact of port manipulations when the tumor is close to somatosensory pathway. Alternatively, adjunctive use of transcranial motor evoked potentials may provide a useful supplement to the monitoring approach. A large patient series is needed to completely evaluate the utility of SSEPs during resection of deep brain tumors through a minimal access port.

Conflicts of Interest

The authors have no financial conflicts of interest.

REFERENCES

1. Bejjani GK, Nora PC, Vera PL, Broemling L, Sekhar LN. The predictive value of intraoperative somatosensory evoked potential monitoring: review of 244 procedures. *Neurosurgery* 1998;43:491-498; discussion 498-500.
2. Wiedemayer H, Sandalcioglu IE, Armbruster W, Regel J, Schaefer H, Stolke D. False negative findings in intraoperative SEP monitoring: analysis of 658 consecutive neurosurgical cases and review of published reports. *J Neurol Neurosurg Psychiatry* 2004;75:280-286.
3. Wilber RG, Thompson GH, Shaffer JW, Brown RH, Nash CL Jr. Postoperative neurological deficits in segmental spinal instrumentation. A study using spinal cord monitoring. *J Bone Joint Surg Am* 1984;66:1178-1187.
4. Jones SJ, Edgar MA, Ransford AO, Thomas NP. A system for the electrophysiological monitoring of the spinal cord during operations for scoliosis. *J Bone Joint Surg Br* 1983;65:134-139.
5. Brown RH, Nash CL Jr, Berilla JA, Amaddio MD. Cortical evoked potential monitoring. A system for intraoperative monitoring of spinal cord function. *Spine (Phila Pa 1976)* 1984;9:256-261.
6. Bradshaw K, Webb JK, Fraser AM. Clinical evaluation of spinal cord monitoring in scoliosis surgery. *Spine (Phila Pa 1976)* 1984;9:636-643.
7. Kombos T, Picht T, Derdilopoulos A, Suess O. Impact of intraoperative neurophysiological monitoring on surgery of high-grade gliomas. *J Clin Neurophysiol* 2009;26:422-425.
8. Gentili F, Lougheed WM, Yamashiro K, Corrado C. Monitoring of sensory evoked potentials during surgery of skull base tumours. *Can J Neurol Sci* 1985;12:336-340.
9. 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* 1988;2:63-68.
10. Stejskal L, Kramár F, Ostrý S, Benes V, Mohapl M, Limberk B. Experience of 500 cases of neurophysiological monitoring in carotid endarterectomy. *Acta Neurochir (Wien)* 2007;149:681-688; discussion 689.
11. Lopéz JR, Chang SD, Steinberg GK. The use of electrophysiological monitoring in the intraoperative management of intracranial aneurysms. *J Neurol Neurosurg Psychiatry* 1999;66:189-196.
12. Friedman WA, Kaplan BL, Day AL, Sypert GW, Curran MT. Evoked potential monitoring during aneurysm operation: observations after fifty cases. *Neurosurgery* 1987;20:678-687.
13. Lam AM, Manninen PH, Ferguson GG, Nantau W. Monitoring electrophysiologic function during carotid endarterectomy: a comparison of somatosensory evoked potentials and conventional electroencephalogram. *Anesthesiology* 1991;75:15-21.
14. Schwartz DM, Sestokas AK, Hilibrand AS, Vaccaro AR, Bose B, Li M, et al. Neurophysiological identification of position-induced neurologic injury during anterior cervical spine surgery. *J Clin Monit Comput* 2006;20:437-444.
15. Kamel IR, Drum ET, Koch SA, Whitten JA, Gaughan JP, Barnette RE, et al. The use of somatosensory evoked potentials to determine the relationship between patient positioning and impending upper extremity nerve injury during spine surgery: a retrospective analysis. *Anesth Analg* 2006;102:1538-1542.
16. Chung I, Glow JA, Dimopoulos V, Walid MS, Smisson HF, Johnston KW, et al. Upper-limb somatosensory evoked potential monitoring in lumbosacral spine surgery: a prognostic marker for position-related ulnar nerve injury. *Spine J* 2009;9:287-295.
17. Schwartz DM, Drummond DS, Hahn M, Ecker ML, Dormans JP. Prevention of positional brachial plexopathy during surgical correction of scoliosis. *J Spinal Disord* 2000;13:178-182.
18. Kassam AB, Engh JA, Mintz AH, Prevedello DM. Completely endoscopic resection of intraparenchymal brain tumors. *J Neurosurg* 2009; 110:116-123.
19. Harris AE, Hadjipanayis CG, Lunsford LD, Lunsford AK, Kassam AB. Microsurgical removal of intraventricular lesions using endoscopic visualization and stereotactic guidance. *Neurosurgery* 2005;56(1 Suppl):125-132; discussion 125-132.
20. Engh JA, Lunsford LD, Amin DV, Ochalski PG, Fernandez-Miranda J, Prevedello DM, et al. Stereotactically guided endoscopic port surgery for intraventricular tumor and colloid cyst resection. *Neurosurgery* 2010;67(3 Suppl Operative):ons198-ons204; discussion ons204-ons205.
21. Thirumala PD, Kassasm AB, Habeych M, Wichman K, Chang YF, Gardner P, et al. Somatosensory evoked potential monitoring during endoscopic endonasal approach to skull base surgery: analysis of observed changes. *Neurosurgery* 2011;69(1 Suppl Operative):ons64-ons76; discussion ons76.
22. York DH, Chabot RJ, Gaines RW. Response variability of somatosensory evoked potentials during scoliosis surgery. *Spine (Phila Pa 1976)* 1987;12:864-876.

23. Chen ZY, Wong HK, Chan YH. Variability of somatosensory evoked potential monitoring during scoliosis surgery. *J Spinal Disord Tech* 2004;17:470-476.

24. Balzer JR, Rose RD, Welch WC, Sclabassi RJ. Simultaneous somatosensory evoked potential and electromyographic recordings during lumbosacral decompression and instrumentation. *Neurosurgery* 1998; 42:1318-1324; discussion 1324-1325.

25. Mizoi K, Yoshimoto T. Permissible temporary occlusion time in aneurysm surgery as evaluated by evoked potential monitoring. *Neurosurgery* 1993;33:434-440; discussion 440.

26. 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* 1974;45:195-208.

27. Astrup J, Symon L, Branston NM, Lassen NA. Cortical evoked potential and extracellular K+ and H+ at critical levels of brain ischemia. *Stroke* 1977;8:51-57.

28. Lopez JR. Intraoperative neurophysiological monitoring. *Int Anesthesiol Clin* 1996;34:33-54.

29. Symon L. The relationship between CBF, evoked potentials and the clinical features in cerebral ischaemia. *Acta Neurol Scand Suppl* 1980; 78:175-190.

30. Branston NM, Symon L, Strong AJ. Reversibility of ischaemically induced changes in extracellular potassium in primate cortex. *J Neurol Sci* 1978;37:37-49.

31. Nuwer MR, Dawson EG, Carlson LG, Kanim LE, Sherman JE. Somatosensory evoked potential spinal cord monitoring reduces neurologic deficits after scoliosis surgery: results of a large multicenter survey. *Electroencephalogr Clin Neurophysiol* 1995;96:6-11.

32. Nuwer MR. Spinal cord monitoring. *Muscle Nerve* 1999;22:1620-1630.

33. Macdonald DB. Intraoperative motor evoked potential monitoring: overview and update. *J Clin Monit Comput* 2006;20:347-377.