

Utility of neuromonitoring during lumbar pedicle subtraction osteotomy for adult spinal deformity

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OBJECTIVE The benefits and utility of routine neuromonitoring with motor and somatosensory evoked potentials during lumbar spine surgery remain unclear. This study assesses measures of performance and utility of transcranial motor evoked potentials (MEPs) during lumbar pedicle subtraction osteotomy (PSO).

METHODS This is a retrospective study of a single-surgeon cohort of consecutive adult spinal deformity (ASD) patients who underwent lumbar PSO from 2006 to 2016. A blinded neurophysiologist reviewed individual cases for MEP changes. Multivariate analysis was performed to determine whether changes correlated with neurological deficits. Measures of performance were calculated.

RESULTS A total of 242 lumbar PSO cases were included. MEP changes occurred in 38 (15.7%) cases; the changes were transient in 21 cases (55.3%) and permanent in 17 (44.7%). Of the patients with permanent changes, 9 (52.9%) had no recovery and 8 (47.1%) had partial recovery of MEP signals. Changes occurred at a mean time of 8.8 minutes following PSO closure (range: during closure to 55 minutes after closure). The mean percentage of MEP signal loss was 72.9%. The overall complication rate was 25.2%, and the incidence of new neurological deficits was 4.1%. On multivariate analysis, MEP signal loss of at least 50% was not associated with complication ($p = 0.495$) or able to predict postoperative neurological deficits ($p = 0.429$). Of the 38 cases in which MEP changes were observed, the observation represented a true-positive finding in only 3 cases. Postoperative neurological deficits without MEP changes occurred in 7 cases. Calculated measures of performance were as follows: sensitivity 30.0%, specificity 84.9%, positive predictive value 7.9%, and negative predictive value 96.6%. Regarding the specific characteristics of the MEP changes, only a signal loss of 80% or greater was significantly associated with a higher rate of neurological deficit (23.0% vs 0.0% for loss of less than 80%, $p = 0.021$); changes of less than 80% were not associated with postoperative deficits.

CONCLUSIONS Neuromonitoring has a low positive predictive value and low sensitivity for detecting new neurological deficits. Even when neuromonitoring is unchanged, patients can still have new neurological deficits. The utility of transcranial MEP monitoring for lumbar PSO remains unclear but there may be advantages to its use.

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KEYWORDS complications; osteotomy; neuromonitoring; spinal deformity; three-column; lumbar; surgical technique

THE surgical correction of fixed spinal deformities often requires extensive osteotomies, aggressive spinal manipulation, and long-construct instrumented reconstruction. Deformity surgery can be associated with high risks for major complications, neurological morbidity, and large-volume blood loss.^{1–3,8,16–18} The previously reported intraoperative complication rate is around 7%, and

new neurological deficits are observed in approximately 7% to 10% of patients.^{3,8} Major permanent neurological injury occurs in fewer than 3% of cases.⁵ Multimodality intraoperative neuromonitoring (IONM) via somatosensory evoked potentials (SSEPs), motor evoked potentials (MEPs), and electromyography has played a critical role in minimizing and preventing neurological injury when

ABBREVIATIONS ASD = adult spinal deformity; IONM = intraoperative neuromonitoring; MEP = motor evoked potential; NPV = negative predictive value; PPV = positive predictive value; PSO = pedicle subtraction osteotomy; SSEP = somatosensory evoked potential; UIV = uppermost instrumented vertebra.

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used in appropriate settings. IONM allows the surgeon to be notified of early neurological changes (a surrogate for impending or active injury) and proactively reverse the offending mediator.

IONM, specifically monitoring of SSEPs and MEPs, has been shown to be beneficial when operating in the cervical and thoracic region where the spinal cord is present.¹⁴ The use of IONM has been studied thoroughly in pediatric spinal deformity patients and has been demonstrated to be highly valuable.⁴ The sensitivity and specificity of IONM changes to detect neurological deficits during deformity surgery in pediatric patients are both greater than 80%.^{20,21} However, the false-positive rate is significantly higher for IONM used during deformity surgery in adults.⁷ Pediatric spinal cord physiology and anatomical spinal deformities are very different from what is seen in adult patients, who oftentimes require very different surgical interventions. Pediatric cases often involve scoliosis, coronal imbalance, and the thoracic region, whereas adult spinal deformity (ASD) cases more often involve sagittal imbalance, spinopelvic mismatch, and the lumbar spine. Thus, the results from pediatric studies may not be directly applicable to an ASD population.

MEP use is best understood in spine surgery as a measure for spinal cord monitoring, and the true utility of using transcranial MEPs when operating in the lumbar spine (a region of thecal sac and nerve roots without spinal cord) remains controversial, even for ASD cases that require 3-column osteotomies. However, MEP monitoring should detect any injuries along the motor pathways from the cortex down to the muscle fibers; disruption or injury along the pathway should result in MEP changes if the monitoring is sensitive enough. For these reasons, it is important to understand the efficacy and utility of MEP monitoring during lumbar cases. While not all spine surgeons use IONM, there is a general recommendation to utilize neuromonitoring in all spinal deformity cases; this recommendation was based mainly on the pediatric literature and consensus by an expert panel.^{12,24} There is a lack of studies evaluating the utility of transcranial SSEPs and MEPs for thoracolumbar 3-column osteotomies in ASD patients. The available studies have small sample sizes,¹⁰ and even among the limited publications there are mixed conclusions.²³ Therefore in this study we aim to evaluate the utility of IONM, specifically monitoring of MEPs, to detect new neurological deficits during lumbar pedicle subtraction osteotomy (PSO) in ASD patients. We report the performance measures and examine MEP signal characteristics that may be associated with neurological deficits.

Methods

This study was formally approved by the Committee of Human Research at the University of California, San Francisco.

Patients

All adult patients (18 years or older) with ASD who underwent a 3-column osteotomy for spinal deformity correction performed by the senior author (C.P.A.) during 2006–2016 were retrospectively identified. This was

a consecutive series of patients. From this cohort, patients who underwent a PSO at L1, L2, L3, L4, L5, or S1 were further isolated for final analysis. Patients who had diagnoses of infection, acute trauma, and/or tumor were excluded. Indications for surgery were fixed, nonmobile spinal deformity causing abnormal spinal imbalance, significant dysfunction, debilitating axial back pain, and/or neurological deficits (radiculopathy and/or myelopathy). All patients underwent open PSO with long-segment instrumentation; there were no minimally invasive cases.

MEP Monitoring

Intraoperative monitoring of transcranial MEPs was utilized for all lumbar PSO cases. MEP responses are recorded for transcranial electrical stimulation delivered through 2 corkscrews placed at C3' and C4' sites (International 10–20 system) with a train of 9 pulses, 200 to 500 V, 75-msec pulse width, and 1–3 msec of interstimulus interval using a Cadwell Cascade system (Cadwell Laboratories Inc.). Anodal stimulation was applied to trigger contralateral MEP responses. Baseline MEPs are established after positioning. MEP responses were recorded from paired needle electrodes placed bilaterally in the psoas, adductors, quadriceps (rectus femoris/vastus medialis), tibia anterior, extensor hallucis longus, extensor digitorum brevis, gastrocnemius, abductor hallucis muscle, and hand muscle as control.

MEPs were checked intermittently during instrumentation and PSO. Prior to closure of the PSO, MEP signals were checked, and immediately following PSO closure MEP signals were checked again. Following this, MEPs were checked every 2 to 3 minutes for 20 minutes and then every 5 to 10 minutes until the first layer of fascia was closed for cases without MEP changes. If there were MEP changes, MEP signals were checked more frequently based on specific circumstances and continued until the completion of the case. In the event of an MEP change, standardized assessments were taken: checking for changes in anesthesia, temperature, hypotension, hypovolemia, technical difficulties with neuromonitoring, or direct neural compression. In all cases, patients underwent maneuvers in attempt to reverse the cause to MEP changes such as augmenting blood pressure (correction hypotension or increasing mean arterial pressure) and/or further decompression/reversal of spinal correction.

Data Points

Neuromonitoring records and complete MEP data strips for all patients were examined by a single neurophysiologist in a retrospective fashion. The neurophysiologist was blinded to the patient's clinical course and neurological status. The specific data points of interest were the incidence of changes/reduction in MEP signals, whether the signal change was transient or permanent, extent of recovery of the signal, timing of the signal change relative to PSO closure, and degree of signal loss relative to baseline measurements. The threshold of a positive MEP signal change was defined as a reduction in MEP amplitude of at least 50% as compared to baseline recordings.

Demographics, baseline clinical variables, and surgical

details were retrospectively reviewed and recorded: age (< 50, 50–64, 65–79 years), sex, weight (< 80 or ≥ 80 kg), neurological deficit (normal strength vs weakness on neurological examination), osteotomy level (L1, L2, L3, L4, L5, or S1), number of instrumented levels (≤ 10 vs > 10), uppermost instrumented vertebra (UIV) (upper vs lower thoracic), prior surgery, staged anterior/lateral-posterior, and comorbidities (cardiac disease, hypertension, vascular disease, diabetes, pulmonary disease, renal disease, stroke, psychiatric disease, hyperlipidemia, and thyroid disease). Preoperative neurological deficit was based on lower-extremity strength; if the strength examination did not result in a score of 5/5, the patient was categorized as having weakness. Upper thoracic levels were defined as T1, T2, T3, T4, T5, T6, and T7. Thoracolumbar junction levels were defined as T8, T9, T10, T11, T12, L1, and L2.

The primary outcome of interest was any new or worsened neurological deficit that was present following surgery; the neurological deficit could be motor or sensory in nature. Motor deficit was determined based on strength examination, and sensory deficit was based on light touch sensation examination of both lower extremities. The secondary outcome of interest was perioperative complication. A complication was defined as any unforeseen event requiring additional medical and/or surgical intervention. The perioperative period was defined as time from surgical intervention to time of discharge from the hospital.

Statistical Analysis

First, descriptive statistics were used to summarize the cohort, and univariate statistics (via 2-tailed chi-square tests) were used to assess for associations between covariates and outcomes of interest. A multivariate logistic regression model was then employed to assess whether MEP change was an independent predictor of the outcomes of interest. All covariates with *p* values less than 0.200 were included in the multivariate model. Performance measures for the ability of MEP changes to detect new neurological deficits were derived from a 2 × 2 contingency table: accuracy, positive predictive value (PPV), negative predictive value (NPV), sensitivity, and specificity. Lastly, univariate 2-tailed chi-square tests were used to evaluate whether specific MEP change characteristics were associated with neurological deficits. A *p* value < 0.050 was used as the threshold of statistical significance. All statistics were performed with the use of SAS 9.4 software (SAS Institute).

Results

A total of 242 patients were included in the final analysis. Their mean age was 65.1 years and 39.7% were male. The levels at which PSO was performed were as follows: L1 (5.8%), L2 (7.9%), L3 (51.7%), L4 (30.2%), L5 (2.5%), and S1 (2.1%). Among the cohort, 48.3% had 10 or more levels instrumented, and the construct's UIV was in the upper thoracic spine 48.3% of the time. Of the 242 patients, 72.7% had undergone prior surgery. Intraoperative MEP signal changes occurred in 38 cases (15.7%). Of the 38 MEP changes, 2 changes (5.3%) were not directly correlative to the myotomes of the associated PSO level (nerve root above and below). In one case a decrease in

extensor hallucis longus response was observed during an L1 PSO and in the other there was decrease in all bilateral lower-extremity myotomes during an L3 PSO. These changes were transient in 55.3% of cases and permanent (no or only partial recovery of MEP signal) in 44.7% of cases. Among the 38 cases with MEP signal changes, 9 (23.7%) had no recovery of MEP signal, 8 (21.1%) had partial recovery, and 21 (55.3%) had complete recovery (transient changes). The mean time of MEP signal change was 8.8 minutes after PSO closure (range: during PSO closure to 55 minutes after PSO closure). The average reduction in MEP signal amplitude was 72.9%. Three (7.9%) of the MEP changes observed were secondary to hypotension and resolved with normalization of blood pressures.

Table 1 shows the results of univariate analyses assessing associations between MEP changes and other covariates and outcomes of interest. The overall perioperative complication rate was 25.2%, and new postoperative neurological deficits occurred in 4.1% of cases. There were no significant differences in the rates of complication (31.6% vs 24.0%, *p* = 0.324) or neurological deficit (7.9% vs 3.4%, *p* = 0.204) between patients with and without intraoperative MEP changes. A multivariate model was then deployed to determine whether MEP signal change was an independent predictor of complications or neurological deficits (Table 2). MEP signal change was not an independent predictor of complications (OR 1.38, 95% CI 0.55–3.45, *p* = 0.495) or neurological deficits (OR 1.90, 95% CI 0.39–9.37, *p* = 0.429). There were no identified independent predictors of neurological deficits. Baseline renal disease was identified to be an independent predictor of complications. Patients with renal disease had 6.30 higher odds of complication compared to patients without renal disease (95% CI 1.90–20.92, *p* = 0.003).

Calculated performance measures of at least 50% change in transcranial MEP monitoring for the detection of new postoperative neurological deficits during lumbar PSO can be seen in Table 3. A total of 3 true positives were detected among the whole cohort of 242 patients. The overall accuracy was 82.6%, with most of the accuracy accounted for by true negatives; there were 187 true negatives and 7 false negatives. Overall, the PPV was 7.9% and the NPV was 96.6%. In other words, neurological deficit occurred in 7.9% of cases in which MEP signal change was observed. When no MEP signal changes were observed, 96.6% of patients did not have new neurological deficits. The sensitivity and specificity were 30.0% and 84.9%, respectively.

Table 4 shows details and assesses whether certain characteristics of MEP changes are associated with new neurological deficit. Permanent MEP changes trended toward higher rates of neurological deficit, but this was not statistically significant (11.8% vs 4.8%, *p* = 0.426). Related to this, patients who had MEP changes with no recovery or only partial recovery had higher neurological deficit rates, but the difference was not statistically significant: 11.1% (no recovery), 12.5% (partial recovery), and 4.8% (complete recovery) (*p* = 0.724). Patients with MEP changes occurring after 9 minutes of PSO closure or at other times not related to PSO closure had higher rates of neurological deficits, but this again was a trend and not statistically significant: 0 to 9 minutes from PSO closure

TABLE 1. Univariate analysis of covariates to complications and neurological deficits following lumbar PSO

	All Cases		Complications			Neurological Deficits		
	n	%	n	%	p Value	n	%	p Value
Total	242		61	25.2		10	4.1	
Neuromonitoring changes					0.324			0.204
Yes	38	15.7	12	31.6		3	7.9	
No	204	84.3	49	24.0		7	3.4	
Age (yrs)					<0.001			0.770
<50	17	7.0	2	11.8		1	5.9	
50 to 64	88	36.4	11	12.5		4	4.5	
65 to 79	115	47.5	38	33.0		5	4.3	
>79	22	9.1	10	45.5		0	0.0	
Sex					0.250			0.983
Male	96	39.7	28	29.2		4	4.2	
Female	146	60.3	33	22.6		6	4.1	
Weight (kg)								0.957
<80	119	49.2	28	23.5		5	4.2	
≥80	123	50.8	33	26.8		5	4.1	
Strength					0.968			0.238
Normal	179	74.0	45	25.1		9	5.0	
Abnormal	63	26.0	16	25.4		1	1.6	
Osteotomy level					0.125			0.025
L1	14	5.8	3	21.4		0	0.0	
L2	19	7.9	6	31.6		1	5.3	
L3	125	51.7	24	19.2		1	0.8	
L4	73	30.2	24	32.9		6	8.2	
L5	6	2.5	1	16.7		1	16.7	
S1	5	2.1	3	60.0		1	20.0	
No. of instrumented levels					0.138			0.553
≤10	117	48.3	26	22.2		6	5.1	
>10	115	47.5	35	30.4		4	3.5	
UIV					0.494			0.397
Upper thoracic	104	43.0	29	27.9		3	2.9	
Lower thoracic	138	57.0	32	23.2		7	5.1	
Prior surgery					0.432			0.099
Yes	176	72.7	42	23.9		5	2.8	
No	66	27.3	19	28.8		5	7.6	
Two-stage anterior-posterior					0.046			0.551
Yes	54	22.3	8	14.8		3	5.6	
No	188	77.7	53	28.2		7	3.7	
Comorbidities								
Cardiac disease					0.006			0.076
Yes	56	23.1	22	39.3		0	0.0	
No	186	76.9	39	21.0		10	5.4	
Hypertension					0.017			0.952
Yes	143	59.1	44	30.8		6	4.2	
No	99	40.9	17	17.2		4	4.0	
Vascular disease					0.679			0.707
Yes	17	7.0	5	29.4		1	5.9	
No	225	93.0	56	24.9		9	4.0	

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TABLE 1. Univariate analysis of covariates to complications and neurological deficits following lumbar PSO

	All Cases		Complications			Neurological Deficits		
	n	%	n	%	p Value	n	%	p Value
Comorbidities (<i>continued</i>)								
Diabetes					0.407			0.763
Yes	40	16.5	8	20.0		2	5.0	
No	202	83.5	53	26.2		8	4.0	
Pulmonary disease					0.528			0.476
Yes	45	18.6	13	28.9		1	2.2	
No	197	81.4	48	24.4		9	4.6	
Renal disease					<0.001			0.753
Yes	18	7.4	13	72.2		1	5.6	
No	224	92.6	48	21.4		9	4.0	
Stroke/TIA					0.033			0.284
Yes	9	3.7	5	55.6		1	11.1	
No	233	96.3	56	24.0		9	3.9	
Psychiatric disease					0.965			0.279
Yes	59	24.4	15	25.4		1	1.7	
No	183	75.6	46	25.1		9	4.9	
Hyperlipidemia					0.205			0.211
Yes	57	23.6	18	31.6		4	7.0	
No	185	76.4	43	23.2		6	3.2	
Thyroid disease					0.055			0.635
Yes	37	15.3	14	37.8		1	2.7	
No	205	84.7	47	22.9		9	4.4	

TIA = transient ischemic attack.

(3.8%), more than 9 minutes (11.1%), and other situations (33.3%) ($p = 0.184$). The only signal characteristic that was significantly associated with neurological deficit was the extent of MEP reduction. Patients with MEP signal loss of 80% or more had a significantly higher rate of neurological deficit (23.1% vs 0.0%, $p = 0.021$).

Table 5 shows the performance measures of minimum 80% change in transcranial MEP monitoring for the detection of new postoperative neurological deficits during lumbar PSO. A total of 2 true positives were detected. The overall accuracy was 92.6%, with most of the accuracy accounted for by true-negative rates; there were 222 true negatives and 8 false negatives. Overall, the PPV was 16.7% and the NPV was 96.5%. The sensitivity and specificity were 20.0% and 95.7%, respectively.

Images from an illustrative case are shown in Figs. 1 and 2. This 69-year-old man underwent T10 to pelvis posterior spinal fusion and instrumentation and L3 PSO for rigid flat back syndrome and scoliosis. Approximately 3 minutes after closing down the L3 osteotomy, there was greater than 80% loss in the rectus femoris, a 50% loss in the vastus medialis, and a 60% loss in the anterior tibialis (Fig. 2). The surgeon released the correction at the osteotomy site and identified compression of the L3 nerve root by a small bone fragment. MEP signals did not recover to baseline. Postoperatively, the patient had 2/5 strength in leg extension. This case exemplifies the importance of

multimiotomal monitoring, even for the individual muscles of the quadriceps.

Discussion

One of the most devastating complications that can occur during spinal deformity surgery is spinal cord or nerve root injury resulting in neurological dysfunction. Patients who undergo extensive osteotomies and curvature correction are at a high risk: new neurological deficits are observed in approximately 7% to 14% of patients following 3-column osteotomy in ASD.^{3,5,8,23} Fortunately, major permanent neurological injury occurs less than 3% of the time.⁵ Multimodality transcranial monitoring has been a mainstay as a result of its ability to offer real-time information regarding neurological status and warn the surgeon of impending or present neurological injury. It has been shown to be most helpful in pediatric deformity cases and when manipulating areas around the spinal cord.¹⁴ However, its utility in lumbosacral surgery is controversial, given concern regarding low sensitivity for detecting new neurological deficits. In a study of 1036 patients who underwent posterior spinal fusion, the sensitivity to detect a neurological deficit was 13% when there was complete loss of signal and 20% when changes in response were present.¹³ In addition, the rate of nerve injury when operating in the lumbar region is quite low, around 2%.¹⁹

TABLE 2. Multivariate analysis showing no association between neuromonitoring changes and outcomes following lumbar PSO

	Complications			Neurological Deficits		
	OR	95% CI	p Value	OR	95% CI	p Value
Neuromonitoring changes						
Yes	1.38	0.55–3.45	0.495	1.90	0.39–9.37	0.429
No	Ref	Ref	Ref	Ref	Ref	Ref
Age (yrs)						
<50	Ref	Ref	Ref			
50–64	0.92	0.17–4.86	0.919			
65–79	2.47	0.49–12.48	0.274			
>79	2.92	0.45–18.78	0.259			
Osteotomy level						
L1	Ref	Ref	Ref	Ref	Ref	Ref
L2	1.17	0.19–7.21	0.864	>999.99	<0.01 to >999.99	0.953
L3	0.85	0.21–3.54	0.826	>999.99	<0.01 to >999.99	0.963
L4	1.83	0.43–7.74	0.411	>999.99	<0.01 to >999.99	0.952
L5	1.38	0.09–21.43	0.818	>999.99	<0.01 to >999.99	0.949
S1	7.91	0.72–86.82	0.091	>999.99	<0.01 to >999.99	0.947
No. of instrumented levels						
≤10	Ref	Ref	Ref			
>10	1.31	0.65–2.62	0.454			
Prior surgery						
Yes				0.52	0.13–2.05	0.350
No				Ref	Ref	Ref
Two-stage anterior-posterior						
Yes	0.80	0.31–2.02	0.630			
No	Ref	Ref	Ref			
Cardiac disease						
Yes	1.31	0.61–2.83	0.488	<0.01	<0.01 to >999.99	0.892
No	Ref	Ref	Ref	Ref	Ref	Ref
Hypertension						
Yes	1.37	0.67–2.80	0.385			
No	Ref	Ref	Ref			
Renal disease						
Yes	6.30	1.90–20.92	0.003			
No	Ref	Ref	Ref			
Stroke/TIA						
Yes	2.98	0.66–13.40	0.155			
No	Ref	Ref	Ref			

Many of the studies evaluating the utility of transcranial MEPs in spinal deformity surgery stem from the pediatric literature or include mixed cohorts, limiting the applicability of those findings for ASD patients with sagittal imbalance secondary to lumbosacral pathology.^{4,7,9,20–22,24} Even among the ASD population, the utility of MEP monitoring should be evaluated within a specific deformity type and a specific procedure. One of the most common posterior-based osteotomies performed for the correction of fixed lumbar spinal deformities is a PSO (Schwab grade 3 and 4 osteotomy).¹⁵ There is a paucity of published studies (2

retrospective studies) investigating the value of transcranial MEPs during lumbar PSO and their conclusions are contradictory.^{10,23}

The first study was published by Lieberman et al.¹⁰ Their study included 35 patients who underwent posterior-based lumbar osteotomies for fixed sagittal imbalance over a 2-year period; 26 patients underwent PSO and the rest underwent Smith-Petersen osteotomies (SPOs). The authors retrospectively examined MEP responses and associated clinical outcomes. Among their cohort, 25 patients (71%) had greater than 80% reduction in MEP

TABLE 3. Performance measures of MEP monitoring to detect new neurological deficits at minimum 50% decrease

	Neurological Deficits			% Value
	Positive	Negative	Total	
MEP changes				
Positive	3	35	38	
Negative	7	197	204	
Total	10	232	242	
Performance				
Accuracy				82.6
PPV				7.9
NPV				96.6
Sensitivity				30.0
Specificity				84.9

amplitude in at least 1 muscle group. Their reported incidence of MEP changes was remarkably higher than that observed in our study (overall incidence of MEP changes at 15.7%, and MEP changes greater than 80% at 5.4%). The persistence of reduced MEP signal seems to be an important characteristic associated with a true neurological deficit. In Lieberman and colleagues' study, 15 patients had improvement in their MEP signals and all of those patients had no new neurological deficit postoperatively. All patients who continued to have reduced MEP signals at the end of the case demonstrated evidence of neurological injury. We observed a similar trend of higher rates of neurological deficits when signal reduction was permanent and did not completely return to baseline.

The question of whether transcranial monitoring is essential when performing lumbar PSO depends on its ability to detect and prevent neurological complications. According to the published literature, we understand the sensitivity and specificity of MEP monitoring during deformity surgery to be greater than 80% when operating near the spinal cord.^{7,20,21} The performance measures for lumbar spine surgery, specifically in cases requiring lumbar PSO, are less clear. While reporting of performance measures of intraoperative transcranial MEPs depends on the threshold used, past studies report sensitivities and specificities of 67%–100% and 81%–98%, respectively.^{10,14,19} There is support for the notion that sensitivity decreases and specificity increases when osteotomies are performed for deformity correction.¹⁴ Contrary to past studies, our observed sensitivity of MEPs for detecting a new neurological deficit was much lower at 30.0%, but the specificity was comparable (84.9%). This difference may be due to the fact that past studies were composed of cohorts with mixed spinal deformities (heterogeneous sample), utilized a variety of surgical techniques, established different threshold criteria (MEP reduction) as “signal change,” and had small patient cohort sizes (relative to the incidence of neurological deficit). The largest series included 102 patients, only 29 of whom underwent an osteotomy. With neurological deficits being uncommon (particularly when osteotomies are not performed), it would be very difficult to assess the true performance of a test

TABLE 4. Associations between MEP changes to incidence of neurological deficits

	All Pts		Pts w/ Neurological Deficit		p Value
	n	%	n	%	
Total	38		3	7.9	
Change					0.426
Transient	21	55.3	1	4.8	
Permanent	17	44.7	2	11.8	
Neuromonitoring recovery					0.724
None	9	23.7	1	11.1	
Partial	8	21.1	1	12.5	
Complete	21	55.3	1	4.8	
Time from PSO closure (mins)					0.184
0–9	26	68.4	1	3.8	
>9	9	23.7	1	11.1	
Other	3	7.9	1	33.3	
Percentage loss (%)					0.021
<80	21	61.8	0	0.0	
≥80	13	38.2	3	23.1	

Pts = patients.

in a cohort of this size. One would expect to see greater sensitivities with lower MEP reduction thresholds. Within the published literature, MEP reduction thresholds ranged from 50% to 80%. Our study used MEP reduction of at least 50% and still observed very low sensitivity. Based on our large, homogeneous cohort of ASD patients who underwent PSO performed by the same surgeon and monitoring by the same neuromonitoring team, it seems intraoperative MEP monitoring is not sensitive in the detection of neurological deficits at a 50% threshold. In fact, only 3 true positives were observed among 242 patients, a PPV of 7.9%. This is further supported by multivariate analysis showing MEP signal changes were not associated with neurological deficits or complications. While specificity of approximately 85% is significantly better, this still means

TABLE 5. Performance measures of MEP monitoring to detect new neurological deficits at minimum 80% decrease

	Neurological Deficits			% Value
	Positive	Negative	Total	
MEP changes				
Positive	2	10	12	
Negative	8	222	230	
Total	10	232	242	
Performance				
Accuracy				92.6
PPV				16.7
NPV				96.5
Sensitivity				20.0
Specificity				95.7

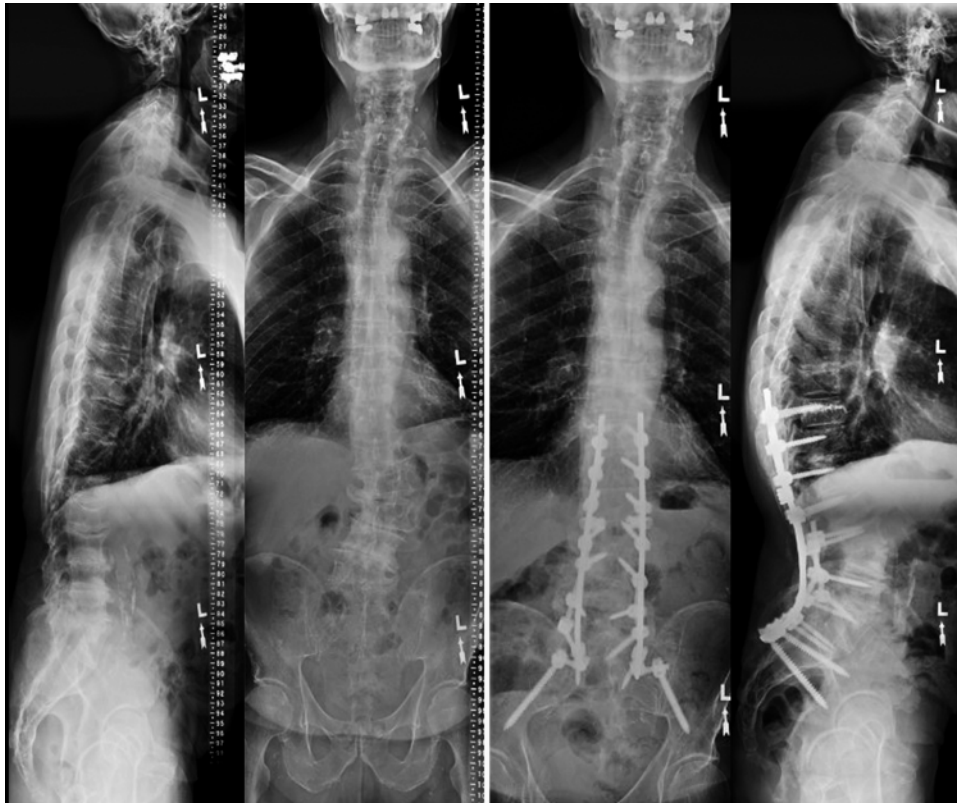


FIG. 1. Preoperative (left) and postoperative (right) standing radiographs of a patient who underwent an L3 PSO. The preoperative standing radiographs demonstrate flattening of the thoracic and lumbar spine and scoliosis. Lumbar pelvic parameters were as follows: sagittal vertical axis (SVA) of 3.0 cm, lumbar lordosis (LL) of -7° (kyphosis), pelvic incidence (PI) of 46° , and scoliosis of 29° . After L3 PSO, the lumbar pelvic parameters were as follows: SVA of 3.5 cm, LL of 65° , and scoliosis of 7° .

that 15% of the patients in whom no signal change is detected will nevertheless have a postoperative neurological deficit. Further supporting this observation, Buchowski et al. performed a retrospective study of 102 patients who underwent lumbar PSO. The neurological complication rate was 11%, but IONM did not detect any of the observed deficits (false negatives).⁵ Similarly, in a study by Eager et al. of 2095 spinal fusion cases, 17 were noted to have IONM changes, but none of the patients in these cases had postoperative neurological deficits (i.e., the changes were false positives).⁶

Given these mixed findings, there are questions regarding whether transcranial MEP monitoring should be used during lumbar PSO, and if so how the signals should be interpreted. In addition to low performance measures, there are direct and indirect drawbacks to using MEP monitoring, such as increased operating time and cost and the intrinsic risks associated with interventions done in the setting of signal change (such as blood pressure augmentation, greater spinal decompression, and intraoperative delay of surgical case).¹¹ A study by Trobisch et al. reported the perioperative outcomes of 22 patients who underwent lumbar PSO without neuromonitoring.²³ Two patients experienced a new unilateral foot drop, a neurological complication rate of 9%, which is similar to the published rate in patients undergoing lumbar PSO.⁵ Use of MEP monitoring for specific patients and settings may be more appro-

priate. There is evidence that patients with baseline neurological defects are at higher risk for nerve injury and are more likely to demonstrate changes in IONM; it is possible this specific cohort should be monitored.¹³

According to the findings from our study, the majority (68.4%) of MEP changes occurred within 10 minutes of PSO closure, and most surgeons would agree it is important to monitor the patient during this critical period. While MEP changes occur less frequently outside this critical period, it seems that neurological deficits are present more often when there is change in MEPs. This may be due to the nature of the nerve injury and reversibility (i.e., release of PSO closure can improve or reverse the neuromonitoring changes). Monitoring after PSO closure may be beneficial if neuromonitoring is utilized.

The first step in utilizing IONM effectively is to establish reliable baseline MEP signals. Among our cohort of 242 patients, 30 were categorized as having unreliable proximal MEP signals, and 3 (10.0%) of those patients had postoperative neurological deficits. Therefore, establishing reliable baseline signals is critical when using IONM. However, there will always be cases in which MEP signals are deemed unreliable despite all measure of optimization, and this study accounts for this actuality. There is a lack of standardization and consensus regarding the “threshold” of MEP changes to use as an indicator for real neurological injury. A drop of 80% in MEP signal was a strong indi-

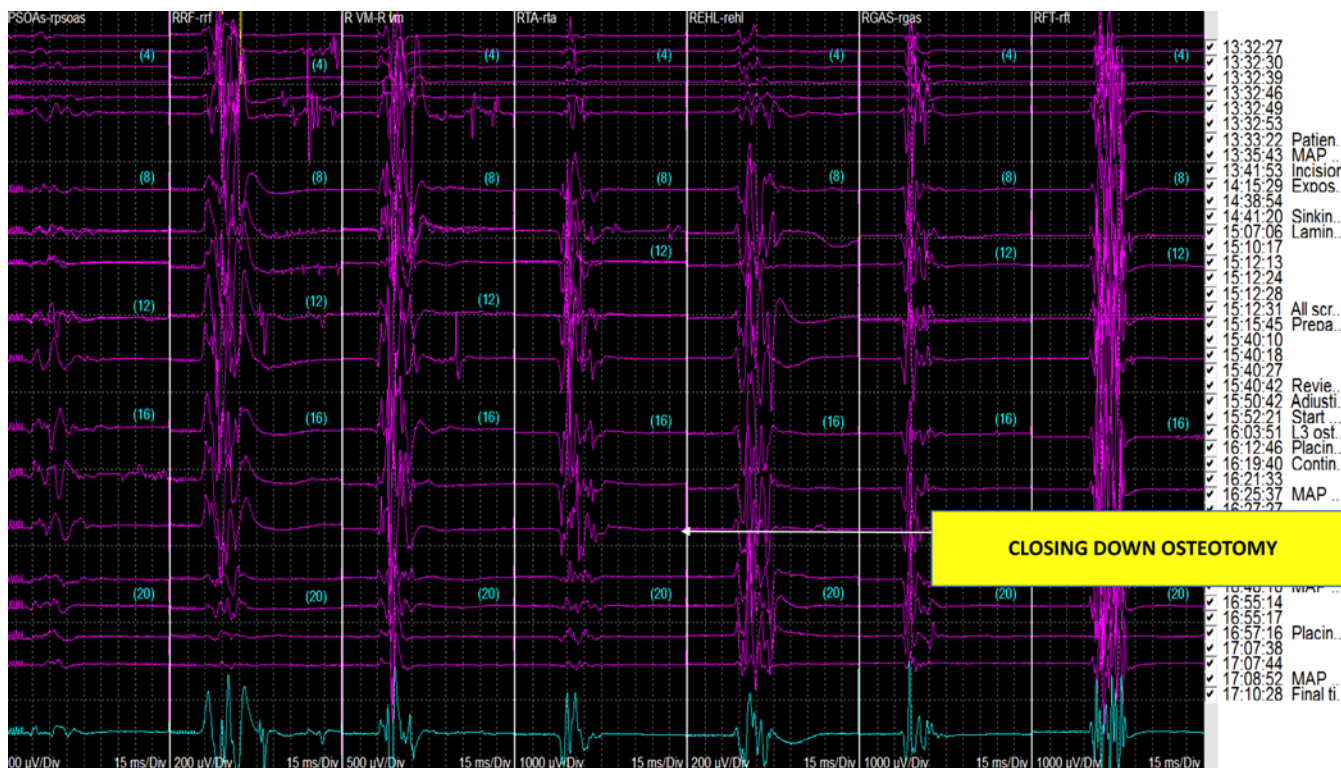


FIG. 2. Loss of multimyotomal signals following closure of L3 PSO. A greater than 80% loss in the right rectus femoris, 50% loss in the right vastus medialis, and 60% loss in the right anterior tibialis can be seen 3 minutes following closure of the osteotomy. REHL = right extensor hallucis longus; RFT = right foot; RGAS = right gastrocnemius; RRF = right rectus femoris; RTA = right tibialis anterior; R VM-R = right vastus medialis. Figure is available in color online only.

indicator for the presence of neurological deficit in our study. Establishment of a “signal change” threshold is important because sensitivity and specificity changes are based on an established threshold. Other studies have shown that a simple change of threshold from 65% to 50% decreases specificity by 5%.¹⁹ In this study, performance measures for thresholds of both 50% and 80% MEP decrease were calculated. From the 50% to the 80% threshold, there was a 10-percentage-point increase in accuracy, an 8.8-percentage-point increase in PPV, a 0.1-percentage-point decrease in NPV, a 10.0-percentage-point decrease in sensitivity, and a 10.8-percentage-point increase in specificity. While a threshold of 80% offers generally better performance measures, there should be caution that there is a higher chance of not detecting a postoperative neurological deficit (decreased sensitivity).

In addition, the specific lower-extremity muscle group used plays a role in the performance of MEP monitoring.¹⁰ Data suggest that lower-extremity adductors have high sensitivity (89%) and specificity (93%) for detecting new neurological deficits. Sensitivity increases to 100% when there are signal reductions in all muscle groups (rectus, adductors, vastus, and tibialis). Therefore, interpretation of “significant” signal changes should include evaluation of multiple myotomes, especially when performing lumbar PSO, because an insufficient number of myotomes reduces sensitivity and can lead to false negatives.

Based on the findings and data from this study, it seems the utility of transcranial MEPs during lumbar PSO is limited,

and MEP monitoring may not be required for every case or throughout the case. However, there are drawbacks to this study, mainly related to the low incidence of neurological deficits and the retrospective design. The main limitation in relying on a retrospective review of medical records relates to the general accuracy and granularity of medical documentation. Evaluating outcomes like neurological deficit can be difficult and our analysis thus most likely results in underestimation of the true neurological deficit rate. In this study, only 10 (4.1%) patients had new neurological deficits and only 38 cases had at least 50% reduction in MEP. These are promising findings in regard to patient outcomes, but the power of the study analysis is limited. Specifically, we saw trends of higher neurological deficit rates with permanent signal reduction and with signal changes that do not recover to baseline. Even though this is the largest cohort study on this topic to date, a study with greater power might demonstrate significance where ours did not. Because of the retrospective nature of this study, there are factors that may not be easily accounted for in the analysis and may impact the interpretation of the results. An important consideration is to take into account what is done when signal changes occur during surgery. Upon review of the cases, the senior author (C.P.A.) consistently reverses correction when possible, performs wide decompressions, increases mean arterial pressures, assesses anesthesia regimen, and checks neuromonitoring leads. These maneuvers may have helped minimize neurological injury and clinical neurological deficit; this may

indirectly explain the low sensitivity and PPV and indicate that neuromonitoring may be useful.

Conclusions

Lumbar PSO is a formidable but effective surgical technique for the correction of fixed spinal deformities. The utility of IONM via transcranial MEP monitoring is controversial for lumbar spine surgery, and there are very few studies on this specific topic. Based on this large contemporary data set of ASD patients who underwent lumbar PSO, the neurological complication rate is low and the incidence of neuromonitoring changes is infrequent. Reduction of at least 50% MEP signal was not a predictor of complications or neurological deficits. In fact, the sensitivity and PPV (true-positive rate) were very low (30.0% and 7.9%, respectively). The specificity and NPV are comparable to those reported in past studies, but false negatives still exist, with patients without MEP changes being found to have a new neurological deficit. Our results show that patients who experience a reduction of 80% or greater in MEP signals have much higher rates of postoperative neurological deficits. Permanent reductions in MEP signals and MEP changes that occur outside the time period of PSO closure are associated with higher rates of neurological deficit, but this was not statistically significant. Overall, transcranial MEP monitoring seems to be limited in its ability to reliably predict neurological injury during lumbar PSO. But it may still be able to offer the surgeon an opportunity to reverse potential injuries. Its application should be re-evaluated in a prospective manner.

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Author Contributions

Conception and design: Ames. Acquisition of data: Lau, Dalle Ore. Analysis and interpretation of data: Lau, Dalle Ore, Ames. Drafting the article: Lau, Dalle Ore. Critically revising the article: Lau, Reid, Safae, Ames. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Lau. Statistical analysis: Lau. Study supervision: Deviren, Smith, Shaffrey, Ames.

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