Severe Cerebral Palsy and Short Stature Predict Absent Baseline IONM Signals in Pediatric Neuromuscular Scoliosis Surgery

Pochih Shen, MD, PhD,†‡ Alan Robert Bielsky, MD,*§ Thanh Nguyen, MD,*§ Carson Keeter, MS,‖ Susan Walczak, MS,* Tyler Sullivan, BS,* Nancy Hadley-Miller, MD,*‖¶ and Mark A. Erickson, MD*‖¶*

Background: Intraoperative neurophysiological monitoring (IONM) is essential for detecting potential neurological injury during scoliosis surgery, but obtaining recordable baseline signals can be challenging in neuromuscular scoliosis (NMS) patients. Absent baseline IONM signals, characterized by unattainable initial IONM responses despite technical and anesthetic optimization, present significant challenges to intraoperative neurological assessment and surgical risk stratification. This study aims to identify predictive factors for absent baseline IONM signals in pediatric NMS patients and establish a clinically applicable risk prediction model.

Methods: This retrospective study initially identified 118 nonambulatory NMS patients under 18 years old who underwent spinopelvic fusion between 2013 and 2022. All patients received standardized total intravenous anesthesia (TIVA) protocol to optimize signal acquisition. After excluding 3 patients with spinal cord injuries, 115 patients were analyzed. Multimodality IONM, including somatosensory evoked potentials (SSEPs) and transcranial electrical motor evoked potentials (TcMEPs) was attempted in all cases. Clinical data and radiographic measurements were analyzed to determine predictive factors for absent baseline IONM signal. ROC curve analysis and logistic regression were used to determine optimal thresholds and predictive factors for absent baseline IONM signals.

Results: Thirty-eight (33%) had absent baseline lower extremity IONM signals. Cerebral palsy (CP) was the most significant predictive factor [odds ratio (OR): 9.615, $P < 0.001$], with 53.1% of CP patients having absent baseline IONM signals. Within the CP cohort, Gross Motor Function Classification System (GMFCS) level V (OR: 11.501, $P = 0.028$) and body height < 128.5 cm (OR: 4.097, $P = 0.044$) were significant risk factors. Three patients developed new-onset urinary incontinence postoperatively, though the relationship to IONM status remains undetermined.

Conclusion: Severe CP and shorter stature significantly increase the risk of absent baseline IONM signals in pediatric NMS patients. These findings inform preoperative risk assessment, enhance patient-specific surgical planning, and suggest the need for alternative monitoring approaches in high-risk cases. Such early identification of monitoring challenges can improve surgical preparation, consent processes, and ultimately patient care in this vulnerable population.

Level of Evidence: Level III.

Key Words: neuromuscular scoliosis, Intraoperative neurophysiological monitoring, somatosensory evoked potentials, transcranial electrical motor evoked potentials

(*J Pediatr Orthop* 2026;46:e164–e171)

Intraoperative neurophysiological monitoring (IONM) provides real-time assessment of neural structures during scoliosis surgery, facilitating early detection and prevention of iatrogenic neurological injury.[1–3] Spinal deformity surgery is often indicated for patients with nonambulatory neuromuscular diseases to prevent deformity progression and improve quality of life.[4,5] Despite the increased use of IONM in NMS surgery, its efficacy remains debated in this population.[5,6] Verifying IONM benefits in NMS patients is challenging due to the potential absent baseline IONM signals and difficulties in

Received for publication April 8, 2025; accepted August 4, 2025.

From the *Children's Hospital Colorado, Aurora, CO; †Department of Orthopedic Surgery, Kaohsiung Medical University Hospital, Kaohsiung, Taiwan; ‡School of Post-Baccalaureate Medicine, College of Medicine, Kaohsiung Medical University, Kaohsiung, Taiwan; §Department of Anesthesiology, University of Colorado School of Medicine, CO; ‖Department of Orthopedics, University of Colorado Anschutz Medical Campus, Aurora, CO; and ¶Musculoskeletal Research Center, Children's Hospital Colorado, Aurora, CO.

The data supporting this study's findings are available from the corresponding author upon reasonable request.

The authors declare that they received no funding from any external organization for the submitted work.

The institutional review board approved (BLIND FOR REVIEW) this retrospective study, and the requirement for informed consent was waived due to the retrospective study design.

The authors declare no conflicts of interest.

Reprints: Mark Alan Erickson, MD, Children's Hospital Colorado, 13123 East 16th Avenue, Aurora, CO 80045. E-mail: mark.erickson@childrenscolorado.org.

Supplemental Digital Content is available for this article. Direct URL citations are provided in the HTML and PDF versions of this article on the journal's website, www.pedorthopaedics.com.

DOI: 10.1097/BPO.0000000000003096

identifying changes in postoperative neurological function.[7–11]

The risk of postoperative neurological deficit increases without IONM.[12] Weight-bearing ability, motor function, and intracranial lesions influence baseline IONM signals.[6,13] However, most nonambulatory NMS patients cannot bear weight and have different neurological impairments. The decision-making and preparation for addressing potential absent baseline IONM signals in these patients pose significant challenges due to a lack of accompanying information.

Despite IONM's standard use in spinal deformity surgery, its utility in NMS patients is limited by frequently absent baseline signals. We aim to identify predictors of absent baseline IONM signals in pediatric NMS patients to create a risk stratification model for preoperative planning and alternative monitoring strategies.

METHODS

Study Participants

Following approval by the institutional review board, we retrospectively reviewed nonambulatory NMS patients under 18 years old who underwent spinopelvic fixation at our single institution between 2013 and 2022. The demographic data, intraoperative reports, and other associated medical records were collected through the electronic health medical record (Epic Systems Corporation).

Radiographic Measurements and Surgical Procedure

The major curve, T2-T12 kyphosis, and T12-S1 lordosis were assessed using the Cobb method.[14] Pelvic obliquity was measured using the Osebold method.[15] The sagittal vertical axis, sacral slope, and pelvic incidence were also carried out following the established protocol.[16] The surgery consisted of the usual posterior spinal fusion (PSF) with either pedicle screws or hooks. The fusion level extended from the upper thoracic spine to the pelvis, with most patients having their upper instrumented vertebrae (UIV) at T2 (n = 48) or T3 (n = 54), and a minority at other levels (C5/C6, T1, or T4). All patients received S2-alar-iliac (S2AI) screws for pelvic fixation.

Anthropometric Assessment

The Brooks growth charts were utilized to transform measurements of body height (BH), body weight (BW), and body mass index (BMI) into standardized percentiles for the cerebral palsy (CP) cohort.[17] These growth charts were individualized based on sex, Gross Motor Function Classification System (GMFCS) level, and gastrostomy tube use (specifically for children at GMFCS level V).

Anesthesia Procedure

Patients received standardized anesthesia protocols supervised by an anesthesiologist. Standard anesthesia monitoring was applied, and anesthesia was induced by inhalation or IV induction. If inhalational induction was used, volatile anesthetics were then discontinued after induction, washed out with high-oxygen and air gas flows for at least 30 minutes, and all patients received total intravenous anesthesia (TIVA) with propofol, ketamine, and remifentanil or sufentanil. Muscle relaxants were only given to facilitate intubation and, if utilized, reversed to preserve motor monitoring.

IONM Procedure

The neurophysiologist conducted multimodality IONM using a team approach supervised by the pediatric neurologist. Baseline measurements for all monitoring modalities were obtained after positioning and before incision. After addressing all correctable factors such as anesthetic management, patient positioning, and technical issues, the inability to obtain reliable lower extremity responses was documented as absent baseline IONM signals.[6] The protocol initially recommended discontinuing the IONM procedure if baseline IONM signals for both somatosensory evoked potentials (SSEPs) and transcranial motor evoked potentials (TcMEPs) could not be obtained from the lower extremity. This situation was documented as absent baseline IONM signals. In all cases where reliable lower extremity IONM signals could not be obtained despite optimization, the surgical team held a multidisciplinary discussion to exclude reversible causes. Families were informed of the monitoring limitations and potential risks associated with the surgery. In our cohort, all patients proceeded with the planned spinal procedure without modification, regardless of IONM availability. In some cases, IONM was continued selectively to monitor upper extremity function or assist with pedicle screw placement.

For the SSEPs monitoring, stimulating electrodes were placed over the ulnar and posterior tibial nerves, and recording electrodes consisted of needle electrodes positioned to capture the peripheral, cervical, and cortical potentials. Both cortical and subcortical signals were recorded for SSEPs. A significant signal change of SSEPs was defined as a complete loss, more than a 50% decrease in amplitude, or a 10% increase in latency.[18]

For the TcMEPs monitoring, stimulating electrodes were placed bilaterally over the motor strip area of the cranium. Compound muscle action potentials were recorded from bilateral upper and lower extremity muscles. The monitored muscles included the first dorsal interosseous, iliopsoas, adductor, quadriceps, tibialis anterior, abductor hallucis, abdominal, and anal sphincter muscles. A significant signal change of TcMEPs was defined as a 50% or more decrement in MEP amplitude or an increase in the stimulation threshold of 100 V or more from the baseline.[19]

The physiological status of the innervated roots was monitored through spontaneous and electrically triggered EMG (sEMG and tEMG) activity. Subdermal needle electrodes were placed in key muscle groups. Both legs were monitored for the iliopsoas, adductor, quadriceps, tibialis anterior, and abductor hallucis muscles. Bilateral recordings were also made for the abdominal and anal sphincter muscles. During the procedure, sEMG was

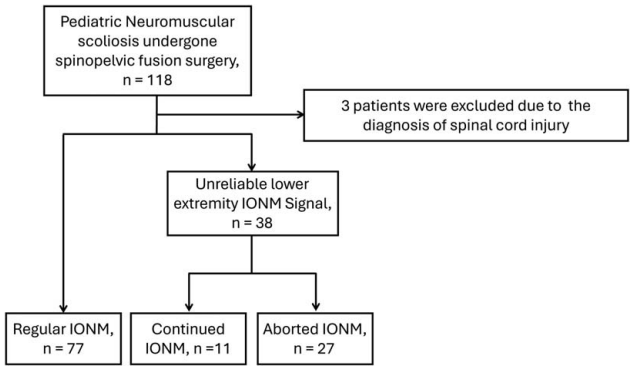

FIGURE 1. Flow chart of patient inclusion/exclusion. IONM, intraoperative neurophysiological monitoring.

continuously monitored for bursts or persistent changes. tEMG was used during pedicle screw implantation to detect potential breaches, with the stimulus threshold set to 7 mA.

Statistical Analysis

Continuous variables were compared using *t* tests, while ROC curve analysis identified optimal thresholds for predictors. Univariate and multivariate logistic regression determined significant predictors of absent baseline IONM signals, with results expressed as odds ratios and 95% CI. All statistical analysis was conducted using IBM SPSS Statistics (Version 25, SPSS Inc.).

RESULTS

Patient Characteristics and Group Difference

We initially identified 118 patients with NMS who underwent spinopelvic fusion surgery at our institution. Three cases were excluded because the patients had spinal cord injuries and did not exhibit any baseline IONM signals, making predictions unnecessary (Fig. 1). Thus, the final analysis included 115 patients, of whom 38 (33%) had absent baseline IONM signals from the lower extremities. IONM was discontinued in 27 patients as per protocol. IONM continued in 11 patients despite absent baseline IONM signals in the lower extremities for monitoring arm position or pedicle screw implantation as the surgeon's request.

Patients with absent baseline IONM signals in the lower extremities were classified into the signal-absent group (n = 38). In contrast, the remaining patients were assigned to the signal-detected group (n = 77). Among the 38 patients with absent baseline IONM signals, 34 had a diagnosis of CP. The remaining 4 patients were diagnosed with Krabbe disease, Aicardi-Goutières syndrome, traumatic brain injury, and a KIF1A-related disorder, respectively. Table 1 summarizes the demographic data and clinical characteristics between groups. Patients in the signal-absent group had significantly lower BH ($P < 0.001$), BW ($P < 0.001$), and BMI ($P = 0.048$) than the signal-detected group. The signal-absent group also had a higher incidence of unclosed triradiate cartilage ($P = 0.002$), CP diagnosis ($P < 0.001$), and preoperative incontinence ($P = 0.013$). However, the 2 groups had no significant difference in the severity of preoperative spinopelvic deformities (Table 2).

Intraoperative IONM Signal Changes and Clinical Outcomes

Among the patients with IONM during surgery, 33 IONM signal change events occurred. Five events were associated with the arm or leg position; the signal was returned to baseline after the limbs were repositioned. A total of 28 patients experienced signal changes during pedicle screw insertion or deformity correction. Postoperatively, 3 patients developed new-onset urinary incontinence; one recovered within 1 year, whereas the other 2 had no documented resolution. However, given the retrospective design and incomplete follow-up, the relationship between these events and IONM signal changes remains unclear, and causality cannot be established.

TABLE 1. Demographic Results of the Patients

Variable	Total cohort	Baseline IONM signals of lower extremity		*P*
		Signal-absent group	Signal-detect group	
Patient number	115	38	77	
Age at surgery (y)	12.8 ± 2.3	12.3 ± 2.6	13.1 ± 2.0	0.078
Male:female	50:65	18:20	32:45	0.554
Body height (cm)	139.8 ± 14.8	133.1 ± 14.9	143.1 ± 13.7	0.001
Body weight (kg)	35.4 ± 14.0	29.6 ± 8.1	38.3 ± 15.4	0.001
Body mass index (kg/m^2)	17.8 ± 4.3	16.6 ± 2.9	18.3 ± 4.7	0.045
Triradiate cartilage closed (Y:N)	82:33	22:16	60:17	0.026
Neuromuscular disease				
Cerebral palsy	64	34	30	< 0.001*
Spinal muscular atrophy	7	0	7	
Muscular dystrophy	13	0	13	
Rett syndrome	7	0	7	
Others	24	4	20	
Preoperative incontinence (Y:N)	87:28	35:3	52:25	0.005

*Compared cerebral palsy versus non-cerebral palsy cases; IONM indicates intraoperative neurophysiological monitoring.

TABLE 2. Preoperative Radiographic Measurements of the Patients

Variable	Total cohort	Baseline IONM signals of lower extremity		P*
		Signal-absent group	Signal-detect group	
Major curve Cobb angle (°)	76.9 ± 23.2	79.5 ± 19.3	75.6 ± 24.9	0.404
Pelvic obliquity (°)	17.7 ± 11.6	18.9 ± 13.8	17.1 ± 10.4	0.428
T2-T12 kyphosis (°)	37.2 ± 21.6	42.7 ± 19.5	34.5 ± 22.1	0.061
Lumbar lordosis (°)	34.6 ± 27.4	36.7 ± 27.2	33.5 ± 27.7	0.577
Sacral slope (°)	33.3 ± 20.5	33.8 ± 21.8	33.1 ± 20.0	0.876
Pelvic incidence (°)	49.4 ± 22.3	45.8 ± 23.3	51.2 ± 21.7	0.258
Sagittal vertical axis (mm)	54.6 ± 59.3	43.3 ± 60.6	60.2 ± 58.2	0.173

*The statistical analysis was performed using *t* tests.
IONM indicates intraoperative neurophysiological monitoring.

TABLE 3. Logistic Regression Analysis for Predicting Absent Baseline IONM Signals (Entire Cohort)

Variable	Univariate model		Multivariate model	
	Odds ratio (95% CI)	P	Odds ratio (95% CI)	P
Age at surgery (y)	0.840 (0.703-1.004)	0.056	—	—
Sex (male)	0.790 (0.362-1.726)	0.555	—	—
Cerebral palsy (Y:N)	13.317 (4.290-41.337)	< 0.001	9.615 (2.806-32.953)	< 0.001
Preoperative incontinence (Y:N)	5.609 (1.572-20.011)	0.008	1.911 (0.443-8.235)	0.385
Body mass index (kg/m^2)	0.894 (0.799-1.000)	0.049	0.978 (0.517-1.849)	0.944
Body weight (kg)	0.927 (0.884-0.972)	0.002	1.011 (0.744-1.374)	0.944
Body height (cm)	0.949 (0.919-0.980)	0.001	0.961 (0.830-1.112)	0.590
Preop MCCA (°)	1.007 (0.990-1.024)	0.401	—	—
Preop pelvic obliquity (°)	1.014 (0.980-1.048)	0.426	—	—
Preop T2-T12 kyphosis (°)	1.018 (0.999-1.038)	0.065	—	—
Preop lumbar lordosis (°)	1.004 (0.989-1.019)	0.574	—	—
Preop sagittal vertical axis (mm)	0.995 (0.988-1.002)	0.174	—	—

IONM indicates intraoperative neurophysiological monitoring; MCCA, major curve Cobb angle; preop, preoperative; Y:N, yes versus no.

TABLE 4. Demographic Result and Preoperative Deformity of Patients With and Without Cerebral Palsy

Variable	Total cohort	Neuromuscular siagnosis		P
		Cerebral palsy	Non-cerebral palsy	
Patient number	115	64	51	
Absent baseline IONM signals, (%)	38 (33)	34 (53.1)	4 (7.8)	< 0.001
Age at surgery (y)	12.8 ± 2.3	12.7 ± 2.5	13.1 ± 2.0	0.336
Sex (male:female)	50:65	27:37	23:28	0.754
Body height (cm)	139.8 ± 14.8	136.1 ± 15.1	144.4 ± 13.1	0.003
Body weight (kg)	35.4 ± 14.0	30.1 ± 7.4	42.2 ± 17.1	< 0.001
Body mass index (kg/m^2)	17.8 ± 4.3	16.2 ± 2.8	19.7 ± 4.9	< 0.001
Triradiate cartilage closed (Y:N)	82:33	24:40	9:42	0.019
Preop incontinence (Y:N)	87:28	58:6	29:22	< 0.001
Preop major curve Cobb angle (°)	76.9 ± 23.2	80.5 ± 22.3	72.3 ± 23.6	0.060
Preop pelvic obliquity (°)	17.7 ± 11.6	19.0 ± 13.1	16.0 ± 9.1	0.153
Preop T2-T12 kyphosis (°)	37.2 ± 21.6	40.0 ± 20.3	33.7 ± 22.8	0.133
Preop lumbar lordosis (°)	34.6 ± 27.4	34.4 ± 27.1	34.7 ± 28.1	0.960
Preop sacral slope (°)	33.3 ± 20.5	33.6 ± 18.4	33.0 ± 23.1	0.884
Preop pelvic incidence (°)	49.4 ± 22.3	49.1 ± 21.3	49.8 ± 23.7	0.864
Preop sagittal vertical axis (mm)	54.6 ± 59.3	44.9 ± 59.2	67.1 ± 57.6	0.059

IONM indicates intraoperative neurophysiological monitoring; Y:N, yes versus no.

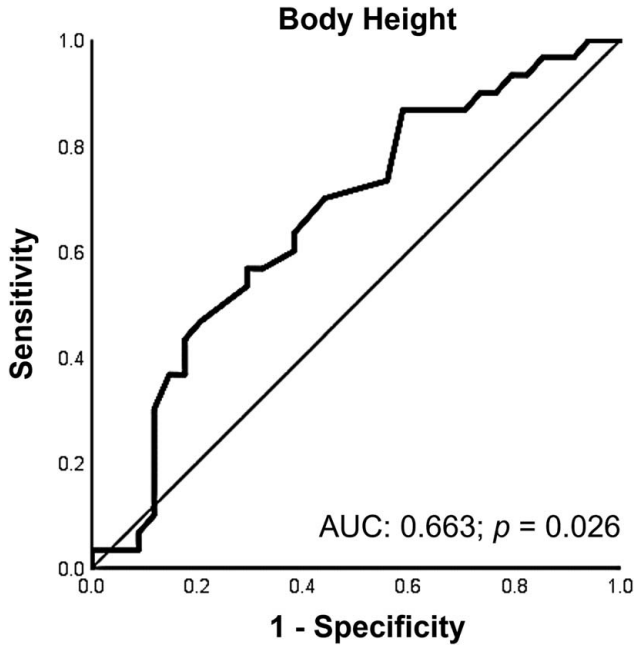

FIGURE 2. The receiver operating characteristic (ROC) curve analysis of body height for predicting absent baseline IONM signals in the cerebral palsy cohort. (IONM, intraoperative neurophysiological monitoring; AUC, area under the curve).

Risk Factors for Absent IONM Signals in Entire Neuromuscular Scoliosis Cohort

Logistic regression analysis identified several predictors for the absent baseline IONM signals in the lower extremities (Table 3). In the univariate model, CP was the strongest predictor [odds ratio (OR): 13.317, 95% CI: 4.290-41.337, $P < 0.001$], followed by preoperative incontinence (OR: 5.609, 95% CI: 1.572-20.011, $P = 0.008$), BW (OR: 0.927, 95% CI: 0.884-0.972, $P = 0.002$), and BH (OR: 0.949, 95% CI: 0.919-0.980, $P = 0.001$). BMI also showed a significant association (OR: 0.894, 95% CI: 0.799-1.000, $P = 0.049$). In the multivariate model, which included all variables with $P < 0.05$ from the univariate analysis, only CP retained statistical significance (OR: 9.615, 95% CI: 2.806-32.953, $P < 0.001$).

Risk Factors for Absent IONM Signals in CP Cohort

Given the dominant effect of CP, we focused on the CP cohort (n = 64), which comprised 53 patients classified as GMFCS level V, ten as level IV, and one as level III (Table 4). Seizure history was documented in 55 patients (85.9%), and gastrostomy tube (G-tube) dependency in 61 patients (95.3%). Fisher exact test revealed a significant association between seizure history and the absence of baseline IONM signals ($P = 0.049$); however, no significant association was found for G-tube dependency ($P = 0.452$).

To identify a clinically applicable cutoff for a continuous predictor, we performed ROC curve analysis, which demonstrated that BH was a significant predictor of absent baseline IONM signals in the CP cohort [area under the curve (AUC): 0.663; 95% CI: 0.528-0.797, $P = 0.026$], with an optimal cutoff of 128.5 cm (sensitivity: 86.7%; specificity: 41.2%) (Fig. 2).

When analyzing the CP cohort using logistic regression, univariate analysis revealed several significant predictors: GMFCS level V showed the strongest association (OR: 16.5, 95% CI: 1.978-137.683, $P = 0.010$), followed by BH < 128.5 cm (OR: 4.550, 95% CI: 1.296-15.978, $P = 0.018$) and ITB pump use (OR: 4.024, 95% CI: 1.147-14.124, $P = 0.030$) (Table 5). The multivariate model identified GMFCS level V (OR: 11.501, 95% CI: 1.311-100.966, $P = 0.028$) and BH < 128.5 cm (OR: 4.097, 95% CI: 1.038-16.185, $P = 0.044$) as independent predictors of

TABLE 5. Logistic Regression Analysis for Predicting Absent Baseline IONM Signals (Cerebral Palsy Cohort)

	Univariate model		Multivariate model	
Variable	**Odds ratio (95% CI)**	***P***	**Odds ratio (95% CI)**	***P***
Age at surgery (y)	0.915 (0.747-1.121)	0.393	—	—
Sex (male)	1.092 (0.404-2.952)	0.862	—	—
GMFCS level V (Y:N)	16.5 (1.962-138.754)	0.010	11.501 (1.298-101.903)	0.028
Preoperative Incontinence (Y:N)	2.462 (0.417-14.517)	0.320	—	—
G-tube dependent (Y:N)	2.357 (0.203-27.390)	0.493	—	—
Seizure history (Y:N)	4.870 (0.926-25.618)	0.062	—	–
ITB pump use (Y:N)	4.024 (1.142-14.180)	0.030	3.681 (0.919-14.739)	0.066
BMI (kg/m^2)	1.120 (0.932-1.346)	0.225	—	—
BW (kg)	0.970 (0.906-1.038)	0.376	—	—
BH (cm)	0.963 (0.928-0.998)	0.040	–	—
BH < 128.5 cm (Y:N)	4.550 (1.297-15.958)	0.018	4.097 (1.038-16.163)	0.044
BH < 50th percentile (Y:N)	2.300 (0.775-6.823)	0.133	—	—
Preop major Cobb angle (°)	1.002 (0.980-1.024)	0.871	—	—
Preop pelvic obliquity (°)	1.008 (0.970-1.047)	0.687	—	—
Preop T2-T12 kyphosis (°)	1.015 (0.990-1.042)	0.244	—	—
Preop lumbar lordosis (°)	1.008 (0.989-1.028)	0.393	—	—
Preop sagittal vertical axis (mm)	0.997 (0.988-1.006)	0.475	—	—

BH indicates body height; BMI, body mass index; BW, body weight; GMFCS, gross motor function classification system; IONM, intraoperative neurophysiological monitoring; ITB pump, intrathecal baclofen pump; preop, preoperative; Y:N, yes versus no.

TABLE 6. Absent IONM Baseline Signals in Pediatric Neuromuscular Scoliosis: A Literature Review

Author (y)	Patients number	NM diagnoses	CP proportion, %	IONM modalities	Absent IONM baseline signals (%)
Ashkenaze, 1993[9]	104	CP, DMD, Polio, SMA, CMT, and others	52.9	SCEP	28
Owen, 1994[23]	60 (idiopathic scoliosis), 47 (NM scoliosis)	CP, Others	66	SSEPs, MEPs	27% for SSEPs in NM scoliosis group
Padberg, 1996[10]	74	CP, MD, Polio, FA, CMT, SMA, others	39.2	SSEPs, NMEPs	15% for SSEPs and 14% for NMEPs
Noordeen, 1997[20]	99	DMD, SMA, others	NA	SSEP	2.1
DiCindio, 2003[12]	68	CP, others	57.4	SSEPs, TceMEPs	30% for SSEP and 10% for MEP in severe CP
Thuet, 2005[13]	4310 (total), 324 (NM scoliosis)	Various spinal conditions	NA	SSEPs, NMEPs	1.37% overall, 6.80% in NM scoliosis
Master, 2008[21]	7	Rett syndrome	0	SSEPs	0
Hammett, 2013[7]	109	CP, DMD, SMA, Rett syndrome, others	56.0	SSEPs, TceMEPs	12.1% of attempted cases
Aleem, 2015[11]	82	Various spinal cord pathologies	NA	SSEPs, DNEPs, MEPs	18
Pastorelli, 2015[8]	40	CNS and PNS diseases	79.3% in CNS Group	SSEPs, TceMEPs	6.8% in CNS group, 0% in PNS group
Dulfer, 2020[22]	30 (total), 15 (DMD)	DMD, AIS	0	SSEPs, TceMEPs	6.7% (SSEP) in DMD
Present study	115	CP, SMA, DMD, others	55.7	SSEPs, TceMEPs	33% of overall, 53.1% in CP

AIS indicates adolescent idiopathic scoliosis; CMT, Charcot-Marie-Tooth disease; CNS, central nervous system; CP, cerebral palsy; DMD, duchenne muscular dystrophy; DNEP, descending neurogenic evoked potential; IONM, intraoperative neurophysiological monitoring; MEPs, motor evoked potentials; NA, not available; NM, neuromuscular; NMEP, neurogenic motor evoked potentials; PNS, peripheral nervous system; SCEP, spinal cord evoked potentials; SMA, spinal muscular atrophy; TceMEP, transcranial MEPs.

absent baseline IONM signals.

Distribution of Growth Parameters in CP Cohort

Given the identified relationship between BH and absent IONM signals, we analyzed the distribution of anthropometric parameters within our CP cohort using specialized CP-specific growth standards to determine whether nutritional status might be a confounding factor.[17] Assessment utilizing these standards demonstrated predominantly normative growth patterns in our cohort (Supplemental Fig. 1, Supplemental Digital Content 1, http://links.lww.com/BPO/A964). BH measurements revealed no patients below the 5th percentile, with the majority distributed within the 25 to 75th percentile range. Similar patterns were observed for BW and BMI, with minimal occurrence below the 5th percentile.

DISCUSSION

The efficacy of IONM in NMS surgery remains controversial despite its widespread adoption. Our finding that one-third of NMS patients had absent baseline IONM signals at the onset of spinopelvic fusion surgery highlights a significant clinical challenge that impacts surgical planning and patient safety protocols. The disproportionate prevalence among CP patients, especially those with GMFCS level V and shorter stature, suggests the need for clinical teams to anticipate monitoring challenges and develop protocols for these high-risk patients.

Obtaining reliable baseline IONM signals during spine surgery for NMS varies based on monitoring method, diagnosis, and disease severity. Multiple monitoring modalities are often used to enhance success rates.[7] The literature and our study indicate higher success rates in obtaining baseline IONM signals for non-CP patients, particularly those with muscular dystrophy, spinal muscular atrophy, and Rett syndrome (Table 6).[8,20–22] This variability suggests that IONM strategies should be tailored to the specific underlying pathology instead of applying uniformly across all neuromuscular disorders.

CP presents challenges for obtaining reliable IONM signals. While previous studies reported 21.6% to 30% unsuccessful in CP populations, our cohort showed a markedly higher failure rate of 53.1%.[7,9,13,23] This difference likely reflects our inclusion of more severely affected patients, as 98.4% of our CP cohort were classified as GMFCS levels IV-V. By demonstrating a clear relationship between functional severity and monitoring challenges, our work extends previous research in this area. Importantly, such real-world data provides crucial information to inform preoperative planning for surgical teams managing severe CP patients.

BH <128.5 cm emerged as an independent predictor of absent baseline IONM signals in CP patients. Our anthropometric analysis using CP-specific growth charts demonstrated that patients predominantly exhibited typical growth patterns, with no patients falling below the 5th percentile for BH. This confirms that the increased risk of absent baseline IONM signals in CP patients with BH <128.5 cm is not from malnutrition or growth restriction.

This height threshold may be associated with practical monitoring limitations in smaller patients. In CP patients, underlying neurological factors may potentially interact with these physical size considerations, though the exact mechanisms require further investigation. While our multivariate analysis identified BH <128.5 cm as a significant predictor, this should be interpreted cautiously, given our sample size limitations. Further research is needed to validate these findings and develop appropriate monitoring equipment for smaller patients.

ITB pumps are frequently used to manage severe spasticity in CP patients unresponsive to oral medications or botulinum toxin injections.[24] While previous studies demonstrated that the ITB pump abolished the H-reflex but did not affect the MEP, EMG, or cutaneous silent period in spinal cord injury cases, its impact on IONM signals in CP remains unclear.[25,26] Our univariate analysis identified ITB pump use as a significant predictor of absent IONM signals, though this relationship did not persist in multivariate analysis. Whether this reflects direct pharmacological effects or correlates with disease severity remains uncertain, as most ITB patients (16/17) were GMFCS level V. Future research should explore if temporary baclofen adjustments before surgery could improve IONM signal acquisition.

The indication of IONM for patients with severe neurological impairment remains a topic of debate.[7] In our study, IONM enabled early detection of issues such as improper limb positioning and suboptimal pedicle screws placement. Moreover, IONM's ability to monitor spinal cord function and perfusion helps the surgical and anesthesia team maintain ideal mean arterial pressure (MAP) during anesthesia. Even in patients without lower limb signals, monitoring upper limb function helps protect remaining upper extremity function, which is beneficial for patients with limited overall functionality. Future research should investigate whether advanced technologies, such as new intraoperative imaging, navigation systems, or robotic-assisted surgery, could enhance or potentially replace IONM in this population, while also evaluating their cost-effectiveness and clinical utility.

Our study had several limitations. First, this is a retrospective single-center report, which may have bias and limited generalizability. Second, our cohort consisted of predominantly nonambulatory patients with severe motor impairments, making routine neurological assessment challenging and potentially underestimating iatrogenic deficits. We also did not address the impact of IONM unavailability on surgical outcomes and long-term neurological function. Third, we did not comprehensively classify CP patients by type and distribution, which could influence IONM signal acquisition. Fourth, although seizure history demonstrated a statistically significant association with absent baseline IONM signals, it was not independently predictive in logistic regression analysis, possibly due to limited sample size. Moreover, our retrospective design precluded detailed analysis of seizure severity, medication types, and dosing regimens. Future larger prospective studies should systematically examine these seizure-related variables to clarify their potential role in baseline IONM signal acquisition.

However, our strengths included enrolling a substantial sample size from a specialized population of pediatric NMS patients, maintaining homogeneity in surgical approach and fusion levels, comprehensively assessing predictive factors, and specifically focusing on severe CP patients. This approach provided valuable insights into specific factors influencing the success rate of obtaining baseline signals during spine surgery in this challenging patient group.

CONCLUSIONS

This study highlights the challenges of obtaining reliable baseline IONM signals in pediatric NMS surgery, particularly in severe CP patients. We identified that the underlying diagnosis, especially CP, significantly affects signal acquisition, with functional severity (GMFCS level V) and shorter stature (BH <128.5 cm) emerging as key predictors of monitoring challenges.

We recommend a risk-stratified approach to intraoperative neuromonitoring in this population. Surgical teams should proactively identify high-risk patients preoperatively using CP diagnosis, GMFCS level, and body height. This information should be incorporated into surgical planning and informed family counseling. For cases in which reliable signals are unlikely, adjunctive strategies such as intraoperative navigation, O-arm imaging, or robotic assistance may be considered to maintain procedural safety. Surgical teams should consider establishing predefined protocols for managing cases with anticipated IONM limitations, rather than relying solely on intraoperative decision-making. Future research should focus on alternative monitoring technologies and evaluate their impact on intraoperative safety and long-term outcomes in this vulnerable population.

ACKNOWLEDGMENTS

The authors thank Miao-Pei Su for this study's initial conceptualization and consultation.

REFERENCES

1. Angelliaume A, Alhada TL, Parent HF, et al. Intraoperative neurophysiological monitoring in scoliosis surgery: literature review of the last 10 years. *Eur Spine J*. 2023;32:3072–3076.
2. Halsey MF, Myung KS, Ghag A, et al. Neurophysiological monitoring of spinal cord function during spinal deformity surgery: 2020 SRS neuromonitoring information statement. *Spine Deform*. 2020;8:591–596.
3. Kerdoncuff A, Henry P, Compagnon R, et al. Feasibility, safety and reliability of surgeon-directed transcranial motor evoked potentials monitoring in scoliosis surgery. *Children*. 2023;10:1560.
4. Murphy RF, Mooney JF 3rd. Current concepts in neuromuscular scoliosis. *Curr Rev Musculoskelet Med*. 2019;12:220–227.
5. Rumalla K, Yarbrough CK, Pugely AJ, et al. Spinal fusion for pediatric neuromuscular scoliosis: national trends, complications, and in-hospital outcomes. *J Neurosurg Spine*. 2016;25:500–508.
6. Floccari L, Murphy J, Glotzbecker M, et al. Absent baseline intraoperative neuromonitoring signals part 2: neuromuscular scoliosis: current concept review. *J Pediatr Orthop Soc North Am*. 2022;4:1–6.
7. Hammett TC, Boreham B, Quraishi NA, et al. Intraoperative spinal cord monitoring during the surgical correction of scoliosis due to cerebral palsy and other neuromuscular disorders. *Eur Spine J*. 2013; 22 (suppl 1):S38–S41.

8. Pastorelli F, Di Silvestre M, Vommaro F, et al. Intraoperative monitoring of somatosensory (SSEPs) and transcranial electric motor-evoked potentials (tce-MEPs) during surgical correction of neuromuscular scoliosis in patients with central or peripheral nervous system diseases. *Eur Spine J*. 2015;24(suppl 7):931–936.
9. Ashkenaze D, Mudiyam R, Boachie-Adjei O, et al. Efficacy of spinal cord monitoring in neuromuscular scoliosis. *Spine*. 1993;18: 1627–1633.
10. Padberg AM, Russo MH, Lenke LG, et al. Validity and reliability of spinal cord monitoring in neuromuscular spinal deformity surgery. *J Spinal Disord*. 1996;9:150–158.
11. Aleem AW, Thuet ED, Padberg AM, et al. Spinal cord monitoring data in pediatric spinal deformity patients with spinal cord pathology. *Spine Deform*. 2015;3:88–94.
12. Thuet ED, Padberg AM, Raynor BL, et al. Increased risk of postoperative neurologic deficit for spinal surgery patients with unobtainable intraoperative evoked potential data. *Spine*. 2005;30: 2094–2103.
13. DiCindio S, Theroux M, Shah S, et al. Multimodality monitoring of transcranial electric motor and somatosensory-evoked potentials during surgical correction of spinal deformity in patients with cerebral palsy and other neuromuscular disorders. *Spine*. 2003;28:1851–1855.
14. Addai D, Zarkos J, Bowey AJ. Current concepts in the diagnosis and management of adolescent idiopathic scoliosis. *Childs Nerv Syst*. 2020;36:1111–1119.
15. Kisacik P, Bazancir-Apaydin Z, Apaydin H. Intraobserver and interobserver reliability of pelvic obliquity measurement methods in patients with idiopathic scoliosis. *J Pediatr Orthop*. 2024;44:225–231.
16. Qi H, Zhao Z, Gao X, et al Normal spinopelvic parameters and correlation analysis in 217 asymptomatic children. *Eur Spine J*. 2024; 33:2569–2576.
17. Brooks J, Day S, Shavelle R, et al. Low weight, morbidity, and mortality in children with cerebral palsy: new clinical growth charts. *Pediatrics*. 2011;128:e299–e307.
18. Buhl LK, Bastos AB, Pollard RJ, et al. Neurophysiologic intraoperative monitoring for spine surgery: a practical guide from past to present. *J Intensive Care Med*. 2021;36:1237–1249.
19. Acharya S, Palukuri N, Gupta P, et al. Transcranial motor evoked potentials during spinal deformity corrections-safety, efficacy, limitations, and the role of a checklist. *Front Surg*. 2017;4:8.
20. Noordeen MH, Lee J, Gibbons CE, et al. Spinal cord monitoring in operations for neuromuscular scoliosis. *J Bone Joint Surg Br*. 1997; 79:53–57.
21. Master DL, Thompson GH, Poe-Kochert C, et al. Spinal cord monitoring for scoliosis surgery in Rett syndrome: can these patients be accurately monitored? *J Pediatr Orthop*. 2008;28:342–346.
22. Dulfer SE, Lange F, Wapstra FH, et al. Intraoperative neurophysiological monitoring during scoliosis surgery in patients with Duchenne muscular dystrophy. *Eur Spine J*. 2020;29:2029–2035.
23. Owen JH, Lenke LG, Danto J. Spinal cord monitoring in patients undergoing surgery for neuromuscular scoliosis. *Spine (Phila Pa 1976)*. 1994;19:2371–2372.
24. Masrour M, Zare A, Presedo A, et al. Intrathecal baclofen efficacy for managing motor function and spasticity severity in patients with cerebral palsy: a systematic review and meta-analysis. *BMC Neurol*. 2024;24:143.
25. Stetkarova I, Kofler M. Differential effect of baclofen on cortical and spinal inhibitory circuits. *Clin Neurophysiol*. 2013;124:339–345.
26. Bowden M, Stokic DS. Clinical and neurophysiologic assessment of strength and spasticity during intrathecal baclofen titration in incomplete spinal cord injury: single-subject design. *J Spinal Cord Med*. 2009;32:183–190.