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THE NEUROLOGIC DEFICIT RISK OF THREE DIFFERENT KINDS OF SPINAL OSTEOTOMIES AND PERIOPERATIVE MANAGEMENT

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ABSTRACT

Objective: We reported intraoperative and early postoperative new neurologic deficits and acute management of this problem in patients who underwent correction surgery with spinal osteotomies [posterior vertebral column resection (PVCR), pedicle subtraction osteotomy (PSO), and Smith-Peterson osteotomy (SPO)] for complex spinal deformity, and we secondarily compared osteotomies in terms of observing neurologic deficits.

Materials and Methods: Between 2017 and 2021 were retrospectively reviewed from our medical records. A total 83 patients who underwent correction of severe spine deformity with various spinal osteotomies were included in the study.

Results: In total, 3 patients with SPO (2 perioperative and 1 postoperative) and 2 patients with PVCR (both postoperative) had a new neurologic deficit. From these 5 patients, 3 of them had adult deformity and 2 had pediatric congenital kyphoscoliosis. Three patients had no intraoperative signal loss and were normal postoperatively, but they gradually lost motor power in their legs within 6 h of surgery.

Conclusion: We did not observe any neurologic event perioperatively and postoperatively in patients who underwent PSO, and the neurologic complication rate was slightly higher in patients who underwent PVCR. Intraoperative neuromonitoring should be used in every spinal deformity correction surgery even if false-negative results can occur. Perioperative signal loss should be taken seriously, and a necessary management protocol should start immediately. The last check of neuromonitoring should be done before waking up, and a routine neurologic exam must be carried out during hospitalization time. Because of postoperative neurologic deficits can occur.

Keywords: Spine osteotomy, neurologic deficits, scoliosis, khyposis

INTRODUCTION

The rate of new neurologic deficits associated with spine surgery was reported as 1% based on data from the Scoliosis Research Society on 108,419 adult patients with a primary diagnosis of degenerative disease[1]. A new neurologic deficit is one of a devastating complications of severe spine deformity surgery. This may occur intraoperatively or in the early postoperative period[2]. Neuromonitoring is an essential tool used during spine deformity surgery to detect any potential neurologic deficit early[3]. Intraoperative signal loss may occur unilaterally or bilaterally. In addition, false-negative and false-positive instances may occur during surgery. The signal loss could be due to a direct mechanical injury to the cord or nerve root, vascular compromise, or inadequate decompression of the dura or nerve roots after osteotomies[4]. Vale et al.[5] demonstrated that patients with an acute neurologic deficit or injury could improve when mean arterial pressure (MAP) is maintained above 85 mmHg. Unfortunately, given the paucity of data on delayed neurologic deficits, there are no existing guidelines for the treatment of this relatively rare phenomenon[5]. Wang et al.[6] reported that patients with higher ratios of coronal and sagittal angulation were at risk of spinal cord monitoring events and new neurologic deficits. The authors evaluated the patients who only underwent posterior vertebral column resection (PVCR) for deformity correction[6]. Alternatively, Trobisch et al.[7] reported the outcomes of pedicle subtraction osteotomy (PSO) for the deformity correction without neuromonitoring, and the authors discovered similar neurologic complications compared to previous studies. We hypothesized that intraoperative neuromonitoring, which should be performed during complex spinal deformities that require spinal osteotomies, could detect similar signal losses, independent of the type of corrective spinal osteotomy. Therefore, we reported intraoperative and early postoperative new neurologic deficits, as well as acute management of this problem in patients who underwent

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correction surgery with spinal osteotomies for complex spinal deformity, and we secondarily compared osteotomies in terms of observing neurologic deficits.

MATERIALS AND METHODS

Study Population

This retrospective study was approved by the İstinye University Ethical Review Board (3/2022.K-34) and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all the patients. Data of patients who underwent corrective spinal surgery between 2017 and 2021 were retrospectively reviewed from our medical records. Patients who underwent correction of spine deformity with various spinal osteotomies were included in the study. Patients with preoperative neurological deficits and those who underwent revision surgeries were excluded.

Surgical Technique and Neuromonitoring

All surgeries were performed by a spine surgeon with over 25 years of experience and the senior author of this study. PVCR, PSO, the and Smith-Peterson osteotomy (SPO) were performed if needed. No preoperative halo traction or intraoperative traction was used. Intraoperative neuromonitoring was performed with transcranial motor-evoked potentials (MEP), somatosensory-evoked potentials (SSEP), free-run electromyography (EMG), and pedicle screw stimulation EMG under total intravenous anesthesia. No intraoperative wake-up test was used routinely. All cases followed MEP and SSEP neuromonitoring modalities during the operation. Immediate action was taken for those who lost signals intraoperatively with normalization of blood pressure (systolic pressure to 100 mmHg and above), normalization of PO2 (100), removal of rod, loosening of correction, and further decompression of dura and nerve roots. The signals returned to normal within 15 min after necessary interventions in all patients. After the normalization of the MEP and SSEP signals, re-correction of the deformity was performed uneventfully.

Statistical Analysis

Data analysis was performed using SPSS (IBM, Armonk, NY, USA). Chi-square test was used to compare neurologic deficit risk for osteotomy groups. A p value of <0.05 was considered statistically significant.

RESULTS

This study included 83 patients (52 female, 31 male) with a mean age of 32 years (range; 6-53 years old) (29 pediatric and 54 adult patients). The mean coronal Cobb's angle was 98° (range; 60-100°), and the mean sagittal Cobb's angle was 78° (range; 55-95°). PVCR was performed in 35 patients, PSO was performed in 20 patients, and SPO was performed in 28 patients for rigid deformity correction (Table 1). We encountered intraoperative signal loss in two patients on one side after the affected side had been instrumented and corrected. Immediate action was performed in these patients, as previously described (Figure 1). Three patients had no intraoperative signal loss and were normal postoperatively, but they gradually lost motor power in their legs within 6 h of surgery. These patients had postoperative bilateral neurologic deficits, so we started to apply a high dose of steroid protocol. Immediate computed tomography and magnetic resonance imaging were also conducted. These patients underwent immediate exploratory surgery, and we applied necessary decompression of the dura and nerve roots. In two of the three patients, there was no evidence of hardware malposition, hematoma, or obvious cord compression. All patients hgb levels were above 10 g/dL. In total, three patients with SPO (two perioperative and one postoperative) and two patients with PVCR (both postoperative), had a new neurologic deficit (Table 2). Four of these patients recovered completely within six months postoperatively, and one patient remained Frankel grade C at two years postoperatively. Of these five patients, three had adult spinal deformity, and two had pediatric congenital kyphoscoliosis. There was no statistically significant difference between the osteotomy groups for neurological deficit risk (p>0.05).

DISCUSSION

The most important finding of this study was observing two perioperative signal losses during spine deformity surgery; however, we also observed three early postoperative neurologic deficits despite having normal signals during surgery. Intraoperative signal loss and postoperative early and late new neurological deficits are the most significant complications of spine deformity surgery. Therefore, intraoperative neuromonitoring is used as a tool to recognize possible new neurologic deficits. Based on several studies, neuromonitoring is significantly effective in reducing the incidence of new neurologic deficits[3,8,9]. However, some other studies have also reported false-negative results[10,11]. The MAP has a significant effect on the SSEP results. If MAP drops below 60 mmHg, significant changes in SSEP may occur[12,13]. Mentioned

Table 1. Demographic variables

Gender	**52 female, 31 male**
Age	32 y (6y-53y)
Coronal Cobb	98° (60-100°)
Sagital Cobb	78° (55-95°)
Pediatric patients	29 patients
Adult patients	54 patients
SPO	28 patients
PSO	20 patients
PVCR	35 patients

PVCR: Posterior vertebral column resection, PSO: Pedicle subtraction osteotomy, SPO: Smith-Peterson osteotomy

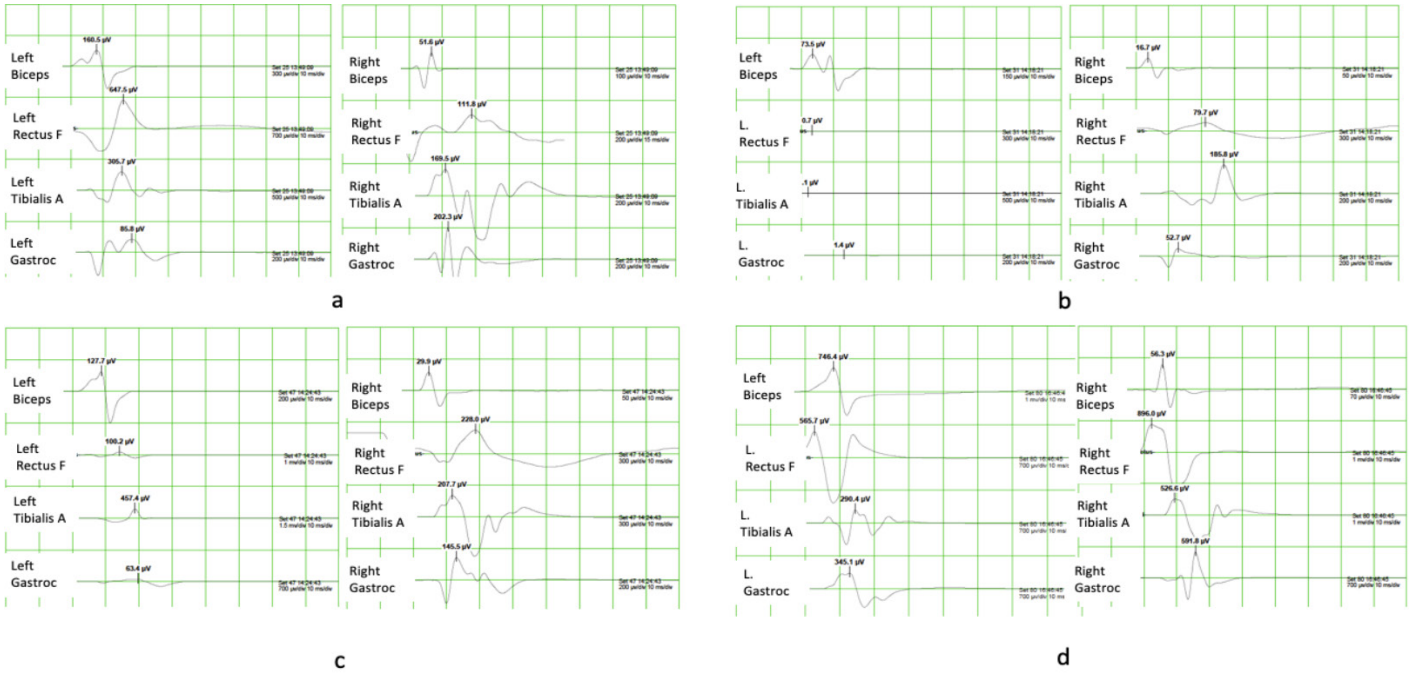

Figure 1. **a)** After a pedicle screw, **b)** osteotomy and correction, left lower extremity signal loss, **c)** after the standard protocol, signal recovery, **d)** final

Table 2. Neurologic deficit

Neurologic deficit	SPO	PSO	PVCR
Intraoperative	2	0	0
Postoperative	1	0	2

PVCR: Posterior vertebral column resection, PSO: Pedicle subtraction osteotomy, SPO: Smith-Peterson osteotomy

that high lability in MAP usually results in false-positive results[13]. The authors did not observe any false-negative results, but they remarked that postoperative neurologic status could not be predicted by neuromonitoring changes[13]. Raynor et al.[10] reported 0.36% (45 patients) false-negative outcomes out of 12,375 spine surgeries. Among this group, only two had permanent new neurologic deficits. In this study, we observed three postoperative neurologic deficits out of 83 patients (3.6%) that could not be detected intraoperatively, which can be considered a false-negative result. Among the three patients, only one had a permanent neurologic deficit (1.2%). According to our experience, we observed two perioperative signal losses during spinal correction surgery managed by a standard protocol as follows:

1. We increased the systolic blood pressure above 100 mmHg.
2. We increased the PO_2 to 100%.
3. We started to replace the estimated blood loss.
4. We removed the rod and reduced the correction.
5. We checked all implants using anterior-posterior and lateral views with an image intensifier.
6. We checked with a second look for nerve roots and dura, and if needed, enlarged the decompression.
7. If the signal returned to normal within 30 min, we replaced the rod and reperformed the correction.
8. We checked neuromonitoring before wound closure.
9. If the signals did not return, we started the wake-up test.

Another devastating problem after deformity correction surgery is an early postoperative neurologic deficit despite stable intraoperative monitoring and postoperative normal neuro exam. This may occur within hours or even days after surgery[14]. The main reason for this type of event is usually delayed ischemic injury of the spinal cord or compression of the cord because of inadequate decompression, specifically after osteotomies or even postoperative hematoma development[14]. According to our results, we had three new neurologic deficits within 6 h postoperatively. All patients underwent computed tomography and magnetic resonance imaging to rule out the malposition of screws, inadequate decompression, or postoperative hematoma. All patients received a high dose of steroid protocol for 24 h after the first detection of neurologic decline with full monitorization of blood pressure (systolic above 100 mmHg), setting oxygenation PO_2 to 100%, and setting hemoglobin level above 10 mg/dL. The same management was also been recommended by Auerbach et al.[14,15]. After this systematic approach, all patients underwent an immediate exploration of the surgical field. All necessary spinal cord decompression was performed. Two of them returned to normal within three months. After two years, one patient improved neurologically, but is still in Frankel grade C. Our study also compared the neurologic deficits in terms of the type of corrective osteotomy performed during surgery. We encountered perioperative signal loss in two patients who underwent SP osteotomy and three with postoperative neurologic deficits (one SP osteotomy and two PVCR). We did not observe any neurologic event perioperatively or postoperatively in patients who underwent

PSO, and the neurologic complication rate was slightly higher in those who underwent PVCR. Wang et al.[6] evaluated 202 consecutive pediatric and adult spine deformities who underwent PVCR. The authors observed 140 signal losses without neurologic deficits and 36 (17.8%) true-positive results, and the overall neurologic deficit ratio was 8/202 (4%)[6]. Daubs et al.[16] reported a 6% neurologic deficit in 84 patients who underwent PSO at a tertiary spine center. In their systematic review and meta-analysis, Liu et al.[17] discovered a higher risk of permanent neurologic deficit in SPO than in PSO (6% vs 5%). Our overall neurologic deficit rate was 3.6%, and our overall permanent neurologic deficit rate was 1.2%. The lower number of permanent neurologic deficits can be explained by the use of intraoperative neuromonitoring and early management of the signal losses.

Study Limitations

The main limitation of our study is that it is a retrospective analysis of a relatively small number of a heterogeneous patient population that contains both pediatric and adult spine deformities. However, we performed a retrospective analysis of a prospectively monitored patient group treated by a single surgeon using the same approaches. Besides, our osteotomy groups had a relatively similar patient number, allowing us to compare neuromonitoring results and neurologic deficits. This study focused on intra and postoperative neurological deficit risk and manegement for spinal osteotomies. Therefore type and level of curvature, curve flexibility, correction rate, comorbidities, intraoperative blood loss, and operation time were ignored. The main strength of our study is that it is the first research to report neuromonitoring results and the management of neurologic events in patients with SPO, PSO, and PVCR.

CONCLUSION

We did not observe any neurological event perioperatively and postoperatively in patients who underwent PSO, and the neurologic complication rate was slightly higher in patients who underwent PVCR. According to the results acquired from this study, intraoperative neuromonitoring should be used in every spinal deformity correction surgery, even if false-negative results occur. Perioperative signal loss should be taken seriously, and the necessary management protocol should be started immediately. The last check should be performed before waking up, and a routine neurologic exam must be conducted during hospitalization.

Ethics

Ethics Committee Approval: This retrospective study was approved by the İstinye University Ethical Review Board (3/2022.K-34, date: 06.09.2014) and conducted in accordance with the Declaration of Helsinki.

Informed Consent: Written informed consent was obtained from all the patients.

Peer-review: Externally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: G.K.K., Ç.Ö., U.A., Concept: G.K.K., K.T., Y.Ç., Ç.Ö., U.A., Design: G.K.K., K.T., Y.Ç., Ç.Ö., U.A., Data Collection or Processing: G.K.K., K.T., Y.Ç., Analysis or Interpretation: G.K.K., Y.Ç., Ç.Ö., U.A., Literature Search: G.K.K., Y.Ç., Writing: G.K.K., K.T., Y.Ç.

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