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DEFORMITY

An Analysis of the Incidence and Outcomes of Major *Versus* Minor Neurological Decline After Complex Adult Spinal Deformity Surgery

A Subanalysis of Scoli-RISK-1 Study

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Study Design. A subanalysis from a prospective, multicenter, international cohort study in 15 sites (Scoli-RISK-1).

Objective. To report detailed information regarding the severity of neurological decline related to complex adult spine deformity (ASD) surgery and to examine outcomes based on severity.

Summary of Background Data. Postoperative neurological decline after ASD surgeries can occur due to nerve root(s) or spinal cord dysfunction. The impact of decline and the pattern of recovery may be related to the anatomic location and the severity of the injury.

Methods. An investigation of 272 prospectively enrolled complex ASD surgical patients with neurological status measured by American Spinal Injury Association Lower Extremity Motor Scores (LEMS) was undertaken. Postoperative neurological decline was categorized into ''major'' (≥5 points loss) *versus* ''minor'' (<5 points loss) deficits. Timing and extent of recovery in LEMS were investigated for each group.

Results. Among the 265 patients with LEMS available at discharge, 61 patients (23%) had neurological decline, with 20 (33%) experiencing major decline. Of note, 90% of the patients with major decline had deficits in three or more myotomes. Full recovery was seen in 24% at 6 weeks and increased to 65% at 6 months. However, 34% continued to experience some neurological decline at 24 months, with 6% demonstrating no improvement. Of 41 patients (67%) with minor decline, 73% had deficits in one or two myotomes. Full recovery was seen in 49% at 6 weeks and increased to 70% at 6 months. Of note, 26% had persistence of some neurological deficit at 24 months, with 18% demonstrating no recovery.

Conclusion. In patients undergoing complex ASD correction, a rate of postoperative neurological decline of 23% was noted with 33% of these being ''major.'' Although most patients showed substantial recovery by 6 months, approximately one-third continued to experience neurological dysfunction.

Key words: acute neurologic complications, adult spinal deformity, American Spinal Injury Association neurologic exam, incidence, lower extremity motor score, outcome, recovery, severity, spinal osteotomy, time course.

Level of Evidence: 2

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The prevalence of adult spinal deformity (ASD) in the general population has been reported as between 2% and 32%.[1–3] As techniques and technologies in spine surgery have evolved, surgical correction of severe complex ASD has become achievable by the use of pedicle screw

fixation and complex spinal osteotomies. One of the most devastating perioperative complications is a deterioration of neurological function.[4] The Scoli-RISK-1 study was an international multicenter prospective cohort study designed and performed to collect data on neurologic complications associated with surgical correction of complex ASD.[5]

Despite the fact that there have been many studies discussing the incidence of postoperative neurological deficits after spinal deformity surgery, very few have focused on an objective, quantitative assessment of the severity, recovery rates, and time course. It is imperative for surgeons to understand the natural history and prognosis of these surgical complications to counsel patients and to plan postoperative management including medical and physical therapies. Furthermore, the etiologies of these neurologic events are not uniform. Spinal cord and/or nerve roots can be impaired to various extents at various locations. Therefore, neurological deficits associated with ASD surgery can range from solitary radiculopathy to complete paraplegia. All of these have different recovery potentials and prognosis, and can show various reactions to treatment. Our hypothesis was that the rate, extent, and time course of recovery of these neurological deficits might vary depending on their severity. The objective of the present investigation is to report detailed information about the severity of neurological deficits related to complex ASD surgery and to identify outcomes based on their severity.

MATERIALS AND METHODS

Patients

Data from the Scoli-RISK-1 study were used for the present investigation.[5] Key inclusion criteria were patients between 18 and 80 years of age and having a diagnosis of complex ASD with an apex of the major deformity between C7 and L2 inclusive. To focus on high-risk events, we enrolled patients based on the procedure performed. For this study, the procedure was defined as complex ASD surgery when one or more of the following criteria were met: corrective surgeries for curvatures with a major Cobb angle of 80° or more in the coronal and/or sagittal plane; corrective osteotomies for congenital spinal deformity; revision of a previous spinal deformity surgery requiring corrective osteotomies; three-column osteotomy (*i.e.*, pedicle subtraction osteotomy [PSO], vertebral column resection); reconstruction for myelopathy due to spinal deformity; or deformity reconstruction with concomitant spinal cord decompression for ossification of the ligamentum flavum or ossification of the posterior longitudinal ligament. Patients with a history of substance dependency, psychosocial disturbance, active malignancy, active bacterial infection, recent history of significant spinal trauma/malignancy, complete long-term paraplegia, pregnancy, prisoners, and institutionalized individuals were excluded.

Nine spinal deformity centers in North America, three in Europe, and three in Asia participated. The respective ethical committees or institutional review boards at all participating sites granted study approval. All patients provided informed consent before enrollment. Enrollment of 272 consecutive patients was completed from September 2011 to October 2012 by 44 surgeons. Surgical approach, instrumentation, corrective maneuvers, and use of neuromonitoring (motor-evoked potentials [MEP], somatosensory-evoked potentials, and/or electromyography) were at the discretion of the operating surgeon.

Measurements

An American Spinal Injury Association (ASIA) neurologic examination was performed by an ASIA-certified examiner within 6 weeks before surgery, at hospital discharge, and at 6 weeks, 6 months, and 24 months after surgery. Muscle strength was rated on a scale of 0 (no motor function) to 5 (full function) for each of five lower extremity key muscle groups: hip flexors (L2), knee extensors (L3), ankle/toe dorsiflexors (L4), great toe extensors (L5), and plantar flexors (S1). The lower extremity motor score (LEMS) evaluates motor function as a sum of the five scores on each side with a maximum of 50 points (25 points per side). It has been demonstrated to be correlated to ambulatory function as measured with gait analysis in patients with incomplete spinal cord injury.[6] Neurological decline was defined as loss of LEMS at discharge in comparison to preoperative (baseline) status. Declines observed were divided into two groups; ''major'' and ''minor'' decline. In the present study, we defined ''major'' decline as a LEMS loss of 5 points or more and ''minor'' decline as a loss of less than 5 points because a 5-point change in LEMS has been reported to be the minimal clinically important difference in spinal cord injury patients.[7] Neurological recovery was defined as any recovery of LEMS points at any time point through the follow-up period in comparison to LEMS at discharge. Recovery was categorized into three groups: full (back to baseline status or better), partial, and no recovery. Some of the patients lacked LEMS at long-term follow-up, but full recovery at both 6 weeks and 6 months was assumed to be sustained for the rest of the follow-up period. For comparisons between the groups, Wilcoxon rank sum tests were used for continuous variables and the chi-square test or Fisher exact test was used for categorical variables. The statistical analysis was performed using the software SAS version 9.4 (SAS Institute Inc., Cary, NC), and P values less than 0.05 were considered statistically significant.

RESULTS

Among the 272 patients enrolled in the Scoli-RISK-1 study, preoperative LEMS and discharge LEMS were available for 265 patients. These patients were included in the present investigation. There were 180 women (68%) and 85 men (32%). Mean age was 56.8 years old (standard deviation: 15.4). The most common indication for inclusion in the present study was three-column osteotomy (n = 201, 76%), followed by revision ASD surgery requiring osteotomy (n = 161, 61%), major Cobb angle of 80° or higher (n = 77, 29%), congenital deformity (n = 12, 5%), deformity-related

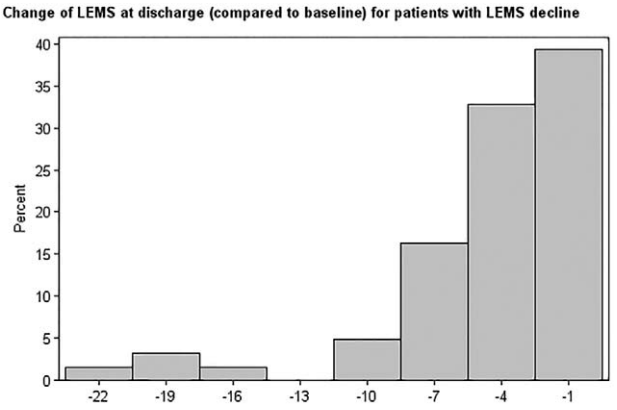

Figure 1. Histogram of decline of American Spinal Injury Association (ASIA) lower extremity motor score (LEMS) at discharge compared to baseline.

myelopathy (n = 12, 5%), and ossification of the ligamentum flavum/ossification of the posterior longitudinal ligament (n = 5, 2%). A total of 199 patients (75%) had no neurological deficits (LEMS = 50) preoperatively, whereas 66 patients (25%) had preoperative deficits (LEMS < 50). The median number of days which elapsed between surgery and discharge was 9.0 days (interquartile range: 7.0–16.0). Neuromonitoring was done for 259 of the 265 patients (98%).

In comparison to preoperative status, 61 patients (23%) showed a decline in LEMS at discharge. Among them, major neurological decline (≥5 LEMS points loss) occurred in 20 patients (33%), whereas 41 (67%) experienced a minor decline (<5 points). A histogram of severity of neurological decline is presented in Figure 1. The worst postoperative decline at discharge among the current cohort was a 22-point loss of LEMS. A comparative table of demographic data between major and minor decline groups is shown in Table 1. There was no significant difference in the demographics between the two groups. Eighteen out of 20 patients with major decline (90%) had dysfunction in three or more myotomes, whereas 30 of 41 patients with minor decline (73%) had deficits in one or two myotomes.

Because of the incomplete follow-up, LEMS was recorded only in 58, 59, and 45 patients at 6 weeks, 6 months, and 24 months, respectively, but full recovery at both 6 weeks and 6 months was assumed to be sustained for the rest of the follow-up period. Full recovery was observed in 41% at 6 weeks, increasing to 68% at 6 months and 71% at 24 months, leaving 28% with some extent of permanent decline (16 of 265 patients included, 6%). No recovery was seen in 14% at 24 months (8 of 265 patients included, 3%).

TABLE 1. Demographics of the Patients With Major *Versus* Minor American Spinal Injury Association Lower Extremity Motor Score Decline at Discharge

	Size of Decline in LEMS Score at Discharge		
	Major Decline (≥5 Points) n = 20	**Minor Decline (<5 Points) n = 41**	***P***
Age, median (IQR)	61.5 (56.0; 69.5)	63.0 (57.0; 70.0)	0.424
Female	11 (55%)	29 (71%)	0.225
Race			
White or Caucasian	18 (90%)	38 (93%)	
Black or African-American	1 (5%)	0 (0%)	
East Asian	1 (5%)	3 (7%)	
Diagnosis (inclusion criteria)			
Three-column osteotomy	17 (85%)	36 (88%)	>0.999
Revision surgery	14 (70%)	29 (71%)	0.953
Cobb ≥80°	6 (30%)	11 (27%)	0.795
Congenital deformity	1 (5%)	1 (2%)	
Deformity-related myelopathy	0 (0%)	2 (5%)	
OPLL/OLF	0 (0%)	1 (2%)	
Smoker	1 (5%)	4 (10%)	
Preoperative neurological deficits	8 (40%)	9 (22%)	0.140
Surgical approach			
Posterior only	15 (75%)	33 (80%)	0.741
Anterior + posterior approach	5 (25%)	8 (20%)	
Number of surgical stages			
Single-stage	17 (85%)	32 (78%)	0.734
Multistage	3 (15%)	9 (22%)	

IQR indicates interquartile range; OLF, ossification of ligamentum flavum; OPLL, ossification of posterior longitudinal ligament.

Changes in the average LEMS in the minor and major decline groups over time are presented in Figure 2. For 20 patients with a major decline of 5 or more LEMS points loss at discharge, full recovery was seen in four patients (24%) at 6 weeks, increasing to 65% at 6 months and 67% at 24 months, with the rest remaining with some decline. Four patients (24%) showed no recovery at 6 weeks, but only one patient showed no recovery at 6 months (5%). In contrast, 20 of 41 patients (49%) with a minor decline of less than 5 LEMS points loss at discharge showed full recovery at 6 weeks, increasing to 70% at 6 months and 74% at 24 months, with some decline persisting in the rest. No recovery was observed in 18 of 41 patients (44%) at 6 weeks, with the rate decreasing to 20% at 6 months and 18% at 24 months. The time course and extent of neurological recovery in the prospective cohort are summarized in Table 2. Changes in MEP signals were observed in 4 out of 15 patients (27%) for whom MEP was monitored in the major decline group, whereas they were observed in 5 out of 39 patients (13%) in the minor decline group. Actions were taken accordingly, including ensuring blood pressure, checking anesthesia status, and confirming and/or adding decompression.

A representative case is described in Figure 3(A–F).

DISCUSSION

The present subanalysis of the Scoli-RISK-1 study provided three major findings. First, among those who experienced postoperative neurological decline after complex ASD surgery, one third experienced a major decline ($\geq$5 LEMS points), whereas the remainder had a minor decline ($<$5 points). Second, the majority of recovery occurred in the first 6 months, and one third of the postoperative neurological deficits showed some persistence long term. Third, although a quicker full recovery was seen in the patients with a minor decline than in those with a major decline, patients with minor deficits showed full recovery less frequently than those with major deficits.

The etiologies of neurologic decline in ASD surgery are diverse. The spinal cord is at risk during decompression maneuvers particularly when cord compression or dysfunction is preexistent due to spinal deformity or ligamentous ossifications. Placement of spinal instruments can cause encroachment of the spinal canal and damage the cord. More importantly, angular deformity correction can lead to impingement of bony structure into the cord or stretch of the anterior spinal artery resulting in vascular injury,[8] whereas over-shortening of the spinal column is also known to be associated with decreased spinal cord blood flow.[10] In

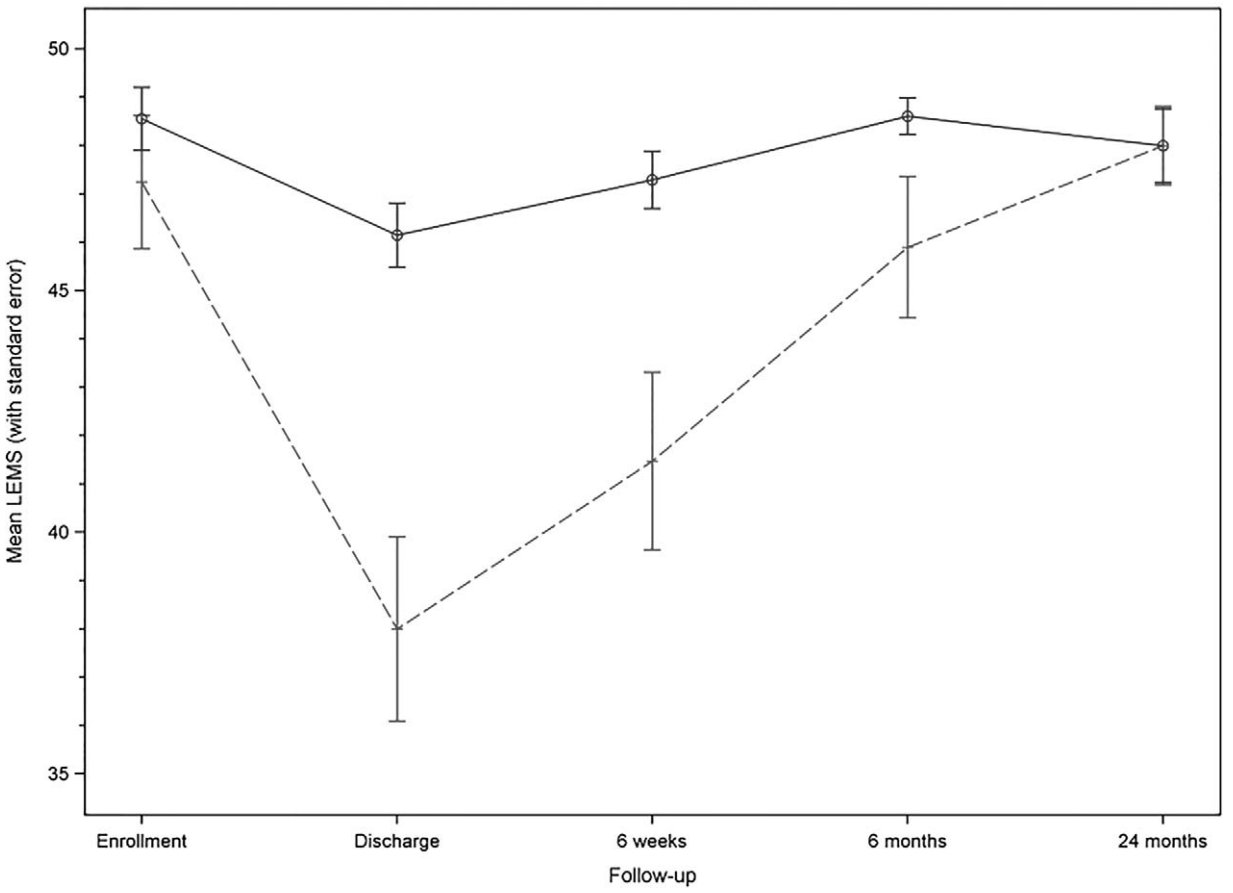

Figure 2. Change of the mean American Spinal Injury Association (ASIA) lower extremity motor score (LEMS) over time (±standard error). Solid line: average in minor declines group, broken line: average in major decline group.

TABLE 2. Summary of American Spinal Injury Association Lower Extremity Motor Score Recovery Among the Patients With Lower Extremity Motor Score Decline at Discharge

	Size of Decline in LEMS Score at Discharge		
	Major Decline (≥5 Points) n = 20	**Minor Decline (<5 Points) n = 41**	**Total n = 61**
Recovery at 6 wk (n)	17	41	58
No recovery	4 (24%)	18 (44%)	22 (38%)
Partial recovery	9 (53%)	3 (7%)	12 (21%)
Full recovery	4 (24%)	20 (49%)	24 (41%)
Recovery at 6 mo (n)	20	40	60
No recovery	1 (5%)	8 (20%)	9 (15%)
Partial recovery	6 (30%)	4 (10%)	10 (17%)
Full recovery	13 (65%)	28 (70%)	41 (68%)
Recovery at 24 mo (n)	18	38	56
No recovery	1 (6%)	7 (18%)	8 (14%)
Partial recovery	5 (28%)	3 (8%)	8 (14%)
Full recovery	12 (67%)	28 (74%)	40 (71%)

LEMS indicates lower extremity motor scores.

contrast, nerve roots are at risk during the surgical approach from either the anterior or posterior side, and these can get trapped in the foraminal compression through the corrective maneuvers that entail posterior column shortening techniques such as Smith-Petersen osteotomy and PSO. As a result, neurological deficits encountered after ASD surgery can range from complete paraplegia to solitary radiculopathy. In the present study, we recorded neurological function using ASIA LEMS to ensure objective assessment. Given the nature of impact on neurological status, declines in three myotomes or more are unlikely to be radiculopathy, although multilevel and/or bilateral lesions can still occur in theory. From that perspective, it is likely that at least half of the postoperative neurological deficits (48%) were due to spinal cord dysfunction.

The extent of postoperative deficits at discharge showed a wide range with 67% being ''minor'' in nature with <5 points of LEMS lost. Despite numerous studies reporting neurological outcome after ASD surgeries, few have mentioned the severity of decline.[11–21] The definition of ''major'' decline as opposed to ''minor'' decline has not been established, but Bridwell *et al*[11] defined major neurologic deficits as immediate postoperative motor weakness in lower extremities without antigravity strength, as did many

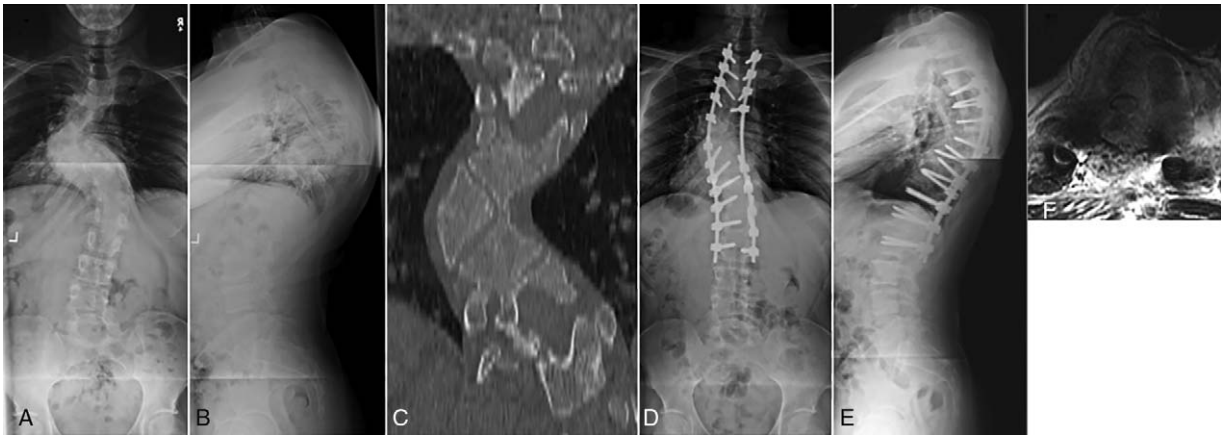

Figure 3. A 50-year-old man with congenital scoliosis (T7–8 hemivertebrae) presented with worsening spastic myelopathy (LEMS 50) due to spinal cord compression at the concavity in the apex. Preoperative coronal (**A**), sagittal (**B**) x-rays, and computed tomography (CT) image (**C**). Decompression with deformity correction by vertebrectomy was performed, but motor-evoked potentials were completely lost intraoperatively. After the optimal blood pressure was confirmed, the decision was made to proceed with deformity correction by spinal column shortening, and recovery of small waves was observed and maintained until the end of surgery.[8] Coronal (**D**) and sagittal (**E**) x-rays 1 year after surgery. Complete decompression was confirmed postoperatively by T2-weighted magnetic resonance imaging (MRI) (**F**). Although the patient was completely paralyzed temporarily after the surgery (LEMS 0), 100 mg of Riluzole, a neuroprotective agent,[9] per day was delivered and neurological function gradually recovered to LEMS 30 at discharge (''major'' decline). LEMS recovered to 40 at 6 weeks, and a cane gait was eventually regained at 3 months. The patient showed complete recovery at 6 months (LEMS 50) postoperatively.

other authors. Some of the studies discriminated spinal cord injury from nerve root injury, and others reported neurological function using ASIA or Frankel grades. Of note, the majority of "major" decline patients (90%) in the present study had declines in three or more myotomes and were thought to have myelopathy, whereas three quarters of patients with "minor" decline (73%) had declines in one or two myotomes, which were more likely to be radiculopathy. It is not uncommon that only minor deficits or nerve root injuries are reported, particularly in studies with small patient populations.[13,15,17,18] However, some larger-scale studies have reported the incidence of major deficits and minor deficits separately. Qiu *et al*[14] reported seven patients with severe postoperative deficits and 19 patients with minor deficits in their cohort of 1373 patients with scoliosis correction. Kim *et al* reported 53 major neurologic deficits, which necessitated additional intervention with longer length of stay, and 67 minor deficits among their 608 cases of ASD. In contrast, Boachie-Adjei *et al*[20] argued that they observed more spinal cord injury (23 cases) than nerve root injury (four cases). However, many of the studies in the literature were in line with our present finding of higher reported incidences of minor declines than major declines.

The percentage of patients who showed no, partial, or full recovery was almost equivalent at 6 and 24 months postoperatively. This suggests that the majority of neurological recovery occurred within 6 months postoperatively and reached a plateau thereafter, as shown in Figure 2. This new finding is almost in line with previously reported recovery course in traumatic spinal cord injury. It is generally believed that most of the acute recovery of neurological function occurs within the first 6 months after the injury, with the greatest amount occurring within the first 3 months, although a small amount of recovery can be expected up to 2 years.[22,23] Similarly, neurapraxia, the pathology of most of the reversible nerve root injuries, generally resolves within 3 months, although motor paralysis may take up to 6 months.[24] In the present study, residual neurologic decline at 24 months was observed in 6% of the patients included. Several previous studies with small cohorts reported that all neurological deficits completely resolved regardless of their severity,[13,16,17,19,25,26] but in large-scale studies, permanent damage has been reported in 12% to 50% of those with neurological complications.[14,20,27–31]

Lastly, a lack of recovery at long-term was much higher in minor decline than in major decline. This could be explained by the fact that the major decline group seemed to include more spinal cord injuries, which are mostly reversible in nature, whereas the minor decline group consisted of more resilient nerve root injuries. The leading causes of intraoperative spinal cord insult are vascular compromise and direct mechanical compression from bony structures or instrumentation. However, ischemia caused by a correction maneuver is often transient and readily manageable by maintaining blood pressure. As for mechanical compression, thorough decompression is achieved by a laminectomy and/or vertebrectomy and visually confirmed intraoperatively. On the other hand, foraminal stenosis after correction leading to the entrapment of nerve roots may not be picked up intraoperatively, and postoperative radiologic screening can also be challenging due to the metallic artifacts of instrumentation. As opposed to transient ischemia of neural tissue, these injuries caused by ongoing mechanical compression will not recover spontaneously. For example, Buchowski *et al*[27] reported, among their 108 lumbar PSO cases, that all of the permanent neurological deficits occurring were single muscle group deficits (nerve root issues) caused by mechanical compression or impingement.

Although the design of the Scoli-RISK-1 study has many strengths, it is important to acknowledge the limitations of the current work. First, the patient cohort in Scoli-RISK-1 was heterogeneous in diagnosis and treatment options. Therefore, neurological decline recorded was also likely to be diverse in etiology in terms of neurological components involved or mechanism of injury. Specifically, differential diagnosis between radiculopathy and myelopathy based on examiners' clinical impression was not used in discriminating minor *versus* major declines in the present study, although the number of myotomes affected in each group indicated that this categorization functioned as a surrogate with limitation. However, it is possible that the major decline group contained patients with minimal declines in more than 5 myotomes, and conversely, the minor decline group could have included patients with almost complete loss of function in a single myotome, which could potentially cause severe functional impairment. This granularity of neurological decline made the interpretation slightly challenging. Second, we only reported the motor aspect of neurological function by ASIA LEMS. This outcome measure was chosen to ensure subjectivity and sensitivity of examination. However, sensory deficits were not captured by LEMS, and sphincter function was not reported either. The absence of this information could have caused the underestimation of neurological decline rates. Also, the closest time point when LEMS was reported after the surgery was at discharge. Therefore, we could not identify how many of the patients experienced significant declines immediately after the surgery or what the recovery rate was at follow-up.

In conclusion, in this series of complex ASD surgeries, a higher incidence of minor *versus* major neurological decline was observed. The majority of recovery occurred in the first 6 months. Minor decline showed quicker full recovery in 6 weeks, but more commonly remained unrecovered at 24 months than major decline. This information regarding the natural history and prognosis of these surgical complications is useful for counseling patients, and it could also be used as a reference for the possible future management modalities for perioperative neurological injury. Future studies should assess the relationship between postoperative neurological deficits and health-related quality of life.

➢ Key Points

- ❑ Among 265 patients in the Scoli-RISK-1 study, 61 patients (23%) had a neurological decline at discharge, with 20 (33%) experiencing major decline (≥5 LEMS points loss) and 41 (67%) experiencing minor decline (<5 LEMS points loss).
- ❑ Approximately 90% of the patients with major decline had declines in three or more myotomes, whereas 73% of the patients with minor decline had declines in 1 or 2 myotomes.
- ❑ For patients with major decline, full recovery was seen in 24% at 6 weeks, increasing to 65% at 6 months. Approximately 24% showed no recovery at 6 weeks, decreasing to 5% at 6 months.
- ❑ For patients with minor decline, full recovery was seen in 49% at 6 weeks, increasing to 70% at 6 months. Approximately 44% showed no recovery at 6 weeks, and 20% remained unrecovered at 6 months.
- ❑ Whether the patient had a major or minor neurologic decline, the majority of recovery occurred within 6 months after surgery.

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