ISCoS
The International
Spinal Cord Society

Check for updates

# ARTICLE

# Perioperative major neurologic deficits as a complication of spine surgery

Kody K. Barrett[1 ✉], Dudley Fukunaga[2] and Kevin W. Rolfe[1,2]

**STUDY DESIGN:** Retrospective review of spine surgery patients with new major neurologic complication.
**OBJECTIVE:** To define the causes and severity of new neurologic damage to the spinal cord or cauda equina caused by spinal surgery.
**MATERIALS AND METHODS:** Consult records were reviewed for all postoperative spine surgery patients referred to a tertiary spinal cord injury rehabilitation center over a 12-year period. Any patients with a new perioperative surgery-related decrement in American Spinal Injury Association (ASIA) Impairment Scale (AIS), loss of bowel or bladder function, or loss of ability to ambulate were examined and final 1-year gaps for neurologic loss reported.
**RESULTS:** 64 patients had a new perioperative major neurologic event with: 41% thoracic, 39% cervical, and 20% lumbar; 61% intraoperative, 31% in the immediate 2-week postoperative period, 8% unknown. Chronic myelopathy (44%) was the most common indication. The causes of neurologic injury were postoperative fluid collection (25%), malposition of instrumentation (14%), traumatic decompression (14%), cord infarct (11%), deformity correction (2%), and unknown (34%). Overall, 87% lost the ability to ambulate and 66% lost volitional bowel-bladder control. AIS decrement and loss of ambulation and bowel-bladder function did not differ statistically significantly by surgical indication. However, among the main root causes, traumatic decompressions and cord infarcts had significantly worse neurologic deterioration than fluid collections or malposition of instrumentation.
**CONCLUSION:** The relative rate of major neurologic injury in spine surgery is higher in thoracic and cervical cases at spinal cord levels, especially when done for myelopathy, even though lumbar surgeries are most common. The most common causes of neurologic injury were potentially avoidable postoperative fluid collections, malposition of instrumentation, and traumatic decompression.

*Spinal Cord Series and Cases* (2021)7:81 ; https://doi.org/10.1038/s41394-021-00444-z

## INTRODUCTION

Major new neurologic deficit of the spinal cord or cauda equina is one of the most feared perioperative complications in spinal surgery. Because of the rarity, few studies have characterized the nature or detail of these events, much less using validated criteria of severity such as the American Spinal Injury Association impairment scale (AIS) [1–7]. One large scale study of 11,817 spine surgeries identified only 21 new major neurologic injuries, but without description of ambulatory or bowel-bladder status, two highly critical neurological functions, nor inclusion of fluid collections beyond 12 h, even if attributable to the surgery [3]. Such studies have produced good estimates for the incidence or rate of occurrence of these injuries, but lack specific details and often include C5 nerve root or other isolated or minor nerve palsies with major injuries. One large data-base study of 1.8 million spine surgery patients, in particular, provided basic data on overall incidence and associated comorbidities for patients sustaining any neurologic deficit, but with little detail about the severity of the deficits sustained and unable to separate minor

palsies from major neurologic injuries; rather, rates were estimated using assumed extrapolations based on prior smaller studies [7].

The goal of this study was to report on a series of new major neurologic complications as a result of spine surgery on a more granular level for both severity and etiology of injury. Studies of this size with a high degree of specificity are largely absent from the literature, with large numbers of neurologic injuries seen primarily through nonspecific database type analysis. This data comes from a pool of patients referred to a tertiary national rehabilitation center over 12 years that sustained new surgically related major neurologic deficits attributable to a spine surgery.

## MATERIALS AND METHODS

Retrospective chart review of all spine consults for patients referred for new postoperative neurologic deficit to a tertiary spinal cord injury rehabilitation center over a 12-year period from 2004 to 2016 was conducted. Spinal surgery consultation, examination, and chart review was performed per protocol by our team upon initial admission to our center for all spinal cord injured patients who were subsequently followed

**Table 1.** Causes of new neurologic deficits due to spine surgery.

| Causes | Percentage | Neurologic severity | | |
| --- | --- | --- | --- | --- |
| | | Ave. AIS grade loss | % lost B/B | % lost ambulation |
| Postoperative fluid collection | 25% (16/64) | 1.9 | 33%[a] | 87% |
| Instrumentation malposition | 14% (9/64) | 1.8 | 75% | 100% |
| Traumatic decompression | 14% (9/64) | 2.8[a] | 89% | 89% |
| Cord infarct | 11% (7/64) | 2.7[a] | 100% | 100% |
| Correction of deformity | 2% (1/64) | 3.0 | 100% | 100% |
| Unknown | 34% (22/64) | 1.9 | 77% | 87% |

*B/B* volitional bowel and bladder.

[a]Traumatic decompression and cord infarct had statistically significantly greater average AIS losses than postoperative fluid collections and instrumentation malposition ($p = 0.02$). There was no statistically significant difference for loss of ambulation stratified by cause. Fluid collections, however, had a lower rate of loss of volitional bowel-bladder control ($p = 0.004$). Deformity correction could not be assessed due to only a single case in the series.

**Table 2.** Indications for spine surgeries with new surgery-related major neurologic event.

| Indication | Percentage |
| --- | --- |
| Degenerative myelopathy | 44% (28/64) |
| Pain | 19% (12/64) |
| Tumor/Infection/Syrinx | 11% (7/64) |
| Fracture | 9% (6/64) |
| Deformity | 8% (5/64) |
| Radiculopathy | 6% (4/64) |
| Treatment of cauda equina syndrome | 3% (2/64) |

No statistically significant differences for neurologic severity (AIS loss, loss of volitional bowel-bladder function, loss of ability to ambulate) stratified by indication.

thereafter to obtain 1-year data. Patients were included only if the new neurologic loss involved damage to the spinal cord or cauda equina with a reduction by 1 grade or more in the preoperative American Spinal Injury Association (ASIA) impairment scale (AIS), loss of volitional bowel or bladder control, or loss of ability to ambulate with final assignment made at 1-year follow-up [4]. Nerve root injuries, foot drops, C5 palsies, and other peripheral neurologic injuries were excluded.

Chart review included patient demographics, details of the surgical indication, spinal region and approach, and cause and severity of the neurologic injury. Intraoperative and postoperative deficits were separated with postoperative deficits included up to 2 weeks post-surgically if, and only if, the complication was attributable to the surgery or its performance. Though the current perioperative period is often extended to 30-days, the 2-week cut-off was chosen since there were none which developed a new perioperative major neurologic deficit in this series beyond that time point.

All patients were examined and assigned postoperative AIS grades, ambulatory status, and bowel-bladder function by rehabilitation and spine staff who are trained and are experts in the exam. Preoperative examinations and neurologic status were determined from retrospective review of medical records sent from the referring institution and were confirmed with patients' own reported histories and assessments. Patient preoperative records and self-reported history of function were concordant in all cases. Access to the patients' referring surgeons was available to clarify any information or details as needed or known. All patients had an equivalent AIS grade of D (used for myelopathy patients) or higher (i.e., AIS E or patients without myelopathy or spinal cord disease or injury) preoperatively.

Statistical analysis was performed using IBM SPSS Statistics (IBM Corp., Armonk, New York, United States). Analysis of variance was performed for continuous variables across groups and Fisher exact test for categorical variables. For purposes of statistical analysis, AIS grades were assigned values as follows: AIS $A = 0$, $B = 1$, $C = 2$, $D = 3$, E or normal $= 4$. Preoperative to final postoperative losses in AIS were assigned 1 point per interval. A drop from AIS D to A, for example, equated to a 3-point change or from normal to AIS C a 2-point change. Final postoperative AIS grade,

bowel-bladder status, and ambulatory status were assigned after minimum 1-year follow-up. Institutional review board approval was obtained.

## RESULTS

64 patients were identified with a new surgery-related spinal cord or cauda equina deficit. Males were 64% (41/64) and females 36% (23/64) with the overall average age 48 years (range 11–82). There was an average decrease of 2.1 AIS grades from preoperative to postoperative status overall at 1 year.

Differences in neurologic severity stratified by *cause* and the statistically significant findings are highlighted in Table 1. Table 2 shows the relative distributions of the *indications* for surgery. No statistical differences in neurologic severity stratified by indication were found. Degenerative myelopathy, however, was the most common indication and represented 44% of cases, more than 2.3 times the nearest competitor. Excluding unknown or missing data points, overall, 87% (54/62) of patients lost the ability to ambulate and 66% (40/61) lost volitional bowel-bladder control.

The majority of surgeries were primary (92%) vs. a revision (8%), and 61% (39/64) of deficits occurred intra-operatively, 31% (20/64) postoperatively, and 8% (5/64) unknown. Table 3 shows the deficits distributed by region, approach, and timing. Finally, Table 4 shows the cause-specific details for the deficits, when known.

## DISCUSSION

This report characterizes major neurological complications of the spinal cord or cauda equina which occur as a direct result of spine surgery. This is a rare, dreaded complication in that it involves paralysis, loss of ambulation, and loss of volitional control of the bowel and/or bladder which are catastrophic to function and often largely irreversible or only partially recoverable. AIS impairment grades were used to quantify the severity of each new neurologic deficit and ambulatory and bowel-bladder status were also included, in contrast to prior studies using unvalidated scales or reports. Our results describe the final or permanent loss of function for these events from preoperatively to postoperatively with a minimum of 1-year follow-up. This ensures that any temporary deficits would have sufficient time for improvement or recovery if it were to be made [8, 9]. In addition, for rare events such as these, a tertiary spinal cord injury referral center represents the ideal setting to study perioperative neurologic complications since prospective study of a series of spine surgeries would require tens of thousands of patients to generate 64 major neurologic events [3, 7]. Our center also has a strong infrastructure for granular analysis of important neurologic outcomes including AIS grade, bowel/bladder, and ambulatory status.

**Table 3.** Additional variables among new neurologic deficits due to spine surgery.

| Timing of deficit | Intraoperative 61% (39/64) | Postoperative 31% (20/64) | Unknown 8% (5/64) |
|---|---|---|---|
| Region of spine[a] | Cervical 39% (25/64) | Thoracic 41% (26/64) | Lumbar 20% (13/64) |
| Approach | Cervical | Thoracic | Lumbar |
| | Anterior 28% (7/25) | Anterior 8% (2/26) | Anterior 8% (1/13) |
| | Posterior 60% (15/25) | Posterior 92% (24/26) | Posterior 92% (12/13) |
| | Combined 12% (3/25) | | |

[a]80% (51/64) new deficits occurred at a spinal cord level vs 20% (13/64) at a cauda equina level.

**Table 4.** Cause-specific details for new major neurologic deficits due to spine surgery by etiology.

| Etiology | Cause-specific details | No. |
|---|---|---|
| Postoperative fluid collection $n = 16$ | Abcess or purulent fluid collection compressing the cauda equina or spinal cord due to acute postoperative infection | 6 |
| | Compressive seroma, reason unknown | 2 |
| | Compressive hematoma, 2 associated with overlooked arteriolar bleeders occurring after release of gelpi retractors just prior to closure, 6 hematomas of reason unknown | 8 |
| Instrumentation malposition $n = 9$ | Medial pedicle screw breach of thoracic adolescent scoliosis case causing intra-canalicular hematoma compressing cord recognized on post-op imaging and exam | 1 |
| | Traumatic cord injury when final tightening torque-limited screwdriver for lateral mass screw caps slipped off the screw-rod construct and traumatized the cord | 1 |
| | 2 mal-reductions of occipitocervical (OC) fusions when plate was installed at 180 degrees rather than the 109 recommended (Kirkpatrick reference) | 2 |
| | Loss of neuromonitoring signals during installation of shunt tubing into myelotomy site for syrinx | 1 |
| | Postoperative interbody cage extruded into lumbar canal | 1 |
| | Acute loss of pedicle screw-rod construct fixation with collapse, 1 thoracic, 1 cervical | 2 |
| | Traumatic cord injury while placing interbody graft during anterior cervical case | 1 |
| Traumatic decompression $n = 9$ | Traumatic cord injury while attempting to take-down the posterior longitudinal ligament during anterior cervical discectomy | 2 |
| | Bone fragment pushed into canal while decompressing disc space during anterior cervical discectomy | 1 |
| | Traumatic cord injury while attempting to remove posterior cervical lamina | 1 |
| | Cord pierced while placing epidural needle | 1 |
| | Cauda equina injury during lumbar decompression with high-speed burr with dural tear and wrapping up of multiple roolets | 1 |
| | Instability and collapse during partial thoracic corpectomy | 1 |
| | Extruded disc into spinal canal after anterior lumbar discectomy preparation causing cauda equina | 1 |
| | Traumatic cervicothoracic stretch due to excess reverse Trendelenburg positioning for posterior cervical case with bodyweight pulling against fixed head to Mayfield clamp | 1 |
| Cord infarct $n = 7$ | Tying off a thoracic segmental artery, 1 during posterior thoracic approach and one during lateral thoracic approach | 2 |
| | Hypotension during anesthetic induction | 2 |
| | Intraoperative hypotension | 3 |
| Correction of deformity $n = 1$ | Distraction and correction of kyphoscoliosis deformity | 1 |

A study undertaken in 1974 (published in 1982) with numbers similar to our own was conducted using mail surveys sent to surgeons about their anterior cervical spine surgeries and associated complications [1]. From this, 70 patients were identified as having a major spinal cord complication due to surgery: 76% occurring upon awakening from anesthesia and 24% in delayed fashion. Severity, however, was not quantified and paresis and plegia were grouped together as a single entity. Among the study's 45 known etiologies: 84% were presumed due to intraoperative trauma, 9% from epidural hematoma, and 7% from infection. This compares to our study's 42 known etiologies: 38% postoperative fluid collection (14% infection), 21% instrumentation complication, 21% traumatic decompression, 17% cord infarct, and 2% deformity correction. Surgeons surveyed were unable to identify a cause in 35% (25/70) of the cases, similar to our rate of 34% (22/64). Thus, despite more modern technology available today, including increased use of neuromonitoring, the data suggest that surgeons can identify a cause of a major neurologic deficit after spine surgery when it occurs only about two-thirds of the time.

As we do not know the total number of cases that were performed for a denominator, we cannot directly ascertain rates of occurrence of these injuries. We can, however, consider rates and distributions in other studies to make additional inferences and extrapolations about potential high-risk factors. A 2009 study on major neurologic complications from an academic spine center in Cincinnati involving 11,817 spine surgery patients revealed 21 with new major neurologic deficit for an overall rate of 0.178% (~1

4

in 561 cases): cervical spine 0.293% (~1 in 341 cases), thoracic spine 0.488% (~1 in 205 cases), and lumbar/sacral spine 0.0745% (~1 in 1342 cases) [3]. The distribution of total cases was 35% cervical, 7% thoracic, and 58% lumbar. If that represents a typical distribution, then the thoracic spine seems especially susceptible to neurologic deficit as it represented 41% of the 64 major deficits in our series, though likely only 5–10% of all cases performed in the denominator. Of special note, the thoracic spine contained 5 of 7 cord infarcts in this series which was also disproportionate relative to the cervical and lumbar regions. There are a number of possible mechanisms that might account for the increased rate of deficits in the thoracic spine including it being a watershed area with relatively poorer blood supply, or perhaps simply a region that surgeons have less experience operating around, in general [10–12].

Though Cramer et al. [3] suggest an overall major event rate of 0.178%, this does not take account of the wide array of indications for surgery, particularly myelopathy. In our series, 80% of the major neurologic complications occurred at a spinal cord level (cervical or thoracic) and the most common indication was myelopathy in 44% of all cases. In a study of 384 spine surgeries done solely for myelopathy, 8 sustained neurologic injury to the spinal cord (vs. root) for a rate of 2.1% [2]. Similarly, data from the two cohorts of the AOSPine studies on cervical spondylotic myelopathy revealed 5 major perioperative (non-root) deficits of 302 patients for 1.65% in the first study and 6 of 479 for 1.25% in the second [5, 6]. This might be expected given a vulnerable spinal cord with some degree of pre-existing myelopathic damage, as well as the associated smaller canal space and margin for error in performing the decompression. Myelopathy, therefore, appears to involve a disproportionately higher baseline risk of major neurologic injury.

For myelopathy, regarding region and approach, cervical myelopathy is more common with thoracic cases representing less than one-tenth as many as cervical [13]. Among our 28 myelopathic patients sustaining injury, 50% were thoracic, far in excess of the expected background incidence unless thoracic myelopathy is of greater risk. Among the 14 cervical myelopathy cases, 5 had unknown causes of neurologic injury (2 anterior, 2 posterior, 1 combined approach), 3 were postoperative fluid collections (2 posterior, 1 anterior), 2 were instrumentation related (1 posterior, 1 combined approach), and 4 were traumatic decompressions (3 anterior, 1 posterior). Though we have previously reported a statistically higher rate of inadequate decompression with subsequent neurologic decline (not a perioperative event) requiring revision surgery in myelopathic patients after anterior cervical approaches compared to posterior approaches, best approach overall is still debated and we can make no statement about whether the perioperative neurologic event rate is higher for one or the other [14–17]. In the Yonebu et al. [2] study of 384 cervical myelopathy patients, though the authors did not analyze the difference themselves, anterior and posterior approaches were not different statistically for rates of perioperative major neurologic complications at 5/199 anterior and 3/177 posterior (Chi square $p = 0.59$). Similarly, in a study comparing anterior and posterior cervical approaches using combined data from the AOSpine myelopathy studies, there was no statistical difference between the approaches for new major neurologic event: 5/255 (2%) for anterior and 3/180 (1.7%) for posterior [17].

Of final note are the 31% of major neurologic deficits that arose in the postoperative period, most of which were due to fluid collections: 8 hematomas, 2 seromas, and 6 abscesses. This illustrates the importance of aseptic surgical technique and prevention as well as postoperative surveillance in avoiding these complications. The problem is that postoperative fluid collections are a common occurrence following spine surgery, but most of these are asymptomatic, neither known to the patient nor creating a neurologic deficit [18]. The efficacy of surgical drains to prevent postoperative fluid collections has mixed evidence and whether the decrease in observable fluid volume correlates with a protective effect against postoperative neurologic compromise remains uncertain [19–21]. Notably, postoperative fluid collections had a lower rate of loss of bowel-bladder function than all other causes and were at the lower end of AIS decrement among known causes.

This study does have important limitations including not knowing the total number of cases over which these injuries occurred to determine actual rates of injury, though these rates have been established in other studies and a determination of incidence was not a study goal. Though the data and consults were collected prospectively by our spine team, the retrospective nature of review is subject to the inaccuracies inherent to any chart review and extraction. Retrospective studies are lower grade evidence than prospective ones, but rare events often necessitate this type of study as employed herein to gain more granular detail not otherwise easily achievable. The major advantage is that we have a large number of modern events at 64 and the series includes cases referred from a broad sample of spine surgeries performed by orthopedic spine and neurosurgeons at small and large, academic, private and health maintenance organization centers throughout the Los Angeles, California basin, the second largest metropolitan area in the United States.

## REFERENCES

1. Flynn TB. Neurologic complications of anterior cervical interbody fusion. Spine. 1982;7:536–9.
2. Yonenobu K, Hosono N, Iwasaki M, Asano M, Ono K. Neurologic complications of surgery for cervical compression myelopathy. Spine. 1991;16:1277–82.
3. Cramer DE, Maher PC, Pettigrew DB, Kuntz C. Major neurologic deficit immediately after adult spinal surgery: incidence and etiology over 10 years at a single training institution. J Spinal Disord Tech. 2009;22:565–70.
4. Kirshblum SC, Burns SP, Biering-Sorensen F, Donovan W, Graves DE, Jha A, et al. International standards for neurological classification of spinal cord injury (revised 2011). J Spinal Cord Med. 2011;34:535–46.
5. Fehlings MG, Smith JS, Kopjar B, Arnold PM, Yoon ST, Vaccaro AR, et al. Perioperative and delayed complications associated with the surgical treatment of cervical spondylotic myelopathy based on 302 patients from the AOSpine north America Cervical Spondylotic Myelopathy Study. J Neurosurg Spine. 2012;16:425–32.
6. Fehlings MG, Ibrahim A, Tetreault L, Albanese V, Alvarado M, Arnold P, et al. A globalperspective on the outcomes of surgical decompression in patients with cervical spondyloic myelopathy: results from the porspective multicenter AOS-pine international study on 479 patients. Spine. 2015;40:1322–8.
7. Thirumala P, Zhou J, Natarajan P, Balzer J, Dixon E, Okonkwo D, et al. Perioperative neurologic complications during spinal fusion surgery: incidence and trends. Spine J. 2017;17:1611–24.
8. Spiess MR, Müller RM, Rupp R, Schuld C, EM-SCI Study Group, van Hedel HJ. Conversion in ASIA Impairment Scale during the First Year after Traumatic Spinal Cord Injury. J Neurotrauma. 2009;26:2027–36.
9. Kirshblum SC, O'Connor KC. Predicting neurologic recovery in traumatic cervical spinal cord injury. Arch Phys Med Rehabil. 1998;79:1456–66.
10. Dommisse GF. The blood supply of the spinal cord. A critical vascular zone in spinal surgery. J Bone Jt Surg Br. 1974;56:222–35.
11. Cheshire WP, Santos CC, Massey EW, Howard JF Jr. Spinal cord infarction: etiology and outcome. Neurology. 1996;47:321–30.
12. Alleyne CH Jr, Cawley CM, Shengelaia GG, Barrow DL. Microsurgical anatomy of the artery of Adamkiewicz and its segmental artery. J Neurosurg. 1998;89:791–5.
13. Aizawa T, Sato T, Tanaka Y, Ozawa H, Hoshikawa T, Ishii Y, et al. Thoracic myelopathy in Japan: epidemiological retrospective study in Miyagi Prefecture during 15 years. Tohoku J Exp Med. 2006;210:199–208.
14. Lukasiewicz AM, Basques BA, Bohl DD, Webb ML, Samuel AM, Grauer JN. Myelopathy is associated with increased all-cause morbidity and mortality following anterior cervical discectomy and fusion: a study of 5256 patients in American College of Surgeons National Surgical Quality Improvement Program (ACS-NSQIP). Spine. 2015;40:443–9.
15. Jiang L, Tan M, Dong L, Yang F, Yi P, Tang X, et al. Comparison of anterior decompression and fusion with posterior laminoplasty for multilevel cervical compressive myelopathy: a systematic review and meta-analysis. J Spinal Disord Tech. 2015;28:282–90.

16. Bhalla A, Rolfe KW. Inadequate surgical decompression in patients with cervical myelopathy: a retrospective review. Glob Spine J. 2016;6:542.

17. Kato S, Nouri A, Wu D, Nori S, Tetreault L, Fehlings MG. Comparison of anterior and posterior surgery for degenerative cervical myelopathy: an MRI-based propensity-score-matched analysis using data from the prospective multicenter AOSpineCSM North America and International Studies. J Bone Jt Surg Am. 2017;99:1013–21.

18. Kotilainen E, Alanen A, Erkintalo M, Helenius H, Valtonen S. Postoperative hematomas after successful lumbar microdiscectomy or percutaneous nucleotomy: a magnetic resonance imaging study. Surg Neurol. 1994;41:98–105.

19. Kou J, Fischgrund J, Biddinger A, Herkowitz H. Risk factors for spinal epidural hematoma after spinal surgery. Spine 2002;27:1670–3.

20. Lawton MT, Porter RW, Heiserman JE, Jacobowitz R, Sonntag VK, Dickman CA. Surgical management of spinal epidural hematoma: relationship between surgical timing and neurological outcome. J Neurosurg. 1995;83:1–7.

21. Mirzai H, Eminoglu M, Orguc S. Are drains useful for lumbar disc surgery? A prospective, randomized clinical study. J Spinal Disord Tech. 2006;19:171–7.

## COMPETING INTERESTS

The authors declare no competing interests.

## ADDITIONAL INFORMATION