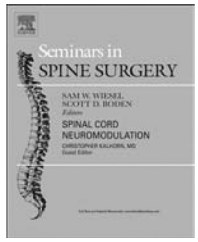
Available online at www.sciencedirect.com
ScienceDirect
www.elsevier.com/locate/semss



Complications from the surgical treatment of lumbar stenosis

Ananth S. Eleswarapu*

University of Miami Miller School of Medicine, United States

ABSTRACT

Symptomatic lumbar spinal stenosis, with resultant neurogenic claudication, is a major source of pain and disability in modern societies. Surgical decompression has been shown to improve pain and disability, albeit with an increased risk of adverse events relative to non-operative care. In this review we discuss complications of lumbar stenosis surgery, including cerebrospinal fluid leak, iatrogenic instability, infection, epidural hematoma, as well as positioning-related and medical complications. An emphasis is placed on prevention, diagnosis, and management of these commonly-seen complications.

1. Introduction

Symptomatic lumbar spinal stenosis, with resultant neurogenic claudication, is a significant cause of pain and disability in modern societies. In the United States alone, 200,000 patients suffer from symptomatic lumbar stenosis.[1] Surgical decompression has been shown in several large clinical studies to result in significant improvements in pain and function relative to non-surgical care.[2,3] Unfortunately, studies have also shown high rates of complications in surgically-managed patients, as shown in Table 1. The safe surgical management of lumbar stenosis requires thorough knowledge of possible complications, as well as techniques for avoidance and management.

2. Cerebrospinal fluid leak

Incidental durotomy is a commonly-seen complication of lumbar decompression. The rate of incidental durotomy has been reported at 3–11%.[4–17] A substantially higher rate can be expected in patients who are elderly, obese, those undergoing deformity correction, and in the revision setting.[7,17,18] When recognized and addressed appropriately, incidental durotomy does not lead to a worse long term outcome.[9,17] Persistent post-operative cerebrospinal fluid (CSF) leak, however, can result in a wide range of adverse outcomes, including postural headache, wound drainage, meningitis, pseudomeningocele, radiculopathy, intracranial hemorrhage, cerebral herniation, cranial nerve palsies, and mortality.[19–24] Delayed recognition and mismanagement of CSF leak is a major source of medical malpractice claims following spine surgery.[25]

Given the unpredictable nature of incidental durotomies, it is difficult to structure a Level-I study to evaluate management options, and the majority of available evidence consists of either small retrospective series or expert opinion. There is substantial heterogeneity in treatment patterns among spine surgeons.[15] There are, however, several principles which can guide management.

Durotomy can be recognized by extravasation of clear CSF and loss of turgor of the thecal sac. The patient should be placed in the Trendelenburg position to limit hydrostatic pressure in the lumbar spine and prevent excessive CSF loss.[13] The first step is to localize the tear. Bony resection should be carried out to the extent necessary to fully visualize

* *Correspondence to:* (c/o Karen Daley), 1201 NW 14th Street, 12th Floor, Miami, FL 33136, United States.
E-mail address: axe500@med.miami.edu

https://doi.org/10.1053/j.semss.2019.04.005
1040-7383/

Table 1 – Rates of common complications following lumbar stenosis surgery.

Complication	Rate
Iatrogenic instability	5–10%[30,31]
Incidental durotomy	3–11%[4-17]
Urinary tract infection	1.7%[82,83]
Deep wound infection	1.2–1.3%[41,82]
Superficial wound infection	0.8–1.9%[41,82]
Pneumonia	0.7–2.7%[82,86]
Congestive heart failure	0.6%[82]
Systemic sepsis	0.6%[82]
Myocardial infarction	0.3%[82]
Deep vein thrombosis	0.2–0.34%[82,94]
Pulmonary embolism	0.18–0.2%[82,94]
Epidural hematoma	0.16–0.22%[72,73]
Post-operative vision loss	0.138%[78]
Acute renal failure	0.1%[82]
Stroke	0.1–0.13%[82,95]
Mortality	0.1–0.6%[82,93]

the margins of the tear.[13] If necessary, the operative dissection should be widened. CSF extravasation can be temporarily halted with placement of a cottonoid patty or hemostatic sponge over the durotomy. Once exposure of the tear is complete, meticulous hemostasis should be obtained to prevent ingress of blood into the thecal sac. Bone fragments that have penetrated the dura should be gently removed.[13]

Primary repair of the dura should be performed whenever possible.[15] Herniated nerve rootlets must be reduced into the thecal sac.[13] To aid in reduction of nerve rootlets, sutures can be placed initially parallel to the tear. This allows for gentle traction on the ends of the tear to hold the durotomy open and allow for reduction of rootlets.[26] An assistant can depress the intradural contents with a Woodson or Penfield 4 retractor during suturing to prevent reherniation or capture of the rootlets by the suture.

There are a variety of suture materials and suturing techniques described for dural repair. The principle of watertight repair is more important than a particular suture material or technique. Either interrupted or continuous locked suture technique is acceptable.[27] A tapered needle should be used, and the ratio of needle to suture diameter should be minimized.[27] A hydrosgel or fibrin sealant may be used to limit leakage from needle holes.[27] Expansile sealants should be used with caution as they may cause postoperative neurologic compression.[13] Following closure, the patient should be returned to reverse Trendelenburg position and a Valsalva maneuver applied to 40 mmHg of positive end-expiratory pressure to verify watertight closure.[13]

There are certain scenarios in which a dural tear may not be amenable to primary repair: the tear may be too large, or overlying the root sleeve. In cases where the tear is too large for direct approximation, either fat, muscle, or synthetic dural patch can be used for dural closure.[15] This should be done as a last resort, however. Woodroffe et al. reviewed 60 patients who received a patch alone without primary closure or lumbar drain and found a failure rate of 39.5%.[15]

Wound closure must be meticulous in cases in which durotomy has occurred. Epidural dead space should be limited. A subfascial drain may be placed but intrinsic suction should not be applied to avoid damage to the repair and drainage of CSF.[16]

Post-operatively, patients should be closely monitored for postural headache, nausea, and wound drainage. It is important to inform the patient of the durotomy, both for medicolegal reasons and also so they can monitor for signs of persistent leak. Drain output should be monitored for both color and quantity. If there is uncertainty as to whether a persistent CSF leak is present, the drain fluid can be sent for a β2-transferrin assay. Flat bedrest can be prescribed to limit hydrostatic pressure against the repair. The risks of bedrest (venous thromboembolism, aspiration pneumonia, urinary tract infection) must be weighed against the potential benefits.[28] Radcliff et al. reviewed 42 patients who underwent primary dural repair for incidental durotomy, with 18 prescribed bed rest <24 h and 24 for >24 h. The authors found a 50% rate of medical complications in the >24 h group, vs 0% in the <24 h group. No difference was seen in neurologic complications, wound complications, or need for revision surgery.[28]

Several management options are available in the event of a persistent postoperative CSF leak. CT-guided epidural blood patch can be used to close the dural defect in patients with small (<4 mm) durotomies and no pseudomeningocele.[29] For larger defects, a subarachnoid drain can be placed cranial to the level of the durotomy to divert CSF flow.[13] The drain is usually left in place for 3-5 days.[13,15] Finally, there is the option of return to the operating room for revision repair.

3. Iatrogenic instability

Iatrogenic spondylolisthesis is a frequent complication when lumbar decompression is performed without arthrodesis. It has been reported that 5–10% of patients without preoperative instability will develop postoperative spondylolisthesis after undergoing open laminectomy without fusion.[30,31] Postoperative spondylolisthesis can result in low back pain, radiculopathy, recurrent stenosis, and need for revision surgery.[31]

Iatrogenic instability can be avoided by fusing the involved levels at the index operation. The proportion of patients undergoing fusion for isolated lumbar stenosis (with no associated instability or scoliosis) has increased dramatically in recent years.[32,33] Addition of fusion has several downsides, however, including increased operative time, blood loss, cost, complication risk, and risk of adjacent segment disease.[34,35] Proper selection of patients who are good candidates for decompression alone, and meticulous surgical technique to prevent destabilization, can prevent unnecessary use of fusion.

There are several preoperative risk factors that put patients at increased risk for instability following lumbar decompression. Decompression of multiple contiguous levels, preoperative disc height >6 mm, preoperative listhesis, sagittal facet orientation, and degenerative scoliosis are established risk factors for post-operative instability.[30,31,36,37] In particular, patients with preoperative spondylolisthesis that are treated with decompression alone have a high rate of progressive instability requiring revision surgery (22–38%)[34,36,38] (Fig. 1). While these risk factors do not necessitate fusion, the risk of

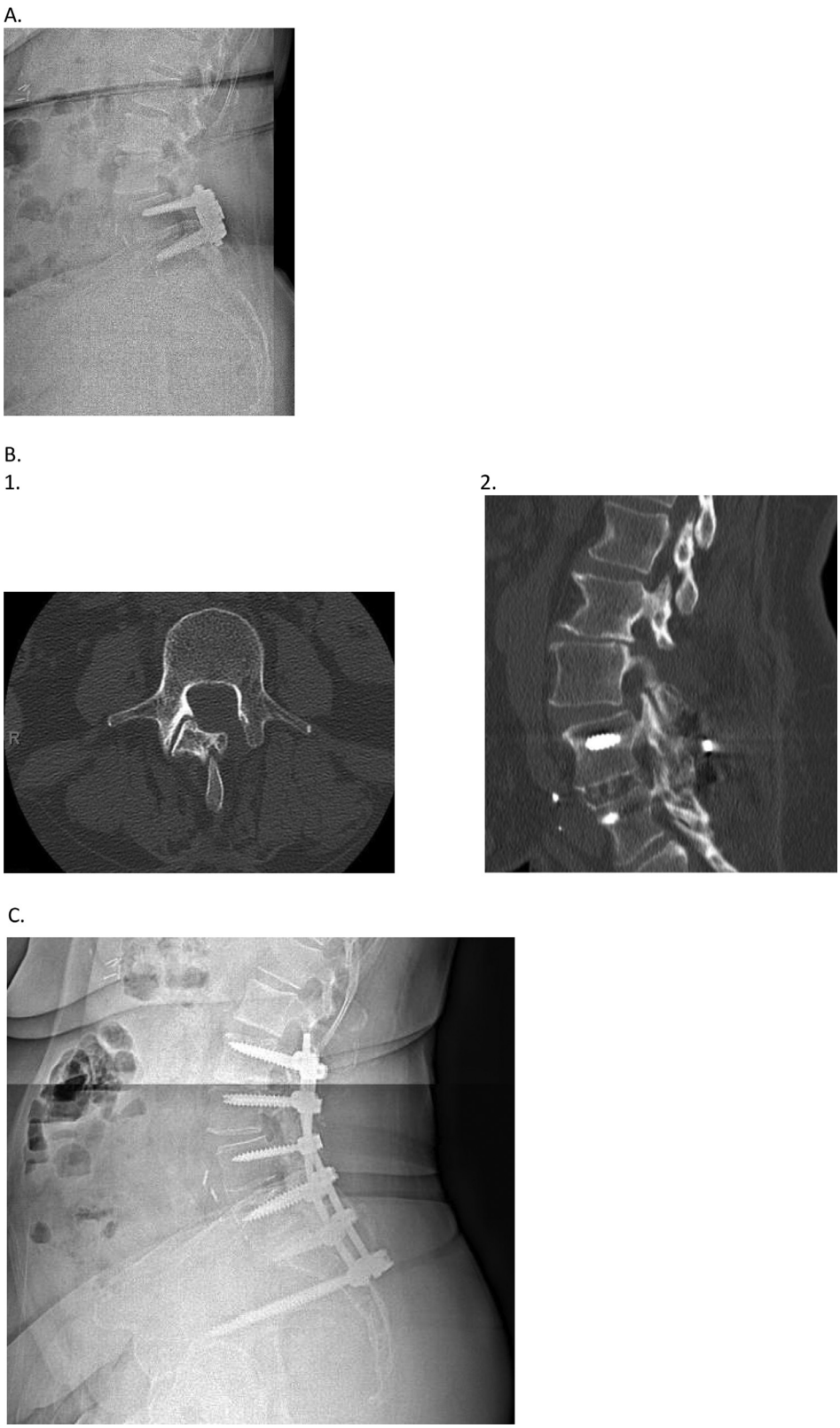


Fig. 1 – Iatrogenic instability. A 57 year old female presented with unrelenting back and leg pain. She had a history of an L4/5 fusion, done 20 years ago, and an L2/3 and L3/4 decompression for adjacent segment disease, done 6 months ago. 1A: The patient was noted to have retrolisthesis of L2/3 and anterolisthesis of L3/4 on her upright xrays. 2A/B: Her CT scan showed excessive resection of the left-sided facet at L2/3 and excessive thinning of the left-sided pars of L3, resulting in pars fracture. 3. Following stabilization, the patient had improvement in her back and leg pain.

revision surgery being needed in the future should be discussed with patients that are at high risk for instability.

When decompression is performed without fusion, it is Important to preserve as much of the posterior osseoligamentous complex as possible without sacrificing the quality of the decompression. In particular, preservation of >50% of the facet joint and >5 mm of the pars interarticularis will limit the potential for iatrogenic spondylolisthesis.[30] The posterior midline structures (spinous processes, supra- and interspinous ligaments) are also important for preservation of stability. Several midline-preserving decompression techniques have been developed, such as spinous process splitting laminectomy, bilateral hemilaminotomy, and unilateral hemilaminotomy with crossover decompression.[39,40] These techniques have shown excellent clinical outcomes with decreased risk of iatrogenic instability.[39,40]

4. Infection

Post-operative infection following lumbar decompression is associated with a range of adverse outcomes, including pseudarthrosis, chronic pain, failure of wound healing, sepsis, and neurologic injury.[41–43] The rate of mortality associated with spinal surgical site infections (SSI) is 0.8–2.3%, relative to a baseline of 0.09% in patients without an SSI.[44,45] Fortunately, the risk of infection can be lowered dramatically with appropriate patient selection and antibiotic prophylaxis.

A wide range of preoperative risk factors have been identified for lumbar spine SSI. These factors can help guide patient selection and optimization prior to surgery. Hikata et al., in a retrospective review of diabetic patients undergoing instrumented thoracolumbar surgery, found a preoperative HbA1C level of >7.0% to be associated with a 35% risk of postoperative SSI, as opposed to a 0% risk in patients with HbA1C < 7.0%.[46] Interestingly, perioperative serum glucose was not correlated with risk of infection in their study. Nutrition is also critical in prevention of SSI. Tempel et al., in their review of 83 patients who developed a postoperative spine SSI at their institution, found that 82/83 had evidence of malnutrition on preoperative labwork, as assessed by prealbumin <19 mg/dL.[47] Smoking is also a major risk factor for infection. In their systematic review of studies investigating the link between tobacco use and complications following spine surgery, Jackson et al. reported an odds ratio of 1.2–2.5 for infection in smokers.[48] The authors recommended a period of smoking cessation of at least 4 weeks prior to elective spine surgery and 6 months postoperatively.

Obesity is a major risk factor for infection, particularly in patients with body mass index >35.[49] Body fat distribution is important as well, with a recent study finding fat thickness >5 cm overlying the lumbar spine to be associated with a 4-fold increase in risk of SSI.[50] Referral for bariatric surgery prior to spine surgery is an option that should be discussed with obese patients. Jain et al., in a review of 740 patients who underwent bariatric surgery prior to posterior lumbar fusion, found the risk of infection was lower than in severely obese patients, although the risk remained elevated relative to that in patients with no history of obesity.[51]

A wide variety of medical comorbidities are associated with increased risk of infection, including anemia, coronary artery disease, and chronic kidney disease.[41,45] To the extent possible, management of these comorbid conditions should be optimized prior to elective surgery. Any non-spinal infections should also be eradicated prior to elective spine surgery.[42] Lumbar epidural injections within 3 months prior to surgery are also associated with an increased risk of postoperative infection.[52]

Surgical technique and perioperative management play an important role in prevention of infection. Surgical invasiveness contributes greatly to infection risk, and should be limited to the extent necessary to achieve an adequate clinical outcome. In a meta-analysis of 161 studies, Patel et al. found an SSI rate of 3.8% for instrumented fusion, relative to 1.8% for decompression alone.[44] Operative time and need for allogeneic blood transfusion are also associated with infection risk.[53,54] Minimally invasive techniques have also shown promise in lowering infection rates.[55] Incidental durotomy is not a risk factor for SSI.[12]

Antibiotic prophylaxis is critical for reducing both the frequency and severity of SSI.[42] The most common pathogen in spinal SSI is Staphylococcus aureus, although coagulase-negative staphylococci and gram-negative bacteria are also frequently isolated.[44,56] Historically, an intravenous first-generation cephalosporin, such as cefazolin, has been used for pre-incision prophylaxis. In the modern era, however, resistance to cefazolin has been reported as high as 40%, and many institutional antibiotic protocols now recommend a second intravenous agent, such as vancomycin or clindamycin. Lopez et al. reported a 50% decrease in the rate of SSI at their institution following the addition of intravenous vancomycin to cefazolin in their institutional protocol.[57] Vancomycin can also be administered as a topical agent. Several studies have shown a lower rate of SSI following vancomycin powder administration with minimal systemic absorption or adverse effects.[41,58,59] Use of vancomycin prophylaxis has shifted the microbial profile of spine SSI's. In particular, Pseudomonas aeuruginosa now represents an increasing portion of spine SSi's.[58] This may necessitate further changes in prophylactic protocols in the future.

As the majority of spine SSI's are the result of direct inoculation at the time of surgery, meticulous sterile technique is essential.[60] Skin preparation with chlorhexidine has been shown to decrease SSI rate relative to iodine skin preps.[61] Irrigation with dilute povidone-iodine solution prior to decortication and grafting has been shown to decrease risk of infection as well.[41] In a study in which spinal implant trays were cultured intraoperatively, Bible et al. found a 16.7% rate of contamination of sterile implants,[62] Simply covering the implants with a drape when not being used was able to reduce contamination to 2%.[62] Although not commonly used for pedicle screw instrumentation in the United States, single-use instrumentation may represent an opportunity to further reduce SSI rates. Litrico et al. reported a bi-institution study in which infection rates for lumbar fusion were decreased from 6% to 2% following adoption of single-use instrumentation at their centers. Removal of outer gloves immediately prior to insertion of instrumentation has also been shown to dramatically lower infection rate.[63] The post-

Table 2 – Postoperative infection treatment score for the spine (PITSS).[70]

Predictor		PITSS Score
Location	Cervical	1
	Thoracolumbar	2
	Lumbosacral	4
Comorbidities	None / other	0
	Cardiovascular	1
	Diabetes	4
Microbiology	Gram positive	2
	Gram negative / polymicrobial without MRSA	4
	MRSA	6
Distant Site Infection	None	1
	Urinary / respiratory tract infection	3
	Bacteremia	5
	Bacteremia + urinary / respiratory tract infection	6
Instrumentation	Yes	6
	No	2
Bone Graft	None	1
	Autograft	3
	Other (allograft, bone morphogenetic protein, synthetic)	6

operative use of closed suction drains does not appear to have an impact on infection rates.[42,64] Occlusive closure with 2-octyl-cyanoacrylate and a waterproof dressing can reduce wound drainage and infection risk.[41,64]

Post-operative SSI can be subclassified into superficial or deep, on the basis of whether the infection extends below the level of the fascia, as well as early vs late, with 4 weeks as the cutoff for a late infection.[42] Deep and late-presenting infections portend a worse prognosis.[42,65] Infection with a resistant organism (such as methicillin-resistant Staphylococcus aureus) is also a poor prognostic factor.[65]

Clinicians should have a high suspicion for SSI in the early postoperative period. Wound drainage that persists beyond 10 days after surgery is suggestive of SSI.[41,60] Pain, hyperemia surrounding the wound, and fever are also suggestive of infection although non-specific.[41] If infection is suspected, serum inflammatory markers can be assessed. Leukocytosis is common after surgery and the presence of leukocytosis alone is not suggestive of an SSI.[42] C-reactive protein (CRP) is the most sensitive marker for an SSI.[66] CRP levels should peak at day post-operative day 3 and normalize by week 2.[41,42] A "second peak" of CRP is suggestive of infection.[60] CRP can also be used as a marker to assess response to antibiotic therapy once an SSI has been diagnosed. MRI with gadolinium infusion is the most sensitive imaging study in the diagnosis of SSI.[42] Factors consistent with infection include marrow signal change (hyperintense on T2 and hypointense of T1), rim enhancement of fluid collection, disc involvement, fluid surrounding the pedicle screw heads, and early osseous destruction.[42,67,68] CT can also be useful to identify osseous destruction and lucency surrounding instrumentation.[42]

While treatment of superficial SSI can be attempted with antibiotics alone, deep SSI requires operative debridement.[42] If the patient is not septic, antibiotics can be held prior to incision in an attempt to obtain deep tissue culture. Any tissue that appears necrotic should be debrided.[60] In the setting of attempted fusion, if the arthrodesis is not yet solid, the instrumentation should be retained or exchanged.[42,60] The wound can either be closed primarily or left open with a negative-pressure dressing for serial debridement.[69] The Postoperative Infection Treatment Score for the Spine (PITSS) can help to determine the need for single-stage vs multiple debridement, with a score >21 indicating a high risk of failure with single stage debridement.[70] The components of the PITSS are shown in Table 2. The post-operative antibiotic course is generally six weeks of targeted intravenous antibiotics.[60] If a resistant organism is cultured from the wound, or if hardware is retained, six months to a year of oral suppressive antibiotics are also commonly prescribed.[42,60] In the setting of significant soft tissue loss, plastic surgery consultation for rotational or free flap coverage may be necessary.[41,60]

5. Epidural hematoma

Postoperative epidural hematoma is a rare but potentially devastating complication of lumbar decompression. As development of an asymptomatic hematoma is almost universal after spine surgery, it is unclear why a subset of patients develop compressive symptomatic hematomas.[71] The rate of symptomatic epidural hematoma after lumbar surgery has been reported at 0.16–0.22%.[72,73] Multilevel decompression, revision status, alcoholism and hypertension have been identified as risk factors for symptomatic epidural hematoma.[72,73]

Unfortunately, an effective protocol for preventing epidural hematoma has not been identified. The use of expansile hemostatic agents directly on the dura should be avoided.[72] Subfascial closed-suction drains decrease the rate of asymptomatic hematoma, but have not been shown to decrease the rate of symptomatic hematoma.[71] Post-operative anticoagulation may raise the rate of symptomatic hematoma. Awad et al., in a review of 14,392 patients, found an increased risk in patients treated with Coumadin if the INR rose above 2.0 within the first 48 h.[74] McLynn et al., in a review of the National Surgical Quality Improvement Program (NSQIP) database, found a relative risk of 7.37 for hematoma requiring surgical decompression in patients receiving unfractionated heparin vs. no prohpylaxis.[75]

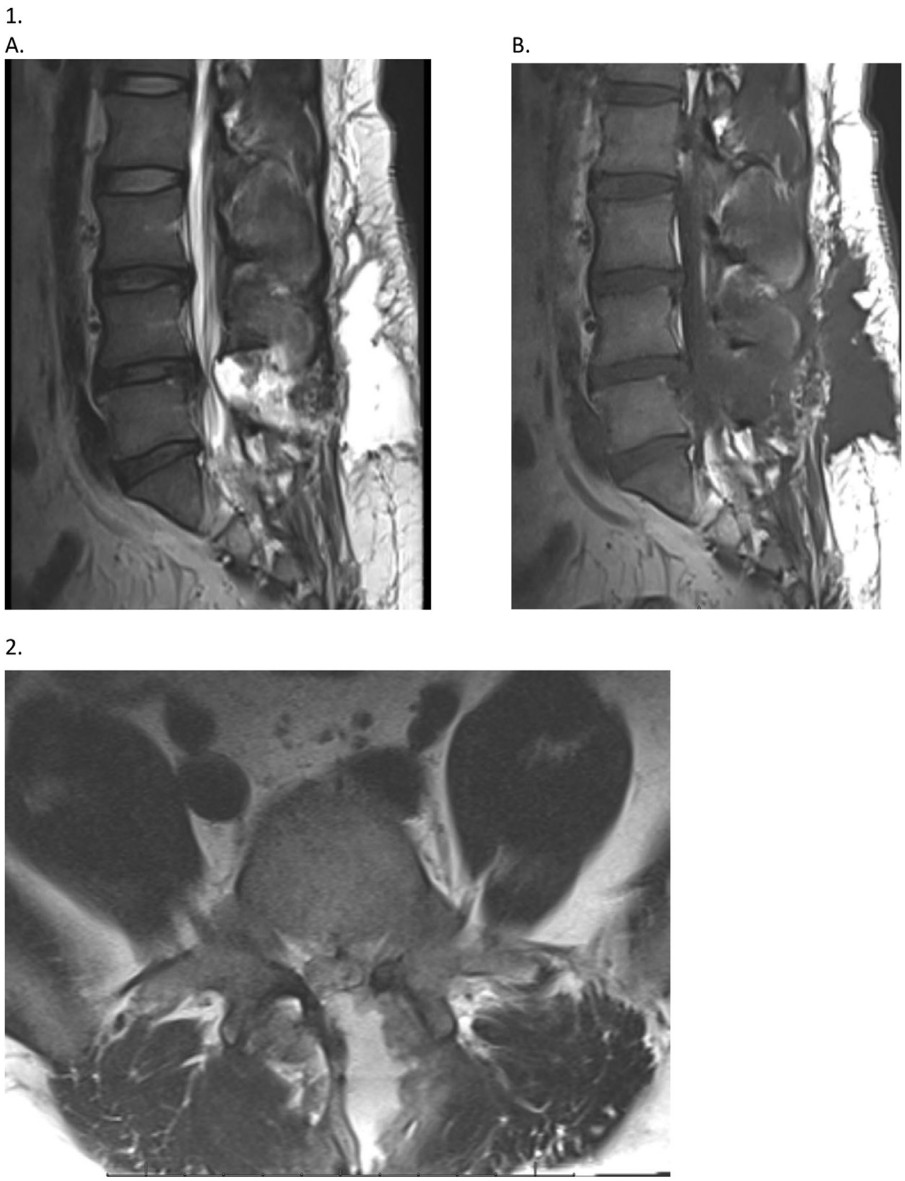


Fig. 2 – Cerebrospinal fluid leak. A 24 year old male underwent a left L4/5 microdiscectomy. Intraoperatively, an incidental durotomy was observed and repaired primarily. At 10 days post-operative, he presented to clinic with serosanguinous wound drainage, back pain, and recurrent radiculopathy. 1A/B: A large, homogenous fluid collection which is isointense with CSF on T1. Note the absence of disc space or marrow signal changes which would indicate infection. 2: Note the minimal effacement of the thecal sac, as is characteristic of CSF leak. The patient was treated with an epidural blood patch and 24-hour bedrest with complete resolution of his symptoms.

Kao et al., in a review of 15,562 patients undergoing lumbar decompression surgery at their institution, identified 25 patients with symptomatic epidural hematoma.[72] They report lower extremity weakness (80%), saddle anesthesia (76%), and sudden severe increase in pain (56%) as the most common presenting symptoms.[72] In any patients that have new post-operative neurologic deficits, MRI should be obtained emergently to evaluate for the possibility of compressive hematoma. Sokolowski et al. reviewed pre and post-operative lumbar spine MRIs and found the ratio of thecal sac diameter on post-operative vs pre-operative MRI to be correlated with patient symptoms. An average ratio of 0.8 was seen in asymptomatic patients, 0.5 in patients with pain, and 0.2 in patients with cauda equine syndrome.[76]

It is important to distinguish epidural hematoma from other causes of post-operative fluid collection such as infection or cerebrospinal fluid leak, as the treatment protocol will differ for each diagnosis. Radcliff et al. reviewed 33 patients at their institution who had a post-operative MRI within 2 weeks of lumbar spine surgery to identify imaging characteristics of each diagnosis.[67] Epidural hematoma can be differentiated from CSF leak from the presence of a mass effect on the thecal sac and fluid signal of increased T1 intensity relative to CSF.[67] Differentiation from infection is seen in absence of

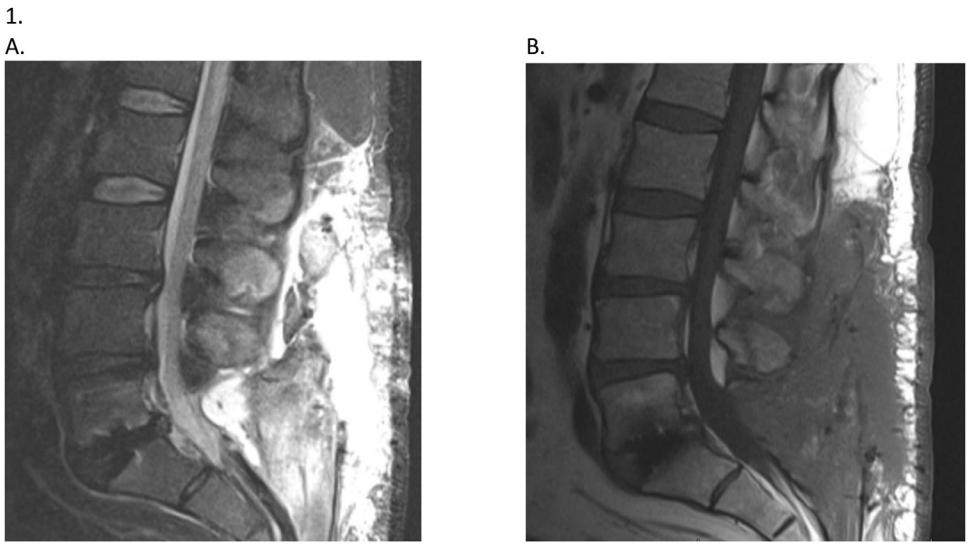


Fig. 3 – Surgical site infection. A 49 year old male underwent an L5/S1 TLIF. At 4 weeks post-operative, her presented to the emergency room with wound drainage and back pain. 1A/B: He was found to have a large fluid collection on MRI. The fluid is hyperintense relative to CSF on both T1 and T2, which differentiates it from CSF leak. There is marrow signal change on both sequences in L5, which is characteristic of infection. The patient underwent operative irrigation and debridement, with operative cultures positive for methicillin-sensitive Staphylococcus aureus. He was treated with six weeks IV oxacillin and 6 months oral amoxicillin with resolution of the infection.

disc space involvement, marrow signal change, and osseous destruction.[67] Example MRI images of CSF leak, infection, and epidural hematoma are seen in Figs. 2–4.

Upon diagnosis of symptomatic epidural hematoma, decompression of the fluid collection should be performed in an urgent manner. Mukerji and Todd, in a meta-analysis of 31 studies on treatment of spinal epidural hematoma, found the two main factors influencing outcome to be severity of neurologic deficit prior to decompression and the time to decompression.[77] The authors recommend decompression within 12 h of symptom onset.[77]

6. Positioning-related complications

There is limited literature on positioning-related complications following lumbar spine surgery and it is likely that these complications are both underappreciated and underdiagnosed. There are, however, several basic principles which can guide prevention of these complications.

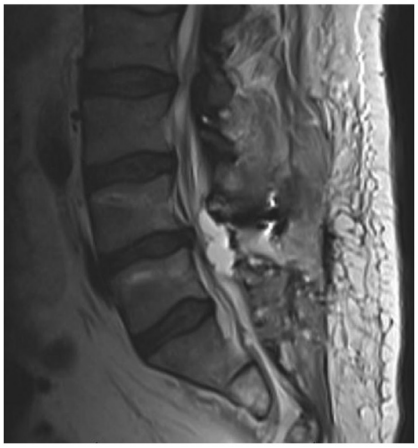
Post-operative visual loss (POVL) is perhaps the most feared positioning-related complication of lumbar spine surgery. Li et al. performed a meta-analysis and systematic review of 4 large-scale studies and 27 case reports of POVL.[78] The authors report a 0.138% incidence of POVL, with three main causes: ischemic optic neuropathy, central retinal artery occlusion, and cortical blindness. Presentation of vision loss can be either immediately following surgery or with several days delay. Risk factors including vasculopathy (history of coronary artery disease, diabetes, or smoking), large volume crystalloid infusion during surgery, and prolonged operative time. The authors recommend several techniques for avoiding visual loss, including positioning the head above the level of the heart, minimizing direct pressure on the eyes, maintaining neutral position of the neck, and avoiding intraoperative hypotension.[78] Emery et al. evaluated intraocular pressure in 52 patients undergoing lumbar spine fusion and noted significantly lower intraocular pressure with the head elevated 10° as opposed to neutral. The authors recommended head elevation to minimize intraocular pressure and improve optic nerve perfusion.[79] Presence of any visual symptoms following spine surgery warrants urgent ophthalmologic consult.[78]

A variety of positioning-related complications are attributable to positioning on the prone frame. These include peripheral neuropathy (of ulnar, median, or lateral femoral cutaneous nerves), brachial plexopathy, compartment syndrome (of abdomen or thigh), or pressure ulcers.[80] To avoid these complications, key pressure points should be checked for padding prior to draping. These include the medial elbow and anterior superior iliac spine. Great caution should be taken to avoid patient rotation which can cause asymmetric pressure.[81] Arms should be positioned at less than 90° glenohumeral abduction and secured. The abdomen should be allowed to hang free. Minimizing operative time will also decrease risk of pressure-related complications.

7. Medical complications

Medical complications in the perioperative period following lumbar spine surgery are related to the physiologic toll of the procedure, perioperative medications (including anesthetic agents and opiod pain medications), as well as baseline comorbidities of the patient. Deyo et al. reviewed 12,154 patients from the Veterans Affairs National Surgical Quality

1.



2.

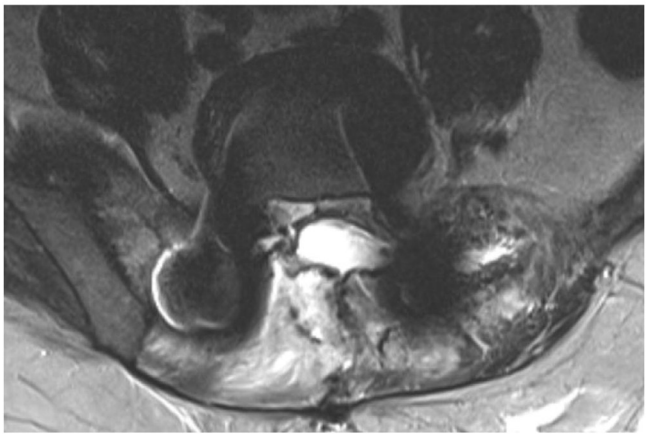


Fig. 4 – Epidural hematoma. A 50 year old male was treated with an L4/5 decompression/fusion. On post-operative day 1 he developed left leg pain and foot drop. 1. MRI showed a fluid collection at the laminectomy site. Note the difference in signal intensity between the fluid collection and CSF. Also note the absence of disc or marrow signal change. 2. Axial T2 image shows significant effacement of the thecal sac resulting in central stenosis and compression of the traversing left L5 nerve root. The patient was referred to interventional radiology for CT-guided drainage of the fluid collection, with immediate resolution of his symptoms.

Improvement Database who underwent surgery for a diagnosis of lumbar stenosis.[82] The authors report a rate of major medical complications of 2.1%. American Society of Anesthesiologists class and age were strongly correlated with risk of major medical complications.

Urinary tract infection (UTI) is among the most common medical complications seen after lumbar spine surgery, with a rate of 1.7%.[82,83] The risk of systemic sepsis following UTI was found to be 11.5% in the NSQIP database, relative to a baseline risk of 0.6% in patients undergoing lumbar fusion.[82] Nunez Pereira et al. reviewed 88 patients with post-operative UTI following instrumented spine surgery at their institution and found an SSI rate of 25%.[84] In 38% of patients with both UTI and SSI, the same gram-negative organism was isolated from both infections. Although not specific to spine surgery, adherence to 24 h catheter-removal protocols has decreased UTI rates dramatically following colonic surgery.[85]

The incidence of pneumonia following posterior lumbar fusion has been reported at 0.59% in the NSQIP database, with a mortality of 2.7%.[86] A pneumonia-prevention protocol of head of bed elevation to 30° for meals, twice-daily oral hygiene with chlorhexidine, and encouragement of ambulation and incentive spirometry was able to reduce pneumonia rates by 44% among inpatients at a Veterans Affairs hospital.[87]

Clostridium difficile colitis was found in the Nationwide Inpatient Sample to have an incidence of 0.11% following lumbar spine surgery, with a mortality rate of 4%.[88] The unnecessary use of antibiotics should be avoided to help reduce the frequency of this complication. In particular, the empiric use of post-operative antibiotics has not shown a benefit in reducing the rate of SSI.[89]

The occurrence of asymptomatic venous thromboembolism (VTE) following spine surgery is quite common. A study in which 588 patients undergoing lumbar spine surgery received routine ultrasonagraphy at 1 week post-operative found a deep vein thrombosis (DVT) rate of 32%. The rate of clinically significant VTE is much lower, at 0.4–0.9%.[82,90] There is no consensus guideline at this time as to whether anticoagulation should be routinely used postoperatively following lumbar spine surgery. Review of the NSQIP database found 54% of patients received postoperative anticoagulation following elective spine surgery.[75] Cox et al. reviewed results before and after their institution began a prophylaxis protocol that consisted of 5000u subcutaneous heparin three times daily. The authors found a decreased rate of DVT with no change in the rate of PE or epidural hematoma.[91]

Given that lumbar stenosis is a degenerative condition, a large proportion of patients with disabling symptoms are elderly. It is common for patients and surgeons alike to be concerned about the risk of complications in elderly patients. Rihn et al. reviewed four-year outcomes from the Spine Patient Outcomes Research Trial (SPORT) to compare complication rates between octogenarians and those younger than 80.[92] They found patients over the age of 80 to have a significant improvement in patient-reported outcomes with no increase in complication rates relative to younger patients. Prabhu et al. reviewed outcomes from the NSQIP database to compare patients over the age of 80 undergoing lumbar decompression to those aged 45–65. In this study they did find a higher rate of complications and mortality, but noted the overall complication rate to be low.[93]

8. Conclusion

Surgical decompression of symptomatic lumbar stenosis is associated with substantial improvements in pain and disability. Complications are common but with appropriate management most complications can be resolved without a detriment to long term patient outcomes. Surgeons should adhere to the principles of pre-operative optimization and use of the least invasive treatment that can achieve the desired clinical outcome. Awareness of commonly-seen complications and a high degree of vigilance in the early

post-operative period will help to ensure early recognition and response to adverse events.

Disclosures

The authors note no proprietary or commercial interest in any product mentioned or concept discussed in this article.

REFERENCES

1. Lurie J, Tomkins-Lane C. Management of lumbar spinal stenosis. *Br Med J*. 2016;352:h6234.
2. Ma X, Zhao X, Ma J, et al. Effectiveness of surgery versus conservative treatment for lumbar spinal stenosis: A system review and meta-analysis of randomized controlled trials. *Int J Surg*. 2017;44:329–338.
3. Lurie JD, Tosteson TD, Tosteson A, et al. Long-term outcomes of lumbar spinal stenosis: eight-year results of the Spine Patient Outcomes Research Trial (SPORT). *Spine*. 2015;40:63–76.
4. Adogwa O, Huang MI, Thompson PM, et al. No difference in postoperative complications, pain, and functional outcomes up to 2 years after incidental durotomy in lumbar spinal fusion: a prospective, multi-institutional, propensity-matched analysis of 1,741 patients. *Spine J*. 2014;14:1828–1834.
5. Yohihara H, Yoneoka D. Incidental dural tear in lumbar spinal decompression and discectomy: analysis of a nationwide database. *Arch Orthop Trauma Surg*. 2013;133:1501–1508.
6. Murray NJ, Demetriades AK, Rolton D, et al. Do surgeon credentials affect the rate of incidental durotomy during spine surgery? *Eur Spine J*. 2014;23:1767–1771.
7. Chen Z, Shao P, Sun Q, et al. Risk factors for incidental durotomy during lumbar surgery: a retrospective study by multivariate analysis. *Clin Neurol Neurosurg*. 2015;130:101–104.
8. Buck JS, Yoon ST. The incidence of durotomy and its clinical and economic impact in primary, short-segment lumbar fusion: an analysis of 17,232 cases. *Spine*. 2015;40:1444–1450.
9. Ulrich NJ, Burgstaller JM, Brunner F, et al. The impact of incidental durotomy on the outcome of decompression surgery in degenerative lumbar spinal canal stenosis: analysis of the Lumbar Spinal Outcome Study (LSOS) data – a Swiss prospective multi-center cohort study. *BMC Musculoskelet Disord*. 2016;17:170.
10. Kothe R, Quante M, Engler N, et al. The effect of incidental dural lesions on outcome after decompression surgery for lumbar spinal stenosis: results of a multi-center study with 800 patients. *Eur Spine J*. 2017;26:2504–2511.
11. Nandyala SV, Elboghdady IM, Marquez-Lara A, et al. Cost analysis of incidental durotomy in spine surgery. *Spine*. 2014;39:E1042–E1051.
12. Desai A, Ball PA, Bekelis K, et al. SPORT: does incidental durotomy affect longterm outcomes in cases of spinal stenosis? *Neurosurgery*. 2015;76(Suppl 1):S57–S63.
13. Papavero L, Engler N, Kothe R. Incidental durotomy in spine surgery: first aid in ten steps. *Eur Spine J*. 2015;24:2077–2084.
14. Herren C, Sobotke R, Mannion AF, et al. Incidental durotomy in decompression for lumbar spinal stenosis: incidence, risk factors and effect on outcomes in the Spine Tango registry. *Eur Spine J*. 2017;26:2483–2495.
15. Woodroffe RW, Nourski KV, Helland LC, et al. Management of iatrogenic spinal cerebrospinal fluid leaks: a cohort of 124 patients. *Clin Neurol Neurosurg*. 2018;170:61–66.
16. Kamenova M, Leu S, Mariani L, et al. Management of incidental dural tear during lumbar spine surgery. To suture or not to suture? *World Neurosurg*. 2016;87:455–462.
17. Iyer S, Klineberg EO, Zebala LP, et al. Dural tears in adult deformity surgery: incidence, risk factors, and outcomes. *Global Spine J*. 2018;8:25–31.
18. Burks CA, Werner BC, Yang S, et al. Obesity is associated with an increased rate of incidental durotomy in lumbar spine surgery. *Spine*. 2015;40:500–504.
19. Sporns PB, Shwindt W, Cnyrim CD, et al. Undetected dural leaks complicated by accidental drainage of cerebrospinal fluid (CSF) can lead to severe neurologic deficits. *Rofo*. 2016;5:451–458.
20. Kim YJ. Incarceration of spinal nerve root through incidental durotomy as a cause of sciatica. *Korean J Spine*. 2017;14:103–105.
21. Wichmann TO, Karabegovic S, Rasmussen MM, et al. Cranial nerve palsies due to incidental durotomy in lumbar spine surgery: a case report. Br J Neurosurg (In Press), 2019.
22. Raudenbush BL, Molinari A, Molinari RW. Large compressive pseudomeningocele causing early major neurologic deficit after spine surgery. *Glob Spine J*. 2017;3:206–212.
23. Graffeo CS, Perry A, Wijdicks EF. Subarachnoid hemorrhage and spinal subdural hematoma due to acute CSF hypotension. *Neurocritical Care*. 2017;26:109–114.
24. Lin TY, Chen WJ, Hsieh MK, et al. Postoperative meningitis after spinal surgery: a review of 21 cases from 20,178. *BMC Infect Dis*. 2014;14:220.
25. Durand WM, Eltorai AEM, Shantharam G, et al. Medical malpractice claims following incidental durotomy due to spinal surgery. *Spine*. 2018;43:940–945.
26. Rahyussalim AJ, Djaya YP, Saleh I, et al. Preservation and tissue handling technique on iatrogenic dural tera with herniated nerve root and cauda equina level. Case Rep Orthop (In Press), 2019.
27. Dafford EE, Anderson PA. Comparison of dural repair techniques. *Spine J*. 2015;15:1099–1105.
28. Radcliff KE, Sidhu GD, Kepler CK, et al. Complications of flat bed rest after incidental durotomy. *Clin Spine Surg*. 2016;29:281–284.
29. Mihlon F, Kranz PG, Gafton AR, et al. Computed tomography-guided epidural patching of postoperative cerebrospinal fluid leaks. *J Neurosurg Spine*. 2014;21:805–810.
30. Guha D, Heary RF, Shamji MF, et al. Iatrogenic spondylolisthesis following laminectomy for degenerative lumbar stenosis: systematic review and current concepts. *Neurosurg Focus*. 2015;39:E9.
31. Rahmdani S, Xia Y, Xu R, et al. Iatrogenic spondylolisthesis following open lumbar laminectomy: case series and review of the literature. *World Neurosurg*. 2018;113:E383–E390.
32. Raad M, Donaldson CJ, El Dafrawy MH, et al. Trends in isolated lumbar spinal stenosis surgery among work US adults aged 40-64 years, 2010-2014. *J Neurosurg Spine*. 2018;29:169–175.
33. Bae HW, Rajaee SS, Kanim LE. Nationwide trends in the surgical management of lumbar spinal stenosis. *Spine*. 2013;38:916–926.
34. Forsth P, Olafsson G, Carlsson T, et al. A randomized controlled trial of fusion surgery for lumbar spinal stenosis. *N Engl J Med*. 2016;374:1413–1423.
35. Chang W, Yuwen P, Zhu Y, et al. Effectiveness of decompression alone versus decompression plus fusion for lumbar spinal stenosis: a systematic review and meta-analysis. *Arch Orthop Trauma Surg*. 2017;137:637–650.
36. Blumenthal C, Curran J, Benzel EC, et al. Radiographic predictors of delayed instability following decompression without fusion for degenerative grade I lumbar spondylolisthesis. *J Neurosurg Spine*. 2013;18:340–346.
37. Houten JK, Nasser R. Symptomatic progression of degenerative scoliosis after decompression and limited fusion surgery for lumbar spinal stenosis. *J Clin Neurosci*. 2013;20:613–615.

38. Ghogawala Z, Dziura J, Butler WE, et al. Laminectomy plus fusion versus laminectomy alone for lumbar spondylolisthesis. *N Engl J Med*. 2016;374:1424–1434.
39. Nomura H, Yanagisawa Y, Arima J, et al. Clinical outcome of microscopic lumbar spinous process-splitting laminectomy: clinical article. *J Neurosurg Spine*. 2014;21:187–194.
40. Chang HS, Fujisawa N, Tsuchiya T, et al. Degenerative spondylolisthesis does not affect the outcome of unilateral laminotomy with bilateral decompression in patients with lumbar stenosis. *Spine*. 2014;39:400–408.
41. Radcliff KE, Neusner AD, Millhouse PW, et al. What is new in the diagnosis and prevention of spine surgical site infections? *Spine J*. 2015;15:336–347.
42. Pawar AY, Biswas SK. Postoperative spine infections. *Asian Spine J*. 2015;10:176–183.
43. Andres-Cano P, Cervan A, Rodriguez-Solera M, et al. Surgical infection after posterolateral lumbar spine arthrodesis: CT analysis of spinal fusion. *Orthop Surg*. 2018;10:89–97.
44. Patel H, Khoury H, Girgenti D, et al. Burden of surgical site infections associated with select spine operations and involvement of Staphylococcus aureus. *Surg Infect (Larchmt)*. 2017;18:461–473.
45. De la Garza Ramos R, Abt NB, Kerezoudis P, et al. Deep-wound and organ space infection after surgery for degenerative spine disease: an analysis from 2006–2012. *Neurol Res*. 2016;38:117–123.
46. Hikata T, Iwanami A, Hosogane N, et al. High preoperative hemoglobin A1C is a risk factor for surgical site infection after posterior thoracic and lumbar spinal instrumentation surgery. *J Orthop Sci*. 2014;19:223–228.
47. Tempel Z, Grandhi R, Maserati M, et al. Prealbumin as a serum biomarker of impaired perioperative nutritional status and risk for surgical site infection after spine surgery. *J Neurosurg Part A Central Eur Neurosurg*. 2015;76:139–143.
48. Jackson KL, Devine JG. The effects of smoking and smoking cessation on spine surgery: a systematic review of the literature. *Glob Spine J*. 2016;6:695–701.
49. Bono OJ, Poorman GW, Foster N, et al. Body mass index predicts risk of complications in lumbar spine surgery based on surgical invasiveness. *Spine J*. 2018;18:1204–1210.
50. Lee JJ, Odeh KI, Holcombe SA, et al. Fat thickness as a risk factor for infection in lumbar spine surgery. *Orthopedics*. 2016;39:e1124–e1128.
51. Jain D, Berven SH, Carter J, et al. Bariatric surgery before elective posterior lumbar fusion Is associated with reduced medical complications and infection. *Spine J*. 2018;18:1526–1532.
52. Singla A, Yang S, Werner BC, et al. The impact of preoperative epidural injections on postoperative infection in lumbar fusion surgery. *J Neurosurg Spine*. 2017;26:645–649.
53. Jannsen SJ, Braun Y, Wood KB, et al. Allogeneic blood transfusions and postoperative infections after lumbar spine surgery. *Spine J*. 2015;15:901–909.
54. Ogihara S, Yamazaki T, Inanami H, et al. Risk factors for surgical site infection after lumbar laminectomy and/or discectomy for degenerative disease in adults: a prospective multicenter surveillance study with registry of 4027 cases. *PLoS One*. 2018.
55. Mueller K, Zhao D, Johnson O, et al. The difference in surgical site infection rates between open and minimally invasive spine surgery for degenerative lumbar pathology: a retrospective single center experience of 1442 cases. Oper Neurosurg (In Press), 2019.
56. Norton TD, Skeete F, Dubrovskaya Y, et al. Orthopedic surgical site infections: analysis of causative bacteria and implications for antibiotic stewardship. *Am J Orthop*. 2014;43:E89–E92.
57. Lopez W, Rider SM, Nwosu K, et al. The impact of vancomycin and cefazolin as standard pre-operative antibiotic prophylaxis on surgical site infections following instrumented spinal fusion. Spine (In Press), 2019.
58. Hey HW, Thiam DW, Koh ZS, et al. Is intraoperative local vancomycin powder the answer to surgical site infections in spine surgery? *Spine*. 2017;42:267–274.
59. Haimoto S, Schar RT, Nishimura Y, et al. Reduction in surgical site infection with suprafascial intrawound application of vancomycin powder in instrumented posterior spinal fusion: a retrospective case-control study. *J Neurosurg Spine*. 2018;29:193–198.
60. Butler JS, Wagner SC, Morrissey PB, et al. Strategies for the prevention and treatment of surgical site infection in the lumbar spine. *Clin Spine Surg*. 2018;31:323–330.
61. Lee I, Agarwal RK, Lee BY, et al. Systematic review and cost analysis comparing use of chlorhexidine with use of iodine for preoperative skin antisepsis to prevent surgical site infection. *Infect Control Hosp Epidemiol*. 2010;31:1219–1229.
62. Bible JE, O'Neill KR, Crosby CG, et al. Implant contamination during spine surgery. *Spine J*. 2013;13:637–640.
63. Rehman A, Rehman AU, Rehman TU, et al. Removing outer gloves as a method to reduce spinal surgery infection. *J Spinal Disord Tech*. 2015;28:E343–E346.
64. Glennie AR, Dea N, Street JT. Dressings and drains in posterior spine surgery and their effect on wound complications. *J Clin Neurosci*. 2015;22:1081–1087.
65. Maruo K, Berven SH. Outcome and treatment of postoperative spine surgical site infections: predictors of treatment success and failure. *J Orthop Sci*. 2014;19:398–404.
66. Gunne AF, Hosman AJ, Cohen DB, et al. A methodological systematic review on surgical site infections following spine surgery: part 1: risk factors. *Spine*. 2012;37:2017–2033.
67. Radcliff KE, Morrison WB, Kepler C, et al. Distinguishing pseudomeningocele, epidural hematoma, and postoperative infection on postoperative MRI. *Clin Spine Surg*. 2016;29:E471–E474.
68. Kimura H, Shikata J, Odate S, et al. Pedicle screw fluid sign: an indication on magnetic resonance imaging of a deep infection after posterior spinal instrumentation. *Clin Spine Surg*. 2017;30:169–175.
69. Mehbod AA, Ogivlie JW, Pinto MR, et al. Postoperative deep wound infections in adults after spinal fusion: management with vacuum assisted wound closure. *J Spinal Disord Tech*. 2005;18:14–17.
70. Diapola CP, Saravanja DD, Boriani L, et al. Postoperative infection treatment score for the spine (PITSS): construction and validation of a predictive model to define need for single versus multiple irrigation and debridement for spinal surgical site infection. *Spine J*. 2012;12:218–230.
71. Schroeder GD, Kurd MF, Kepler CK, et al. Postoperative epidural hematomas in the lumbar spine. *J Spinal Disord Tech*. 2015;28:313–318.
72. Kao FC, Tsai TT, Chen LH, et al. Symptomatic epidural hematoma after lumbar decompression surgery. *Eur Spine J*. 2015;24:348–357.
73. Amiri AR, Fouyas IP, Cro S, et al. Postoperative spinal epidural hematoma (SHE): incidence, risk factors, onset, and management. *Spine J*. 2013;13:134–140.
74. Awad JN, Kebaish KM, Donigan J, et al. Analysis of the risk factors for the development of post-operative spinal epidural hematoma. *J Bone Joint Surg*. 2005;87:1248–1252.
75. McLynn RP, Diaz-Collado PJ, Ottesen TD, et al. Risk factors and pharmacologic prophylaxis for venous thromboembolism in elective spine surgery. *Spine J*. 2018;18:970–978.
76. Sokolowski MJ, Garvey TA, Perl 2 J, et al. Postoperative lumbar epidural hematoma: does size really matter? *Spine*. 2008;33:114–119.
77. Mukerji N, Todd N. Spinal epidural haematoma: factors influencing outcome. *Br J Neurosurg*. 2013;27:712–717.
78. Li A, Swinney C, Verravagu A, et al. Postoperative visual loss following lumbar spine surgery: a review of risk factors by diagnosis. *World Neurosurg*. 2015;84:2010–2021.

79. Emery SE, Daffner SD, France JC, et al. Effect of head position on intraocular pressure during lumbar spine fusion: a randomized, prospective study. *J Bone Joint Surg*. 2015;97:1817–1823.
80. Shriver MF, Zeer V, Alentado VJ, et al. Lumbar spine surgery positioning complications: a systematic review. *Neurosurg Focus*. 2015;39:E16.
81. Agarwal N, Mistry JB, Khandge PV, et al. Meralgia paresthetic after spine surgery on the Jackson table. *Clin Spine Surg*. 2018;31:53–57.
82. Deyo RA, Hickam D, Duckart JP, et al. Complications following surgery for lumbar stenosis in a veteran population. *Spine*. 2013;38:1695–1702.
83. Bohl DD, Ahn J, Tabaraee E, et al. Urinary tract infection following posterior lumbar fusion procedures: an American College of Surgeons National Surgical Quality Program Study. *Spine*. 2015;40:1785–1791.
84. Nunez-Pereira S, Rodriguez-Pardo D, Pellise F, et al. Postoperative urinary tract infection and surgical site infection in instrumented spinal surgery: is there a link? *Clin Microbiol Infect*. 2014;20:768–773.
85. Okrainec A, Aarts MA, Conn LG, et al. Compliance with urinary catheter removal guidelines leads to improved outcome in enhanced recovery after surgery patients. *J Gastrointest Surg*. 2017;21:1309–1317.
86. Bohl DD, Mayo BC, Massel DH, et al. Incidence and risk factors for pneumonia after posterior lumbar fusion procedures: an ACS-NSQIP study. *Spine*. 2016;41:1058–1063.
87. Kazaure HS, Martin M, Yoon JK, et al. Long-term results of a postoperative pneumonia prevention program for the inpatient surgical ward. *JAMA Surgery*. 2014;149:914–918.
88. Skovrlj B, Guzman JZ, Silvestre J, et al. Clostridium difficile colitis in patients undergoing lumbar spine surgery. *Spine*. 2014;39:E1167–E1173.
89. Lewis A, Lin J, James H, et al. A single-center intervention to discontinue postoperative antibiotics after spinal fusion. *Br J Neurosurg*. 2018;32:177–181.
90. Bouyer B, Rudnichi A, Dray-Spira R, et al. Thromboembolic risk after lumbar spine surgery: a cohort study on 325,000 French patients. J Thromb Haemost (In Press), 2019.
91. Cox JB, Weaver KJ, Neal DW, et al. Decreased incidence of venous thromboembolism after spine surgery with early multimodal prophylaxis: clinical article. *J Neurosurg Spine*. 2014;21:677–684.
92. Rihn JA, Hillibrand AS, Zhao W, et al. Effectiveness of surgery for lumbar stenosis and degenerative spondylolisthesis in the octogenarian population. *J Bone Joint Surg*. 2015;97:177–185.
93. Prabhu AV, Lieber BA, Henry JK, et al. Early postoperative complications for elderly patients undergoing single-level decompression for lumbar disc herniation, ligamentous hypertrophy, or neuroforaminal stenosis. *Neurosurgery*. 2017;81:1005–1010.
94. Fineberg SJ, Oglesby M, Patel AA, et al. The incidence and mortality of thromboembolic events in lumbar spine surgery. *Spine*. 2013;38:1154–1159.
95. Marquez-Lara A, Nandyala SV, Fineberg SJ, et al. Cerebral vascular accidents after lumbar spine fusion. *Spine*. 2014;39:673–677.