ISSN: 0975 -8542

Journal of Global Pharma Technology

Available Online at: www.jgpt.co.in

CASE REPORT

Aphasia after Spinal Deformity Correction Surgery in Adolescent Idiopathic Scoliosis: A Case Report

Luthfi Gatam[1], Harmantya Mahadhipta[2], Phedy[1], Asrafi Rizki Gatam[1], Aldo Fransiskus Marsetio[1], Aji Antoro[1*]

[1]Spine Division, Orthopaedic and Traumatology Department, Faculty of Medicine, Universitas Indonesia/ Fatmawati General Hospital, Jakarta.

[2]Spine Division, Orthopaedic and Traumatology Division, Tangerang General Hospital, Tangerang.

***Corresponding Author: Aji Antoro**

Abstract

Introduction: Advancement in operative techniques, better understanding of biomechanics and breakthrough in technology making spinal deformity correction surgery become more feasible and conducted more often. Majority of the neurological complications were related to spinal cord disruption due to technical issues. However, neurological complications that were attributed to brain pathology were more likely to be of ischemic origin. Case Illustration: We performed spinal deformity correction surgery on a 20-year old man with adult idiopathic scoliosis classification Lenke 2AN, with Cobb angle of 68°. On the second day, the serum sodium level showed a reduction. MRI examination revealed hyper acute infarct lesion on the posterior lobe of left temporo-parietal aspect. However, DSA examination was unremarkable. On the third postoperative day, his motoric and sensory functions, and sodium level returned to normal state. His cognitive function and communication abilities were able to return to normal gradually. Discussion: Stroke that occurs during spinal deformity correction surgery is reported in 5.8% of operative cases. Prone positioning accompanied by extension of the neck can compress vertebral arteries, causing reduction in vascular flow. Therefore, inappropriate prone positioning on the operating table for a prolonged period may cause cerebral ischemia. Conclusion: This case may serve as a reminder to surgeons and anesthesiologists towards the existence of such complications. Full awareness of the risk, good preoperative assessment, good surgical training, good communication, good teamwork, and good intraoperative and postoperative monitoring will minimize the risk for neurological complications.

Keywords: *Aphasia, Spinal Deformity, Adolescent Idiopathic Scoliosis.*

Introduction

Scoliosis is a spinal deformity disorder, in which the spine is curved sideways. Along with the improvement of medical screening and diagnosis, more people are detected with the spinal deformity. Additionally, advancement in operative techniques, better understanding of biomechanics and breakthrough in technology making spinal deformity correction surgery become more feasible and conducted more often. Spinal deformity correction surgery is a major operation with high impact on the physiology of the patient and carries high risk of complications. The complications may be categorized as infection, neurological, mechanical, and general/peri-operative [1]. One systemic review found 40% risk of adverse peri-operative events, [2] while a multicenter study reported overall complication rate of 39% [3]. On the other hand, morbidity and mortality registry of adult scoliosis surgery by Scoliosis Research Society only find 13.4% complication rate [4]. One study in Europe found that the most complications were related to mechanical or neurological, while the neurological

complications were related to technical issues causing spinal cord disruption [1].

The most commonly found postoperative neurological complication is delirium. It affects up to 70% of elderly patients undergoing major surgery, with adverse outcomes varying from persistent cognitive decline up to mortality [5, 6]. It is a marker of susceptibility of the brain, and most frequently, underlying neurological disease presents in these patients [7]. Complication of perioperative stroke in general surgery is varying widely, between 0.08-1.0% [8, 9]. However, the highest risk found on cardiac surgery and carotid endartectomy (4-10%) [10, 11].

Some studies also found that the incidence of perioperative stroke is attributable to perioperative metoprolol use in non-cardiac surgery [12, 13]. Spinal cord ischemia (SCI) postoperative complication is closely related to thoracoabdominal aneurysms and dissections repair surgery [11]. The etiology of immediate onset SCI is thought due to spinal cord blood supply interruption.

The postoperative mortality due to SCI may be as high as 50%, with 5-year mortality rate up to 75% [14, 15]. Visual loss after surgery is rare, with the highest rate can be found in posterior spine surgery (0.03%) [16]. It can be caused by occlusion of central retinal artery, cortical blindness, and ischemic optic neuropathy (ION)[11].

One study found that postoperative visual loss is related to blood loss ≥1 liter or anesthetic duration ≥6 hours [17]. Cholelithiasis and acalculous cholecystitis, pancreatitis, superior mesenteric artery syndrome, ileus, coagulopathy, chylothorax, pneumothorax, hemothorax are other rare complications that also associated with scoliosis surgery, but causing no neurologic symptom. Complication of stroke may occur during or after surgery, or may even be delayed up to 30 days. Mostly the brain pathology occurs is of ischemic origin, less likely of haemorrhagic. The risk is not related to general anesthesia procedure [18].These patients have the same risk of stroke even without having the surgery. However, the mortality risk in these setting of surgery is higher than in the community [19]. Stroke neurologist consultation and necessary neuroimaging need to be done shall postoperative stroke occur.

Additionally, physiological perturbations, such as hypotension, hypoxia, hyperthermia, hyperglycemia, should be avoided because it can exacerbate the neural injury [11].

Case Report

A 20-year old man presented to our outpatient clinic and we diagnosed him with adult idiopathic scoliosis classification Lenke 2AN, with Cobb angle of 68°. The patient was planned for spinal deformity correction surgery. Preoperative neurology, cardiology, and pulmonology assessment was unremarkable. No previous history of any cardiac or vascular disease. Preoperative medication were given as the following: 500 miligram injection of tranexamic acid (Tranexid, Dexa Medica); 125 miligram injection of methylprednisolone (Medixon, Ferron); and inhalation with 2.5 miligram of albuterol (Ventolin, GlaxoSmithKline Pharmaceuticals). The patient also had preoperative physiotherapy deep breathing exercise.

However, preanesthesia assessment revealed ASA physical status III with low central venous pressure (0-4 cmH_2O). In order to maintain the blood pressure and central venous pressure, 20 miligram injection of furosemide (Lasix, Sanofi Group Indonesia), 250 miligram injection of dobutamine hydrochloride (Dobuject, Dexa Medica), and 250 mililiter infusion of 5% albumin (Octalbin, Kalbe Farma) were also given by the anesthesiologist.

The patient had additional preoperative medication of 1 gram injection of meropenem (Merofen, Kalbe Farma), 40 miligram injection of pantoprazole (Pranza, Kalbe Farma), and calcium supplement (Epocaldi, Novell Pharma). Spinal deformity correction was performed, and the surgery went uneventful. Intraoperative bispectral index were maintained at the value of 75-79. Preoperative medication appeared to increase the blood pressure up to 170/90 mmHg and heart rate up to 130 bpm.

However, along the course of the surgery it was able to be maintained into the range of 110-90 mmHg on the diastolic, 70-50 mmHg on the systolic, and 85-100 heart bpm. Body temperature was maintained between 34.8°C to 35.4°C**.** The duration of the surgery was five hours with 1600 ml bleeding? As much as 1500 ml crystaloids, 250 ml coloids, 989 ml packed red cells and 551 fresh frozen

plasmas were transfused during the surgery. Eighteen pedicle screws and nuts, two long rods, and one cross-link were implanted at the level of third thoracal up to second lumbar vertebrae.

Corrections up to Cobb angle of 16° were able to be achieved. Intraoperative nerve monitoring were used (somatosensory evoked potentials, motor evoked potentials, free run electromyography), and it showed no significant changes on wave latency and amplitudes until the end of the surgery. Postoperatively, the patient was treated in Intensive Care Unit (ICU) for tight observation. After being conscious, the patient's vital sign was unexceptional, despite slightly tachycardia. However, the patient had altering levels of consciousness and had difficulty in communicating.

Cognitive function of the patient was good, a simple command was able to be comprehended, and simple words could be expressed. However, he was having difficulties to explicate complex sentences on his mind. Additionally, the patient also felt that the coordination of his right side of the body was poor. The urine output was slightly increased (2.4/ml/kg/hour), laboratory evaluation revealed increased leukocyte (15,780/uL), metabolic acidosis, hyperglycemia (371 mg/dL), and hyperlactatemia (10.15 mEq/L).

Other than those, laboratory results were within normal ranges, including electrolytes (Sodium 135 mEq/L; Potassium: 4.0 mEq/L; Chloride: 93mEq/L). Cardiometry examination revealed tachycardia, low stroke volume, very high stroke volume variation. Other parameters were within normal ranges. On the following day, the tachycardia was still persisted intermittently, while other vital signs were stable.

Diuresis was still slightly increased (2.4/ml/kg/hour). Serum level of sodium showed a reduction of 131mEq/L. Previously abnormal laboratory values were returned to within normal ranges. All cardiometry parameters were returned within normal ranges. Numbness and poor coordination of the right upper extremity persisted.

As much as 0,625 mcg/kgBW/minute infusion of Dobutamine (Dobuject, Dexa Medica) and 0.05 mcg/kgBW/minute infusion of norepinephrine (Vascon, Fahrenheit) were given to control the blood pressure and CVP. Mean arterial pressure was maintained for 100-120 mmHg. For the neurovascular problem, the patient received medication of mannitol 20% infusion, citicholine (Brainact, Kalbe Farma), and 25,000 IU heparin (Inviclot, Fahrenheit) infusion.

Additionally, the patient had seizure once. Thus, 5 mg Midazolam (Miloz, Novell Pharmaceutical Laboratories) injection and twice 100 mg Phenytoin infusion drip were given. Magnetic resonance imaging (MRI) examination revealed hyper acute infarct lesion on the posterior lobe of left temporo-parietal aspect. There was no sign of intra- or extra-parenchymal intracranial bleeding.

Digital subtraction angiography (DSA) of the brain was also performed. It revealed that the diameter of vessels on the brain were within normal caliber, no stenosis, no malformation, no aneurysm, good and symmetrical perfusion of both hemisphere of cerebrum and cerebellum, and no signs of cerebral vein thrombosis. On the third postoperative day, his hemodynamic was stable, motoric and sensory function returned to normal state.

Serum sodium was returned to normal value (138 mEq/L), diuresis was decreased to normal (1.1 ml/kgBW/hour). His cognitive function and communication ability were improved on the 4th postoperative day. With some cognitive therapy and speech physiotherapy, the cognitive function and communication ability gradually returning to normal. After being considered as generally stable, the patient was then discharged at the 10th postoperative day without any residual symptom.

Discussion

There are some known neurology complications of spinal deformity correction surgery in scoliosis. Spinal cord injury may be caused by direct injury or vascular injury. Once suspected, anesthesiologist should be informed and optimize the blood pressure, hematocrit, and oxygenation [20].Our patient indeed had reduced motoric function postoperatively; however, the recovery was very well over time. Despite intraoperative mechanical injury may be excluded; other mechanism may still be suspected.

Leakage of dural content due to accidental durotomy during thoracolumbar surgery may induce intracerebellar hemorrhage or

subdural or extradural hematoma [21].Unusual headache or central neurological effect is the alarm signal for emergency cerebral imaging with suspicion of dural leakage [22].

Despite no intraoperative accidental durotomy and careful instrumentation, unnoticed dural tear is still possible. Nevertheless, MRI examination of our patient revealed no cerebral hemorrhage. Stroke that occurs during spinal deformity correction surgery is reported in 5.8% of operative cases [19].

The second postoperative day is reported to be the critical time for occurrence of perioperative brain infarction [23]. One epidemiology study regarding cerebral vascular accidents after lumbar spine fusion found deformity correction as one of the associated risk factors [24].

On the other hand, Huang et al [25].Found protective effect of deformity surgery towards stroke. Stroke was approximately 25% less likely to happen in patients who underwent spinal fusion surgery, in this study of more than 13,000 adult spinal deformity patients within 10 years. The exact pathophysiology of stroke as complication in spine surgery is still unclear.

Hyponatremia as a part of syndrome of inappropriate antidiuretic hormone (SIADH) has been associated with spine fusion and deformity correction procedure, including scoliosis surgery [26].The anti diuretic hormone levels could be elevated postoperatively for the first 72 hours. Increased level of antidiuretic hormone causes excessive free water retention and hyponatremia with hypoosmolality, despite normal function of kidney and adrenal.

It leads to fluid shifts into intracellular fluid compartment and cerebral edema [27]. The syndrome should be suspected in patient with oliguria after surgery, serum and urine electrolyte including osmolality should be performed. Postoperatively, although serum electrolyte examination revealed borderline normal sodium level of135 mEq/L, it was decreased into 131 mEq /L at the second postoperative day.

However, the serum sodium level returned to normal at the third postoperative day (138mEq/L). Unfortunately, however, the urine electrolyte and urine/serum osmolality was not checked. Additionally, our patient had an increased urine output of 2.4/ml/kg/hour. This shows that no fluid retention occur on the patient.

The increased urine output occurs because of a positive fluid balance protocol were applied on the patient due to low CVP. Thus, the occurrence of SIADH as the cause of cerebral edema might be exluded. Fat embolism is one important complication in any kind of surgeries such as long bone fracture or arthroplasty surgery, but rarely occurs in scoliosis surgery.

Free fat droplets may originated from bone marrow or surrounding soft tissue, and enter circulation from torn vein. However, available literature is still unable to explain whether neurologic symptoms are caused by fat emboli to the brain [27]. Based on DSA examination, our patients was revealed that there was with symmetrical perfusion of both hemisphere of cerebrum and cerebellum no occlusion on the vessels, and the diameter was still normal.

Therefore, the mechanism possibility of vessels occlusion can be excluded. Inappropriate prone positioning on the operating table for a prolonged period may cause several complications, such as ophthalmologic injuries (either due to central retinal artery occlusion or ischemic optic neuropathy) peripheral nerve injury (for example ulnar nerve, brachial plexus, lateral femoral cutaneous nerve, or peroneal nerve) [28].

Additionally, it is also be considered as the cause of cerebral ischemia. Studies have shown that prone position, accompanied by rotation and/or extension of the neck, can compress vertebral arteries and causing reduction in vascular flow [29, 30]. Furthermore, it may even lead to neurologic complications, such as cerebral and cerebellar infarction, hemiparesis, quadriplegia and phrenic nerve injury [31].

The exact position of our patient's head and neck during the surgery was not well documented. However, this could be one of the possible contributing factors. Surgeons and anesthesiologist should pay attention toward the head and neck placement during positioning.

Suggestions for Clinical Practice

Central nervous system is a unity from the brain to cauda equina. Any damage or surgery done to any part of it may complicate another. Perhaps, there are some preventive measures to reduce these kinds of complications upon spine surgery. First and the foremost, knowledge and awareness towards these possible complications are important. Good surgical training, good communication, and good teamwork are necessary for a surgery to be successful. Every team members must be alert towards all possible complications of the surgery.

Preoperative clinical condition of the patient must be optimized to prevent it from happening. Additionally, every patient should have recent clinical examination, updated laboratory and imaging. Risk of postoperative paraplegia and brain infarction may be reduced by good preoperative neurologic assessment. MRI prior to surgery may also useful in patients with congenital spine anomaly [28].

Good surgical planning should be performed, including patient's positioning, approach, and instruments. Nowadays, the application of intraoperative neurophysiologic monitoring (IONM) is becoming more common in spine surgery. Studies have shown that the use of IONM is able to decrease neurological complications of spine surgery, including in scoliosis surgery [32, 34]. It is found to be sensitive and specific for detecting intraoperative neurologic injury, with the motor-evoked potentials (MEP) examination is more sensitive [35].

Free-running EMG can also continuously monitor peripheral nerves or roots at risk for potential injury. Intraoperative blood pressure management is another important factor. Generally, 70 mmHg of mean arterial blood pressure (MAP) is the safe pressure to decrease intraoperative blood loss.

However, MAP of less than 55-60 mmHg may have increased risk of spinal cord ischemia.[36] Additionally, blood pressure is also related to cerebral perfusion pressure, which is important to be kept maintained during surgery. Study found that only about 50% of people who have normal or complete configuration of the circle of Willis, while the rest of them have occlusion and/or incompetent circle of Willis [37].

Recent study even found 73% of people has atypical circle of Willis pattern [38]. With the addition of severe internal carotid artery stenosis, combination of these risk factor will increase the risk of watershed infarction, leading to ischemic stroke [39, 40].Postoperatively, patient's vital function and fluid balance must be closely monitored. Therefore, the patient should be treated in ICU for tight observation at least 24 hours postoperatively.

MAP and overall hemodynamic status should be closely assessed. Any decrease in urine output, laboratory examination of serum and urine electrolyte including osmolality should be checked. Postoperative complete and thorough neurology examination also should be performed. Besides sensory and motor function, higher function of the brain should be assessed as well, including mental status, memory, orientation, speech, reflexes, and others. Other possible complications such as ophthalmology and brachial plexus should also be examined.

Conclusion

Good functional and anatomic results can be achieved by spinal deformity surgery. However, the procedure exposes patients to a variety of risks, including neurological. The case we presented is a case of aphasia after spinal deformity correction surgery. The exact cause is unknown; however, we suspected that intraoperative positioning might contribute to the condition.

This case report may serve as an alert and a reminder to all surgeons and anesthesiologists towards the existence of such complications. Full awareness of the risks, good preoperative assessment, good surgical training, good communication, good teamwork, and good intraoperative and postoperative monitoring will minimize the risk for neurological complications. Appropriate laboratory, imaging, and neurophysiological examination would be a significant aid.

Statement of Disclosure

All authors would like to declare NO conflict of interest or financial ties with any vendor or organization that might have been mentioned in the submitted article.

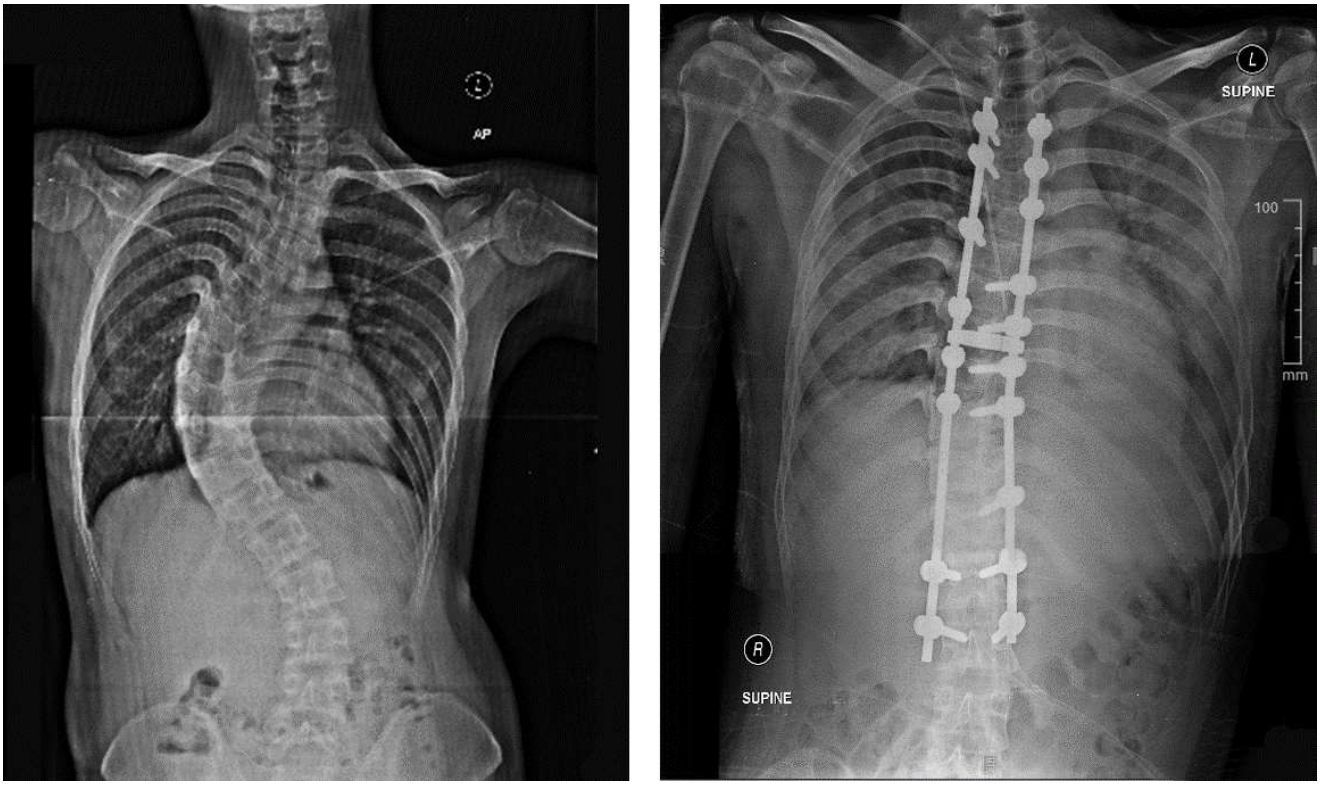

Figure 1: Spine x-ray in anteroposterior projection. Preoperative (Left); Postoperative (Right)

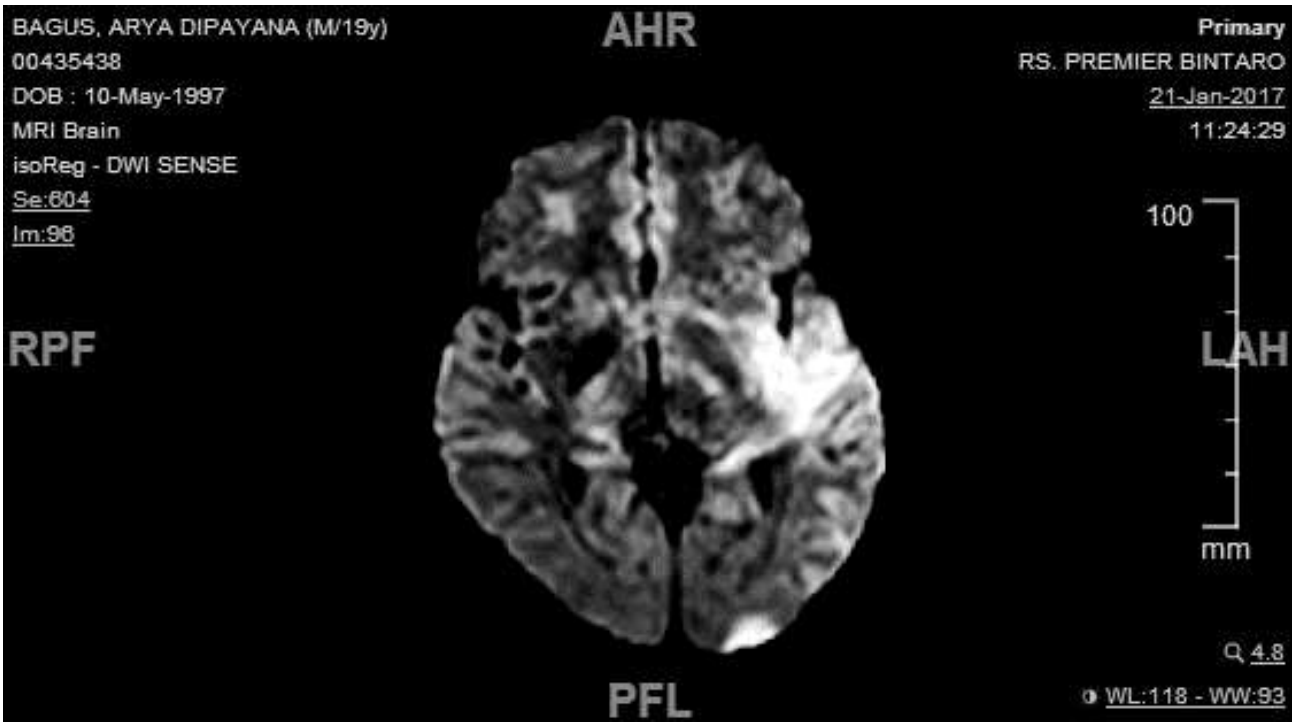

Figure 2: Postoperative brain magnetic resonance imaging showed hyper acute infarct lesion on the posterior lobe of left temporo-parietal aspect

References

1. Bhagat S, Vozar V, Lutchman L, Crawford RJ, Rai AS (2013) Morbidity and mortality in adult spinal deformity surgery: Norwich Spinal Unit experience. European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society, 22 (1):S42-6.
2. Yadla S, Maltenfort MG, Ratliff JK, Harrop JS (2010) Adult scoliosis surgery outcomes: a systematic review. Neurosurgical focus, 28(3):E3.
3. Charosky S, Guigui P, Blamoutier A, Roussouly P, Chopin D (2012) Study Group on S. Complications and risk factors of primary adult scoliosis surgery: a

multicenter study of 306 patients. Spine, 37(8):693-700.

4. Sansur CA, Smith JS, Coe JD, Glassman SD, Berven SH, Polly DW, Jr et al (2011) Scoliosis research society morbidity and mortality of adult scoliosis surgery. Spine, 36(9):E593-7.
5. Tsai MC, Chou SY, Tsai CS, Hung TH, Su JA (2013) Comparison of consecutive periods of 1-, 2-, and 3-year mortality of geriatric inpatients with delirium, dementia, and depression in a consultation-liaison service. International journal of psychiatry in medicine, 45(1):45-57.
6. Neufeld KJ, Leoutsakos JM, Sieber FE, Wanamaker BL, Gibson Chambers JJ, Rao V, et al (2013) Outcomes of early delirium diagnosis after general anesthesia in the elderly. Anesthesia and analgesia, 117(2):471-8.
7. Davis DH, Muniz Terrera G, Keage H, Rahkonen T, Oinas M, Matthews FE, et al (2012) Delirium is a strong risk factor for dementia in the oldest-old: a population-based cohort study. Brain : a journal of neurology, 135(Pt 9):2809-16.
8. Selim M (2007) Perioperative stroke. The New England journal of medicine, 356(7):706-13.
9. Bateman BT, Schumacher HC, Wang S, Shaefi S, Berman MF (2009) Perioperative acute ischemic stroke in noncardiac and nonvascular surgery: incidence, risk factors, and outcomes. Anesthesiology, 110(2):231-8.
10. Bucerius J, Gummert JF, Borger MA, Walther T, Doll N, Onnasch JF, et al (2003) Stroke after cardiac surgery: a risk factor analysis of 16,184 consecutive adult patients. The Annals of thoracic surgery, 75(2):472-8.
11. Mashour GA, Woodrum DT, Avidan MS (2015) Neurological complications of surgery and anaesthesia. British journal of anaesthesia, 114(2):194-203.
12. Mashour GA, Sharifpour M, Freundlich RE, Tremper KK, Shanks A, Nallamothu BK, et al (2013) Perioperative metoprolol and risk of stroke after noncardiac surgery. Anesthesiology, 119(6):1340-6.
13. Ashes C, Judelman S, Wijeysundera DN, Tait G, Mazer CD, Hare GM, et al (2013) Selective beta1-antagonism with bisoprolol is associated with fewer postoperative strokes than atenolol or metoprolol: a single-center cohort study of 44,092 consecutive patients. Anesthesiology, 119(4):777-87.
14. Conrad MF, Ye JY, Chung TK, Davison JK, Cambria RP (2008) Spinal cord complications after thoracic aortic surgery: long-term survival and functional status varies with deficit severity. Journal of vascular surgery, 48(1):47-53.
15. Coselli JS, LeMaire SA, Koksoy C, Schmittling ZC, Curling PE (2002) Cerebrospinal fluid drainage reduces paraplegia after thoracoabdominal aortic aneurysm repair: results of a randomized clinical trial. Journal of vascular surgery, 35(4):631-9.
16. Shen Y, Drum M, Roth S (2009) The prevalence of perioperative visual loss in the United States: a 10-year study from 1996 to 2005 of spinal, orthopedic, cardiac, and general surgery. Anesthesia and analgesia, 109(5):1534-45.
17. Lee LA, Roth S, Posner KL, Cheney FW, Caplan RA, Newman NJ, et al (2006) The American Society of Anesthesiologists Postoperative Visual Loss Registry: analysis of 93 spine surgery cases with postoperative visual loss. Anesthesiology, 105(4):652-9; quiz 867-8.
18. Group GTC, Lewis SC, Warlow CP, Bodenham AR, Colam B, Rothwell PM, et al (2008) General anaesthesia versus local anaesthesia for carotid surgery (GALA): a multicentre, randomised controlled trial. Lancet., 372(9656):2132-42.
19. Ng JL, Chan MT, Gelb AW (2011) Perioperative stroke in noncardiac, nonneurosurgical surgery. Anesthesiology, 115(4):879-90.
20. Bridwell KH, Lenke LG, Baldus C, Blanke K (1998) Major intraoperative neurologic deficits in pediatric and adult spinal deformity patients. Incidence and etiology at one institution. Spine, 23(3):324-31.
21. Garreau de Loubresse C (2014) Neurological risks in scheduled spinal surgery. Orthopaedics & traumatology, surgery & research : OTSR, 100(1):S85-90.
22. Khalatbari MR, Khalatbari I, Moharamzad Y (2012) Intracranial hemorrhage following lumbar spine surgery. European spine journal : official publication of the European Spine Society, the European

Spinal Deformity Society, and the European Section of the Cervical Spine Research Society, 21(10):2091-6.

23. Parikh S, Cohen JR (1993) Perioperative stroke after general surgical procedures. New York state journal of medicine, 93(3):162-5.
24. Marquez-Lara A, Nandyala SV, Fineberg SJ, Singh K. Cerebral vascular accidents after lumbar spine fusion. Spine. 2014;39(8):673-7.
25. Huang LC, Chung WF, Liu SW, Chang PY, Chen LF, Wu JC, et al (2015) Lower Risk of Stroke after Deformity Surgery: Long Term Benefit Demonstrated by a National Cohort Study. International journal of environmental research and public health, 12(10):12618-27.
26. Callewart CC, Minchew JT, Kanim LE, Tsai YC, Salehmoghaddam S, Dawson EG, et al (1994) Hyponatremia and syndrome of inappropriate antidiuretic hormone secretion in adult spinal surgery. Spine, 19(15):1674-9.
27. Shapiro G, Green DW, Fatica NS, Boachie-Adjei O (2001) Medical complications in scoliosis surgery. Current opinion in pediatrics, 13(1):36-41.
28. Mooney JF, 3rd, Bernstein R, Hennrikus WL, Jr, MacEwen GD (2002) Neurologic risk management in scoliosis surgery. Journal of pediatric orthopedics, 22(5):683-9.
29. Harman F, Yayci F, Deren S, Hasturk AE, Tarhan B, Yaman M, et al (2012) Acute cerebellar ischemia after lumbar spinal surgery: a rare clinical entity. Journal of anesthesia, 26(6):947-8.
30. Pamphlett R, Raisanen J, Kum-Jew S (1999) Vertebral artery compression resulting from head movement: a possible cause of the sudden infant death syndrome. Pediatrics, 103(2):460-8.
31. Kumar D, Hamid M, Kumar K (2017) Another approach for prone positioning under general anaesthesia. Indian journal of anaesthesia, 61(3):278-9.
32. Sala F, Palandri G, Basso E, Lanteri P, Deletis V, Faccioli F, et al (2006) Motor evoked potential monitoring improves outcome after surgery for intramedullary spinal cord tumors: a historical control study. Neurosurgery, 58(6):1129-43; discussion -43.
33. Nuwer MR, Dawson EG, Carlson LG, Kanim LE, Sherman JE (1995) Somatosensory evoked potential spinal cord monitoring reduces neurologic deficits after scoliosis surgery: results of a large multicenter survey. Electroencephalography and clinical neurophysiology, 96(1):6-11.
34. Dawson EG, Sherman JE, Kanim LE, Nuwer MR (1991) Spinal cord monitoring. Results of the Scoliosis Research Society and the European Spinal Deformity Society survey. Spine, 16(8):S361-4.
35. Fehlings MG, Brodke DS, Norvell DC, Dettori JR (2010) The evidence for intraoperative neurophysiological monitoring in spine surgery: does it make a difference? Spine, 35(9):S37-46.
36. Owen JH (1999) The application of intraoperative monitoring during surgery for spinal deformity. Spine, 24(24):2649-62.
37. Kapoor K, Singh B, Dewan LI (2008) Variations in the configuration of the circle of Willis. Anatomical science international, 83(2):96-106.
38. Klimek-Piotrowska W, Rybicka M, Wojnarska A, Wojtowicz A, Koziej M, Holda MK (2016) A multitude of variations in the configuration of the circle of Willis: an autopsy study. Anatomical science international, 91(4):325-33.
39. Momjian-Mayor I, Baron JC (2005) The pathophysiology of watershed infarction in internal carotid artery disease: review of cerebral perfusion studies. Stroke, 36(3):567-77.
40. Ballotta E, Renon L, Da Giau G, Barbon B, De Rossi A, Baracchini C (2005) Prospective randomized study on asymptomatic severe carotid stenosis and perioperative stroke risk in patients undergoing major vascular surgery: prophylactic or deferred carotid endarterectomy? Annals of vascular surgery, 19(6):876-81.

