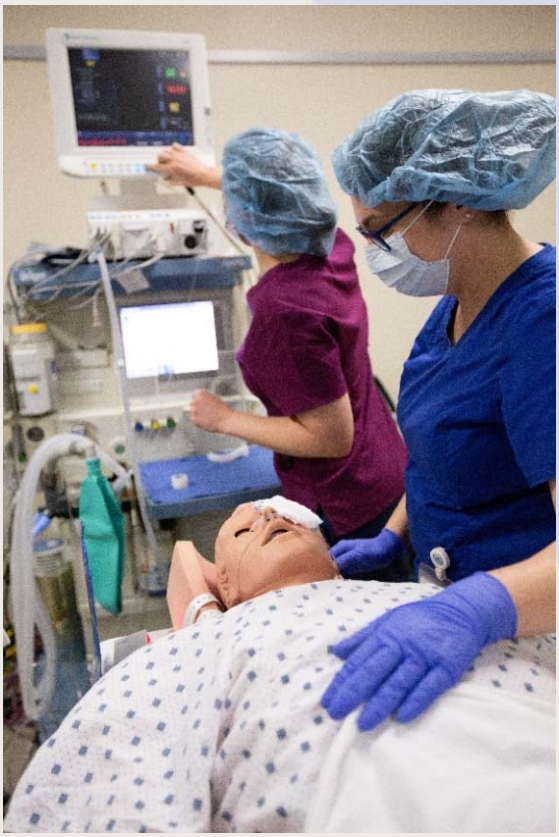
Volume 18 Issue 3 Fall 2019

The International Student Journal of Nurse Anesthesia

TOPICS IN THIS ISSUE

Lambert-Eaton Myasthenic Syndrome
Transcarotid Artery Revascularization
Pericapsular Nerve Group block
Suboccipital Craniotomy
Mustard procedure
Spinal vs. General
Lung donation
Sugammadex
Cosyntropin
Bleomycin



INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA
Vol. 18 No. 3 FALL 2019

Contributing Editors for this Issue

Nathan Adams, DNP, CRNA	National University
Jennifer Debban, MS, CRNA	National University
Clay Freeman, DNP, CRNA	Texas Wesleyan University
Amber Johnson, PhD, CRNA	University of North Dakota
Lee Ranalli, DNP, CRNA	Midwestern University
Art Shimata, DNAP, CRNA	Westminster College

Reviewers for this Issue

Erik Ark, MSN, CRNA	University of Wisconsin, Madison
Kristine S. Faust, DNAP, MS, MBA, CRNA	Michigan State University
Richard Flowers, DNP, CRNA	Wake Forest School of Medicine
Renee George, MS, CRNA	Albany Medical College
Clayton Karimi, MSN, CRNA	Washington University School of Medicine; St. Louis, MO
Johnathon T. Lannan, DNP, CRNA	Fort Campbell, Kentucky
David O'Connor, PhD, DNAP, CRNA	Memorial Sloan Kettering
MAJ Julie Petsche, DNP, CRNA, USAF, NC	Uniformed Services University
LCDR Michael Rucker, DNP, CRNA	Uniformed Services University
Julie D. Vaughan, MSN, CRNA	Nebraska Medicine, University Hospital; Omaha, NE
Brad Wagner, MSN, CRNA	University of Wisconsin, Madison
Matthew A. Wecker, DNAP, CRNA	Kearney Regional Medical Center; Kearney, NE

Front Cover:
Jason Schultz, BSN, RN, Maribel Freund, BSN, RN, Lynda Vielbig, BSN, RN, Lacey Anderson, BSN, RN, Brittany Petersen, BSN, RN, Stephanie Kuntz, BSN, RN, Allison Nierode, BSN, RN, Inshirah Robinson, BSN, RN, Melissa Beck, BSN, RN, Jessie Bozelka, BSN, RN, and Melissa Henneman, BSN, RN, graduate students enrolled in the University of Wisconsin Oshkosh Nurse Anesthesia Program, practice clinical and decision making skills by participating in simulated anesthesia scenarios, and then debrief in the School of Nursing's Simulation Learning Center. All photographs were taken by Mary Manchester.

Publication Information:

The International Student Journal of Nurse Anesthesia (ISSN 2688-5263) is published three times a year in the spring, summer, and fall. Current and past issues, and the Guide for Authors of this free, open access, electronic journal can be found at:

www.aana.com - Member Resources → Students → International Student Journal
https://www.aana.com/studentjournal
https://ifna.site/international-publications/international-student-journal-for-nurse-anesthesia/

For additional information, please contact:

Vicki Coopmans, PhD, CRNA
Webster University Department of Nurse Anesthesia
Browning Hall, ISB 107
8274 Big Bend Blvd.
St. Louis, MO 63119
314-246-5928; Intsjna@aol.com

Table of Contents

Case Reports

Total Intravenous Anesthesia in a Patient with Lambert-Eaton Myasthenic Syndrome

Kristin Couch, MSNA
Texas Wesleyan University

Keywords: gastrectomy**,** laparoscopic gastric sleeve, Lambert-Eaton myasthenic syndrome, LEMS, neuromuscular, total intravenous anesthesia (TIVA)

This is a case review of an anesthetic experience on a 57-year-old female with Lambert-Eaton myasthenic syndrome (LEMS) undergoing an elective laparoscopic sleeve gastrectomy. LEMS is a rare auto-immune disorder affecting the neuromuscular junction (NMJ) with symptoms consisting of muscle weakness and fatigue, as well as autonomic dysfunction.[1,2] Specifically, patients with this disease have IgG mediated autoantibodies targeted against the pre-synaptic voltage-gated calcium channels, leading to a decrease in acetylcholine (Ach) release at the NMJ.[1,3] Described in this case report is the anesthetic approach to avoid residual neuromuscular blockade, as well as other concerns, in patients with LEMS.

Case report

A 58-year-old female patient presented for elective laparoscopic sleeve gastrectomy. Past medical history included LEMS, diagnosed in 2006 with ptosis and generalized weakness beginning in 2000. The patient received monthly intravenous immunoglobulin (IVIG) therapy with prior exacerbation 5 months before surgery. The patient received 3 additional doses of IVIG during the week before surgery. The patient reported previous postoperative intensive care unit (ICU) admissions requiring prolonged intubation. Other comorbidities included attention deficit disorder, multiple deep venous thromboses and pulmonary emboli requiring IVC filter, hypothyroidism, hyperparathyroidism, wheelchair dependence, lymphedema, generalized weakness, arthritis, and morbid obesity. Her medication regimen included dextroamphetamine-amphetamine, levothyroxine, furosemide, and IVIG.

The patient's cardiac functional status in metabolic equivalents was <2. Her physical exam was unremarkable, except for morbid obesity and generalized weakness. Pre-surgical chest x-ray revealed low lung volumes, with no evidence of other pulmonary abnormalities.

The patient took levothyroxine the morning of surgery. While in preoperative holding no anxiolytics or opioids were administered. Once in the operating room (OR), standard monitors were applied and mask preoxygenation at 10 L/min was provided. Three minutes later, general anesthesia was induced with remifentanil 150 mcg, lidocaine 100 mg, propofol 150 mg, and ketamine 25 mg. Volatile anesthetics and neuromuscular blockers were avoided.

Direct laryngoscopy and tracheal intubation was performed, and an additional dose of propofol 20mg administered. Respiration was controlled with mechanical ventilation and a mixture of O_2 1 L/min and air 1 L/min. General anesthesia was maintained with propofol 150 mcg/kg/min and remifentanil 0.1 mcg/mg/min. A phenylephrine infusion was started at 0.05 mcg/kg/min and titrated as needed to maintain mean arterial pressure >65 mmHg.

After intubation, the patient received a transversus abdominis plane (TAP) block with 0.5% ropivacaine 60 mL. An additional dose of ketamine 15 mg was given approximately one hour after induction of anesthesia.

Throughout the procedure plasmalyte 1L was infused, estimated blood loss was 20 mL, and the patient remained hemodynamically stable. Acetaminophen 1 g and ketorolac 30 mg were given before emergence from anesthesia. The propofol and remifentanil infusions were discontinued simultaneously with dressing application. Nine minutes later the patient opened her eyes and demonstrated purposeful movement. The patient was spontaneously breathing 8-10/per minute, achieving tidal volumes of 400 mL, maintaining SpO2 >97%, and following commands. Successful tracheal extubation occurred and the patient was transferred to the post-anesthesia care unit. The immediate postoperative course was unremarkable.

Discussion

Lambert-Eaton myasthenic syndrome is a rare auto-immune disorder of the NMJ caused by antibodies formed against the pre-synaptic P/Q type calcium channels.[1,4] The IgG antibodies decrease the amount calcium entering the nerve endings leading to a reduced amount of Ach at the NMJ.[1,5] Approximately half of patients with LEMS also have small-cell lung cancer.[2,4] LEMS differs from Myasthenia Gravis (MG) in that muscular strength increases with exercise, whereas MG muscular strength decreases with exercise.[1,3] The patient in this case was unable to increase strength with exercise due to wheelchair dependence and morbid obesity. Clinical features of LEMS include muscle weakness, fatigue, respiratory and autonomic dysfunction, and sensitivity to all neuromuscular blockers (NMB).[1-4] Approximately 30% of these patients have autonomic dysfunction which can lead to hemodynamic instability.[3] To counteract this effect, the patient was started on a phenylephrine infusion on induction of anesthesia.

A multitude of treatment options include 3,4-diaminopyridine 15-30 mg, pyridostigmine 30-180 mg, intravenous immune globulin (IVIG) 2 g/kg, plasmapheresis, and glucocorticoids.[1-3] This patient received monthly doses of IVIG, which has been shown to exert anti-inflammatory effects that deactivate the intracellular inflammatory cascade.[6] Although the patient was extubated in the OR and had an uneventful immediate postoperative period, they had a LEMS exacerbation in the ICU the night of surgery. Neurology consult notes revealed the patient had episodes of bradycardia, dry mouth, slurred speech, and notable ptosis. Other symptoms of an exacerbation may include muscle weakness of the trunk and proximal limb muscles.[9] The patient received an additional dose of IVIG, symptoms improved, and was sent to the medical-surgical unit the next day.

Patients with LEMS require unique perioperative considerations when receiving care by anesthesia practitioners. Postoperative respiratory failure is a significant concern for patients with neuromuscular disorders.[4] Preoperative assessment and optimization is vital, as these patients may present anesthetic challenges.[3,7] Specifically, a thorough preoperative pulmonary evaluation is imperative to assess the risk for perioperative pulmonary complications.[7] These patients also have an increased sensitivity to pre-anesthetic anxiolytics and opioids which could worsen sleep apnea and hypoventilation, leading to further respiratory compromise.[7] A preoperative course of IVIG could be beneficial in preventing a postoperative exacerbation;

however, there is minimal evidence stating this helps reduce anesthesia-related complications.[7] LEMS patients that have nocturnal hypoxemia should be further evaluated for pulmonary hypertension prior to receiving anesthesia.[7] In fact, all patients with neuromuscular disorders with suspected cardiac dysfunctions should undergo an electrocardiogram (ECG) and echocardiogram within 12 months before surgery.[7] This patient had a an ECG the morning of surgery and an echocardiogram four months before surgery.

LEMS patients have a significant sensitivity to both nondepolarizing and depolarizing NMB's.[3] It is therefore advisable to avoid these drugs when possible.[3,7] In this case, the patient was successfully intubated without the use of NMB. Muscle relaxation achieved from inhalational anesthetic agents might be acceptable since LEMS patients are not susceptible to the development of malignant hyperthermia.[3,7] Additionally, medications known to potentiate NMB should be minimized or avoided due to their mechanism of action on the NMJ.[3,7] These include certain antibiotics, antiarrhythmics, and benzodiazepines.[3]

In LEMS patients with compromised pulmonary function, regional anesthesia should be utilized and general anesthesia (GA) avoided.[7] If avoidance of GA is not possible, then TIVA with propofol and remifentanil are preferable due to their ultra-short acting mechanisms of action.[7] It is also noted the use of the Bispectral Index Monitor (BIS) should be utilized in these cases to avoid the potential of medication overdose and intraoperative awareness.[7] In this particular case, propofol, remifentanil, and the BIS were all utilized. The use of regional anesthesia in patients with neuromuscular disorders carries risks such as nerve damage and local anesthetic toxicity.[7] However, the use of regional and local anesthesia over GA presents advantages such as avoidance of certain anesthetics and postoperative pulmonary complications.[5,7] Since local anesthetic agents can block neuromuscular transmission, neuraxial anesthesia doses should be reduced.[7]

Furthermore, peripheral nerve blocks are associated with fewer side effects than epidural anesthesia and have been shown to provide similar postoperative analgesia.[7] A recent systematic review and meta-analysis concluded TAP blocks are safe and should be considered for multimodal analgesia in patients having abdominal surgery.[8] After abdominal surgery, TAP blocks are associated with a decrease in postoperative morphine requirements, a possible reduction in pain severity, and a decrease in postoperative nausea and vomiting.[8] Although the patient received a TAP block, she was started on a hydromorphone PCA postoperatively. If postoperative opioids are used the dose should be decreased. [3,7]

In summary, while patients with LEMS can pose many anesthetic challenges, the perioperative management can be uneventful if the patient is diagnosed and managed per best evidence-based practice.[1] The anesthesia implications involved in this case firmly adhered to the literature recommendations for perioperative management of patients with LEMS. If anesthesia professionals are faced with caring for patients with LEMS, it is important to understand the disease pathophysiology, obtain a thorough preoperative pulmonary exam, avoid all NMB's, and utilize total intravenous anesthesia.

References

1. Weingarten TN, Araka CN, Mogensen ME, et al. Lambert-Eaton myasthenic syndrome during anesthesia: a report of 37 patients. *J Clin Anesth.* 2014;26(8):648-53. DOI: 10.1016/j.jclinane.2014.09.009
2. Abenroth DC, Smith AG, Greenlee JE, Austin SD, Clardy SL. Lambert-Eaton myasthenic syndrome: Epidemiology and therapeutic response in the national veterans affairs population. *Muscle Nerve.* 2017;56(3):421-426. DOI: 10.1002/mus.25520
3. Romero A, Joshi GP. Neuromuscular disease and anesthesia. *Muscle Nerve.* 2013;48(3):451-60. DOI: 10.1002/mus.23817
4. Katz JA, Murphy GS. Anesthetic consideration for neuromuscular diseases. *Curr Opin Anaesthesiol*. 2017;30(3):435-440. DOI: 10.1097/ACO.0000000000000466
5. Mcswain JR, Doty JW, Wilson SH. Regional anesthesia in patients with pre-existing neurologic disease. *Curr Opin Anaesthesiol.* 2014;27(5):538-43. DOI: 10.1097/ACO.0000000000000107
6. Gilardin L, Bayry J, Kaveri SV. Intravenous immunoglobulin as clinical immune-modulating therapy. *CMAJ*. 2015;187(4):257-64. DOI: 10.1503/cmaj.130375
7. Racca F, Mongini T, Wolfler A, et al. Recommendations for anesthesia and perioperative management of patients with neuromuscular disorders. *Minerva Anestesiol.* 2013;79(4):419-33.
8. Johns N, O'neill S, Ventham NT, Barron F, Brady RR, Daniel T. Clinical effectiveness of transversus abdominis plane (TAP) block in abdominal surgery: a systematic review and meta-analysis. *Colorectal Dis.* 2012;14(10):e635-42. DOI:10.1111/j.1463-1318.2012.03104.x
9. Hiroi Y, Nakao T, Tsuchiya N, et al. Exacerbation of Lambert-Eaton myasthenic syndrome caused by an L-type Ca2+ channel antagonist. *Jpn Heart J*. 2003;44(1):139-44.

Mentor: Clay Freeman, DNP, CRNA

Management of a Postdural Puncture from Epidural Placement

Brandon Gebke, MS
Webster University

Keywords: Postdural Puncture, Cosyntropin, Epidural

Accidental dural puncture (ADP) is a potential risk of epidural anesthesia, occurring in approximately 1 out of 67 instances of epidural placement.[1] Postdural puncture headache (PDPH) occurs in up to 50% of young patients following ADP with large diameter epidural needles. Epidural blood patch (EBP) has been the mainstay treatment for PDPH. However, no clear consensus exists on how to best prevent PDPH from occurring after ADP.[2] Recent studies have shown cosynotropin may prove to be an attractive method of prevention for PDPH.

Case Report

A 29 year-old, gravida 1 para 2 female with a twin intrauterine pregnancy presented in active labor. The patient was 175 cm and 75 kg with a body mass index (BMI) of 24.5 kg/m^2. Her medication regimen consisted of a daily prenatal vitamin. The patient had a history of headaches, but no other pertinent medical or surgical history. The patient had no known drug allergies and all laboratory tests were within normal limits.

As labor progressed the patient requested an epidural be placed for pain management. After discussing possible risks and benefits of the procedure, the patient consented to proceed. A noninvasive blood pressure cuff, pulse oximeter, and electrocardiogram were placed for monitoring. A timeout was performed confirming patient identity, allergies, and type of procedure being performed. The patient was placed in the sitting position and the spinous processes were palpated to identify the L4-L5 interspace. The patient was then prepped and draped. Lidocaine 1% was administered for local anesthesia at the intended epidural needle insertion site. A 17 gauge Tuohy needle was guided between the spinous processes.

Advancement was performed by using a glass syringe filled with normal saline while continually checking for loss of resistance. Upon advancement of the epidural needle, ADP occurred as evidenced by cerebrospinal fluid (CSF) filling the glass syringe. The epidural needle was removed. The procedure was repeated at the L3-L4 interspace and loss of resistance was achieved. An epidural catheter was then threaded into the epidural space. Flow or aspiration of CSF via the epidural catheter was not observed. A test dose of 1.5% lidocaine with 1:200,000 epinephrine 3 mL was administered. Intravenous and intrathecal placement were ruled out as evidenced by no change in patient's vital signs and absence of any patient reported symptoms. An infusion of 0.2% ropivicaine with fentanyl 2mcg/ml was initiated at a rate of 10 mL/hr along with the option of bolus dosing.

Upon completion of epidural catheter placement the patient received an intravenous injection of 0.75 mg of cosyntropin reconstituted with normal saline 3 mL.[6,7] The patient was encouraged to drink plenty of fluids and instructed to inform staff if any signs of a headache were to arise. Follow-up was conducted by anesthesia twice daily for 72 hours after dural puncture. The patient never reported any symptoms of headache, light sensitivity or nausea.

Discussion

Post dural puncture headache is described as a bilateral, non-throbbing pain, usually fronto-occipital, which is aggravated in the standing position and alleviated in the supine position.[3] The signs and symptoms of PDPH usually develop within the first 24 hours after dural puncture, and result from loss of cerebrospinal fluid, traction on the cranial contents, and reflex cerebral vasodilation. Factors for increased risk of PDPH include female gender, pregnancy, younger age, use of a large gauge needle, and previous headaches.[3] This patient met all of these criteria placing her at an increased risk of developing PDPH. Further studies have shown that patients with a lower BMI are also at an increased risk of developing PDPH.[4] This patient had a BMI less than 25 kg/m ^{2}, placing her at further risk of developing PDPH after ADP. Post dural puncture headache has the potential to cause significant morbidity in the obstetric patient. Postdural puncture headache can prolong hospital stay for both mother and child, and consequently contribute to an increase in the cost of health care in the maternity ward.[3] In light of these potential complications and not being able to predict which patients will develop PDPH after dural puncture, a therapy or medication is warranted to prevent PDPH from occurring.

Epidural blood patch continues to be the optimal treatment when PDPH actually presents. However, a prophylactic EBP placed after ADP has not been shown to be effective at preventing a PDPH.[5] Other conservative measures such as hydration and bed rest have a history of being ineffective in preventing PDPH.[3] Alternatively, studies have been conducted showing success of

using adrenocorticotrophic hormone (ACTH) or its analogues, such as cosyntropin, to treat PDPH.[6] There is also evidence suggesting cosynotropin may be effective as a preventative measure for PDPH.[7] In light of current literature and the patient's increased likelihood of developing PDPH, cosyntropin was administered to the parturient following dural puncture.

Cosyntropin mimics ACTH and when released stimulates the adrenal cortex to secrete both mineralocorticoids and glucocorticoids. It has been proposed that ACTH stimulates the release of aldosterone, which enhances salt and water retention and affects an expansion of blood volume. This could favor the closure of the dural tear by inducing dural edema or by simple overlap of the edges of the dural hole. Other proposed mechanisms are an increase in cerebrospinal fluid production involving active transport of sodium ions or an increase in brain endorphins that could modulate the perception of pain. Since cosyntropin releases glucocorticoids it also provides an anti-inflammatory effect that is postulated to have an effect on preventing PDPH as well.[3]

Due to dose variation in literature more studies need to be conducted to further evaluate the effective dose of cosyntropin. A study performed Hanling and colleagues chose a dose of 0.5 mg diluted in 1,000 mL of normal saline as a treatment for PDPH. Furthermore, in a study by Hakim in 2010 patients received 1.0 mg reconstituted with 4 mL of normal saline intravenously for prophylaxis of PDPH.[7] The patient in this case study received 0.75 mg of cosyntropin. In this case a higher dose was chosen assuming it would be more likely to demonstrate an effect. Since the incidence of PDPH is 50% after ADP it is possible that this patient fell into the category of patients who do not develop a headache. Furthermore, the patient was instructed to take in extra fluids and to stay in bed when possible which could have decreased the risk of PDPH. Overall multiple factors could have aided in the prevention of PDPH in this patient. However, given this patients multiple risk factors and current evidence on the effectiveness of cosyntropin, it is hypothesized that cosyntropin may have played a role in the prevention of PDPH.

References

1. Choi P, Galinski S, Takeuchi L, Lucas S, Tamayo C, Jadad A. PDPH is a common complication of neuraxial blockade in parturients: a meta-analysis of obstetrical studies. *Can J Anaesth*. 2003;50(5):460-9.
2. Apfel C, Saxena A, Cakmakkaya O, Gaiser R, George E, Radke O. Prevention of postdural puncture headache after accidental dural puncture: a quantitative systematic review. *Br J Anaesth.* 2010;105(3):255-63. doi:10.1093/bja/aeq191
3. Macaruthur A. Postpartum headache. In Chestnut D, Wong C, Tsen L, Eds. *Chestnut's Obstetric Anesthesia: Principles and Practice.* 5th ed. Philadelphia, PA: Elsevier/Saunders; 2014:718-33.
4. Peralta F, Higgins N, Lange E, Wong C, McCarthy R. The relationship of body mass index with the incidence of postdural puncture headache in parturients. *Anesth Analg.* 2015;121(2):451-6. doi:10.1213/ANE.0000000000000802
5. Scavone B, Wong C, Sullivan J, Yaghmour E, Sherwani S. Efficacy of a Prophylactic Epidural Blood Patch in Preventing Post Dural Puncture Headache in Parturients after

Inadvertent Dural Puncture. *Obstetric Anesthesia Digest*, vol. 25, no. 2, 2005, pp. 70–71., doi:10.1097/00132582-200506000-00015.
6. Hanling S, Lagrew J, Colmenar D, Quiko A, Drastol C. Intravenous cosyntropin versus epidural blood patch for treatment of postdural puncture headache. *Pain Med.* 2016;17(7):1337-1342. doi: 10.1093/pm/pnw014
7. Hakim S. Cosyntropin for prophylaxis against postdural puncture headache after accidental dural puncture. *Anesthesiology*. 2010;113(2):413-20. doi:10.1097/ALN.0b013e3181dfd424

Mentor: Vicki Coopmans, PhD, CRNA

Management of Transcarotid Artery Revascularization

Kerry M. Dolan, BSN
University of Pennsylvania

Keywords: carotid stenosis, revascularization, transcarotid artery revascularization, TCAR

The surgical management of carotid stenosis using carotid artery stenting has historically been associated with an increased risk of embolic events secondary to plaque embolization.[1] This complication can be minimized, or even eliminated, with the use of devices that are protective against embolic events. Transcarotid artery revascularization (TCAR) is a novel approach in this area of vascular surgery with promising outcomes relating to a reduction in the incidence of adverse neurologic events. This approach utilizes flow reversal in conjunction with angioplasty and stenting for the management of carotid stenosis.[2] Specific anesthetic considerations are warranted to promote success in this procedure.

Case Report

A 63-year-old, 93 kg, 185 cm Caucasian male presented for transcatheter placement of intravascular carotid stents to treat left sided carotid stenosis. The patient's past medical history included chronic obstructive pulmonary disease, hypertension and hypercholesterolemia. During a cardiac workup, the patient was found to have carotid bruits with bilateral carotid stenosis on Doppler ultrasound. A computed tomography angiogram revealed complete occlusion of the proximal right internal carotid artery and 70-80% stenosis of the left proximal internal carotid artery. Left ventricular size and systolic performance were found to be normal with an ejection fraction estimated at 60-70% via echocardiogram. Chest radiography demonstrated grossly stable chronic emphysematous and interstitial lung disease. A baseline neurological exam was performed, and findings were within normal limits. Preoperative laboratory values were unremarkable. The patient's active medications included aspirin 81 mg, clopidogrel 75 mg and lisinopril 10 mg. All three medications were administered pre-operatively. Pre-operative vital signs were as follows: blood pressure 144/72 mmHg, heart rate 77/min, respiratory rate 20/min, SpO_2 99% and temperature 36.2°C. Midazolam 1 mg was administered intravenously (IV) in the preoperative area.

Upon arrival to the operating room, standard noninvasive monitors were applied. Preoxygenation was accomplished with O_2 10 L/min via facemask. An inhalational induction was performed using an expired sevoflurane concentration of 3%. A laryngeal mask airway (LMA) was placed without difficulty. General anesthesia was maintained with an expired sevoflurane concentration of 2% in a mixture of O_2 1 L/min and air 1 L/min. Spontaneous ventilation was maintained throughout the procedure. After placement of the LMA, a 20-gauge arterial catheter was inserted in the right radial artery. Prior to incision, cefazolin 2 g was administered. A baseline activated clotting time (ACT) was measured from an arterial blood sample and determined to be 131 seconds.

After the patient was prepped and draped, heparin 7,000 units was given IV, per surgeon request with the goal of attaining an ACT of 250 seconds. Additionally, glycopyrrolate 0.2 mg was administered IV for maintenance of a heart rate greater than 60/min. Five minutes after heparin was administered, another ACT was measured from an arterial blood sample and was determined to be 280 seconds. A phenylephrine infusion was initiated at 50 mcg/hr IV and was titrated throughout the procedure to maintain a systolic blood pressure greater than 140 mmHg. Fentanyl 25 mcg was administered IV prior to incision. Flow reversal from the left common carotid artery to the left femoral vein was carried out using the ENROUTE Transcarotid Neuroprotection System (Silk Road Medical, Sunnyvale, CA) for a total duration of 12 minutes. During that time period, a stent was successfully placed in the left internal carotid artery. Vital sign changes noted during this time period included an increase in blood pressure to a maximum of 192/87 mmHg, which was managed with down titration of the phenylephrine infusion, with a heart rate that remained unchanged in the range of 77-80/min.

At the conclusion of the procedure, an ACT was measured from an arterial blood sample and was determined to be 255 seconds. Subsequently, protamine 20 mg was administered IV. A repeat ACT drawn 5 minutes after protamine administration was determined to be 134 seconds. Total procedure time from incision to closure was 132 minutes. A total of 1 L of lactated ringer's was administered IV throughout the procedure. The LMA was removed without incident and immediate postoperative neurologic examination was unchanged from baseline assessment. The patient was transported to the postanesthesia care unit for postoperative monitoring with O_2 3 L/min administered via nasal cannula. Following postanesthesia care unit discharge, the patient was transferred to the cardiovascular intensive care unit for monitoring of hemodynamic and neurologic status. The patient was discharged on postoperative day one with no signs of hemodynamic or neurologic instability.

Discussion

Carotid artery disease involves narrowing of the carotid arteries due to atherosclerotic plaque formation within the vascular endothelium.[3] The primary complication related to carotid artery disease is embolization of plaque segments to the cerebral vasculature, resulting in a transient ischemic attack (TIA) or stroke. Diagnostic imaging and testing performed to confirm the existence and severity of carotid artery disease includes carotid ultrasound, computed tomographic angiography or magnetic resonance angiography.[3] Approaches to revascularization of the carotid vasculature range from conservative management to surgical intervention. Conservative management consists of pharmacological treatment with statins and antiplatelet

agents, as well as control of modifiable risk factors associated with vascular disease. Patient modifiable risk factors include hypertension, smoking and diabetes.[3] Surgical management of carotid artery disease is recommended by the American Heart Association when patients experience a cerebrovascular ischemic event as a direct result of carotid arterial stenosis greater than 50% as documented by either invasive or noninvasive imaging. Surgery is also recommended in asymptomatic patients who demonstrate greater than 70% stenosis.[3]

There are 2 major approaches to the surgical management of carotid artery disease: open carotid endarterectomy (CEA) and endovascular carotid artery stenting (CAS). While CAS offers the advantage of a minimally invasive, endovascular approach to carotid artery revascularization, numerous studies demonstrate a higher risk of long-term stroke with CAS versus CEA due to minor periprocedural strokes commonly seen with CAS.[4] Increased periprocedural stroke with CAS is attributed to inadequate embolic protection provided during traditional CAS procedures. To mitigate the risk of periprocedural stroke associated with CAS, a transcarotid artery revascularization device was developed to combine the benefits of endovascular repair with a protective device against plaque embolization. This combined approach is termed transcarotid artery revascularization (TCAR).[5] A multicenter clinical trial conducted to evaluate the adverse event rates associated with TCAR found that the stroke rate associated with the use of this approach was significantly lower than that reported of any other stenting technique.[5] Results from clinical studies have driven the increased demand for the use of such neuroprotective strategies in order to improve the safety of carotid revascularization procedures.

As the use of TCAR becomes more widespread in vascular surgery, a greater number of anesthesia providers must understand the implications of this approach for their practice. This includes an understanding of the mechanics used to achieve flow reversal, which is the central feature of the TCAR device. This device, the ENROUTE Transcarotid Neuroprotection System (Silk Road Medical Inc., Sunnyvale, CA), consists of a flow line connected on either end to two separate vascular sheaths. Food and Drug Administration approval for the ENROUTE system was obtained in May of 2015. The first sheath is placed in the common carotid artery through a small incision in the neck, while the second is placed in the ipsilateral femoral vein. Clamping of the common carotid artery proximal to the area of sheath placement results in temporary reversal of arterial blood flow from the common carotid artery through the ENROUTE device and directly into the venous sheath in the femoral vein.[5] The flow line also contains a filter that traps embolic material to prevent delivery into the venous circulation. During flow reversal, a carotid stent is deployed through the carotid sheath. Once the stent is properly placed, the clamp on the common carotid artery is released, flow reversal ceases and the arterial and venous sheaths are removed.[2]

Anesthetic considerations unique to the TCAR procedure include specific pharmacologic and hemodynamic parameters. Although either sedation or general anesthesia can be utilized to carry out the TCAR procedure, vascular surgeons are encouraged by device manufacturers to request general anesthesia in their first attempts to carry out this procedure in order to facilitate optimal surgical conditions and minimize external complicating factors while learning the technical aspects of the procedure.[5] In the case presented, the procedure was being performed for the first time by the surgeon and thus general anesthesia was employed. Preoperative considerations for patients undergoing TCAR include pharmacological management with dual-antiplatelet therapy.

The specific recommendation followed by the patient in this case, included aspirin 75-325 mg and clopidogrel 75 mg for 3 days preoperatively. Intraoperatively, systemic anticoagulation must be administered to achieve an ACT of 250 to 300 seconds prior to cannulation. In the case presented, heparin was administered resulting in an ACT of 280 seconds.[2] In order to ensure the achievement of flow reversal through the flow line, systolic blood pressure must be maintained between 140-160 mm Hg, as measured by an invasive arterial line. There is no preferred site for arterial line placement. In the case submitted, the patient's arterial pressures were continuously transduced and a phenylephrine infusion was titrated to maintain systolic blood pressure in the target range. After sheath removal, heparin is reversed to restore baseline ACT level, which was achieved through protamine administration at the close of the procedure. Postoperative pharmacologic management includes maintenance of aspirin and clopidogrel therapy.

Anesthesia practitioners play a key role in the management of patients with carotid artery disease undergoing a TCAR procedure. Knowledge and consideration of the anesthetic implications of this procedure facilitates success in this patient population.

References

1. Valentine E, Ochroch E. Anesthesia for Vascular and Endovascular Surgery. In: Barash PG, Cullen BF, Stoelting RK, CahalanMK, Stock, C, Ortega R, Sharar SR, Holt NF, eds. *Clinical Anesthesia.* 8th ed. Philadelphia, PA: Wolters Kluwer; 2017: 1077-1111.
2. Malas MB, Leal J, Kashyap V, Cambria RP, Kwolek CJ, Criado E. Technical aspects of transcarotid artery revascularization using the ENROUTE transcarotid neuroprotection and stent system. *J Vasc Surg.* 2017;*65*(3):916-920. doi:10.1016/j.jvs.2016.11.042.
3. Montorsi P, Galli S, Ravagnani PM, Roffi M. Symptomatic carotid artery disease: revascularization. *Prog Cardiovasc Dis.* 2017;59(6):601-611. doi: 10.1016/j.pcad.2017.04.002
4. Sardar P, Chatterje, S, Aronow HD, Kundu A, Ramchand P, Mukherjee D, Nairooz R, Gray WA, White CJ, Jaff MR, Rosenfield K, Giri J. Carotid artery stenting versus endarterectomy for stroke prevention. *J Am Coll Cardiol.* 2017;69(18):2266-2275. doi: 10.1016/j.jacc.2017.02.053.
5. Kwolek CJ, Jaff MR, Leal JI, Hopkins LN, Shah RM, Hanover TM, Macdonald S, Cambria RP. Results of the ROADSTER multicenter trial of transcarotid stenting with dynamic flow reversal. *J Vasc Surg*. 2015:62(5):1227-1234. doi: 10.1016/j.jvs.2015.04.460

Mentor: Lori Ann Winner, MSN, CRNA, APN-A

Anesthetic Implications for Patients Status Post Mustard Procedure

Erica Piehl, MSNA
Westminster College

Keywords: d-transposition of the great arteries, congenital heart defects, mustard procedure, atrial switch, pulmonary hypertension

According to the Centers for Disease Control, the occurrence of transposition of the great arteries in the United States is estimated at around 1,250 babies each year.[1] The first surgical procedure to correct this abnormality was the Mustard procedure, performed by Dr. Mustard in 1963.[2] The procedure was replaced by the arterial switch method developed in the late 1980s.[2] Therefore, Mustard procedure patients are now between the ages of 22 and 50 when encountered in the operating room. Anesthetic management requires an understanding of the surgical procedure prior to the arterial switch and the alterations in anatomy and physiology that result.

Case Report

A 37-year-old, 77 kg male presented to the hospital for laser lead pacemaker extraction and reimplantation of an implantable cardiac defibrillator (ICD), with superior vena cava baffle stenosis stenting. He was born with d-transposition of the great vessels and a ventricular septal defect (VSD). He underwent a Mustard procedure at 10 months of age. At 14, he developed complete heart block and had an implantation of a dual-chamber permanent pacemaker which was later replaced at the age of 27. Recently, he sustained a syncopal episode when he sat up in bed and was investigated with an EP study, which demonstrated atrial flutter and/or atrial tachycardia. He then underwent an AV node ablation for rate control and was anticoagulated. Further investigations included an echocardiogram, showing depressed systemic ventricular function. He was referred for an upgrade to an ICD device. Device replacement was performed in the electrophysiology lab; however, was not successful due to the discovery of baffle stenosis. Catheterization showed systemic ventricular pressures of 120/24 mm Hg, aortic pressure 119/76 mm Hg, pulmonary ventricle 84/7 mm Hg, left and right pulmonary arteries 44/14 mm Hg, left pulmonary artery wedge pressure mean 22 mm Hg with a large V-wave. His cardiac index was 2.29 L/min/m^2. Pressures measured in the systemic baffle were 13/16/11 mm Hg. Preoperative echocardiography indicated moderate to severe systemic ventricular dysfunction, severe mitral and tricuspid valve regurgitation, and pulmonic stenosis.

Upon arrival to the preoperative holding area, an intravenous (IV) catheter and radial arterial line were placed. Midazolam 2 mg was administered to the patient. Once transferred to the operating room, standard noninvasive monitors were placed. After denitrogenation, proper end-tidal CO_2 waveform, and an SpO_2 of 100%, general anesthesia was induced. A slow and controlled IV induction included 1% lidocaine 50 mg, etomidate 12 mg, fentanyl 100 mcg, and rocuronium 50 mg. Following successful intubation, another large bore IV was established, and epinephrine and vasopressin drips were connected to be available as needed. Prior to incision cefazolin 2 g was administered. Maintenance of anesthesia was established with sevoflurane 1.5 - 2.5% with boluses of fentanyl and rocuronium. Ventricular fibrillation occurred twice during the case. The first event was related to wire manipulation from the surgeon while attempt to guide the wire for

stenting of the baffle. The second occurred during testing of the newly implanted pacemaker, which functioned correctly converting the patient to normal sinus rhythm. During the dilation and stenting of the SVC, simultaneous TEE during balloon inflation was performed to assess the pulmonary venous channel, which displayed no further obstruction. TEE monitoring continued after deployment of the stent to monitor ventricular performance and for any evidence of compromise to the pulmonary venous channel. The anesthetic course was otherwise uneventful with the patient requiring no hemodynamic support. The patient's neuromuscular blockade was antagonized with neostigmine 5 mg and glycopyrrolate 0.8 mg. and extubation occurred without any problems. A total of 1300 mL of crystalloids was administered throughout the case. The estimated blood loss was approximately 100 mL and the urine output 600 mL, over 4 hours and 21 minutes. Recovery in the post-anesthesia care unit was uneventful.

Discussion

Transposition of the great arteries (TGA) is one of many congenital heart defects that can occur during fetal development. TGA belongs explicitly in a group of cyanotic congenital heart lesions. With this heart defect, the pulmonary artery originates from the morphological left ventricle, and the aorta originates from the morphological right ventricle.[3,4] The dextro represents the transposition of the aorta and pulmonary artery; inferring that the aorta is primarily to the right (dextro) of the pulmonary artery. The combination of concordant atrioventricular and discordant ventricular and arterial connections creates parallel circulation systems in which recirculation of oxygenated blood occurs within the pulmonary circuit via the left ventricle and pulmonary artery while deoxygenated systemic blood recirculates to the body via the right ventricle and aorta that results in systemic cyanosis.[3,4] Uncompensated TGA is fatal due to the lack of intracardiac mixing of oxygenated and deoxygenated blood. Fortunately, TGA is usually associated with other congenital heart lesions that allow for intracardiac mixing that include: VSD, atrial septum defect (ASD), patent foramen ovale (PFO), or a patent ductus arteriosus (PDA). These defects provide an opportunity for surgical correction due to the intracardiac mixing of oxygenated and deoxygenated blood.[3,4.] The patient's TGV was accompanied by a congenital VSD.

The surgical procedure performed on the case study patient to correct the d-TGA was the Mustard Procedure, also known as an atrial switch. This switch involves creating a systemic venous baffle to the left ventricle from the inferior vena cava and the superior vena cava, allowing the deoxygenated blood to travel to the pulmonary artery and lungs.[2] The oxygenated blood travels to the right atrium and ventricle to be pumped to the body. The primary problem that develops from the surgery is right ventricular failure.[2] The morphologic right ventricle becomes the systemic ventricle and is unable to maintain the pressures required for adequate systemic circulation due to the structure and arrangement of the myofibers which results in a less muscular ventricle when compared to the left ventricle. Right ventricular failure and tricuspid regurgitation ultimately result, both of which existed in the patient in this case study. Additionally, the patient presented with atrial flutter, which is commonly seen in these patients, from trauma and scarring of the sinus node.[2] Another commonly encountered complication the patient presented with was baffle stenosis due to the increased pressures the baffle must overcome. The baffle can become leaky or stenotic leading to blood clots or systemic venous hypertension.[2,4]

Anesthesia providers must be acutely aware of the new anatomy and sequela created by the Mustard procedure. Increases in pulmonary vascular resistance must be avoided during any surgical procedure. Any increases in pulmonary vascular resistant can lead to an acute ventricular failure event resulting in decreased cardiac output and rapid progression to cardiac arrest. Pulmonary hypertension can be a late complication of atrial-level repair, with an incidence reported at 7% in those patients surviving to adulthood. Pulmonary hypertension was present in this patient.[5] During the case, special focus was on prevention of hypoxia, systemic hypoxemia, metabolic acidosis, hypercarbia and sympathetic nervous system stimulation due to the light plane of anesthesia and pain. All are potential triggers that result in a rapid rise in pulmonary vascular resistance, and even pulmonary hypertensive crisis.[3] To minimize any rise in pulmonary pressures, precise ventilation strategies were used during the case. These included the use of high inspired fraction of oxygen concentrations, low tidal volumes of 6 mL/kg of the predicted body weight, a slightly elevated respiratory rate allowing mild hypocarbia, and positive end-expiratory pressure of 5 cm H_2O to mitigate atelectasis.[4] Isoflurane and sevoflurane are associated with pulmonary vasodilation and are safe to administer to patients with pulmonary hypertension.[4] Furthermore, benzodiazepines, opioids, etomidate, neuromuscular blocking agents, and propofol are regarded as safe for patients with pulmonary hypertension, and have little to no effect on pulmonary vascular resistance.[6] The clinical presentation of pulmonary hypertension does not normally manifest until adulthood, due to the left pulmonary ventricle's ability to overcome higher pressures.[7]

During this case, attention was focused on ensuring that the systemic vascular resistance was maintained within normal values, due to the right systemic ventricle failing overtime. Increases in systemic vascular resistance could potentially lead to a pulmonary hypertensive crisis. Care was taken to prevent as little alteration as possible in cardiac output, pulmonary vascular resistance, and systemic vascular resistance. Using etomidate for anesthesia induction and ensuring an adequate depth of anesthesia was achieved before direct laryngoscopy was vital. Blood pressures throughout the case were maintained within 20% of the patient's normal blood pressure by adjusting the sevoflurane, and administering fentanyl as needed. Fluid management was guided by intraoperative transesophageal echocardiogram to optimize ventricular preload while avoiding volume overload, distention, and increased tricuspid regurgitation.

Overall, the anesthesia course for this patient was smooth and uneventful. Careful consideration was taken into everything administered, recognizing the unique anatomy and clinical presentation of the patient. An arterial line was started pre-induction to help control the alterations in cardiac depression and hemodynamics. Hemodynamic pressor support was obtained by pharmacy beforehand to be adequately prepared in case any adverse events were encountered during the surgery. Due to careful anesthetic planning, all potential outcomes that could occur were addressed. The patient had an uneventful anesthetic course and recovered well. Mustard patients are becoming increasingly uncommon. However, anesthesia providers must always be prepared to treat patients with congenitally corrected d-TGA to provide safe research-based anesthesia.

References

1. Parker SE, Mai CT, Canfield MA, et al. for the National Birth Defects Prevention Network. Updated national birth prevalence estimates for selected birth defects in the United States, 2004-2006. *Birth Defects Res A Clin Mol Teratol*. 2010;88:1008-1016.
2. Marathe SP, Talwar S. Surgery for transposition of great arteries: A historical perspective. *Ann Pediatr Cardiol*. 2015;8(2):122–128. doi:10.4103/0974-2069.157025
3. Gupta S, Saiyed A, Meena R, Dogra N. Anaesthetic Management during Transposition of Great Arteries (TGA) Correction: Points to be Focused. *Br J Med Med Res*. 2017;19(8):1-5. doi:10.9734/bjmmr/2017/30290.
4. Nicole Elsey N, Tobias JD. Perioperative management following an atrial level switch (Mustard or Senning procedure) for transposition of the great arteries (Review Article). *Anaesth Pain & Intensive Care*. 2011;15(3):185-194.
5. Love BA, Mehta D, Fuster VF. Evaluation and management of the adult patient with transposition of the great arteries following atrial-level (Senning or Mustard) repair. *Nat Clin Pract Cardiovasc Med*. 2008;5:454-67.
6. Rieker, M. Anesthesia for Thoracic Surgery. In Nagelhout JJ, Plaus KL, eds. Nurse Anesthesia. 6th ed. St. Louis: Saunders Elsevier; 2014:597.
7. Ebenroth ES, Hurwitz RA, Cordes TM. Late onset of pulmonary hypertension after successful mustard surgery for d-transposition of the great arteries. *Am J Cardiol*. 2000; 85(1):127–130. A10.

Mentor: Mandarie Shimata, DNAP, CRNA

Anesthesia for Suboccipital Craniotomy and Medulloblastoma Resection

Paul Nicholson, MSNA, MS
Westminster College

Keywords: Craniotomy, EVD, suboccipital, park bench position, medulloblastoma.

Craniotomy for tumor resection is a routine procedure performed at hospitals across the United States. Posterior fossa tumors, arising within the cerebellum or brainstem, such as medulloblastoma, often cause obstructive hydrocephalus.[1] Medulloblastoma is quite rare with only 500 children diagnosed each year in the United States.[2] It occurs exclusively in the cerebellum, affecting children 10 times more often than adults.[2] Hydrocephalus increases intracranial pressure (ICP) and may complicate the clinical picture. External ventricular drains (EVD) or other decompressive devices are often used to treat hydrocephalus. Anesthesia practitioners must balance these unique challenges while providing care for such complex neurosurgical patients.

Case Report

A 26-year-old, 82 kg, 153 cm gravida 4, para 3 female at 34 weeks gestation presented with a cerebellar mass, for a sub-occipital craniotomy. When she arrived at the hospital, she was

complaining of lethargy, headache, and weakness for the past 15 days. Computed tomography (CT) and magnetic resonance imaging (MRI) confirmed a mass within the posterior fossa causing compression of the fourth ventricle and pons resulting in obstructive hydrocephalus. After admission, she had a successful cesarean section delivery of a male neonate and placement of an external ventricular drain (EVD). She was then transferred to the neuro intensive care unit (ICU) and presented for a sub-occipital craniotomy on post-partum day 2. Additional medical history included Bell's palsy. Her surgical history was only significant for the cesarean section under general anesthesia, after which she remained intubated in the neuro ICU.

Her hospital intravenous (IV) medication regime included propofol 25mcg/kg/min, fentanyl 100 mcg/hr, oxytocin 42 units/min, 2% hypertonic saline 60 ml/hr, and Metronidazole 500 mg every 12 hours. A preoperative echocardiogram showed normal heart function with an ejection fraction of 65%. Preoperative laboratory values were normal with the exception of:, RBC 2.8 M/UL, hemoglobin 7.9 g/dL, hematocrit 23.4%, platelets 116 K/UL, sodium 133 mEq/L, and serum osmolality 279 mOs/kg. The oxytocin infusion was discontinued in an effort to increase her serum sodium. She received midazolam 2 mg IV, and the 2% hypertonic saline at 60 ml/hr were continued into the operating room (OR) suite, and the EVD was placed at 15cm H2O. Care was taken to monitor and maintain the patient's endotracheal tube, right radial arterial line, and right subclavian triple lumen catheter. All other IV medications were discontinued prior to transfer from the ICU to the OR.

Standard noninvasive monitors were applied upon arrival to the OR. Anesthesia was induced using propofol 150mg, remifentanil at 0.2 mcg/kg/minute, and rocuronium 50 mg IV. The patient was given cefazolin 2 g and metronidazole 500 mg IV for infection prophylaxis. After induction, mechanical ventilation was maintained with volume control, and sevoflurane at 1.8% expired concentration, in a mixture of air and oxygen. An intravenous infusion of norepinephrine at 2mcg/minute was initiated to maintain a mean arterial pressure of at least 65 mm Hg. An esophageal temperature probe was inserted, a lower body forced-air warmer was used, and a second peripheral intravenous catheter was placed in the patient's foot. The patient was positioned into park-bench position on the left lateral side, and the patient's head was placed in the Mayfield, head holder. The park-bench position differs from the traditional beach-chair position for neurosurgery because it keeps the head at the level of the heart, rather than elevating the head above the heart. The posterior fossa was then accessed, and surgical resection of the tumor began.

After about five hours of procedure time, the posterior fossa tumor was successfully resected and sent to pathology for identification. The EVD was also removed. Estimated blood loss totaled to 400 mL, and 3 units of packed red blood cells were administered to the patient as guided by a transfusion trigger of hemoglobin less than 7 g/dL. Intraoperative point of care arterial blood gas measurements were done hourly to assess hemoglobin and hematocrit. The remifentanil infusion was discontinued, and a nicardipine infusion at 10 mg/hr was initiated near the end of the procedure to maintain systolic blood pressures below 140 mm Hg. The patient emerged from anesthesia uneventfully. The patient was extubated and transferred to the post-anesthesia care unit (PACU) for continued care and monitoring. No neurological deficits were noted. Pathology determined the tumor to be a medulloblastoma.

Discussion

Tumors of the posterior fossa, such as medulloblastoma, often lead to increased ICP and hydrocephalus because the posterior fossa is a rigid compartment with poor compliance.[1,3] The posterior fossa contains the cerebellum, the brainstem, and the lower cranial nerves, all of which may be affected by increased pressure with the various sequela.[3] Anesthetic management of hydrocephalus is directed at controlling ICP and relieving the obstruction.[1]

Management of this case included an oxytocin infusion used to augment uterine contraction and reduce post-partum blood loss. Because oxytocin is structurally similar to vasopressin, it is known to have an anti-diuretic like effect.[4] Prolonged infusion may result in water retention and hyponatremia.[3] Because hyponatremia is associated with increased ICP, and poor outcomes in neurosurgical patients, hyponatremia should be avoided. In the current case study, oxytocin was discontinued due to the risk of causing hyponatremia.

Another concern for this particular surgery is a severe complication known as venous air embolism (VAE). A venous air embolism is of particular concern in all surgeries in the head-up position and is most associated with neurosurgery performed in the sitting, or beach-chair position.[3] Because the surgical site is above the level of the heart, venous pressure at the surgical wound can be less than atmospheric pressure and may result in air entrainment into the venous system. The incidence of VAE varies from 25-76% in sitting position surgeries.[3,5] In comparison, neurosurgical positions that maintain the head at the same level as the heart, such as park-bench or prone positioning have a much lower incidence of VAE at 11-17%. [6]

One well-described treatment option of VAE is known as the Durant Maneuver.[3] The patient is positioned left side down, and in steep Trendelenburg in order position the air-lock within the right ventricle away from the outflow tract (pulmonic valve).[3] One possible additional advantage of the park bench position is that it places the patient in the left lateral position, such that if VAE were to occur, the anesthesia practitioner could more easily and quickly position the patient for the Durant Maneuver.

Sitting positions facilitate cerebral blood and cerebrospinal fluid (CSF) drainage due to gravity and thus help to reduce ICP.[1] Therefore, potential disadvantages of horizontal positions such as prone, and park-bench is that ICP must be reduced via mechanisms other than blood or CSF drainage. The use of the park bench position and the associated advantages were deemed to outweigh potential disadvantages in the current case study.

Trigeminocardiac reflex (TCR) is defined as the onset of sudden hypotension, parasympathetic dysrhythmias, apnea, or gastric hyper-motility during stimulation of any of the sensory branches of the trigeminal nerve.[7] The oculocardiac reflex (OCR) is a more well-known sub-variant of TCR. Just as stimulation of the ophthalmic division of the trigeminal nerve causes bradycardia in the OCR, stimulation of any branch of the trigeminal nerve may cause vagal stimulation via the same reflex arc.[7] Posterior fossa neurosurgery is particularly capable of stimulating the TCR because of the surgical proximity to the trigeminal and vagus nerves. One retrospective study by Schaller et al. found the occurrence of TCR to be 11% in those undergoing cerebellar tumor resection.[8] Treatment options for TCR include cessation of the stimulus, administration of

vagolytic agents or sympathomimetics such as atropine, glycopyrrolate, or epinephrine.[7] Anesthesia practitioners must be quick in the identification and treatment of TCR. The anesthesia practitioner prepared intravenous atropine in case of TCR in this particular case study.

Finally, postoperative nausea and vomiting (PONV) is a risk of any surgery. However, posterior fossa neurosurgery poses a particular risk of PONV due to the surgical site proximity to the vomiting center.[3] The use of opioids to control pain associated with neurosurgery further increases the risk of PONV. Dexamethasone is particularly useful in neurosurgery because it reduces cerebral edema as well as helping prevent PONV.[3] Post-operative vomiting may also increase blood pressure, ICP, postoperative bleeding; thus prevention should be paramount.

References

1. Pasternak J, Lanier W. Diseases affecting the brain. In: Hines RH, Marschall KE. *Stoelting's Anesthesia and Co-existing Disease.* 7th ed. Philadelphia, PA: Elsevier Saunders; 2017.
2. Pomeroy S. Clinical presentation, diagnosis, and risk stratification of medulloblastoma. In: Eichler A, ed. *UpToDate.* Waltham, Mass.: UpToDate, 2018. https://www.uptodate.com/contents/clinical-presentation-diagnosis-and-risk-stratification-of-medulloblastoma
3. Jagannathan S, Hari K. Anaesthetic considerations for posterior fossa surgery. *Continuing Ed in Anaesthesia, Critical Care and Pain*. 2014;4(5):202-206.
4. Ufuk E, Gulay K, Aysun U, Tugrul A. Case report: hyponatremia and generalized convulsion after oxytocin infusion. *J Turkish-German Gynecol Assoc.* 2009; 10: 47-48.
5. Spence N, Faloba K, Sonabend A, et al. Venous air embolus during scalp incision. *J of Clinical Neuroscience.* 2016; 28: 170-171.
6. Rushatamukayanunt P, Seanho P, Muangman S, et al. Severe venous air embolism related to positioning in posterior cranial fossa surgery in Siriraj Hospital. *J Med Assoc Thai.* 2016; 99(5): 511-516.
7. Arasho B, Sandu N, Spiriev T, et al. Management of the trigeminocardiac reflex: facts and own experience. *Neurol India.* 2009; 57: 375-380.
8. Schaller B, Probst R, Strebel S, Gratzl O. Trigeminocardiac reflex during surgery in the cerebellopontine angle. *J Neurosurg.* 1999; 90: 215-20.

Mentor: Mandarie Shimata, DNAP, CRNA

Spinal versus General Anesthesia for Hip Fracture Repair

Nicole Higgins, DNAP, BSN
National University

Keywords: spinal, general anesthesia, hip fracture, perioperative morbidity, perioperative mortality, geriatric surgery

Broken hips are a significant cause of mortality in the geriatric population. Over 250,000 incidences of hip fractures affect adults in the U.S. annually.[1] Of these patients, 4-14% will die within 30 days of the injury.[2] Although general anesthesia remains the most common method of anesthesia for repair of hip fractures, the evidence for spinal anesthesia has shown to be effective

for this procedure. The evidence of its superiority, however, remains in question. This case report describes a geriatric hip fracture patient and investigates the literature of whether the anesthetic affects morbidity and mortality in the 30-day post-operative period.

Case Report

The patient was a 90-year-old woman, who weighed 43.4 kg, and was 152 cm tall. Past medical history was significant for dementia, hypertension, congestive heart failure (ejection fraction 50%), hyperlipidemia, osteoarthritis, osteoporosis, and type 2 diabetes mellitus. She appeared malnourished, dehydrated, and with poor dentition. Her neck had a limited range of motion and she had a small mouth opening. A complete airway assessment was unable to be performed due to the patient's dementia and limited cooperation. Daily home medications included lisinopril 20 mg, metformin 500 mg twice daily, glipizide 2.5 mg, aspirin 81 mg daily, donezepil 5 mg, galantamine 10 mg twice daily, and alendronate 10 mg. The last dose of aspirin was given 3 days before the operation. Coagulation laboratory values were within normal limits, and her hemoglobin was 9.8 mg/dL. Two units of packed red blood cells were typed and cross matched in case transfusion was necessary. Platelets were 208,000/μL.

Once the patient was transported into the operating theater, noninvasive monitors were placed on the patient. Baseline vital signs were within normal limits. Oxygen 6 L/min was delivered via simple face mask. A liter of ringer's lactate was infusing through a 22-gauge access catheter in the patient's wrist. Ketamine 50 mg was delivered intravenously, and the patient was turned onto the affected hip in the left lateral decubitus position one minute after ketamine administration and she tolerated laying on the broken hip. While in the lateral position, using aseptic technique, a spinal anesthetic was delivered between the L2 and L3 interspace using a 25g Whitacre needle. Only one attempt was necessary, and 0.75% bupivacaine 12.5 mg was administered intrathecally. The patient continued to lay on the affected hip to allow the hyperbaric solution to settle onto the nerve roots. The patient was then repositioned supine, prepped, and draped for surgery.

Additional intravenous access was gained by placing a 20-gauge catheter in the patient's left forearm and another liter of ringer's lactate was administered as a slow infusion. Seven minutes before incision, a propofol infusion was initiated at 25 mcg/kg/h. Phenylephrine was administered in 100 mcg boluses prior to surgery start and three times after it began. Fifteen minutes into the surgery, a phenylephrine infusion was initiated at 25 mcg/min. It was discontinued at the end of the procedure. Ephedrine 50 mg was administered intramuscularly 10 minutes prior to the end of the surgery and the patient was uneventfully transferred to the post anesthesia care unit.

Discussion

The question researched most often in the literature asks which method of anesthesia has the greatest effect on morbidity and mortality. Several studies found that there is no difference in thirty-day mortality rates.[2-4] The differences lie is in the adverse events that happen in the immediate post-operative period. Some studies found differences in length of stay with different methods favored in each study.

A meta-analysis comparing general and spinal anesthesia found no difference in thirty-day mortality but did find that in-hospital mortality related to adverse events was higher in patients receiving general anesthesia, specifically rates of myocardial infarction and respiratory failure. They also found a shorter length of stay for the spinal block patients, though the authors concluded that because of selection bias and other limitations that neither anesthetic had superior perioperative outcomes over the other.[2]

A large retrospective study of 55,000 patients and over 7 years found that there was no significant difference in thirty-day mortality between general anesthesia and spinal blocks. The authors did find there was a modest difference in the length of stay which favored the spinal block patients.[4]

A study conducted by the American College of Surgeons National Safety Quality and Improvement Program (ACS-NSQUIP) that studied short term complications in hip fracture surgery found that subarachnoid blocks were favorable compared to general anesthesia. Rates of urinary tract infection, deep vein thrombosis, and blood transfusion were lower in patients receiving spinal blocks. They also had shorter operating room times, though this is likely related to extubation post-operatively.[5]

Another retrospective cohort study that was conducted utilizing the ACS-NSQUIP database compared adverse events related to the two anesthesia types. They found that general anesthesia is associated with higher rates of blood transfusion, embolic events like deep vein thrombosis and pulmonary embolism, but that spinal blocks were associated with higher rates of urinary tract infections, pneumonia, and longer lengths of stay in the hospital.[3]

Another study that compared cost between general anesthesia and spinal blocks found that spinals are cheaper, likely due to equipment, volatile agent, and drug costs associated with general anesthesia. Costs savings ranged from 3.2-5.3% of total OR cost, or 1.3% per case.[6]

Stress response has been studied as well. This study divided patient groups into general anesthesia, neuraxial anesthesia, and regional blocks. It measured levels of cortisol, thyroid hormones, insulin, glucose, and C-reactive proteins pre-operatively, and 4, 12, and 24 hours post-operatively. Patients receiving general anesthesia had the highest levels of glucose and cortisol at the 4-hour mark. They found that groups that received regional or neuraxial blocks had significantly lower levels of cortisol at the 4-hour mark, and those that had a catheter with local anesthetic infusion had still significantly lower cortisol levels at the 12-hour mark as well. The authors concluded overall that the use of regional anesthesia diminishes the surgical stress response.[7]

Another technique to consider is the use of combined spinal and epidural (CSE). One study utilized CSE and compared it to a spinal block. In this study, patients receiving CSE were given 1 mL of 0.5% hyperbaric bupivacaine with 25 mcg of fentanyl and an epidural catheter was dosed with increments of 1-1.5 mL of isobaric 0.5% bupivacaine to achieve a T10 sensory block. Patients receiving just the spinal block were given 2.5 mL of 0.5% hyperbaric bupivacaine with 25 mcg of fentanyl. Patients in the CSE group had substantially lower rates of hypotension and need for vasopressors (6.7% versus 67%) and lower rates of bradycardia (6.7% versus 30%).

Patients receiving the spinal alone had a sensory block up to T6, whereas the combined group remained at a T10 block.[8]

In light of the evidence, it is reasonable to conclude that one method of anesthesia is not favorable over the other, however in older patients with cardiac or pulmonary compromise neuraxial anesthesia may be a safer option. The rates of blood transfusion and embolic events are lower in patients with neuraxial block patients, however it is unclear if rates of urinary tract infections are affected by the anesthetic. Given the age of the patient population most affected by this type of injury, it is prudent to consider the CSE technique because of the stability in hemodynamics with this technique. More research is necessary to come to any definitive conclusions about which method of anesthesia is superior, but for now considering the individual patient, their comorbidities, and the unique circumstances surrounding their injury remains the best course of action to decide the most suitable method of anesthesia.

References

1. Neuman, Mark D. (2016). Pilot Randomized Controlled Trial of Spinal Versus General Anesthesia for Hip Fracture Surgery. *Journal of the American Geriatrics Society.,64*(12), 2604.
2. Van Waesberghe J, Stevanovic A, Rossaint R, Coburn M. General vs. neuraxial anaesthesia in hip fracture patients: a systematic review and meta-analysis. *BMC Anesthesiology*. 2017;17:1-25. doi:10.1186/s12871-017-0380-9.
3. Basques BA, Bohl DD, Golinvaux NS, Samuel AM, Grauer JG. General versus spinal anaesthesia for patients aged 70 years and older with a fracture of the hip. *Bone & Joint Journal*. 2015;97-B(5):689-695. doi:10.1302/0301-620X.97B5.35042.
4. Neuman MD, Rosenbaum PR, Ludwig JM, Zubizarreta JR, Silber JH. Anesthesia technique, mortality, and length of stay after hip fracture surgery. *JAMA: Journal of the American Medical Association*. 2014;311(24):2508-2517. doi:10.1001/jama.2014.6499.
5. Fields AC, Dieterich JD, Buterbaugh K, Moucha CS. Short-term complications in hip fracture surgery using spinal versus general anaesthesia. *Injury*. 2015;46(4):719-723. doi:10.1016/j.injury.2015.02.002.
6. Chakladar A, White SM. Cost estimates of spinal versus general anaesthesia for fractured neck of femur surgery. *Anaesthesia*. 2010;65(8):810-814. doi:10.1111/j.1365-2044.2010.06382.x.
7. Kendrišić M, Šurbatović M, Djordjević D, Jevdjić J. Surgical stress response following hip arthroplasty regarding choice of anesthesia and postoperative analgesia. *Vojnosanitetski Pregled: Military Medical & Pharmaceutical Journal of Serbia*. 2017;74(12):1162-1169. doi:10.2298/VSP160416153K.
8. Tummala V, Rao LN, Vallury MK, Sanapala A. A comparative study-efficacy and safety of combined spinal epidural anesthesia versus spinal anesthesia in high-risk geriatric patients for surgeries around the hip joint. *Anesth Essays Res*. 2015;9(2):185-8.

Mentor: Jennifer A. Debban, MS, CRNA

Anesthesia Management of the Bleomycin Treated Patient

Deonna N. Carlson, MS
Midwestern University

Keywords: Bleomycin, pulmonary toxicity, chemotherapy, anesthesia

Testicular cancer is often treated with a radical orchiectomy and chemotherapy using bleomycin, etoposide, and cisplatin (BEP) as the cornerstone treatment.[1] Nearly half of testicular cancer patients will undergo some reduced form of chemotherapy, but long-term patient survival remains very high. Multiple complications from patient management stem from the cytotoxic nature of bleomycin. Bleomycin-induced lung injuries occur in 10% of patients and can further complicate their medical condition. Roughly 3% of those who develop lung injuries can develop severe interstitial pneumonia.[2] Bleomycin causes damage by forming a complex with iron and oxygen, which creates free radicals that destroy deoxyribonucleic acid (DNA) resulting in pulmonary toxicity and fibrosis. Improved anesthesia management and treatment of these patients is key to positive patient outcome, as inappropriate care can exacerbate bleomycin-related complications.

Case Report

A 27-year-old, 80 kg male presented for a retroperitoneal lymph node dissection. The patient had a history of a left radical orchiectomy and completed four cycles of BEP therapy 2 months prior to presenting for surgery. The patient had no drug allergies, no other significant medical history and was otherwise in good health. Laboratory values were unremarkable, and chest computed tomography showed clear lungs with no metastasis in the surrounding structures. No pulmonary function tests were available but the patient reported no shortness of breath and scored > 4 metabolic equivalents (METS). Physical examination exhibited clear breath sounds with no crackles present. The anesthesia plan of general endotracheal intubation was discussed and agreed upon with the patient and surgeon.

The patient received midazolam 2 mg intravenously (IV) preoperatively. Once in the operating room, standard monitors were applied, and the patient was only administered air (~21% O_2) prior to induction. Oxygen saturations began in the high 90s while preparation for induction was completed. An IV induction with cricoid pressure was conducted using lidocaine 1 mg/kg, sufentanil citrate 2 mcg/kg, propofol 2 mg/kg, and succinylcholine 1 mg/kg IV. Direct laryngoscopy with a Miller 2 blade revealed a grade 2 view resulting in successful placement of a cuffed 7.5 mm endotracheal tube (ETT) on the initial attempt. After induction, a right arterial line was placed as well as a left subclavian central line under sterile technique. General anesthesia was maintained with 2% inspired sevoflurane in air 2 L/min. Inspired O_2 was maintained at 21% throughout the procedure. The patient was placed on mechanical ventilation and once train-of-four (TOF) was established, neuromuscular blockade was maintained with rocuronium with a one to two twitch titration intraoperatively. Pain was managed during surgery with sufentanil citrate 0.5 mcg/kg boluses, not to exceed a total of 1 mcg/kg/hr.

Prior to extubating, a transversus abdominis plane (TAP) block was placed using sterile technique with ultrasound by instilling dexamethasone 4 mg in 30 mL ropivacaine 0.25% bilaterally to facilitate postoperative deep breathing. After return of spontaneous ventilation, the patient's neuromuscular blockade was antagonized by administering neostigmine concurrently with glycopyrrolate. After adequate tidal volumes, sustained tetanus, and regular ventilation rate were established, the ETT was removed and the patient transported to the post anesthesia care unit with no significant events.

Discussion

Retroperitoneal lymph node dissection has both a diagnostic and therapeutic role. In higher-grade cancers it is offered to patients who have residual masses following combination BEP therapy.[3] It is important for anesthesia personnel to note the pulmonary risk bleomycin imposes. Bleomycin is inactivated by bleomycin hydrolase but because this enzyme is not found in the lung and skin, bleomycin can lead to toxicity in these areas. In normal metabolic breakdown, inactivated bleomycin is nontoxic. However, in forming an iron and oxygen complex, bleomycin exacerbates respiratory failure by increasing superoxide release by neutrophils in response to stimuli.[4] When superoxide radicals interact with cells, especially pneumocytes, trademark bleomycin toxicity is apparent.

Risk factors that increase pulmonary toxicity include high inspired oxygen fraction, patients older than 70-years-old due to impaired kidney excretion, and fluid overload. Bleomycin has also been known to cause spontaneous pneumomediastinum.[4] Complications often present with dyspnea, dry cough, tachypnea and cyanosis. In addition, radiographic presentation is seen to have bibasilar reticular or fine nodular infiltrates causing crackles upon auscultation. Differential diagnoses include metastasis or infection pneumonitis. However, in bleomycin toxicity there is a decreased functional vital capacity and less effect on the transfer capacity of the lungs for carbon monoxide (TLCO) in comparison to what would be seen with either differential diagnoses.[4] Treatment includes steroidal therapy and intubation as necessary.[2]

Suggestions for best care range greatly within the literature. This is essential when choosing mode of anesthesia and pain management control. Concerning ventilation status, reduced oxygen concentrations should be used while maintaining peripheral oxygen saturation between 88%-92% (SpO_2) with positive end-expiratory pressure.[5]

It is important to note that most chemotherapy drugs are metabolized in the liver and thus regional anesthesia may be contraindicated in cases of associated coagulopathy.[5] However, regional technique may influence cancer recurrence with lidocaine and bupivacaine inhibiting transcription pathways often linked to metastasis and cell proliferation.[5] Regional anesthesia provides reduced opioid associated side effects, inflammatory response, and immune suppression postoperatively. It has been found that local anesthetics augment the natural killer cell activity while volatile agents can increase risk of metastasis.[5]

Non-volatiles such as nitrous oxide and intravenous agents are now being studied in cancer recurrence. Low-dose ketamine has been associated with immune suppression and increased risk of cancer recurrence while propofol may have an anticancer effect. This mechanism is thought to

be related to inhibiting tumor size and angiogenesis of cancer and inducing cell apoptosis.[5] Furthermore, when researching prophylactic management, it has been found that steroids prior to surgery may bolster against pulmonary toxicity.[6] It has been studied how both methylprednisolone succinate and Nigella sativa have an effect on pulmonary diseases resulting in decreased pulmonary inflammation and fibrosis with treatment.[6]

Considering the literature review, there are several factors that could have offered an improved anesthetic plan. Concerning the fact that no pulmonary function tests were available preoperatively, it would have been better to have had these completed prior to the surgery for baseline values. However, the patient presented with > 4 METS and otherwise insignificant medical history. Additionally, administering an intraoperative steroid stress dose of methylprednisolone to decrease pulmonary inflammation and address potential adrenal suppression should have been implemented into the anesthetic plan.[2] This is the same steroid used in the event of pulmonary toxicity complication with the bleomycin patient. Also, the concept that propofol may have an anticancer effect should be further studied, whichmay lead to future implementation of total intravenous venous anesthesia and avoidance of all volatiles in these patients. Overall, oxygen concentration was kept at 21% with satisfactory SpO_2 and fluid management was maintained judiciously. The TAP block was successful on follow up to foster deep breathing and the use of incentive spirometer.

Depending on the environment in which anesthesia professionals practice, particularities of oncology anesthesia is important for anesthesia personnel to recognize the differing risk factors chemotherapy drugs place on the patient. In recognizing these risks and optimization of perioperative care, the decisions by anesthesia personnel can ultimately mitigate perioperative morbidity and mortality. It is vital to consider the effect each anesthetic plan may have on the immediate and long-term disease process. It is suggested to further study the different modes in which prophylaxis, intraoperative. and postoperative care would best optimize the patient for better recovery and quality of life.

References

1. Karim S, Wei X, Leveridge M, et al. Delivery of chemotherapy for testicular cancer in routine practice: A population-based study. *Urol Oncol.* 2018;18:1078-1439.
2. Biva J, Stoclin A, Dury S, et al. Consortium for detection and management of lung damage induced by bleomycin. *Cancer Bull*. 2016;103(7):651-661.
3. Farquhar-Smith P, Wigmore T. *Anaesthesia, Intensive Care, and Pain Management for the Cancer Patient*. Oxford, NY: Oxford University Press; 2011.
4. Frusch N, Servais S, De Prijck B, et al. Spontaneous Pneumomediastium Caused By Bleomycin-Induced Pneumonitis. *Acta Clinica Belg*. 2012;65(5):370-371.
5. Gudaitytė J, Dvylys D, Šimeliūnaitė I. Anaesthetic challenges in cancer patients: current therapies and pain management. *Acta Med Litu*. 2017;24(2):121-127.
6. Poursalehi HR, Samareh Fekri M, Sharifi Far F, et al. Early and late preventive effect of *Nigella sativa* on the bleomycin-induced pulmonary fibrosis in rats: An experimental study. *Avicenna J Phytomed*. 2018;8(3):263–275.

Mentor: Lee Ranalli, DNP, CRNA

Pericapsular Nerve Group block in the Total Hip Arthroplasty Patient

Jacob Wolf, DNAP, BSN
National Univeristy

Keywords: PENG block, Peripheral nerve block, fascia iliaca nerve block, femoral nerve block, 3-in-1 block, total hip arthroplasty, THA, opioid sparing, post-operative recovery

The fascia iliaca nerve block (FIB), femoral nerve block (FNB), and 3-in-1 nerve block, have been used to help control pain from hip fractures and total hip arthroplasty (THA) for many years. In August 2018, the PEricapsular Nerve Group (PENG) block was introduced for the purpose of better pain control.[1] Giron-Arango et al. found the accessory obturator nerve and the femoral nerve which are covered by the PENG block to have a greater reduction in opioid use and better pain control post-operatively.[1]

Case Report

An 85-year-old, 72 kg, 172 cm female presented for a left THA. She had a significant past medical history that included hypertension, heart failure, atrial fibrillation, a pacemaker, osteoarthritis, obesity, asthma, and gastroesophageal reflux disease. She had allergies to levaquin, xarelto, celebrex, and amoxicillin. Daily home medications included hydroclorothiazide/triamterene 50/75 mg, metoprolol 50 mg extended release, and pantoprazole 20 mg. Past surgical history included nasal carcinoma excision, right THA, coronary catheterization, parathyroidectomy, carpal tunnel release, pacemaker placement, and melanoma excision. No prior complications with anesthesia were reported. Lab values were within normal limits. Risks, benefits, and alternatives were discussed, and the patient consented to spinal anesthesia, monitored anesthesia care and the PENG block.

In the preoperative unit, 2 hours prior to surgery the patient received gabapentin 600 mg and acetaminophen 1000 mg by mouth. In the operating room, the patient was transferred to the operating table and placed in the sitting position for administration of a spinal anesthetic. Standard American Association of Nurse Anesthetist monitors were placed on the patient and baseline vital signs were assessed as stable. Oxygen 2 L/min via nasal cannula was administered. Using aseptic technique, 0.75% bupivacaine 1.8 mL with an epinephrine wash was administered at L3-L4 intervertebral space with minimal difficulty. The patient was immediately placed in the left lateral position for 3 minutes then placed supine. The patient was then placed in the right lateral position by the surgeon. Dermatome levels were evaluated, and the patient had an adequate anesthetic block to T10 bilaterally.

During the procedure, monitored anesthesia care was maintained using a propofol infusion at 50 mcg/kg/min. A total of 800mg of propofol was administered throughout the 147-minute case. Intermittent doses of 5-10 mg of ephedrine were used to maintain systolic blood pressure above 100 mmHg. She received 1300 mL of lactated ringers. Estimated blood loss was 200 mL. The patient maintained spontaneous respirations throughout the case.

The PENG block was administered in the following manner following the conclusion of the surgery prior to the patient leaving the OR. With the patient in the supine position, a curved ultrasound probe was used to find the left anterior inferior iliac spine. The probe was then turned approximately 45 degrees and moved distally until the femoral artery, iliopsoas tendon, and pectineus muscle came into view. A 22 gauge 120 cm needle with catheter was inserted parallel to the iliac spine to the pubic ramus, and under the iliopsoas tendon. Following negative aspiration, 0.5% bupivacaine 10 mL with 1% lidocaine 10 mL and normal saline 5 mL was injected. The initial dosing with bupivacaine for the block was weight-based, location sensitive, and followed local anesthetic limits. After injection, the catheter was left in place and put on an On Q-pump using 0.2% ropivacaine with a starting rate of 6 mL/hr. The On Q-pump was discontinued 5 days later.

Follow-up with the patient in the post anesthesia care unit (PACU) demonstrated pain scores from 0-3. The patient did not require any additional pain management in the PACU. On postoperative day 1, the patient remained on the On Q-pump at a rate of 10 mL/hr and received oral acetaminophen 650 mg when her pain level reached a 2/10. On postoperative day 2, the patient received another dose of oral acetaminophen 650 mg for a pain score of 3/10 and was discharged home. Reports from the nursing and physical therapy staff stated a stronger mobility compared to their patients who received FIB. The patient also stated a stronger mobility compared to her previous THA on the contralateral side 12 months prior, using FIB. This resulted in a 36 hour hospital stay compared to 2 days for patients at this hospital who received FIB.

Discussion

The purpose of a peripheral nerve block is to minimize opioid use and maximize pain control in the postoperative phase. Peripheral nerve blocks are proven to be more effective in decreasing long term post-operative pain when compared to opioids.[2] Nerve blocks don't carry the same side effects of nausea, vomiting, constipation and delirium which leads to improved recovery and outcomes, especially in the elderly population.

The femoral nerve block, the facia iliaca nerve block, and the 3-in-1 block are the standard peripheral blocks used during THA cases. The femoral nerve block covers sensory innervation to the anterior thigh, anteromedial knee, and the medial aspect of the lower leg, ankle, and foot. It also effects motor function to the anterior portion of the leg. The fascia iliaca block blocks sensation to the hip, anterolateral thigh, anteromedial knee and the medial aspect of the lower leg. The 3-in-1 block provides regional anesthesia to the femoral, obturator and lateral cutaneous nerves.

The pioneers of the PENG block mapped out the nerves that innervate the hip capsule and found the femoral nerve in the capsule wasn't being completely covered by the standard nerve blocks, and the accessory obturator nerve (AON), a branch of the obturator nerve, wasn't being blocked at all.[1,2] Nelson et al. conducted a cadaver ultrasound study and found the articulating branches of the AON and femoral nerve make up most of the sensory innervation of the hip. They compared these different blocks to the PENG block and found the FIB, FNB, and 3-in-1 block don't cover deep or cephalad enough resulting in moderate post-operative pain.[3,4] The Nelson et

al. study also showed a 7 point reduction in pain scores when using the PENG block compared to the previously mentioned blocks.[2] This is because the PENG block targets the sensory nerves of the hip capsule, namely the AON, while also preserving motor nerves of the femoral nerve to the hip and lower leg. This was the basis for the decision to perform this block on the patient; an attempt for a superior outcome after moderate success with other block methods, while also minimizing the need for opioid use.

Twelve months prior to this case, the patient received a THA on the contralateral hip. Preoperatively, the patient received a fascia iliaca block and intraoperative management was the same. Post-operatively, during her hospital stay, the patient received a total of fentanyl 50 mcg IV, oxycodone 15 mg by mouth, acetaminophen 700 mg by mouth, and ketorolac 60 mg IV. In her most recent hip replacement on the left side, the intra-operative management was the same. The patient received the PENG block post-operatively. During her 36-hour hospital stay, she received a total of acetaminophen 650 mg by mouth.

In the PACU the patient was awake and oriented. She complained of minimal pain, 2/10. She received an ON-Q pump using 0.2% ropivacaine. She was discharged to the floor while receiving no additional medication for pain in PACU. The patient had minimal movement in her leg immediately post-operative due to the effects of the spinal anesthetic. After she moved to the medical surgical unit, she was able to ambulate with minimal pain reported. Her pain scores show decreased pain levels after this surgery compared her previous THA and the only difference was the block that was used, the PENG block.

In elderly patients, the use of opioids cause delirium and can increase the risk for falls. Using the PENG block with a continuous catheter allows for adequate pain control as shown in this case study, and decreases the amount of opioids needed, if at all. The block also allows for early postoperative mobility due to their muscle-sparing properties and a faster, more cost-effective discharge, without compromising patient safety. Elderly patients can receive this block in both a surgical setting or emergency room setting where hip pain may present itself. Future research is needed to compare: pain scores, muscle strength, postoperative mobility, opioid use, and more specific dosing. A study is currently in the planning stages to compare the PENG block to the FIB.

References

1. Girón-Arango L, Peng P, Chin K, Brull R, Perlas A. Pericapsular nerve group (PENG) Block for hip fracture. *Reg Anesth Pain Med*. 2018;43(8):859-863. doi: 10.1097/AAP.0000000000000847.
2. Weinstein E, Levene J, Cohen M, Andreae D, Chao J, Johnson M, Hall C, Andreae M. Local aneaesthetics and regional anaesthesia versus conventional analgesia for preventing persistent postoperative pain in adults and children. *Cochrane Database Syst Rev.* 2018;20(6). DOI: 10.1002/14651858.CD007105.pub4
3. Nielsen T, Moriggl B, Søballe K, Kolsen-Petersen J, Børglum J, Bendtsen T. A cadaveric study of ultrasound-guided subpectineal injectate spread around the obturator nerve and its hip articular branches. *Reg Anesth Pain Med.* 2017;42(3):357-361.

4. Short AJ, Barnett JJG, Gofeld M, Baig E, Lam K, Agur AMR, Peng PH. Anatomic study of innervation of the anterior hip capsule: Implication for image-guided intervention. *Reg Anesth Pain Med.* 2018;43(2):186-192. doi: 10.1097/AAP.0000000000000701.
5. Guay J, Parker M, Griffiths R, Kopp S. Peripheral nerve blocks for hip fractures: A Cochrane review. *Anesth Analg*. 2018;126(5):1695-1704. doi: 10.1213/ANE.0000000000002489
6. Yu H, Moser J, Chu A, Montgomery S, Brown N, Endersby R. Inadvertent quadriceps weakness following the pericapsular nerve group (PENG) block. *Reg Anesth Pain Med*. 2019;44(5):611-613.

Mentor: Nathan Adams, DNP, CRNA

Perioperative Ventilation Strategies for Lung Donation

Elizabeth R. Chambers, BSN
University of Pennsylvania

Keywords: ventilation, lung donation, donor management, lung preservation, protocol

In organ transplantation, donor lungs are particularly scarce, creating a significant issue of lack of organ supply to meet demand. Viable lungs for transplant are procured from only 10-20% of donors, with a 7.9-25% incidence of primary graft dysfunction among recipients.[1, 2] Compromise of donor lungs may be the result of direct trauma, resuscitation efforts, neurogenic edema, aspiration, and poor ventilation in the patient meeting brain death criteria.[3] Recent strategies to optimize lung tissue include protective ventilation and recruitment maneuvers during medical management.[1] This case study examines the perioperative ventilatory management of a patient presenting for multiple organ donation.

Case Report

A 42-year-old, 87 kg, 165 cm female found unresponsive prehospital, presented for donor organ procurement following declaration of brain death in the intensive care unit (ICU). Timing and transport to the operating room was coordinated with the organ procurement teams. The patient was transferred to the care of the anesthesia team with vascular access, a 7.0 mm endotracheal tube (ETT), orogastric tube, and urinary catheter in situ. The patient's vascular access included a left radial arterial line, right internal jugular central line infusing continuous levothyroxine 10 mcg/hr, and one 20-gauge peripheral intravenous (IV) line in the right hand.

After transferring the patient to the operating room table, standard noninvasive monitors and arterial blood pressure monitoring were applied. Correct positioning of the ETT was confirmed with positive end tidal Carbon Dioxide ($ETCO_2$), equal chest expansion, and bilateral breath sounds. The anesthesia machine breathing circuit was connected to the ETT and ventilation was initiated on volume control ventilation. Settings on volume control ventilation included O_2 2L/min, tidal volume (VT) 500 mL, respiratory rate (RR) 12/min, and positive end-expiratory pressure (PEEP) of 5 cm H_2O. No inhalational agent was used. Baseline vital signs included heart rate 62/min, SpO_2 98%, arterial blood pressure 90/50 (63) mm Hg, and temperature of

35.8°C. An esophageal temperature probe and upper body forced air heating blanket were applied to maintain normothermia greater than 35°C.

Heart, lung, liver, and kidney surgical teams were present for time-out procedure with nursing and anesthesia professionals. After time-out, evaluation of the lungs was conducted via bronchoscopy with adult flexible bronchoscope prior to sternotomy incision. No excessive suctioning was performed, nor were any medications instilled by the pulmonologist during the exam. Ventilation was held for sternotomy and resumed. VT was increased to 10 mL/kg, RR decreased to 10/min, and PEEP increased to 8 cm H_2O. Intermittent manual recruitment maneuvers to achieve peak inspiratory pressures of 30 cm H_2O were performed. Mannitol 25 g IV was given. Hypotension due to cardiac manipulation was treated with 1L Isolyte and 300 mLs albumin IV. A continuous phenylephrine infusion 20-200 mcg/min was initiated and titrated to maintain a mean arterial pressure greater than 65 mm Hg. Heparin 30,000 units were administered.

An additional liter of Isolyte was rapidly infused to treat continued variable hypotension. Intermittent manual recruitment maneuvers were implemented to achieve peak inspiratory pressures of 30 cm H_2O, and maximal lung inflation continued. Oxygen was lowered to 0.5 L/min with 1.5 L/min air. Respiratory rate was decreased from 10 to 8/min with VT increased to 12 mL/kg. PEEP was further increased to 10 cm H_2O. The aorta was cannulated and cross-clamped. Cardioplegia solution was infused by the perfusion team. The heart was excised and transferred for cooling. Ventilation with frequent Valsalva maneuvers was continued until the complete excision of bilateral lungs. Lungs were maximally expanded under surgeon visualization immediately prior to removal. Liver and kidneys were procured last. After the removal of both the heart and lungs, anesthesia team involvement was no longer required.

Discussion

No standardized protocol for intraoperative pulmonary management during organ procurement exists.[1] On an individual case basis, the anesthesia team defers to the expert opinions of the procurement team to achieve the best care and conditions of the patient presenting for organ donation. Following brain death diagnosis, the literature regarding donor care focuses on management in the intensive care unit (ICU). As normalization of donor physiology maximizes the long-term viability of organs for donation, the strategy should continue intraoperatively.[4] Fortunately, new approaches to optimizing lung tissue in the intraoperative course have widened the donor pool to lungs that might have traditionally been rejected.[5] Conditions that result in poor pulmonary function include aspiration pneumonitis, pulmonary contusions, substantial smoking history, and tracheobronchial injuries.[5] Brain death further complicates these conditions and otherwise healthy lungs, resulting in neurogenic pulmonary edema, atelectasis, catecholamine and cytokine release, and low partial pressure of arterial oxygenation of less than 300 mmHg.[4]

Employment of a protective versus conventional lung strategy, consisting of recruitment maneuvers, and airway pressure release ventilation, have resulted in decreased atelectasis and improved PaO_2 greater than 300 mmHg, therefore increasing the number of accepted lungs.[5,6] Protective lung strategy uses 6-8 mL/kg VT for ventilation and PEEP of 8-10 cm H_2O, compared

to conventional strategy of 10-12 mL/kg VT with PEEP between 3 and 5 cm H_2O.[1,6] This lower volume strategy has been shown to increase the number of lungs transplanted while preserving 6-month survival rates of recipients.[7] In this case, initial ventilation settings employed smaller tidal volumes, yet PEEP was set at 5 cm H_2O, consistent with conventional strategy.

Recruitment maneuvers aim to re-expand collapsed and edematous lung tissue.[3] Specifically, transpulmonary pressure is transiently increased to reopen alveoli that are not aerated or poorly aerated but reopenable.[3] Contraindications include, but are not limited to, severe bronchospasm, bullous emphysema, untreated pneumothorax, unilateral lung disease, and hemodynamic instability.[8] Many of these findings may be detected via bronchoscopy. The findings of the bronchoscopy in this case did not preclude the use of recruitment maneuvers.

Lung recruitment may be approached in different ways. In the ICU setting, literature describing recruitment maneuvers recommends the ventilator to be set to pressure control mode, pressure control ventilation 25-30 mmHg, with the application of PEEP 10-15 cm H_2O to obtain tidal volumes 8-10 mL/kg lasting two hours.[3] In this study, marginal lungs were defined in patients with normal chest X-ray or bilateral infiltration, with PaO_2 200-300 mmHg with 100% FiO_2, and monitored pre- and post- arterial blood gases.[3] Another recruitment maneuver involves doubling ventilation with low tidal volumes for ten breaths, especially after any disconnection from the ventilator.[1,6] Lastly, the ventilator may be set to a continuous positive airway pressure of 40 cm H_2O for 30 seconds.[8] This process is repeated every 20 mins for a total of 3 times and may be done at a lower pressure if not tolerated by the donor.[8] This maneuver resembles the manual maneuvers done in this case, although the pressure set with the adjustable pressure-limiting (APL) valve was variable from 30-70 cm H_2O to achieve airway pressures of 30 cm H_2O.

Airway pressure release ventilation (APRV) is another approach to donor lung management in the ICU that is uncommon in the operating room, though newer anesthesia machines may contain expanded ventilation options. APRV is a pressure-limited, time-cycled, volume-variable mode of ventilation using continuous positive airway pressure with an intermittent pressure release phase.[5] The continuous airway pressure maintains adequate lung volume and improves alveolar recruitment while the release phase allows for carbon dioxide removal.[5] Compared with alternative modes of ventilation, APRV produces similar or improved oxygenation with lower peak airway pressures with no significant hemodynamic effects.[5] Additionally, APRV overcomes the critical opening pressure of quiescent alveoli without shear stress and barotrauma associated with assist control ventilation.[5] Unfortunately in this case, APRV was not an available function on the anesthesia machine and volume control ventilation was the principle mode used.

Anesthetic pharmacological management in this case was largely dictated from guidelines provided by the donation coordinator. This included instruction for fluids, mannitol, and heparin administration. Like many other organ procurement organizations there were no guidelines on pulmonary management,[7] creating an opportunity for interest and review. In retrospect, many of the choices made for ventilation were in concordance with protective lung strategy and frequent recruitment maneuvers. The pulmonary team requested higher levels of PEEP, lower FiO_2, Valsalva maneuvers, and higher volumes during critical parts of the case. Having reviewed the literature and parameters for lung suitability, one lesson learned from this case study is to pay closer attention to the effects of the ventilation strategy on the arterial blood gases in a consistent

manner, with a focus on PaO_2. Though the lungs were suitable for transplant, one limitation in this case is unknown recipient course and/or impact of this ventilatory management.

References

1. Miñambres E, Coll E, Duerto J, et al. Effect of an intensive lung donor-management protocol on lung transplantation outcomes. *J Heart Lung Transplant.* 2014;33(2):178-184.
2. Porteous M, Diamond J, Christie J. Primary graft dysfunction: lessons learned about the first 72 hours after lung transplantation. *Curr Opin Organ Transplant.* 2015;20:506-514.
3. Parto S, Shafaghi S, Khoddami-Vishteh H, et al. Efficacy of recruitment maneuver for improving the brain dead marginal lungs to ideal. *Transplantation Proceedings.* 2013;45:3531-3533.
4. Anderson TA, Bekker P, Vagefi PA. Anesthetic considerations in organ procurement surgery: a narrative review. *Can J Anesth.* 2015;62:529–539.
5. Hanna K, Seder CW, Weinberger JB, et al. Airway pressure release ventilation and successful lung donation. *Arch Surg.* 2011;146(3):325-328.
6. Swanson JA, D'Cunha J. Increasing the lung donor pool: recruitment of the gift of life. *Semin Thoracic Surg*. 2011;23:91-92.
7. Ream RS, Armbrecht ES. Variation in pediatric organ donor management practices among US organ procurement organizations. *Prog Transplant.* 2018;28(1):4-11.
8. Kirschbaum CE, Hudson S. Increasing organ yield through a lung management protocol. *Prog Transplant.* 2010;20(1):28-32.

Mentor: Lori Ann Winner, MSN, CRNA, APN-A

The Safety and Efficacy of Sugammadex versus Neostigmine

Nicole Landen, MSN, BSN
University of North Dakota

Keywords: Sugammadex, Neostigmine, Neuromuscular blockade (NMB), Train of four (TOF), Post-tectonic count (PTC)

Neuromuscular blockade (NMB) is utilized for the purpose of skeletal muscle paralysis during surgery. The depth of the NMB is monitored to ensure optimal surgical conditions and aid in determining the appropriate method to reduce the risk of residual NMB. Residual NMB can lead to various complications (i.e. upper airway obstruction, hypoxemia, aspiration risk, and hypoventilation) associated with increased perioperative morbidity and longer hospital stays.[1] A survey found that majority of anesthesia providers perceived the incidence of residual NMB to be 1-10%.[2] However, it has been shown to occur in 30-50% of patients following surgery.[2,3] To compare the safety and efficacy of sugammadex and neostigmine on NMB reversal, numerous studies have been analyzed. [3-7]

Case Report

A 73-year-old, 98.5 kg, 157 cm female presented for a ventral hernia repair. Significant past medical history included hypertension, chronic kidney disease-stage 3, and gastroesophageal

reflux disease. Pertinent past surgical history included gastric bypass and ventral hernia repairs. No noted anesthesia complications. She had no known allergies and had been NPO for 8 hours. Pre-operative labs were unremarkable.

Airway assessment included Mallampati class I, full neck range-of-motion, and adequate thyromental distance of 6 cm. Preoperative vitals included blood pressure 100/64 mm Hg, heart rate 78/min, respirations 16/min, SpO_2 97% on room air, and temperature 36.4ºC. Auscultation of her heart and lungs revealed a regular heart rate and rhythm with clear, bilateral breath sounds.

The patient was transported to the operating room (OR) via a transport stretcher and was positioned with assistance on OR bed. Lactated ringer (LR) was infusing from the preoperative unit via an 18-guage, peripheral intravenous (IV) line; 2mg of midazolam was administered. Standard noninvasive monitors were applied and monitoring commenced. The patient was pre-oxygenated with 8 L/min of oxygen via face mask. Induction was initiated with fentanyl 100 mcg, lidocaine 40 mg, rocuronium 5 mg, and a dexmedetomidine bolus of 0.25 mcg/kg IV. Propofol 200 mg IV was administered, and cessation of spontaneous respirations was noted. Rocuronium 40 mg IV was administered for paralysis. Patient ventilated with ease via bag mask ventilation, chest rise noted and EtCO2 within normal limits. A Miller 2 laryngoscope was inserted into the patient's oropharynx, and vocal cord visualization was confirmed. A size 7.0 mm endotracheal tube (ETT) was passed through vocal cords successfully. The ETT was secured and pressure control ventilation was provided. Desflurane was initiated and fresh gas flows were incrementally decreased to provide a 0.8 to 1.0 MAC. Additional prophylactic medications, ondansetron 4 mg IV, dexamethasone 4 mg IV, cefazolin 2 g IV, and heparin 5,000 units subcutaneous were administered. Ketamine 50 mg and Fentanyl 50 mcg IV were given prior to incision and a dexmedetomidine infusion (0.5mcg/kg/min IV) was provided through the duration of the case. During the 4 hour case, additional fentanyl 200 mcg, hydromorphone 1 mg, LR solution 3,000 mL, and 5% albumin 500 mL IV were administered. The neuromuscular blockade was monitored with a peripheral nerve stimulator via the orbicularis oculi muscle. Paralysis was maintained via rocuronium boluses to a train-of-four (TOF) count of 2-3 out of 4 twitches. In total, 130 mg of rocuronium was administered throughout the procedure.

At the end of the case a TOF count showed 4/4 twitches. Sugammadex 400mg IV was administered to antagonize NMB. Dosing of 4mg/kg of sugammadex was considered based on provider preference due to level of relaxation and total amount of paralytic administered. Shortly after the sugammadex administration, spontaneous respirations were noted. Desflurane was discontinued and the patient was withdrawn from mechanical ventilation. The patient was spontaneously breathing and following commands before she was extubated to spontaneous mask ventilations, followed by 3 L/min via nasal canula. The patient was successfully transferred to the PACU without any complication and remained hemodynamically stable throughout her recovery.

Discussion

Neuromuscular blocking agents induce muscle relaxation, which is commonly used during surgery to facilitate tracheal intubation and provide optimal surgical conditions via suppression of skeletal muscle tone. It is vital at the end of surgery these medications can be reversed to

allow return of normal neuromuscular function to prevent postoperative complications (ie. hypoventilation, airway obstruction, and hypoxia). Traditionally, cholinesterase inhibitors, such as neostigmine, have been the primary agents used to counteract the neuromuscular blocking agents. These agents increase the amount of ACh available to compete against the nondepolarizing agent, ultimately reestablishing normal neuromuscular transmission.[1] Neostigmine also allows the ACh within the cleft to have a longer lifespan, allowing for more antagonistic dissociation time and reactivation of the nicotinic receptor site via ACh.[1] This excess ACh available in the synaptic cleft can also bind to muscarinic receptors, which contribute to the parasympathomimetic side-effects commonly seen following neostigmine administration.[1] These unwanted side-effects are usually minimized by prior or co-administration of an anticholinergic medication, commonly glycopyrrolate.[1] Recently, sugammadex has become available to inactivate steroidal neuromuscular blocking agents. Sugammadex works by encapsulating the steroidal neuromuscular blocking agent, creating a complex that completely inactivates its ability to produce paralysis.[1] Once the encapsulation occurs there is no dissociation and the sugammadex-relaxant complex is eliminated in the urine.[1]

Common methods of monitoring the level of paralysis includes: TOF, Tetany, and post-tetanic count (PTC). Upon onset of NMB with nondepolarizing relaxant there is a progressive reduction in the twitches response and strength measured via TOF stimulator.[1] A TOF-ratio compares twitch 4 to twitch 1 and can aid in approximating the degree of paralysis.[1] PTC is assessed following a 50-Hz tetanic stimulation for five seconds followed by a series of single 1 Hz stimulations.[1] The response to the single twitches, correlates with the approximate depth of the block and can indicate how long it may take for spontaneous reversal to occur.[1]

Evidence of a moderate neuromuscular blockade is indicated by visual or palpable T1 and T2 twitches via the TOF stimulator.[5,6] One meta-analysis found sugammadex to be 6.6 times faster than neostigmine to produce a TOF ratio of 0.9 or greater following a moderate rocuronium-neuromuscular blockade.[6] Additional studies support sugammadex to be faster than neostigmine in reversing this level of NMB.[4,5,6]

Deep paralysis is indicated by a PTC of 1 to 4.[5,6] It was found sugammadex produced an average recovery time of 2.2 minutes and 3.8 minutes following rocuronium and vecuronium induced paralysis, respectively.[5] Within 5 minutes, 95% and 77% (correspondingly for rocuronium and vecuronium) of the sugammadex groups had full recoveries while only 7% of the rocuronium-neostigmine group recovered within 5 minutes.[5] In the vecuronium group, no participants were able to recover within 5 minutes following neostigmine administration.[5] Research established that the neostigmine group, required 30-60 minutes for majority of its patients to fully recover to TOF ratio of 0.9 or greater following administration at PTC 1-2.[6]

For immediate reversal of a profound NMB, such that following 1.2 mg/kg IV rocuronium administration, sugammadex 16 mg/kg IV required an average of 1.7 minutes.[5] Neostigmine was not studied in this setting due recommendations that some indication of spontaneous recovery must be seen before it can safely be administered.[5]

Overall, hemodynamic changes were minimal and without statistically significant differences between neostigmine and sugammadex administration throughout the literature reviewed.[6]

Sugammadex was suggested to be associated with lower respiratory and cardiovascular adverse events (bradycardia), as well as less postoperative weakness.[4,6]

Sugammadex is renally eliminated thus posing risk to worsen renal failure.[5] The minimal effects on glomerular filtration and tubular function produced only slightly slower recovery times and were not associated with any clinical evidence of renal dysfunction.[4,5] As far as severe renal impairment (i.e. dialysis), sugammadex is currently not recommended for use due to the limited data available.[5]

Research has shown sugammadex is associated with an increase in Prothrombin Time (PT) and Activated Partial Thromboplastin Time (aPTT) occurring within 10 minutes following sugammadex administration.[6,7] However, it was seen to resolve within 60 minutes.[6,7] As a whole, the studies support that these increases in PT and aPTT are transient and unlikely to be clinically relevant. [6,7]

The incidence of anaphylaxis associated with sugammadex or the sugammadex-rocuronium complex has been increasing throughout the past few years.[7] The most frequent symptoms reported accompanying sugammadex-induced anaphylaxis include, rash, hypotension, and tachycardia.[7] It has been reported that most reactions occurred within 4 minutes after administration. [7] Making this time frame, a critical period to be vigilant and ensure adequate identification of allergic reaction are managed in a timely manner.

Overall, the literature supports sugammadex as a safer and more efficacious medication in the reversal of all depths of neuromuscular blockades. Research provides support for sugammadex to provide a faster and more reliable reversal, thus providing a great benefit for patient safety. Other important considerations when using sugammadex include its incompatibility with verapamil, ondansetron, and ranitidine.[8] Also, it's important to note that sugammadex can interact with hormonal contraceptives, posing risk of them becoming less effective.[8] It is imperative to ensure patients are aware of sugammadex administration and the risks post-operatively that coincide for 7 days following administration.[8] Ultimately, the research concluded sugammadex provided a more reliable reversal independent of neuromuscular blockade depth, had significantly less risk of residual paralysis postoperatively, and a lower adverse event profile.[1,4,6]

References

1. Nagelhout JJ. Neuromuscular blocking agents, reversal agents, and their monitoring. In Nagelhout JJ, Elisha S. *Nurse anesthesia.* 6th ed. Elsevier Saunders; 2018:140-164
2. Murphy G. Presentation of the APSF collaborative panel on neuromuscular blockade and patient safety at the 2017 ASA annual meeting. apsf.org. https://www.apsf.org/wp-content/uploads/newsletters/2018/Feb/pdf/APSF201802.pdf#page=12. Published February 2018. Accessed March 3, 2019.
3. Brull SJ, Kopman AF. Current status of neuromuscular reversal and monitoring: challenges and opportunities. *Anesthesiology* 2017;126(1):173-190.

4. Carron M, Zarantonello F, Tellaroli P, Ori C. Efficacy and safety of sugammadex compared to neostigmine for reversal of neuromuscular blockade: A meta-analysis of randomized controlled trials. *J Clin Anesth*. 2016;35:1-12.
5. Herring WJ, Woo T, Assaid CA, et al. Sugammadex efficacy for reversal of rocuronium- and vecuronium-induced neuromuscular blockade: A pooled analysis of 26 studies. *J Clin Anesth*. 2017;41:84-91. doi: https://doi.org/10.1016/j.jclinane.2017.06.006.
6. Hristovska AM, Duch P, Allingstrup M, Afshari A. Efficacy and safety of sugammadex versus neostigmine in reversing neuromuscular blockade in adults. *Cochrane Database Systematic Review*. 2017;8(8):CD012763. doi:10.1002/14651858.CD012763
7. Iwasaki H, Renew JR, Kunisawa T, Brull SJ. Preparing for the unexpected: special considerations and complications after sugammadex administration. *BMC Anesthesiology*. 2017;17(1):140. doi:10.1186/s12871-017-0429-9
8. Merck. Bridion (Sugammadex). merck.com. 2018. https://www.merck.com/product/usa/pi_circulars/b/bridion/bridion_pi.pdf. Updated December 2018. Accessed March 2, 2019.

Mentor: Amber Johnson, PhD, CRNA

Editorial

The annual ISJNA meeting will be held at the Assembly of Didactic and Clinical Educators on Friday, February 21st from 6:30-7:15 AM in the Cannes meeting room located in the Chateau Elan. Anyone involved with or interested in learning more about the ISJNA is welcome to attend!

I am always impressed with the variety of case report topics we receive, but we also welcome submissions on topics that have already been published. As clinical practitioners, we know that every case is different. As lifelong learners we can appreciate the nuanced variations between patients and procedures, and gain insights from the experiences of others as we continually work to refine and perfect our craft. I hope everyone who reads this journal takes from it a valuable piece of information that helps improve their practice.

I am pleased to announce that the International Student Journal of Nurse Anesthesia now has an International Standard Serial Number (ISSN) - 2688-5263. We are now also indexed with ProQuest (in addition to EBSCO)! This enhances the visibility and accessibility of the ISJNA, allowing us to reach a wider audience.

Sincerely,

Vicki C. Coopmans, PhD, CRNA
Editor

"***The International Student Journal of Nurse Anesthesia*** **is produced exclusively for publishing the work of nurse anesthesia students. It is intended to be basic and introductory in its content. Its goal is to introduce the student to the world of writing for publication; to improve the practice of nurse anesthesia and the safety of the patients entrusted to our care.**"

INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA
GUIDE FOR AUTHORS

MISSION STATEMENT

The International Student Journal of Nurse Anesthesia (ISJNA) is produced exclusively for publishing the work of nurse anesthesia students. It is intended to be basic and introductory in its content. Its goal is to introduce the student to the world of writing for publication; to improve the practice of nurse anesthesia and the safety of the patients entrusted to our care.

ITEM PREPARATION & SUBMISSION

Case reports, research abstracts, evidence-based practice (EBP) analysis reports, evidence-based practice project abstracts, and letters to the editor may be submitted. These items must be authored by a student under the guidance of an anesthesia practitioner mentor (CRNA or physician). Case and EBP analysis reports must be single-authored, while abstracts may have multiple authors. Submissions may list only one mentor. **Mentors should take an active role** in reviewing the item to ensure appropriate content, writing style, and format prior to submission. The mentor must submit the item for the student and serve as the contact person during the review process. Items submitted to this journal should not be under consideration with another journal. Authors and mentors should critically evaluate the topic and quality of the writing – multiple reviews of the item by the mentor, faculty, and peers (fellow graduate students) prior to submission is recommended. If the topic and written presentation are beyond the introductory publication level we strongly suggest that the article be submitted to a more prestigious publication such as the *AANA Journal*.

The journal is committed to publishing the work of nurse anesthesia students. The review process is always initiated with the following rare exceptions. We are conservative in accepting reports where the patient has expired, realizing that you can do everything right and still have a negative outcome. Submissions that report a case demonstrating failure to meet the standard of care (by any practitioner involved in the case) will not be accepted. Unfortunately, while the experiences in these cases can offer valuable insight, these submissions will not be accepted for review due to potential legal risks to the author, journal, and anyone else involved in evaluating the report.

It is the intent of this journal to publish items while the author is still a student. In order to consistently meet this goal, all submissions must be received by the editor at least **3 months prior** (4-6 months recommended) to the author's date of graduation. Manuscripts must be submitted by the mentor of the student author via e-mail to **INTSJNA@aol.com** as an attachment. The subject line of the e-mail should use the following format: ISJNA Submission_submission type_author last name_mentor last name. The item should be saved in the following format – two-three word descriptor of the article_author's last name_school abbreviation_mentor's last name_date (e.g. PedsPain_Smyth_GU_Pearson_5.19.09)

REVIEW PROCESS

Items submitted for publication are initially reviewed by the chief editor. If the chief editor does not acknowledge receipt of the item within two weeks, please inquire to ensure receipt. Upon receipt, the chief editor will review the submission for compliance with the Guide to Authors. If proper format has not been followed, the item will be returned to the mentor for correction. This is very important as all reviewers serve on a volunteer basis. Their time should be spent ensuring appropriate content, not making format corrections. It is the mentor's responsibility to ensure formatting guidelines have been followed prior to submission.

All accepted submissions undergo a formal process of blind review by at least two reviewers. After review, items may be accepted without revision, accepted with revision, or rejected with comments. Once the item has been accepted for review the chief editor will send a blinded copy to an editor, who will then coordinate a blinded review by two reviewers who are not affiliated with the originating program. The editor will return the item to the chief editor, who will return it to the mentor for appropriate action. Every effort is made to complete the process in an efficient, timely matter. Again, the goal is for all articles submitted by students to be published while the author is still a student. If an item is not ready for publication within 6 months after the student author has graduated it will no longer be eligible for publication. Mentors will be listed as contributing editors for the issue in which the item is published.

PHOTOS

Photos of students for the front cover of the Journal are welcome. Please contact the chief editor at intsjna@aol.com to submit photos for consideration. Only digital photos of high quality will be accepted. If the photo is accepted, consent forms must be completed and returned by all identifiable individuals in the photo, and the individual who took the photo.

ACADEMIC INTEGRITY

Issues of academic integrity are the responsibility of the author and mentor. Accurate and appropriate acknowledgement of sources is expected. The two most common breaches of academic integrity that have been identified in submissions to this journal are (AMA 10th ed., p. 158):

1. Direct plagiarism: verbatim lifting of passages without enclosing the borrowed material in quotation marks and crediting the original author.
2. Paraphrase: restating a phrase or passage, providing the same meaning but in a different form without attribution to the original author.

Please note that changing one or two words in a reference source passage (e.g. 'of' for 'in', or 'classified' for 'categorized') and then citing it as a paraphrase or summary is also not appropriate, and still falls within the definition of direct plagiarism. If plagiarism in any form is identified, review of the item will be suspended and it will be returned to the mentor. Repeated instances of plagiarism will result in rejection of the item.

Plagiarism detection software (TurnItIn, PlagScan, SafeAssign, etc . . .) can be used to analyze the document prior to submission to ensure proper citation and referencing, but is not required.

"Plagiarism is the presentation of someone else's ideas, writings, or statements as one's own. Plagiarism is a serious breach of academic integrity, and anyone who is found to have committed plagiarism will be subject to disciplinary action.
Paraphrase is the act of putting someone else's ideas into one's own words. The use of paraphrase can be an acceptable practice under some circumstances if it is used sparingly and if the original text is properly acknowledged. Unacknowledged paraphrase, like plagiarism, is a serious breach of academic integrity. Any improper use of sources may constitute plagiarism. Every quotation from another source, whether written, spoken, or electronic, must be bound by quotation marks and be properly cited. Mere citation alone is not sufficient when a scholar has used another person's words. Similarly, every paraphrase or summary (a more concise restatement of another's ideas) must be properly cited."
https://sites.google.com/a/georgetown.edu/gsas-graduate-bulletin/vi-academic-integrity-policies-procedures

GENERAL GUIDELINES

Items for publication **must adhere to the *American Medical Association Manual of Style*** (AMA 10th ed., the same guide utilized by the *AANA Journal* and such prominent textbooks as *Nurse Anesthesia* by Nagelhout and Plaus). Page numbers are provided for easy reference in the AMA Manual of Style throughout this document. The review process will not be initiated on items submitted with incorrect formatting and will be returned to the mentor for revision. Please note the following:

1. Use complete sentences.
2. Acronyms/Initialisms (p. 379) - spell out with first use, do not capitalize the words from which the acronym/initialism is derived unless it is a proper noun or official name. If you are using the phrase only once, do not list the acronym/initialism at all. Avoid beginning sentences with acronym/initialisms.
3. Abbreviations (p. 441)
4. Use *Index Medicus* journal title abbreviations (p. 472, http://www.ncbi.nlm.nih.gov/nlmcatalog/journals)
5. Always provide units of measure (p. 521 & 795). In most cases The International System of Units (SI) is used. Abbreviations for units of measure do not need to be spelled out with first use. Report height in cm, weight in kg, temperature in ^{o}C, pressure in mm Hg or cm H_2O. Report heart and respiratory rate as X/min (e.g. the patient's heart rate increased to 145/min).
6. In general, first use of pulmonary/respiratory abbreviations should be expanded, with the following exceptions: O_2, CO_2, PCO_2, $PaCO_2$, PO_2, PaO_2, $EtCO_2$, N_2O. Please use SpO_2 for oxygen saturation as measured by pulse oximetry.

7. Use the nonproprietary (generic) name of drugs (p. 568) - avoid proprietary (brand) names. Type generic names in lowercase. When discussing dosages state the name of the drug, *then* the dosage (midazolam 2 mg).
8. Use of descriptive terms for equipment and devices is preferred. If the use of a proprietary name is necessary (for clarity, or if more than one type is being discussed), give the name followed by the manufacturer and location in parenthesis (p. 583, e.g. a GlideScope (Verathon Inc., Bothell, WA) was used) Please note, TM and ® symbols are not used per the AMA manual.
9. Infusion rates and gas flow rates:
    a. Use mcg/kg/min or mg/kg/min for infusion rates. In some cases it may be appropriate to report dose or quantity/hr (i.e. insulin, hyperalimentation). If a mixture of drugs is being infused give the concentration of each drug and report the infusion rate in ml/min.
    b. Report gas flow of O_2, N_2O and Air in L/min (not %) and volatile agents in % as inspired or expired concentration (e.g. General anesthesia was maintained with sevoflurane 3% inspired concentration in a mixture of O_2 1 L/min and air 1 L/min.)
10. Only Microsoft Word file formats will be accepted with the following criteria:
    a. Font - 12 point, Times New Roman
    b. Single-spacing (except where indicated), paragraphs separated with a double space (do not indent)
    c. One-inch margins
    d. End the sentence with the period before placing the superscript number for the reference.
    e. Do not use columns, bolds (except where indicated), or unconventional lettering styles or fonts.
    f. Do not use endnote/footnote formats.
11. Do not use Endnotes or similar referencing software – any embedded formatting must be removed prior to submission.
12. Remove all hyperlinks within the text.
13. Avoid jargon and slang terms. Use professional, scholarly, scientific language.
    a. *'The patient was reversed'* - Did you physically turn the patient around and point him in the opposite direction? "Neuromuscular blockade was antagonized."
    b. *The patient was put on oxygen.* "Oxygen 2 L/min was administered via face mask."
    c. *The patient was intubated and put on a ventilator*. "The trachea was intubated and mechanical ventilation was initiated.
    d. *An IV drip was started.* "An intravenous infusion was initiated."
    e. Avoid the term "MAC" when referring to a sedation technique - the term sedation (light, moderate, heavy, unconscious) may be used. Since all anesthesia administration is monitored, pharmacologic, rather than reimbursement, terminology should be used.
14. Direct quotes are discouraged for reports of this length – please express in your own words.
15. Use the words "anesthesia professionals" or "anesthesia practitioners" when discussing all persons who administer anesthesia (avoid the reimbursement term "anesthesia providers").
16. Do not include ASA Physical Status unless it is germane to the report.
17. Do not use the phrase "ASA standard monitors were applied". Instead, "standard noninvasive monitors" is acceptable – additional monitoring can be detailed as needed.
18. References
    a. The **AMA Manual of Style must be adhered to** for reference formatting.
    b. All sources should be published within the past 8 years. Seminal works essential to the topic being presented will be considered.
    c. Primary sources are preferred.
    d. **A maximum of one textbook (must be most recent edition available) may be used as reference for case report submissions only**.
    e. All items cited must be from peer-reviewed sources – use of sources found on the internet must be carefully considered in this regard. URLs must be current and take the reader directly to the referenced source.

Heading – for all submission types (Case Report, Abstract, EBPA Report) use the following format.

1. **Title** is bolded, centered, 70 characters (including spaces) or less
2. Author name (academic credentials only) and NAP are centered, normal font,.
3. *Graduation date and email address* are centered, italicized, and will be removed prior to publication)
4. **Keywords** is left-justified, bolded – list keywords that can be used to identify the report in an internet search

Title

Author Name
Name of Nurse Anesthesia Program
Anticipated date of graduation
E-mail address

Keywords: keyword one, keyword two, etc . . .

Case Reports - The student author must have had a significant role in the conduct of the case. The total word count should be between 1200 – 1400 words (references not counted). Case reports with greater than 1400 words will be returned to the mentor for revision prior to initiation of the review process. The following template demonstrates the required format for case report submission.

Heading (see above)

A brief introductory paragraph of less than 100 words to focus the reader's attention and interest them to continue reading. This may include historical background, demographics or epidemiology (with appropriate references) of the problem about to be discussed. It is written in the *present tense*. Although it is introductory, the heading word '***Introduction'*** is not used. Be certain to cite references in this section, especially statistics and demographics.
[space]
Case Report (bold, 400-600 words)
[space]
This portion discusses the case performed and is written in the *past tense*. Do not justify actions or behaviors in this section; simply report the events as they unfolded. Present the case in an orderly sequence. Some aspects need considerable elaboration and others only a cursory mention. Under most circumstances if findings/actions are normal or not contributory to the case then they should not be described. Events significant to the focus of the report should be discussed in greater detail. The purpose of the case report is to set the stage (and 'hook' the reader) for the heart of your paper which is the discussion and teaching/learning derived from the case.

- Give dosage and schedule only if that information is pertinent to the consequences of the case.
- **Significant** laboratory values, x-rays or other diagnostic testing pertinent to the case. Give the units of measure after the values (eg. Mmol/L or mg/dL).
- Physical examination/pre-anesthesia evaluation - **significant** findings only.
- Anesthetic management (patient preparation, induction, maintenance, emergence, post-operative recovery).

[space]
Discussion (bold, 600-800 words)
[space]
Describe the ***anesthesia*** implications of the focus of the case report citing current literature. Describe the rationale for your actions and risk/benefits of any options you may have had. This section is not merely a pathophysiology review that can be found in textbooks. *Relate the anesthesia literature with the conduct of your case noting how and why your case was the same or different from what is known in the literature.* Photographs are discouraged unless they are essential to the article. Photos with identifiable persons must have a signed consent by the person photographed forwarded to the editor via first class mail. Diagrams must have permission from original author. This is the most important part of the article. In terms of space and word count this should be longer than the case presentation. End the discussion with a summary lesson you learned from the case, perhaps what you would do differently if you had it to do over again.
[space]
References (bold)
[space]
A minimum of 5 references is recommended, with a maximum of 8 allowed. One textbook may be used as a reference – it must be the most recent edition. All references should be no older than 8 years, except for seminal works essential to the topic. This is also an exercise in searching for and evaluating current literature.
[space]
Mentor: (bold, followed by mentor name and credentials in normal text)
E-mail address: (normal text, will be removed prior to publication)

EBP Analysis Reports - Evidence-based practice analysis reports are limited to 3000 words. Please do not include an abstract. The report should provide a critical evaluation of a practice pattern in the form of a clinical question about a specific intervention, population, and outcome. The manuscript should:

1. Articulate the practice issue and generate a concise question for evidence-based analysis. A focused foreground question following either the PICO or SPICE format should be used.
2. Describe the methods of inquiry used in compiling the data.
3. Critically analyze the quality of research reviewed and applicability to different practice settings.
4. Draw logical conclusions regarding appropriate translation of research into practice.

The same general format guidelines apply with the exception of the section headings as below. Textbooks and non-peer reviewed internet sources may not be used, and sources of reference should be less than 8 years old unless they are seminal works specifically related to your topic of inquiry. A maximum of 16 references is allowed.

Heading

Introduction (bold)
[space]
Briefly introduce the reader to the practice issue or controversy, describe the scope or significance or problem, and identify the purpose of your analysis. Describe the theoretical, conceptual, or scientific framework that supports your inquiry.
[space]
Methods (bold)
[space]
Include the format used for formulating the specific question you seek to answer, search terms and methods used, and levels of evidence.
[space]
Literature Analysis (bold)
[space]
Analyze and critique the literature relevant to your question, determining scientific credibility and limitations of studies reviewed. Your synthesis table is included in this section. Your review and discussion of the literature should logically lead to support a practice recommendation. Subheadings may be used if desired.
[space]
Conclusions (bold)
[space]
Summarize the salient points that support the practice recommendation and make research-supported recommendations that should improve the practice issue, while also acknowledging any limitations or weaknesses
[space]
References (bold, 16 maximum)
[space]
Mentor: (bold, followed by mentor name and credentials in normal text)
E-mail address: (normal text, will be removed prior to publication)

Evidence Based Practice Project Abstracts - Evidence-based practice project abstracts are limited to 600 words. References do not impact the word count - a maximum of 5 are allowed. Note that the abstract is different from a project proposal. The following format should be used:

Heading

Introduction (bold)
[space]
A brief introductory paragraph including purpose (what change is intended) and rationale (why change is needed/evidence to support the change) here.
[space]

Design and Methods (bold)
[space]
Include population, intervention, and measures
[space]
Outcome (bold)
[space]
Present results from statistical analysis – do not justify or discuss here.
[space]
Conclusion (bold)
[space]
Discuss results (implications). Optionally include limitations, suggestions for future projects/research.
[space]
References (bold, 5 maximum)
[space]
Mentor: (bold, followed by mentor name and credentials in normal text)
E-mail address: (normal text, will be removed prior to publication)

Research Abstracts - Research abstracts are limited to 600 words. References do not impact the word count - a maximum of 5 are allowed. Note that the abstract is different from a research proposal. The following format should be used:

Heading

Introduction (bold)
[space]
A brief introductory paragraph including purpose and hypotheses.
[space]
Methods (bold)
[space]
Include sample and research design
[space]
Results (bold)
[space]
Present results from statistical analysis – do not justify or discuss here.
[space]
Discussion (bold)
[space]
Discuss results (implications, limitations, suggestions for future research)
[space]
References (bold, 5 maximum)
[space]
Mentor: (bold, followed by mentor name and credentials in normal text)
E-mail address: (normal text, will be removed prior to publication)

Letters to the Editor - Students may write letters to the editor topics of interest to other students. Topics may include comments on previously published articles in this journal. Personally offensive, degrading or insulting letters will not be accepted. Suggested alternative approaches to anesthesia management and constructive criticisms are welcome.
The length of the letters should not exceed 100 words and must identify the student author and anesthesia program.

AMA MANUAL OF STYLE

The following is brief introduction to the *AMA Manual of Style* reference format along with some links to basic, helpful guides on the internet. The website for the text is http://www.amamanualofstyle.com/oso/public/index.html. It is likely your institution's library has a copy on reserve. Some helpful websites are listed below:
https://guides.nyu.edu/amastyle
https://owl.english.purdue.edu/owl/resource/1017/01/

Journal names should be in *italics* and abbreviated according to the listing in the PubMed Journals Database. The first URL below provides a tutorial on looking up correct abbreviations for journal titles; the second is a link to the PubMed where you can perform a search.
http://www.nlm.nih.gov/bsd/viewlet/search/journal/journal.html
http://www.ncbi.nlm.nih.gov/pubmed

The International Student Journal of Nurse Anesthesia (ISJNA) is not listed in the PubMed Database. For the purpose of citing the ISJNA *in this Journal* use "**Int Student J Nurse Anesth**" as the abbreviation.

Journals - A comma is placed after the first initials until the last author, which has a period. If there are six or less authors **cite all six**. If there are more than six authors **cite only the first three** followed by "et al." Only the first word of the title of the article is capitalized. The first letters of the major words of the journal title are capitalized. There is no space between the year, volume number, issue number, and page numbers. If there is no volume or issue number, use the month. If there is an issue number but no volume number use only the issue number (in parentheses). Page numbers are inclusive - **do not omit digits** (note - some online journals do not use page numbers). Some journals may be available both as hard copies and online. When referencing a journal that has been accessed online, the DOI (digital object identifier) or PMID (PubMed identification number) should be included (see example below).

Journal, 6 or fewer authors:
Han B, Liu Y, Zhang X, Wang J. Three-dimensional printing as an aid to airway evaluation after tracheotomy in a patient with laryngeal carcinoma. BMC Anesthesiol. 2016;16(6). doi:10.1186/s12871-015-0170-1.

Journal, more than 6 authors:
Chen C, Nguyen MD, Bar-Meir E, et al. Effects of vasopressor administration on the outcomes of microsurgical breast reconstruction. *Ann Plast Surg*. 2010;65(1):28-31. PMID: 20548236.

Elayi CS, Biasse L, Bai R, et al. Administration of isoproterenol and adenosine to guide supplemental ablation after pulmonary vein antrum isolation. *J Cardiovasc Electrophysiol*. 2013;24(11):1199-1206. doi: 10.1111/jce.12252.

Electronic references - Only established, peer-reviewed sources may be referenced. Please do not reference brochures, fact sheets, or informational websites where a peer-review process cannot be confirmed. The URL must be functional and take the reader directly to the source of the information cited. The accessed date may be the only date available.

Author (or if no author, the name of the organization responsible for the site). Title. *Name of Website*. Year;vol(issue no.):inclusive pages. URL. Published [date]. Updated [date]. Accessed [date].

Examples:
Kamangar N, McDonnell MS. Pulmonary embolism. *eMedicine*. http://www.emedicine.com/med/topic1958.htm. Updated August 25, 2009. Accessed September 9, 2009

Howlader N, Noone AM, Krapcho M, Garshell J, Miller D, et al. SEER Cancer statistics review, 1975-2012. National Cancer Institute. http://seer.cancer.gov/csr/1975_2012/. Published April 2015. Updated November 18, 2015. Accessed February 29, 2016.

Textbooks - There are two types of books – 1) those that are fully authored by one or more individuals, and 2) those that are edited by one or more individuals, with chapters authored by different individuals. Edited textbooks give

primary credit to the chapter authors, who are listed first, and the inclusive page numbers of the entire chapter are provided at the end. Textbooks that are authored do not have different chapter authors and the chapter titles are not listed, but the inclusive page numbers where the information was found are provided, unless the entire book is cited.

Authored text:
Shubert D, Leyba J, Niemann S. *Chemistry and Physics for Nurse Anesthesia*. 3rd ed. New York, NY: Springer; 2017:405-430.

Chapter from an edited text:
Pellegrini JE. Regional anesthesia. In Nagelhout JJ, Elisha S, eds. *Nurse Anesthesia*. 6th ed. St. Louis:Elsevier; 2017:1015-1041.

SUBMISSION CHECK LIST

___ **Adheres to AMA Manual of Style and all other format instructions**
___ Total word count not exceeded (1400 for case report, 600 for abstracts, 3000 for EBPA report)
___ The item is one continuous Word document without artificially created page breaks
___ All matters that are not common knowledge to the author are referenced appropriately
___ Generic names for drugs and products are used throughout and spelled correctly in lower-case
___ Units are designated for all dosages, physical findings, and laboratory results
___ Endnotes, footnotes not used
___ Jargon/slang is absent

Heading
___ Concise title less than 70 characters long
___ Author name, credentials, nurse anesthesia program, graduation date and email are included
___ Three to five **Keywords** are provided

Case Report
___ Introduction is less than 100 words.
___ Case Report section states only those facts vital to the account (no opinions or rationale)
___ Case report section is 400-600 words and not longer than the discussion
___ Discussion section is 600-800 words
___ Discussion of the case management is based on a review of current literature
___ Discussion concludes with lessons learned and how the case might be better managed in the future

Abstracts
___ The 600 word count maximum is not exceeded
___ Appropriate format used depending on type of abstract (research vs. EBP project)

EBPA Report
___ The 3000 word count maximum is not exceeded
___ A critical evaluation of a practice pattern in the form of a precise clinical question about a specific intervention, population, and outcome is presented
___ A focused foreground question following either the PICO or SPICE format is used
___ Includes Introduction, Methodology, Literature Analysis (with synthesis table), and Conclusion sections

References
___ Adheres to AMA Style format
___ Reference numbers are sequenced beginning with 1 and superscripted
___ References are from anesthesia and other current (within past 8 years) primary source literature
___ Journal titles are abbreviated as they appear in the PubMed Journals Database
___ Number of references adheres to specific item guidelines (1 textbook allowed for case reports only)
___ Internet sources are currently accessible, reputable, and peer reviewed

Transmission
___ The article is sent as a attachment to **INTSJNA@AOL.COM**
___ The file name is correctly formatted (e.g. PedsPain_Smyth_GU_Pearson_5.19.09)
___ Item is submitted by the mentor
___ Subject heading format - ISJNA Submission_submission type_author last name_mentor last name