

Volume 15 Issue 1 Spring 2016

The International Student Journal of Nurse Anesthesia

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Preventing OR Fires



INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA
Vol. 15 No. 1 Spring 2016

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Front Cover:

Graduate students enrolled in the Florida Gulf Coast University Nurse Anesthesia program benefit from curricular activities in simulation and scholarship. On the left is recent graduate Kristen Frye practicing simulated ultrasound-guided arterial line placement, and above Ginette Peterson performs a simulated subarachnoid block placement with the assistance of Katherine Register. Pictured bottom right are Radhika Patel, BSN (right) and faculty member Ann Miller, CRNA, DNP (left) next to Ms. Patel's Blue Ribbon winning poster at the 2015 AANA Foundation State of the Science Poster Session.

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Anesthetic Management of Carnitine Palmitoyltransferase Deficiency

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Keywords: carnitine palmitoyltransferase deficiency, malignant hyperthermia, non-triggering anesthesia, total intravenous anesthesia, and rhabdomyolysis

Carnitine Palmitoyltransferase (CPT) is an enzyme that is essential for fatty acid oxidation. Long chain fatty acids attach to carnitine and enter the mitochondria. Once inside, CPT removes carnitine to allow for fatty acid oxidation. Mutations of the CPT gene decrease the activity of the CPT enzyme. During periods of fasting, stress, and exercise muscle cells are unable to utilize fatty acids as an energy source, glucose stores become depleted, and rhabdomyolysis ensues.¹ In recent studies, CPT deficiency has been associated with causing signs of a malignant hyperthermia (MH) like syndrome during anesthesia.²

Case Report

A 31-year-old, 168 cm, 69 kg, Caucasian female presented to an ambulatory facility for a diagnostic laparoscopy, possible bilateral salpingo-oophorectomy, and possible laparotomy. The patient was recently diagnosed with endometriosis and was undergoing a diagnostic laparoscopy for a full evaluation of her disease. Her medical history was significant for a mild myopathy form of CPT II deficiency with a subsequent history of severe rhabdomyolysis, and a decrease in exercise tolerance. Her current home medications consisted of oxycodone hydrochloride, acetaminophen, magnesium supplements, and levocarnitine. Ibuprofen was listed as a drug allergy with the patient stating a reaction of severe muscle weakness. The patient's surgical history included a tonsillectomy with adenoidectomy and bilateral thigh fasciotomies. None of these surgical interventions were associated with any anesthetic complications. A complete metabolic panel and a complete blood count were obtained. All lab values were within normal limits, and the patient reported no oral intake for 8 hours. The patient's preoperative blood glucose level was 110 mg/dL.

A 20 gauge peripheral intravenous (IV) line was inserted into the dorsal side of the patient's left hand and a liter of 10 % dextrose IV solution was initiated as a continuous infusion. The patient was then administered IV midazolam 2 mg preoperatively. The patient was scheduled as the first case in the operating room (OR). Hospital policy and recommendations provided by the Malignant Hyperthermia Association of the United States (MHAUS) were followed to prepare the OR and anesthetic equipment. Once in the operating room noninvasive monitors were applied, including core temperature. Denitrogenation was initiated with 100% oxygen via facemask using an oxygen flow of 12 L/min and having the patient take six tidal volume breaths. General anesthesia was induced with IV lidocaine 60 mg, propofol 200 mg, fentanyl 100 mcg. A baseline train of four (TOF) was assessed over the patient's ulnar nerve, with a pre-induction baseline of four out of four twitches. Rocuronium 50mg was then given intravenously. Once apnea ensued, mask ventilation with 100% oxygen was initiated. Direct laryngoscopy with a MacIntosh three blade was performed and a Grade I view was obtained. A 7.0 mm endotracheal tube was advanced through the glottic opening and secured at 20 cm at the teeth. Positive

bilateral breath sounds were auscultated and positive end-tidal carbon dioxide (ETCO₂) was noted. Volume control ventilation was utilized to maintain an ETCO₂ of 30 to 33 mmHg. A non-triggering anesthetic was maintained through total intravenous anesthesia (TIVA) consisting of a propofol infusion at 200 mcg/kg/min. A total of morphine 2mg and fentanyl 300 mcg were administered IV throughout the case. The previous one liter bag of 10% dextrose was discontinued after the patient had received approximately 500 milliliters. A one liter bag of lactated ringer's was then initiated intravenously, and used as the continuous maintenance fluid.

At the conclusion of the case the patient's TOF was documented as three out of four twitches. The neuromuscular blockade was antagonized with neostigmine 3mg and glycopyrrolate 0.6mg IV and TIVA was discontinued. Once the patient was noted to begin to breathe over the set respiratory rate on the ventilator, the patient was placed on a manual spontaneous mode. The patient was able to follow simple commands, demonstrated a regular respiratory rate with tidal volumes of 500 milliliters, and maintained a sustained head lift for 5 seconds. At this point the patient's oropharynx was suctioned, the pilot balloon was deflated, and the endotracheal tube was removed. A simple facemask with 6 liters of oxygen flow was then applied to the patient's face as she was transported to the post anesthesia care unit (PACU). The patient was discharged to home later that day after following the MHAUS post operative procedure, which include a minimum of one hour monitoring in PACU with vital signs being documented at least every 15 minutes, with an additional 1 hour monitoring in phase 2 PACU. Monitoring for the absence of myoglobin using a chemstrip is also recommended but was not completed for this patient.³

Discussion

Carnitine Palmitoyltransferase II (CPT II) deficiency is an autosomal recessive disorder involving oxidation of long chain fatty acids. The disorder was first described in 1973 and since then more than three hundred case reports have been published. Little is known about the mechanism of CPT II deficiency. It is thought that with a CPT II deficiency the body cannot metabolize fatty acids as an energy source. Therefore, during periods of fasting, exercise, and stress, including infection, cold, and emotional stress, muscle cells become depleted of glucose and rhabdomyolysis ensues.¹ Common medications such as non-steroidal anti-inflammatory drugs like ibuprofen, high doses of diazepam, valproate sodium, and the use of general anesthesia have all been associated with triggering a CPT II attack.⁴

There are currently three distinct and isolated clinical manifestations of CPT II deficiency. These include: mild myopathy, a severe infantile disorder, and a fatal neonatal form. Both the age of onset and involvement of organ systems are considered during diagnosis.^{4,5} The mild myopathy form of CPT II deficiency is characterized by recurrent episodes of myalgia and weakness. Exercise was found to be the most common triggering factor for myalgia, noted in 62% of CPT II deficiency patients.¹ The mild myopathy form of CPT II can also be accompanied by rhabdomyolysis, which can lead to myoglobinuria and renal failure. Myoglobinuria is known to be the hallmark of mild myopathy CPT II deficiency in 62% of patients. The myopathic form of CPT II deficiency can manifest from infancy to adulthood and is the most common disorder of lipid metabolism.¹

The severe infantile form of CPT II often occurs within the first year of life and is characterized by liver failure, cardiomyopathy, seizures, hypoketotic hypoglycemia, peripheral myopathy, abdominal pain, and headaches. The fatal neonatal form often presents with many of the same characteristics as the severe infantile form except that with the fatal neonatal form facial abnormalities, cardiac arrhythmias, and seizures after fasting or an infection often occur. The fatal neonatal form of CPT II deficiency also presents within days after birth. Both the infantile and neonatal forms of CPT II deficiency are associated with an increased risk of demise when compared to the mild myopathy form.¹ Only eighteen patients with the fatal neonatal form and twenty-eight patients with the severe infantile form of CPT II deficiency have been diagnosed and reported. The increase in mortality is due to the dramatic and multi system effects that the neonatal and infantile form of CPT II cause to the pediatric patient.⁴

Due to its signs of myalgia, muscle weakness, myoglobinuria, and rhabdomyolysis, CPT II deficiency has been linked with MH. Unlike CPT II, MH is an autosomal dominant genetic disorder of muscle hypermetabolism that occurs when exposed to inhalational agents and succinylcholine. Early signs of MH include: elevated carbon dioxide production, increased oxygen consumption, metabolic and respiratory acidosis, diaphoresis, tachycardia, cardiac arrhythmias, masseter spasm, and generalized muscle rigidity. Later signs of MH include: rapid increase in core body temperature, hyperkalemia, elevated blood creatine phosphokinase and myoglobin levels, myoglobinuria, cardiac arrhythmias leading to cardiac arrest, and disseminated intravascular coagulation.⁶ Like MH, CPT II can lead to elevated creatine phosphokinase and myoglobin levels. It can also lead to myoglobinuria, cardiac arrhythmias, and even cardiac arrest if not ultimately treated with dantrolene. However, the most important trigger for CPT II deficiency is exercise or a stress induced event.²

The American Society of Anesthesiologists and the American Association of Nurse Anesthetists have not indicated anesthetic measures that should be taken with CPT II patients. However, knowing the pathophysiology and signs and symptoms of a patient with a CPT II deficiency it would be prudent to undertake the same MH precautions as provided by the hospital and/or the Malignant Hyperthermia Association of the United States (MHAUS).³ Anesthesia professionals should consider the triggers for a CPT II attack and avoid any undue emotional stress for the patient. Warming the operating room and providing a continuous means of providing warmth to the patient should also be considered. Since most individuals requiring general anesthesia must fast for a prolonged period of time and low nutritional intake can be a trigger for a CPT II attack, consider infusing a dextrose containing solution and monitoring glucose levels throughout the case. High doses of nonsteroidal anti-inflammatory drugs, diazepam, and valproate sodium should also be avoided. Although there has been no direct link to specific muscle relaxants and inhaled anesthetics, they should still be avoided in patients with a CPT II deficiency.

In this particular case presentation, the patient was previously diagnosed with a mild myopathy form of CPT II deficiency. Therefore, a non-triggering anesthetic was provided. A forced air-warming device was used throughout the case to keep the patient normothermic, and glucose monitoring was utilized every hour throughout the case to maintain blood glucose levels between 100-180mg/dL according to the discretion of the anesthesia team. In conclusion, anesthesia professionals need to know how to optimize a CPT II deficient patient prior to surgery, how to prepare the operating room for such a patient, and recognize the signs of a CPT II attack, and

how to treat such an incidence.

References

1. Joshi PR, Deschauer M, Zierz S. Carnitine palmitoyltransferase II (CPT II) deficiency: Genotype-phenotype analysis of 50 patients. *J Neurol Sci.* 2014;338(1-2):107-11. doi:10.1016/j.jns.2013.12.026.
2. Hogan KJ, Vladutiu GD. Malignant hyperthermia-like syndrome and carnitine palmitoyltransferase II deficiency with heterozygous R503C mutation. *Anesth Analg.* 2009;109(4):1070-2. doi:10.1213/ane.0b013e3181ad63b4.
3. Post Operative Procedure. *Malignant Hyperthermia Association of the United States.* 2016. <http://www.mhaus.org>.
4. Wieser T. Carnitine palmitoyltransferase II deficiency. *GeneReviews.* 2014;1-40. <http://www.ncbi.nlm.nih.gov/books/NBK1253>. Updated May 15, 2014. Accessed June 2, 2014. PMID:20301431.
5. Isackson PJ, Bennett MJ, Lichter-konecki U, et al. CPT2 gene mutations resulting in lethal neonatal or severe infantile carnitine palmitoyltransferase II deficiency. *Mol Genet Metab.* 2008;94(4):422-7. doi:10.1016/j.ymgme.2008.05.002.
6. Glahn KP, Ellis FR, Halsall PJ, et al. Recognizing and managing a malignant hyperthermia crisis: Guidelines from the European Malignant Hyperthermia Group. *Br J Anaesth.* 2010;105(4):417-20. doi:10.1093/bja/aeq243.

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Preventing Operating Room Fires in Anesthesia

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Keywords: anesthesia; operating room fire; fuels; oxidizers; ignition

Operating room fires are thought to be a rare occurrence. According to the National Center for Health Statistics there are “approximately 600 surgical fires each year, with some resulting in injury, disfigurement, or even death”.¹ In recent studies, a fire triad has been identified which includes fuels, oxidizers and ignition sources.² All are prevalent in any operating room. The most frequently reported sources for ignition include electrocautery (68%) and laser equipment (13%).² All members of every surgical team must be cognizant of potential fire risks.

Case Report

A 78-year-old female diagnosed with squamous cell carcinoma of the right alveolar ridge presented for a partial mandibulectomy with plating, radial neck dissection, free flap of the radial forearm and tracheostomy. The malignant mass lacked outward visualization. Neck range of motion was not limited and the patient denied pain, paresthesia or numbness with movement.

The patient had a medical history significant for atrial fibrillation, hypothyroid and lymphoma treated with chemo radiation therapy.

The patient weighed 72 kg and was 71 cm tall. A recent 12-lead electrocardiogram (ECG) showed normal sinus rhythm. Oxygen saturation was 98% without supplemental oxygen. Anesthetic airway assessment was unremarkable. Auscultation of the lungs revealed diminished breath sounds in the bases. Endotracheal tube and tracheostomy placement were planned with the anesthesiologist with the goal of maintaining the patient's airway while optimizing surgical exposure.

Pre-medication with midazolam 2 mg was administered through a 20-gauge peripheral intravenous (IV) catheter that was placed in the holding area. Once in the operating room, non-invasive monitors were applied with the patient in the supine position. Pre-oxygenation occurred for three to five minutes with O₂ 10 L/min via a bag valve mask. General anesthesia was induced with fentanyl 50 mcg, lidocaine 40 mg, propofol 120 mg, and rocuronium 50 mg IV. Correct placement of a 7.0 endotracheal tube (ETT) was placed without complication. Inhalation anesthesia was maintained by administering isoflurane 1% and O₂ at 2 L/min. In preparation for tracheostomy placement, fresh gas flows were decreased and the fraction of inspired oxygen (FiO₂) was decreased to 0.38. Sterilization with betadine was used and sterile surgical drapes were placed two to three inches from the tracheal surgical site.

Opening of the trachea was performed by the surgeon, and electrocautery was utilized to prevent bleeding. Once direct visualization of the seated endotracheal tube was made, the tracheostomy tube was placed and the endotracheal tube was slowly retracted by the nurse anesthetist following the surgeon's instruction. Correct placement of the tracheostomy tube was confirmed by bilateral chest auscultation, chest rise and positive end tidal carbon dioxide. The surgical procedure progressed smoothly and lasted approximately 8 hours. The patient was transferred to the intensive care unit for observation and airway management. She was discharged to home after an uneventful post-operative course.

Discussion

Surgical fires with airway involvement are more common than once thought. Three principles (known as the fire triad) have been identified as risk factors: fuels, oxidizers and ignition sources.² Members of the surgical team need to understand the fire triad and how to respond effectively when a fire develops in order to ensure patient and staff safety. Fuels such as towels, gowns, drapes, sponges, petroleum and surgical skin prepping agents should be monitored by the surgical technicians and surgeons. Electrocautery, lasers and fiberoptic light are ignition sources must be well controlled by surgeons, whereas oxidizers, such as fresh gas flows, undergo continuous manipulation by anesthesia professionals.²

To ensure patient safety, pre-procedure time outs have been developed to incorporate a fire risk assessment. A fire risk assessment takes very little time and should be implemented before the start of every surgical procedure to increase intraoperative awareness and mitigate hazards of associated risk factors.³ Some facilities have created risk evaluation scores ranking from 1 (low risk) to 3 (high risk). One point is awarded for the presence of: procedure site above the xiphoid

process, open oxygen source (facemask or nasal cannula) and ignition source (cautery, laser or fiberoptic light).⁴ Although a scoring system was not utilized in preparation for this case, the anesthesia professionals verbally identified fire risk factors before the start of the procedure.

Anesthesia professionals have been identified as key players in optimizing fire safety.⁵ The circulating nurse is often busy coordinating procedural activities. Surgical technicians are pre-occupied with maintaining instruments for the surgeon's use. The surgeon and resident surgeons are focused on human anatomy and the procedure at hand. Anesthesia professionals have the greatest advantage of focusing on the patient.⁶ However, the anesthesia practitioner is just one part of the surgical team and all staff members are obligated to act on behalf of patient safety.

Oxygen has been identified as the key contributor to fire in the operating room. The American Society of Anesthesiologists (ASA) supports that supplemental O₂ is not always a medical necessity and its use should be limited or omitted if unnecessary.⁶ Containment of O₂ should also be optimized by using room air via a well seated tracheal cuff. All surgical cases and procedures do not meet requirements for endotracheal tube placement or limited oxygen. Sedation cases may only warrant nasal cannulas and facemasks which contribute to an oxygen rich environment.⁴

During placement of the tracheostomy in this case, steps were taken to prevent airway fire. Anesthesia professionals maintained the FiO₂ below 0.40 and identified that surgical draping was maintained at an appropriate distance from the surgical site. However, site prepping with povidone-iodine (Betadine) was not identified as being dry before electrocautery began by the operating technologist or nurse. Povidone-iodine (Betadine) is "slightly flammable to flammable in the presence of heat".⁷ In addition, members of the surgical team did not state surgical factors present which would contribute to the risk of operating room combustion. To demonstrate OR fire knowledge, nurses could state the risk evaluation score for the procedure and specific fuels, oxidizers and electrical currents being utilized.³ Based on this surgical case, team members outside of anesthesia should not be assumed to possess the knowledge to ensure fire safety. Although risk factors were identified by anesthesia professionals, understanding of the fire triad and combustion were not clearly demonstrated by the other surgical team members involved in this case report.

This surgical case had the potential for developing an airway fire. Oxygen, surgical skin prepping agents, electrocautery, sterile draping and sponges were all used during tracheostomy placement. Active contributors to the fire triad: fuels, oxidizers and ignition sources were present. Improvements could have been made in this case by surgeons, surgical technicians, nurses and anesthesia professionals by actively identifying case specific contributors to combustion. This could be easily carried out by verbally utilizing the fire risk assessment scoring system. If utilized, this case would have received 3 points, indicating a high procedure risk.

The case ended in the absence of an airway fire, but this case report identifies valuable lessons. This case highlights areas for improvement by anesthesia professionals and all members of the surgical team to ensure continued safety for all patients in the future.

References

1. Durso F. Operation fire safety. *NFPA J.* January-February 2012. <http://www.nfpa.org/newsandpublications/nfpa-journal/2012/january-february-2012/features/operation-fire-safety>. Accessed February 14, 2016.
2. Moskowitz M. Fire in the operating room during open heart surgery: a case report. *AANA J.* 2009;77(4):261-264.
3. Hughes, AB. Implementing AORN recommended practices for a safe environment of care. *AORN J.* 2013;98(2):153-163. doi: 10.1016/j.aorn.2013.06.007.
4. Anonymous. Fire prevention: avoid oxygen to face. *OR Manager.* 2010;26(1):19-21.
5. Hart SR, Yajnik A, Ashford J, Springer R, Harvey S. Operating room fire safety. *Ochsner J.* 2011 Spring; 11(1): 37-42.
6. American Society of Anesthesiologists. Practice advisory for the prevention and management of operating room fires: a report by the American Society of Anesthesiologists task force on operating room fires. *Anesthesiology.* 2008;108:786-801. doi:10.1097/ALN.0b013e31827773d2
7. Science Lab. Material Safety Data Sheet, Povidone-Iodine MSDS. www.sciencelab.com/msds.php?msdsId=9924704. Published October 9, 2005. Updated May 21, 2013. Accessed February 14, 2016.

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Airway Management of a Tracheostomy Patient Undergoing General Anesthesia

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Keywords: general anesthesia, tracheostomy, airway management, intraoperative, endotracheal tube

Intraoperative airway management of a patient presenting with a tracheostomy is an important part of ensuring a good patient outcome but sometimes anesthesiologists make the mistake of assuming that the tracheostomy tube can simply be connected to the ventilator and the patient anesthetized. Variables such as stoma maturity and the type of tracheostomy tube will have an impact on how the airway is managed intraoperatively. It is imperative that the anesthesiologist is aware of safe airway management techniques for a tracheostomy patient presenting for general anesthesia, but there is scant evidence-based research on the topic.

Case Report

A 58-year-old, 58 kg, 163 cm female presented for a left cranioplasty due to a cranial deformity resulting from a prior craniotomy. The patient's past medical history included hypertension, coronary artery disease, multiple intracranial aneurysms, left middle cerebral artery (MCA) aneurysm rupture, subarachnoid hemorrhage (SAH), right-sided hemiparesis, and methamphetamine abuse. Six months prior to presenting for the cranioplasty, the patient

underwent a craniotomy for left temporal lobe hematoma evacuation due to a SAH and was transferred to another facility four days later for coiling of a giant left MCA aneurysm. Post-coiling, the patient remained in a coma and underwent a tracheostomy and percutaneous endoscopic gastrostomy tube placement. Prior to cranioplasty the patient presented alert and oriented and able to speak in 2-3 word sentences. The patient did not have any anesthetic complications during previous surgeries. The patient denied any drug allergies and current medications included pravastatin, levetiracetam, lorazepam, and ranitidine. The patient's subcutaneous heparin injections were discontinued one week prior to surgery.

The patient presented with a Shiley 6.5 uncuffed tracheostomy tube (Covidien Healthcare, Minneapolis, MN) that was capped. Glycopyrrolate 0.2 mg and ranitidine 50 mg were administered in the preoperative holding area. The patient was transferred to the operating room where noninvasive monitors and a Bispectral Index (BIS) monitor (Covidien Healthcare, Minneapolis, MN) were applied. Lidocaine 4% (80 mg) was provided via a laryngotracheal anesthesia kit through the patient's tracheostomy tube by removing and then replacing the cap. No sedation was given because the anesthesia team felt that if the patient became sleepy, there could be difficulty securing the airway.

The patient was preoxygenated with O₂ 10 L/min for 5 minutes via face mask. With the patient awake and breathing spontaneously, the patient's Shiley 6.5 tracheostomy tube was exchanged for a size 6.5 reinforced endotracheal tube (ETT). The ETT was secured and correct placement of the ETT was confirmed via capnography and auscultation of equal bilateral breath sounds.

Induction commenced with propofol 100 mg and sevoflurane 1.4% inspired concentration in a mixture of O₂ 1 L/min and air 1 L/min. Upon loss of lash reflexes, the patient's eyes were lubricated and a transparent dressing was applied to each eye. After confirming the ability to manually ventilate the patient's lungs, rocuronium 30 mg and esmolol 10 mg were administered prior to suturing of the ETT to the skin.

An arterial line was placed in the left radial artery under sterile technique and secured. Sevoflurane was continued and the inspired concentration titrated to maintain a BIS score of 40-60 throughout surgery. An additional 10 mg dose of rocuronium was administered to maintain 1-2 twitches on the train-of-four stimulation; the total amount of rocuronium administered was 40 mg. The patient was breathing spontaneously on pressure support when the surgeon started to close the skin incision and glycopyrrolate 0.6 mg IV and neostigmine 4 mg IV were given to antagonize the neuromuscular blockade; sustained tetanus for 5 seconds at 50 hz was noted. With the patient awake and spontaneously breathing, the ETT was removed and replaced with the patient's uncuffed tracheostomy tube. After auscultatory confirmation of bilateral breath sounds, the patient was transferred to the post-anesthesia care unit.

Discussion

Very little literature exists regarding the intraoperative airway management of patients who present with a tracheostomy. However, resources focusing on the different types of tracheostomy tubes, and the indications for each type, are available. By knowing the type of tracheostomy tube the patient presents with and understanding its use, the anesthetist can formulate a safe plan for

intraoperative airway management. Even without expert knowledge on the various types of tracheostomy tubes that exist, simply determining whether the patient's tracheostomy tube is cuffed or uncuffed will enable the anesthetist to create a plan for safe airway management.

Tracheostomy tubes can be grouped into two main categories: cuffed and uncuffed; within these two categories, tracheostomy tubes can be further subdivided into single-cannula or dual-cannula.¹ Additional types of tracheostomy tubes such as fenestrated, metal (as opposed to plastic) tubes, and adjustable length tubes are beyond the scope of this discussion. The ability to ventilate the patient via a tracheostomy tube is the main concern of anesthesia professionals and knowing whether or not the tracheostomy tube is cuffed must be ascertained prior to beginning an anesthetic. Cuffed tracheostomy tubes are used when a patient cannot protect his/her airway and are always utilized for the first several days after tracheostomy creation.¹ A cuffed tracheostomy tube allows the patient to receive positive pressure ventilation when the cuff is inflated because a seal is created to prevent air from escaping around the tube in the trachea.² If the tracheostomy tube is uncuffed, positive pressure ventilation is not possible and an alternative method must be used to secure the airway. This is why an ETT was utilized rather than the tracheostomy tube in this particular case.

While a cuffed tracheostomy tube may decrease the risk of aspiration¹ it does not necessarily prevent aspiration. Therefore, measures should be taken to prevent aspiration in patients who cannot protect their airway.² Preoperative ranitidine was administered in anticipation of using lidocaine to blunt the patient's airway reflexes. Due to the increased risk of aspiration from blunted airway reflexes, ranitidine was given to increase pH and reduce gastric volume.³

Another important factor to consider is whether the patient's stoma has healed adequately to allow for removal of the tracheostomy tube in order to exchange it for an ETT. The time required for stomal maturity is controversial. Vallamkondu and Visvanathan¹ suggest as few as 2-3 days after tracheostomy placement,¹ yet others state that the tube should not be changed until 7-10 days post-tracheostomy.⁴ Mitchell et al⁷ varies their recommendation depending on how the tracheostomy was performed; they recommend waiting 3-7 days after surgical tracheostomy or as long as 10-14 days after percutaneous dilational tracheostomy. Many authors that state a time frame for the first tracheostomy tube change also mention that there is no data to support the suggested time frames. Additionally, there are no recommendations on how long to wait after tracheostomy creation prior to exchanging the tracheostomy tube for an ETT to perform general anesthesia. Since this patient presented for surgery six months after the tracheostomy creation, the anesthesia team determined that it would be safe to exchange the tubes.

Since no guidelines exist for intraoperative airway management for patients with tracheostomies, induction and emergence were undertaken with the utmost caution and the patient was treated as a potentially difficult airway. Preoperative sedation was avoided and lidocaine was used to topically anesthetize the patient's trachea so she could remain awake and spontaneously breathing while the airway was secured. Additionally, glycopyrrolate was given in the preoperative holding area to dry any airway secretions. These interventions comply with the anticipated difficult airway strategy described by Law et al.⁵

Induction of anesthesia occurred after ETT placement confirmation, thus preventing any difficulties that could arise due to airway musculature relaxation. Extubation was undertaken with similar caution since a decreased level of consciousness and decreased muscular strength can lead to airway compromise.⁶ Due to this risk, 3 mg of neostigmine was given to antagonize the neuromuscular blockade with subsequent full return of muscle strength as evidenced by sustained tetany and adequate spontaneous ventilation. Additionally, we waited for the patient to be fully awake and following commands prior to removing the ETT and replacing the tracheostomy tube.

Management of a tracheostomy patient requiring general anesthesia is not necessarily difficult, but it requires the anesthesia professional to ascertain the type of tracheostomy tube in place and the age of the stoma prior to being able to formulate an anesthetic plan. While this case study may serve as a starting point for intraoperative airway management in tracheostomy patients, further research is needed to establish a set of evidence-based practice guidelines on this topic.

References

1. Vallamkondu V, Visvanathan V. Clinical review of adult tracheostomy. *J Perioperative Pract.* 2011;21(5):172-176.
2. Morris, LL. Fitting and Changing a Tracheostomy Tube. In: Morris LL, Afifi MS, eds. *Tracheostomies: The Complete Guide.* New York, NY. Springer Publishing Company, LLC. 2006;115-157.
3. American Society of Anesthesiologists Committee. Practice guidelines for preoperative fasting and the use of pharmacologic agents to reduce the risk of pulmonary aspiration: Application to healthy patients undergoing elective procedures: An updated report by the American Society of Anesthesiologists Committee on Standards and Practice Parameters. *Anesthesiology.* 2011;114:495-511.
4. Russell, C. Tracheostomy Tubes. In: Russell C, Matta B, eds. *Tracheostomy: A Multiprofessional Handbook.* Cambridge, Great Britain. Cambridge University Press; 2004;85-114.
5. Law JA, et al. The difficult airway with recommendations for management- Part 2- The anticipated difficult airway. *Can J Anaesth.* 2013;60:1119-1138.
6. Popat M, Mitchell V, Dravid R, Patel A, Swampillai C, Higgs A. Difficult airway society guidelines for the management of tracheal extubation. *Anaesthesia.* 2012; 67:318-340.
7. Mitchell RB, Hussey HM, Setzen G, et al. Clinical consensus statement: Tracheostomy care. *Otolaryngol Head Neck Surg.* 2013;148(1):6-20.

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Causes of Reintubation in the Postanesthesia Care Unit

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Keywords: reintubation, hypoxia, recovery unit, postanesthesia care unit

Reintubation in the postanesthesia care unit (PACU) is an adverse event and can result in increased length of stay in the PACU, intensive care unit (ICU) admission, prolonged mechanical ventilation, increased pulmonary complications and increased economic burden. The causes are multifactorial, and fall into two broad categories: patient and/or anesthesia related. Patients with morbid obesity and obstructive sleep apnea (OSA) are at increased risk of complications such as postoperative respiratory depression, hypoxia, and reintubation.¹ The anesthesia related causes can involve residual neuromuscular blockade and respiratory depression from opioids and/or sedatives. A combination of these anesthesia and patient related factors may lead to airway complications in the PACU.

Case report

A 30-year-old male (162 cm, 125 kg) presented for an elective rhinoplasty for a nasal fracture under general anesthesia. During the preoperative evaluation on the day of the surgery, it was revealed the patient had signs and symptoms of OSA such as snoring, tiredness, obesity, and high blood pressure, but the patient was never diagnosed with or treated for OSA. His preoperative blood pressure was 149/91 mm Hg and blood glucose was 140 mg/dL. Preoperatively, the patient's oxygen saturation (SpO₂) was 99% on room air. His electrolytes, renal panel, and complete blood count were within normal limits. Chest auscultation was clear and the electrocardiogram showed normal sinus rhythm with a heart rate of 75/min. The patient took no prescription medications and denied surgical history and family history of anesthesia complications.

Preoperative sedation was not administered. The patient was transferred to the operating room and noninvasive monitors were applied. Anesthesia was induced with propofol 200 mg, fentanyl 50 mcg, and rocuronium 50 mg intravenously (IV). The trachea was intubated with a 7.5 mm endotracheal tube (ETT), and placement confirmed with auscultation and end-tidal carbon dioxide monitoring. Anesthesia was maintained with sevoflurane 3% inspired concentration in a mixture of oxygen 1 L/min and air 1 L/min. Neuromuscular blockade was monitored with a peripheral nerve stimulator (PNS). A second dose of rocuronium 10 mg was given IV one hour after induction when the PNS showed four twitches. Additional increments of fentanyl 25 mcg IV were titrated after the patient started to breathe spontaneously with a tidal volume (VT) of 7 mL/kg. A total dose of fentanyl 125 mcg was administered to the patient. In light of the patient's obesity, he received 5 recruitment breaths with 100 % oxygen (O₂) three times intraoperatively in an attempt to prevent atelectasis. Continuous positive airway pressure (CPAP) of 5 mm Hg was provided during controlled and spontaneous ventilation.

Surgery duration was two hours. The patient received ondansetron 4 mg IV thirty minutes prior to the end of the surgery. Neuromuscular blockade was antagonized with neostigmine 6 mg IV

and glycopyrrolate 1.2 mg IV at the end of surgery when the PNS showed four twitches. Subsequently four twitches without tetanic fade were noted. The ETT was removed with the head of the bed elevated, patient following commands and with an end tidal sevoflurane concentration of 0.3%. A respiratory rate of 14/min and VT of 7 mL/kg were observed following extubation. The patient was transported to PACU with O₂ at 6 L/min via face mask.

The initial vital signs were stable with a heart rate 86/min, blood pressure 138/72 mm Hg, respiratory rate 10/min, temperature 36.4⁰C and SpO₂ 100% on 6L/min of O₂ via facemask. Within 5 minutes of arrival to the PACU the patient's SpO₂ declined to 92%, with a respiratory rate of 6/min. An obstructed breathing pattern was noted and the patient became less arousable. Initial interventions included a jaw thrust, insertion of an oral airway, and increasing the O₂ flow to 10 L/min. The SpO₂ continued to decline to 85% and the patient became less responsive to verbal commands. Manual positive pressure ventilation with 15L/min of O₂ via an ambu bag was initiated. Despite these interventions, the patient's SpO₂ did not improve and he was reintubated with a 7.5 mm ETT and the placement was verified. Blood gases were drawn following reintubation revealing: pH 7.29, pCO₂ 55 mm Hg, HCO₃ 20 mEq/L and a pO₂ 70 mm Hg. The patient was transferred to the ICU, and the endotracheal tube was removed the following day without further complications.

Discussion

Reintubation continues to occur in the PACU despite adherence to evidence based extubation criteria including: return of spontaneous breathing, return of consciousness, neuromuscular blockade fully antagonized, normothermia, and ability to follow commands.² The patient in this case report met the extubation criteria with an end tidal sevoflurane concentration of 0.3%. A study conducted by Katoh et al. showed the cerebral anesthetic concentration for awakening to be an end tidal sevoflurane concentration of 0.3%.³ This patient did not receive preoperative sedation, opioids were carefully titrated after return of spontaneous ventilation, and neuromuscular blockade was fully antagonized considering the patient's risk factors for OSA.

Despite careful monitoring and appropriate interventions, patients with OSA are at risk for postoperative complications due to the likelihood of airway collapse following general anesthesia as anesthetics alter the upper airway muscle tone.¹ A sleep study can diagnose and determine the severity of OSA by monitoring periods of apnea and obstruction during sleep, both of which are likely due to relaxation of the throat muscles leading to a collapsible airway and obstruction.¹ Repetitive episodes of airway obstruction, nocturnal hypoxemia leading to excessive sympathetic stimulation, chronic hypertension, and pulmonary hypertension from hypoxic pulmonary vasoconstriction are associated with severe sleep apnea.¹

A retrospective study conducted by Munish et al. showed an increased incidence of adverse surgical outcomes in patients with high risk OSA following general anesthesia to include hypoxia (16.8 % vs 10.2%; $p < .01$) and an increased incidence of reintubation (4.9% vs 0.7%; $p < .001$) when compared to a low risk OSA group.⁴ A historical cohort study conducted by Vasu et al. found an increased incidence of postoperative complications in patients with high risk vs. low risk OSA (19.6% vs 1.3%; $p < .001$).⁵ This study also showed a higher rate of postoperative complications in patients with obesity (17.6% vs 5.9%; $p=0.04$).⁵

CPAP can be administered intraoperatively to prevent postoperative ventilatory complications. In a study by Liao et al., the application of auto titrated continuous positive airway pressure in the perioperative setting was shown to reduce postoperative complications, and improve the apnea- hypopnea index in the postoperative period in patients with untreated OSA.⁶ The patient in this case report received CPAP and recruitment breaths at the end of the surgery to prevent hypoventilation and atelectasis.

Neuromuscular blocking agents are used intraoperatively to facilitate endotracheal intubation and surgical relaxation but residual neuromuscular blockade is a risk factor for postoperative complications.⁷ Recent data suggests the gold standard for measuring the depth of neuromuscular blockade is acceleromyography (AMG) to provide quantitative neuromuscular function monitoring indicating a train of four (TOF) ratio. A TOF ratio >0.9 likely denotes a minimal acceptable level of neuromuscular recovery.⁷ The patient in this case study was monitored with a PNS, had four twitches before reversal agents were administered, and had four twitches without post tetanic fade following administration of reversal agents.

A randomized control study by Murphy et al. suggested the incidence and severity of residual muscle relaxation was higher in patients with a TOF ratio <0.9.⁸ The patients in this study were randomly assigned to two groups: acceleromyography (AMG) group using an AMG device to objectively measure the TOF ratio intraoperatively and in the PACU, and a control group using a PNS measuring observed twitches and post tetanic fade intraoperatively and acceleromyography in PACU. This study showed 63% of the patients in the AMG group had a TOF ratio >0.9 and only 23% of the patients in the control group had a TOF ratio >0.9 ($p < 0.0001$).⁷

Reintubation in the PACU results from multifactorial causes. The anesthesia related causes may be due to residual effects of anesthetics and neuromuscular blocking agents. Patients with OSA are more susceptible to airway complications following general anesthesia because of the likelihood of airway collapse. It is highly unlikely the hypoxia in this patient was due to the residual effects of volatile anesthetics or opioids. The exact PACU diagnosis remains unclear as a PNS was not utilized to check for residual neuromuscular blockade. This patient had signs and symptoms of OSA such as snoring, tiredness, obesity, high blood pressure and male gender. The likelihood of OSA and a possibility of residual neuromuscular blockade could have contributed to the reintubation of this patient in the PACU.

References

1. Bigatello LM, Srinivasa V. Chronic pulmonary disease. In: Miller RD, Pardo MC ed. *Basics of Anesthesia*. 6th ed. Philadelphia, USA: Elsevier Saunders; 2011:435-436.
2. Howie W, Dutton RP. Implementation of an evidence-based extubation checklist to reduce extubation failure in patients with trauma: a pilot study. *AANA J*. 2012;80(3).
3. Katoh T, Suguro Y, Kimura T, Ikeda K. Cerebral awakening concentration of sevoflurane and isoflurane predicted during slow and fast alveolar washout. *Anesth Analg*. 1993;77(5):1012-7.
4. Munish M, Sharma V, Yarussi KM, Sifain A, Porhomayon J, Nader N. The use of practice guidelines by the American Society of Anesthesiologists for the identification of surgical

patients at high risk of sleep apnea. *Chron Respir Dis*. 2012;9(4):221-30. doi:10.1177/1479972312458680.

5. Vasu TS, Doghramji K, Cavallazzi R, et al.. Obstructive sleep apnea syndrome and postoperative complications: clinical use of the STOP-BANG questionnaire. *Arch Otolaryngol Head Neck Surg*. 2010;136(10):1020-4. doi: 0.1001/archoto.2010.1020.
6. Liao P, Luo Q, Elsaid H, Kang W, Shapiro CM, Chung F. Perioperative auto-titrated continuous positive airway pressure treatment in surgical patients with obstructive sleep apnea: A randomized controlled trial. *Anesthesiology*. 2013;119(4):837-47. doi:10.1097/ALN.0b013e318297d89a.
7. Nicholau D. Post anesthesia recovery. In: Miller RD, Pardo MC ed. *Basics of Anesthesia*. 6th ed. Philadelphia, USA: Elsevier Saunders; 2011:633-634.
8. Murphy GS, Szokol JW, Avram MJ, et al. Postoperative residual neuromuscular blockade is associated with impaired clinical recovery. *Anesth Analg*. 2013;117(1):133-41. doi: 10.1213/ANE.0b013e3182742e75.

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The Development of a Hematoma Following Thyroidectomy

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Keywords: thyroidectomy, Grave's disease, hematoma, airway obstruction, postoperative complications, hemorrhage, deep extubation

Grave's disease is an autoimmune disorder that leads to excessive production of thyroid hormones, producing hyperthyroidism. Grave's disease can be treated medically with antithyroid medications or surgically with removal of the thyroid gland. Anesthetists should have a thorough knowledge of Grave's disease, thyroid gland function, and any complications that may occur during a thyroidectomy. The most prevalent postoperative complications include hypocalcemia, recurrent laryngeal nerve damage, and hematoma at the surgical site.¹ The formation of a hematoma postoperatively is a serious complication that can lead to airway obstruction and possible asphyxiation. A thorough preoperative evaluation and modern surgical techniques have been associated with a decreased risk of complications following a thyroidectomy.¹

Case Report

A 70-year-old, 75 kg, 165 cm, female presented for an elective total thyroidectomy. Her medical history included Grave's disease, hypertension, gastroesophageal reflux disease, and a 50 pack year smoking history. A preoperative electrocardiogram (ECG) was done and recorded a rhythm of sinus tachycardia at a rate of 115 beats per minute, along with a complete blood count and basic metabolic panel with laboratory values within normal range as determined by generalized laboratory references. Preoperatively, the patient received midazolam 2 mg intravenously (IV).

Upon arrival to the operating room a noninvasive monitors were applied. The initial vital signs were acceptable for the patient's medical history and current status. The patient was preoxygenated with 100% oxygen via face mask prior to rapid sequence induction (RSI) with cricoid pressure. Anesthesia induction was performed using fentanyl 100 mcg IV, lidocaine 80 mg IV, propofol 150 mg IV, and succinylcholine 100 mg IV. Following the induction of general anesthesia and neuromuscular blockade, direct laryngoscopy was performed using Macintosh (MAC) 3 blade and the trachea was intubated with a 7.0 mm nerve integrity monitor (NIM) endotracheal tube (ETT). After successful intubation, the stomach was suctioned thoroughly using an orogastric tube with minimal yellow tinged secretions observed. General anesthesia was maintained using a mixture of sevoflurane at a 2.3% end-expired sevoflurane concentration, oxygen (O₂) at 1.0 L/min, and air at 1.5 L/min.

Post intubation neuromuscular blockade was not used due to the need to monitor the function of the recurrent laryngeal nerve throughout the case. Ondansetron 4 mg IV and dexamethasone 8 mg IV were administered after induction for prophylactic treatment of postoperative nausea and vomiting. Fentanyl 150 mcg was administered IV in divided doses throughout the maintenance phase of anesthesia for analgesia. General anesthesia was maintained in stage III as indicated by central gaze, constricted pupils, and regular respirations. Following the closure of the surgical site and prior to extubation, the patient was returned to spontaneous ventilation at an adequate tidal volume and respiratory rate. Throughout the surgery there was no apparent damage to the recurrent laryngeal nerve based on readings from the NIM ETT. The oropharynx was suctioned thoroughly prior to extubation. Lidocaine 80 mg IV was administered and deep extubation was performed to decrease the potential risk of hematoma formation due to excessive coughing by the patient secondary to her 50 pack year smoking history. Immediately after extubation the patient maintained adequate gas exchange, no signs of obstruction were observed, and the airway remained patent.

Five minutes after extubation an enlarging mass, approximately the size of a baseball, was noted in the area of the surgical site. The suture line remained clean and no active bleeding from the wound was observed. The trachea was reintubated successfully after two attempts. The first laryngoscopy was performed using a MAC 3 blade. The initial attempt provided a Cormack-Lehane grade III view with deviation of the glottic opening to the right. Due to the deviation, tracheal intubation was not achieved. The trachea was successfully intubated on the second attempt using an Eschmann stylet with a 7.0 mm NIM ETT. The enlarging mass, which was identified to be a hematoma, was evacuated and hemostasis was achieved. The patient's oropharynx was suctioned and the trachea was safely extubated. No further signs of hematoma formation were noted and the patient was transferred to the post anesthesia care unit (PACU) in an acceptable condition established by the patient's medical history and intraoperative assessment. The patient remained in the PACU for 1.5 hours post-surgery; afterwards she was admitted on a medical-surgical floor for overnight observation. No other airway issues were observed with the patient in the postoperative period.

Discussion

Hematoma formation after thyroidectomy is a rare but potentially fatal complication. Hematoma formation is estimated to develop in 0.1-1.1% of cases.² Close observation and prompt intervention is required after thyroidectomy. The risk of developing a hematoma in the postoperative period is the highest within six hours following surgery.³ Hematomas can cause airway obstruction by compressing the trachea and obstructing the laryngeal opening. If there are any indications suggesting airway obstruction, the trachea should be intubated immediately to ensure airway patency. A difficult intubation should always be anticipated following a hematoma formation because of a physiological obstruction, distorted anatomy, and the potential presence of laryngopharyngeal edema. If intubation is unsuccessful, the surgical site should be decompressed to alleviate the obstruction and preparation for a surgical airway should be initiated.⁴ Cardiovascular collapse can occur secondary to hypoxia from an obstructed airway.

Postoperative hematoma formation can be attributed to the slipping of ligature from major vessels, reopening of cauterized veins, or oozing from the site of surgery. The reopening of cauterized veins can occur due to vomiting, asynchronous breathing with the ventilator during emergence, or Valsalva maneuver during the postoperative period.³ Total airway obstruction can occur rapidly with compression below the strap muscles of the neck (sternohyoid, sternothyroid, thyrohyoid, and omohyoid) which impairs venous and lymphatic drainage leading to laryngopharyngeal edema.⁵ A study conducted by Dehal et al⁴ suggest not closing the strap muscles too tightly to allow early visualization of the hematoma in the subcutaneous region. The larger size thyroid nodules tend to result in larger dissections which have an increased occurrence of hematoma formation. This is related to the large volume of potential dead space which boosts hematoma formation.³ Studies have shown that there is an insignificant difference in the rate of hematoma formation between hemithyroidectomy and a total thyroidectomy.³

Intubation of the trachea during airway obstruction should be immediate and performed by the most experienced anesthesia practitioner available. Multiple intubation attempts can increase edema formation and worsen hypoxia. If successful tracheal intubation cannot be achieved surgical decompression and a surgical airway should be immediately performed. If stridor is present the sutures should be removed to allow for evacuation of the hematoma.

Potential interventions for preventing a hematoma from developing may include placing the patient in a 30 degree head down position prior to wound closure in order to observe any venous bleeding. Other interventions include meticulous hemostasis, and suturing of the strap muscles in the midline to allow blood flow into the subplatysmal space where it can be detected easily.⁵ The anesthesia practitioner should implement techniques to reduce or avoid coughing and gagging during tracheal extubation. Coughing can increase the risk of laryngospasm and bleeding after a thyroidectomy. Administration of dexmedetomidine 0.5 mcg/kg IV during emergence reduces the severity of coughing without serious adverse effects but awakening may be prolonged.⁶ Lidocaine 1.5mg/kg IV can also be used to attenuate airway responses during tracheal extubation.⁷ In this particular case report, attempts were made to minimize coughing during tracheal extubation by extubating the patient deeply and by administering IV lidocaine. However, the first attempt at re-intubation was not successful due to tracheal deviation and other airway techniques were utilized to achieve a patent airway in a prompt manner.

Although rare, a post-thyroidectomy hematoma can be fatal. It is imperative that the anesthesia professional be prepared and remain vigilant. The use of emergency airway adjuncts may be required if the patient is unable to be ventilated or intubated. Airway management is crucial in the prevention of respiratory and cardiovascular collapse.

References

1. Dixon J, Snyder S, Lairmore T, et al. Risk factors target in patients with post-thyroidectomy bleeding. *Int J Clin and Exp Me*. 2014;7(7):1837-1844.
2. Giorgio C, Erdas E, Medas F, et al. Late bleeding after total thyroidectomy: report of two cases occurring 13 days after operation. *Clin Me Ins*. 2013:6.
3. Hung-Hin L, Chun-Ling Y, Chung-Yau L. A review of risk factors and timing for postoperative hematoma after thyroidectomy: is outpatient thyroidectomy really safe? *World J Surg*. 2012;36:2497–2502.
4. Dehal A, Abbas A, Hussain F, Johna S. Risk factors for neck hematoma after thyroid or parathyroid surgery: ten-year analysis of the nationwide inpatient sample database. *Perm J*. 2015;19(1):22-28.
5. Kemparaj T, Mohammed A, Janakirama J, et al. Thyroidectomies: to drain or not to drain? *J Evol Me and Dent Sci*. 2014; 3(57):12927-12932.
6. Lee S, Kim J, Kang L, et al. Effect of single-dose dexmedetomidine on coughing during emergence from sevoflurane-remifentanil anaesthesia after thyroidectomy. *Euro J Anes*. 2013;30(8):26-27.
7. Sharma V, Prabhakar H, Rath G, et al. Comparison of dexmedetomidine and lidocaine on attenuation of airway and pressor responses during tracheal extubation. *J Neuro Anesth Crit Care*. 2014;1(1):50-55.

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Ventilation of a Preterm Neonate with Tracheoesophageal Fistula

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Keywords: tracheoesophageal fistula, mechanical ventilation, premature neonate

Congenital esophageal atresia (EA) and tracheoesophageal fistula (TEF) occur in 1 out of 2,500-3,000 live births. The most common malformation is EA with a distal fistula (C/IIIb), which occurs in 86% of patients with TEF. This is frequently seen as a component of the VACTERL association, which includes vertebral, anal, cardiovascular, tracheoesophageal, renal and limb defects. Patients with associated cardiac pathology have a higher incidence of intraoperative critical events.¹ In infants less than 1500 g, it has been shown that a staged attempt at repair, with definitive repair occurring when the infant is over 2000 g offers improved outcomes.²

Case Report

A 36-hour-old, 37.8 cm, 1385 g male born at twenty-nine weeks gestation presented for a bronchoscopy, right-sided thoracotomy for tracheoesophageal fistula ligation, and placement of a gastrostomy tube. The pregnancy was complicated by molar changes to the placenta and premature rupture of membrane 4 days prior to the patient being born via cesarean section. Neonatal resuscitation included positive pressure ventilation, chest compressions for 2 minutes, tracheal intubation, and administration of beractant. Inability to place a nasogastric tube necessitated abdominal radiograph leading to the diagnosis of tracheal atresia with a distal tracheoesophageal fistula (TEF). Near complete opacifications of the lungs were noted on chest radiograph.

Upon physical exam breath sounds were diminished. An echocardiogram showed a patent ductus arteriosus, patent foramen ovale, and a small to moderate ventricular septal defect with left to right shunt. Laboratory values revealed blood glucose values of 20-30 mg/dL. The patient was receiving total parenteral nutrition and dextrose 20% via a double lumen umbilical vein catheter (UVC). An umbilical arterial catheter (UAC) was also present and used for intraoperative monitoring. Mechanical ventilation with synchronized intermittent mandatory ventilation with a peak inspiratory pressure (PIP) of 18 cm H₂O, rate of 60/minute, positive end-expiratory pressure (PEEP) of 6 cm H₂O, and FiO₂ of 40% was used to maintain SpO₂ at 90-95%. Due to progressing respiratory failure and the presence of bilious tracheal aspirate, the decision was made to proceed for surgical ligation of the TEF.

The patient was transported to the operating room intubated and manually ventilated with noninvasive monitoring in place. General anesthesia was induced and maintained with sevoflurane 0.5% in a mixture of O₂ 1.5 L/min and air 1.5 L/min via the existing endotracheal tube and vecuronium 0.2 mg was administered. The surgeon performed a direct laryngoscopy and bronchoscopy. The existing 3.0mm ID-uncuffed endotracheal tube (ETT) was removed and replaced with the same sized uncuffed ETT, following the unsuccessful attempt at advancing a 3.0mm ID-cuffed ETT. Fiberoptic bronchoscopy showed a fistula just superior to the carina. The ETT placement was confirmed by fiberoptic bronchoscopy and noted to be distal to the fistula. The patient was repositioned in a 45-degree semi-prone position for surgery. A right-sided thoracoscopy was initiated and significant retraction of the right lung was required. During retraction, hypotension occurred and oxygen saturations decreased to 70-85%. Interruptions in retraction were required to facilitate decreased peak inspiratory pressures and allow manual ventilation to increase SpO₂ and decrease hypercarbia. Due to frequent episodes of desaturation, the surgeon converted to an open thoracotomy and proceeded with ligation of the fistula.

Ventilator settings were adjusted based on arterial blood gas results. Ventilation and oxygen saturations were improved once the fistula was ligated. Hypotension did occur during the open repair, but was responsive to IV fluid, packed red blood cell, and albumin administration. The patient was repositioned supine and a gastrostomy tube was placed without incident. The patient remained intubated and was transported to the neonatal intensive care unit in stable condition.

Discussion

Ventilation of patients with tracheoesophageal fistula can impose a significant challenge for anesthesia professionals. Optimally, one-lung-ventilation (OLV) would be used intra-operatively to improve surgical exposure and allow for a complete repair of the fistula. Other techniques for managing a TEF may include placement of the endotracheal tube distal to the fistula or occlusion of the fistula with a Fogarty embolectomy catheter (Edwards Lifesciences, Irvine, CA).^{3,4} Tracheal intubation is best performed while the patient is spontaneously breathing using either topical anesthetic or an inhalation induction. Spontaneous ventilation typically limits airflow through the fistula, minimizing gastric distension. Positive pressure ventilation can result in gastric distension, which can decrease lung volumes, cause atelectasis and worsen ventilation efficiency.

In this patient, several factors complicated the anesthetic and surgical plan. This patient required positive pressure mechanical ventilation. There was concern that placing a gastrostomy without ligating the fistula would create a low resistance passage for ventilation due to the poorly compliant lungs. This would result in significant difficulty with positive pressure mechanical ventilation. In patients with a low birth weight, definitive repair of the esophagus is typically delayed, and a staged repair consisting of gastrostomy tube placement is done following ligation or occlusion of the fistula.² This patient was also at high risk of neonatal respiratory distress syndrome due to prematurity. Beractant, modified bovine pulmonary surfactant, was administered at birth to improve alveolar surface tension and facilitate ventilation. The left to right shunting also complicated management of CO₂ and O₂ tensions and balancing pulmonary and systemic vascular resistance.

Initiating OLV can be performed using a variety of techniques and equipment. Double lumen endotracheal tubes, balloon tipped bronchial blockers, Uni-vent endotracheal tubes (Fuji Systems Corporation, Fukushima, Japan), or intentional mainstem intubation can be used for OLV. Limitations of most equipment for OLV include the large airway diameter required to place these tubes. Sutton et al. describe successful OLV using an Arndt endobronchial blocker (Cook Medical, Bloomington, IN) in patients as small as 2.5 kg.⁵ In this patient a 3.0mm ID endotracheal tube was the largest size that would pass through the airway, making mainstem intubation the only possible technique to initiate OLV. The lack of a cuffed endotracheal tube also would make OLV difficult, as the only way to achieve a seal would be to wedge the tube in the bronchus. Significant volumes could potentially be lost to the non-isolated lung or to the fistula.

Fiberoptic bronchoscopy is the preferred technique for confirmation of correct placement of the endotracheal tube because breath sounds can be referred in small neonates.³ The anesthetic plan for this patient included positioning the tube into the left mainstem bronchus if right lung ventilation or gastric insufflation became problematic. OLV in this patient, however, would have unpredictable effects on pulmonary vascular resistance that could increase the risk of developing a right to left shunt through the VSD, resulting in worsening hypoxemia. The effects of hypercarbia may increase pulmonary vascular resistance which can worsen hypoxemia. This may affect the shunt direction when the balance between PVR and SRV is altered. Patients with

coexisting congenital heart defects experience a 23% mortality rate. In addition, birth weight less than 1500g increases morbidity with TEF.¹

Hypercarbia has been described as a complication of OLV. Due to small tidal volumes and leak around the endotracheal tube, ETCO₂ measurements can be unreliable.⁶ For this reason, it is recommended to adjust ventilation based on arterial blood gas results to prevent hypercarbia, hypoxemia, and acidosis.⁵ Supplemental oxygen, including the use of 100% oxygen, is recommended, especially during OLV to prevent desaturations. In this case, increasing the FiO₂, PIP, and respiratory rate were necessary to manage CO₂ and O₂ tensions that were exacerbated by the left to right shunt. In addition, the use of opioids and the risk of postoperative apnea that is associated with infants less than 60 weeks postconceptual age typically require postoperative ventilation while CO₂ and pH normalize prior to extubation.⁶

Many aspects of this case provide an opportunity to improve the care of premature infants undergoing TEF repair. Thoracoscopic repair and open repair of TEF have been shown to have comparable outcomes. Decreased trauma is associated with thoracoscopic repairs.⁷ It is necessary to evaluate the ability to provide optimal conditions for thoracoscopic surgery. OLV can be difficult to implement, especially in low weight premature infants. Managing the anesthetic by continuous assessment of lung compliance, gastric distension, oxygen saturation, blood gas analysis, and hemodynamics is required to maintain adequate ventilation during repair of a TEF.

References

1. Diaz LK, Akpek EA, Dinavahi R, Andropoulos DB. Tracheoesophageal fistula and associated congenital heart disease: Implications for anesthetic management and survival. *Paediatr Anaesth*. 2005;15(10):862-9.
2. Petrosyan M, Estrada J, Hunter C, et al. Esophageal atresia/tracheoesophageal fistula in very low-birth-weight neonates: Improved outcomes with staged repair. *J Pediatr Surg*. 2009;44(12):2278-81.
3. Broemling NN, Campbell FF. Anesthetic management of congenital tracheoesophageal fistula. *Paediatr Anaesth*. 2011;21(11):1092-1099.
4. Bachiller PR, Chou JH, Romanelli TM, Roberts JD. Neonatal emergencies. In: Cote CJ, Lerman J, Anderson B, eds. *A practice of anesthesia for infants and children*. 5th ed. Philadelphia, PA: Elsevier Saunders; 2013:746-765.
5. Sutton CJ, Naguib A, Puri S, Sprenker CJ, Camporesi EM. One-lung ventilation in infants and small children: Blood gas values. *J Anesth*. 2012;26(5):670-674.
6. Krosnar S, Baxter A. Thoracoscopic repair of esophageal atresia with tracheoesophageal fistula: Anesthetic and intensive care management of a series of eight neonates. *Paediatr Anaesth*. 2005;15(7):541-6.
7. Borruto FA, Impellizzeri P, Montalto AS, et al. Thoracoscopy versus thoracotomy for esophageal atresia and tracheoesophageal fistula repair: Review of the literature and meta-analysis. *Eur J Pediatr Surg*. 2012;22(6):415-419.

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Total Intravenous Anesthesia for Gastroenterology Procedures

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Keywords: total intravenous anesthesia (TIVA), colonoscopy, esophagogastroduodenoscopy (EGD), high risk patient population.

Total Intravenous Anesthesia (TIVA) has been successfully used in gastroenterology for short duration procedures such as colonoscopy and esophagogastroduodenoscopy (EGD). This technique has a solid safety record; however, adverse outcomes continue to occur. Although this method of anesthesia may appear easy to perform, it must be administered by trained anesthesia professionals. These individuals can institute prompt, appropriate interventions in case of emergency to avoid devastating outcomes. Increased vigilance and extra safety measures are needed when administering TIVA to patients, particularly for those who are overweight or have multiple comorbidities.

Case Report

A 63-year-old, 193 cm, 189 kg male with a diagnosis of Barrett's esophagus presented to undergo EGD. The patient's medical history included Barrett's esophagus, gastroesophageal reflux disease (GERD), insulin dependent diabetes mellitus, seasonal allergies, hypertension, neuropathy to bilateral feet, osteoarthritis, and hypothyroidism. Previous anesthetic complication consisted of post-operative nausea and vomiting (PONV). The patient had no known drug allergies. The patient's medication profile included escitalopram, insulin aspart, insulin glargine, levothyroxine, diltiazem, enalapril, acetaminophen, terazosin, sitagliptin, dexlansoprazole, and glipizide. Electrocardiogram (ECG) showed normal sinus rhythm. Chest X-ray revealed mild pulmonary vascular congestion and cardiac silhouette at the upper limits of normal in size. Preoperative laboratory analysis included a complete blood count with differential and a basic metabolic panel with results within normal limits. The patient's preoperative vital signs were blood pressure 159/70 mmHg, heart rate 74/min, respiratory rate 20/min, SpO₂ 95% on room air, and temperature 37.7°C. Airway assessment revealed Mallampati Class III, small oral aperture, and no visualization of front teeth upon smiling.

On arrival to the gastrointestinal laboratory (GI Lab), the patient was connected to noninvasive monitors, including continuous end-tidal CO₂ (ETCO₂) monitoring. Oxygen was administered via simple face mask at 6 L/min. The plan was to perform total intravenous anesthesia (TIVA) via intermittent propofol boluses and continuous infusion if needed. Emergency airway management equipment and medications were at bedside before the administration of IV medications. These included King Vision portable video laryngoscope (King Systems, Noblesville, IN), a gum elastic bougie, bag valve mask resuscitator, suction, succinylcholine 200 mg in a 10 mL syringe, 90 mm and 100 mm oral airways, and an endotracheal tube (ETT) with internal diameter of 8.0 mm. The patient was asked to position himself in left lateral position to facilitate easier access for the gastroenterologist. An initial propofol bolus of 30 mg intravenously (IV) was administered. Anesthesia was maintained with intermittent propofol

boluses of 10-20 mg IV as needed. Particular attention was paid to the depth of sedation and maintenance of spontaneous respirations.

At the conclusion of the case, a total of 160 mg of propofol was administered. The patient tolerated the procedure well with minimal fluctuations in vital signs. After the end of procedure, the patient was closely monitored until he was fully awake and able to maintain his airway. The face mask was then removed and the patient was able to maintain his SpO₂ > 90% on room air. The patient care was transferred at this time after report was given to the registered nurse who was attending the procedure.

Discussion

Total Intravenous Anesthesia has become more widely used for therapeutic, diagnostic and interventional procedures in both adults and children. It is widely used for procedures in the operating room (OR), as well as for procedures away from the OR including the GI Lab¹. In the GI Lab, TIVA can be used for short duration diagnostic and interventional procedures such as EGD and colonoscopy. A variety of hypnotic drugs are currently available for use during TIVA, however, it is clear that the “ideal” IV anesthetic is yet to be developed.¹ There is a list of characteristics that an “ideal” IV anesthetic should possess but, one of the most important is the absence of cardiovascular and respiratory depression. A number of factors can influence the pharmacokinetics of IV sedative-hypnotic drugs and they vary from patient to patient. These factors include the degree of protein binding, the efficiency of hepatic and renal elimination processes, physiologic changes with aging, pre-existing disease states, the operative site, body temperature, and drug interactions.¹ Furthermore, individual patient characteristics such as difficult airway, morbid obesity, and history of obstructive sleep apnea make provision of safe anesthesia more challenging. Continuous monitoring, assessment, and interpretation of vital signs and cardiorespiratory parameters is essential in early identification and prevention of potential complications.

Over the past several years, propofol has increasingly been used as the sedative agent for endoscopy.² Compared to other IV anesthetic agents, propofol offers advantages such as rapid onset of action, rapid elimination, and antiemetic effect. The result is faster return of respiratory and cognitive function once the propofol administration has ceased. However, propofol is not the “ideal” IV anesthetic because it is associated with hemodynamic effects such as hypotension as well as respiratory depression and airway obstruction.³ Propofol’s narrow therapeutic range makes the level of sedation less predictable and its lack of a reversal agent leaves few options if deeper than expected depth of sedation is achieved.³ The margin between sedation and general anesthesia is narrow and may be crossed unexpectedly. Often times, patients vacillate between sedation and general anesthesia, even with relatively small alterations in the dose of IV anesthetic.⁴

There are a number of benefits that TIVA can offer during gastrointestinal endoscopy and other procedures. These include decreased anxiety and discomfort, sedation and amnesia. Additionally, patients remain immobile for the procedure, avoid the side effects of inhaled anesthesia, and experience faster recovery and discharge times.³ However, some significant adverse events from TIVA have also been reported that may increase morbidity and mortality.³ Recent sentinel events

involving the use of propofol in celebrities have drawn more attention to this medication and to IV anesthesia/sedation performed in settings outside of the OR.

Most adverse outcomes can be attributed to the effects of propofol on cardiovascular and respiratory systems. Therefore, a thorough preoperative assessment and identification of risks is essential in administration of a safe anesthetic. The main focus of preoperative anesthesia evaluation is the airway assessment. It consists of various parameters, the most important ones being Mallampati classification, thyromental distance, evaluation of the mouth opening, and head and neck mobility.⁵ Other factors that can affect successful ventilation and intubation are the presence of a beard, receding chin, prominent incisors, airway edema/abscess, and increased neck circumference. A medical history of GERD, hiatal hernia, diabetes mellitus, and liver disease all increase the risk of aspiration during anesthesia. As was noted above, the patient in this case had multiple risk factors present that would classify him as having a potentially difficult airway.

The hospital where the patient was undergoing the procedure had King Vision video laryngoscope (King Systems, Noblesville, IN) available which was brought to the GI Lab. Video laryngoscopes may offer little advantage in patients with uncomplicated airways; however, they do improve visualization of laryngeal structures in difficult airways.⁵ It is recommended to use some sort of an ETT stylet or gum elastic bougie with video laryngoscopes. The stylet helps to form the ETT into a shape that matches the angle of the video laryngoscope in order to facilitate the endotracheal intubation.⁵ The use of gum elastic bougie is superior to stylet-assisted intubation of the airway and it should be readily available for patients with suspected difficult airway.⁶

In this case, the concern was that the patient presented with a difficult airway. It was discovered during preoperative assessment and necessary preparations were completed before the case started. Although, there is no clear evidence as to which airway management device is the most superior, having multiple options is the best strategy. Despite the adverse effects and risks mentioned above, TIVA offers great benefits for short duration gastroenterology procedures when administered by properly prepared and vigilant anesthesia professionals.

References

1. White PF, Eng MR. Intravenous anesthetics. In Barash PG, Cullen BF, Stoelting RK, Cahalan MK, Stock MC, eds. *Clinical Anesthesia*. 6th ed. Philadelphia: Lippincott Williams & Wilkins; 2009:444-464.
2. Hsieh YH, Lin HJ, Tseng KC. Which should go first during same-day bidirectional endoscopy with propofol sedation? *J Gastroenterol Hepatol*. 2011;26(10):1559-1564.
3. Hession PM, Joshi GP. Sedation: not quite that simple. *Anesthesiol Clin*. 2010;28(2):281-294.
4. Thomson A, Andrew G, Jones DB. Optimal sedation for gastrointestinal endoscopy: review and recommendations. *J Gastroenterol Hepatol*. 2010;25(3):469-478.
5. Butterworth JF, Mackey DC, Wasnick JD. Airway management. In Butterworth JF, Mackey DC, Wasnick JD, eds. *Morgan & Mikhail's Clinical Anesthesiology*. 5th ed. New York: McGraw-Hill; 2013:309-341.

- Messa MJ, Kupas DF, Dunham DL. Comparison of bougie-assisted intubation with traditional endotracheal intubation in a simulated difficult airway. *Prehosp Emerg Care*. 2011;15(1):30-33.

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Anesthetic Management of a Veteran with History of Emergence Delirium

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Keywords: emergence delirium, combat veteran, safety, post traumatic stress disorder, traumatic brain injury

Repercussions from the September 11, 2001 terrorist attacks have been extended combat operations in Iraq and Afghanistan. During these conflicts more than three million U.S. military personnel were deployed, some more than once.¹ Emergence delirium (ED) following general anesthesia in recent combat veterans became a point of concern among military anesthesia practitioners in 2005.¹ Research suggests a 20% incidence of emergence delirium in combat veterans compared to 5% in the general adult population.² These veterans are transitioning back into society and are having routine surgery in non-military hospitals requiring all anesthesia professionals be informed and prepared.³

Case Report

A 36-year-old, 177.8 cm, 98.8 kg male with a history of combativeness during general anesthesia emergence presented to a non-military hospital for a scheduled L4/L5, L5/S1 fusion. The patient's medical history included low back pain, radiculopathy, traumatic brain injury (TBI), posttraumatic stress disorder (PTSD) and six back surgeries without symptom relief. He had experienced low back pain and radiculopathy following injury from a roadside explosive while deployed to Iraq with the U.S. Army in 2006. His home prescription medications included clonazepam, alprazolam, sertraline and hydrocodone/acetaminophen.

The patient verbalized a feeling of anxiety in the pre-operative holding area and received midazolam 2 mg intravenously (IV). He confirmed documentation of multiple combative episodes upon anesthesia emergence. These episodes resulted in injuries to the operating room staff.

The patient was transferred to the operating room and noninvasive monitors were applied. After pre-oxygenation with 6 L/min via mask, lidocaine 60 mg, fentanyl 100 mcg, propofol 200 mg and rocuronium 35 mg were given IV for induction. The patient's trachea was intubated. Respirations were controlled by mechanical ventilation, and he was turned prone without incident.

Intraoperative anesthesia was maintained with sevoflurane 2 % inspired concentration in a mixture of oxygen 1 L/min and air 1 L/min for the 4.5 hour procedure. Medications included fentanyl 350 mcg, hydromorphone 2 mg, ephedrine 10 mg, ondansetron 4 mg, and dexamethasone 8 mg.

At completion of surgery spontaneous ventilation was re-established and sevoflurane decreased to an end tidal concentration of 1.6%, in preparation for a deep extubation. The patient was turned supine on the stretcher. The staff was reminded of potential combativeness during emergence. Strict noise reduction was maintained. After the oropharynx was suctioned, sevoflurane was discontinued; oxygen flow was increased to 8 L/min, and the patient's trachea was extubated without incident. A mask seal was re-established, and manual chin lift was required briefly.

Oxygen 2 L/min via nasal cannula was administered and the patient placed in the semi fowler's position. Prior to leaving the operating room the patient attempted to punch anesthesia personnel. He was verbally reoriented and re-assured. The patient was transferred to the post anesthesia care unit (PACU). He did not have any combative episodes or confusion while in the PACU and was transferred to his hospital room alert and oriented.

Discussion

Post September 11, 2001 combat veterans are making an impact on civilian health care. Combat veterans are seeking treatment in civilian and military hospitals following completion of service or retirement.^{3,4} An increased incidence of emergence delirium among recent combat veterans (20%) compared to a 5% incidence in the general adult population carries implications for military and civilian anesthesia professionals.² Risk factors that increase incidence of ED in combat veterans include history of anxiety, depression, PTSD, TBI, acute and chronic pain.⁵ Risk factors for ED in the non-military population with implications for veterans are extended surgical time, preoperative benzodiazepine administration, abdominal or breast surgeries.^{4,5} Awake extubation following volatile anesthesia increases ED when compared to balanced anesthesia with opioids and deep extubation.^{4,5,6} Emergence delirium symptoms have a positive relationship with high pain scores in the PACU and greater opioid requirement.²

Signs of ED start with anesthesia emergence continuing until resolution before PACU discharge and do not fluctuate.¹ Behaviors observed include one or more of the following: thrashing in an aggressive manner; pulling at monitoring equipment, intravenous lines, endotracheal tubes or drains; yelling, punching, biting, speaking unintelligibly; and attempting to leave the operating room, resulting in falls.¹ These behaviors impact the patient and staff at a time when anesthesia practitioners are directly responsible for patient safety.

Interventions that reduce ED behaviors include identification of potential patients, environmental changes, physiologic alteration, and alternative anesthetic approaches.^{1,2,4,5,7} Identifying this typically stoic subculture is difficult. Veterans may have undiagnosed PTSD which increases anxiety and memory flashback potential.⁶ Mental health concerns or exposure to traumatic events are rarely discussed during preoperative evaluation. Attention to the patient's current medication list and questioning the indication for the prescription will help to identify a potential

ED patient. Prazosin, a selective alpha₁ antagonist prescribed for hypertension is currently used for persistent nightmares associated with PTSD.⁴ Paroxetine and sertraline, selective serotonin reuptake inhibitors, often assumed for depression, carry a specific indication for the treatment of PTSD.⁴

Although little research was found, clinical evidence from military anesthesia providers demonstrates management strategies for veterans susceptible to ED. Preoperative benzodiazepine administration for anxiolysis is controversial. Benzodiazepines can cause confusion, which exacerbates ED symptoms.⁴ Midazolam may enhance memories associated with PTSD; therefore lorazepam is preferred when this diagnosis is present.^{4,6} Combat veterans have heightened situational awareness that is attenuated by general anesthesia. Verbal assurance through induction and on emergence, coined “vocal local,”^{7 p. 264} can make emergence smoother. Emergence delirium is an excitatory response influenced by the sympathetic nervous system.⁴ Removing loud noise from the operating room and maintenance of normothermia may diminish excitatory stimulation.⁵ Intraoperative administration of clonidine or dexmedetomidine, alpha₂ agonists, is associated with less emergence delirium in susceptible veteran patients.⁴ Alpha₂ agonist’s inhibit sympathetic outflow, provide sedation and analgesia.^{4,5} A multimodal approach to analgesia is superior in reduction of ED to fentanyl used alone.⁴ Ketorolac, local anesthetic infiltration at the surgical site and longer acting opioids such as morphine and hydromorphone, are used successfully.⁴

Ketamine, often avoided because of hallucination and psychotic potential, has recently been used on patients with a history of PTSD or ED.^{7,8} Ketamine antagonizes the excitatory action of N-methyl-D-aspartate receptors preventing afferent pain signals from reaching the brain.^{6,8} Ketamine has been used for multimodal pain control in low doses for veterans with PTSD without exacerbation of symptoms.^{7,8} Total intravenous anesthesia with ketamine 1 mg per propofol 10 mg has been used in patients reporting a history of ED and PTSD.⁷ Ketamine 100mg and propofol 100 mg have been used for IV induction on previously combative ED patients.⁷ A balanced anesthetic approach, minimizing or eliminating volatile agent, combined with IV anesthetics and deep extubation can decrease ED and improve emergence.^{4,7}

The most effective intervention when faced with acute ED is injury prevention while allowing time to pass.⁴ Attempting to treat the combative behavior with reason, reorientation or family presence is not effective once the episode has escalated.^{4,6}

Comparing the presented case to current literature demonstrates that awareness and a proactive approach improved the outcome related to ED. A focused preoperative assessment for ED risk factors to include a diagnosis of PTSD, current symptoms, and medications is essential in the veteran population. The presented patient had a positive history of emergence delirium. Operating room staff awareness, reduction of environmental stimuli, deep extubation and additional long acting opioid minimized the symptoms of ED. Retrospectively, avoiding midazolam, using ketamine with propofol to supplement the intraoperative anesthesia, and reducing volatile gas could have been beneficial.

Civilian anesthesia practitioners are likely to encounter post September 11 combat veterans in their practice. Early identification of potential ED patients can reduce the negative effects

associated with combative patients. Small reductions to sympathetic outflow will reap big rewards in excitatory reduction. Utilization of multimodal pain relief and current anesthetic alternatives is vital. These strategies might improve staff and patient safety.

References

1. Wilson JT. Army anesthesia providers' perceptions of emergence delirium after general anesthesia in service members. *AANA J.* 2013;81(6):433-440.
2. McGuire J. The incidence of and risk factors for emergence delirium in U.S. military combat veterans. *J Perianesth Nurs.* 2012;4:236-245.
3. Scannell D, Doherty M. Experiences of U.S. military nurses in the Iraq and Afghanistan wars. *J Nursing Scholar.* 2010;42(1):3-12.
4. Lovstrand D, Phipps P, Lovstrand S. Posttraumatic stress disorder and anesthesia emergence. *AANA J.* 2013;81(3):199-203.
5. McGuire J, Burkard J. Risk factors for emergence delirium in U.S. military members. *J Perianesth Nurs.* 2010;6:392-401.
6. Shoum S. Posttraumatic stress disorder: a special case of emergence delirium and anesthetic alternatives. *A&A Case Reports.* 2014;3:58-60.
7. Wilson JT, Pokorny ME. Experiences of military CRNAs with service personnel who are emerging from general anesthesia. *AANA J.* 2012;80(4):260-265.
8. McGhee L, Maani C, Garza T, Gaylord K, Black I. The correlation between ketamine and posttraumatic stress disorder in burned service members. *J Trauma.* 2008;64:S195-S199.

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Hyperthermic Intraperitoneal Chemotherapy for Colorectal Cancer

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Keywords: Hyperthermic Intraperitoneal Chemotherapy, HIPEC, Colorectal Cancer, Cytoreductive Surgery, peritoneal malignancy

Hyperthermic intraperitoneal chemotherapy (HIPEC) is a therapeutic surgical treatment offered to patients with various metastatic abdominal carcinomas. Patients with extensive disease may present for surgical 'debulking' of tumor(s). After exploration and reduction of solid tumor, the laparotomy incision is temporarily closed with sutures and a heated chemotherapeutic agent is instilled into the peritoneal cavity for 90 minutes via perfusion cannulae. Fluid shifts, insensible loss, and temperature regulation are three common anesthetic challenges that require methodical management. Patients typically present with complex co-morbidities and may experience significant blood loss and hemodynamic instability throughout the 6-14 hour procedure.¹

Case Report

A 39-year-old, 168 cm, 72 kg male with history of colon cancer and recurrent carcinomatosis, presented for exploratory laparotomy, tumor debulking, and HIPEC. His comorbidities included hypertension, hyperlipidemia, gastroesophageal reflux, and deep venous thrombosis following a surgical procedure two years earlier. An inferior vena cava (IVC) filter was placed three weeks prior to scheduled cytoreductive surgery. Medication regime included propranolol, omeprazole, welchol, and warfarin. Past surgical history included previous HIPEC, and ileostomy placement, which was recently reversed. A preoperative physical assessment included a complete blood count and basic metabolic panel. All blood samples indicated normal values.

On the day of surgery, an 18 gauge intravenous catheter was placed in the left hand. The patient was premedicated with 2 mg midazolam, 50 mcg fentanyl, and loaded with 800 ml of plasmalyte prior to thoracic epidural insertion in the preoperative holding area. An epidural catheter was placed at the T9 level. The epidural was dosed for intraoperative use with 8 ml of 0.25% bupivacaine. The anesthesia provider continued to use the epidural throughout the case via intermittent bolus dosing. General anesthesia was induced after three minutes of preoxygenation. Induction medications included 40 mg lidocaine, 200 mcg fentanyl, 200 mg propofol, and 50 mg rocuronium. A Mac 3 blade was used to facilitate oral intubation with a size 8.0 endotracheal tube. The patient was placed on volume mode ventilation and maintained on 2% expired sevoflurane, 1 L/min air, and 1 L/min oxygen. A 7 French triple lumen internal jugular catheter was placed in sterile fashion under ultrasound guidance and covered with chlorhexidine impregnated dressing. A 20-gauge catheter was inserted in the left radial artery and transduced for hemodynamic monitoring. One gram of ertapenem and heparin 5000 units subcutaneously were given as prophylaxis for infection and thrombosis, respectively. Standard noninvasive monitors, bispectral index, arterial blood pressure, central venous pressure (CVP), and esophageal temperature were monitored throughout the case. Four units of packed red blood cells were available in the room.

After six hours of abdominal exploration, a partial colectomy was performed, along with removal of a portion of the mesentery and ostomy placement. The surgeon decided that HIPEC might prove to be more harmful with the overwhelming extent of the patient's peritoneal disease. Estimated blood loss was 300 ml and no blood products were given. Total fluids amounted to five liters of plasmalyte. After reversal of neuromuscular blockade, the patient was placed on a spontaneous ventilation mode and showed adequate tidal volumes. When expired sevoflurane had decreased to 0.2%, the patient was able to follow commands and was successfully extubated to face mask 10 L oxygen. A continuous epidural infusion of ropivacaine 0.1% with 3mcg/ml fentanyl was initiated for postoperative analgesic management. The patient was transferred to the postanesthesia recovery unit.

Discussion

Hyperthermic intraperitoneal chemotherapy is a technique used to attack tumor cells on organ tissue surfaces and throughout the pelvic cavity, and was first introduced in 1980 for treatment of psuedomyoma peritonei (cancer of the appendix). It has since gained favor as a therapeutic and palliative treatment for other metastatic abdominal carcinomas including gastrointestinal and

ovarian cancers.² Widespread peritoneal metastases occur in 30% of patients diagnosed with colorectal cancer, making these patients excellent candidates for HIPEC procedures. When combined with cytoreductive surgery via laparotomy, high heat chemotherapy can be instilled into the peritoneum at a temperature of 39-42°C for 60-120 minutes. The most common alkylating agent used is mitomycin C. The heated agent enhances the chemotherapeutic drug effect. The therapeutic mechanism of action increases membrane permeability in the malignant cell, impairs DNA repair, and triggers protein denaturation. Research shows that combining surgical and systemic therapy may increase the five-year survival rate for patients with recurrent disease confined to the peritoneal cavity.³

According to recent reports, survival rates have improved from 12 to 92 months when combining cytoreductive surgery and HIPEC for colorectal cancers.^{2,4,5} Complete cytoreduction (or tumor ‘debulking’ to decrease cancer cells) correlates with better outcomes.⁴ Criteria for complete cytoreduction include no evidence of extra abdominal disease, biliary obstruction, ureteral obstruction, intestinal obstruction (at more than one site), or gross disease in small bowel mesentery. There must be only small volume disease in the gastro-hepatic ligament and the patient must be a performance status two (or lower) according to the Eastern Cooperative Oncology Group guidelines (ECOG).³ The most commonly associated morbidity is due to complications with leakage from anastomosis sites, intraperitoneal sepsis, or abscesses.²

The anesthesia provider must perform a thorough preoperative assessment prior to HIPEC procedure. In addition to general demographics, allergies, medication list, and previous surgical history, a comprehensive review of systems and physical exam should be performed. More commonly patients may present with decreased functional residual capacity (FRC) due to abdominal content displacement and pathologic fluid accumulation. Therefore, these patients may demonstrate rapid desaturation on induction. The patient may be at increased risk of aspiration of gastric contents as well. Both of these factors may warrant consideration of a rapid sequence induction. Cardiac disease should be noted, as induced hyperthermia with heated chemotherapy may increase myocardial oxygen demand. Lab work, including coagulation panel and appropriate cardiac testing, should be completed and reviewed prior to the procedure.⁶

Hemodynamic monitoring remains a cornerstone in the anesthetic management of patients undergoing HIPEC procedures. In addition to arterial blood pressure, pulse pressure variation (PPV) may be monitored to measure volume responsiveness. Fluid shifts are common throughout these cases. Central venous pressure is another tool to measure third space loss. Blood loss should be anticipated. Cross matched packed red blood cells and colloid replacement should be readily available.⁴ The anesthesia provider must ensure meticulous fluid management. Fluid overload may result in pulmonary edema. Temperature rise during instillation is common and may induce vasodilation and hypotension. Extremes in body temperature may lead to an increase in heart rate, cardiac output, oxygen consumption, and end tidal carbon dioxide. There may also be decreases in systemic vascular resistance.⁴ Blood pressure should be stabilized with vasopressor agents. Appropriate heating and cooling blankets or devices may be used to facilitate normothermia. Antibiotic administration and correction of electrolyte imbalances should be attended to regularly throughout the case. Analgesic management, as described in the case study, may be achieved with thoracic epidural bolus or infusion, intravenous narcotics, and local infiltration.⁶

The future of cytoreductive surgery and HIPEC for colorectal cancer carcinomatosis (CRC-C) patients begins with dissemination of the information. A study by Spiegle et al. showed that most physicians were unaware of the benefits of HIPEC as treatment for CRC-C.⁵ Eighty-six percent of physicians had awareness that cytoreductive surgery and HIPEC were used for psuedomyoma while only 46% were familiar with its use for CRC-C.⁵ Educational strategies must be in place to inform both patients and medical teams of all therapeutic options, including HIPEC, when devising treatment plans for patients with CRC-C. There remains a lack of easily reproducible staging and scoring systems to fully interpret the outcome data from HIPEC procedures with precision.³ Further research is needed to determine the pathologic and quality of life outcomes for patients seeking this type of therapy.

References

1. Wurm H, Balonov K, Schumann R. Practice advisory for anesthetic management of patients undergoing cytoreductive surgery and hyperthermic intraperitoneal chemotherapy. *Tufts Medical Center Department of Anesthesiology*. 2015;1-3.
2. Rotruck S., Wilson JT, McGuire J. Cytoreductive Surgery With Hyperthermic Intraperitoneal Chemotherapy: A Case Report. *J AANA*, 2014;82(2):140-143.
3. Esquivel J. Cytoreductive surgery for peritoneal malignancies-development of standards of care for the community. *Surg Oncol Clin N Am*. 2007;16(3):653-666.
4. Karadayi K, Turan M, Sen M, et al. Cytoreductive surgery followed by hyperthermic intraperitoneal chemotherapy: Morbidity and mortality analysis of our patients. *Turkiye Klinikleri J Of Med Sci*. 2012;32(1):162-170.
5. Spiegle G, Schmocker S, Kennedy E, et al. Physicians' awareness of cytoreductive surgery and hyperthermic intraperitoneal chemotherapy for colorectal cancer carcinomatosis. *Can J Surg*. 2013;56(4):237-242.
6. Rothfield K, Crowley K. Anesthesia considerations during cytoreductive surgery and hyperthermic intraperitoneal chemotherapy. *Surg Oncol Clin N Amer*. 2012; 21(4):533-541.

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Off Site Venous Air Embolism

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Keywords: Venous air embolism, acute myocardial infarction, off-site anesthesia, electrophysiology

Venous air embolism or vascular air embolism (VAE) has been seen in many different invasive procedures and is associated with varying hemodynamic outcomes ranging from mild hypotension to cardiac arrest requiring cardiopulmonary bypass. This case report describes a patient who suffered a VAE during a minimally invasive electrophysiology procedure that led to the need for resuscitation and subsequent percutaneous cardiac intervention.

Case Report

A 59-year-old, 182 cm, 85.45 kg male with no known drug allergies presented for cryoablation for atrial fibrillation (AF). His past medical history included paroxysmal AF and hypercholesterolemia. An electrocardiograph (ECG) showed AF with a ventricular rate of 94 and trans esophageal echocardiography (TEE) revealed mild left ventricular hypertrophy and a left ventricular ejection fraction (LVEF) of 60%. The patient reported a metabolic equivalent of task score (METs) greater than 7. His medications were rivaroxaban, metoprolol, atorvastatin, multivitamin and fish oil supplements. Past procedural history included synchronized cardioversion and tonsillectomy.

An anesthesia timeout was performed in the EP suite with the nursing and cardiology team. Noninvasive monitors were applied. Intravenous induction facilitated endotracheal intubation using a video laryngoscope. Maintenance of anesthesia was achieved with 1.6% end tidal sevoflurane and a remifentanyl infusion at 0.1 mcg/kg/minute. A phenylephrine infusion maintained systolic blood pressures (SBPs) within 20% of baseline values.

After procedural timeout was performed, the EP team obtained venous and arterial access. Electrophysiology intracardiac monitoring equipment, including an intracardiac echocardiography (ICE) catheter, was inserted. Once the atrial septum was crossed, the EP procedural RN administered heparin.

Air was noted on ICE in a trans-septal catheter. It was promptly withdrawn across the septum and removed. Immediately the SBP fell. A 500 ml bolus of lactated ringers solution, ephedrine 10mg and neosynephrine 120 mcg IV were administered, Sevoflurane was discontinued and anesthesia help was summoned. Bradycardia and hypotension led to pulseless electrical activity (PEA). Advanced Cardiovascular Life Support (ACLS) algorithms were initiated, chest compressions started, and epinephrine 1 mg IV was administered. Routine pulse checks revealed return of spontaneous circulation (ROSC) and the monitor showed sinus tachycardia with ST segment elevations. No significant changes were noted in the ETCO₂, but air was seen in the right atrium via the ICE catheter. A catheter was inserted by the EP team to retrieve air from the RA and RV, but no significant air returned with aspiration.

Shortly after the attempt at air aspiration, the patient's SBP acutely decreased again, despite a 250 ml lactated ringers bolus, ephedrine 10mg IV and neosynephrine 120 mcg IV administration. There was no visual evidence of air or pericardial effusion via the ICE catheter. Subsequent bradycardia led to loss of pulses and ACLS was initiated with chest compressions and epinephrine administration. A subsequent pulse check revealed ROSC and sinus rhythm with ST elevation and frequent premature ventricular contractions. Ventricular tachycardia ensued, and the patient was defibrillated successfully with return to sinus rhythm.

A transthoracic echocardiogram revealed no pericardial effusion, but severe global ventricular hypokinesis and a LVEF of 25%. Given the depressed ventricular function, the procedure was aborted. Dopamine and epinephrine infusions were initiated, and titrated to stabilize hemodynamics. After one hour, the patient was awakened and his neurologic function was

assessed as normal. He was then admitted to the cardiac care unit (CCU) for serial cardiac markers and echocardiograms.

In the CCU, the troponin level peaked at 0.62 and echocardiogram showed severely reduced LV function with an LVEF of 20-25% and significant anteroseptal hypokinesis. The decision was made to transport the patient to the cardiac catheterization lab. A 30% proximal stenosis of the left anterior descending artery and a 90% focal stenosis of the LAD were noted distal to the two diagonal origins, and the vessel was stented. Echocardiography on post op day four showed an improved LVEF of 45%.

Discussion

Venous air embolism is a potentially devastating complication seen during invasive procedures. The severity of VAE is directly related to the rate of air accumulation and the volume of air entrained.⁶ VAE is manifested through a number of symptoms: decreased cardiac output, decreased SBP, arrhythmias, increased central venous pressure, decreased ETCO₂, increased pulmonary artery pressure. Electrocardiogram changes most often demonstrate right heart strain from air entrainment in the right coronary artery (RCA), because the right coronary cuff is located at the superior aspect of the heart in the supine position.⁵ The most sensitive tool for VAE detection for as little as 0.02 ml/kg of air is TEE.⁶

Treatment includes rapid identification and cessation of air entrainment. Supportive therapy should then be initiated: vasopressor agents to stabilize the hemodynamics, volume administration to increase the CVP and decrease air entrainment, Trendelenburg positioning, insertion of pulmonary artery catheter for air aspiration, hemodynamic resuscitation and the potential initiation of cardiopulmonary bypass.⁶

VAE is known to occur during minimally invasive electrophysiology procedures. Kuwahara et al followed 2976 patients undergoing AF ablation. Five of those patients had complications of severe air embolism. Two of the air embolisms were associated with air entering through the hemostasis valve of the long sheaths, and three cases of air entry occurred during the exchanging of mapping catheters. All of the study participants were spontaneously breathing and it was proposed that breath holding after sedation boluses increased the negative intrathoracic pressure and increased the risk of air entry. It is proposed that air may entrain during the exchange of the mapping catheters, because the catheter shape makes catheter insertion difficult. It was also noted that rapidly removing catheters, creates a vacuum within the sheath and the increased negative pressure further draws air through the hemostasis valve.² Although our patient was not spontaneously breathing, his trans-septal catheter was rapidly removed, as soon as air was noted, in an effort to prevent an air embolism.

Additionally, as a late complication, multiple case reports describe air embolism resulting from atrio-esophageal fistula (AEF) following catheter ablations. AEF is a rare complication with a world-wide incidence of <0.25%.⁴ Patients present with fever and neurological status changes¹. The mechanism of action is not well understood, but is thought to be related to thermal injury to the esophagus directly behind the atria. However, the long latency period suggests that other factors could be involved, such as vascular necrosis of the esophagus or esophagitis.⁴ The

outcomes associated with AEF are poor with 100% mortality when untreated and 65% mortality with treatment.⁴

Our case encouraged the review of the 2014 American College of Cardiology/American Heart Association guidelines for the pre operative cardiac assessment (ACC/AHA). Fleisher et al followed 15,122 patients having non-cardiac surgery who required overnight admission. It was noted that 11.6% of the patients had an elevation of troponin greater than 0.02ng/dl.³ Given the stress of surgery and risk of cardiac ischemia, a thorough evaluation of cardiac status is required for all patients prior to the administration of anesthesia. The ACC/AHA guidelines stress functional status is a strong predictor of perioperative and long-term cardiac outcomes. Patients with good functional status are at decreased risk of cardiac events and can proceed without further cardiac evaluation. Patients with less than four METs are at increased risk of perioperative and long-term cardiac events. Patients with stable AF require no modification of treatment, only adjustment of anticoagulation.³ Given our patients pre-operative strong functional status, greater than seven METs, no further cardiac evaluation was required.

Our case describes untoward events following a VAE that occurred in the electrophysiology procedure suite. For our patient, a prompt diagnosis of acute ischemia and new impaired myocardial function was made. This, combined with clear, concise communication and use of resources for rapid treatment, helped to mitigate the deleterious effects of the VAE. Although our patient's insult developed after air entry, every patient undergoing a surgical procedure is at some risk for the development of coronary ischemia. Knowing baseline cardiac function helped to identify the acute change in cardiac function and therefore guide appropriate care needed for preservation of myocardial function.

References

1. Aryana A, Arthur A, O' Neill PG, D' Avila A. Catastrophic manifestations of air embolism in a patient with atrioesophageal fistula following minimally invasive surgical ablation of atrial fibrillation. *J Cardiovasc Electrophysiol*. 2013;24(8):933-934.
2. Doppalapudi H, Yamada T, Kay GN. Complications during catheter ablation of atrial fibrillation: Identification and prevention. *Heart Rhythm*. 2009;6(12):S18-S25.
3. Fleisher LA, Fleischmann KE, Auerbach AD, et al. 2014 ACC/ AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: Executive summary: A report of the American College of Cardiology/ American Heart Association task force on practice guidelines. *Circulation*. 2014;130(24):2215.
4. Koa-Wing M, Jamil-Copley S, Ariff B, et al. Haemorrhagic cerebral air embolism from an atrio- oesophageal fistula following atrial fibrillation ablation. *Perfusion*. 2015;30(6):484.
5. Kuwahara T, Takahashi A, Takahashi Y, et al. Clinical characteristics of massive air embolism complicating left atrial ablation of atrial fibrillation: Lessons from five cases. *Europace*. 2012;14(2):204-208.
6. Mirski MA, Lele AV, Fitzsimmons L, Toung TJK. Diagnosis and treatment of vascular air embolism. *Anesthesiology*. 2007;106(1):164.

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Ketamine Induction for an Awake Intubation for Airway Mass Excision

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Keywords: awake intubation, ketamine induction, airway mass, nasal intubation, difficult airway

Anticipated difficult airways give rise to an opportunity to properly prepare the patient for a safe, atraumatic intubation in spite of difficult circumstances. Patients who are morbidly obese, have an anatomical abnormality, or who have an obstructive growth should be treated as anticipated difficult airways, and awake intubation should be considered.¹⁻³ Preparations should take into account the patient's specific risk factors, the skill and experience level of the anesthesia provider, the availability of emergency backup airways, and the patient's ability to cooperate with airway placement.

Case Report

A 67-year-old, 167 cm, 98 kg male presented to have a submandibular mass removed and evaluated for possible malignancy. His airway exam revealed tracheal deviation away from the side of the mass, a Mallampati class IV, an inter-incisor distance of 2 cm and a class II upper lip bite test (lower teeth can bite the upper lip below the vermilion line).² The patient reported dyspnea with mild activity and while lying flat. Notably, the patient kept his head turned away from the side of obstruction and reported he had difficulty breathing with his head in a neutral position. The patient denied any other health history. The patient exhibited extreme anxiety when a fully awake intubation was discussed, but readily consented to an approach that involved sedation.

Before transport to the operating room (OR) the patient was given viscous lidocaine 4% on cotton swabs that were applied to the back of his oropharynx and were left in place for 15 minutes. Phenylephrine 1% nasal spray was self-administered twice in each naris. Immediately before moving into the OR the patient was given fentanyl 25 mcg intravenously (IV), glycopyrrolate 0.2 mg IV, and midazolam 1 mg IV for anxiolysis. The patient appeared to be resting comfortably during transport.

Upon arrival the patient was placed in a semi-recumbent position on the OR table at an angle of 30 degrees. Noninvasive monitors were placed including electrocardiogram, a blood pressure cuff, and pulse oximetry probe. The patient was pre-oxygenated via face mask at 8 L/min for approximately 10 minutes. The patient denied discomfort or shortness of breath. After pre oxygenation, the patient was given a total of ketamine 70 mg IV administered in 10 mg increments and lidocaine 100 mg IV. Once the patient was no longer responsive to verbal stimuli, the endotracheal tube was placed into his right naris and was advanced into the back of his oropharynx. Visualization of the vocal cords was obtained using a video laryngoscope. The endotracheal tube (ETT) was advanced past the cords without difficulty. ETT placement was verified by auscultation and capnography. The patient's vital signs were stable during induction with a < 10% change in baseline blood pressure and the SpO₂ was > 96%. Spontaneous ventilation was maintained throughout induction. Anesthesia was maintained using sevoflurane

3% inspired concentration in a mixture of oxygen 1 L/min and air 1 L/min. Fentanyl was administered in 25 mcg increments IV throughout the procedure.

After uneventful excision of the mass, tracheal deviation appeared to be completely resolved. Hemostasis was obtained by the surgeon with no apparent complications. At the completion of the case the sevoflurane was discontinued. A leak check was performed prior to extubation to ensure that air could pass around the inflated ETT cuff at a pressure of 20 cm H₂O. Once the patient was awake and able to raise and maintain a head lift for 5 seconds, the ETT was removed. He was transported to PACU and recovered without incident.

Discussion

Anticipated difficult airways are situations in which the anesthesia practitioner can make a detailed plan for intubation that may be very different from a standard induction with propofol and muscle relaxation. The planning phase should include consideration of whether an awake intubation would be tolerated and whether maintaining spontaneous ventilation would be beneficial. Maintaining an open airway in the presence of an airway mass depends partially on skeletal muscle support³, which may be ablated by the use of skeletal muscle relaxants. When skeletal muscles decrease in tone, as happens with a standard IV induction using skeletal muscle relaxation, there is a propensity for the mass to completely obstruct the airway resulting in an emergency situation. Because our patient experienced dyspnea at rest, there was a concern that his airway could be totally obstructed if muscle relaxation was used during IV induction. Although a fully awake procedure would have been beneficial, it would have required a fully cooperative patient.³ This patient expressed extreme anxiety when we explained an awake procedure, so we elected to use a light ketamine induction with the goal of maintaining spontaneous ventilation. An inhalation induction may have also been considered but was not utilized in our case.

In a related study, Kundra, Kutralam, and Ravishankar⁴ compared patient's reactions to awake fiber optic nasal intubation using topical lidocaine vs. conventional airway nerve blocks. Patients received either 4 ml of nebulized lidocaine 4% or a regimen consisting of a bilateral superior laryngeal and translaryngeal nerve blocks and lidocaine soaked swabs in the nose. The patients with the blocks and nasal swabs reported less discomfort with the intubation and had a smaller physiologic response to intubation (heart rate increase was not as dramatic and was of shorter duration), and although statistical significance was achieved, clinically the numbers were quite similar. The block group had a rise in heart rate to around 100 beats per minute (bpm) on average vs. 120bpm in the nebulizer group. Patient reported satisfaction with the procedure was 83% in the block group and 79% in the nebulizer group. It is surprising and interesting that nebulized lidocaine is able to so closely approximate the block group and suggests that the extra time and resources spent with airway blocks may not be necessary in some cases. Although it is certainly possible to think of situations where heightened tachycardia would be undesirable (i.e., congestive heart failure, coronary artery disease, valvular disease, etc.) other patients may benefit from the less invasive approach. Our patient reported satisfaction with the procedure and had no recall of the event.

The use of a paralytic in tracheal intubation has become common practice, but is not without risks⁵, particularly in patients with obstructive airway masses.⁶ In this case, we wanted to avoid muscle relaxation, to avoid the risk of complete airway obstruction and maintain spontaneous ventilation. As reviewed above, there are a variety of ways to anesthetize the trachea and thereby avoid the need for relaxation including airway blocks, the application of lidocaine directly to the vocal cords, lidocaine given intravenously, or lidocaine applied topically to the oropharynx.^{3,5,7} This patient's anxiety and the nature of his mass led us to use a technique that would require as little preoperative discomfort as possible. Goudra and Singh⁶ suggest that patients may consider airway blocks to be an unacceptable alternative and should therefore not be the first choice for an awake intubation. These authors suggest that topical lidocaine as well as nebulized lidocaine 4% are good alternatives.

Awake procedures require a cooperative patient that is willing to experience some degree of discomfort under sedation, especially when nasal intubation is required.⁴ Ramkumar⁷ describes the preparation that is required to make awake intubations successful. He discusses the necessity of both psychological and pharmacological preparation. He states that ketamine is a beneficial choice for these procedures because of its ability to alter consciousness while having minimal effect on spontaneous ventilation. In our case, we were able to induce complete unconsciousness, eliminate recall, and maintain adequate ventilation throughout induction and intubation. Furthermore, we found that ketamine and lidocaine was enough to prevent a cough or gag response during nasal intubation. Ramkumar⁷ suggests that one reason people describe topical lidocaine as inadequate is because they don't allow for enough time to fully numb the area. In this case, 15 minutes was adequate in combination with other agents given.

Ketamine is not without side effects, and these too must be considered⁷. Notably, ketamine is known to increase oral secretions. This can obstruct the view of airway structures, particularly when using video technology or a fiber optic scope. Either of these devices are susceptible to have decreased visibility in the presence of copious secretions⁷. In our case, we gave glycopyrrolate to decrease secretions, but this can also increase heart rate, which may itself be undesirable, especially in patients with a cardiac history.

Awake intubations require patient cooperation, provider skill, and the time necessary for locally administered medications to take effect. Ketamine may be a useful adjunct for patients who require an awake intubation. Our case demonstrates that carefully titrated ketamine is adequate to prevent recall and discomfort during the procedure.

References

1. Gupta B, Kohli S, Farooque K, Jalwal G, Gupta D, Sinha S, et al. Topical airway anesthesia for awake fiberoptic intubation: comparison between airway nerve blocks and nebulized lidocaine by ultrasonic nebulizer. *Saudi J Anaesth.* 2014;8(1):15-19.
2. Honarmand A, Safavi M, Yaraghi A, Attari M, Khazaei M, Zamani M. Comparison of five methods in predicting difficult laryngoscopy: neck circumference, neck circumference to thyromental distance ratio, the ratio of height to thyromental distance, upper lip bite test and Mallampati test. *Adv Biomed Res.* 2015;4:122.
3. Peiris K, Frek C. Awake intubation. *J Perioper Pract.* 2008;18(3):96-104.

4. Kundra P, Kutralam S, Ravishankar M. Local anaesthesia for awake fiberoptic nasotracheal intubation. *Acta Anaesthesiol Scand*. 2008;44(5):511-516.
5. Goudra BG, Singh PM. Best of both worlds-endotracheal intubation without paralysis. *J Anaesthesiol Clin Pharmacol*. 2014;30:172-173.
6. Blank RS, de Souza DG. Anesthetic management of patients with an anterior mediastinal mass: continuing professional development. *Can J Anaesth*. 2011;58(9):853-867.
7. Ramkumar V. Preparation of the patient and the airway for awake intubation. *Indian J Anaesth*. 2011;55(5):442-447.

Mentor: James Stimpson, CRNA, DNP

Continuous Adductor Canal versus Femoral Nerve Block after Total Knee Arthroplasty

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Keywords: total knee arthroplasty, continuous adductor canal block, continuous femoral nerve block, sciatic nerve block.

Introduction

Total knee arthroplasty (TKA) is considered to be one of the more painful surgical procedures that individuals can undergo. The purpose of this study was to explore an alternative treatment strategy for patients undergoing TKA that will decrease the consumption of opioids in the postoperative period. This research study investigated the impact of either a continuous adductor canal block (CACB) or continuous femoral nerve block (CFNB), both with a single injection sciatic block (SISB), on opioid consumption during the first 24 hours following TKA.

The null hypothesis stated there will be no difference in opioid consumption between patients who received a SISB with CACB compared to patients who received a SISB with CFNB in the first 24 hours following surgery. The alternative hypothesis stated that patients who received a CACB or CFNB, both utilizing SISB, will demonstrate a difference in opioid consumption in the first 24 hours following surgery.

Methods

A retrospective chart review was conducted to assess the impact of CACB and CFNBs on opioid consumption following TKA. Group A included patients receiving a CFNB, while patients in group B received a CACB. A morphine equivalent calculator (<http://www.globalrph.com/narcoticonv.htm>) was used to determine the total morphine equivalent (ME) consumption of each individual. Raw data was recorded into GraphPad Prism 6 (GraphPad Software, Inc., La Jolla, CA). An unpaired two-tailed *t*-test was used to compare ME, ages, and body mass index (BMI) between the groups. A chi-squared test was utilized to determine any significance between genders.

Results

Group A consisted of eight patients and group B consisted of ten patients. The mean age for group A was 70.25 years \pm 10.12, and group B was 70.60 years \pm 12.77, and were not statistically different ($p = 0.9504$). The mean BMI in group A was 31.65 kg/m² \pm 5.230, and group B was 30.70 kg/m² \pm 8.166, and were not statistically different ($p = 0.8711$). There was no statistical difference found between gender as well ($p = 0.4570$). Mean ME consumption for group A was 30.92 mg \pm 8.801 versus 51.23 mg \pm 10.14 for group B, and when compared were not found to be statistically different ($p = 0.1611$).

Discussion

To our knowledge, this is the first retrospective study to evaluate ME consumption during the first 24-hours post-operatively after TKA when using CFNB and CACB, both with a SISB. Other studies have compared single injection adductor canal block (ACB) versus single injection femoral nerve block without using a SISB.

In conclusion, this study revealed a difference in mean ME between the groups, but it was not statistically significant. The null hypothesis of no difference in postoperative opioid consumption in the first 24 hours postoperatively between the groups was accepted. A limitation of this study is that it was underpowered. A *G*power* power analysis determined that a sample size of 102 patients would be sufficient; a larger population could have theoretically shown a significant difference. We were unable to determine which block was superior, however, studies have shown that, while more technically challenging to place, quadriceps strength is preserved with an ACB. Therefore specific populations, such as patients at high risk for falling, may benefit from this technique.

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Spinal-induced Hypotension and Bradycardia Prevention with Ondansetron

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Keywords: spinal-induced hypotension, bradycardia, ondansetron, Bezold-Jarisch reflex, cesarean section

Introduction

Spinal anesthesia is the most common method of anesthesia provided for the parturient undergoing cesarean section. Spinal anesthesia is often preferred over epidural and general anesthesia because of its superior ease of administration, safety, and effectiveness.¹ Spinal anesthesia is chosen by many anesthesiologists because it avoids risks of general anesthesia, provides better postoperative pain relief, and allows the mother to remain awake to see the baby immediately after birth.² Although spinal anesthesia is a safe and an effective route of anesthesia

for cesarean section, it is associated with serious risks and complications to both the mother and baby.

The most common side effects associated with spinal anesthesia are hypotension and bradycardia, which occur at rates of 33% and 13% in the non-obstetric population.³ Hypotension occurs at an even higher rate of 50-60% in non-laboring obstetric patients.³ A prospective cross sectional study conducted by Somboonviboon et al⁴ involving 722 parturients undergoing cesarean section under spinal anesthesia confirmed the high incidence of hypotension and bradycardia. The research study observed hypotension to occur at a rate of 52.5% and bradycardia to occur at a rate of 2.5% and concluded that the risk of hypotension after spinal anesthesia for cesarean section is increased in cases where estimated blood loss was greater than 500mL and an analgesic level blockade higher than T4.⁴

After a review of the literature concerning cardiovascular effects after spinal anesthesia administration, all of the research studies reviewed confirm the Bezold-Jarisch reflex plays an important role in spinal-induced hypotension and bradycardia. Owczuk et al.⁵ describes the Bezold-Jarisch reflex as a triad of hypotension, bradycardia, and peripheral vasodilation. The origin of the Bezold-Jarisch reflex lies within cardiac sensory receptors, and activation of these receptors increases parasympathetic nervous system activity, inhibits sympathetic nervous system activity, and explains cardiovascular collapse that is associated with spinal and regional anesthesia.⁵

The Bezold-Jarisch reflex is a cardioinhibitory reflex and is composed of non-myelinated, type C afferent vagal fibers.⁵ Cardiac receptors that mediate the Bezold-Jarisch reflex cause inhibition of vasomotor centers when stimulated, promoting vasodilation and hypotension.² The receptors of the Bezold-Jarisch reflex located within the walls of the heart respond to systemic responses to hypervolemia and hypovolemia. After spinal blockade, there is a decreased venous return of blood to the heart that results in deformation of the cardiac wall and irritation of cardiac mechanoreceptors, leading to the activation of the Bezold-Jarisch reflex and thus inducing hypotension and bradycardia.⁵ The chemoreceptors that mediate the Bezold-Jarisch reflex are of the serotonin 5-HT₃ type. The activation of peripheral 5-HT₃ receptors located in intracardiac vagal nerve endings by serotonin further elicits the Bezold-Jarisch reflex resulting in hypotension and bradycardia.⁵ The stimulation of 5-HT₃ receptors located on sensory vagal nerve endings results in a lowered heart rate and an initial short-lasting hypotension followed by a longer-lasting hypotension, attributed to the Bezold-Jarisch reflex.⁵

It has been hypothesized that inhibition of serotonin 5-HT₃ receptors can result in the inhibition of the Bezold-Jarisch reflex, ultimately resulting in a decreased incidence of hypotension and bradycardia after spinal anesthesia administration.¹ Ondansetron was the 5-HT₃ receptor antagonist administered to negate the effects of the Bezold-Jarisch reflex in all clinical trials reviewed. Pasternak et al⁶ investigated the risk of adverse fetal outcomes associated with ondansetron in a study that included 608,385 pregnancies from the period of January 2004 through March 2011 and concluded there was no significantly increased risk of adverse fetal outcomes when ondansetron was taken during pregnancy.⁶

Hypotension and bradycardia can be harmful to the mother and fetus during cesarean section by placing the mother at risk for loss of consciousness, aspiration and cardiac arrest leading to hypoperfusion of the placenta.² Rates of hypotension and bradycardia following spinal anesthesia in the obstetric population are high, however, what is most concerning is the paucity of preemptive techniques to minimize the occurrence. Methods utilized by anesthesia professionals to prevent maternal hypotension after spinal anesthesia include fluid boluses, physical positioning to enhance blood return to the heart, vasopressors, and leg bindings.² These methods are often ineffective or harmful to the fetus.

Methodology

Evidence-based Practice Model

The PICO format was used to create a clinical question that would guide the search criteria. The PICO parameters were P (patient population) parturients undergoing spinal anesthesia for cesarean section, I (current intervention) administration of intravenous ondansetron five minutes prior to subarachnoid block, C (comparison) administration of placebo five minutes prior to subarachnoid block, O (outcome) decrease incidence of hypotension, bradycardia, and vasopressor administration.

Purpose

The purpose of this review is to evaluate the current body of literature regarding the effectiveness of the 5-HT₃ receptor antagonist ondansetron on reducing incidence of hypotension, bradycardia, and vasopressor usage in the obstetric population undergoing spinal anesthesia for cesarean section.

Search Terms

Spinal-induced hypotension, bradycardia, ondansetron, Bezold-Jarisch reflex, and cesarean section.

Search Methods

An electronic database search using CINAHL, ScienceDirect, DynaMed, Pubmed, Cochrane Database, ProQuest and Google Scholar were searched for studies published from 2008-2015. Keywords were searched within databases utilizing search terms, boolean operators, synonyms and truncation to gather additional results. Inclusion criteria included randomized controlled trials published from 2008-2015, English speaking journals, studies evaluating administration of a 5-HT₃ antagonist prior to spinal anesthesia on attenuation of hypotension, bradycardia, or vasopressor usage.

Levels of Evidence

Four studies met criteria for this review. The four studies were double-blinded randomized control trials.

Literature Review

Hypotension

All studies reviewed concluded that a significant reduction in hypotension was observed when administering ondansetron prior to spinal anesthesia. Marashi et al¹ defined hypotension as a MAP < 80mm Hg and observed mean arterial pressures that were statistically different between patients groups who were given 6 mg ondansetron (n = 70) and 12 mg ondansetron (n = 70) vs. normal saline (n = 70) 5 minutes prior to spinal anesthesia. In the control group, 12 patients (17%) experienced hypotension that was statistically significant compared to the ondansetron induced groups (P = 0.04).¹ There were no significant differences in MAP between the ondansetron groups (P = 0.06).¹

Sahoo et al³ examined 52 parturients scheduled for elective cesarean section and randomly allocated them into two groups. Before induction of spinal anesthesia Group O (n = 26) received 4 mg ondansetron and Group S (n = 26) received normal saline. MAP was recorded at one-minute intervals in both groups. Significant decreases in MAP were observed in both groups.³ Differences were observed at 5 min (Group O 88 ± 11.7 vs Group S 82.2 ± 10.5 mm Hg, P = 0.038) and 6 min (Group O 87.5 ± 11.3 vs. Group S 80.4 ± 10.8 mm Hg, P = 0.025).³ Those in Group S had a significantly lower MAP between 14 and 35 min.³

Rashad and Farmawy² examined 60 patients undergoing spinal anesthesia for cesarean section and randomly divided them into 3 equal groups. Five minutes prior to spinal anesthesia, Group O (n = 20) ondansetron 4mg, Group G (n = 20) granisetron 1mg, and Group S (n = 20) normal saline all received their respected doses. MAP was measured at 5-minute intervals in each group. In regards to decreases in MAP, there was significant differences between group O and both groups G and S at 5 (P = 0.03), 10 (P = 0.02), 15 (P = 0.001), 20 (P = 0.002), and 25 minutes (P = 0.013).²

Owczuk et al⁵ studied 71 patients undergoing spinal anesthesia and divided them into two groups. Ondansetron 8 mg (n = 36) and control normal saline (n = 35) were administered 5 minutes prior to spinal anesthesia. Systolic arterial pressures (SAP), diastolic arterial pressures (DAP) and mean arterial pressures were measured before blockade and at 5-minute intervals after spinal anesthesia.⁵ SAP values were significantly higher in the ondansetron group at the 10, 15, and 20 minute time points (P < 0.05).⁵ SAP below 90 mm Hg was recorded in 7 patients (20%) in the placebo group vs. 1 patient (2.8%) in the ondansetron group (P = 0.028).⁵ There were no significant differences in MAP and DAP between the 2 groups.⁵

Bradycardia

Evaluating the clinical effects regarding the administration of ondansetron prior to spinal anesthesia on the incidence of bradycardia was difficult due to the less frequent occurrence of bradycardia (13%) compared to hypotension (33%) under spinal anesthesia.¹ Marashi et al¹ defined bradycardia as HR < 50 bpm. Ten patients (14%) in the control group experienced bradycardia while no patients in the ondansetron 6 mg and ondansetron 12 mg groups experienced bradycardia (P = 0.02).¹ There were no significant differences in HR between the two ondansetron groups.¹

Sahoo et al³ compared HR at minute intervals between an ondansetron 4mg treatment group (Group O) and a normal saline group (Group S). Decreases in HR were more common in Group S, but differences were only statistically significant at 24 minutes (Group O: 93.9 ± 16.5 vs. Group S 82.9 ± 14.1 bpm, $P=0.031$) and at 45 minutes (Group O: 94.3 ± 16.2 vs. 83.1 ± 7.5 bpm, $P = 0.02$)³.

Rashad and Farmawy³ defined bradycardia as HR < 50 bpm and reported no significant differences in HR or occurrences of bradycardia between the ondansetron 4 mg (Group O), granisetron 1mg (Group G), and normal saline (Group S) groups. Bradycardia did not occur in Group O but occurred three times (15%) in Group G and twice (10%) in Group S ($P = 0.21$).³

Owczuk et al.⁵ observed no significant difference in HR between the ondansetron 8 mg and normal saline group at the same time points. No patients experience HR < 50 bpm in the ondansetron group while one patient in the control group received atropine 0.5 mg because of a HR of 48 bpm.⁵

It is worthy to note that although not all studies found significant reductions in bradycardia between ondansetron induced groups and control groups, no incidences of bradycardia (HR<50 bpm) were reported in any of the ondansetron treatment groups across all four of the studies examined.

Vasopressor Usage

The usage of vasopressors in the obstetric population can have adverse effects on uterine blood flow and may contribute to fetal acidosis.⁵ Two studies specifically examined the use of vasopressors. In the study conducted by Marashi et al¹, 12 patients (17%) in the control group experienced hypotension that was statistically significant and required vasopressor usage compared to the ondansetron treatment groups ($P = 0.04$).¹ In the study conducted by Rashad and Farmawy³, a significant increase in ephedrine administration related to hypotension was seen in the normal saline and granisetron groups compared to the ondansetron group. Ephedrine was administered in 35% of patients in the normal saline group and 25% of patients in the granisetron group, compared to only 5% in the ondansetron group ($P = 0.05$).³

Conclusions

The literature supports the administration of ondansetron 5 minutes prior to spinal anesthesia to prevent hypotension and decrease vasopressor usage. The findings for 5-HT₃ agents significantly decreasing the rate of bradycardia when compared to placebo were mixed; however, results suggest a possible benefit. A strength of the studies reviewed is that multiple dosages of ondansetron were tested in each study. Marashi et al¹ administered dosages of 6 mg and 12 mg, Sahoo et al³ administered 4 mg, Rashad and Farmawy² administered 4 mg, and Owczuk et al⁵ administered 8 mg. All dosages of ondansetron appeared to have similar effectiveness and significance. Marashi et al¹ saw no differences between dosages of 6 mg and 12 mg of ondansetron. No adverse events were reported with these larger dosages.

Reference	Hypotension	Bradycardia	Vasopressor Usage
Marashi et al., (2014). Comparing Two Different Doses of Intravenous Ondansetron with Placebo on Attenuation of Spinal-induced Hypotension and Shivering.	<p>-210 patients undergoing spinal anesthesia divided into 3 equal groups: ondansetron 6mg (n = 70), ondansetron 12mg (n=70), and control saline group (n = 70)</p> <p>-In control group 12 patients (17%) had MAP < 80mm Hg compared to experimental groups (P = 0.04)</p> <p>-No significant differences in MAP between experimental groups (P = 0.06)</p>	<p>-10 patients (14%) in control group had HR < 50 bpm compared to experimental groups (P = 0.02)</p> <p>-No significant differences in HR between experimental groups (P = 0.06)</p>	<p>In control group 12 patients (17%) had MAP < 80 mm Hg requiring vasopressor usage compared to experimental groups (P = 0.04)</p>
Sahoo et al., (2013). Reduction in Spinal-induced Hypotension with Ondansetron in Parturients Undergoing Cesarean Section.	<p>-52 parturients scheduled for elective cesarean section randomly allocated into two groups. Before induction of spinal anesthesia Group O (n = 26) received ondansetron 4 mg; Group S (n = 26) received normal saline</p> <p>-Significant decreases in MAP were observed in both groups. =Differences were observed at 5 min (Group O 88 ± 11.7 vs. Group S 82.2 ± 10.5 mm Hg, P = 0.025). -Those in group S had a significantly lower MAP between 14 and 35 min.</p>	<p>-Decreases in HR were more common in group S, but differences were statistically significant only twice: at 24 min (Group O: 93.9 ± 16.5 vs. Group S: 82.9 ± 14.1 beats/min, P = 0.031) and at 45min (Group O: 94.3 ± 16.2 vs. Group S: 83.1 ± 7.5 beats/min, P = 0.02)</p>	
Rashad & Farmawy, (2013). Effects of Intravenous Ondansetron and Granisetron on Hemodynamic	<p>-60 patients undergoing spinal anesthesia for cesarean section divided into 3 equal groups: Group O: ondansetron 4mg (n = 20), Group G: granisetron 1mg (n = 20), Group S:</p>	<p>-No significant differences in HR among the 3 groups</p> <p>-No statistically significant differences in occurrences of</p>	<p>- Ephedrine was administered in 35% of patients in the Group S and 25% of patients in Group G, compared to</p>

Changes and Motor and Sensory Blockade Induced by Spinal Anesthesia in Parturients Undergoing Cesarean Section.	control normal saline (n = 20) 5 min prior to spinal anesthesia. -In regards to decreases in MAP, there was a significant difference between group O and both groups G and S at 5 (P = 0.03), 10 (P = 0.02), 15 (P = 0.001), 20 (P = 0.002), and 25 min (P = 0.013).	bradycardia but 10% of patients in group S and 15% of patients in group G suffered from bradycardia and treated with atropine (P = 0.21).	only 5% in Group O (P = 0.05).
Owczuk et al., (2008). Ondansetron Given Intravenously Attenuates Arterial Blood Pressure Drop due to Spinal Anesthesia.	-71 patients undergoing spinal anesthesia divided into 2 groups: ondansetron 8mg (n = 36) and control normal saline (n = 35) administered 5 min prior to spinal anesthesia -SAP values were significantly higher in the ondansetron group at the 10, 15, and 20 minute time points (P < 0.05) -SAP below 90mm Hg was recorded in 7 patients in the placebo group and in 1 patient in the ondansetron group (20% vs. 2.8%) (P = 0.028) -There were no significant differences in MAP and DAP between the 2 groups	-There were no significant differences in HR between the 2 groups at the same time points	

Table 1. Recent literature regarding ondansetron administration and prevention of spinal-induced hypotension, bradycardia, and vasopressor usage

Several inconsistencies and limitations were encountered while evaluating the bodies of literature. Varying definitions of bradycardia and hypotension were described across all of the studies reviewed. For example, Marashi et al.¹ defined hypotension as mean arterial pressure less than 80 mm Hg and bradycardia as heart rate less than 50 bpm while Sahoo et al.³ compared mean arterial pressures and heart rates between control and experimental groups at minute intervals.

The largest study group consisted of 210 patients while the smallest group consisted of 52 patients. Bradycardia occurs at a rate of nearly three times less than hypotension after spinal anesthesia.⁵ In small-observed groups of patients, frequency of bradycardia may be too small to

observe significant differences between ondansetron-induced groups and control groups.⁵ This proved to be the case in multiple studies where differences in heart rates were observed, but were not frequent enough to be statistically significant.

After a thorough review of the literature, several recommendations for the anesthesia professional are suggested. A large-scale double-blinded randomized controlled trial should be performed on only obstetric patients undergoing spinal anesthesia for cesarean section. Dosages of ondansetron administered suggested 4 mg and 12 mg to determine if there is any difference between low dose and high dose regarding efficacy and adverse effects. Parameters for evaluating hypotension and bradycardia must be clearly defined. Most prevalent parameters in the literature reviewed were hypotension defined as mean arterial pressure less than 80 mm Hg or a 20% decrease from baseline and bradycardia defined as a heart rate less than 50 beats per minute. Vasopressor usage must be measured quantitatively in both the ondansetron-administered groups and control group.

The literature review on the use of ondansetron to prevent spinal-induced hypotension and bradycardia provided a comprehensive and meaningful background for the development of an evidence-based practice analysis report. Hypotension and bradycardia occur at alarming rates in patients undergoing spinal anesthesia and have been reported to be as high as 60% and 13% in the obstetric population.³ In spite of study limitations, current evidence supports the prophylactic use of ondansetron to reduce the incidence of spinal blockade mediated bradycardia and hypotension. Ondansetron dosage ranges of 4 mg-12 mg given the intravenous route have consistently demonstrated significant reductions in hypotension, bradycardia and vasopressor usage.

References

1. Marashi SM, Soltani-Omid S, Mohammadi SS, Aghajani Y, Movafegh A. Comparing two different doses of intravenous ondansetron with placebo on attenuation of spinal-induced hypotension and shivering. *Anesthesiol Pain Med*. 2014;3:1-5
2. Rashad M, Farmawy M. Effects of intravenous ondansetron and granisetron on hemodynamic changes and motor and sensory blockade induced by spinal anesthesia in parturients undergoing cesarean section. *Egyptian J Anaesth*. 2013;29(4):369-374.
3. Sahoo, SenDasgupta, Goswami, Hazra. Reduction in spinal-induced hypotension with ondansetron in parturients undergoing cesarean section. *Obstetric Anesthesia Digest*. 2013;33(1):31-32.
4. Somboonviboon W, Kyokong O, Charuluxananan S, Narasethakamol A. Incidence and risk factors of hypotension and bradycardia after spinal anesthesia for cesarean section. *J Med Assoc Thai*. 2008;91:181-187
5. Owczuk R, Wenski W, Polak-Krzeminska A, et al. Ondansetron given intravenously attenuates arterial blood pressure drop due to spinal anesthesia. *Reg Anesth Pain Med*. 2008;33(4):332-339.
6. Pasternak B, Svanström H, Hviid A. Ondansetron in pregnancy and risk of adverse fetal outcomes. *Surv Anesthesiol*. 2013;57(5):231-232.

7. Palmese S, Manzi M, Visciano V, Scibilia A, Natale A. Reduced hypotension after subarachnoid anaesthesia with ondansetron most colloids in parturients undergoing caesarean section. a retrospective study. *The Internet Journal of Anesthesiology*. 2012;30(4).
8. Kee WND, Khaw KS, Tan PE, Ng FF, Karmakar MK. Placental transfer and fetal metabolic effects of phenylephrine and ephedrine during spinal anesthesia for cesarean delivery. *Anesthesiology*. 2009;111(3):506-512.

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The Role of Tranexamic Acid in Trauma Surgical Patients

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Keywords: tranexamic acid, trauma, antifibrinolytic agents, hemorrhage, bleeding

Introduction

Trauma is the sixth leading cause of death in all age groups and the first leading cause of death in individuals under age thirty-five worldwide.¹ Hemorrhage during the first few hours of trauma accounts for 80% of all deaths in trauma patients.¹ Trauma and major surgeries trigger similar hemostatic responses in which blood vessels contract, platelet plugs form, and coagulation begins to stop the bleeding.² At the same time, the fibrinolytic system is activated to dissolve clots and start remodeling of the damaged tissue.² Thus, severe blood loss presents a challenge in both trauma and major surgeries.

Currently, blood transfusions remain a key treatment modality, after establishment of hemostasis, in bleeding trauma patients and during emergency surgeries. Approximately 12 million units of packed red blood cells are transfused each year in the United States (US) with trauma patients accounting for 10-15%.³ Although the safety of blood transfusions has been established with screening of donated blood for infectious diseases such as Human Immunodeficiency Virus (HIV), Hepatitis B, and Hepatitis C, blood transfusion is still an independent risk factor for adverse patient outcomes.⁴ Blood transfusions are linked to increased mortality, increased length of hospital stay, infections, sepsis, and multi-organ system dysfunction.⁴ In addition, the estimated cost of one unit of packed red blood cells is between \$700-\$1200 U.S. dollars when all the processes involved in blood transfusion are taken into account.⁴

Tranexamic acid, an antifibrinolytic drug, prevents blood clots from breaking down and reduces blood loss by inhibiting plasminogen activation and plasmin activity.² Tranexamic acid binds ten times more strongly than aminocaproic acid, an older antifibrinolytic drug, at the receptor sites of plasminogen molecule and is proven to be more effective than aminocaproic acid.² Tranexamic acid, thus, should be the leading treatment option in hemorrhaging trauma patients to decrease exposure to allogeneic blood transfusions.

Methodology

Evidence-based Practice Model

The PICO format was used to formulate an evidence-based practice question that would guide the search criteria. The acronym PICO stands for population (P), intervention (I), comparison of interest (C), and outcome (O). The questions is: In adult trauma patients undergoing surgery, how does the use of intravenous (IV) tranexamic acid compared to placebo or no treatment affect mortality, blood transfusion requirements, safety, and cost peri-operatively?

Purpose

The purpose of this evidence-based practice analysis is to evaluate empirical evidence on the effects of tranexamic acid with regards to mortality, blood transfusion requirements, safety, and cost.

Search Terms

Tranexamic acid, trauma, antifibrinolytic agents, hemorrhage, bleeding

Search Models

An electronic database search was conducted using ScienceDirect, Cochrane Database of Systematic Reviews, CINAHL, PubMed, and Google Scholar. The inclusion criteria were peer-reviewed journals published between 2010 and 2015, articles published in English, adult subjects age 18 and older undergoing emergency surgeries, and articles evaluating the effects of tranexamic acid on mortality, blood transfusion requirements, safety, and cost.

Levels of Evidence

Studies found using the described search model were assessed for their levels of evidence based on their design. For this evidence-based practice analysis, eight studies were selected. Four systematic reviews of randomized controlled trials provided level I evidence. A randomized controlled trial provided level II evidence. Two prospective studies and one retrospective cohort study provided level IV evidence.

Literature Review

Mortality

The relationship between administration of tranexamic acid and mortality in hemorrhaging trauma patients has been extensively studied. Clinical Randomisation of an Antifibrinolytic in Significant Haemorrhage 2, widely known as CRASH-2, is a groundbreaking study conducted to examine the effects of administration of tranexamic acid on death, vascular occlusive events, and the receipt of blood transfusions in trauma patients.⁵ This randomized, double-blind, placebo-controlled trial involving 20,211 patients was undertaken in 274 hospitals in 40 countries.⁵ In this study, hemorrhaging trauma patients were randomly assigned to either tranexamic acid regimen or placebo regimen: each containing a loading dose of 1 g infusion over 10 minutes, followed by a 1 g infusion over 8 hours.⁵ The researchers from the CRASH-2 trial concluded the administration of tranexamic acid to trauma patients with, or at risk of, bleeding significantly reduces overall mortality by 14.5% (n=1463) when compared to placebo at 16% (n=1613) with a *P* value of 0.0035.⁵

A systematic review of four randomized controlled trials involving 20,548 patients further solidifies the effects of tranexamic acid on reducing mortality.⁶ This systematic review established tranexamic acid reduces the risk of death by 10% and the risk of death due to bleeding by 15% when administered within three hours of injury.⁶

A retrospective observational study, titled Military Application of Tranexamic Acid in Trauma Emergency Resuscitation (MATTERs), assessed the effects of tranexamic acid in patients with combat injuries.⁷ The researchers selected a total of 896 patients with combat injury, of which 293 patients received tranexamic acid, from prospectively collected U.K. and U.S. trauma registries.⁷ The researchers concluded the group receiving tranexamic acid had lower mortality than the group without treatment.⁷

Blood Transfusion

There is significant research about blood transfusion requirements in trauma patients receiving tranexamic acid. A systematic review of five randomized controlled trials concluded that tranexamic acid reduces the probability of receiving a blood transfusion by 30% when compared to either no treatment with tranexamic acid or treatment with desmopressin in patients undergoing emergency or urgent surgery.⁸

A meta-analysis of 129 randomized controlled trials totaling 10,488 patients compared patients treated with tranexamic acid with either no treatment or placebo in elective and emergency surgeries.⁹ The researchers concluded that tranexamic acid reduces blood transfusion requirement by 38% ($P < 0.001$).⁹

A third systematic review of 65 randomized controlled trials compared the use of tranexamic acid to no treatment. Of the 4842 participants, 2528 patients were randomly assigned to receive tranexamic acid and remaining 2314 patients were assigned to control group. The researchers concluded that the group receiving IV tranexamic acid had a decreased need for allogeneic blood transfusion by 39%.¹⁰

Safety

The safety of tranexamic acid has been questioned due to its mechanism of prevention of clot breakdown. The CRASH-2 trial examined the effects of tranexamic acid on thromboembolic complications such as myocardial infarction (MI), stroke, pulmonary embolism (PE), and deep vein thrombosis (DVT).⁵ The researchers of this randomized-controlled trial concluded tranexamic acid does not increase the risk of fatal or non-fatal vascular occlusive events when compared to placebo.⁵ In fact, 201 patients allocated to placebo had one or more vascular occlusive events compared to 168 patients from tranexamic acid group.⁵ Findings from the MATTERs study also revealed that tranexamic acid was independently associated with less coagulopathy when compared to no treatment ($P = 0.003$).⁷

The systematic review by Perel et al⁸ established tranexamic acid did not result in any events of PE or MI.⁸ The systematic review by Roberts et al⁶ also supported tranexamic acid is safe and no correlation was found between use of tranexamic acid and thrombosis.⁶ The authors concluded there was no evidence that tranexamic acid increased the risk of vascular occlusive events or need for surgical intervention.⁶

Cost-Effectiveness

In addition to decreasing mortality and blood transfusion requirements, tranexamic acid is also a cost-effective intervention. Guerriero et al¹¹ conducted an analysis of data from the CRASH-2 trial to assess the cost effectiveness of administering tranexamic acid to trauma patients in Tanzania, India, and the United Kingdom (UK).¹¹ These three countries represented low, middle, and high income settings respectively.¹¹ The primary outcome measured in this study was the number of life years (LY) gained.¹¹ Cost effectiveness was measured in international dollars per LY.¹¹ The researchers concluded that administering tranexamic acid to hemorrhaging trauma patients within three hours of injury saved an estimated 372, 315, and 755 LYs per 1000 trauma patients in Tanzania, India, and the UK respectively.¹¹ The cost of administering tranexamic acid to 1000 patients was \$17,483 in Tanzania, \$19,550 in India, and \$30,830 in the UK.¹¹ On the other hand, the cost of not administering tranexamic acid was \$18 025 in Tanzania, \$20 670 in India, and \$48 002 in the UK.¹¹ The analysis of data revealed that early administration of tranexamic acid is more cost-effective than no treatment.¹¹

To further strengthen the cost-effectiveness of tranexamic acid, the cost of administering blood transfusion was taken into account. Shander et al¹² conducted a prospective study utilizing blood transfusion data from two U.S. and two European hospitals.¹² The researchers' aim was to analyze the total cost of blood, accounting for all of the processes involved in administering a blood transfusion in the surgical setting.¹² The researchers found one unit of packed red blood cells costs between \$522 and \$1183 U.S. dollars.¹² Annual expenditures on blood transfusions in surgical patients ranged from \$1.62 to \$6.03 million U.S. dollars per hospital.¹² Thus, when the cost of administering tranexamic acid is compared to either the cost of no treatment or administering blood transfusions, it is clear that tranexamic acid is a cost-effective solution to saving lives in hemorrhaging trauma patients.

Author & Date	Study Design	Sample & Setting	Major Findings
Shakur et al, 2010 ⁵	Randomized, double-blind, placebo-controlled trial	20,211 patients from 274 hospitals in 40 countries. 10 096 patients allocated to tranexamic acid group vs. 10,115 patients allocated to placebo group. A loading dose of 1 g over 10 minutes, followed by 1 g continuous infusion over 8 hours of tranexamic acid or placebo.	The administration of tranexamic acid to trauma patients with, or at risk of, bleeding significantly reduces overall mortality when compared to placebo with a <i>P</i> value of 0.0035. Tranexamic acid does not increase the risk of fatal or non-fatal vascular occlusive events when compared to placebo.
Roberts et al, 2012 ⁶	Systematic review of randomized controlled trials	Two randomized controlled trials with a combined total of 20,451 patients. Tranexamic acid 1 g loading dose over 10 minutes, followed by 1 g	Tranexamic acid reduces the risk of death by 10% and the risk of death due to bleeding by 15% when administered within three hours of injury. There was

		continuous infusion over 8 hours vs. matching placebo.	no evidence that tranexamic acid increased the risk of vascular occlusive events.
Morrison et al, 2012 ⁷	Retrospective observational study	896 patients with combat injuries selected from U.K. and U.S. trauma registries. 293 patients from tranexamic acid group vs. 603 patients with no treatment. Tranexamic acid dose included 1 g loading dose and repeated as determined by clinician.	The group receiving tranexamic acid had lower mortality than the group without treatment. Tranexamic acid was independently associated with less coagulopathy with a <i>P</i> value of 0.003 when compared to no treatment.
Perel et al, 2013 ⁸	Systematic review of randomized controlled trials	260 patients from three randomized controlled trials assigned to either tranexamic acid vs. no treatment or treatment with desmopressin.	Tranexamic acid reduces the probability of receiving a blood transfusion by 30% when compared to either no treatment or treatment with desmopressin in patients undergoing emergency or urgent surgery. Tranexamic acid group did not result in any events of PE or MI.
Ker et al, 2012 ⁹	Meta-analysis of randomized controlled trials	10,488 patients assigned to either tranexamic acid vs. no treatment.	Tranexamic acid reduces the probability of receiving a blood transfusion by 38% with a <i>P</i> value of less than 0.001.
Henry et al, 2011 ¹⁰	Systematic review of randomized controlled trial	65 randomized controlled trials with a total of 4842 patients. 2528 patients from tranexamic acid group vs. 2314 patients from control group. Tranexamic acid bolus dose ranged from 2.5 mg/kg to 100 mg/kg. The maintenance dose ranged from 0.25 mg/kg/hr to 4 mg/kg/hr over 1 to 12 hours.	The use of tranexamic acid significantly reduces the need for allogeneic blood transfusion by 39%.
Guerriero et al,	Prospective study	Data from CRASH-2	The cost of administering

2011 ¹¹		trial was analyzed. The dose of tranexamic acid administered was 1 g loading dose over 10 minutes, followed by 1 g continuous infusion over 8 hours vs. matching placebo.	tranexamic acid to 1000 patients was \$17,483 in Tanzania, \$19 550 in India, and \$30 830 in the UK. The cost of not administering tranexamic acid was \$18 025 in Tanzania, \$20 670 in India, and \$48 002 in the UK. Early administration of tranexamic acid would cost \$48, \$66, and \$64 per LY saved in Tanzania, India, and the UK respectively.
Shander et al, 2010 ¹²	Prospective study	Blood transfusion data from two U.S. and two European hospitals were used. A combined 21,614 units of packed red blood cells were transfused in surgical patients.	One unit of packed red blood cells costs between \$522 and \$1183 when all steps involved in blood transfusion are considered. Annual expenditures on blood transfusions in surgical patients ranged from \$1.62 to \$6.03 million per hospital.

Conclusions

The widespread use of tranexamic acid has the potential to save 70,000 to 100,000 lives per year around the world.¹¹ Tranexamic acid use in hemorrhaging trauma patients correlates with reduced mortality and blood transfusion requirements without increasing the risk of thromboembolic events. Tranexamic acid is also cost-effective if routinely administered within three hours of injury in a broad range of clinical settings. After recent breakthroughs in research, tranexamic acid has been included in the World Health Organization's list of essential medicines.² Based on the empirical evidence, tranexamic acid is recommended as a first-line treatment to reduce mortality, blood transfusion requirements, and cost in hemorrhaging trauma patients.

References

1. Alberdi F, Garcia I, Atutxa L, Zabarte M. Epidemiology of severe trauma. *Medicina Intensiva*. 2014;39(9):580-588.
2. Tengborn L, Blomback M, Berntorp E. Tranexamic acid: an old drug still going strong and making a revival. *Thrombosis Research*. 2014;135:231-242.

3. Fraga GP, Bansal V, Coimbra R. Transfusion of blood products in trauma: an update. *J Emerg Med.* 2010;39(2):253-260. doi:10.1016/j.jemermed.2009.02.034.
4. Spahn DR, Goodnough LT. Alternatives to blood transfusion. *Lancet.* 2013;381:1855-1865. doi:10.1016/S0140-6736(13)60808-9.
5. Shakur H, Roberts I, Bautista R, et al. Effects of tranexamic acid on death, vascular occlusive events, and blood transfusion in trauma patients with significant haemorrhage (crash-2): a randomised, placebo-controlled trial. *Lancet.* 2010;376:23-32. doi:10.1016/S0140-6736(10)60835-5.
6. Roberts I, Shakur H, Ker K, Coats T. Antifibrinolytic drugs for acute traumatic injury. *Cochrane Database Syst Rev.* 2012. doi:10.1002/14651858.CD004896.pub3.
7. Morrison JJ, Dubose JJ, Rasmussen TE, Midwinter MJ. Military application of tranexamic acid in trauma emergency resuscitation (MATTERs) study. *Arch Surg.* 2012;147(2):113-119.
8. Perel P, Ker K, Morales Uribe CH, Roberts I. Tranexamic acid for reducing mortality in emergency and urgent surgery. *Cochrane Database Syst Rev.* 2013:1-3. doi:10.1002/14651858.CD010245.pub2. Copyright.
9. Ker K, Edwards P, Perel P, Shakur H, Roberts I. Effect of tranexamic acid on surgical bleeding: systematic review and cumulative meta-analysis. *BMJ.* 2012;344:e3054-e3054. doi:10.1136/bmj.e3054.
10. Henry DA, Carless PA, Moxey AJ, et al. Anti-fibrinolytic use for minimising perioperative allogeneic blood transfusion. *Cochrane Database Syst Rev.* 2011. doi:10.1002/14651858.CD001886.pub4.
11. Guerriero C, Cairns J, Perel P, Shakur H, Roberts I. Cost-effectiveness analysis of administering tranexamic acid to bleeding trauma patients using evidence from the crash-2 trial. *PLoS One.* 2011;6. doi:10.1371/journal.pone.0018987.
12. Shander A, Hofmann A, Ozawa S, Theusinger OM, Gombotz H, Spahn DR. Activity-based costs of blood transfusions in surgical patients at four hospitals. *Transfusion.* 2010;50:753-765.

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Editorial

Please join me in thanking Robert Hawkins, PhD, DNP, MS, MBA, CRNA for his dedicated service to the ISJNA, and wish him the best as he steps down from the editorial board. Dr. Hawkins served as a reviewer for several years prior to joining the editorial board in 2009. He recruited countless other individuals to join our volunteer ranks, and I can't thank him enough for his contributions and support. As we wish Dr. Hawkins a fond farewell, I would like to acknowledge our newest editorial board members:

Marianne Cosgrove, CRNA, DNAP – Yale-New Haven Hospital School of Nurse Anesthesia

Anne Marie Hranchook, CRNA, DNAP – Oakland University – Beaumont

Ann Miller, CRNA, DNP – Florida Gulf Coast University

J. Dru Riddle, CRNA, DNP, PhD – Texas Christian University

Tina Williams, CRNA, MSN – Carl R. Darnall Army Medical Center

Welcome to the student journal family!

Sincerely,

A handwritten signature in cursive script that reads "Vicki C. Coopmans".

Vicki C. Coopmans, CRNA, PhD
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.”

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INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA GUIDE FOR AUTHORS

MISSION STATEMENT

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.

ITEMS ACCEPTED FOR PUBLICATION

Case reports, research abstracts, evidence-based practice (EBP) analysis reports, 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). 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. We encourage authors and mentors to critically evaluate the topic and the quality of the writing. If the topic and the 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*.

ITEM PREPARATION & SUBMISSION

Student authors prepare case reports, abstracts, EBP analysis reports, and letters to the editor with the guidance of a mentor. Only students may be authors. Case and EBP analysis reports must be single-authored. Abstracts may have multiple authors. **Mentors should take an active role** in reviewing the item to ensure appropriate content, writing style, and format prior to submission.

The original intent of this journal was 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** to the author's date of graduation.

PEER REVIEW

Items submitted for publication are initially reviewed by the editor. Items may be rejected, or returned to the mentor with instructions for the author to revise and resubmit prior to initiation of the formal review process. All accepted submissions undergo a formal process of blind review by at least two ISJNA reviewers. After review, items may be accepted without revision, accepted with revision, or rejected with comments.

General guidelines

1. Items for publication must adhere to the *American Medical Association Manual of Style* (AMA, the same guide utilized by the *AANA Journal* and such prominent textbooks as *Nurse Anesthesia* by Nagelhout and Plaus). The review process will not be initiated on reports submitted with incorrect formatting and will be returned to the mentor for revision. Please note the following:
 - a. Use of abbreviations is detailed in Section 14. Spell out acronyms/initialisms when first used. If you are using the phrase once, do not list the acronym/initialism at all.
 - b. Instructions regarding units of measure can be found in Section 18. 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. Some examples: height/length should be reported in cm, weight in kg, temperature in °C, pressure in mm Hg or cm H₂O.
 - c. In general, first use of pulmonary/respiratory abbreviations should be expanded, with the following exceptions: O₂, CO₂, PCO₂, PaCO₂, PO₂, PaO₂. Please use SpO₂ for oxygen saturation as measured by pulse oximetry.
 - d. Use the nonproprietary (generic) name of drugs - avoid proprietary (brand) names. Type generic names in lowercase. When discussing dosages state the name of the drug, *then* the dosage (midazolam 2 mg).
 - e. 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:

“A GlideScope (Verathon Inc., Bothell, WA) was used to”

Please note, TM and ® symbols are not used per the AMA manual.
 - f. Examples of referencing are included later in this guide.

2. Report appropriate infusion rates and gas flow rates:
 - a. When reporting infusion rates report them as mcg/kg/min or mg/kg/min. 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. Keep the gas laws in mind when reporting flow rates. Report the liter flows of oxygen and nitrous oxide and the percent of the volatile agent added to the gas mixture. Statements such as “40% oxygen, 60% nitrous oxide and 3% sevoflurane” do not = 100% and are thus incorrect. For example, “General anesthesia was maintained with sevoflurane 3% inspired concentration in a mixture of oxygen 1 L/min and air 1 L/min”.
3. 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. Place one space after the last punctuation of sentences. 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.
4. Do not use Endnotes or similar referencing software. Please remove all hyperlinks within the text.
5. Avoid jargon.
 - 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 was administered by face mask."
 - c. *The patient was intubated and put on a ventilator.* “The trachea was intubated and respiration was controlled by a mechanical ventilator.
 - d. *The patient had been on Motrin for three days.* “The patient had taken ibuprofen for three days.”
 - e. Avoid the term “MAC” when referring to a sedation technique - the term sedation (light, moderate, heavy, unconscious) sedation may be used. Since all anesthesia administration is monitored, the editors prefer to use specific pharmacology terminology rather than reimbursement terminology.
6. Use the words “anesthesia professionals” or “anesthesia practitioners” when discussing all persons who administer anesthesia (avoid the reimbursement term “anesthesia providers”)
7. References
 - a. Again, the **AMA Manual of Style** must be adhered to for reference formatting.
 - b. All should be within the past 8 years, except for seminal works essential to the topic being presented.
 - c. Primary sources are preferred.
 - d. All items cited must be from peer-reviewed sources – use of internet sources must be carefully considered in this regard.
 - e. Numbering should be positioned at the one-inch margin – text should begin at 1.25”.
8. See each item for additional information.
9. **Heading** for each item (Case Report, Abstract, EBPA Report) must adhere to the following format:

Title (bold, centered, 70 characters or less)

[space]

Author Name (centered, include academic credentials only)

Name of Nurse Anesthesia Program (centered)

[space]

Anticipated date of graduation (italics, centered, will be removed prior to publication)

E-mail address (italics, centered, will be removed prior to publication)

[space, left-justify from this point forward]

Keywords: (‘Keywords:’ in bold, followed by keywords (normal font) that can be used to identify the report in an internet search.)

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 do not count against the word count. 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 #9 above in General Guidelines)

[space]

A brief introductory paragraph of less than 100 words to focus the reader's attention. 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 pertaining to your topic.

[space]

Case Report (bold, 400-500 words)

[space]

This portion discusses the case performed in *400 words or less*, 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.

Patient description: height, weight, age, gender.

History of present illness

Statement of co-existing conditions/diseases

Mention the current medications, **generic names only**. (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 after the values (eg. Mmol/L or mg/dL).

Physical examination/Pre-anesthesia evaluation - **significant** findings only. Include the ASA Physical Status and Mallampati Classification **only** if pertinent to the case.

Anesthetic management (patient preparation, induction, maintenance, emergence, post-operative recovery).

Despite the detail presented here it is only to help the author organize the structure of the report. 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 real point of your paper which is the discussion and teaching/learning derived from the case.

[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. Diag 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. No more than 2 textbooks may be included in the reference list, and all references should be no older than 8 years, except for seminal works essential to the topic. This is also an exercise in evaluating and using current literature.

[space]

Mentor: (bold, followed by mentor name and credentials in normal text)

E-mail address (italics, will be removed prior to publication)

Research Abstracts

Research abstracts are limited to 500 words. References are not desired but may be included if considered essential. Note that this abstract is different from a research proposal. This abstract reports the *outcome* of your study. Use the same format described for the case report with the exception of the section headings:

Heading (see #9 above in General Guidelines)

[space]

Introduction (bold)

[space]

A brief introductory paragraph including purpose and hypotheses.

[space]

Methods (bold)

[space]

Include research design and statistical analyses used

[space]

Results (bold)

[space]

Present results – do not justify or discuss here.

[space]

Discussion (bold)

[space]

Discuss results

[space]

References (bold)

[space]

Not required, but a maximum of 5 references is allowed.

[space]

Mentor: (bold, followed by mentor name and credentials in normal text)

E-mail address (italics, 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 and population. 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. Please note that text books and non-peer reviewed internet sources should be avoided, and sources of reference should be less than 8 years old unless they are seminal works specifically related to your topic of inquiry:

Heading (see #9 above in General Guidelines)

[space]

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]

Methodology (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]

Review and critique the pertinent and current literature, determining scientific credibility and limitations of studies reviewed. Your synthesis table would be 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]

[space]

A minimum of 8 references is recommended, with a maximum of 12 allowed.

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.

<http://www.docstyles.com/amastat.htm#Top>

<http://healthlinks.washington.edu/hsl/styleguides/ama.html>

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. The titles of text books are also printed in *italics*. Please pay close attention to ensure correct punctuation.

Journals

Note there is a comma 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). The pages are inclusive - **do not omit digits**.

Some journals (and books) 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:

Hamdan A, Sibai A, Rameh C, Kanazeh G. Short-term effects of endotracheal intubation on voice. *J Voice*. 2007;21(6):762-768.

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.

Texts

There is a difference in citing a text with one or more *authors* from a text with one or more *editors*. Texts that are *edited* give credit to the authors of the chapters. They must be annotated and the **inclusive** pages of the chapter are noted. Texts that are *authored* do not have different chapter authors, the chapter is not cited by heading **but the inclusive pages where the information was found are cited**, unless the entire book is cited.

Text:

Stoelting R, Dierdorf S. *Anesthesia and Co-Existing Disease*. 3rd ed. Philadelphia: Churchill Livingstone; 1993:351-354.

Chapter from a text:

Burkard J, Olson RL, Vacchiano CA. Regional anesthesia. In Nagelhout JJ, Plaus KL, eds. *Nurse Anesthesia*. 4th ed. St. Louis:Elsevier; 2010:977-1030

Each chapter was written by a different author. Note the chapter's author gets the prominent location. The chapter title is cited; "editor" is abbreviated in a lowercase. The word "edition" is also abbreviated and in lower case. The inclusive pages of the chapter are cited.

Electronic references

Only established, peer-reviewed sources may be referenced. Please do not reference brochures or informational websites where a peer-review process cannot be confirmed. Authors are cautioned to not copy and paste from these without full credit and quotation marks where appropriate. Electronic references are cited using the following format:

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

For online journals, the accessed date may be the only date available, and in some cases no page numbers.

Examples:

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

Gupta A, Aggarwal N, Sharma D. Ultrasound guided ilioinguinal block. *The Internet Journal of Anesthesiology*. 2011;29(1). http://www.ispub.com/journal/the_internet_journal_of_anesthesiology/volume_29_number_1/article/ultrasound-guided-ilioinguinal-block.html. Accessed August 1, 2011.

ACADEMIC INTEGRITY

Issues of academic integrity are the primary responsibility of the author and mentor. Accurate and appropriate acknowledgement of sources is expected. **Any violation will be cause for rejection of the article.**

"Plagiarism is defined as the act of passing off as one's own the ideas, writings, or statements of another. Any act of plagiarism is a serious breach of academic standards, and is considered an offense against the University subject to disciplinary action. Any quotation from another source, whether written, spoken, or electronic, must be bound by quotation marks and properly cited. Any paraphrase (a recapitulation of another source's statement or idea in one's own words) or summary (a more concise restatement of another's ideas) must be properly cited."

http://grad.georgetown.edu/pages/reg_7.cfm

HOW TO SUBMIT AN ITEM

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 be "Submission to Student Journal". 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 AND PUBLICATION

If the editor does not acknowledge receipt of the item within one week, assume that it was not received and please inquire. Upon receipt, the Editor will review the submission for compliance with the Guide to Authors. If proper format has not been following 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.

Once the item has been accepted for review the Editor will send a blinded copy to a Section Editor, who will then coordinate a blinded review by two reviewers who are not affiliated with the originating program. The reviewers recommend publication to the Section Editor or make recommendations for changes to be addressed by the author. The Section Editor will return the item to the Editor, who will return it to the mentor for appropriate action (revision, approval to print). If the article is returned to the author for repair it is usually to answer a specific question related to the case that was not clear in the narrative or it asks the author to provide a reference for a statement. Every effort is made to place the returned article in the earliest next issue.

The goal is for all articles submitted by students to be published while the author is still a student. Therefore, deadlines must be met and the entire process must be efficient. If an item is not ready for publication within 3 months after the student author has graduated it will no longer be eligible for publication. For this reason it is recommended that case reports be submitted at least 4-6 months prior to the student author's anticipated graduation date.

Mentors of the papers may be asked to serve as reviewers of case reports by student authors from other prog and 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. Include a legend describing the activity and who is in the photo and identify the photographer. Only digital photos of high quality will be accepted via email to INTSJNA@aol.com. There must be a follow up hard copy signed by all present in the photo, as well as the photographer/ owner of the original photo, giving consent to publish the photo. Mail that consent to:

Vicki C. Coopmans, CRNA, PhD
Webster University
470 E. Lockwood Ave. Suite 15
St. Louis, MO 63119

SUBMISSION CHECK LIST

<p><input type="checkbox"/> AMA Manual of Style and other format instructions are adhered to.</p> <p><input type="checkbox"/> Total word count not exceeded (1400 for case report, 500 for abstract, 3000 for EBPA).</p> <p><input type="checkbox"/> The item is one continuous Word document without artificially created page breaks.</p> <p><input type="checkbox"/> Verbatim phrases and sentences are quoted and referenced.</p> <p><input type="checkbox"/> All matters that are not common knowledge to the author are referenced.</p> <p><input type="checkbox"/> Generic names for drugs and products are used throughout and spelled correctly in lower-case.</p> <p><input type="checkbox"/> Units are designated for all dosages, physical findings, and laboratory results.</p> <p><input type="checkbox"/> Endnotes, footnotes not used.</p> <p><input type="checkbox"/> Jargon is absent.</p> <p>Heading</p> <p><input type="checkbox"/> Concise title less than 70 characters long</p> <p><input type="checkbox"/> Author name, credentials, nurse anesthesia program, graduation date and email are included.</p> <p><input type="checkbox"/> Five Keywords are provided</p> <p>Case Report</p> <p><input type="checkbox"/> Introduction is less than 100 words.</p> <p><input type="checkbox"/> Case Report section states only those facts vital to the account (no opinions or rationale)</p> <p><input type="checkbox"/> Case report section is 400-500 words and not longer than the discussion.</p> <p><input type="checkbox"/> Discussion section is 600-800 words.</p> <p><input type="checkbox"/> Discussion of the case management is based on a review of current literature</p> <p><input type="checkbox"/> Discussion concludes with lessons learned and how the case might be better managed in the future.</p> <p>Abstract</p> <p><input type="checkbox"/> The 500 word count maximum is not exceeded.</p> <p><input type="checkbox"/> Abstract reports the <i>outcome</i> of your study.</p> <p><input type="checkbox"/> Includes Introduction, Methods, Results, and Conclusion sections.</p> <p>EBPA Report</p> <p><input type="checkbox"/> The 3000 word count maximum is not exceeded.</p> <p><input type="checkbox"/> A critical evaluation of a practice pattern in the form of a precise clinical question about a specific intervention and population is presented.</p> <p><input type="checkbox"/> A focused foreground question following either the PICO or SPICE format is used.</p> <p><input type="checkbox"/> Includes Introduction, Methodology, Literature Analysis, and Conclusion sections.</p> <p>References</p> <p><input type="checkbox"/> AMA Style for referencing is used correctly.</p> <p><input type="checkbox"/> Reference numbers are sequenced beginning with one and superscripted.</p> <p><input type="checkbox"/> References are from anesthesia and other current <u>primary</u> source literature.</p> <p><input type="checkbox"/> All inclusive pages are cited, texts as well as journals.</p> <p><input type="checkbox"/> Journal titles are abbreviated as they appear in the PubMed Journals Database.</p> <p><input type="checkbox"/> Number of references adheres to specific item guidelines.</p> <p><input type="checkbox"/> Internet sources are currently accessible, reputable, and peer reviewed.</p> <p>Transmission</p> <p><input type="checkbox"/> The article is sent as a attachment to INTSJNA@AOL.COM</p> <p><input type="checkbox"/> The file name is correctly formatted (e.g. PedsPain_Smyth_GU_Pearson_5.19.09)</p> <p><input type="checkbox"/> It is submitted by the mentor with cc to the student author</p> <p><input type="checkbox"/> The words "Submission to Student Journal" are in the subject heading.</p>
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