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TOPICS IN THIS ISSUE

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Front Cover:
Midwestern University faculty member Rodney Fisher, PhD, CRNA instructs graduate nurse anesthesia students on epidural technique during a simulation lab utilizing lumbar back task trainers. Pictured from left to right are Ryan Boynton, BSN, RN, Krysten Dillsworth, BSN, RN, Gwladys Best, BSN, RN, Rodney Fisher, PhD, CRNA, and Kelli Gleason, BSN, RN.

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Charcot-Marie-Tooth Disease

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Keywords: Charcot-Marie-Tooth, muscular dystrophy, bispectral index monitoring, myelin, heavy sedation

Charcot-Marie-Tooth (CMT) disease, a form of muscular dystrophy, is one of the most common inherited neurological disorders.[1] Charcot-Marie-Tooth is a rare, non-treatable, hereditary motor and sensory neuropathy.[2] There are multiple subtypes of CMT with varying effects on the neuromuscular functions of the body.[1,2] Under normal un-diseased processes, peripheral nerves are wrapped in myelinating Schwann cells that conduct neurological impulses to the periphery.[2] When these myelin sheaths are damaged, transmission of neurologic signals are either delayed or completely interrupted.[2] This disruption in signal neurotransmission leads to muscle wasting and weakness.[2]

Case Report

A 40-year-old, 100 kg, 185 cm male patient presented for incision and debridement, and hardware removal from infected right first and second toes. The hardware had been placed one month prior as part of a hammer toe correction procedure. The patient had undergone multiple procedures, over the past 18 months, in attempt to straighten the interphalangeal and metatarsophalangeal joints of both feet in hopes that the patient would be able to wear shoes once again and improve overall quality of life.

The patient's mobility was limited to a wheel chair due to CMT muscular dystrophy. His health history was significant for depression, gastric esophageal reflux disease, osteomyelitis with abscess, joint contractures, >20 year, 1 pack per day tobacco smoking history, and prior hospitalization with prolonged ventilation of 3 days following laparoscopic cholecystectomy. Surgical history included left ankle incision and debridement, bilateral feet interphalangeal and metatarsophalangeal joint procedures with metal implantation of hardware, and left ankle fusion. Current home medications included: amitriptyline, ibuprofen, omeprazole, sertraline, veranicline. Known drug allergies included ondansetron and zonisemide. Current laboratory values were reported to be within normal limits with the exception of a mild decrease in creatinine at 0.4 mg/dL.

Pre-oxygenation was administered with O_2 10 L/min via simple face mask with end-tidal CO_2 ($EtCO_2$) detection sampling. Intraoperative antibiotics were held due to infection culture sampling. Induction and maintenance of heavy sedation were performed with a propofol infusion titrated between 100 – 200 mcg/kg/min. Fentanyl 100 mcg was titrated in 2 doses of 50 mcg during the first 10 minutes of the procedure. Hydromorphone 2 mg was gradually titrated in throughout the remaining 25 minutes of procedure. The propofol infusion was discontinued at the beginning of wound dressing. By the time of final dressing placement and tourniquet deflation the patient was able to fully follow commands and denied any sensation of pain.

Standard monitoring was utilized with vital signs remaining stable throughout the case. Mean arterial pressure (MAP) was maintained between 60-75 mm Hg, heart rate ranged from 64-93/min, respiratory rate was noted at 16/min pre-induction and 12-14/min throughout the case. The patient's SpO_2 remained >97%, and $EtCO_2$ detection was present, as evidence of spontaneous ventilation, throughout the case. Bispectral (BIS) index monitoring (Medtronic Covidien, Minneapolis, MN) was utilized with readings ranging from 50-70%.

A tourniquet was placed on the right upper thigh and inflated at 250 mm Hg for surgical hemostasis. The infected areas were opened, culture samples were taken with sterile curettes, effected tissue were irrigated, and hardware removed. The wounds were then packed with sterile gauze and wounds dressed and secured. The tourniquet was then deflated after 53 minutes and antibiotics were started post-operatively of sulfamethoxazole 800mg/trimethoprim 160 mg by mouth every 12 hours.

Discussion

Charcot-Marie-Tooth disease is caused by genetic defects in myelinating Schwann cells which wrap around the neural axons of the peripheral nervous system (PNS).[2] Overexpression leads to the accumulation of mutated proteins which results in demyelination.[2] The existing Schwann cells fail to sustain axonal support which results in progressive axonal and neuronal loss which causes neurogenic muscle atrophy and weakness.[2] Slowed conduction of sensory and motor nerve impulses is believed to be the cause of the muscle weakness and numbness experienced by patients with CMT.[2]

Anesthesia administration for orthopedic procedures to correct deformed joints, improve overall balance, or to repair fall related fractures is common.[3] Special anesthetic considerations must be made to avoid toxicity or exacerbation of neuropathy due to the use of neuromuscular blocking agents.[3] The use of succinylcholine is contraindicated with CMT due to the greater risk of hyperkalemia. Increased paralytic sensitivity occurs with all non-depolarizing neuromuscular blocking agents, with the exceptions of atracurium and mivacurium.[4] Though quite rare with CMT, respiratory muscle weakness can be a serious complication when experienced. Incidence of prolonged ventilation or need for reintubation has been noted.[3]

Bispectral index monitoring is the first quantitative electroencephalogram (EEG) index used in clinical practice as a monitor to assess the depth of anesthesia.[5] BIS monitoring has the potential advantages of monitoring the depth of anesthesia, thus reducing the amount of anesthetic agent required to achieve the desired anesthetic depth, more rapid emergence from anesthesia, as well as a decrease in phase II nausea and vomiting.[6] BIS monitoring serves to guide the titration of anesthesia along with other hemodynamic parameters such as blood pressure, pulse and respiratory rate.[5] BIS monitoring protocol (maintaining BIS values between 40 and 60%) has been reported to decrease intraoperative awareness.[7] A BIS value below 60% is associated with a low probability of response to commands as well as decreased incidence of intraoperative recall.[5]

Taking into consideration the anesthetic implications for CMT, the decision to avoid intubation and neuromuscular blocking agents, and elected to maintain spontaneous ventilation. The patient's previous history of prolonged intubation and ventilation, though the specific indication

for this episode of respiratory support was not clear, was taken in to consideration when formulating an anesthetic plan. By administering heavy sedation in conjunction with BIS monitoring, it was anticipated that the patient would have an optimized outcome and anesthetic emergence. Overall, the anesthetic plan proved beneficial to the patient, as he maintained stable vital signs, preserved autoregulation of respiratory function, and experienced prompt emergence at which time the patient was able to follow all commands and denied pain.

References

1. Braathen GJ, Sand JC, Lobato A, Hoyer H, Russell MB. Genetic epidemiology of Charcot-Marie-Tooth in the general population. *Eur J Neurol*. 2011;18(1):39-48. doi: 10.1111/j.1468-1331.2010.03037.x.
2. Ekins S, Litterman NK, Arnold RJ, et al. A brief review of recent Charcot-Marie-Tooth research and priorities. *F1000Res*. 2015;4(53). doi: 10.12688/f1000research.6160.1
3. Vinci P, Lapi G. Anesthetic management in Charcot-Marie-Tooth disease type 2 due to a mutation in the mitofusin-2 gene. *J Anaesthesiol Clin Pharmacol*. 2011;27(2):286-287. doi: 10.4103/0970-9185.81845.
4. Nagelhout JJ. Neuromuscular blocking agents, reversal agents, and their monitoring. In Nagelhout JJ, Elisha S, eds. *Nurse Anesthesia*. 6th ed. St Louis:Saunders Elsevier; 2018:140-164.
5. Health quality Ontario. Bispectral index monitor: An evidence-based analysis. *Ont Health Technol Assess Ser*. 2004;4(9):1-70. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3387745/
6. Marley RA, Clapp TJ. Outpatient anesthesia. In Nagelhout JJ, Elisha S, eds. *Nurse Anesthesia*. 6th ed. St Louis:Saunders Elsevier; 2018:889-903.
7. Diehl MR, Sanders KK. Anesthesia complications. In Nagelhout JJ, Elisha S, eds. *Nurse Anesthesia*. 6th ed. St Louis:Saunders Elsevier; 2018:1216-1235.

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Cardiac Rhythm Management Devices and Electrocautery

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Keywords: electrocautery, pacemaker, automatic implantable cardioverter-defibrillator (AICD)

Pacemakers and automatic implantable cardioverter-defibrillators (AICD) are commonly encountered in clinical practice. The operating room presents situations where electrical interference may affect proper function of these devices. Information found in current literature can be confusing and contradictory.[1] Electromagnetic interference during surgical procedures, particularly from the use of monopolar cautery, is a cause of major concern for patients with cardiac rhythm management devices. Several adverse responses to electromagnetic interference include failure to pace, inappropriate pacing, inappropriate anti-tachycardia therapies,

unintentional device reprogramming, lead-tissue interface damage, and damage to the device circuitry.[2]

Case Report

A 70-year-old, 82 kg, 165 cm female presented for elective L2-S1 transforaminal lumbar interbody and sacroiliac joint fusion. The patient's medical history included anemia, transient ischemic attack, stage III hypertension, and hypertension-induced chronic kidney disease. Surgical history included appendectomy and bilateral carpal tunnel release. Preoperative hemoglobin and hematocrit were 12.7 g/dL and 38.5% respectively. Preoperative blood pressure was recorded at 143/85mm Hg. The patient was noted to have an implanted pacemaker/AICD upon day of surgery examination, but was unable to provide an indication or documentation regarding the device. A postoperative cardiology consultation revealed that the device was placed due to sick sinus syndrome. An echocardiogram completed three weeks prior to surgery showed an ejection fraction of 55-65%, and moderate mitral valve regurgitation. Perioperative electrocardiogram showed an atrial paced rhythm of 70/min.

The patient was transferred to the operating room where general anesthesia was induced with fentanyl 50 mcg, propofol 150 mg, and succinylcholine 100 mg. The trachea was intubated without difficulty, a radial arterial line was placed, and anesthesia was maintained with desflurane 6% inspired concentration in O_2 0.5 L/min and air 0.5 L/min. A doughnut pacemaker magnet was secured to the upper left chest over the implanted pacemaker/AICD. The electrocautery grounding pad was secured to the lateral right thigh. The patient was placed in the prone position and the surgical procedure commenced.

Upon dissection with monopolar electrocautery, the patient became hypotensive with a blood pressure of 68/44 mm Hg and bradycardic with a heart rate of 30-40/min. The surgeon was informed of the hemodynamic instability and asked to discontinue electrocautery. Upon discontinuation, the heart rate returned to an atrial paced rate of 70/min and the blood pressure increased to 129/72 mm Hg. Prior to resuming electrocautery, glycopyrrolate 0.2 mg was administered and a continuous infusion of phenylephrine 40 mcg/min was started. As electrocautery resumed, the heart rate again slowed to 56/min and blood pressure decreased to 87/58 mm Hg with resolution after discontinuation of electrocautery. Hemodynamic instability was noted several times throughout the procedure and supported with titration of a phenylephrine infusion, glycopyrrolate administration, and limiting electrocautery use. The procedure was later converted to a total intravenous anesthetic, as somatosensory evoked potential monitoring was requested by the surgeon. Desflurane was titrated off and a propofol infusion was utilized to maintain anesthesia.

One hour prior to the conclusion of the surgical procedure, the propofol infusion was discontinued and desflurane was restarted for maintenance of anesthesia. At the conclusion of the case, desflurane was discontinued and the patient demonstrated adequate respiratory function but was unable to follow commands. The patient was hemodynamically stable, and was taken to the recovery area intubated, and placed on a T-piece. The pacemaker was interrogated in the recovery room upon arrival by the post anesthesia care unit (PACU) staff, and a report was sent

for evaluation. The patient was extubated one hour after arrival in the PACU and neurological status had returned to baseline function.

Discussion

Ideally, patients with cardiac rhythm management devices (CRMD) will have the device interrogated preoperatively. Consultation with the manufacturer, cardiologist, or electrophysiology service is essential.[3] Information regarding the type of procedure, anatomic location, positioning, use of monopolar electrocautery, and surgical venue should be provided to the consultant.[4] Situations such as urgent or emergent surgery may preclude device interrogation and precautions for perioperative device failure should be made.

Electromagnetic interference (EMI) generated by electrocautery can temporarily inhibit pacemaker output or give rise to a temporary increase in pacing rate.[5] For AICDs, there is a possibility that interference may be misinterpreted as ventricular tachycardia or ventricular fibrillation, causing inappropriate initiation of therapy.

In earth grounded electrosurgical systems, failure of the return electrode connection can result in shunting of current to alternative radiofrequency ground sites, resulting in threshold increase or loss of capture.[4] Optimal grounding of the electrosurgical system involves the use of a split foil return electrode, which allows for detection of proper application to the patient.[4] Failure of the return electrode could be considered as a causative factor in this case. Potential threshold increase or loss of capture may have resulted in sinus bradycardia as capture was lost upon use of monopolar electrocautery. However, a split foil return electrode was utilized in this case. No indication of grounding system malfunction was noted, and the electrocautery functioned properly throughout the procedure.

Clinical magnets can expedite care of patients with CRMDs. Although CRMDs vary in their responses to magnets, some generalizations can be made. All pacemakers change to asynchronous pacing mode with magnet application, and revert to the original programming when the magnet is removed.[1] No change in the pacing mode occurs in AICDs with magnet application. The general response of AICDs to magnet application is suspension of all anti-tachycardia therapies.[1] Disabling or altering function can be accomplished by placing a magnet over the device or by reprogramming the device. The principal disadvantage of reprogramming is that changes that are made with the programmer are not readily reversible.[4] If a patient develops sinus tachycardia or an arrhythmia, asynchronous pacing may have deleterious effects and the AICD will not respond.[4] There is also a risk of failure to re-enable tachycardia therapies following the procedure, leaving the patient unprotected. A study by Boston Scientific showed that of 67,410 remote follow up patients, the most common “red alert” was ventricular fibrillation detection with therapies inactivated.[4]

The principal advantage of magnet use is that it can be quickly removed. For example, if a patient suffers ventricular tachycardia or fibrillation, the magnet can be removed and the tachyarrhythmia will be treated.[4] As previously stated, magnet response can be varied between equipment manufacturers. Postoperatively, it was discovered that the patient in this case had a Biotronik (Biotronik Inc., Lake Oswego, OR) AICD. Biotronik AICDs suspend their anti-

tachycardia therapy while the bradycardia pacing function remains unaffected.[1] There is no audible tone response to magnet application. Application of a magnet was appropriate for this surgical case, as EMI from monopolar cautery was anticipated. Magnet application should not disable pacemaker function with the equipment utilized in this case. It could be considered that the device was programmed to ignore magnet inhibition, however, this function is almost never used in clinical practice.[1] In this case, it can be assumed that the use of monopolar electrocautery resulted in an inhibition of pacemaker output, as the paced rhythm of 70/min changed to sinus bradycardia at a rate of 30-40/min. It should also be considered that poor magnet contact may have contributed to the arrhythmia issues in this case, as the patient was in the prone position.

Recommendations from a joint statement from the Heart Rhythm Society and American Society of Anesthesiologists advise that devices should be interrogated postoperatively. Rationale includes assuring the device has not entered a backup mode, function was not impaired, and restoration of preprocedural programming if changes were made.[4] Patients undergoing monopolar electrosurgery are recommended to have CRMD evaluation within one month of the procedure.[1] These guidelines do not recommend additional periprocedural device interrogation beyond routine management previously described for patients undergoing nerve conduction studies or electromyography. In the presented case, the patient's CRMD was interrogated immediately following surgery in the PACU using remote CRMD evaluation technology. Results of interrogation were not available at the time of publication. In retrospect, a pre-operative pacemaker interrogation and cardiology consultation may have provided additional information regarding the specifics of the AICD.

Successful perioperative management of patients with CRMDs requires a thorough evaluation of the patient's medical history, CRMD indication, and specific details on the implanted device. As technology rapidly advances, there are several variances in CRMD responses to EMI and magnet usage. Having the most current information regarding the responses and nuances of the specific equipment that will be encountered in the case will lead to greater success in incident free management of the CRMD patient.

References

1. Jacob S, Panaich SS, Maheshwari R, Haddad JW, Padanilam BJ, John SK. Clinical applications of magnets on cardiac rhythm management devices. *Europace*. 2011;13:1222-1230. doi:10.1093/europace/eur137.
2. Mahlow WJ, Craft RM, Misulia NL, et al. A Perioperative Management Algorithm for Cardiac Rhythm Management Devices: The PACED-OP Protocol. *Pacing Clin Electrophysiol*. 2013;36:238-248. doi: 10.1111/pace.12049.
3. Gerlich RM, Sweitzer BJ. Overview of Preoperative Assessment and Management. In: Longnecker DE, Newman MF, Zapol WM, Mackey SC, Sandberg WS, eds. *Anesthesiology*. 3rd ed. New York: McGraw-Hill Professional; 2017:35-55.
4. Crossley GH, Poole JE, Rozner MA, et al. The Heart Rhythm Society (HRS)/American Society of Anesthesiologists (ASA) Expert Consensus Statement on the Perioperative Management of Patients with Implantable Defibrillators, Pacemakers, and Arrhythmia Monitors: Facilities and Patient Management. *Heart Rhythm*. 2011;8(7):1114-1154. doi: 10.1016/j.hrthm.2010.12.023.

5. Peter NM, Ribes P, Khooshabeh R. Cardiac Pacemakers and Electrocautery in Ophthalmic Surgery. *Orbit*. 2012;31:408-411. doi: 10.3109/01676830.2012.711888.

Mentor: Vicki Coopmans, PhD, CRNA

Pneumothorax Following Endotracheal Tube Exchange over Bougie Introducer

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Keywords: pneumothorax, bougie, introducer, Eschmann Stylet (Bell Medical Inc., St. Louis, MO), endotracheal tube exchange

The intubating stylet is an adjunct in the management of difficult airways and endotracheal tube exchange. The use of any airway adjunct, however, is not without possible risk. The following case study presents an occurrence of perioperative pneumothorax following endotracheal tube exchange over a bougie introducer. Considerations unique to this case are addressed, including the use of an intubating stylet as an exchange catheter following suspected cuff leak. Indications and insertion techniques for the intubating bougie are also discussed.

Case Report

A 34-year-old male presented for an exploration of spinal fusion and possible removal or reinsertion of lumbar hardware. Radiographs taken for continued pain indicated dislodgement of spinal hardware. The patient was 185cm and 94 kg with no known drug allergies. Two previous lumbar surgeries were performed for recurrent left S1 pain and radiculopathy. These surgeries took place at five and eight weeks respectively prior to the cited day of surgery (DOS). Past medical history was significant for chronic left sided S1 radiculopathy and lumbar pain and posttraumatic stress disorder (PTSD). The patient developed a febrile lower respiratory tract infection immediately following the original surgery eight weeks prior. A subsequent chest radiograph indicated a likely viral pneumonia; however, aspiration pneumonitis was not ruled out. The physical exam on the DOS was unremarkable with normal heart sounds and lungs clear to auscultation bilaterally.

Intravenous induction of general anesthesia was performed and a video laryngoscope was utilized to place an 8.0 mm endotracheal tube (ETT) into the trachea with mild difficulty directing the rigid stylet between the premolars. The ETT was secured at a depth of 24 mm at the lip. Adequate tidal volumes were not achieved with the use of manual ventilation due to an inability to maintain positive pressure. The anesthesia practitioner attempted to add air to the pilot balloon, but was unable to due to a suspected ETT cuff leak. A single use 15 Fr, 70 cm straight tip introducer adult bougie (SunMed Medical, Largo, FL) was then directed into the ETT by the student nurse anesthetist until a change in tactile sensation was experienced. The existing ETT was removed, and a new 8.0 ETT was placed over the bougie introducer. Resistance was met at the larynx and both the ETT and the bougie introducer were removed without further attempt to advance the ETT. The patient was mask ventilated and the video laryngoscope was

then utilized to place a new 8.0 ETT. Upon auscultation for confirmation of tube placement, severely diminished breath sounds were noted throughout the right lung fields. The patient was placed on volume control ventilation and at that time, the patient's arterial oxygen saturation (SpO_2) was 99% on a mixture of 1L/min Oxygen and 1L/min air with peak inspiratory pressures of 14 cm H_20 and tidal volumes ranging from 500-550 ml. A portable chest radiograph showed an appropriately placed ETT 6.2 cm above the carina; no focal consolidation, pleural effusion, or pneumothorax; and near complete resolution of diffuse interstitial opacities seen on the chest radiograph taken eight weeks prior. It was determined between the anesthesia professionals and surgeon that the case could proceed.

Following almost three hours of surgical time, extubation criteria were met and the patient's SaO_2 remained greater than 95% on 6L simple facemask. After one hour in the recovery unit, the patient was unable to be weaned from oxygen and had absent breath sounds throughout the right lung fields. A portable chest radiograph showed complete collapse of the right lung with minimal leftward mediastinal shift. Cardiothoracic surgery was consulted and a chest tube was placed. The pneumothorax resolved and the chest tube was removed without additional incident. A follow up computed tomography (CT) scan was negative for structural airway damage or blebs.

Discussion

The original multi-use gum-elastic bougie (Eshmann Healthcare Tracheal Tube Introducer, SIMS Portex, Hythe, Kent, UK), as well as new single use varieties, such as the Portex introducer (Portex Tracheal Tube Introducer, SIMS Portex) and Frova single–use introducer (Frova Intubating Introducer, Cook (UK) Limited, Letchworth, Hertfordshire, UK), are well known for their use as airway adjuncts during difficult intubation scenarios. Some sources suggest a higher success rate for both first attempt and overall intubation success when they are employed.[1] Although used in over 50% of non-operating room difficult intubation cases by primary responders at a University hospital during a recent retrospective study, the bougie itself was not identified as an independent risk factor for complication rates associated with emergency intubation.[1] However, multiple adverse events, including airway trauma, have been reported with use of the intubating bougie.[2-4]

Two strategies are often utilized for determining proper placement and depth of the intubating bougie: the presence of 'clicks' indicating tracheal rings, and the more clinically sensitive hold up sign.[5] Although both are commonly accepted practice with gum-elastic bougies, newer studies indicate that utilizing the hold up sign, which is felt as increased resistance as the bougie tip enters smaller airways, can generate a significantly increased peak force at the tip of the bougie which may lead to inadvertent airway damage, specifically with the Frovia Intubating Introducer (Cook UK Ltd, Letchworth, UK).[5] Single-use bougies are notable for a more rigid design and pointed tip.[2] Additional research reveals that there is a significant difference in success of tracheal placement between different types and styles of bougie introducers, as well as a significant difference in mean force generated during insertion, with the Portex single-use introducer (Portex Tracheal Tube Introducer, SIMS Portex) creating the greatest force when held at the 10 cm markings. Also of note, forces generated by both single-use bougie introducers are more than double the forces generated by the multi-use bougie introducer.[6]

Single-use bougies are notable for the replacement of braided polyester and resin coating with an outer plastic shell, more rigid design, and pointed tip.[2,6] Nonetheless, the manufacturer and design of an intubating bougie is often unknown by the anesthesia professionals within each facility.[4] Because of decreased costs associated with single-use bougie introducers, they appear to be more prevalent despite higher reports of complications and trauma.[4,7] Previous case reports exist implicating the use of bougie-introducers with unanticipated airway trauma, including bleeding and pneumothorax; while some specify the use of single-use bougie introducers in cases of airway trauma,[2,4] others do not report the style of bougie introducer.[3]

The exact cause of the perioperative pneumothorax in this case remains unknown. A follow up CT scan failed to reveal any mucosal damage, however, several previous case studies also failed to show the source of pneumothorax via imaging following intubation with a bougie introducer.[3] In this case, because the ETT was being exchanged, utilizing tracheal 'clicks' to determine depth was not possible. A single click could indicate the end of the ETT, however this is not guaranteed. The hold up sign was utilized to ensure adequate depth of the bougie-introducer, however because the original ETT was placed under direct visualization, a safer approach would have been to use the depth of the current ETT as a guide for bougie placement. The necessary depth of the bougie should have been calculated, and the proximal end stabilized during ETT exchange. Also, appropriate positive control of the bougie introducer was not maintained at all times. Hand placement changed multiple times as the bougie was placed through the existing ETT and a new ETT was advanced over the introducer. Advancement too far into the airway could not be ruled out. This could have been avoided with proper handling of the bougie introducer and assistance from an operating room nurse or other staff member to maintain positive stabilization of the device.

Lubrication of the bougie introducer is recommended by several sources[2,4], however, when exchanging the new ETT in this case the only resistance encountered was likely when the ETT approached the glottic opening. Direct visualization of the bougie passing through the glottis could have been attempted during both the initial placement or during replacement of the ETT via railroading over the bougie introducer. This may have also may have facilitated the success of the attempt. Similarly, the video laryngoscope could have been utilized for indirect visualization.

In summary, this patient had a documented recent lower respiratory infection eight weeks prior and had several physical characteristics of those who suffer spontaneous pneumothoraces, including age and body habitus.[8] Differential diagnoses of bleb rupture, spontaneous pneumothorax, and airway trauma do exist. Although a definitive cause of pneumothorax was not determined, the association between the single-use bougie introducer and possible airway trauma cannot be overlooked.

References

1. Martin L, Mhyre J, Shanks A, Tremper K, Kheterpal S. 3,423 emergency tracheal intubations at a university hospital: Airway outcomes and complications. *Anesthesiol*ogy. 2011;114(1):42-48.

2. Kumar K, Batra R, Dhir R, Sharma S. Inadvertent pneumothorax caused by intubating bougie. *J Anaesthesiol Clin Pharmacol*. 2015;31(2):271-272.
3. Rajagopal R, Jayaseelan V, George M. A case of pneumothorax following bougie-guided intubation in a patient undergoing excision of an intraventricular space occupying lesion. *J Neuroanaesthesiol Crit Care*. 2017;4:117-119.
4. Staikou C. Airway injury caused by a Portex single-use catheter. *J Clin Anesth*. 2009;21:616-620.
5. Marson BA, Anderson E, Wilkes AR, Hodzovic I. Bougie-related airway trauma: dangers of the hold up sign. *Anaesthesia*. 2014;69:219-223.
6. Hodzovic I, Latto I, Wilkes A, Hall J, Mapleson W. Evaluation of Frova, single-use intubation introducer, in a manikin. Comparison with Eschmann multiple-use introducer and Portex single-use introducer. *Anaesthesia*. 2004;59:811-816.
7. Mushambi M, Ali P, Dyson, L, Malik, D. A national survey of tracheal tube introducers and associated complications. *Anaesthesia*. 2016;71:847-861.
8. Deshpande R, Kurup V. Restrictive respiratory diseases and lung transplantation. In Hines RL, Marschall KE, eds. *Anesthesia and Co-Existing Disease*. 7th ed. Philadelphia:Elsevier; 2018:33-52.

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Adenosine to Treat Ventricular Dysfunction after Cardiopulmonary Bypass

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Keywords: adenosine, contractility, cardiopulmonary bypass, aortic valve, balloon pump

Cardiopulmonary bypass (CPB) is a technique used to create a bloodless field during invasive cardiac surgery. The physiologic stress of this technique on a suboptimal cardiac system can manifest as decreased contractility and ventricular dysfunction.[1] Separating these patients from CPB is often more difficult and requires vigilant monitoring. The additional use of volume replacement, inotropic drugs, vasopressors, and mechanical assistance is also frequently required.[2] Adenosine is not commonly indicated for a hypokinetic heart, yet studies are beginning to clearly identify adenosine receptor's role in coronary blood flow, cardiac contractility, and ischemic pre- and post-conditioning.[3]

Case Report

A 55-year-old male presented to the emergency department with chest pain radiating to his left arm. Past medical history included hyperlipidemia, angina for the past three months, shortness of breath at rest, and dyspnea on exertion. Social history was significant for a 37-pack year smoking history, cocaine and alcohol use. Chest x-ray showed bilateral pleural effusions and interstitial pulmonary edema. An echocardiogram revealed an ejection fraction of 30-35%, severe aortic stenosis (AS), mild mitral regurgitation, pulmonary hypertension, and left ventricular (LV)

hypokinesia. Electrocardiogram (EKG) showed sinus tachycardia with premature ventricular contractions and anterolateral ischemia.

The patient was admitted to the intensive care unit (ICU) and consented for aortic valve replacement. Dobutamine and furosemide drips (gtt) were initiated, and oxygenation supported with oxygen 2 liters via nasal cannula. Blood pressure (BP) was maintained at 95/48 mm Hg, and heart rate (HR) 82/min.

In the operating room, American Society of Anesthesiologists standard monitors were applied and insertion of a right radial arterial line was performed. After adequate preoxygenation, an IV induction was completed with midazolam 5mg, fentanyl 250mcg, propofol 50mg, rocuronium 100mg, and phenylephrine 100mcg. The trachea was intubated and isoflurane was initiated.

A left subclavian central venous catheter was inserted, through which a pulmonary artery catheter was threaded. A transesophageal echocardiography (TEE) probe was placed and cardiac function assessed. Cefazolin 2g and tranexamic acid 850mg were given. Bypass was initiated, and the native aortic valve was replaced with a mechanical valve without complication.

Separation from CBP was unsuccessful due to ventricular dysrhythmia and hypotension. The patient remained dysrhythmic and hypotensive despite the initiation of an Epinephrine gtt at 20 mcg/min and lidocaine 100 mg IV push. Defibrillation was attempted twice with no improvement and patient was transitioned back onto bypass.

The decision was made to perform a left anterior descending coronary artery bypass graft (CABG) utilizing the right femoral vein. A dobutamine infusion at 20 mcg/kg/min and a vasopressin infusion at 6 units/hr were initiated. A second attempt was made to bring the patient off bypass but he remained dysrhythmic and hypotensive. Boluses of phenylephrine 100 mcg, amiodarone 150 mg, and lidocaine 100 mg were given. TEE assessment showed continued LV hypokinesia. Defibrillation was performed a third time with no improvement.

The patient was transitioned back onto bypass and an intra-aortic balloon pump (IABP) was placed. A third attempt was made to transition the patient off bypass. He was now in sinus rhythm yet remained severely hypotensive. The TEE continued to show LV hypokinesia.

Adenosine 6 mg was given per surgeon request. The BP increased from 70/30 mm Hg on balloon pump to 145/90 mm Hg and LV function showed improvement on TEE. The balloon pump was titrated off; after 10 minutes the patient maintained a BP of 125/70 mm Hg. Cardiopulmonary bypass was discontinued and the patient was transported to the ICU with the IABP in place. Upon arrival, the dobutamine infusion was at 5 mcg/kg/min, epinephrine infusion at 5 mcg/min, and the vasopressin infusion was off. The patient's BP remained 130/50 mm Hg with a HR of 100/min.

Discussion

Patients at an increased risk for ventricular dysfunction following CPB include those with advanced age, decreased LV systolic and/or diastolic dysfunction, chronic beta-blocker use,

recent heart attack, and end-organ comorbidities.[2] Prolonged bypass and increased surgical complexity are intraoperative risk factors for ventricular dysfunction upon CPB cessation.[2]

Patients with AS maintain normal ventricular function until late in the disease course.[1] A triad of classic AS symptoms come with worsening ventricular function; angina, syncope, and congestion are correlated with a 5-, 3-, and 2-year survival rate respectively.[1] These patients are dependent on atrial contribution to left ventricular end diastolic volume (LVEDV) which can be as much as 40%.[1] This makes them intolerant of dysrhythmias such as atrial fibrillation.[1] Induction of anesthesia is a critical time for these patients, and BP must be tightly controlled.[1]

The use of TEE has become the 'gold standard' for intraoperative assessment in cardiac surgery. It is utilized to assess fluid status and guide volume replacement.[2] If volume and preload are acceptable, TEE can indicate one of four other scenarios: a structural abnormality, a dynamic abnormality, systolic dysfunction or impaired diastolic relaxation, or vasoplegic syndrome.[2]

The presence of ventricular dysfunction may indicate the use of an inotrope such as epinephrine, dopamine, or dobutamine.[1,2] A vasoconstrictor is added when an inotrope alone does not improve hypotension, most often norepinephrine or neosynephrine.[1,2] Additional adjuncts may include milrinone or vasopressin.[2] Persistent instability could be due to a mechanical problem that is surgically correctable.[1] Cardiac stunning may be present if TEE assessment finds no anatomical problem, requiring a period of resting perfusion on bypass.[1]

Vasoplegic syndrome is a type of vasodilatory shock that occurs after CPB.[1] It is defined as hypotension with normal to high cardiac output. Phenylephrine is the first line treatment, adding norepinephrine or vasopressin if resistance to phenylephrine occurs.[1] Though cardiac output is not depressed, a low dose inotrope may help maintain contractility during the course of vasodilatory shock.[1] The causes of vasoplegic syndrome vary widely from preoperative medications (angiotensin-converting enzyme inhibitors, calcium channel blockers, amiodarone, IV heparin) and diabetes, to poor LV function.[1,2]

An IABP may be required with a heart that cannot adequately separate from CPB despite volume resuscitation and pharmacological support.[1,4] The balloon is seated in the aorta where the arch meets the descending limb, distal to the subclavian artery.[1] It provides counter-pulsation by inflating as the aortic valve closes and deflating as systole begins.[1,4] The IABP decreases afterload and improves diastolic blood flow to the heart which increases subendocardial perfusion, reduces myocardial oxygen demand, and increases CO 10-15%.[1,2,4] It requires the LV to have a certain level of function in order to be effective. The use of an IABP in chronic heart failure patients with severely reduced (<35%) EF has not been shown to significantly improve outcomes.[1,4] The IABP, when placed as a prophylactic measure following cardiac surgery, decreased length of hospital stay but had no effect on 7- and 30-day survival rates.[4]

Adenosine is a vasodilator and anti-arrhythmic used clinically to treat supraventricular tachycardias and reduce pulmonary hypertension.[1] Four types of Adenosine Receptors (AR) have been described: A_1AR, A_{2a}AR, A_{2b}AR, and A_3AR.[5] A_1AR is known to have negative chronotropic and dromotropic effects at the sinoatrial and atrioventricular nodal receptors.[5] A_1AR within the ventricular myocardium has been found to have antiadrenergic effects.[5]

Stimulation of $A_{2a}AR$ and $A_{2b}AR$ causes coronary vasodilation and an increase in coronary flow.[5] $A_{2b}AR$ is also responsible for a modest increase in contractility, but shows the lowest affinity for adenosine.[5] $A_{2a}AR$ potentiates the antiadrenergic effects of A_1AR but does not improve contractility directly.[5] A_3AR has shown no direct effects with isolated stimulation.[5]

The A_1 and A_3 receptors are also known to play a role in cardiac preconditioning, a cardioprotective mechanism that prevents further myocardial infarction after exposure to a brief initial ischemia.[6] These cardioprotective effects occur with pathophysiologic events such as myocardial infarction, but can be mimicked with pharmacologic intervention.[6] Activation of A_1AR releases several protein kinases which improve cell survival and regulate mitochondrial function.[6] Cardiac postconditioning is a cardioprotective mechanism that can prevent reperfusion ischemia after a long ischemic period, such as with cardiac surgery.[6] Pre- and post-conditioning utilize the same A_1AR receptors/pathways.[6]

Cardiac surgery requiring CPB has inherent risks that surpass those of less invasive interventions. The benefits of adenosine administration in the case described above warrant further research into the role of adenosine receptors on ventricular function and the dose required for the physiologic goal of improved ventricular contraction. The previously described aortic valve replacement followed the order of optimization closely described in text, yet new interventions were explored. Adenosine administration significantly improved the cardiac function of this patient and could be utilized as an end-line treatment in similar scenarios.

References

1. Contrera M, Patterson M, Cushing M. Anesthesia for cardiac surgery. In: Nagelhout J, Plaus K. *Nurse Anesthesia.* 6th ed. St. Louis, MO. Elsevier, Inc. 2018:479-524.
2. Licker M, Diaper J, Cartier V, et al. Clinical Review: Management of weaning from cardiopulmonary bypass after cardiac surgery. *Ann Card Anaesth.* 2012;15(1):206-23.
3. Mitchell R, Foppiano L, Siegel L, Oakes D. Cardiac surgery. In: Jaffe R, Schmiesing C, Golianu B. *Anesthesiologist's Manual of Surgical Procedures.* 5th ed. Philadelphia, PA. Wolters Kluwer Health. 2014:574-601.
4. Yildirim Y, Pecha S, Kubik M, et al. Efficacy of prophylactic intra-aortic balloon pump therapy in chronic heart failure patients undergoing cardiac surgery. *Artif Organs.* 2014;38(11):967-72.
5. Chandrasekera P, McIntosh V, Cao F, Lasley R. Differential effects of adenosine A 2a and A 2b receptors on cardiac contractility. *Am J Physiol Heart Circ Physiol.* 2010;299(1):H2082-89.
6. Fretwell L, Dickenson J. Role of large-conductance Ca2+-activated K+ channels in adenosine A1 receptor-mediated pharmacological postconditioning in H9c2 cells**.** *Can. J. Physiol. Pharmacol.* 2011;89(1):24–30.

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Anesthesia in the Non-traditional Operating Room

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Keywords: Anesthesia in remote locations, out of the OR Anesthesia care, out of department anesthesia, Non-operating room anesthesia, NORA.

Surgical procedures are performed in traditional operating rooms (OR) and non-traditional ORs outside the OR suite, referred to as non-operating room anesthesia (NORA). Anesthesia practitioners are expected to administer the same quality and safe anesthesia management for surgical procedures performed in both environments. Non-operating room anesthesia areas are located in different hospital departments, and sometimes different buildings. Therefore, anesthesia practitioners may be presented with several logistic challenges related to patient safety that are beyond OR suite safety concerns.[1] Some examples of NORA locations are the electrophysiology laboratory, endoscopy suites, intensive care units (ICU), interventional radiology (IR), cardiology non- invasive procedure rooms, and brachytherapy.

Case Report

A 70-year-old, 74.8 kg, 200.7 cm male with a BMI of 23.7 kg/m^2 presented to the hospital for sudden onset of headache, aphasia, and right-sided weakness with a National Institutes of Health stroke scale score of 23. Pertinent medical history included atrial fibrillation, hypertension, coronary artery disease, liver disease, and a surgical history of cholecystectomy. Home medications included digoxin, oxycodone-acetaminophen, and lisinopril. Since his history included atrial fibrillation and mitral valve replacement, he took warfarin daily, and was not a candidate for intravenous tissue plasminogen activator (tPA) as his INR was 2.5. Computed tomography angiography showed a left internal carotid artery occlusion extending to the main stem of the middle cerebral artery. He presented to IR for emergent thrombectomy and revascularization.

The patient was brought to the IR suite accompanied by an Emergency Room nurse and resident. The surgeon requested that the mean arterial pressure (MAP) and systolic blood pressure (SBP) remain above 85 mm Hg and 150-160 mm Hg, respectively, until revascularization. An airway assessment revealed right sided facial drooping, which caused an inability to widely open the mouth; therefore, a Mallampati score was not assessed. Further inspection showed an oral aperture of 4cm, thyromental distance of 6 cm, full neck range of motion, and poor dentition including a chipped incisor. Neurological assessment deteriorated prior to anesthesia and surgical procedures. Pre-oxygenation with O_2 10 L/min was initiated along with simultaneous placement of standard monitors.

A rapid sequence induction was performed with propofol 30 mg and remifentanil 300 mcg. Using a Macintosh 3 blade, the trachea was intubated with a 7.5 mm endotracheal tube under direct laryngoscopy while maintaining cricoid pressure. A left radial arterial line was placed after induction. In response to induction, the BP decreased to 100/60 mm Hg. Phenylephrine 240 mcg was administered, and a phenylephrine infusion was initiated at 50 mcg/min and titrated to

surgeon's requested parameters for MAP and SBP. A total dose of 360 mcg of phenylephrine was administered in incremental boluses. General anesthesia was maintained with isoflurane 0.5% expired concentration in air 1 L/min and O_2 1 L/min, rocuronium 50 mg, and a remifentanil infusion titrated between 0.08 – 0.1mcg/kg/min. After revascularization, SBP was maintained at 130 mm Hg.

The surgical procedure lasted 72 minutes. The patient's trachea remained intubated after the procedure and the remifentanil infusion was maintained during transport to the ICU with standard monitors. The remifentanil infusion was discontinued while a propofol infusion was initiated at 30 mcg/kg/min upon arrival to the ICU. The patient's trachea was extubated on postoperative day (POD) 1. The patient was transferred from the ICU to the medical floor on POD 3. The patient demonstrated minimal right sided weakness and was scheduled for physical, occupational, and speech therapy activities.

Discussion

Anesthesia practitioners are expected to provide care in traditional OR suites as well as in non-traditional OR's referred to as NORA.[2] Often the comfort and familiarity of the environment and resources of the traditional OR may not be present in NORA locations, and could affect anesthesia care.[1,3] Anesthesia practitioners work in NORA areas with personnel who may not be aware of the support needed to deliver anesthesia care; therefore, the anesthesia practitioner must have knowledge of the logistics of NORA areas, paricularly during unexpected events. Additional challenges that anesthesia practitioners may encounter include differences in equipment, distance from the OR suites, and difficulty in procuring needed pharmacological agents.[4] Effective communication between anesthesia practitioners and other health care staff is imperative when working in NORA locations. This will help ensure that in times of urgency, needed equipment, pharmacological agents, or additional personnel will be readily available.

The environment of NORA areas may also pose health hazards to the anesthesia practitioners. The limited space for them to work and use of radiation may cause injury.[3] Knowledge of the location of personal protective equipment (PPE) and protective devices is imperative. Unfamiliar electrical or equipment cords may cause a tripping hazard. Therefore, familiarity with NORA areas for each facility will help ensure proper preparation and communication to not only achieve patient safety, but also the safety of anesthesia practitioners providing anesthesia in these areas.[5]

Anesthesia practitioners continuously train and receive education on facility specific logistics. This is to ensure that anesthesia practitioners familiarize themselves and satisfactorily prepare for the surgical procedures in NORA locations. For this case, the anesthesia practitioners were well trained and familiar with providing anesthesia care in IR. Standard monitors, pharmacological interventions, and additional personnel were readily available to assist, particularly when the patient's neurological status deteriorated. The surgical team and anesthesia practitioners communicated well on the objective outcomes of the procedure such as the threshold for blood pressure. The care for the patient was tailored according to his medical history and current co-morbidities. The anesthesia practitioners had adequate PPE and were shielded from radiation. The combination of these factors contributed to safe anesthesia care and was instrumental in ensuring that the patient received quality care that enhanced chances for a quick recovery.

References

1. Chang B, Urman RD. Non-operating room anesthesia:the principles of patient assessment and preparation. *Anesthesiol Clin*. 2016:34;223-240.
2. Kolker A, Mascarenhas J. Anesthesia for outfield procedures in cancer patients. *Curr Opin Anaesthesiol*. 2015:28;464-468.
3. Landrigan-Ossar M, McClain CD. Anesthesia for interventional radiology. *Pediatric Anesthesia.* 2014:28:439-440.
4. Ferrari, LR. Anesthesia outside the operating room. *Curr Opin Anaesthesiol*, 2014:28;439-440
5. Nagrebetsky A, Gabriel RA, Dutton RP, Urman RD. Growth of nonoperating room anesthesia care in the united states: A contemporary Trends Analysis. *Anesth Analg*. 2017:124;1261-1267

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Anesthetic Management of Placenta Accreta

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Key word: cesarean section, hysterectomy, placenta accreta, placenta previa, risk factors

Placenta accreta is defined as an abnormal adherence of the placenta to the uterine wall.[1] Its most common risk factors are previous cesarean section (CS), placenta previa, or any previous uterine curettage surgeries.[1,2] With the rate of cesarean section increasing, (3 per 1000 deliveries compared to 1 per 10,000 deliveries in the 1960s),[1-3] the incidence is only expected to grow.[1] Management of placenta accreta is centered upon early diagnosis and adequate preparation to treat associated hemorrhage, coagulopathy and shock.[1] It is imperative that anesthesia practitioners are aware and understand the consequences of this obstetrical emergency.[1]

Case Report

A 34-year-old, gravida 5, para 3, 80 kg, Hispanic female was scheduled for a CS with possible hysterectomy for complete placenta previa and possible accreta. Pertinent medical history was significant for uterine fibroids, gestational thrombocytopenia and anxiety. The patient had two previous cesarean sections. Preoperative hemoglobin and hematocrit (H&H) values were 11.7 g/dl and 33.3% and platelet count of 125,000/μL. She was premedicated with oral citric-Na citrate 334-500 mg/5 mL 30 mL and metoclopramide 10 mg intravenous (IV) prior to arrival to the operating room (OR). A 500 mL bolus of lactated ringer's (LR) solution was initiated through a left forearm 18-gauge peripheral IV catheter as she was being transported to the OR.

In the OR, O_2 6 L/min was administered via simple face mask and noninvasive monitors were applied. She was placed in a sitting position for combined spinal-epidural (CSE) administration

using sterile technique. Subcutaneous infiltration of 1% lidocaine was performed at the L2-L3 intervertebral space. Using a 17-gauge Tuohy needle into the intervertebral space and following loss of resistance, a 26-gauge X 124 mm Gertie Marx® spinal needle was advanced slowly through the Touhy needle until free flow of cerebrospinal fluid was confirmed. Bupivacaine 0.75% (1.7 mL) was injected through the 26-gauge needle. The spinal needle was removed after injection and an epidural catheter was advanced through the Touhy needle into the epidural space. The Tuohy needle was removed, and the catheter secured in place. The patient was assisted to a supine position with left uterine displacement. Once a T4-T5 sensory block was achieved, the surgical team was advised to start the surgical delivery. The blood bank was alerted of immediate need for blood products in the OR.

Upon delivery of the neonate, oxytocin 40 units in one liter LR was titrated to adequate uterine tone. The surgical team determined the patient had placenta accreta and a hysterectomy was necessary. The patient was preoxygenated with O_2 10 L/min, and general anesthesia initiated with a rapid sequence induction using propofol 130 mg and succinylcholine 100 mg. Following fasciculations, the trachea was intubated under direct laryngoscopy with a 7.0-cuffed oral endotracheal tube (ETT). Placement of the ETT was confirmed with positive chest rise, breath sounds and end-tidal CO_2. The ETT was secured and mechanical ventilation was initiated. General anesthesia was maintained with sevoflurane 2.1% inspired concentration in a mixture of O_2 and air at 1 L/min and 1 L/min respectively. An arterial line was placed in the right radial artery. Two additional large bore peripheral IVs were established for fluid and blood products replacement.

Fentanyl, duramorph (epidural) and rocuronium were administered intermittently for pain management and muscle relaxation. Ephedrine and phenylephrine were also used for blood pressure management. Dexamethasone 10 mg and ondansetron 8 mg were used for antiemetics. Intraoperative lab values were H&H of 7.8g/dl and 23.5%. Two units of packed red blood cells (PRBC) and a unit fresh frozen plasma (FFP) were transfused using the level-1® rapid infuser. A total of 3250 mL crystalloids, 750 mL 5% albumin, 4 units PRBCs, 4 units FFPs and 2 units of platelets were administered. Urine output was 1950 mL. Estimated blood loss was 2500 mL. The case duration was 8 hours and 35 minutes.

Upon completion of the case, the neuromuscular blockade was antagonized with sugammadex 400 mg IV. Once extubation criteria were met, the trachea was extubated, and 6 L/min oxygen was administered. The patient was transferred to the post anesthesia care unit in stable condition. She was spontaneously breathing without any distress. Vital signs were stable. She denied any pain or nausea. Immediate postoperative H&H was 10.1g/dl and 29.9%. Postoperative day one revealed no anesthetic complications and the H&H was 9.6g/dl and 27.6%.

Discussion

The spectrum of abnormal implantation include placenta accreta, placenta increta, and placenta percreta.[1-3] Placenta accreta refers to an abnormal adherence of the placenta to the uterine wall.[1] When the invasion of the placenta extends into the myometrium, it is defined as increta and an invasion of placenta villi through the myometrium into the serosa is known as percreta.[1,3,4] The presence of any of these abnormal implantations makes an attempt to separate the placenta following delivery of the neonate difficult and often accompanied by major hemorrhage.

Although the extent of implantation differs amongst these abnormal placenta presentations, the management is similar.[1,5] The patient in this case study presented with placenta accreta.

The risk factors associated with placenta accreta include a history of multiple cesarean sections, placenta previa, advanced maternal age, multiple pregnancies or any past procedure that might have compromised the integrity of the uterus such as myomectomy or curettage.[1,3,4,6] Kong et al. and Humphrey however, report a strong correlation between previous CS, placenta previa, previous uterine curettage and placenta accreta.[3,5] Silver et al.[4] reported a 60 fold increase in the rate of CS since the 1970s (1 in 30,000 pregnancies to 1 in 533).[4] Notably, an increase in the rate of abnormal placenta implantation has followed.[1,3,4] Recognizing patients with risk factors, coordinating appropriate personnel, and optimizing timing of delivery between 34–37 weeks of gestation, are crucial to the survival of the mother and neonate.[1,3,4,6]

Kong et al.[3] conducted a retrospective study that involved 29,220 parturients during a 3-year period with goals of analyzing the risk factors and diagnosis of placenta accreta.[3] The study revealed 318 of the 14,529 patients who underwent CS had placenta previa, an incidence of 10.9 per 1000 births.[3] Of the 47 who met a diagnosis for placenta accreta, 91% had prior uterine curettage, 70% with history of multiple CS, 72% with placenta previa while 60% had a combination of placenta previa and CS.[3] This supports a strong correlation between these risk factors and placenta accreta. As seen in the preoperative history of 2 prior CS and placenta previa, this patient was at increased risk of developing placenta accreta.

In another study, Lilker et al.[6] analyzed the anesthetic management used in 23 patients with placenta accreta. The anesthetic management for CS included regional anesthesia or regional with general anesthesia. 26% of the patients had a planned combined epidural and general anesthesia while the other 74% had only regional anesthesia (15 epidurals and 2 CSE). Of the 17 patients with regional anesthesia, a total of 5 cases were later converted to general anesthesia due to excessive intraoperative blood loss (>2 liters) in four patients and inadequate anesthesia in one patient.[6] The average EBL for all cases in this study was 1.5 liters. Uterine artery embolization was also used in this study to control excessive blood loss and a possibility of preserving the uterus.[6] The anesthetic management in this case study began with a CSE for the CS and general anesthesia for the hysterectomy due to anticipated hemodynamic instability. Uterine artery embolization was not performed by the surgical team.

Garmi et al.[7] and Humprey[5] concluded that while there is still no general consensus on a clear advantage of one technique (general or neuraxial) over the other, anesthetic management considerations should include insertion of large-bore venous access to allow rapid infusion of crystalloids and blood products transfusion, availability of high flow rate infusion and suction devices, hemodynamic monitoring capabilities, measures to prevent thromboembolism, padding and positioning to prevent nerve compression, and avoidance and treatment of hypothermia.[5,7]

The complications from placenta accreta are best managed by preparedness through a multidisciplinary team approach that includes a skilled gynecologic surgeon, urologist, anesthesia practitioner, blood bank team and an interventional radiologist should artery catheterization be used.[1,5,6,7] For this case, recommended preparations were made in advance and the CS was performed in the main operating room, where additional resources were immediately

available. Hemodynamic monitoring was accomplished by application of standard ASA monitors and insertion of an arterial line which was used for blood pressure monitoring and obtaining frequent blood samples to guide fluid replacement. Blood products were available in the room and transfused expeditiously using a Level-1 rapid infuser (Smiths Medical, Dublin, OH). The institution does have a massive transfusion protocol, but it was not initiated, although other cases may be advantaged by the protocol if necessary. Normothermia was maintained using Bair Hugger (3M, St. Paul, MN) and warm fluids.

In conclusion, the increase in the rate of cesarean section, placenta previa and other risk factors for placenta accreta, indicates knowledge of the anesthetic management of this obstetrical emergency is important. To provide the appropriate care to these patients, risk factors must be identified, and the surgical and anesthesia teams must communicate to develop a coordinated plan to ensure hemodynamic monitoring, timely administration of blood products, and to perform surgery in a setting with appropriate emergency capabilities to support the health of the mother and neonate.

References

1. Hull AD, Moore TR. Multiple repeat cesareans and the threat of placenta accreta: incidence, diagnosis, management. *Clin Perinatol.* 2011(38):285-296.
2. Kasson BJ. Obstetric anesthesia. In: Nagelhout JJ, Plaus KL. eds. Nurse anesthesia. 5th ed. St. Louis, MO: Elsevier Saunders; 2014:1127-1158.
3. Kong X, Kong Y, Yan J, Hu J, Wang F, Zhang L. On opportunity for emergency cesarean hysterectomy and pregnancy outcomes of patients with placenta accreta. *Medicine,* 2017(96):1-5. http://dx.doi.org/10.1097/MD.0000000000007930.
4. Silver RM, Barbour KD. Placenta accreta spectrum: accreta, increta, percreta. *Obstet Gynecol Clin N Am.* 2015(42):381-402.
5. Humphrey J. Primary cesarean delivery results in emergency hysterectomy due to placenta accreta: A case study. *AANA J*. 2015;83(1):28-34.
6. Lilker SJ, Meyer RA, Downey KN, Macarthur AJ. Anesthetic considerations for placenta accreta. *Intl J Obstet Anes.* 2011 Oct;20(4):288-92. doi: 10.1016/j.ijoa.2011.06.001.
7. Garmi G, Salim R. Epidemiology, Etiology, Diagnosis, and management of placenta accreta. *Obstet Gynecol Int.* 2012(8)*;* 1-7. http://dx.doi.org/10.1155/2012/873929.

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Fluid Management in a Heart Transplant Recipient for Thoracic Surgery

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Keywords: heart transplant, fluid administration, fluid management, thoracic surgery, lung surgery, goal directed therapy, ALI, acute lung injury, one lung ventilation

Heart transplantation remains the only curative treatment for end-stage heart failure,[1] with approximately 3500 Americans undergoing the procedure annually.[2] Since the first orthotopic heart transplant (OHT) performed in 1967, survival rates for heart transplant recipients continue to improve.[2,3] Physiological alterations in the transplanted heart result in cardiac output being preload dependent.[2] Therefore, careful consideration must be given to fluid administration in OHT patients undergoing thoracic surgery to maintain adequate cardiac output. However, excessive fluid administration during thoracic surgery is associated with pulmonary complications, notably acute lung injury (ALI), a major source of morbidity and mortality following lung resection.[4,5]

Case Report

A 68-year-old male, weighing 84 kg and measuring 170 cm, presented for a robotic-assisted right middle lung lobectomy secondary to right middle lobe squamous cell carcinoma without evidence of metastasis. His past medical history included hypertension, hyperlipidemia, diabetes mellitus type II and OHT performed 19 years prior due to ischemic cardiomyopathy. The patient's medications included cyclosporine, everolimus, captopril, metformin, rosuvstatin and aspirin. The patient reported taking his anti-rejection medications on the day of surgery.

An electrocardiogram (ECG) performed the week before surgery demonstrated a normal sinus rhythm at a rate of 98/min. A transthoracic echocardiogram performed five months prior demonstrated an ejection fraction of 60-65% with normal systolic function, normal wall motion and trace mitral regurgitation. A stress echocardiogram (performed five months prior) was negative for ischemia and indicated a metabolic equivalent of 6. Additionally, pulmonary function tests revealed a mild obstructive pattern (FEV_1/FVC ratio of 64%). All other lab results were normal.

Anesthetic risks and benefits were discussed and patient consent was granted. The patient was administered midazolam 2 mg intravenous (IV) before transferring to the operating room (OR). Standard monitors were applied including a pulse oximeter, ECG monitoring in leads II and V, noninvasive blood pressure, capnography and a nerve stimulator. Arterial cannulation was performed before induction and hemodynamic parameters were monitored using the FloTrac system (Edwards Lifesciences, Irvine, CA). Since survival rates and life expectancy of OHT recipients continue to improve, an increasing number of these patients present for elective non-cardiac surgery. Management of a patient with a heart transplant includes consideration of the altered physiology of a denervated heart.[2]

General anesthesia was induced with lidocaine 100 mg IV, fentanyl 100 mcg IV, propofol 200 mg IV and succinylcholine 100 mg IV. The trachea was intubated under direct laryngoscopy with a left-sided 39 French double lumen endotracheal tube (DLT). Breath sounds and end-tidal carbon dioxide was confirmed. Dexamethasone 8 mg IV and cefazolin 2 g IV were administered after induction. The patient was positioned in the left lateral decubitus position. Double lumen endotracheal tube placement was confirmed with a fiberoptic scope before and after positioning and one lung ventilation (OLV) was initiated utilizing a tidal volume of 500 mL, a respiratory rate of 14/min, and PEEP 5 cm H_2O. Anesthesia was maintained with sevoflurane 2% inspired concentration in a mixture of O_2 1 L/min and air 1 L/min, and inspired O_2 was titrated for SpO_2 greater than 92%. Neuromuscular blockade was maintained with titrated doses of cisatracurium for a train-of-four count of 1-2 twitches. A Foley catheter was inserted to monitor urine output. The intraoperative course lasted four hours. Plasmalyte totaling 1100 mL and albumin 250 mL were administered. A phenylephrine infusion ranging from 10-25 mcg/min was titrated to maintain a blood pressure within 20% of the patient's preoperative baseline (128/83 mm Hg). The infusion ran for approximately one hour. The blood pressure ranged from 90-140/60-80 mm Hg and heart rate remained stable at approximately 90/minute. Cardiac index ranged from 2.2-2.4 L/min/m^2. Urine output totaled 500 mL, which averaged 125 mL/hour. Ondansetron 4 mg IV was administered for antiemetic therapy at the end of the case. Residual neuromuscular blockade was antagonized with glycopyrrolate 1 mg IV and neostigmine 5 mg IV. A total of hydromorphone 2 mg IV was administered upon emergence. The patient's trachea was extubated without incident. The patient remained stable in the post-anesthesia recovery area and was transferred to the intensive care unit after 90 minutes.

Discussion

The transplanted heart has no sympathetic, parasympathetic or sensory innervation,[2,3] and as a result the graft is unable to increase the heart rate in response to acute hypovolemia or hypotension.[3] Therefore, the transplanted heart responds to stress by increasing stroke volume via circulating catecholamines.[3] Based on the Frank-Starling mechanism, stroke volume is heavily dependent on venous return. Unlike the normal heart, the transplanted heart cannot compensate for acute decreases in preload by utilizing neural stimulation to increase cardiac output. Hence, the transplanted, denervated, heart is heavily dependent on preload.[6] The anesthetic management for OHT recipients includes maintaining preload with adequate fluid administration and avoiding acute vasodilation, hypotension, and hypovolemia.[2,3]

Heart transplant recipients may require fluid boluses throughout surgery to maintain cardiac output; however, excessive fluid administration during thoracic surgery is associated with pulmonary complications, notably ALI.[4] Incidence of ALI following thoracic surgery ranges from 2-7% and is associated with a 25% mortality rate. Although one study has indicated mortality rates as high as 70%.[4] While the etiology of ALI includes pulmonary endothelial damage, oxidative stress, reperfusion injury and lung over-inflation,[4] fluid overload has consistently been linked as a major risk factor. Studies show intraoperative crystalloid and colloid administration exceeding 2000 mL and 1000 mL, respectively, are significant risk factors for developing postoperative ALI.[4,5] Conservative fluid administration has been associated with lower incidence of postoperative mechanical ventilation.[4] Avoiding hypervolemia is the simplest conservative measure to decrease the risk of ALI.[5]

Since heart transplant recipients require higher fluid volumes to maintain adequate preload, the excessive fluid administration associated with thoracic surgery would place this patient at an increased risk for ALI. A total crystalloid volume of 1100 mL was administered for the treatment of hypotension (systolic blood pressure less than 100 mm Hg). A phenylephrine infusion and albumin 250 mL were administered to minimize crystalloid volume. The total amount of intraoperative fluids administered remained below the recommended threshold of crystalloid 2000 mL and colloid 1000 mL for ALI risk reduction. The patient was successfully extubated in the OR and did not experience postoperative pulmonary complications such as ALI.

Central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), pulse pressure variation (PPV) and stroke volume variation (SVV), have historically been indicators to assess fluid responsiveness. The FloTrac system uses stroke volume variation in mechanically ventilated patients to guide fluid therapy by predicting fluid responsiveness,[7] defined as a significant increase in stroke volume in response to fluid administration.[5,8] FloTrac analyzes the arterial blood pressure waveform to monitor a patient's hemodynamic status on a beat-to-beat basis.[7] Goal directed therapy (GDT) involves the monitoring of these hemodynamic parameters and fluid responsiveness to guide fluid administration.[5] Studies show SVV guided fluid management in thoracic surgery requiring OLV does not result in fluid overload[9] and is associated with decreased lactate levels at the end of surgery.[7] By predicting fluid responsiveness, cardiac output is optimally maintained while avoiding excessive fluid administration.[5] Goal directed therapy plays an important role in thoracic surgery due to the unfavorable outcomes associated with fluid overload during lung surgery.[5]

Invasive arterial blood pressure monitoring was performed in this patient due to his cardiac transplant status and the risk of significant fluid shifts. FloTrac and GDT were used to monitor SVV and minimize the risk of fluid overload. The goal was SVV of less than 10%.[7] Lung protective ventilation was utilized to decrease the risk of a stretch induced lung injury, which is another risk factor for ALI.[4] Reduced tidal volumes of 5-6 mL/kg to maintain a peak airway pressure of less than 30 cm H_2O were utilized during OLV. Studies indicate that SVV accurately predicts fluid responsiveness in patients undergoing OLV when tidal volumes are at least 8 mL/kg, but are less sensitive with lower tidal volmes.[8] Additionally, increased intrathoracic pressures affect stroke volume, resulting in increased SVV values.[10] Other factors that may limit the accuracy of SVV interpretation include presence of arrhythmias, PEEP, and the use of vasodilatation therapy.[7] Based on these results, the prediction of fluid responsiveness during this case may not have been completely accurate since lower volumes and PEEP were utilized, and the thoracic cavity was insufflated.

The patient presented for surgery with a significant risk for developing postoperative ALI. Evidence-based practice supports conservative fluid management to decrease this risk. Goal directed therapy using SVV analysis was utilized to optimize cardiac output. Although studies indicate the predictive power of fluid responsiveness is not accurate when tidal volumes are less than 8 mL/kg or when thoracic pressure is increased, SVV monitoring nevertheless appeared to be an effective strategy for this patient. He received adequate fluids to maintain hemodynamic stability, as evidenced by his blood pressure, cardiac output and urine output, and fluid overload was avoided - he was successfully extubated and did not demonstrate clinical signs of

postoperative ALI. Based on his favorable outcome, no changes would be made in the anesthetic management of this patient. Considering the detrimental effects of ALI following thoracic surgery and the unique intraoperative considerations of OHT recipients, anesthesia practitioners should utilize GDT to minimize the risk of ALI in this patient population.

References

1. Kobashigawa J, Khush K, Stehlik J, et al. Report from the American Society of Transplantation Conference on donor heart selection in adult cardiac transplantation in the United States. *Am J Transplant*. 2017;17(10):2559-2566.
2. Valerio R, Durra O, Gold ME. Anesthetic considerations for an adult heart transplant recipient undergoing noncardiac surgery: A case report. *AANA J*. 2014;82(4):293-299.
3. Swami A, Kumar A, Rupal S, Lata S. Anaesthesia for non-cardiac surgery in a cardiac transplant recipient. *Indian J Anaesth*. 2011;55(4):405-407.
4. Eichenbaum KD, Neustein SM. Acute lung injury after thoracic surgery. *J Cardiothorac Vasc Anesth*. 2010;24(4):681-690.
5. Chau EHL, Slinger P. Perioperative fluid management for pulmonary resection surgery and esophagectomy. *Semin Cardiothorac Vasc Anesth*. 2014;18(1):36-44.
6. Contrera MA, Patterson M, Cushing M. Anesthesia for cardiac surgery. In Nagelhout JJ, Plaus KL, eds. *Nurse Anesthesia*. 6th ed. St. Louis:Elsevier; 2018:479-534.
7. Benes J, Chytra I, Altmann P, et al. Intraoperative fluid optimization using stroke volume variation in high risk surgical patients: Results of prospective randomized study. *Crit Care*. 2010;14(3):R118. doi:10.1186/cc9070.
8. Suehiro K, Okutani R. Influence of tidal volume for stroke volume variation to predict fluid responsiveness in patients undergoing one-lung ventilation. *J Anesth*. 2011;25(5):777-780.
9. Haas S, Eichhorn V, Hasbach T, et al. Goal-directed fluid therapy using stroke volume variation does not result in pulmonary fluid overload in thoracic surgery requiring one-lung ventilation. *Crit Care Res Pract*. 2012:1-8. doi 10.1155/2012/687018.
10. Kang WS, Kim SH, Kim SY, Oh CS, Lee SA, Kim JS. The influence of positive end-expiratory pressure on stroke volume variation in patients undergoing cardiac surgery: an observational study. *J Thorac Cardiovasc Surg*. 2014;148(6):3139-3145.

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Anesthetic Management for a Patient with Superior Vena Cava Syndrome

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Keywords: Superior vena cava syndrome, anesthesia, mediastinal mass, mediastinoscopy

Providing anesthesia for patients with a mediastinal mass can result in life-threatening complications. A patient with a mediastinal mass may present with superior vena cava (SVC) syndrome, complete airway obstruction, or cardiopulmonary failure.[1-4] Superior vena cava syndrome is a result of compression of mediastinal structures leading to impairment of venous

drainage from the head, face, and upper extremities.[4] Patients with SVC syndrome often present with dyspnea, tachycardia, and upper body edema.[1-4] General anesthesia can worsen extrinsic airway compression as a result of relaxation of bronchial smooth muscle, presenting multiple challenges for perioperative anesthetic management.[2]

Case Report

A 64-year-old, 54 kg female was scheduled to undergo a mediastinoscopy after presenting with complaints of headaches as well as facial and neck swelling. On admission, the patient was discovered to have a mediastinal mass with compression on the SVC causing SVC syndrome. The patient was a former 20 pack-year smoker with a history of emphysema and coronary artery disease. The patient denied any previous surgical history. A preoperative electrocardiogram showed sinus tachycardia at a rate of 114/min, a compensatory mechanism as a result of decreased preload from compression of the SVC. A chest radiography (CXR) and CT scan revealed a 52 x 59 mm mediastinal mass centered on the right pericarinal region with extrinsic compression upon the SVC and direct contact of the carina. An MRI was not performed on admission due to the difficulty in the patient maintaining the supine position for an extended period of time.

Preoperative labs and vitals were within normal limits with the exception of heart rate, which ranged from 110-130/min. The preoperative airway evaluation revealed a modified mallampati grade II, a normal thyromental distance, full neck range of motion, and minimal right sided neck swelling with jugular venous distention. The patient was able to tolerate lying flat and claimed improvement in breathing after receiving methylprednisolone 40 mg IV every 8 hours for the previous three days. The patient's tachycardia was being treated with metoprolol 75 mg PO daily.

The operating room was prepared with emergency airway equipment, which included a GlideScope (Verathon Inc., Bothell, WA), fiberoptic bronchoscope, and rigid bronchoscope. Within the preoperative holding area, two 18-gauge IV catheters were inserted bilaterally in the upper extremities and a 500 mL lactated ringer bolus was administered to preload the patient prior to induction. The right wrist was localized with 1% lidocaine and an arterial line was successfully inserted into the radial artery. After adequate preoxygenation was evident by an end-tidal O_2 concentration >90%, the patient was induced in a semi-upright position in the presence of the cardiothoracic surgeon. General anesthesia was induced with lidocaine 100 mg, fentanyl 100 mcg, propofol 100 mg, etomidate 10 mg, and succinylcholine 100 mg. The trachea was intubated with a size 7.0 mm endotracheal tube (ETT) via laryngoscope without complication or apparent trauma. After ETT placement and adequate ventilation was confirmed, the patient was subsequently dosed with rocuronium 20 mg IV upon return of four twitches via train-of-four (TOF) monitoring.

General anesthesia was maintained with sevoflurane 3% in O_2 2 L/min on volume controlled mechanical ventilation to maintain an expired concentration of 2.5% . Additional IV medications administered intraoperatively included hydrocortisone 100 mg stress dose, fentanyl 100 mcg and ondansetron 4 mg. Total fluids administered during the case included 500 mL of lactated ringers with an estimated blood loss of less than 50 mL. After successful antagonism of neuromuscular

blockade with 3 mg neostigmine and 0.4 mg glycopyrrolate, the patient was extubated fully awake after achieving appropriate extubation criteria as evidenced by tidal volumes of 5ml/kg and a 5 second head lift. Oxygen 10 L/min was administered via simple face mask and the patient was transferred in a sitting position to the intensive care unit for monitoring. The postoperative course was uneventful.

Discussion

Superior vena cava syndrome results from obstruction of blood flow from the SVC to the right atrium.[1] Induction of general anesthesia in patients with a mediastinal mass and corresponding SVC syndrome requires thorough evaluation and planning to decrease the likelihood of life-threatening complications.[2,4] The majority of poor outcomes appear to arise from tracheal or bronchial obstruction leading to hypoxemia and cardiopulmonary arrest. Additionally, a mediastinal mass has the potential to cause obstruction of the airway and major vessels.[2,4] Inadequate preoperative evaluation and preparation for induction of a patient with SVC syndrome could result in fatal complications.[1-4]

A thorough preoperative evaluation is imperative in assessing the risk of major complication in a patient with SVC syndrome presenting for surgery requiring general anesthesia. This allows the anesthesia personnel to prepare for induction as well as plan for proper optimization of the patient. The clinical presentation of a patient with SVC syndrome includes swelling of the upper airway, head, and upper extremities. Additional symptoms include cough, dyspnea, headache, tachycardia, and chest pain.[1-4]

Preoperative testing that may help guide the anesthetic plan include an electrocardiogram, chest CT scan, and echocardiography (echo). The chest CT scan aides in visualizing the relationship between the tumor and potential airway compression. An echo allows the ability to evaluate the overall impact of vessel compression on hemodynamics.[1-4] In addition, the echo should be performed in various positions because different positions may potentiate airway compression or hypotension. If the patient decompensates in the perioperative period, repositioning the patient should be considered.[4] A mediastinal mass may grow quickly and thus a posterior-anterior and lateral chest x-ray films should be taken within 1-week of surgery.[1] Magnetic resonance imaging may also be considered in order to more closely identify soft tissues versus vascular structures and detect position related compression syndromes.[4]

The use of premedication was avoided in this particular patient to avoid potential respiratory compromise. Due to compression of the SVC, a pre-induction bolus was administered to optimize preload. The literature suggests vascular access should be obtained in the lower extremities below the inferior vena cava to bypass any potential occulusions in the upper vasculature due to compression of the SVC. [4,6] Preoperative steroids were also utilized to assist in decreasing upper airway edema associated with the inflammatory response related to tumor invasion.[1,3,4] Regarding induction agents, those which have a minimal effect on hemodynamics (i.e. etomidate) should be favored.[1] Of note, general anesthesia intensifies extrinsic airway compression by relaxing bronchial smooth muscle. Additionally, neuromuscular blockade and positive pressure ventilation result in a narrowing of large caliber airways due to decreasing transpleural pressure gradients.[4] Therefore, if evaluation of the airway and preoperative imaging

is suggestive of a potential difficult airway then awake fiberoptic should be considered. Allowing the patient to maintain spontaneous respirations is the conservative method of choice to avoid exacerbation of airway compression. However, in patients without symptomatic evidence of airway obstruction or signs of a difficult airway IV induction and intubation may be considered.[2,4]

It is important for the anesthesia provider to prepare for the inability to intubate or ventilate and to have emergency airway equipment readily available in the operating room. In cases where the trachea cannot be intubated past the tumor, rigid bronchoscopy can be implemented by the cardiothoracic surgeon. Therefore, it is recommended to consider having the cardiothoracic surgeon present during the induction process.[2,4] If airway compromise post procedure is of concern, then extubating over an intubating stylet or leaving the patient intubated should be considered.[1]

Intraoperatively, positioning of the mediastinoscope against the right brachiocephalic artery by the surgeon may cause vessel compression, which can lead to a decrease in right extremity pulses. Utilizing an arterial line or pulse oximetry on the right upper extremity should be implemented to detect immediate compression of the innominate artery in order to notify the surgeon. Monitoring of the blood pressure on the left is also advised to obtain accurate blood pressure measurements.[5] If there is indication of unpredictable circulation time in the upper extremities due to compression at the SVC, then IV lines in the upper extremities would be contraindicated and lower extremity peripheral lines would be recommended.[1]

The anesthetic management of this case was successful. However based on best practice, vascular access should have been established in the lower extremities due to the presence of upper airway edema which was suggestive of obstruction of the upper vasculature .[6] Initial management of SVC syndrome caused by malignancy is dependent on the severity of symptoms and primary malignant condition. Currently the research supports accurate histological diagnosis prior to beginning radiation therapy and the use of endovenous stents as emergent treatment in severely symptomatic patients.[2,6] For this particular patient, the surgeon felt a mediastinoscopy was necessary to obtain an accurate histological diagnosis and determine the extent of lymph node involvement. The results of the biopsies would dictate the primary course of treatment (resection versus radiation therapy). Although current literature and guidelines on SVC syndrome are lacking, the consensus remains that awake fiberoptic is the safest option for obtaining control of the airway.[1-4] An awake fiberoptic intubation may have been the optimal choice in this particular case considering the mediastinal mass had direct contact with the carina. Overall, the patient was adequately optimized preoperatively and the emergency airway equipment was readily available if a complication were to occur.

References

1. Chaudhary K, Wadhawan S, Bhadoria P, Gupta A, Jain D. Anesthetic management of superior vena cava syndrome due to anterior mediastinal mass. *J Anaesthesiol Clin Pharmacol*. 2012;28(2):242-246.

2. Rath L, Gullahorn G, Connolly N, Pratt T, Boswell G, Cornelissen C. Anterior Mediastinal Mass Biopsy and Resection: Anesthetic Techniques and Perioperative Concerns. *Semin Cardiothorac Vasc Anesth.* 2012;16(4):235-242.
3. Koetters KT. Superior Vena Cava Syndrome. *J Emerg Nurs*. 2012;38(2):135-138.
4. Blank RS, de Souza DG. Anesthetic management of patients with an anterior mediastinal mass: Continuing Professional Development. *Can J Anaesth*. 2011;58:853-867.
5. Cata J, Lasala J, Mena G, Mehran J. Anesthetic Considerations for Mediastinal Staging Procedures for Lung Cancer. *Semin Cardiothorac Vasc Anesth*. 2018;32(2):893-900.
6. Drews RE, Rabkin DJ. Malignancy-related superior vena cava syndrome. In: Post TW, ed. *UpToDate*. Waltham, MA: UpToDate; 2017.

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Perioperative Management of Postural Orthostatic Tachycardia Syndrome

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Keywords: postural orthostatic tachycardia syndrome, POTS, autonomic dysfunction, autonomic nervous system, tachycardia

Postural orthostatic tachycardia syndrome (POTS) is a dysfunction of the autonomic nervous system (ANS) characterized by tachycardia associated with posture changes, a lack of orthostatic hypotension, and presyncopal symptoms.[1,2] The prevalence of POTS is difficult to estimate as the disorder is widely misdiagnosed and subsequently under-diagnosed.[1,3] Females from adolescence to middle-age are most frequently affected.[2,4] There exists a wide range of symptoms and several potential anesthetic implications concerning a patient with POTS, but optimal anesthetic management has not yet been empirically determined.[2,5]

Case Report

A 20-year-old female, 55 kg and 165 cm, presented for an arthroscopic anterior cruciate ligament (ACL) repair of the right knee. Past medical history included chronic migraines and POTS, and the patient reported no prior surgeries and no known allergies. Home medications included fludrocortisone, ivabradine, midodrine, sertraline, pyridostigmine, ferrous sulfate, magnesium, and oral contraceptives. The patient reported being cleared for surgery by her autonomic neurologist, however no documentation was provided.

Preoperative workup included a comprehensive metabolic panel and magnesium level. All results were within normal limits, and a preoperative electrocardiogram was unremarkable. The patient was instructed to continue all medications the morning of surgery. Preoperative assessment the day of surgery was unremarkable. Vital signs included blood pressure (BP) 130/87 mm Hg, heart rate (HR) 58/min, SpO_2 98% on room air, and respiratory rate (RR) 16/min. An 18 gauge intravenous (IV) catheter was placed in the right hand. Midazolam 2 mg

and ondansetron 4 mg IV were administered. The decision was made to place an arterial line after induction.

Upon transferring to the operating room, standard monitors were applied, and O_2 10 L/min was administered via mask. Induction of anesthesia was achieved with fentanyl 50 mcg, lidocaine 60 mg, and propofol 200 mg IV. A size 4 laryngeal mask airway (LMA) was successfully placed. Sevoflurane was used for maintenance of anesthesia at 3% inspired concentration with a fresh gas flow mixture of O_2 at 1 L/min and air at 1 L/min. Respirations were assisted with a mechanical ventilator on pressure support to maintain end-tidal CO2 between 35 - 45 mm Hg. A left radial arterial line was placed.

Care was taken to maintain hemodynamics within 20% of baseline throughout the perioperative period. An additional dose of fentanyl 50 mcg IV was given prior to injection of local anesthetic by the surgeon. A total of 1200 mL of lactated ringer's solution and a phenylephrine IV infusion at 20 mcg/min was used throughout the intraoperative period to maintain BP while avoiding tachycardia. The patient's systolic BP ranged from 85 - 158 mm Hg with an average reading of 108 mm Hg. The HR ranged from 51 - 103/min, with an average reading of 62/min. An additional dose of ondansetron 4 mg IV was given 30 minutes prior to emergence, and the LMA was removed awake. Estimated blood loss was minimal. Total time from induction to emergence was 104 minutes.

Upon transfer to the post-anesthesia care unit, the patient was drowsy yet arousable to voice with O_2 2 L/min via nasal cannula. Vitals signs included BP 134/81 mm Hg, HR 84/min, SpO2 100%, RR 18/min, and temperature 36.9°C.

Discussion

The prevalence and etiology of POTS is unknown, but its onset may be associated with viral illness, primary autonomic disorders, or secondary to autonomic neuropathies seen in other diseases.[2] Subtypes of the syndrome exist, but considering the significant overlap between the subtypes, classification of such may not be clinically relevant.[5] Common pertinent findings in patients with POTS include exercise intolerance, deconditioning, hemodynamic instability, hypovolemia, and increased circulation of catecholamines.[2,5] Treatment includes lifestyle changes and pharmacotherapy, the goals of which are expansion of blood volume, reduction of venous pooling, stabilization of HR and BP, and improvement of deconditioning.[4]

This patient was on several medications that are commonly used in the treatment of POTS. Fludrocortisone is used to increase sodium and fluid retention, thereby increasing volume status and BP, as well as sensitizing alpha-adrenergic receptors to endogenous catecholamines.[2,3] Desmopressin may be used as an alternative but could promote hyponatremia.[1] Ivabradine is a sinoatrial node blocker used to control HR and may be used alone or in conjunction with beta-blockers.[1,3] Midodrine is an alpha-adrenergic agonist which promotes peripheral vasoconstriction thereby increasing venous return and BP, and it may have additional effect on suppression of tachycardia.[1,3,5] It is thought that a disturbance in serotonin production plays a part in the dysregulation of HR and BP in patients with POTS, and selective serotonin reuptake inhibitors such as sertraline may be used as part of pharmacologic therapy.[2,3] Pyridostigmine is an

acetylcholinesterase inhibitor used to increase neurotransmission and enhance function of the ANS.[4] Chronic use of acetylcholinesterase inhibitors presents unique anesthetic considerations. Due to inhibition of plasma cholinesterase which metabolizes succinylcholine, these patients are at increased risk for prolonged duration of succinylcholine. In addition, reversal of nondepolarizing neuromuscular blocking drugs with an acetylcholinesterase inhibitor may be unsuccessful if inhibition is already maximized by chronic use.[6]

Perioperative management of POTS is not well studied or understood.[2,5] It is recommended that the patient continue all therapeutic medications through the morning of surgery in order to maintain autonomic stability. Intravenous substitutions may be used when the oral route is not permissible.[5] The presence of comorbidities should be assessed preoperatively. Autoimmune and connective tissue disorders appear to predominate although a causal relationship has not yet been established.[1] In a study of 84 patients with a diagnosis of POTS, 8% were found to have mitral valve regurgitation or prolapse.[7] An electrocardiogram was done on this patient preoperatively and was unremarkable, and she reported no other medical comorbidities apart from chronic migraines, a frequent finding in patients with POTS.[3,5]

In addition to continuing home medications the day of surgery in order to maintain autonomic stability, medications that may elicit tachycardia or have anticholinergic effects should be avoided. Examples include ketamine, ephedrine, glycopyrrolate, and desflurane.[5] This patient received minimal intravenous agents in an attempt to avoid interaction with the ANS or tachycardic side effects, and her HR remained near baseline with the exception of a slight tachycardia during emergence. As an induction agent, there are concerns that use of propofol may exacerbate intraoperative hypotension, but considering the adrenergic suppression effects of etomidate and the tachycardia associated with ketamine and thiopental, propofol appeared to be the more prudent choice for this patient.[4,5] It is also recommended to minimize sympathetic response by providing early and adequate pain relief.[5]

Case reports show that prolonged hypotension intraoperatively requiring fluid and vasopressor support is a common occurrence for patients with POTS.[2] Fluid boluses are recommended preoperatively and may be used to treat tachycardia associated with POTS, and alpha-adrenergic receptor agonists are recommended for vasopressor treatment.[5] This patient did not receive a preoperative fluid bolus which may have benefited the patient by reducing the need for support by phenylephrine, an alpha-adrenergic agonist. Invasive hemodynamic monitoring may be considered for even minor surgical procedures requiring general anesthesia.[5] Placement of the radial arterial line in this patient allowed for tight control of BP parameters.

Overall, the management of this case followed the current literature well. All home medications were continued through the day of surgery. Something to consider would be an IV stress dose of steroid used for patients on chronic steroid therapy. Drugs that may induce tachycardia or hypotension were avoided, with the exception of propofol for induction of anesthesia. We were able to avoid neuromuscular blocking drugs which could be affected by the patient's home dose of pyridostigmine. A radial arterial line was used for close hemodynamic monitoring, and a phenylephrine IV infusion was used to maintain BP. After learning that hypovolemia is a common occurrence from POTS, a preoperative fluid bolus may have been beneficial in

maintaining hemodynamics. Overall, this patient remained stable throughout the perioperative period and experienced no complications.

References

1. Pavlik D, Agnew D, Stiles L, Ditoro R. Recognizing postural orthostatic tachycardia syndrome. *JAAPA*. 2016;29(4):17-23. doi:10.1097/01.JAA.0000481398.76099.09
2. Rabbitts JA, Groenewald CB, Jacob AK, Low PA, Curry TB. Postural orthostatic tachycardia syndrome and general anesthesia: a series of 13 cases. *J Clin Anesth*. 2011;23(5):384-392. doi:10.1016/j.jclinane.2010.12.013
3. Busmer L. Postural orthostatic tachycardia syndrome. *Prim Health Care*. 2011;21(9):16-20. doi:10.7748/ns2013.01.27.20.44.c9502
4. Bogle JM, Goodman BP, Barrs DM. Postural orthostatic tachycardia syndrome for the otolaryngologist. *Laryngoscope*. 2017;127(5):1195-1198. doi:10.1002/lary.26269
5. Kernan S, Tobias J. Perioperative care of an adolescent with postural orthostatic tachycardia syndrome. *Saudi J Anaesth*. 2010;4(1):23-27. doi:10.4103/1658-354X.62611
6. Blichfeldt-Lauridsen L, Hansen BD. Anesthesia and myasthenia gravis. *Acta Anaesthesiol Scan*. 2012;56(1):17-22. doi:10.1111/j.1399-6576.2011.02558.x
7. Parsaik AK, Singer W, Allison TG, et al. Orthostatic intolerance without postural tachycardia: how much dysautonomia? *Clin Auton Res*. 2013:23(4):181-188. doi:10.1007/s10286-013-0199-5

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Total Intravenous Anesthesia for Endoscopic Sinus Surgery

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Keywords: endoscopic sinus surgery, total intravenous anesthesia, inhalational anesthesia, surgical field, visualization, blood loss

Endoscopic sinus surgery (ESS) is a minimally invasive surgery for the treatment of chronic sinusitis and other sinus conditions.[1,2] A successful operation relies on adequate surgical visualization through the endoscope and a dry surgical field. Bleeding, even a small amount, can increase the risk for complications due to the difficulty in identifying anatomical structures.[1] Some techniques suggested in the literature to decrease surgical bleeding for an ESS include head elevation, administration of local vasoconstrictors, and controlled hypotension.[2] Additionally, researchers have suggested the use of total intravenous anesthesia (TIVA) versus inhalational anesthesia (IA) to reduce bleeding and to improve surgical field visualization.

Case Report

A 35-year old female patient (67 kg, 170 cm) presented for an ESS for recurrent sinusitis. Despite two sinus surgeries and three sinus debridements, she continued to experience sinusitis

requiring antibiotics. The patient had a history of asthma that was well-controlled with daily mometasone inhaler and denied any history of emergency or urgent care admission for asthma related issues. The patient's laboratory values were within normal limits.

Preoperative vital signs included blood pressure (BP) 112/67 mm Hg, heart rate (HR) 72/min and a SpO_2 of 98% on room air. The patient described severe postoperative nausea after her previous surgeries; therefore, a scopolamine 1.5 mg transdermal patch was placed behind the patient's left ear. She received midazolam 2 mg intravenous (IV) prior to entering the operating room. In the operating room, non-invasive blood pressure cuff, 5-lead electrocardiogram, and pulse oximeter monitors were placed. Oxygen 10 L/min was delivered via the anesthesia mask for 5 minutes. Vital signs were reassessed and an IV induction of anesthesia was initiated with fentanyl 50 mcg, lidocaine 50 mg, propofol 150 mg and rocuronium 50 mg. Direct laryngoscopy was performed with a Macintosh blade size 3 and a 7.5 mm internal diameter oral RAE endotracheal tube was inserted and confirmed via end-tidal CO_2 and auscultation of bilateral lung sounds. The patient was placed on mechanical ventilation with O_2 1 L/min and air 1 L/min and the following settings: tidal volume 500 mL, respiratory rate 10/min, and positive end-expiratory pressure 5 cm H_2O. As the primary anesthetic, a continuous propofol infusion was initiated at 150 mcg/kg/min in combination with a remifentanil infusion at 0.1 mcg/kg/min.

Vancomycin 1 g IV was administered over 60 minutes, and dexamethasone 10 mg IV for antiemetic prophylaxis. The head of the bed was elevated to 15 degrees. For the remainder of the case, rocuronium 30 mg IV was administered in 10 mg aliquots to maintain a train-of-four count (TOFC) of 1-2/4 twitches. Throughout the procedure the propofol and remifentanil infusions remained constant as the patient's mean arterial pressure was maintained between 60-70 mm Hg and her HR remained stable at 50-60/min. At the conclusion of the surgery, the TOFC was assessed and the remaining neuromuscular blockade was antagonized with sugammadex 140 mg IV. The peripheral nerve stimulator showed a TOFC of 4/4 twitches, absence of fade, and sustained tetany of 5 seconds. Ondansetron 4 mg IV was administered and the propofol and remifentanil infusions were discontinued. Spontaneous respirations were observed with tidal volumes greater than 400 mL and fentanyl 50 mcg IV was titrated to a respiratory rate of 10 to 12/min. The patient was extubated fully awake with no respiratory difficulty and transported to the post-anesthesia recovery room on room air. The patient received 1,500 mL of 0.9% sodium chloride and her estimated blood loss was 50 mL. The surgeon verbalized no complaints regarding excessive bleeding or poor visualization. The case duration was 2.5 hours. The patient denied postoperative pain, nausea, or vomiting and was discharged home within 2 hours.

Discussion

Controlled hypotension is currently employed to decrease bleeding during an ESS. Controlled hypotension is achieved by reducing systemic vascular resistance (SVR) or cardiac output (CO). This reduction in BP is accomplished with the use of vasodilators such as sodium nitroglycerin or with the use of beta-blockers such as esmolol.[1,2] Unfortunately, controlled hypotension by reducing SVR is not very effective at lowering blood loss and improving surgical field visualization. This is due to a reflex tachycardia caused by these vasodilators and the local dilation of mucosal vessels.[1,2] Beta-blockers appear to be more advantageous as they lower the

BP via a decrease in the CO, as opposed to the SVR although these are not often administered for ESS.[1,2]

Anesthetic agents have also been utilized to reduce both SVR and CO. Inhaled anesthetics increase cerebral blood flow and vasodilate the ethmoid arteries augmenting the risk of bleeding.[2,3] Propofol, an intravenous anesthetic commonly used for TIVA, benefits patients by decreasing cerebral perfusion pressure, which inadvertently lowers pressure at the nasal cavity of the anterior and posterior ethmoid arteries thought to reduce bleeding. [2-4] The result of improved visualization and the potential for decreased bleeding has led to an increase interest in TIVA for ESS by surgeons.[1,5] For these reasons, propofol was chosen as the primary agent on this case.

A study by Ahn et al.[6] compared the amount of blood loss and the surgeon's visibility rating when utilizing target-controlled infusion of propofol to sevoflurane. Forty patients were studied and the surgical lesion of each patient was classified based on the Lund-Mackay (LM) scale as high (>12) or low (≤12). Both the propofol and sevoflurane group received continuous remifentanil infusion at 0.2 mcg/kg/min. The surgeon scores were obtained at the conclusion of the procedure. Ahn et al.[6] found that in patients with high LM scores, the administration of propofol resulted in less blood loss and superior visualization scores. The patients with low LM scores demonstrated no differences in blood loss and visualization scores between the two groups. Patients with high lesions (LM >12) appeared to benefit most with TIVA over IA.

A study by Yoo et al.[4] looked at 60 patients undergoing ESS and showed no differences in surgical field visibility between three types of anesthesia techniques: propofol and remifentanil, sevoflurane and remifentanil, desflurane and remifentanil. However, compared to the study by Ahn et al., these 60 patients had low LM scores and the visibility scores were obtained from the surgeons after 60 minutes of surgery start, which limits a thorough assessment of blood loss throughout the procedure. In our case, the surgeon was solicited to verbalize the clarity of the surgical field once surgery was completed, and blood loss was estimated at the end of the case. Unfortunately, in this case report the patient's lesions were not classified utilizing the LM scale or an equivalent grading tool. The study by Ahn et al. study demonstrated standardization of the scoring system for sinus lesions by the surgical team, as well as standardization of blood loss, calculation could aid in further assessing the significant increase in benefit of TIVA vs IA.

A systematic literature review by Kelly et al.[1] looked at prospective controlled trials assessing the effect of TIVA versus IA on the degree of blood loss and the quality of the surgical field visualization. In their analysis, Kelly et al.[1] ascertained that four of the seven studies reported improvements in surgical conditions with TIVA compared to IA supporting the use of propofol. A larger sample size however may have more strongly illustrated the benefits of TIVA over IA. The utilization of TIVA with propofol with the goal of blood loss reduction for ESS is not a common practice at this facility. This demonstrates the need for further and larger studies that can provide strong evidence of statistically significant benefits of TIVA for ESS.

DeConde et al.[3] performed a systematic review and meta-analysis and found a statistically significant improvement in surgical visibility with TIVA when compared to IA. However, the studies contained confounding factors such as differences in the scales determining surgical visualization and in methods for calculating estimated blood loss. Further research is needed.

Endoscopic sinus surgery is a common procedure performed in an outpatient setting for the treatment of sinusitis. One complication associated with ESS is increased blood loss obstructing the clarity of the operative site. In this case scenario, TIVA with propofol was administered to reduce BP, lessen blood loss, improve the surgeons view, and mitigate the patient's postoperative nausea. The patient's BP and HR were adequately maintained to provide controlled hypotension with TIVA. At the end of the surgery, the surgeon stated satisfaction with the minimal blood loss and the adequate surgical view. Ideally, grading the severity of the lesion with an LM score may support or refute the literature. Further exploration regarding anesthetic management for optimal surgical view and for the disparity between non-comparable lesions is warranted.

Until all ESS patient's lesions are graded and tracked, the literature remains elusive. However, ensuring a bloodless surgical field is imperative to adequately identify anatomical landmarks and reduce the risk of complications. The administration of TIVA with propofol appears to accomplish a safe controlled hypotension for patients undergoing ESS.

References

1. Kelly EA, Gollapudy S, Riess M, Woehlck HJ, Poetker DM. Quality of surgical field during endoscopic sinus surgery: a systematic literature review of the effect of total intravenous compared to inhalational anesthesia. *Int Forum Allergy Rhinol.* 2013;3(6):474-481. doi:10.1002/alr.21125.
2. Carlton DA, Govindaraj S. Anesthesia for functional endoscopic sinus surgery. *Curr Opin Otolaryngol Head Neck Surg.* 2017;25(1):24-29. doi:10.1097/MOO.0000000000000322.
3. DeConde AS, Thompson CF, Wu EC, Suh JD. Systematic review and meta-analysis of total intravenous anesthesia and endoscopic sinus surgery. *Int Forum Allergy Rhinol*. 2013;3(10):848-854. doi:10.1002/alr.21196.
4. Yoo HS, Han JH, Park SW, Kim KS. Comparison of surgical condition in endoscopic sinus surgery using remifentanil combined with propofol, sevoflurane or desflurane. *Korean J Anesthesiol.* 2010;59(6):377-382. doi:10.4097/kjae.2010.59.6.377.
5. Ferrari LR, Nargozian C. Anesthesia for otolaryngologic surgery. In: Barash PG, Cullen BF, Stoelting RK, Cahalan MK, Stock MC, Ortega R, eds. *Clinical Anesthesia.* 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2013:1356-1372.
6. Ahn HJ, Chung SK, Dhong HJ, Kim HY, et al. Comparison of surgical conditions during propofol or sevoflurane anaesthesia for endoscopic sinus surgery. *Br J Anaesth.* 2008;100(1):50-54. doi:10.1093/bja/aem304.

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Closed Loop Control of Propofol Infusions in Anesthesia

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Keywords: propofol, closed loop, target control, feedback control, automated infusions

Introduction

Closed loop control of propofol administration has the capability to drastically improve patient care in the anesthesia setting. Closed loop infusions, also known as feedback control systems, adjust drug infusions automatically to stay within set parameters as a result of data collection and analysis.[1] While closed loop anesthesia was first proposed in the 1950s, more recent developments in feedback mechanisms, such as depth of hypnosis measurements, have catapulted research and opened opportunities for automated infusions.[2] Supporters of closed loop anesthesia claim its use will increase the time spent in the desired state of anesthesia, decrease the workload of the anesthesia professional, and improve safety and quality of anesthesia.[1]

The developing research on closed loop infusions has been devoted to automated administration of propofol. Due to its quick onset, rapid elimination, and relative safety profile, propofol has become the most widely used drug in both general anesthesia and conscious sedation.[3] Propofol elicits an effect by activating the gamma-aminobutyric acid A (GABA) receptor, leading to chloride influx, hyperpolarization of the cell, and therefore inhibition of nerve impulses in the brain.[4] Over 90% of propofol is metabolized in the liver by cytochrome p450 enzymes and phase II drug metabolizing enzymes.[4] Since these GABA receptors, enzymes, and pathways can be affected by numerous genetic and environmental mechanisms, there is marked inter-individual variability in the effect of propofol.[4]

Feedback based closed loop infusions allow for real time and automatic titration of propofol to therapeutic effect, leading to a more individualized anesthetic.[7] While target-controlled infusions have been studied for the administration of propofol, they require no actual feedback of drug levels and therefore lack the precision of closed loop administration. Target controlled infusions use recognized pharmacodynamic and pharmacokinetic principles to estimate serum drug levels at the effect site.[2,5,6] Due to the striking inter-individual variability in the distribution, metabolism, and hypnotic dose of propofol, calculated drug levels are simply approximations and are inconsistent.[4] In practice, patients often respond differently to identical doses of propofol for reasons including co-administration of drugs, receptor up and down regulation, enzyme deficiencies, organ insufficiencies, or genetic polymorphisms.[1,4] Closed loop control can account for the inherent pharmacodynamic and pharmacokinetic patient variability that manual administration and target controlled infusion cannot overcome.[2] However, in order for closed loop infusions to successfully function, the concentration of propofol must be monitored accurately and efficiently.[3]

There are various methods proposed for initiating feedback mechanisms in closed loop infusions, however the use of electroencephalographic (EEG) measurement seems to be the most widely researched. By measuring the patient's depth of hypnosis, comparing it to a target value, a closed

loop system can adjust an infusion to optimize and individualize the anesthetic.[2] Other methods of measurement include testing serum propofol levels and propofol breath analysis. It has been suggested that propofol sensors on the outer surface of an indwelling catheter would be feasible for monitoring blood levels of propofol throughout anesthesia.[3] In addition, propofol concentrations measured through an exhaled breath are highly correlated with serum concentrations.[8,9] Propofol can be detected in an exhaled breath within seconds of intravenous injection, at the same time that electroencephalography registers its onset.[8] However, it is important to note that these measures are used to analyze the concentration in the blood which is assumed to be correlated to the concentration at the effect site. One study showed propofol concentrations in the plasma at loss of consciousness can vary among patients.[4] Therefore, this particular study concluded that plasma concentrations cannot always accurately predict action at the effect site.[4]

Further review of the literature reveals that closed loop infusions allow for more time in the defined target anesthetic state with less overshoot in the depth of anesthesia when compared to manual administration.[2,7,10-12] Closed loop infusions also lead to less propofol administered upon induction and less total propofol delivered throughout the case.[7,10,11] Thus, these automated infusions are able to utilize smaller doses while providing a better anesthetic for patients. The automated infusion of propofol also provides a decrease in hemodynamic variability during anesthesia, which could be associated with a decreased total dosage of propofol delivered during the use of these controlled infusions.[7,10,11] Similarly, feedback infusions of propofol have been shown to decrease the unnecessary compensatory use of vasopressors and fluid replacement.[11,12] Closed loop infusions have been associated with a decrease in the time to tracheal extubation and emergence from anesthesia.[1,12] Lastly, automated infusions allow for less manual titration of infusions.[11,12] Closed loop systems make modifications to the dosage more frequently and in smaller increments than manual administration.[11,12] By decreasing operator intervention and variability, safety is increased and overshoot of the anesthetic is reduced.[2]

Methodology

Evidence-based Practice Model

The PICOT format was used to formulate a clinical question that would guide the search criteria. (P) In adult and pediatric surgical patients (I) does closed loop control of intravenous propofol administration (C) compared to manual administration of propofol (O) improve patient safety and quality of anesthesia, and decrease anesthesia provider workload (T) throughout the surgical procedure?

Purpose

The purpose of this literature review is to provide evidence related to the benefits and obstacles of implementing closed loop control of propofol infusions in anesthesia.

Search Terms

Propofol, closed loop infusions, target controlled infusions, pharmacokinetics, pharmacodynamics, feedback infusions

Search Models
A broad electronic database search including PubMed, Cochrane Library, MEDLINE, Cumulative Index to Nursing and Allied Health Literature (CINAHL), Cochrane Database, and Google Scholar were utilized to obtain current, high-level evidence published within the last eight years. Search criteria included studies published from 2009 to 2017 and English speaking journals. Keywords were searched within databases utilizing boolean operators, synonyms and truncation.

Levels of Evidence
Studies examined and utilized include four randomized control trials comprised of one multicenter study and one randomized control phase II trial (Level I evidence) and three studies with no control group classified as observational studies (Level III evidence). The levels of evidence were based on the Johns Hopkins Nursing Evidence-Based Practice Rating Scale.[13]

Literature Review

Depth of Anesthesia Measurement
West et al.[2] utilized a bilateral EEG monitor known as NeuroSENSE. This monitor processes EEG data from sensors on the forehead and provides a value between 0 units and 100 units with 0 representing an isoelectric EEG and 100 representing a fully awake patient. Values between 40 units and 60 units represent the appropriate range for anesthesia and this range was used as the target for the study. During the maintenance phase of anesthesia, the depth of anesthesia was measured within 10 units of the target range 89% of the time.[2] West et al.[2] found that their closed loop system maintained an adequate depth of anesthesia without additional manual administration in 85% of cases. Manual propofol bolus doses administered during the induction phase totaled 0.5mg/kg while bolus doses during the maintenance phase totaled 0.25mg/kg.[2]

Agarwal et al.[10], Biwas et al.[11], Le Guen et al.[7], and Liu et al.[12] utilized the Bispectral Index in four separate randomized control trials. The Bispectral Index (BIS) monitor has been shown to correlate with estimated propofol concentrations in patients older than two years of age.[11] The monitor reduces encephalography data to quantify the EEG into an empirical number ranging from an awake encephalogram at a reading of 100 to isoelectric at zero.[8,10] The BIS monitor is well known to function properly even in an electrically noisy environment such as an operating room.[10] Le Guen et al.[7] found a significant difference (P=0.001) in the time spent under adequate sedation between a closed loop group and a manual administration group with the automated group experiencing adequate sedation an average of 77% of the time while the manual group experienced only 36% adequate sedation.

Hornuss et al.[8] aimed to show that propofol concentrations in exhaled breath detected via ion molecule reaction mass spectrometry can be useful for titrating intravenous anesthesia. This method detects demethylated propofol fragments with a mass of 163 D, a mass at which volatile anesthetics cannot influence the detection of propofol.[8] No significant difference was found (P>0.29) when comparing the time to detection of propofol by ion molecule reaction mass spectrometry and the time to detection of effect on BIS readings.[8] There was also no significant difference between the time to peak expiratory propofol concentration and time to lowest BIS reading (P=0.57).[8] While this suggest that expiratory propofol concentrations and plasma

concentrations mimic concentrations at the effect site, other studies have shown plasma concentrations do not always correlate to the effect site.[4] Khan et al.[4] noted that propofol plasma concentrations at loss of consciousness varied from 2.1 ug/ml to 29.3 ug/ml and therefore plasma concentrations cannot adequately predict action at the effect site.

Propofol Dosing

Agarwal and collegues[10] found a significant difference (P=0.0001) in the dose of propofol needed for induction, with a much lower dose utilized in the closed loop anesthesia delivery system group than in the manual group. Biwas et al.[2] supported the finding that lower doses of propofol were utilized for induction with a closed loop system (2.06 ± 0.79 mg/kg) than the manual group (2.95 ± 1.03 mg/kg). Agarwal et al.[10], Le guen et al.[7], and Biwas et al.[11] concluded less total propofol was used throughout the surgical procedure with the closed loop infusion (13.3 ± 3.8 mg/kg) verses manual administration (17.1 ± 6.9 mg/kg).

Hemodynamic Stability

Agarwal et al.[10] demonstrated that mean arterial pressure and heart rate were maintained within 25% of baseline for a larger percent of the time during closed loop anesthesia than with the manual group. In addition, patients in the closed loop group required an average phenylephrine dose of 172.5 mcg/kg while the manual group required an average of 331.3 mcg/kg (P=0.03). Biwas et al.[11] confirmed these hemodymanic benefits with their trial which found mean arterial pressure was maintained for a longer period of time with significantly less phenylephrine use in the closed loop group than in the manual group (P=0.006) . Le Guen et al.[7] found vasopressor infusions were three times more likely to be discontinued in patients when sedation was provided through automated feedback infusions than with manual administration.

Time to Extubation

Liu et al.[12] found the time to endotracheal tube extubation was shorter in the closed loop group than in the manual group (P<0.2). In this study extubation was only performed when the patient met specific criteria including Spo2 >95% with an oxygen inspiratory fraction < 50%, four twitches with a twitch ratio >90%, no hemodynamic instability, and a cooperative and responsive patient.

Articles	Description	Results	Conclusion
West, Dumont, Vanheusden, et al., 2013[2]	One hundred and eight children were enrolled and sedated using closed loop controlled propofol infusions, which were continually adjusted according to feedback from an electroencephalographic measurement of depth of hypnosis.	The closed loop system maintained an adequate depth of anesthesia in 85% of cases and only required manual intervention in 11% of cases.	Due to the large pharmacodynamic and pharmacokinetic variability observed in pediatric patients, closed loop infusions could be beneficial in optimizing intravenous anesthesia.
Agarwal, Puri, Mathew, 2009[10]	Forty-five adults undergoing cardiopulmonary bypass surgery were enrolled and randomized to receive a closed	The closed loop group required less propofol, had less overshoot of BIS, and maintained	The automated delivery of propofol was safer and more advantageous than

	loop infusion or manual administration of propofol. The closed loop delivery system utilized the BIS monitor to regulate the infusion of propofol.	hemodynamic parameters more effectively than the control group.	manual administration in open heart surgery. While closed loop control of anesthesia has been described in healthy patients, this study revealed its benefits in cardiac surgery patients, who are often characterized by hemodynamic instability and higher risk of awareness.
Biswas, Mathew, Singh, Puri, 2013[11]	Compared the use of closed loop anesthetic delivery of propofol using the Bispectral Index versus manual control of propofol in pediatric patients during surgery requiring cardiopulmonary bypass. Forty children were enrolled in the study and randomly assigned to the closed loop group or manual group.	The maintenance of Bispectral Index and hemodynamic stability were similar between the two groups, however, the induction dose of propofol and total propofol used was less in the closed loop group. There was less overshoot of BIS and less phenylephrine use in the closed loop group. The manual group required an average of eighteen dose adjustments per hour while the closed loop group required none.	Established the potential advantages and feasibility of using closed loop controlled propofol infusions in pediatric cardiac surgery.
Hornuss, Wiepcke, Praun, Dolch, Apfel, Schelling, 2012[8]	Aimed to show that propofol concentrations in exhaled breath detected via ion molecule reaction mass spectrometry can be useful for titrating intravenous anesthesia. To evaluate the utility of propofol detection in expired gas, the time course of expiratory propofol was compared to the estimated cerebral propofol effect using the BIS monitor.	The study showed similar times of onset and peak expired propofol concentrations and onset and peak of the cerebral effect of propofol via the BIS monitor.	Suggests expiratory propofol may be useful in titrating intravenous anesthesia. The ability to measure propofol concentrations in real time would allow for adequate anesthesia and an accurate method of feedback for closed loop systems.

Le Guen, Liu, Bourgeois, et al., 2013[7]	Thirty one intensive care patients were enrolled in a randomized controlled phase II trial in which automated infusions of propofol and remifentanyl guided by BIS were compared to manual administration.	The automated group experienced adequate sedation much more frequently than the manual group. Propofol consumption and vasopressor use was also reduced within the automated group compared to the manual group.	Automated control maintained sedation better than manual control in intensive care patients while utilizing less drug and less vasopressor. While this study was not performed on surgical patients it is further evidence that automated control can be beneficial in the most critical of patients.
Liu, Chazot, Hamada, et al., 2011[12]	A multicenter study of 196 patients were enrolled and randomly assigned to receive propofol and remifentanyl via manual administration or by a dual closed loop infusion guided by BIS. The study was conducted at four different sites and included seventeen anesthesiologist and twenty-two nurse anesthetist.	The percentage of time with an adequate anesthetic as evidence by BIS was higher in the dual loop group. Overshoot, undershoot, and burst suppression of BIS occurred less often in the dual loop group. Modifications to the infusions were more frequent and in smaller increments in the closed loop group. The time until tracheal extubation was less in the dual group.	Controlled closed loop infusion of propofol and remifentanyl allowed for more stable BIS values during general anesthesia compared to manual administration.
Janda, Simanski, Bajorat, Pohl, Noeldge-schomburg, Hofmockel,2011[1]	Twenty-two adults were enrolled in a study to evaluate a closed loop system using the BIS monitor and electromylogram simultaneously to control depth of anesthesia and neuromuscular blockade.	There was no human intervention required during the closed loop administration of propofol and mivacurium. There were no claims of awareness and all patients rated the quality of anesthesia as “good” or higher.	The automatic infusion of anesthesia was able to provide adequate anesthesia by maintaining the target value of the BIS monitor.

Conclusions

According to the literature reviewed, closed loop infusions of propofol have demonstrated benefits to the practice of anesthesia through its effects on anesthesia provider workload and patient safety. Automated infusions provide adequate depth of anesthesia, decreased total propofol use, and decreased hemodynamic variability. The efficacy of closed loop infusions allow for quality anesthesia with increased usage of total intravenous anesthetics, which are associated with improved patient outcomes compared to inhalation anesthetics.[5] In addition, closed loop infusions may decrease time to endotracheal tube extubation.[12] These closed loop systems also require little intervention from the anesthesia practitioner, which allows for more time dedicated to providing a well-rounded anesthetic.[2,11] While the benefits of the electroencephalogram for feedback control infusions has been widely demonstrated in a number of randomized control trials, the use of other methods for "closing the loop" require randomized trials to evaluate their clinical impact. Specifically, more research is needed to determine whether propofol plasma concentrations can adequately predict action of propofol at the GABA receptors in the brain.[4]

References

1. Janda M, Simanski O, Bajorat J, Pohl B, Noeldge-schomburg GF, Hofmockel R. Clinical evaluation of a simultaneous closed-loop anaesthesia control system for depth of anaesthesia and neuromuscular blockade. *Anaesth*. 2011;66(12):1112-1120.
2. West N, Dumont GA, Van heusden K, et al. Robust closed-loop control of induction and maintenance of propofol anesthesia in children. *Paediatr Anaesth*. 2013;23(8):712-719.
3. Kivlehan F, Chaum E, Lindner E. Propofol detection and quantification in human blood: the promise of feedback controlled, closed-loop anesthesia. *Analyst*. 2015;140(1):98-106.
4. Khan M, Zetterlund E, Eintrei C, et al. Pharmacogenetics, plasma concentrations, clinical signs and EEG during propofol treatment. *Basic Clin Pharmacol Toxicol*. 2014;115(6):565-570.
5. Bibian S, Dumont GA, Black I. Closed-loop target-controlled infusion systems: stability and performance aspects. *Milit Med.* 2015;180(suppl 3):S96-S103.
6. Myers MH, Li Y, Kivlehan F, Lindner E, Chaum E. A feedback control approach to organic drug infusions using electrochemical measurement. *IEEE Trans Biomed Eng.* 2016;63(3):506-511.
7. Le Guen M, Liu N, Bourgeois E, et al. Automated sedation outperforms manual administration of propofol and remifentanil in critically ill patients with deep sedation: a randomized phase II trial. *Intensive Care Med*. 2013;39(3):454-462.
8. Hornuss C, Wiepcke D, Praun S, Dolch ME, Apfel CC, Schelling G. Time course of expiratory propofol after bolus injection as measured by ion molecule reaction mass spectrometry. *Analytical Bioanalytical Chem*. 2012;403(2):555-561.
9. Akbar M, Agah M. A microfabricated propofol trap for breath-based anesthesia depth monitoring. *J Microelectromechanical Syst*. 2013;22(2):443-451.
10. Agarwal J, Puri GD, Mathew PJ. Comparison of closed loop vs. manual administration of propofol using the bispectral index in cardiac surgery. *Acta Anaesthesiologica Scandinavica.* 2009;53(3):390-397.

11. Biswas I, Mathew PJ, Singh RS, Puri GD. Evaluation of closed-loop anesthesia delivery for propofol anesthesia in pediatric cardiac surgery. *Paediatr Anaesth*. 2013;23(12):1145-1152.
12. Liu N, Chazot T, Hamada S, et al. Closed-loop coadministration of propofol and remifentanil guided by bispectral index: a randomized multicenter study. *Anesth Analg*. 2011;112(3):546-57.
13. Dang, D. (2012). *Johns Hopkins Nursing Evidence-Based Practice Model and Guidelines, Second Edition*. Sigma Theta Tau International.

Mentor: David Fort, DNP, CRNA

Editorial

First, I would like to announce the annual meeting of the ISJNA during the newly renamed Assembly of Didactic and Clinical Educators. It will be held on Friday, February 15th from 6:45-7:30 AM in Champions Ballroom I&II. Anyone involved with or interested in learning more about the ISJNA is welcome to attend!

It is with much appreciation that I announce the retirement of the following individuals from the Editorial Board:

Rhonda Gee, DNSc, CRNA
Johnnie Holmes, PhD, CRNA
Donna Jasinski, PhD, CRNA

I would also like to welcome the following new Editorial Board Members:

CDR Raymond Bonds, DNP, CRNA, CHSE, NC, USN
LCDR Chad Moore, DNP, CRNA, NC, USN

Several new reviewers have joined our ranks as well, and I am so grateful for all of the hard work and dedication shown by all of these individuals. I truly appreciate all of the time and effort the editorial board members and reviewers commit to sustaining the ISJNA – it would not exist without you!

Sincerely,

Vicki C. Coopmans, PhD, CRNA
Editor

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

INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA

GUIDE FOR AUTHORS

MISSION STATEMENT

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

ITEM PREPARATION & SUBMISSION

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

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

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

REVIEW PROCESS

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

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

PHOTOS

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

ACADEMIC INTEGRITY

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

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

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

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

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

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

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

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

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

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

Title

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

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

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

Heading (see above)

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

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

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

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

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

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

Heading

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

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

Heading

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

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

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

Heading

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

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

AMA MANUAL OF STYLE

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

SUBMISSION CHECK LIST

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

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

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

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

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

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

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