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TOPICS IN THIS ISSUE

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Front Cover:

Timothy Joseph, RN, BSN and Emily Hoffiz, RN, BSN, graduate students enrolled in the Oakland University-Beaumont Graduate Program of Nurse Anesthesia, practice clinical skills with Clinical Coordinator Linda McDonald, DrAP, CRNA in the simulated operating room in the Marcia & Eugene Applebaum Simulation Learning Institute at Beaumont, Royal Oak.

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Case Reports

Remifentanil for Maintenance of General Anesthesia during Carotid Endarterectomy

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Keywords: Carotid endarterectomy, remifentanil, electroencephalography

The use of regional versus general anesthesia for carotid endarterectomy (CEA) is a strongly debated issue. Despite inhalation agents' ability to provide predictable amnesia, they can interfere with neurological monitoring such as electroencephalography (EEG), and can cause hemodynamic lability. A balanced remifentanil and volatile anesthetic technique can cause less hemodynamic variability while maintaining intact EEG signals. Remifentanil also facilitates rapid smooth emergence due to its short elimination half-life, allowing for immediate postoperative neurological assessment.[1] The following report presents a case utilizing a balanced general anesthetic technique for patients undergoing CEA.

Case Report

A 75-year-old male, measuring 167 cm in height and weighing 78 kg, presented for left CEA following a cerebral vascular accident that resulted in right-sided facial droop and weakness in the right upper extremity. Past medical history included chronic obstructive pulmonary disease, hypertension, myocardial infarction, and abdominal aortic aneurysm. Past surgical history involved right pneumonectomy, coronary angioplasty with stent placement, and abdominal aortic aneurysm repair. The patient's medications were aspirin 81 mg, Plavix 75 mg, and lisinopril 2.5 mg, all taken by mouth once per day. Physical examination in the preoperative area revealed decreased breath sounds on the left without signs of increased work of breathing. Peripheral intravenous (IV) access was achieved with an 18-gauge catheter in the left hand and an intra-arterial catheter was inserted for direct blood pressure measurement. Sedation was not administered to the patient prior to transport to the operating room to avoid interference with baseline neurological assessment. Additionally, a 16-channel EEG was placed by an experienced electroencephalographer in the preoperative area, and baseline signals were recorded prior to induction of anesthesia.

Upon arrival to the operating room, the patient was positioned supine on the operating table. A pulse oximeter, five-lead ECG, and arterial line transducer were applied and the patient was pre-oxygenated with 100% oxygen via face mask. Induction of anesthesia was achieved with fentanyl 100 mcg and propofol 100 mg, and rocuronium 50 mg IV was given to attain muscle relaxation. The patient's trachea was intubated atraumatically under direct laryngoscopy with a MacIntosh size 3 blade and an 8.0 mm cuffed endotracheal tube (ETT). Placement of the ETT was confirmed by the presence of end-tidal carbon dioxide (EtCO2) and auscultation of breath sounds on the left. General anesthesia was maintained with sevoflurane 1% inspired concentration in a mixture of O_2 1 L/min and air 1 L/min and a remifentanil infusion at 0.05 mcg/kg/min. The patient was mechanically ventilated to maintain $EtCO_2$ between 35 and 40 mm Hg. During carotid clamping, a phenylephrine infusion was started at 20 mcg/kg/min to maintain systolic blood pressure between 190 and 200 mm Hg, which was 20% above the patient's preoperative blood pressure. Heparin 6000 units IV was administered prior to carotid clamping.

Surgery progressed uneventfully, with the patient's EEG signals remaining intact. The electroencephalographer focused primarily on any changes observed in the amplitude of alpha waves, which would suggest cerebral hypoperfusion during carotid clamping.

At the beginning of skin wound closure, sevoflurane was discontinued and remifentanil was increased to 0.15 mcg/kg/min. Due to its rapid elimination half-time of approximately seven minutes, the remifentanil infusion was not discontinued until the end of skin closure, and no additional opioids were administered. Neuromuscular blockade was antagonized with neostigmine 3 mg and glycopyrrolate 0.6 mg IV during skin closure. At the time of emergence, the monitor demonstrated 0% expired sevoflurane and the patient was breathing spontaneously at a respiratory rate of 16/min and achieving tidal volumes of 400 – 420 mL, with SpO_2 ranging between 94 and 96%. The patient was able to respond appropriately to commands and demonstrated return of protective airway reflexes and respiratory muscle strength. The oropharynx was suctioned for a moderate amount of thin clear secretions and the trachea was extubated without coughing.

The patient was placed on O_2 8 L/min via a non-rebreather mask, and the SpO_2 remained between 95 and 97%. In the post anesthesia care unit, the patient's vital signs remained stable and both pain and nausea were absent. The patient remained alert and oriented, with return of right upper extremity strength and full strength demonstrated in all other extremities. Transfer to a step-down unit for overnight observation took place two hours later.

Discussion

Although the debate over regional and general anesthesia has not been settled, there is information in the literature describing techniques that improve anesthetic management of patients undergoing CEA.[2] Studies have shown that both regional and general techniques can be safe for patients undergoing this surgery. Two serious perioperative complications of CEA are cerebrovascular accidents and myocardial infarction. The incidence of perioperative stroke associated with CEA is approximately 3.4% for asymptomatic patients and 5.6% for symptomatic patients.[3] To reduce this incidence, a number of techniques and devices have been developed to detect cerebral ischemia.[3] Providing heart and brain protection during CEA can be accomplished by either regional or general anesthesia, as long as wide fluctuations in hemodynamics are prevented and anesthetics that do not interfere with intraoperative neurological function and monitoring are utilized. This report focuses on the rationale behind employing a combined narcotic/inhalational general anesthetic technique for CEA. A review of the pharmacology of the specific anesthetic agents as well as the technique's implications on cardiovascular and neurological function will be presented.

Opioid-based general anesthesia is advantageous for carotid endarterectomy because of this technique's ability to blunt responses to noxious stimuli and maintain cardiovascular stability.[4] In particular, remifentanil provides the same hemodynamic stability noted with other opioid-based anesthetic techniques but holds the advantage in that it can be rapidly titrated to a desired depth of anesthesia.[4] It is metabolized quickly by plasma esterases and allows for rapid elimination.[2] Hypotension must be avoided during CEA, as it can have deleterious effects on collateral

cerebral blood flow during carotid clamping. As demonstrated by the patient presented in this report, using remifentanil decreased the inhaled anesthetic requirement and produced less vasodilation and subsequent hypotension. Administering less than 1 minimum alveolar concentration (MAC) sevoflurane allowed for adequate maintenance of systolic blood pressure and resulted in minimal hemodynamic variability during carotid clamping.

A possible disadvantage to utilizing opioids during carotid endarterectomy is that the respiratory depression and sedation they cause may persist into the postoperative period and may distort neurologic evaluation in this patient population[4.] We were concerned about respiratory depression in our patient who had known underlying respiratory disease and decided to avoid long acting narcotics. Reliable postoperative assessment of neurological status in CEA patients is essential for detecting cerebral ischemia and neurological decline. To prevent narcotic-associated sedation, anesthesia practitioners must have a complete understanding of the pharmacokinetic profile of the drug. Due to appropriately-timed initiation, titration, and discontinuation of remifentanil, the patient described in this case report did not display evidence of respiratory or neurological decline following emergence from anesthesia. Most importantly, evaluation of this patient's neurological status was possible immediately post extubation and remained stable throughout the postoperative period.

Use of cerebral function monitoring (the most simple being the EEG), may permit optimization of sevoflurane dose and avoidance of burst suppression and major epileptiform signs in fragile subjects during carotid endarterectomy.[5] Epileptogenic activity, which has been noted in subjects receiving high-dose sevoflurane for maintenance of anesthesia, must especially be avoided in patients undergoing carotid surgery. These patients are inherently at risk of experiencing cerebral deterioration due to decreased global cerebral blood flow and reliance on collateral flow during carotid clamping. The incidence and the periodicity of epileptiform EEG changes correlate with the increasing expired fraction of sevoflurane, which can confuse interpretation of cerebral function monitors.[5] Studies have shown that using a maximum of 1.5 MAC sevoflurane for maintenance of anesthesia can limit epileptogenic activity of sevoflurane, which increases at higher concentrations.[5] However, in order to prevent disturbances in EEG monitoring during CEA, some literature has suggested that a maximum of 1 MAC of inhalational agent be utilized in conjunction with IV narcotics or sedative-hypnotics for maintenance of anesthesia. Narcotics used in large doses can cause a dose-dependent slowing of the EEG. However in current clinical practice, narcotics used in low doses show few effects on EEG.[5]

To date, there remains no definitively superior anesthetic technique for patients undergoing CEA. While general anesthesia is an effective method, anesthesia professionals must be meticulous in their selection of anesthetic agents. They must be fully aware of the implications of general anesthetics on hemodynamics and neurological function during carotid surgery, as well as their effect on neurological monitoring. As demonstrated by the patient presented in this case report, a balanced sevoflurane/remifentanil general anesthetic was effective in providing analgesia, amnesia, and hemodynamic stability while preventing interference with perioperative neurological function and monitoring. Adverse effects associated with remifentanil such as respiratory depression and sedation, can be avoided with appropriate titration of the drug and adequate knowledge of its pharmacokinetic profile. Currently, selection of anesthetics for patients undergoing CEA should be primarily based on how best to maintain cardiovascular and

neurological stability, while providing appropriate levels of analgesia and amnesia and suitable operating conditions.

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Prevention of Awareness in Trauma Patients with Substance Abuse

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Keywords: amphetamines, anesthesia awareness, intraoperative awareness, methamphetamines, recall, substance abuse, trauma

Anesthesia awareness occurs during general anesthesia when a patient is cognizant of events during surgery and later recalls explicit events.[1] The frequency of anesthesia awareness is estimated between 0.1% and 0.2% of all patients undergoing general anesthesia, with 20,000 to 40,000 cases occurring in the United States annually.[1,2] Trauma patients have a higher incidence of awareness ranging from 11-43%. Patients with substance abuse and American Society of Anesthesiologists (ASA) physical status IV or V have a higher risk of intraoperative awareness.[3] Substance abuse not only increases the risk for anesthesia awareness, but also for adverse outcomes and drug interactions during anesthesia.[3,4]

Case Report

A 40-year-old, 73 kg, 185 cm male was transported to the emergency room (ER) after being stabbed. In the field, the patient had a heart rate (HR) of 120/min, Glasgow Coma Score (GCS) of 3 and spontaneous ventilations. A cervical collar was placed and intravenous (IV) access obtained. Needle decompression was performed for diminished lung sounds to the right chest and stab wounds to the lower chest wall with no air or blood expelled. Upon arrival in the ER,

the patient had a GCS of 8 and oxygen was administered at 15 L/min via face mask. Electrocardiogram (ECG) revealed sinus tachycardia at a rate of 122/min and systemic hypotension was treated with 2 L of normal saline. Injuries assessed were multiple stab wounds to the neck, chest, abdomen, extremities, and a right pneumothorax.

Significant laboratory values included: white blood cell count 20.2 K/uL, red blood cell count 3.68 M/uL, hemoglobin 12.5 g/dL and hematocrit 35.9%. Chemistry and coagulation studies were within normal limits. Urine toxicology was positive for opiates, tricyclic antidepressants, and methamphetamines, with levels unavailable. Serum alcohol was negative. Computed tomography with IV contrast showed a right pneumothorax, possible liver and splenic injuries, ascites in the abdomen and pelvis with no evident pneumoperitoneum.

The patient was transported to the operating room (OR) for an emergency exploratory laparotomy, right chest tube placement, and repair of multiple lacerations. Noninvasive blood pressure, oxygen saturation, ECG and temperature monitors were applied. Oxygen 10 L/min was administered via face mask in preparation for a rapid sequence induction. Cricoid pressure was applied, general anesthesia induced with etomidate 40 mg and succinylcholine 140 mg. Video laryngoscopy performed using a C-Mac (Karl Storz-Endoskope, Tuttlingen, Germany) with placement of an 8.0 mm endotracheal tube. Controlled ventilation settings were as follows: respiratory rate 12/min, tidal volume 650 mL, PEEP 5 cm H_2O, peak airway pressure 16 cm H2O, and O_2 ? L/min. A radial intra-arterial catheter was placed and a phenyleprhine infusion initiated for a blood pressure of 80/50 mm Hg and HR of 105/min. An additional 18-gauge IV catheter was inserted for fluid resuscitation and a total of 4,500 mL of normal saline administered via inline fluid warmer. Hemodynamics and $ETCO_2$ remained within normal limits. Sevoflurane was maintained at an average minimum alveolar concentration (MAC) of 0.7, ET% 1.0 to 1.7 in O_2 2 L/min. Hydromorphone 2 mg was administered, and rocuronium 100 mg total bolused throughout the procedure to maintain train of four at one to two twitches.

A right-sided chest tube was inserted. The laparotomy revealed a hemoperitoneum due to a grade II liver laceration. Multiple lacerations to the neck, bilateral arms, chest and abdominal walls were repaired. No blood products were transfused and estimated blood loss in the OR totaled 500 mL. Upon completion of the surgery, neuromuscular blockade was antagonized with neostigmine 5 mg and glycopyrrolate 0.2 mg. The patient emerged from general anesthesia with spontaneous ventilations, stable oxygenation was extubated and transported to the intensive care unit for postoperative care.

On postoperative day 1, the patient received 2 units of packed red blood cells for a hemoglobin of 6.7 G/dL. The patient remained in the hospital for 6 days and was discharged to the county detention center.

Discussion

Awareness, a serious complication of anesthesia, is described as a patient's worst hospital experience.[3] Patients who experience intraoperative awareness may report sensations of paralysis and pain, perception of surgical manipulation, auditory perception, or feelings of panic and helplessness. Post-traumatic stress disorder may develop in patients who experience

intraoperative awareness. While anesthesia awareness can be a major complication, there are situations when anesthesia awareness may be unavoidable to achieve essential lifesaving goals by anesthesia professionals.[3]

The incidence of anesthesia awareness is reported to be higher among patients in which general anesthetics must be administered in smaller doses, such as patients with hemodynamic instability.[1] Other risk factors found to contribute to the development of intraoperative awareness are a low dose of primary anesthetic agent, non-continuous dosing of the primary anesthetic agent, lack of premedication, and female gender. Specific patient history, surgical procedures, and anesthetic management increase a patient's risk for intraoperative awareness. Patient specific factors include previous awareness, history of substance abuse, chronic pain patient on high dose opioids, difficult tracheal intubation, ASA physical status IV or V, and limited hemodynamic reserve. Surgical procedures for trauma, emergent and cardiac surgery are awareness risk factors. Lastly, anesthetic factors include the use of neuromuscular blockers, total intravenous anesthesia, use of nitrous oxide, decreased anesthetic doses with paralysis and surgery requiring evoked potential monitors. Additionally, studies show that the most identifiable cause of awareness during general anesthesia is due to the lack of primary anesthetic agent administration or a reduced concentration, as occurred in this case maintaining sevoflurane at less than one MAC.[3]

Intraoperative awareness is estimated to be as high as 43% in patients experiencing major trauma and a large proportion of these patients have a history of major depression.[3] The patient in this case was a trauma victim who had a urine toxicology positive for tricyclic antidepressants and methamphetamines. Substance abuse not only put this patient at risk for anesthesia awareness, but also adverse outcomes and drug interactions during anesthesia.[3,4]

According to Cruickshank and Dyer, methamphetamines are the second most commonly abused illicit drug worldwide.[6] A high incidence of methamphetamine use is noted in young trauma patients and anesthesia professionals must be aware of the clinical implications of illicit drug use.[5] Methamphetamine is an indirect agonist of dopamine, adrenaline, and serotonin receptors and is structured similarly to amphetamines and monoamines.[7] Therefore, methamphetamine attenuates monoamine metabolism through inhibition of monoamine oxidase and long-term abuse can result in depletion of catecholamine stores in the body.[6,7] Patients acutely intoxicated from ingestion of amphetamines may exhibit hypertension, tachycardia, hyperthermia, and increased requirements for inhalation anesthetics. It has also been reported that intraoperative intracranial hypertension and cardiac arrest have been attributed to amphetamine abuse.[7]

Catecholamine depletion in chronic methamphetamine abusers may result in decreased anesthetic requirements and refractory hypotension. Anesthesia professionals should use direct-acting vasopressors, such as neosynephrine, to treat hypotension as done in this case. Indirect-acting vasopressors like ephedrine can intensify the catecholamine depletion. Monitoring blood pressure using an intra-arterial catheter is also advised.[7]

The confounding characteristic of substance abuse in trauma patients warrants special attention by anesthesia professionals to prevent awareness. A major problem with awareness during general anesthesia is that recall can only be detected postoperatively through interviewing the

patient. The anesthesia professional should monitor anesthetic depth with monitors and clinical patient signs to help avoid awareness.[3] Bispectral Index (BIS) (Covidien, Minneapolis, MN) could have been used in this case to monitor the state of hypnosis during general anesthesia as it is the only brain function monitor, according to Borzova and Smith, associated with a decreased incidence of anesthesia awareness.[3] Practitioners must be aware of the lag time with BIS monitoring and factors that can influence numerical values. While the interaction with acute methamphetamine ingestion is not documented, other medications including ketamine can increase BIS values due to EEG waveform activation.[3]

In the emergent case presented, there is much opportunity for improvement to prevent intraoperative awareness. While it is not known if the patient had any reports of recall while under general anesthesia due to lack of postoperative interview, he should have been treated as high risk for awareness based on several factors. Triggers must be in place to interview all high risk patients for recall under anesthesia. Additionally, premedication with an amnestic drug might have been administered as this has been shown to aid in the prevention of awareness while administering a light anesthetic. Sevoflurane MAC of 0.7 could have been supplemented with nitrous oxide upon resolution of the pneumothorax.[3] Small doses of midazolam, ketamine, or scopolamine are also advantageous in preventing recall and brain function monitoring with a BIS should have been implemented. [3] While anesthesia awareness cannot always be prevented given hemodynamic instability, professionals must manage high risk patients to prevent long-term disabling outcomes and complications from drug interactions in the patient with a history of substance abuse.[1]

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Negative Pressure Pulmonary Edema after an Adult Laryngospasm

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Keywords: negative pressure pulmonary edema, laryngospasm, NPPE

Negative pressure pulmonary edema (NPPE) is a rare but potentially fatal complication of anesthesia. Since little is known about mortality rates, it is important for anesthetists to be able to recognize and treat this emergency.[1] NPPE occurs when there is an inspiratory effort against a closed glottis, commonly during a laryngospasm. These efforts cause a negative pressure inside the pleura and alveoli. Therefore, fluid inside the pulmonary capillaries is "suctioned" into the alveolar and interstitial spaces.[2] Hypoxemia ensues due to lack of gas exchange.[2] The systemic vasculature compensates for hypoxemia by sending more blood to the pulmonary circulation. This increases pulmonary capillary resistance, in turn, worsening hypoxemia and pulmonary edema.[2] This article presents a case of NPPE after postoperative laryngospasm.

Case Report

A 42-year-old, 178 cm, 108 kg male with a full beard presented for a second ankle debridement. The patient's medical history was significant for hypertension and obstructive sleep apnea. His surgical history included a debridement of the ankle and external fixation of the foot fracture without any anesthetic complication. Chronic medications were carvedilol, losartan potassium, amlodipine, and chlorthalidone. Midazolam 1 mg and fentanyl 50 mcg were administered intravenously preoperatively.

The patient was transferred to the operating room and monitors were applied. Following positioning and preoxygenation, anesthesia was intravenously induced with lidocaine 100 mg, propofol 200 mg, and alfentanil 1000 mcg. Direct laryngoscopy was unremarkable, and the trachea was intubated with a 7.0 endotracheal tube. Positive $ETCO_2$ was confirmed, and bilateral breath sounds were auscultated. General anesthesia was maintained with an inspired concentration of isoflurane 0.8-1.2% in a mixture of oxygen 1 L/min and air 1 L/min with stable vital signs throughout surgery.

One hour later the surgical procedure was complete. Isoflurane was discontinued, and O_2 flow was increased to 10 L/min. The patient assessment revealed the ability to squeeze the anesthetist's hand and open his eyes. A minute ventilation of 3.6 L/min was noted. The trachea was extubated, 10 L/min O_2 was administered via facemask, and chest rise was observed. However, $ETCO_2$ readings were absent, and tracheal auscultation revealed absent breath sounds.

Jaw thrust and positive pressure ventilation with 10 L/min O_2 were immediately performed with no return of $ETCO_2$. The patient's SpO_2 decreased to 35%. Succinylcholine 40 mg was administered intravenously, and positive pressure ventilation was accomplished. There was a return of $ETCO_2$; however, SpO_2 remained 75%. The trachea was re-intubated uneventfully and confirmed by auscultation of the chest, the presence of $ETCO_2$ and equal chest rise. SpO_2 then increased to 98%. Tracheal extubation ensued after extubation criteria, as previously noted, were met.

Upon arrival to the recovery unit, the patient had a productive cough and complained of shortness of breath. Despite 6 L/min O_2 administered via facemask, SpO_2 readings were 94%. Bilateral lung field auscultation revealed rhonchi. Furosemide 20 mg was administered intravenously, and a portable chest radiograph was requested due to a high suspicion of NPPE. Bipap with 100% O_2 was used to assist respiratory effort. The chest radiograph demonstrated diffuse interstitial prominence, likely reflecting pulmonary edema in support of NPPE differential. The patient was transferred to the floor without further anesthesia complications or need for re-intubation.

Discussion

A student registered nurse anesthetist frequently encounters didactic and clinical scenarios that address proper response to life-threatening emergencies seen during the perianesthesia period. Laryngospasm is one of those elusive issues. Miller summarizes appropriate treatment when practitioners encounter laryngospasm in the clinical environment.[3] First line therapy requires 100% O_2 administration with positive pressure via facemask and jaw thrust, if necessary to open the airway.[3] Succinylcholine is subsequently administered if first line therapy is ineffective.[3] In the case study presented, the patient, unfortunately, acquired NPPE despite quick recognition and appropriate treatment by the anesthesia team. Understanding laryngospasm pathophysiology, recognizing symptoms and initiating a rapid response is paramount to prevent increased morbidity and mortality, which may include NPPE.

The upper airway has many functions, but protecting the lower airway from foreign bodies is its most important task.[4] The larynx is innervated by sensory and motor pathway nerves utilized to protect against foreign material. The internal superior laryngeal nerve is the afferent or sensory, nerve involved in the laryngeal closure reflex. This nerve detects changes in temperature, sensation and chemical make-up of a foreign body. It innervates the area of the larynx above the glottis. The external branch innervates the cricothyroid muscle. The recurrent laryngeal nerve innervates the other intrinsic muscles of the larynx. More specifically, the lateral cricoarytenoid, cricothyroid and thyroarytenoid muscles are responsible for laryngeal closure during a laryngospasm. These make up the motor, or efferent, pathways of a laryngospasm. So, a laryngospasm occurs when the superior laryngeal nerve senses a foreign body near the glottis and the intrinsic muscles of the larynx close the vocal cords via the recurrent laryngeal nerve.[5]

It is important to recognize a laryngospasm quickly to prevent adverse reactions such as hypoxemia and even cardiac arrest. A laryngospasm can be recognized by an audible "crowing" sound from the patient. However, this is defined as stridor which is not a complete adduction of the vocal cords as it is with laryngospasm.[5] Typically, a laryngospasm occurs rapidly and

relatively silently, as seen with a choking victim.[4] It is possible to see signals such as abdominal movement or supraclavicular or suprasternal retractions. Because the glottic opening is completely closed, there would be an absence of air movement; and the reservoir bag would not move. Also noted would be absent breath sounds and a flat $ETCO_2$ waveform. If the laryngospasm continues, late signs include hypoxemia, bradycardia, and cyanosis.[3] By these definitions; the presented case study was a true laryngospasm. There were no signs or sounds of respiration noted. The onset was rapid, resulting in hypoxia. Chest rise was observed, which delayed the recognition of the laryngospasm. However, after noting the other factors of no breath sounds, a flat capnogram and no movement of the breathing bag, steps were quickly taken to break the spasm. Retrospectively, the chest rise could be seen as the trigger for the NPPE.

Manifestations of NPPE are tachypnea, tachycardia, pink frothy sputum, decreased oxygen saturation, rhonchi, and an abnormal chest radiograph.[3] This must occur, of course, in the appropriate setting for this diagnosis. Typically, it occurs after an acute airway obstruction such as a laryngospasm. The patient in the case study has all of the signs except obvious pink, frothy sputum. The patient did have a productive cough, but the color and texture of the sputum were not consistent with the typical findings of NPPE.

Overall, this case study aligned with literature that addresses laryngospasm and NPPE. The pathophysiology and presentation of signs and symptoms of these phenomena were apparent and treated quickly. The patient was fortunate that the anesthetists intervened swiftly and avoided an adverse outcome.

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Anesthesia for a Patient with Multiple Sclerosis

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Keywords: multiple sclerosis, regional anesthesia, general anesthesia, osteotomy calcaneal, peroneal tendon rupture

Multiple sclerosis (MS) is an incurable autoimmune disease resulting in progressive inflammation and demyelination of the central nervous system (CNS), with periods of relapses and remissions.[1-7] More than 30 per 100,000 persons[1] in northern and southern latitudes are affected, with the greatest prevalence in northern Europe, northern United States, southern Australia[1,2], Canada, and New Zealand.[1] Women are affected 2-3 times more than men,[1,2] and caucasians are the most affected ethinicity.[1] MS is the leading cause of disability in young-adults and average age of onset is 30 years.[4] Events potentially eliciting exacerbations include infection, fever, stress from surgery, and anesthesia.[4,6]

Case Report

A 55-year-old female with an atraumatic left peroneal tendon rupture and aquired left heel varus presented for an elective left calcaneal osteotomy, left lateral ankle ligament reconstruction, left peroneal tendon reconstruction, left flexor tenolysis, and lower leg and ankle medial tendon repair. The patient was 152.5 cm and 47 kg, resulting in a body mass index of 17.2 kg/m^2. She was allergic to interferon beta-1b, and her past medical history included smoking daily (37 pack-years), left ankle sprain/strain with instability, left pes cavus with foot pain, headaches, osteoporosis, MS, neurogenic bladder, peripheral neuropathy, kyphosis, low back pain, and Eagle Syndrome. Her current medications were vitamins A, C, D3, and E, tizanidine, estradiol, gabapentin, methylphenidate, and natalizumab.

In the preoperative holding area, intravenous sedation was provided with fentanyl 50 mcg and midazolam 2 mg through a 20 gauge peripheral catheter in the left wrist. An ultrasound guided left popliteal nerve block with 0.25% bupivacaine with 1:400,000 epinephrine 25 mL and clonidine 41.75 mcg, was sterilely placed, followed by a left saphenous nerve block with 0.25% bupivacaine with 1:400,000 epinephrine 15 mL and clonidine 25.05 mcg, for postoperative pain control. Afterwards, the patient was transported to the operating room and assisted onto the operating room table. Standard monitors for heart rate, blood pressure, and continuous pulse oximetry were applied, and pre-oxygenation with 8 L/min was administered by facemask. Upon reaching an end-tidal O_2 of 90 mm Hg, intravenous induction occurred with propofol 160 mg. A size four, laryngeal mask airway (LMA) was placed without difficulty or trauma. LMA position was confirmed with bilateral breath sounds, symmetrical chest movement, and an end-tidal CO_2 of 36 mm Hg. Next, the patient was positioned in a right lateral decubitus position, with second confirmation of LMA position.

General anesthesia was maintained with sevoflurane 2% inspired concentration in a mixture of oxygen 1 L/min and air 1 L/min, allowing for spontaneous respirations throughout the two-hour procedure. Removal of the LMA occurred prior to moving the patient onto the stretcher, and the

patient received oxygen 4 L/min through a facemask. The patient maintained a patent, spontaneous airway throughout transport, and vital signs were stable upon arrival to the recovery unit. During scheduled postoperative follow-ups with the patient over a course of 2, 4, 6, and 12 weeks, the patient did not have an exacerbation of her MS, nor an onset of new symptoms related to MS.

Discussion

In addition to demyelination and inflammation, neurologic damage in MS also results from atrophy and scarring, ultimately leading to delayed, altered, or blocked nerve conduction.[1] However, a diagnosis of MS is not an absolute contraindication to utilize regional anesthesia[5]. Evidence and data involving MS and regional anesthetic techniques is scant and conflicting.[5,6] Therefore, other patient factors, advantages, and disadvantages need to be considered when choosing an anesthetic technique for MS patients.

The author and other anesthesia practitioners involved chose to perform peripheral nerve blocks for postoperative pain control. Although some studies have shown MS patients may have subclinical involvement of the peripheral nervous system[7], peripheral nerve blocks have been regarded as a theoretically safe approach to anesthesia because the location of local anesthetic (LA) is away from the primary sites of nerve demyelination and destruction in the CNS.[6] Traditionally, postoperative MS exacerbations from peripheral nerve block techniques occur less frequently than other neuraxial anesthetic approaches.[2]

Spinal anesthesia in particular, can be associated with postoperative exacerbations.[2] Theoretically, demyelination of the spinal cord makes the cord more susceptible to injury during a spinal anesthesic from the neurotoxicity caused by the LA.[2] This is known as the "double crush phenomenon," a widely accepted theory hypothesized by Upton and McComas in 1973.[8] They suggested that proximal injury in a nerve makes it more susceptible to injuries along distal sites of that same nerve.[2,8] In theory, MS would be considered the primary/proximal injury. This is important because local anesthetics potentially come in direct contact with MS lesions, which could cause the second (distal) injury.[6]

An additional option for surgical anesthesia was an epidural anesthetic. Epidural anesthetics also have decreased incidence of postoperative MS exacerbations due to a lower concentration of LA in the CNS.[2] However, this case was in an ambulatory setting, and the long-term benefits of postoperative pain control could be addressed through peripheral nerve blocks. Additionally, using a unilateral extremity nerve block avoids delays in discharge from PACU because patients do not need to wait to regain sensory and motor function below the waist (including voiding), before going home. Furthermore, bilateral lower extremity effects of an epidural anesthetic, such as unnecessarily anesthetizing the nonoperative limb, could be avoided through a unilateral extremity approach.

When using regional or neuraxial anesthesia, steps can be taken to minimize toxicity from LA. One way is by avoiding epinephrine which causes vasoconstriction and allows for longer exposure of the nerve to the LA.[3,6] Electric nerve stimulation is used as a safety measure to avoid intraneural injection in peripheral nerve blocks. Another additional safety measure is to use

ultrasound. Utilizing ultrasound can potentially decrease nerve injury caused by mechanical needle trauma and LA toxicity more than nerve stimulation alone.[3,9,10] It is important to avoid intraneural injection, as that increases LA concentration, increases exposure time to the LA, and decreases blood flow to the nerve.[6]

During care for this patient, an ultrasound probe was used to avoid direct intraneural injection. However, the anesthesia practitioners chose to use epinephrine because it was a peripheral nerve block. Also, the patient's disease was well-controlled, with the most recent exacerbation two years prior. Equally important, she had no history of postoperative exacerbations.

General anesthesia is the most common approach for patients with MS.[2] Perhaps this is because no specific interactions between MS and inhalational or intravenous anesthetic medications exist.[2] Additionally, there is no evidence suggesting drug superiority within the realm of general anesthesia in this population.[2,3]

In regards to muscle relaxants, extra caution needs to be taken. MS is an upper motor neuron disease.[1] Subsequently, these patients have a proliferation of extrajunctional nicotinic acetylcholine receptors in the skeletal muscles.[2,3] Because of this, potentially life-threatening hyperkalemic responses follow succinylcholine administration.[2] While the response to depolarizing muscle relaxants is consistent in MS, sensitivity to nondepolarizing muscle relaxants is inconsistent.[2,3] Therefore, the anesthesia practitioners decided to avoid using muscle relaxants. This, coupled with the short duration of the procedure, led to the decision of using an LMA to manage the airway.

By providing anesthesia for a patient with MS, the author learned that peripheral nerve blocks can be safely used in this disease, especially when additional precautions are taken to decrease nerve injury by LA. It was also discovered that evidence about peripheral and neuraxial anesthesia in this population is not consistent. If the same case presented itself again, the author would still choose to perform a peripheral nerve block for postoperative pain control, but would avoid using epinephrine as a precautionary measure to avoid nerve injury and postoperative exacerbation.

In closing, the data on neuraxial and regional anesthesia for MS is sparse and conflicting. The biggest concern with MS patients and anesthesic techniques is postoperative disease exacerbation.[3] Despite careful planning, the stress of surgery can potentially cause relapses of MS, regardless of the anesthetic approach utilized.[2]

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Trendelenburg Position, Pneumoperitoneum, and Airway Pressure

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Keywords: airway pressure, abdominal insufflation, pneumoperitoneum, robotic surgery, laparoscopic, Trendelenburg

Various physiologic changes are associated with abdominal insufflation and steep Trendelenburg positioning during laparoscopic robotic surgery. In particular, increased airway pressure is an alteration that may result from compacting of the lung bases and limited lung expandability secondary to increased intra-abdominal pressure.[1]

Case Report

A 45-year-old, 175 cm, 94 kg, female presented for a scheduled robotic total abdominal hysterectomy for removal of an ovarian neoplasm. The patient's medical history was significant for gastroesophageal reflux disease, autoimmune thyroiditis, polycystic ovarian syndrome, and obesity. The patient previously underwent gastric bypass surgery. The patient's current medications included: biotin, calcium citrate, cholecalciferol, cyanocobalamin, fluticasone, multivitamin, and pantoprazole.

The patient was transported to the operating room, noninvasive monitors were applied, and O_2 10 L/min was administered by face mask. Intravenous (IV) administration of induction agents

included fentanyl 100 mcg, lidocaine 100 mg, propofol 200 mg, and rocuronium 50 mg. The trachea was intubated with a 7.0 mm cuffed endotracheal tube and respiration was controlled by a mechanical ventilator. The ventilator was set on volume control mode with a tidal volume of 575 mL, positive end expiratory pressure (PEEP) of 5 cm H_2O, and a respiratory rate (RR) of 10 breaths per minute. General anesthesia was maintained with isoflurane 1% inspired concentration in a mixture of O_2 1 L/minute and air 1 L/minute.

The patient was positioned in steep Trendelenburg at 20°, and the abdomen was insufflated with CO_2 to reach an insufflation pressure of 15mm Hg. Neuromuscular relaxation was maintained throughout the case with incremental doses of rocuronium 10 mg IV in order to maintain one twitch with the neuromuscular stimulator. At the approximate time of abdominal insufflation, the patient's peak airway pressure acutely increased from 25 to 42 cm H_2O and SpO_2 decreased from 97% to 92%. Breath sounds were auscultated and found to be present and equal bilaterally. No disruptions or kinks were noted in the airway circuit tubing. The mechanical ventilator was changed to pressure control mode with the following settings: RR 12 breaths per minute, PEEP 5 cm H_2O, inspiratory: expiratory (I:E) ratio 1:3, inspiratory pressure 31 cm H_2O, and maximum pressure 37 cm H_2O.
With these ventilator settings, the patient maintained adequate tidal volumes of approximately 500 mL. The surgeon was made aware of the increased airway pressure, and insufflation pressure was decreased to 12mm Hg. The mechanical ventilator remained on pressure control mode until the end of the case. The ventilator was switched to pressure support mode after the neuromuscular blockade was antagonized by the administration of glycopyrrolate 0.8 mg and neostigmine 5 mg IV. Spontaneous ventilation was achieved, isoflurane was discontinued, and oxygen was increased to 10 L/minute. Once the patient had sufficient spontaneous respiratory effort with tidal volumes of 500 mL and followed commands, the endotracheal tube was removed. The patient was transported to the post anesthesia care unit on oxygen 10L/minute via facemask.

Discussion
Abdominal insufflation and steep Trendelenburg positioning during laparoscopic surgery pose various challenges in maintaining adequate intraoperative oxygenation and ventilation. Mechanical effects of pneumoperitoneum include reduced functional residual capacity (FRC), vital capacity, and pulmonary compliance, as well as increased intrathoracic pressure.[1] Additionally, steep Trendelenburg positioning further compounds these pulmonary function changes associated with pneumoperitoneum, leading to the development of atelectasis, impaired oxygenation and increased airway pressures.[1] In this particular case, the patient's body mass index of 30.7 kg/m^2 is another factor which further decreases FRC in steep Trendelenburg position as the diaphragm is displaced cephalad.[2] Given these pulmonary considerations, multiple factors affect the delivery of adequate oxygenation and ventilation to the mechanically ventilated patient undergoing laparoscopic surgery in steep Trendelenburg position. This patient experienced a significant increase in airway pressure and mild desaturation upon insufflation. In order to generate lower peak airway pressures, the ventilator was changed from volume control mode to pressure control mode.

The efficacy of various modes of ventilation in cases involving pneumoperitoneum and Trendelenburg position has been studied. Choi et al[3] compared the use of volume control

ventilation (VCV) and pressure control ventilation (PCV) in steep Trendelenburg position for laparoscopic prostatectomy and concluded PCV provides greater dynamic compliance and lower peak airway pressure than VCV.[3] However, Choi et al[3] noted patients in both the VCV and PVC groups experienced slight decreases in PaO_2, and this study concluded PCV did not improve oxygenation. Because pneumoperitoneum and Trendelenburg positioning cause reduction of lung volume and collapse of small airways, it is important to consider other strategies to enhance pulmonary gas exchange.[4]

Kim et al[4] studied the effects of prolonged inspiratory time as a measure to improve gas exchange in patients undergoing laparoscopic surgery in Trendelenburg position with VCV. The authors[4] determined that prolonged inspiratory time results in better gas exchange as compared with applying external PEEP of 5 cm H_2O. This study implemented an increased inspiratory time with an I:E ratio of 2:1, resulting in reduced airway dead space and improved arterial oxygenation and CO_2 elimination as compared with applying external PEEP.[4]

Kim et al[5] also examined the effects of prolonged I:E ratios on respiratory mechanics and oxygenation in patients undergoing laparoscopic robot-assisted prostatectomy. Kim et al[5] suggested that utilizing VCV with prolonged I:E ratio of 1:1 may be a superior method of ventilation as compared to PCV in the setting of pneumoperitoneum with steep Trendelenburg positioning. Although PCV has been shown to reduce peak airway pressure, there is variability in the tidal volume delivered to patients with PCV.[5] However, VCV with prolonged I:E ratio of 1:1 offers the advantage of guaranteed tidal volume delivery while also reducing peak airway pressure.[5]

As noted in the literature, there are several ventilatory methods to consider for use during laparoscopic robot assisted surgery with abdominal insufflation and steep Trendelenburg positioning. As discussed in this case report, PCV provided adequate ventilation for this patient while also limiting peak airway pressure. Adjusting the ventilator from VCV to PCV is an effective, evidence-based strategy to reduce airway pressure. Prolonging the I:E ratio with VCV is an alternative method that could have been implemented to further reduce airway pressure and improve oxygenation.

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Smith-Lemli-Opitz Syndrome and the Difficult Airway Algorithm

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Keywords: Smith-Lemli-Opitz Syndrome (SLOS), genetic disorder, congenital abnormalities, difficult airway, anesthetic management

Smith-Lemli-Opitz syndrome (SLOS) is a rare autosomal recessive syndrome which causes multiple congenital anomalies and cognitive dysfunction.[1] This syndrome results from a decrease in 7-dehydrocholesterol reductase (DHCR) activity, resulting in a cholesterol synthesis deficiency from a mutation in the DHCR7 gene.[2] Anesthesia for these patients can prove difficult due to several congenital anomalies seen with this syndrome. The presence of microcephaly, micrognathia, high arched palate, and cleft palate predispose these patients to difficult intubation.[3] This case study is presented to discuss the anesthesia care of a patient with SLOS and review the American Society of Anesthesiologists (ASA) difficult airway algorithm.

Case Report

A 16-day-old, 2.9 kg male with a history of poor feeding and failure to thrive, secondary to a primary diagnosis of SLOS, presented for a laparoscopic gastrostomy tube placement. The patient was born full-term and was admitted to the neonatal intensive care unit (NICU) with feeding difficulties and congenital anomalies. The patient's congenital anomalies included microcephaly, cleft palate, high arched hard palate, micrognathia, hypotonia, hypospadias, ambiguous genitalia, undescended testes, bifid scrotum, single palmar crease, and syndactyly of the second and third toes on both feet. Total cholesterol was < 50 mg/dL. An MRI revealed an immature appearing brain, and echocardiogram exposed a small patent foramen ovale (PFO). Genetic testing confirmed the diagnosis of SLOS.

Informed consent for general anesthesia with intravenous (IV) induction and endotracheal intubation was signed by the parents. A difficult airway was anticipated. The difficult airway cart, a fiberoptic bronchoscope (FOS) and a Glidescope (Verathon; Bothell, WA) were present in the operating room (OR). Malignant hyperthermia (MH) precautions were taken, after a literature review, with the installation of Vapor-Clean® charcoal filters, following manufacturer's protocol.

The patient was transported from the NICU to the OR. Standard monitors were applied and the patient was denitrogenated with O_2 at 10 L/min via face mask for 3 minutes. Anesthesia was induced with propofol 10 mg and initiation of a remifentanil infusion at 0.2 mg/kg/min. The ability to mask ventilate was confirmed, and rocuronium 1.5 mg was administered. A subsequent 1 mg dose of rocuronium was given 10 minutes later due to respiratory effort and limb movement.

Initial direct laryngoscopy (DL) with a Miller 1 blade revealed a Cormack-Lehane (CML) grade 3 view that remained unchanged with and without shoulder rolls and head elevation with a pillow. Attempts to intubate with a stylet and curved-tipped bougie also failed. The best view obtained was a grade 2b view with the use of a Glidescope. Attempts to direct the endotracheal tube through the glottis were still unsuccessful. Further attempts were made using the FOS alone, in conjunction with the Glidescope, as well as an intubating laryngeal mask airway. The pediatric surgeon then attempted intubation using a rigid bronchoscope, but was also unable to obtain an adequate view of the glottis. Mask ventilation was possible throughout. However, with repeated laryngeal instrumentation it became increasingly difficult, improving only with the use of an oral airway.

Each attempt was limited by the patient's poor pulmonary reserve. The patient remained stable throughout, and no episodes of bradycardia or severe desaturation occurred. Thick, copious secretions were suctioned repeatedly. After multiple attempts at intubation with a variety of techniques and 3 different anesthesia practitioners, in accordance with the ASA difficult airway algorithm, a decision was made to cancel the procedure. Dexamethasone 0.5 mg IV was given to reduce airway swelling. Mask ventilation was continued and neuromuscular blockade antagonized with glycopyrrolate 0.275 mg and neostigmine 0.3 mg in divided doses after the return of diaphragmatic activity. Upon return of spontaneous respirations, muscular tone, and ability to protect his airway, the patient was transported with monitoring back to the NICU.

Discussion

Smith-Lemli-Opitz syndrome, first described in 1964, is a rare autosomal recessive disorder of cholesterol synthesis, with reported estimated incidence between 1:10,000 to 1:80,000.[1-,4] This syndrome results from a mutation in the DHCR7 gene, causing a decrease in 7-dehydrocholesterol (7DHC) reductase activity, the enzyme responsible for conversion of 7DHC to cholesterol.[1,2] SLOS affects males and females with equal preponderance.[4] Populations of central and northern European origin see the highest prevalence of SLOS, but it is extremely rare in African and Asian populations.[1] In the Caucasian population of North America, the incidence of SLOS falls behind cystic fibrosis and phenylketonuria.[5] It is also extremely likely that the overall prevalence of this disorder is underreported due to mild cases of the disease with subtle phenotypes as well as severe cases of SLOS causing fetal demise. The severity of this disorder and the congenital defects correspond to the severity of cholesterol synthesis dysfunction.[1]

The anesthesia implications of this syndrome stem from the multiple congenital abnormalities that affect the airway of the SLOS patient. The presence of microcephaly, micrognathia, high arched palate, a small abnormally stiff tongue and cleft palate predispose these patients to difficult intubation.[3,5] These patients may have increased aggressive behavior that may be

refractory to the administration of sedative medications.[5] Congenital heart defects affect approximately 50% of SLOS patients, and prior to the administration of anesthesia, the practitioner should ensure that an echocardiogram is available to assess the patient's cardiac status. Cardiac abnormalities commonly seen with SLOS include tetralogy of Fallot, ventricular septal defects, and anomalous pulmonary venous return.[1,5] The echocardiogram from the patient presented in this case study revealed only a small PFO. Therefore, careful attention was paid to meticulously remove air from the IV tubing.

Two isolated case reports demonstrated muscle rigidity after volatile anesthetic in SLOS patients. In these reports, one patient was given succinylcholine but the other was not.[5] A third report of a child developing hyperthermia without muscle rigidity was also reported.[3] Research into the anesthesia care of SLOS patients prior to the start of the presented case study directed the anesthesia care team to employ MH precautions before the induction of anesthesia. Despite two case reports of muscle rigidity in SLOS patients after receiving a volatile anesthetic, according to Baum and O'Flaherty,[5] this syndrome is not associated with MH and precautions in this patient were not necessary.

According to Belanger and Kossick,[7] a difficult intubation occurs when an experienced anesthesia practitioner requires 3 or more attempts at endotracheal tube placement with or without rescue adjuncts. They report that difficult airway situations occur more frequently in the pediatric population than adults. A large retrospective study analyzed intubations in 8,434 pediatric patients, reporting that the incidence of difficult intubation was 1.35%.[8] Further analysis by these researchers suggests that patients less than a year old have higher rates of difficult airway, those with a CML grade III and IV, at 4.7%.[8] The anatomical structure of the neonate's airway is different from that of adults. The neonate's larynx is located at C3-4 or as high as C2-3, which may have prevented tracheal intubation in this case.[7] In addition, micrognathia causes the proportionally large tongue to be displaced posteriorly in the oropharynx, increasing the difficulty to adequately view the vocal cords with DL.[8] An update of the ASA difficult airway algorithm was most recently published in 2013.[6] It outlines the steps to be taken when an anesthetist encounters a patient with a difficult airway. Preparation for a difficult airway includes a thorough evaluation, availability of difficult airway equipment, assigning assistance with the patient, informing the patient (or guardian) of the difficult airway possibility, preoxygenation prior to airway manipulation, and administering supplemental O_2 throughout the airway management.[6]

Presented with this scenario again, it would be prudent of the anesthesia team to initially approach tracheal intubation with the FOS. Airway manipulation was attempted multiple times before the FOS was utilized. Due to this, severe swelling was apparent, as evidenced by the increased difficulty to mask ventilate. If the FOS had been used as the first line device, there could have been less swelling in the airway and the view would likely not have been as obscured. It is also important to remember that if the ability to ventilate is lost, the anesthesia practitioner should consider provisions for a surgical airway without delay in the difficult intubation scenario.[6] In the case presented, the ability to ventilate was always maintained, and the ASA difficult airway algorithm was followed.

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Anesthetic Management of the Detrimental Effects of Protamine

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Keywords: Deep Hypothermic Circulatory Arrest, Jehovah's Witness, Cardio-pulmonary bypass, Protamine, Dissecting Aortic Aneurysm

Type One Dissecting Aortic Aneurysms require unique surgical intervention involving cardio-pulmonary bypass (CPB), profound hypothermia, and rewarming. Protamine sulfate is needed to neutralize and reverse the activity of the large amount of heparin given throughout the case. Administration of protamine is associated with a variety of hemodynamic changes ranging from mild hypotension to significant elevated pulmonary artery pressures and decreased systemic vascular resistance.[1] Incidence of protamine allergy was reported as 0.28-2.6%.[2] Populations at risk for protamine allergy include, patients with salmon allergies, previous vasectomy, and prior exposure to protamine medications.[2]

Case Report

A 53-year-old male, measuring 175 cm and weighing 79 kg, presented with acute chest pain. He had a past medical history of hypertension, non-insulin dependent diabetes mellitus, and no past

surgical history or allergies. The patient's medications included metformin, pravastatin, and amlodipine with benazepril. Following a normal electrocardiogram, a computed tomography noted a DeBakey type one dissecting aortic aneurysm; esmolol and nitroprusside infusions were immediately initiated.

The patient was a practicing Jehovah's Witness who refused whole blood and red blood cell transfusion, but consented to the use of platelets, fresh frozen plasma (FFP) and an uninterrupted CPB circuit. Standard anesthesia monitors were applied, two peripheral intravenous (IV) lines were in position, and a radial arterial line and femoral central venous line were inserted. An 8.0 endotracheal tube was placed and confirmed with exhaled carbon dioxide following an uneventful IV induction with midazolam 7 mg, fentanyl 500 mcg, and vecuronium 10 mg. Cefazolin 2 g and aminocaproic acid infusion was initiated following a 10 g bolus. Anesthesia was maintained with sevoflurane 1% expired concentration in oxygen 2L/min.

Following median sternotomy, heparin 40,000 units were administered and cardiopulmonary bypass was established. Cooling was initiated to 24°C and circulatory arrest ensued for 24 minutes following administration of hyperkalemic cardioplegia. Nearing the completion of the surgical procedure, circulation was restarted following two administrations of internal defibrillation, rewarming occurred over 1.75 hours to 36.2°C, at which time the patient was successfully weaned from CPB and decannulated. Intraoperative blood salvage was returned to the patient.

Upon conclusion of the CPB, protamine sulfate 400 mg slow IV infusion was administered for an activated clotting time (ACT) of 503 seconds. Following repeat ACT, five additional boluses of protamine totaling 1,080 mg were given until an ACT of 142 seconds was obtained. During the administration, the patient's blood pressure (BP) dropped from 130/80 to 68/51 mm Hg, to a low of 36/32 mm Hg, with a heart rate externally paced between 100-115/min. Multiple phenylephrine 200 mcg and norepinephrine 16mcg boluses were administered, a phenylephrine infusion was started, and an epinephrine infusion was incrementally increased from 2 to 6 mcg/min with no notable improvement. There was no significant cardiac dysfunction noted on transesophageal echo. Direct cardiac massage was immediately initiated by the surgeon while epinephrine 1mg IV was administered. The patient's BP increased to 222/138 mm Hg, at which time acute blood loss was estimated at 2000 mL into a clotted cell saver. Sevoflurane was pressurized to 8% and propofol 50 mg was administered, returning the BP to 125/95 mm Hg. Albumin 5% 500 mL, 3 units of FFP, and 1 unit of platelets were administered.

The chest was surgically closed and the patient was transported to the intensive care unit intubated. Epinephrine and phenylephrine infusions were continued IV to maintain a systolic BP greater than 90 mm Hg. The patient's endotracheal tube was removed on post-operative day one.

Discussion

Protamine sulfate is used in most surgical procedures that require large doses of heparin to ensure adequacy of anticoagulation. Protamine sulfate is a positively charged polypeptide extracted from the sperm of salmon that is rich in basic proteins and can thus neutralize the acidic heparin and reverse its activity.[1]

Intravenous administration of protamine sulfate is associated with many adverse hemodynamic changes, such as, hypotension, which is often directly related to the rate of administration, local skin rash, bronchospasm, transient pulmonary hypertension, cardiovascular collapse, and death.[2,3] Although manufacturer guidelines recommend protamine to be administered at a rate not to exceed 5 mg/min, it is often administered much quicker in the clinical environment.

Many of the adverse effects of protamine can be classified as side effects that are directly related to rate of administration. However, anaphylactic reaction resulting in an overproduction of nitric oxide are also well reported.[1] Major adverse reactions related to protamine administration occur with an incidence of 0.28%-2.6% of all cardiac surgical procedures, and are linked to an overall mortality of 2-2.6%.[2] The reported incidence of anaphylactic reactions to protamine in the surgical population independent of surgical procedure range from 0.06%-10.6%.[1] Vasoplegic syndrome is another commonly documented reaction associated with protamine sulfate administration and results in a state of endothelial dysregulation which leads to profound vasodilation and hypotension that is refractory to vasopressors.[1]

Patients at increased risk for protamine reactions are those with fish allergies, previous exposure to protamine, decreased left ventricular function, history of non-protamine medical allergies, previous neutral protamine zinc (NPH) insulin use, history of a vasectomy (due to anti-sperm antibodies), and taking angiotensin-converting enzyme (ACE)- inhibitor therapy or calcium channel blockers.[1,4]

Activation of the complementary system and degranulation of mast cells occurs ten minutes after administration of protamine sulfate, and thus anasarca, a notable rash, audible wheezing, bronchospasm, and increased plateau airway pressures[2] can differentiate a true protamine reaction from mere side effects. Signs of vasoplegic syndrome are elevated cardiac index, low systemic vascular resistance, a central venous pressure of less than 5mm Hg and hypotension that does not typically respond to high doses of vasopressors or fluid therapy.[1] This case was dissimilar, in that CVP was elevated to 30 mm Hg, no rash or respiratory systems were present, and hypotension was eventually responsive to vasopressor support.

Treatments of protamine reactions are similar to that of anaphylaxis. Oxygen, albuterol, IV fluids, diphenhydramine, methoxamine, methylprednisolone, and epinephrine IM can all be used to combat the effects of bronchospasm and extreme hypotension.[2] If vasoplegia is noted and hypotension is not responsive to initial pharmacologic management, methylene blue 1-2mg/kg can be administered IV.[1] Methylene blue competitively inhibits the enzyme cyclic guanosine monophosphate (cGMP), which indirectly reduces the response of vascular endothelium to nitric oxide, and thus opposes massive vasodilation seen with protamine reactions.[1]

There are a few additional factors that could have caused the profound hypotension during this aortic aneurysm repair. Patient rewarming can lead to significant vasodilation and compression of the heart by sternal and chest closure could have caused inadequate preload and hypotension. Although the patient had never had a vasectomy or prior exposure to protamine or NPH, he was on both an ACE-inhibitor and calcium channel blocker, which are risk factors for protamine reactions.[1]

The amount of heparin in autologous blood transfusions vary dependent on transfusion volume, concentration, and drip rate of the anticoagulant heparin solution, and can worsen coagulopathy [5] leading to a higher ACT and need for a larger dose of protamine. Each bolus of protamine was diluted in normal saline and infused by gravity over about five minutes. The rate of administration was significantly quicker than the recommended guidelines, and perhaps the hypotension was related to the repeated doses of protamine. While hypotensive, the patient's SpO_2 remained at baseline, no wheezing or uticaria was noted, and the carbon dioxide waveform was unchanged, acknowledging no suspected bronchospasm. The patient was mechanically paced, and thus no bradycardia was noted.

A pulmonary artery catheter would have been useful in the diagnosis and management of his protamine reaction. A 1mg "test dose" of protamine or a slow initiation of the first dose of protamine may have also been beneficial for this patient.[3] In lieu of absent respiratory or skin issues, the significant hypotension was managed as a side effect of protamine administration, and not a true anaphylactoid reaction. He remained inhaling100% oxygen and was responsive to treatment with vasopressors such as epinephrine, phenylephrine, norepinephrine, and direct cardiac massage.

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Intraoperative Management of Polycythemia in Adrenocortical Carcinoma

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Keywords: anesthetic management, adrenocortical carcinoma, Cushing's syndrome, polycythemia, phlebotomy, adrenalectomy

Adrenocortical carcinomas are extremely rare with an incidence of 1-2 per million population each year.[1] These large carcinomas present with excessive hormone production leading to Cushing's syndrome.[1] Cushing's syndrome is the hallmark of the carcinoma's malignant process and often complicates anesthetic management.[1] One of the major anesthetic implications for adrenocortical carcinomas and the resultant Cushing's syndrome is management of severe polycythemia and increased blood viscosity. Anesthetic management should be emphasized on decreasing the risk of thrombosis development and tissue hypoxia. Phlebotomy has been shown to significantly improve patient outcomes by reducing the morbidity and mortality related to polycythemia.[2-4]

Case Report

A 39-year old, super morbidly obese Caucasian female with body mass index of 40 kg/m^2 presented for a right open adrenalectomy for adrenocortical carcinoma. Medical history included mild mitral regurgitation, current smoker, controlled gastro-esophageal reflux and Cushing's syndrome. Preoperative labs revealed cortisol level of 17.6 mcg/dL with a 24 hour urine cortisol level of 167.4 mcg. Her testosterone level was 86.1 ng/dL with dehydroepiandrosterone (DHEA) level of 1345 mcg/dL. Preoperative hemoglobin and hematocrit showed 19.3 g/dL and 57.9% respectively, and her erythropoietin (EPO) level was 28 mU/mL. Abdominal MRI demonstrated an 8.3x9.3x6.5cm irregular margined right adrenal mass. Current medications included: metoprolol, amlodipine, lisinopril and chlorthalidone. Preoperative vital signs included blood pressure of 184/104 mm Hg, heart rate 74/min and SpO_2 of 96% on room air.

The patient received midazolam 2 mg preoperatively and was transported to the operating room where noninvasive monitors were applied. Oxygen 8L/min was administered by face mask for awake arterial line placement. Preinduction blood pressure was 147/85 mm Hg, heart rate 70/min, and SpO_2 100%. General anesthesia was induced with fentanyl 250 mcg, lidocaine 100 mg, and propofol 200 mg. Mask ventilation was confirmed and muscle relaxation was established with rocuronium 50 mg. The trachea was intubated with a 7.0 mm endotracheal tube, placement confirmed with bilateral breath sounds, chest rise and three end tidal CO_2 readings, and mechanical ventilation initiated. Two additional intravenous lines were then established.

General anesthesia was maintained with sevoflurane 2% in a mixture of air 1 L/min and O_2 1 L/min. Muscle relaxation was maintained with a rocuronium drip. After ten minutes of mechanical ventilation the patient had SpO_2 of 92%, recruitment breaths were given, flows adjusted to air 0.5 L/min and O_2 1.5 L/min which resulted in SpO_2 of 100%. Preincision the patient was given Lactated Ringers 1.5 L bolus. Upon completion of the bolus a hemoglobin was sent indicating a level of 16.2 g/dL. Phlebotomy was then initiated and 600 mls of whole blood

was removed. The patient had an estimated surgical blood loss of 1000 mls. After tumor extraction laboratory set was sent which indicated a hemoglobin of 15.2 g/dL, hematocrit of 50.1% and lactate 2.4 mmol/L.

Upon completion of the procedure, neuromuscular blockade was antagonized and spontaneous ventilation occurred. Respiratory extubation criteria were met and the patient was extubated and placed on O_2 6 L/min via face mask. Prior to transportation to the ICU a set of labs were sent. Hemoglobin value was 13.7 g/dL, hematocrit 40.2% and lactate 3.3 mmol/L. The patient received a total of 6.5 L crystalloids with an estimated urine output of 950 mL.

Discussion

Adrenocortical carcinomas are often incidentally diagnosed during the evaluation for the cause of Cushing's syndrome in patients.[1] Cushing's syndrome, due to adrenocortical carcinomas, results from the malignancy's excessive production of the adrenal cortex hormones which include glucocorticoids, mineralocorticoids and androgens.[1,5]

Cortisol is the most important glucocorticoid produced by the adrenal cortex and is essential in the maintenance of systemic blood pressure, gluconeogenesis, and inhibition of glucose uptake by cells.[5] The major mineralocorticoid produced is aldosterone which plays a major role in potassium excretion and sodium reabsorption from the nephron tubules.[5] Androgens produced by the cortex include DHEA and testosterone.[5] The imbalance of these hormones results in the characteristic Cushing's syndrome presentation: systemic hypertension, glucose intolerance, electrolyte abnormalities, amenorrhea, hirsutism, frequent bruising, weakness and central obesity.[1,5]

Anesthetic considerations for the management of Cushing's syndrome resulting from adrenocortical carcinoma include preoperative and intraoperative management of systemic blood pressure, electrolyte balance and blood glucose control.[5] The patient's electrolytes and glucose levels remained within normal limits throughout the perioperative period. Intraoperative management of hypertension included a systolic blood pressure range of 120-130 mm Hg. This was accomplished with the use of opioids and titration of the Sevoflurane concentration with surgical stimulation. Sevoflurane was chosen as the maintenance anesthetic due to the low blood solubility which allowed for rapid and precise titration, and decreases in systemic vascular resistance without the sympathetic stimulation seen with rapid desflurane titration.[6]

Anesthetic case management was further complicated by the patient's elevated EPO level and resultant polycythemia. Reports of adrenocortical carcinomas directly secreting EPO are extremely rare.[1,7] Postsurgical analysis of the tumor did not show EPO secreting cells; however, increased androgen production from adrenocortical carcinomas may lead to direct stimulation of EPO production and bone marrow stem cell proliferation.[7] Morbid obesity, as seen in Cushing's syndrome, is also associated with polycythemia related to an increased production of EPO to compensate for chronic hypoventilation.[2,5,8]

The cause of the patient's increased EPO is unclear; however, similarities exist with polycythemia vera which results in increased hematocrit and platelets due to bone marrow stem

cell proliferation.[2-4,9] Comprehensive research on the effects, goals and management of polycythemia vera was reviewed and management of this case was based on the research. A special emphasis was placed on hematocrit control. Treatment of polycythemia, no matter the cause, is targeted at controlling symptoms and decreasing complications through phlebotomy and correcting the underlying cause including surgical resection of the tumor.[1,2-4,9]

Polycythemia is an increase in hematocrit which is the primary determinant in blood viscosity.[2-5,9] Blood viscosity exponentially increases at hematocrit level of 55-60%, resulting in critical ischemia and thrombosis development most often manifested as hypoxia, stroke, myocardial infarction, and end organ damage.[2] This may explain the SpO_2 of 92% with fiO2 of 60%. A hematocrit of 33-36% allows for maximal tissue oxygen delivery.[2] A large prospective study determined that a hematocrit of 45-50% resulted in no significant increase in thrombosis development, stroke or cardiovascular collapse in the management of polycythemia vera.[9] However, recommendations from the European LeaukemiaNet include management of hematocrit to a goal of less than 45%.[3] This recommendation is backed by a landmark study completed by the CYTO-PV Collaborative Group, a leader in polycythemia vera research, which found that a hematocrit range of 45-50% was associated with four times the death rate from a major thrombotic event than a goal hematocrit of less than 45%.[4] Additionally, the researchers of the group found that a higher hematocrit (45-50%) resulted in higher rates of deep vein thrombosis and stroke.[4] Current management recommendations for hematocrit control in polycythemia vera include phlebotomy with a goal hematocrit of less than 45%.[2-4]

Phlebotomy was initiated in the patient after reviewing current research recommendations. It was calculated that an allowable blood loss of 1250-1750 mL would reach the goal hematocrit of 40-45%. This calculation was reached from the surgeon's expected surgical blood loss of 500 mL, and the estimated blood volume for the patient. Obese individuals have increased total blood volume but a decreased volume-to-weight basis, with this in mind, the anesthesia team used the morbid obesity calculation of 50 ml/kg to reach an estimated blood volume of 5650 mL.[8] From the surgeon's expected surgical blood loss and calculated allowable blood loss it was determined to phlebotomize 600 mL after intraoperative hemodilution. Risks associated with phlebotomy include infection, hypotension, and decreased oxygen carrying capacity leading to hypoxia. With this in mind, phlebotomy was initiated aseptically, blood loss was replaced with crystalloids and the patient's phlebotomized blood was readily available for re-transfusion if required.

The patient's polycythemia and increased blood viscosity may have developed due to the excessive androgen production and morbid obesity which resulted in an increased EPO level. Elevated EPO levels lead to increased hematocrit levels and a hematocrit greater than 45% carries four times the risk of mortality due to thrombosis development and tissue hypoxia than a hematocrit of less than 45%.[4] Phlebotomy was a reasonable management option in the patient's severe polycythemia in order to significantly decrease her perioperative morbidity and mortality.

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Emergency Care of Patients with Cardiovascular Implantable Electronic Devices

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Keywords: cardiovascular implantable electronic devices, CIED, implantable cardioverter defibrillators, ICD, pacemaker, emergency surgery, magnet, acidosis

More than 400,000 pacemakers and 120,000 automated cardioverter defibrillators (ICDs) are implanted each year in the United States and the presence of such devices may serve as a general marker for patients with increased risk of cardiovascular or general medical complications perioperatively.[1] Safe and effective care for patients with cardiovascular implantable electronic devices (CIEDs) should include a thorough preoperative assessment of the device. Urgent and emergent situations may preclude the opportunity to evaluate CIEDs, however, and the anesthesia professional should be prepared for potential device malfunction. The following case report illustrates how adverse events can arise when urgent and emergent scenarios impede routine device evaluation.

Case Report

An 81-year-old female (81.7 kg, 170 cm) with acute abdominal pain, refractory hypotension, and oliguria was scheduled for emergency laparotomy for suspected ruptured diverticuli. Upon hand-off in the intensive care unit (ICU) the patient's systolic blood pressure (SBP) measured 60 mm Hg and the electrocardiogram (ECG) rhythm demonstrated junctional bradycardia at a rate of 45/min. The patient's trachea had been intubated just prior to the anesthesia practitioners' arrival.

Post-intubation hypotension was being treated with a phenylephrine infusion. The patient's medical history included coronary artery disease (CAD), previous myocardial infarction with coronary artery bypass grafting, atrial fibrillation, ICD placement, hypertension, hyperlipidemia, gastric reflux, and diverticulitis. The patient's medications included digoxin, nebivolol, simvastatin, enoxaparin, and famotidine. No written chart was immediately available.

Two bolus doses of vasopressin, 0.4 units each, and one bolus dose of epinephrine, 10mcg, were administered to increase the patient's SBP to 80 mm Hg. The phenylephrine infusion was discontinued and a norepinephrine infusion was initiated at 0.15mcg/kg/min. After the patient's SBP was sustained at 80 mm Hg or above for at least five minutes, the patient was transported to the operating room. It was not possible to interrogate the CIED at this time. The patient was positioned on the operating room table, standard monitors were applied, transcutaneous pacing and defibrillation pads were positioned on the anterior and posterior aspects of the patient's chest. General anesthesia was induced with midazolam 2 mg, ketamine 10 mg, rocuronium 20 mg, and sevoflurane 0.6%. A magnet was placed over the patient's CIED generator after it was identified in the right subclavian area by palpation. The patient's heart rate increased from 65 to 80/min within a minute of magnet placement, then gradually increased to 100/min over the course of the next 2 to 3 minutes. The patient's SBP increased to 90 mm Hg in association with the rise in heart rate. An arterial blood gas sample revealed a lactate of 3.7 mmol/L and pH 7.2. The magnet was unintentionally dislodged from the right subclavian area during surgical site preparation. The patient's heart rate suddenly fell to 60/min. The magnet was replaced over the center of the CIED and the patient's heart rate returned to 100/min.

One unit of fresh frozen plasma and 1800 mL of crystalloid fluid were administered throughout the case. After surgical repair the patient was prepared for transport back to the ICU. During preparation for transport the magnet was unintentionally moved approximately 0.5 cm and the patient's heart rate and blood pressure decreased. The magnet was again secured over the CIED and the patient was transported to the ICU with norepinephrine 0.15 mcg/kg/min infusing, respirations assisted by bag valve mask through an endotracheal tube, and on a transport monitor. A dedicated anesthesia practitioner ensured magnet placement over the center of the CIED while the patient was transported. Bedside monitors were placed on the patient upon arrival to the ICU and ECG monitoring continued uninterrupted.

Discussion

A thorough preoperative evaluation is necessary in order to safely and effectively care for patients with CIEDs.[2] Situations will arise, however, when an evaluation and consultation with the CIED management team cannot be obtained, such as in urgent or emergent surgery. In situations where device interrogation is not possible, perioperative device malfunction or failure should be anticipated.[3]

In emergency cases, best attempts to discern information about the device should be made by interrogating the patient, utilizing medical records or the patient's device registration card, or by obtaining a chest radiograph.[2,3] The presence of a CIED was known in this case; however the indication for the device was not. The patient could not be interrogated due to her altered mental status. A central venous catheter was in place and thus a chest radiograph may have previously

been obtained, but the image was not located in the patient's record due to the urgency of the situation.

The anesthesia professionals acted in accordance with recommendations for a patient with a CIED undergoing emergent/urgent surgery. Arterial line waveform monitoring was utilized to confirm the presence of an adequate pulse and transcutaneous pacing/defibrillation pads were positioned on the patient in an anterior-posterior orientation.[2-4] The CIED was evaluated postoperatively and the patient remained on continuous ECG monitoring throughout the perioperative period.[2,3] A magnet was placed over the CIED based on the unconfirmed verbal report that the device was an ICD. A magnet was placed over the device with the intention of suppressing tachy-arrhythmia therapy, as is recommended for the patient with an ICD presenting for emergency surgery.[2]

The anesthesia professionals made the assumption that an ICD had been placed for ventricular failure as the patient presented with CAD and was currently prescribed digoxin. Over one million hospitalizations and 58,000 deaths annually result from heart failure due to impaired left ventricle systolic function.[1] ICD implantation is designated for prevention of primary or secondary cardiac death and is indicated in patients with CAD and cardiomyopathies with an ejection fraction less than 35%.[3,4]

The perioperative events suggest that the patient's CIED was likely a pacemaker and not an ICD. The patient's medical history of atrial fibrillation could have been cause for pacemaker placement. Chronic atrial fibrillation with a slow ventricular response, as was demonstrated during preoperative assessment of the patient, is a common reason to place a pacemaker with VVI programming.[3] The code VVI describes ventricular pacing that is inhibited if the device senses intrinsic electrical activity above the set rate in the ventricle.

The patient's heart demonstrated a paced rate of 100/min when the magnet was placed over the CIED. Magnet application over pacemakers causes asynchronous pacing.[3-5] Magnet application over an ICD suspends anti-tachycardia therapy but does not initiate asynchronous pacing; asynchronous pacing can only be accomplished with ICD reprogramming.[3-5] Magnet placement over this CIED resulted in asynchronous pacing, therefore indicating that the device was a pacemaker.

Physiological changes that occur secondary to anesthesia, surgery, and illness can induce unexpected CIED responses.[6] Metabolic abnormalities can result in pacemaker failure to capture or sense.[3,7] The patient's arterial blood gas sample revealed a lactate of 3.7 mmol/L and pH 7.2, consistent with acidosis. Decreased cellular pH has been shown to reduce myofilament calcium sensitivity in animal models studied by Varian, Raman, and Janssen.[8] Schotola et al. demonstrated that mild metabolic acidosis (defined as pH 7.20) impaired contractility in cardiac tissue obtained from patients with heart failure, cardiomyopathy, and ejection fractions below 30%.[9] Specific beta-adrenergic receptors displayed a reduced binding affinity to inotropes and vasopressors in the context of acidosis during an in-vitro study by Modest and Butterworth.[10] Acidosis may explain the presumed failure of the pacemaker to fire prior to magnet placement. A decreased pH may have also contributed to impaired cardiac function that resulted in

compromised hemodynamics. Recommended treatment of CIED malfunction in the context of electrolyte and metabolic abnormalities involves correcting the underlying cause.[4]

The patient's device was interrogated several days postoperatively and was found to be a dual-chamber pacemaker programmed to trigger when the intrinsic heart rate fell below 50/min. At the time of interrogation the device functioned properly, although it cannot be determined whether the device functioned properly during the perioperative period. Potential device failure during the perioperative period should be considered as acidosis is known to interfere with proper device function.[4] The patient's device was reprogrammed during the interrogation to trigger when the intrinsic heart rate fell below 90/min in order to optimize the patient's hemodynamics.

The urgency of the case prevented the anesthesia practitioners from obtaining the routine recommended device evaluation. Although the presence of a CIED was known, the indication for the device was not. The preoperative verbal report included ICD placement as part of the patient's history. The patient's history included disease processes and medications consistent with heart failure, a condition that can prompt ICD placement, as well as a history of atrial fibrillation which necessitated pacemaker placement for this patient. Magnet placement over the patient's pacemaker initiated asynchronous pacing that improved hemodynamic stability. Understanding of CIED indication and function is paramount to interpreting the perioperative events in this case. This case study reinforces that knowledge of CIED operation and management is imperative for the anesthesia professional.

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Transcatheter Aortic Valve Replacement with Dexmedetomidine

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Keywords: anesthesia, aortic valve replacement, deep sedation, dexmedetomidine, transcatheter aortic valve replacement, fascia iliaca nerve block, regional anesthesia, percutaneous aortic valve replacement

Aortic stenosis is the most common diagnosed valve heart disease present in 2-7% of those over the age of 65 years; severe untreated disease is associated with a two-year mortality rate of 50-60%. [1] Surgical aortic valve replacement is considered conventional treatment, however, 33% of those diagnosed older than 75 years are not considered candidates.[1] The U.S. Food and Drug Administration approved Transcatheter Aortic Valve Replacement (TAVR) in 2011 as an alternative option.[2] TAVR is a minimally invasive procedure and while typically performed with general anesthesia, favorable outcomes have been achieved when performed with sedation techniques.[3-4] This case report describes sedation anesthesia for an octogenarian undergoing a TAVR.

Case Report

An 83-year-old, 160 cm, 75 kg female diagnosed with severe aortic stenosis presented for a TAVR. Past medical history was significant for recent syncope, worsening chronic systolic heart failure according to the NYHA, carotid artery stenosis, coronary artery disease, myocardial infarction, 7.25-pack years smoking history, and severe emphysema. Past surgical history included right carotid endarterectomy, esophagogastroduodenoscopy and dilation, hysterectomy, and cataract extraction. Current prescribed medications included: spironolactone, aspirin, citalopram, amlodipine, clonazepam, tiotropium, fluticasone, and omeprazole.

A preoperative echocardiogram estimated the myocardial ejection fraction at 65% with a mean aortic valve gradient of 45 mm Hg and an aortic valve area of 0.5 cm^2. A 12 lead electrocardiogram (ECG) revealed a left ventricular hypertrophy pattern. The computed

tomography angiogram revealed non-obstructive coronary artery disease. Chest radiograph was positive for chronic emphysema and prominent interstitial lung markings with bilateral pulmonary lung nodules. Diffusion capacity of the lung for carbon monoxide was only 5.34 ml/min/mm Hg, or 23% of the predicted value of 22.91 ml/min/mm Hg, which is considered an abnormal diffusion deficit indicating a severe loss of functional alveolar capillary surface. The NYHA functional status was estimated less than 4 metabolic equivalents (METs). All laboratory blood tests were within normal reference ranges.

Due to the severe cardiovascular and pulmonary co-morbid state, the risk benefit ratio of a general anesthesia technique was not deemed favorable, and the anesthetic of choice was intravenous (IV) sedation with maintenance of spontaneous ventilation. Additionally, a fascia iliaca compartment block was performed to provide analgesia and facilitate immobility in the area of catheter insertion - the femoral artery.

Preoperative vital signs were blood pressure 137/67, pulse of 85/min, $Sp0_2$ 100% with 3 L/min nasal cannula, 16 breaths per minute and temperature 37 °C. An ultrasound-guided fascia iliaca compartment nerve block was performed with 40 mLof 2% lidocaine and 1:200,000 epinephrine diluted to 50 mLwith normal saline using sterile technique and was tolerated well. In the operating room, a Dexmedetomidine IV infusion was initiated at 0.7 mcg/kg/hr. A right radial arterial catheter was inserted under sterile technique. After confirming patient comfort verbally,, the surgeon placed a 6-French sheath in to the right femoral artery and a 7-French sheath in to the right femoral vein. A transvenous pacemaker was placed via the right femoral vein. Heparin 10,000 units IV was administered. Blood pressure was maintained within 20% of baseline during the TAVR with IV bolus doses of ephedrine 5 mg, phenylephrine 160 mcg in two 80mcg doses, followed by vasopressin 1 unit, and a norepinephrine IV infusion of 3-5 mcg/min. The Dexmedetomidine infusion was decreased to 0.5 mcg/kg/hr with continued patient comfort noted. Heart rate ranged between 70 and 80 beats per minute and rhythm was normal sinus throughout with the exception of valve deployment.

The Edwards Sapien 23 mm aortic valve (Edwards Lifesciences Corporation, Irvine, CA) was deployed during rapid ventricular pacing at 180/min. A brief period of unconsciousness occurred during valve deployment occurred however spontaneous recovery of baseline consciousness was noted without intervention. The transvalvular gradient decreased from 45 mm Hg to 0 mm Hg with mild aortic regurgitation after the new valve was deployed. At the end of the procedure heparinization was neutralized with 200 mg of protamine. When hemostasis was achieved the Dexmedetomidine infusion was discontinued .The patient was directly transported to the intensive care unit in stable condition. Patient was discharged on postoperative day 5 to a subacute rehabilitation facility.

Discussion

Aortic stenosis is an obstruction of blood flow through the aortic valve during systole usually due to calcification as a result of the aging process.[1] As a result, the left ventricle experiences pressure overload and compensates with increased systolic blood pressure, ejection time, and ventricular mass. Over time, increased oxygen demand and myocardial ischemia occur with left ventricle concentric hypertrophy developing and a decreased ejection fraction.[1] Left untreated,

the three year survival rate for severe symptomatic aortic stenosis is 30%; the gold standard is surgical aortic valve replacement.[1] While the TAVR surgical technique is relatively new, studies suggest it is "noninferior" to traditional open technique in patients with severe aortic stenosis.[2] The patient was not a candidate for the traditional open approach due to her advanced age, stroke history, frailty, and COPD. TAVR is performed by accessing the aortic annulus with a catheter placed into the femoral artery and passing a prosthetic valve through the catheter.[2] Balloon valvuloplasty opens the leaflets of the stenosed aortic valve and the prosthetic valve is deployed during rapid ventricular pacing to allow for proper placement during low cardiac output.[2]

General endotracheal anesthesia (GETA) is typically the anesthetic technique of choice for cardiac surgery. GETA allows for the expeditious management of acute emergent situations such as conversion to cardiopulmonary bypass or prompt repair of an arterial access site.[4-5] GETA does not have superior outcomes for aortic valve replacement secondary to severe aortic stenosis and has an associated mortality risk of 0.03 deaths per 1000.[4]

An alternative technique to GETA for the high risk patients undergoing minimally invasive heart valve procedures is sedation. While still in its infancy stage of overall acceptance, sedation has merit when the risk benefit ratio of general anesthesia is too high, such as in this case. Deep sedation for this patient was calculated to have a better risk benefit ratio (low risk, greater benefits) however success is weighted heavily upon patient acceptance and choosing the best pharmacologic agents to create the needed safety profile and sedation state. It was for these reasons that dexmedetomidine was preferred. Like clonidine, dexmedetomidine is a selective $\alpha 2$-adrenoreceptor agonist.[7] The intrinsic effects create a reduction in sympathetic outflow, an increase in vagal tone, an unclear peripheral ganglionic blocker effect, activation of the $\alpha 2$ receptors within the spinal cord, and minimal to no effects in ventilation.[7-8] The overall clinical picture exhibited is a reduction in heart rate and blood pressure, sedation, hypnosis, anxiolysis, and analgesia, all without affecting ventilation.[7-8] Creating the safe sedation profile without affecting ventilation was crucial due to the underlying severe pulmonary disease; all efforts were aimed at maintaining ventilation, perfusion, and spontaneous respirations. Also noted and considered extremely important, when administered for cardiac surgery, dexmedetomidine is associated with an increase in 1- year survival rates, decreased postoperative complications, and decreased delirium.[8] The sympatholytic properties and sedative effects make it an acceptable and safe agent to create deep sedation during TAVR procedures; a dose of 0.3-0.7 mcg/kg/hr has been noted to facilitate shorter procedure times and as well as hospital length of stay compared to those who received a general anesthetic.[3]

Additionally, and to maximize the comfort state, a fascia iliaca compartment nerve block was performed preoperatively with ultrasound guidance. This technique reliably blocks the lateral femoral cutaneous nerve, femoral nerve and obturator nerve of the lumbar plexus and creates loss of sensation and motor control to the anterior, lateral, and medial thigh.[6] It is this area where the sheaths are placed for the TAVR procedure and if attempted without appropriate adjunct analgesia, could create enough discomfort that the procedure may have to be aborted. The ultrasound technique is recommended rather than the loss of resistance technique due to the increased rate of successful nerve block.[6]

In conclusion, success of a deep sedation technique for a TAVR procedure is dependent upon several factors, including a patient specific and acceptable risk benefit ratio, an adequate fascia iliaca neuraxial block that offers adjunctive analgesia, and last but certainly not least, informed and properly prepared patients and members of the surgery team. The value of an informed, accepting, and involved patient and team cannot be under estimated; it is believed that the success in this situation was attributable to these components of the perioperative process.

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Cesarean Section for the Parturient with Prolonged Prothrombin Time

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Keywords: prothrombin time (PT), activated partial thromboplastin time **(**aPTT), Vitamin K deficiency, clotting cascade, cesarean section, neuraxial anesthesia, coagulopathy

Pregnancy is associated with numerous changes in the coagulation system, many of which become pronounced in the third trimester. Typically, prothrombin time (PT) is not altered, but when prolonged can increase the risk of complications during delivery.[1-3] Neuraxial anesthesia is associated with decreased maternal morbidity and mortality for labor and delivery when compared to general anesthesia, but poses significant risks for the parturient with a bleeding disorder.[2,3] This case report describes a parturient presenting for cesarean section with a prolonged PT secondary to an untreated vitamin K deficiency. The anesthesia care and associated implications are discussed.

Case Report

A 26-year-old female, 152 cm, 67.1 kg, Gravida 3 Para 1, presented for a scheduled repeat cesarean section at full term. Past medical history was significant for headaches, chronic sinusitis, and spontaneous abortion. Past surgical history included one previous cesarean section delivery. Daily by mouth medications consisted of prenatal multivitamins with iron 800 mcg/27 mg and orsythia-D 1-20 mg-mcg. An elevated PT of 14.7 seconds and international normalization ratio (INR) of 1.3 were discovered during preoperative testing the morning of surgery. Review of the medical record identified that coagulation blood test results obtained 21 months prior also showed abnormal PT (13.7 seconds) and INR (1.3) values that had not been corrected. A hematology consultation was requested.

While awaiting the hematology consultation the anesthesia practitioners and surgeon consented the patient for general anesthesia, though a spinal anesthetic was agreed upon as the preferred technique. Extensive explanation was given regarding safe anesthesia care for delivery, and the patient agreed to await evaluation results. Additionally, after further questioning, the patient recalled that she bruises easily and had been instructed by her primary care physician to increase her vitamin K intake.

The hematology consultation included orders for a vitamin K test and factor VII assay and the cesarean section was postponed. An off- site analysis was necessary and took several days to complete; the results - vitamin K level 0.3 ng/mL(normal 0.10-2.20 ng/ml) and factor VII assay 117% (normal 65-180%). As all other test results were normal, and the PT and INR were trending downward, the hematology team recommended to proceed with the surgery with spinal anesthesia.

The patient re-presented for her cesarean section 8 days later. Her repeat PT and INR were within normal range; 12.5 seconds and 1.1, respectively**.** Abnormal hematology results on the day of surgery included: white blood cell count 12.3 $x10^9$/L, fibrinogen 644 mg/dL, hemoglobin

10.8 g/dL, hematocrit 33.7 %, and red blood cell count of 4.07 x 10^{12}/L. All other laboratory results were within normal limits. A spinal anesthetic was administered without difficulty on the first attempt using a 25 gauge pencil point spinal needle via midline approach. Free-flowing, clear cerebrospinal fluid was observed prior to the slow injection of 0.75% hyperbaric bupivicaine1.5 mL(11.25 mg) and fentanyl 10 mcg. The patient was assisted to a supine position with left lateral uterine displacement.

The surgical course proceeded without incident and a healthy baby was delivered. Total estimated blood loss was 500 mLand urine output was adequate and without evidence of heme. Postoperative anesthesia evaluations were negative for complications and the patient was discharged the following day. A follow-up with her primary care practitioner and hematologist was scheduled.

Discussion

Anesthesia for the parturient with coagulopathy of any severity requires a multi-disciplinary approach in developing the anesthetic plan of care.[2] Maximizing safety for both the mother and the baby remains the ultimate goal, even if it requires a delay in delivery.[2-4] The anesthesia plan should include consideration of coagulation system differences of pregnancy, vitamin K deficiency, neuraxial anesthesia and general anesthesia, and the significance of vitamin K in the coagulation process.

The hemostatic profile of the normal parturient differs from the general population in a number of areas. Plasma volume increases up to 40% while red blood cell volume only increases by half this amount, resulting in the physiologic anemia of pregnancy.[2,3] Platelet counts commonly decrease by 20%, and both fibrin stabilization factor and procoagulant factor S may be decreased 50% from pre-pregnancy values.[2] Fibrinogen and factors vWB, VIII, IX, X, XI, and XII may be increased by more than 100% by term.[4,5] Lastly, factor VII may be 1000% greater than in the pre-pregnant state.[2-4] These changes all contribute to a complex profile encompassing both physiologic anemia and hypercoagulability.[4,5] The increased total blood volume and coagulation variations facilitate epistaxis, bleeding gums, and bruising; thus, even when these symptoms are reported, they do not typically initiate further testing in the prenatal period.[2,4] It is worth noting that values for activated partial thromboplastin time (aPTT), PT, and INR, either remain constant or are slightly decreased in the parturient.[3] The most common coagulation variance reported in this population is a marginally shortened aPTT with no change to PT or INR.[3,5]

Vitamin K is a lipid-soluble co-factor required for liver production of clotting factors II, VII, IX, and X; procoagulants C and S; and the anticoagulant glycoprotein Z.[4,6] It is also necessary for the formation of other proteins essential to bone matrix homeostasis, atherosclerosis prevention, and beneficial angiogenesis[6] Vitamin K is acquired through dietary intake of green leafy vegetables, and requires bile from the liver in order to be absorbed for use in the body.[6] Low levels and true deficiency can result from inadequate intake or inadequate absorption by the intestinal flora.[6] The significance of vitamin K to the initiation of the clotting cascade, propagation, and stable clot formation cannot be underestimated. The intrinsic, extrinsic, and common coagulation pathways all rely on at least one vitamin K dependent factor to ultimately achieve stable fibrin clot formation.[3] While the hematology team in the case report did not discover widespread

coagulation abnormalities, they did acknowledge that the parturient's low vitamin K level was the only plausible explanation for the increased PT and INR.

Symptoms of a moderate deficiency include heavy menstrual bleeding, idiopathic bruising, epistaxis, bleeding gums, and hematuria, although patients can be asymptomatic.[1,4,6] Chronic deficiency can increase the risk of osteoporosis, certain cancers, cardiovascular disease, and coagulation abnormalities.[4] Pregnancy can worsen a pre-existing vitamin K deficiency because of placental transfer to the fetus.[4,6] Chronic broad spectrum antibiotic use can cause intestinal bacterial flora depletion and resultant vitamin K deficiency.[6] A prolonged PT, aPTT, or ACT may indicate one of a number of abnormalities; thus, vitamin K level and factor assays are both critically important in establishing a differential diagnosis in the parturient.[3,4,6,7]

Neuraxial anesthesia should not be attempted in cases of unexplained or uncorrected coagulopathies.[2-4] The risks of epidural abscess, bleeding, and hematoma are even more significant in the parturient due to the coagulation system changes discussed.[2-4] The rate of epidural or spinal hematoma in the obstetric population is estimated to be 1:168,000 and the risk is higher in those parturients with coagulopathies.[2,3]

General anesthesia for cesarean section is associated with the greatest risk of maternal mortality, but may be required in cases of acute maternal hemorrhage, rapid fetal deterioration, or when contraindications to neuraxial anesthesia exist.[2,4,6] Failed intubation and resultant hypoxemia are the most common causes of maternal morbidity or demise.[4] Pregnancy results in a restrictive lung disease pattern, increased minute ventilation, and decreased functional residual capacity, contributing to an accelerated time to desaturation and hypoxemia during induction of general anesthesia.[4] Additionally, increased progesterone and estrogen levels in the parturient decrease lower esophageal sphincter tone increasing the risk of aspiration.[4]

This report discussed a case of idiopathic coagulopathy in a parturient presenting for repeat cesarean section with spinal anesthesia. A comprehensive preoperative history and assessment, patient education, collaboration among the interdisciplinary care team, and outside consultation contributed to the favorable outcomes for mother and baby. The evidence is conclusive: an uncorrected, unexplained coagulopathy in the parturient is an absolute contraindication to neuraxial anesthesia.[2,3] Maintaining standard of care and remaining abreast to best practice techniques for obstetric anesthesia is an incontestable responsibility of the anesthesia practitioner. Evidence-based practice dictates that we never assume an outwardly healthy parturient negates the need for thorough pre-anesthetic evaluation prior to neuraxial anesthesia.[2-4]

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Management of Diabetes Insipidus in the Pediatric Neurosurgical Patient

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Keywords: Diabetes insipidus, pediatric anesthesia, neuroanesthesia, vasopressin, pituitary

Recent statistics estimate 10,450 new cancer diagnoses and 1,350 cancer deaths annually among children aged birth to 14 years.[1] Brain and central nervous system tumors are the second leading type of cancer in children, representing 21% of all cancer diagnoses.[1] Pituitary tumors are a unique class of intracranial neoplasms that cause mass effects on nearby vital structures resulting in hormone deficiencies and disorders of water metabolism, such as diabetes insipidus (DI).[2] Perioperative management of DI in children is challenging and frequently wrought with fluid and electrolyte imbalance. In the following case analysis, the pathophysiology and anesthetic implications of pituitary tumors are discussed.

Case Report

An 11-year-old, 152 cm, 59 kg male presented to the emergency department with nausea, vomiting, diarrhea and a 10 kg weight loss over the course of 1 month. Upon further exam, the patient also exhibited headache, vision changes, polyuria, and polydipsia. Pertinent laboratory data included serum osmolarity of 330 mOsmol/kg, urine osmolarity of 118 mOsmol/kg, and serum sodium of 160 mmol/L. MRI of the brain revealed an 8 mm infundibular mass with associated pituitary atrophy. The patient was subsequently diagnosed with central diabetes insipidus. Endocrinology was consulted, and the patient was placed on oral desmopressin therapy, which resulted in dramatic improvements in fluid and electrolyte balance. A neurosurgical consult was also obtained, and the decision was made to perform a craniotomy for excision and biopsy of tumor.

In preparation for surgery, the patient received his usual dose of desmopressin 0.2 mg the evening prior. The morning of surgery, an IV infusion of dextrose 5% in normal saline at 67 mL/hr (2/3 maintenance dose calculated using body surface area) was initiated. The patient was started on an infusion of vasopressin 0.01 mU/ kg/min.

Upon arrival to the operating room, standard ASA monitors were applied to the patient. General anesthesia was induced with fentanyl 100 mcg, lidocaine 50 mg, propofol 150 mg, and rocuronium 40 mg. The trachea was intubated without incident. Respiration was controlled by a mechanical ventilator, and end tidal CO_2 was continuously monitored. An esophageal temperature probe, right radial arterial line, and two 18-gague peripheral intravenous catheters (PIV) were placed in addition to the 20-gauge PIV placed preoperatively. The patient was positioned prone, and his head was placed in a rigid fixation device. General anesthesia was maintained with isoflurane 1% inspired concentration in a mixture of O_2 1 L/min and air 1 L/min, remifentanil infusion 0.05 mcg/kg/min, and vecuronium 0.08 mcg/kg/min. Mannitol 50 mg was administered at the surgeon's request. Normal saline was administered at 67 mL/hr, and additional hydration was only given to replace ongoing blood loss or to maintain hemodynamic stability. A single 200 mL bolus of normal saline was given for a brief period of hypotension. The vasopressin infusion started preoperatively was continued during the procedure. The dose of vasopressin was titrated in 0.01 mU/kg/min increments in approximately 30 minute intervals to establish a urine output between 0.5-2 mL/kg/hr. Urine output was monitored every half hour, and electrolytes were monitored every hour. Serum sodium concentrations were maintained between 141-147 mmol/L.

Isoflurane and vecuronium were discontinued during surgical closure, and an infusion of propofol 100 mcg/kg/min was initiated. Once the patient's head was taken out of fixation and placed supine, neuromuscular blockade was antagonized, and remifentanil and propofol infusions were discontinued. Hydromorphone was titrated to respiratory rate in incremental doses to a total of 0.4 mg. The patient moved all extremities spontaneously and purposefully, and the trachea was extubated. The patient was transported to the PACU with oxygen 10 L/min via non-rebreathing facemask and stable vital signs.

Discussion

Regulation of extracellular fluid osmolality is principally controlled by antidiuretic hormone (ADH), also known as arginine vasopressin.[3] Antidiuretic hormone is synthetized in the hypothalamus and transported to its storage sites in the nerve terminals of the posterior pituitary gland.[3] Antidiuretic hormone works at the distal tubule and collecting duct of the nephron to facilitate reabsorption of solute-free water.[3] In the presence of ADH, the distal tubule and collecting duct are impermeable to water resulting in the formation of a large volume of dilute urine. If fluid deficits are not replaced by an intact thirst mechanism or intravenous hydration, the extracellular fluid becomes hypertonic. Tonicity or osmolality refers to the effect of solutes that do not easily cross cell membranes and thus determine the transcellular distribution of water.[4] Sodium is the primary solute of extracellular fluid.[4] Therefore, a high plasma sodium concentration (hypernatremia) indicates hypertonicity and a decrease in cell volume.[4] Hypernatremia due to water depletion is called dehydration.[4] This is different from hypovolemia,

in which both salt and water are lost.[4] In a deficit of ADH, only water is lost so the patient becomes dehydrated and hypernatremic.[4]

Diabetes insipidus (DI) results from a functional deficit of ADH and may be of neurogenic or nephrogenic origin.[2] Neurogenic DI is caused by destruction of the posterior pituitary, while nephrogenic DI is due to failure of the renal tubules to respond to ADH.[5] Classic manifestations of DI include polydipsia and a high output of dilute urine despite increased serum osmolality.[5] Older children with intact thirst mechanisms usually present with normal serum sodium levels as they are able to replace ongoing water losses.[4] Infants and small children are unable to do so and thus often present hypernatremic.[4] Diabetes insipidus that develops during or immediately after neurosurgery is generally due to reversible trauma to the posterior pituitary and is usually transient.[5] DI that occurs due to mass effects from brain tumors in the regions of the hypothalamus, pituitary, and optic nerve may be permanent.[5]

The principal goal of anesthetic management of pediatric patients with DI presenting for craniotomy is maintenance of fluid and electrolyte balance.[5] There are several different schools of thought with regards to management of DI during the perioperative period. Replacement of ADH is not benign, and is accompanied by the risk of water intoxication.[6] If vasopressin is administered in conjunction with excessive fluid intake, cerebral edema may occur resulting in hyponatremia, seizures, and neurologic injury.[6] To avoid the risk of water intoxication, replacement of ADH may be forgone and the child is hydrated with twice the normal daily maintenance rate.[6] Another school of thought is to replace ADH during the perioperative period with a continuous infusion, restrict intravenous fluids intraoperatively, and allow the child's intrinsic thirst mechanism to regulate fluid intake postoperatively.[6] Conflicting ideology with regards to management of DI during the perioperative period may lead to confusion of the anesthetist and suboptimal management of the patient.

To determine optimal perioperative management of DI, a landmark study compared outcomes of children treated with a standardized protocol to chart reviews of children who presented with or developed DI perioperatively.[6] The mainstay of the standardized protocol consisted of a continuous low-dose vasopressin infusion and fluid restriction.[6] Children in the retrospective control group were managed with a variety of treatment regimens, none of which consisted of a vasopressin infusion.[6] No children in the prospective group had serum sodium concentrations fall outside of the goal range of 130-150 mEq/L during the intraoperative period, while 58% of patients in the control group had sodium concentrations fall out of range.[6]

Management of the patient in the case described, closely mirrors the prospective protocol with one notable exception. In the research protocol, once satisfactory control of urine output was achieved, the dose of vasopressin was not decreased.[6] In the case report, the vasopressin infusion was titrated downward according to urine output. Euvolemic children fully antidiuresed with vasopressin will still continue to produce urine at a rate of approximately 0.5 mL/kg/hr.[6] Therefore, oliguria or anuria is an indication for additional fluid administration, rather than decreasing or discontinuing the vasopressin infusion.[6]

Successful perioperative management of children with DI who present for craniotomy requires precise monitoring and collaboration with an interdisciplinary approach. An evidenced based protocol aids in optimal patient management and reduces confusion among anesthetists.

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Anesthetic Implications for the Parturient with Neurofibromatosis Type 1

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Keywords: neurofibromatosis type 1, pregnancy, cesarean section, anesthesia, regional anesthesia, spinal

Neurofibromatosis Type 1(NF-1) is an autosomal dominant disorder that stems from a mutation of chromosome 17, which controls cell proliferation, resulting in tumors of the peripheral and central nervous system.[1-4] Expressions of the gene are frequently seen as benign, hyperpigmented lesions (café-au-lait spots) or cutaneous tumors. However, more severe forms may include symptoms such as malignant neurofibromas, intracranial tumors, or learning disabilities.[1-3,5] With an incidence of approximately 1:3,000 people, it is likely that many anesthesia professionals will encounter this disease during their careers.[1,2] Pregnancy may further complicate symptoms and result in an increase in size and number of tumors.[1,4,6] This case study reviews the current anesthetic implications for the parturient with NF-1 and the role of regional anesthesia.

Case Report

A 19-year-old primigravida presented for induction of labor at 37 weeks 6 days gestation after ultrasound and non-stress test revealed intrauterine growth restriction (IUGR) <3% and fetal pericardial effusion. The female patient was 162 cm in height and 67 kg in weight. Mallampati classification was a Grade 2. Pertinent medical history included Neurofibromatosis Type 1 (NF-1) and attention-deficit/hyperactivity disorder. Upon assessment, the patient was found to have café-au-lait spots and peripheral subcutaneous and cutaneous tumors consistent with NF-1 diagnosis. The patient reported that she had magnetic resonance imaging (MRI) "a few years ago" that was negative for central nervous system (CNS) tumors, but that she had not had any scans in recent years due to insurance issues. It was recommended to her to contact a neurofibromatosis clinic for a comprehensive follow up with repeat MRI of the head without contrast and ophthalmology exam at 25 weeks gestation. However, the patient did not follow-up with the specialist and did not have a recent MRI performed. The patient reported an increase in number and size of her peripheral lesions during pregnancy, but no focal neurological abnormalities or weakness.

Induction of labor commenced with the administration of an oxytocin infusion and was advanced per institutional protocol. Approximately 10 hours into induction, recurrent variable decelerations in fetal heart rate began to occur. After a variable fetal deceleration to 80 beats per minute, the health care team decided to proceed with an urgent cesarean section. The decision was made by the anesthesia professionals to proceed with a spinal anesthetic for the cesarean section. Standard monitors were applied and oxygen was administered via nasal cannula at two liters per minute. The patient was placed in the sitting position so that the lumbar region could be prepped and draped in sterile fashion. An introducer needle was placed, followed by a 25 gauge pencil-point spinal needle between the L4 and L5 vertebrae via midline approach. After aspiration of cerebrospinal fluid, bupivicaine 0.75% 1.4 mL with 0.2 mg morphine was administered into the intrathecal space without report of paresthesia. A T4 dermatome level of sensory blockade was obtained. The patient's vital signs remained stable after administration of local anesthetic and throughout the subarachnoid block procedure. Cesarean section proceeded uneventfully and a live infant, who did not require any respiratory or cardiovascular support at birth, was delivered. The patient was discharged two days later without incident.

Discussion

Neurofibromas may affect any organ system in the body.[3,4] Disease progression is highly variable from person to person.[3,4] Additional neurofibromas may continue to develop throughout the lifespan.[3,4] Neurofibromatosis Type 1 patients can be a challenge for the anesthesia professional, as each individual afflicted by the disease may have different physical symptoms.[5,6] NF-1 primarily affects the peripheral nervous system, with CNS involvement occurring in less than 10% of those affected with the disease.[6] Neurofibromatosis type 2 is more rare, severe, and primarily involves the CNS.[6] As with any neurological disorder, it is imperative that the anesthesia professional performs a thorough preoperative neurological assessment to detect any deficits, as well as to explore any treatment or therapy currently implemented.[4,6] Hormonal changes, such as pregnancy, can exacerbate symptoms of NF-1 causing tumors to increase in size or new tumors to form.[1,4,6] Airway assessment is of primary concern as this population is at risk

for the development of neck/laryngeal lesions and the potential for general anesthesia with airway instrumentation is always a possibility with any anesthetic.[3,4,5]

It is well documented in the literature that hormonal changes, such as pregnancy, have correlated with an increase in size and number of tumors. [1,4,6] Retrospective studies have shown that NF-1 is associated with increased maternal morbidity in pregnancy: specifically, gestational hypertension, preeclampsia, IUGR, cerebrovascular disease, preterm labor, and higher rates of cesarean section.[1,2,4] These patients are categorized as having a "high risk" pregnancy.[1] With no specific treatment for NF-1 during pregnancy, these patients should be closely monitored at high-risk tertiary centers for early detection of possible complications.[1] Associated morbidities specific to this case include IUGR and an unplanned cesarean section.

Despite pre-existing neurologic conditions at baseline, regional anesthesia may be preferred over a general anesthetic.[5,6] The use of muscle relaxants in NF-1 patients is controversial. Related literature has shown increased sensitivity to non-depolarizing muscle relaxants.[3,4,6] Additionally, there are conflicting reports of increased sensitivity versus resistance to depolarizing muscle relaxants.[3,4,6] Neuraxial anesthesia has successfully been performed on parturients and surgical patients with NF-1 without complication.[5,7] Research studies recommend that radiologic imaging such as computed tomography (CT) scan or MRI should ideally be performed prior to regional anesthesia to rule out any potential spinal cord lesions.[5,6] Spinal anesthesia is contraindicated in patients with known space occupying lesions, as it can result in cerebellar herniation.[6] During the second stage of labor, these patients can have large increases in intracranial pressure (ICP) due to contractions and bearing down (Valsalva maneuver).[4] An elective cesarean section under general anesthesia is the ideal management for the parturient with a known space occupying lesion.[6]

In this case, there were no current images on record to definitively confirm the absence of any CNS tumors. However, the patient and her mother stated that her historic scans were negative for these lesions. The physical exam did not reveal any signs or symptoms suspicious for CNS tumors. The decision was made to proceed with a spinal anesthetic as the anesthesia professional felt the risk of CNS tumors was low and the benefits of regional anesthesia (compared to general anesthesia) for her cesarean section outweighed the risks. A similar case from Korea is documented in the literature with limited pre-operative imaging followed by successful spinal anesthetic in a parturient requiring unplanned cesarean section. [7] Although this case had a positive outcome without any complications, the question remains as to whether neuraxial anesthesia was truly the safest choice for the patient. A safer approach may have been to obtain a CT or MRI when the patient was admitted for induction of labor. The imaging would have posed little to no risk to the patient and fetus, and could have confirmed the absence of CNS lesions. In an urgent situation, where there is not enough time to obtain imaging and the surgery must proceed, it may be in the best interest of the patient to perform general anesthesia. Although the likelihood of CNS tumor presence is small, if an unknown tumor existed, spinal anesthesia could result in severe complications.

Anesthesia professionals utilize extensive knowledge and best evidence resources every day in order to make critical decisions regarding the plan of care for their patients. Both neuraxial and general anesthesia have been documented as safe options for the parturient with NF-1. However,

further research is warranted to develop clearer evidence based practice guidelines for the care of the NF-1 parturient.

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Tracheal Injury following Double Lumen Endotracheal Tube Insertion

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Keywords: Adenocarcinoma, thoracotomy, double lumen endotracheal tube, one lung ventilation, tracheal injury

This case report details the airway management for and unintended complication of a thoracotomy and right upper lobectomy planned for the resection of an adenocarcinoma of the lung. A discussion of airway anatomy, placement of double lumen endotracheal tubes, intubation complications, and indications for one-lung ventilation will be presented.

Case Report

A 67-year-old female with a history of colon cancer was found to have a hypermetabolic 2.8 cm nodule in the upper lobe of her right lung. She underwent a bronchoscopic biopsy and was

subsequently admitted for a thoracotomy with right upper lobe resection. Preoperative airway assessment revealed a Mallampati I with full neck extension and a thyromental distance of 5.5 centimeters. In the operating room, standard monitors were applied and vital signs were within normal limits. An infusion of Ringer's lactate was started and midazolam 2 mg IV was administered. The patient was positioned sitting for a thoracic epidural at the T8 level. The patient was then positioned supine, O_2 10 L/min was administered via face mask, lidocaine 50 mg and propofol 100 mg IV were administered. Manual ventilation was easily performed and rocuronium 40 mg IV was administered to facilitate intubation.

The trachea was intubated with a left-sided double lumen tube (DLT) atraumatically on the first attempt. Once the distal cuff of the DLT tube passed through the vocal cords, the stylet was removed and a fiberoptic bronchoscope (FB) was used to guide the distal end of the tube into the left mainstem bronchus under direct visualization. The DLT passed with ease and placement was re-verified with the FB through the tracheal lumen. There was no apparent air leak during two-lung ventilation. The patient was maintained on volume-controlled ventilation with the following settings: tidal volume 400 mL, respiratory rate 12/min, FiO_2 1.0, peak inspiratory pressure < 25 cm H_2O. After repositioning the patient to the left lateral decubitus position, lacement of the DLT was reconfirmed with the FB and found to be in good position. One-lung ventilation (OLV) to the left lung was instituted by clamping the tracheal side of the DLT. Throughout the surgery, vitals remained stable and SpO_2 was > 95% for the duration of the case. Anesthesia was maintained with sevoflurane 1.8% inspired concentration, fentanyl 100 mcg IV and an additional 20 mg rocuronium IV. After right upper lobe resection and closure of the bronchial stump, the surgeon requested re-inflation of the right lung with 3 Valsalva maneuvers. No air leak was noted and the patient was returned to OLV for the remainder of the surgery.

Before final closure of the chest, the surgeon flooded the field with saline and requested repeat re-inflation of the right lung. During this maneuver, continuous bubbling was noted from the field. An air leak was identified from a linear membranous tear of the posterior trachea. The patient was returned to OLV while the tracheal injury was repaired. No further air leak was noted after closure of the tear. During chest closure, the anesthesia practitioners were unable to dose the thoracic epidural due to the presence of frank heme during catheter aspiration. Hydromorphone 1mg IV was given for post-operative pain. Neostigmine 3 mg and glycopyrrolate 0.6 mg IV were administered to antagonize residual neuromuscular blockade. The patient was extubated awake to a non-rebreather mask and was transported in stable condition to the ICU.

Discussion

The trachea is a part of the lower airway that extends from the bottom of the cricoid cartilage to the carinal bifurcation. The trachea consists of C-shaped cartilages anteriorly while the posterior trachea is comprised of unprotected smooth muscle.[1] The posterior tracheal wall is very susceptible to injury during intubation.[1,2] Major differences between the right and left bronchi include size of the bronchi and angle of bifurcation from the trachea. The right mainstem bronchus has a wider diameter which diverges from the trachea at an angle of 25 degrees whereas the left bronchus diverges at 50 degrees.[1] The right mainstem bronchus divides into upper, middle and lower lobe bronchi whereas the left divides into upper and lower lobe bronchi

only.[2] The orifice of the right upper lobe bronchus is approximately 1-2.5 cm from the carina. The bifurcation of the left main bronchus is typically about 5 cm distal to the carina.[1,3] A left-sided DLT is preferable for OLV due to the difficulty in placing a right-sided DLT which may obscure the more proximal orifice of the right upper lobe. Many anesthesia practitioners will choose to use a left-sided double lumen tube unless it is contraindicated by distorted anatomy of the left mainstem bronchus. This can be due to the presence of a bronchial mass or compression of the left main bronchus due to a descending thoracic aortic aneurysm. Indications for OLV include hemorrhage of one lung, infection, disruption of the tracheobronchial tree and the need for single lung isolation.[1]

When intubating with a L-sided DLT, a direct laryngoscopy is performed and the tube is passed through the glottis. From this point, the anesthetist can either use a blind or a fiberoptic-guided technique when placing the DLT into the left bronchus. With the blind technique, once the distal cuff passes through the vocal cords, the tube is rotated 90° counterclockwise then blindly advanced into the trachea then the L-mainstem.[3,6] Moderate resistance indicates placement of the bronchial lumen into the bronchus and the DLT should not be advanced further.[1] A fiberoptic bronchoscope-guided technique involves removing the rigid stylet after the distal end of the DLT enters the glottis and placing the FB into the bronchial lumen of the DLT. The DLT is then guided into the L-mainstem bronchus under direct visualization.[3,6] To verify proper L-sided DLT placement, the bronchoscope is then inserted through the tracheal port of the DLT. The carina should be visible just distal to the exit point of the bronchoscope and the tip of the blue cuff of the bronchial lumen should be visible just lateral to the carina entering into the L-mainstem bronchus.[1,6] After inflation of both tracheal and bronchial cuffs, tidal volume should be maintained during normal and one-lung ventilation with no apparent air leak and adequate compliance.[3] Re-verification of DLT placement is necessary after the patient is placed in the lateral position as the DLT may migrate. Difficulties that can be encountered with DLT placement include a tube placed too deep or proximal into the bronchus or inadvertent placement into the incorrect mainstem bronchus.[1,6]

Potential complications associated with the placement of DLTs range from failed intubation and damage to the vocal cords and arytenoids to bronchial or tracheal rupture; a rare but serious complication.[3] Signs of tracheobronchial rupture include inability to maintain adequate volumes with mechanical ventilation, subcutaneous emphysema, pneumomediastinum and pneumothorax.[2,4] Delayed manifestations of tracheal injury include acute mediastinitis, chest discomfort and dyspnea.[2] These complications may occur due to overinflation of the bronchial cuff, inappropriate positioning and trauma caused during intubation.[1,2] Most tracheobronchial injuries usually occur as longitudinal tears within the membranous portion of the trachea.[5] This tissue damage may be caused by extension of the rigid stylet beyond the distal end of the DLT, multiple intubation attempts, or weakened membranous tracheal tissue secondary to cancer treatments or steroid use.[1,5]

Early recognition of the complication was fortunate and imperative to the successful outcome of the procedure. The etiology of the tracheal tear was never fully determined. There were no signs of decreased VT intraoperatively or evidence of subcutaneous emphysema throughout the case. The surgeon suspected this complication could have occurred during intubation or be due to the overfilling of the bronchial cuff in the L mainstem. However, the bronchial intubation was

performed under direct visualization via fiberoptic bronchoscopy and the endobronchial cuff inflation was limited to 2cc of air. The tear may have also occurred due to paratracheal lymph node sampling during the surgical procedure. No subcutaneous emphysema was appreciated upon leaving the operating room however, the initial chest x-ray in the ICU revealed a left apical pneumothorax and a chest tube was subsequently placed. In conclusion, despite the complication, the patient was discharged from the ICU after several days and suffered no long-term sequelae.

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Anesthetic Management for Patient with Rubinstein-Taybi Syndrome

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Keywords: Malignant hyperthermia, occipito-atlantic dislocation, occipital to cervical fusion, Rubinstein-Taybi syndrome, total intravenous anesthetic.

Rubinstein-Taybi syndrome (RTS) was first described in 1963 by Rubinstein and Taybi.[1-3] It is a rare autosomal dominant mutation of chromosome 16.[1-3] Some of the pertinent clinical symptoms of RTS are cranio-facial abnormalities, cardiac anomalies and dysrhythmias.[1-,3] Furthermore, these patients are at a higher risk for gastroesophageal reflux, mental retardation, skeletal anomalies and development of tumors.[1,2,3] The purpose of this paper is to present a case study of a patient who has RTS and a history of two episodes that appeared to be malignant hyperthermia (MH).

Case Report

A 49 kg, 180.3 cm, 47-year-old female with no known drug allergies presented with an occipito-atlantic dislocation with cervical (C) spine vertebrae 1 fracture and a C-2 facet fracture. The fracture was sustained after falling down seven stairs. An occipital to C-5 fusion using intraoperative three dimensional (O-arm) imaging (Medtronic, Minneapolis, MN) and navigation was scheduled. Her past medical history was significant for RTS, mental retardation, anxiety, kyphoscoliosis, scoliosis and two episodes of hyperpyrexia attributed to possible MH. The last episode of hyperpyrexia occurred in 1986 during the recovery phase after a general anesthetic.

The patient had facial anomalies with distortion of the lower face and neck, microstomia, beaked nose, and a cervical-collar (c-collar) for C-spine stabilization. She appeared to be restless, tugging on her c-collar and required frequent re-orientation. Her current medication regimen included acetaminophen with codeine phosphate, famotidine, fluticasone propionate, and ibuprofen.

Due to previous anesthetic complications, a pre-surgery huddle was performed and all operating room (OR) staff members were made aware of patient's complications. The patient was pre-medicated with midazolam 1 mg intravenous (IV) in two divided doses. Hemodynamic monitoring included standard monitors, as well as arterial line (A-line) and Bispectral Index monitoring (Covidien, Boulder, CO). General anesthesia was induced using fentanyl 50 mcg IV, lidocaine 40 mg IV, and propofol 100 mg IV. Neuromuscular blockers were avoided. The trachea was intubated using video laryngoscopy without any complications. Anesthesia was maintained using a continuous propofol and remifentanil IV infusions. All volatile anesthetics and succinylcholine were avoided.

The patient's head was placed in a Mayfield (Integra, Plainsboro, NJ) device by the surgeons and the patient was positioned prone on a rotisserie bed. A mean arterial pressure greater than 85 mm Hg was requested by the surgeon. This parameter was maintained with a phenylephrine IV infusion. The patient had occasional premature ventricular arrhythmias and a transient episode with u wave present on the EKG. Baseline arterial blood gases were drawn after A-line placement and again after the transient episode of u wave on the EKG. The u wave was attributed to a decline in the patient's potassium level that had decreased from 3.6 MEq to 3.2 MEq. This alteration was not treated due to the transient nature of the dysrhythmia. The patient's average urine output was approximately 200mLper hour. O-arm imaging was utilized during the surgery requiring the patient to be apneic for less than two minutes per episode. Multiple images were taken at surgeon's discretion in order to precisely secure the cervical hardware. Preparation for the apnea period was accomplished by delivering 100% inspired oxygen to the patient for at least five minutes before anticipated O-arm imaging. The patient tolerated O-arm imaging without any complications. The patient's coagulation function was suboptimal due to a two week history of nonsteroidal anti-inflammatory drug intake for pain relief. Desmopressin acetate IV was given intraoperatively, per the surgeon's request, to support the coagulation function.

Total operative room time was seven and one-half hours. After ensuring that the patient was awake, breathing spontaneously, ventilating with adequate tidal volumes, and following commands, the trachea was extubated at the end of the surgery The patient was transported to an

intensive care unit. First day post anesthesia rounds were done, and the patient was found to be stable without any anesthetic-related complications.

Discussion

Patients with RTS pose special challenges to the anesthesia practitioners as they have an increased possibility of difficult intubations, aspiration, cardiac dysrhythmias, and pose challenges for a possible awake intubation due to cognitive limitations. Extensive preparation is required for cases with RTS due to possibilities of fatal complications. These patients are prone to having facial anomalies with an anterior airway. A difficult airway cart, fiberoptic scopes, video laryngoscopes, four anesthesia practitioners, and a student nurse anesthetist were available in the room during induction and intubation. Administration of succinylcholine, atropine and neostigmine were avoided due to increased risk of dysrhythmias.[1-3] Patients with RTS can have congenital cardiac issues and dysrhythmias resulting in ventricular tachycardia during general anesthesia.[1-3] In preparation for possible cardiac complications, epinephrine infusion and dilution were calculated and placed on the clean table together with pre-filled atropine, and epinephrine syringes.

The anesthetic care of this patient was complicated by previous episodes described as possible MH or anaphylaxis with hyperpyrexia; however, no anesthetic document could be located from 1983 and 1986 hospitalizations. The history of possible MH episode was raised by the patient's mother. Review of all the past medical records revealed MH as a differential diagnosis for the hyperpyrexia. No mention of the anesthetic medications that were administered or any interventions for the possible MH was found in the records. In order to minimize further potential complications, the patient was treated as being MH susceptible, therefore all MH triggering medications were avoided and total intravenous anesthesia was administered. The anesthesia gas machine was flushed and charcoal filters were placed on the inspiratory and expiratory limbs of the breathing circuit per the hospital protocol.[4] MH cart was placed immediately outside the operating room door, and the calculated dose of dantrolene sodium was highlighted to ensure quick access. A crash cart was also immediately available and located outside the room. The staff in the OR were educated about the patient's medical condition and briefed about the location of the MH cart and the crash cart before the patient was brought into the OR. The anesthesia technicians were made aware of the possibility of complications and therefore were ready to assist. Communication among the team members ensured that everyone in the OR was informed and agreed on the plan for treatment and potential complications.

Future practice for anesthetic management of patients with RTS should include an extensive preparation for both airway and cardiac management as each patient is unique and the genotype-phenotype association of RTS is still unclear.[5] Therefore, careful medication selection and titration in the RTS population would be prudent.

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Anesthetic Management for Abdominal Aortic Aneurysm Repair

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Keywords: abdominal aortic aneurysm, open surgical repair, EVAR

An abdominal aortic aneurysm (AAA) is a dilatation of the abdominal aorta with a diameter of 3.0 cm or greater.[1,2] The incidence is 3% -10% of patients beyond age 50.[3] Factors associated with an increased risk include atherosclerosis, high cholesterol, advanced age, and smoking.[3] Often, patients are asymptomatic with only 30%-40% detected on physical exam.[4] The two major approaches to the treatment of an AAA include open surgical repair and minimally invasive endovascular aneurysm repair (EVAR).[1] Although EVAR has quickly replaced open AAA repair and is considered a first-line treatment, not all patients are suitable candidates.

Case Report

A 68-year-old, 170 cm, 101 kg male presented for elective open surgical repair of a 5.2cm x 4.8cm infrarenal abdominal aortic aneurysm. The patient's medical history was significant for tobacco use, hypertension, chronic kidney disease, chronic thrombocytopenia, non-insulin dependent diabetes mellitus, and rheumatoid arthritis. The patient's surgical history included an appendectomy, tonsillectomy and adenoidectomy, vasectomy, and left inguinal hernia repair. His home medications included atorvastatin, lisinopril, metoprolol, and metformin.

Preoperative laboratory values revealed a hemoglobin of 14.2 g/dL, platelet count of 121 bil/L, blood urea nitrogen 25 mg/dL and creatinine 1.73 mg/dL. Stress myocardial perfusion imaging was negative for ischemia and displayed an estimated ejection fraction of 73%. Pulmonary function testing specified mild obstructive airway disease. Airway assessment revealed limited range of motion in the cervical spine. Two 18 gauge peripheral intravenous (IV) catheters were inserted and midazolam 4 mg was administered. A 20 gauge arterial catheter was inserted in the

right radial artery revealing a blood pressure of 154/81 mm Hg, correlating with the automatic blood pressure cuff.

The patient was transferred to the operating room and standard monitors were applied. The patient was pre-oxygenated with O_2 8 L/min via facemask for 5 minutes. Anesthesia was induced with fentanyl 100 mcg, lidocaine 100 mg, propofol 130 mg, and rocuronium 50 mg IV. The trachea was intubated with an 8.0 mm endotracheal tube (ETT) and anesthesia maintained with isoflurane 0.9-1% inspired concentration in a mixture of O_2 1 L/min and air 1 L/min. Adjustment of the inspiration-to-expiration ratio (I:E) to 1:2.5 was required to compensate for the mild obstructive airway disease. A central venous catheter was inserted into the right internal jugular vein.

An initial activated clotting time (ACT) and arterial blood gas was within normal limits. The surgeon requested systolic blood pressure be maintained between 90-110 mm Hg during the repair. Heparin 7,500 units was administered IV to maintain ACT levels between 300-350 seconds. In anticipation of an increase in both preload and afterload following placement of an infrarenal aortic cross clamp, fluid replacement was minimized to 750 mL and the inspired concentration of isoflurane increased to 1.3%. In addition, 12.5 g of mannitol 20% was administered to improve renal blood flow and reduce ischemia.

Prior to aortic unclamping, an intravenous fluid bolus of 500 mL was administered. During release of the aortic clamp, the patient's blood pressure decreased to 72/51 mm Hg. Hypotension was treated with intermittent intravenous boluses of phenylephrine 40-80 mcg, totaling 360 mcg throughout the surgery, ephedrine 10 mg, and decreased inspired concentration of isoflurane to 0.8%. Intravenous calcium chloride 200 mg was administered for positive inotropic effects. Sodium bicarbonate 25 mEq was administered intravenously to counterbalance the metabolic and respiratory acidosis.

The patient received a total of 517 mL of cell saver blood, 2500 mL of Lactated Ringer, and had an estimated blood loss of 900 mL. Urine output totaled 560 mL. Neuromuscular blockade was reversed and the trachea extubated in the operating room. The patient was admitted to the surgical intensive care unit for close monitoring

Discussion

An abdominal aortic aneurysm is a pathologic dilation of the abdominal aorta and has the potential for significant morbidity and mortality. The majority of abdominal aortic aneurysms are infrarenal, between the renal and inferior mesenteric arteries.[4] Abdominal aortic aneurysms can be both asymptomatic and symptomatic; potential clinical manifestations include umbilicus and upper abdominal pain, sudden severe pain in the lower back, and shock.[5] The diagnosis of an AAA can be made through physical examination, abdominal ultrasonography, abdominal computed tomography (CT), and magnetic resonance imaging (MRI).

Medical management is aimed at reducing the risk of aneurysmal rupture and includes administration of aspirin, beta-blockers, ace inhibitors, and statins.[6] Surgical treatments include EVAR or open surgical repair. Multiple aspects are considered to determine if EVAR is an

appropriate surgical option, including anatomy of the aorta, visceral vessels and access vessels, the patient's presenting medical condition and co-morbidities, and the feasibility of performing an open repair.[2] Indications for open surgical repair include symptomatic aneurysm of any size, asymptomatic aneurysm $\geq$ 5.5 cm, and a rapidly expanding AAA.[2] Although elective endovascular AAA repair is associated with lower rates of perioperative morbidity and mortality compared with elective open repair, long term outcomes are similar.[5]

Completing a thorough preoperative assessment is crucial when caring for a high-risk patient with an abdominal aortic aneurysm. Open surgical repair carries a higher risk for perioperative cardiovascular events, thus the anesthesia practitioner needs to be aware of all patient comorbidities.[5] The goal of the perioperative evaluation is to assess the patient's coexisting diseases, optimize the medical status, and develop an anesthetic plan tailored to the individual patient. Patients who are currently taking aspirin, beta blockers, and receiving statin therapy should continue these medications up to the scheduled day of surgery.[4] Diuretics and ACE inhibitors should be considered on a case-by-case basis.[4] Anesthesia for aortic surgery is frequently associated with wide variations in blood pressure, making invasive monitoring pertinent for laboratory analysis and hemodynamic management. Central venous cannulation allows for the measurement of cardiac filling pressures, provides a reliable route for drug delivery and is a site for rapid fluid administration.[1]

General anesthesia is indicated for open surgical repair of an AAA. Aggressive postoperative pain management is necessary for open surgical repair, including the insertion of a thoracic (usually T8-T10) epidural catheter perioperatively.[7] In this population, adding an epidural for post-operative pain control reduces numeric pain scores, time to tracheal extubation, myocardial infarction, postoperative respiratory failure, and time spent in the ICU.[7] In this particular case, the patient refused an epidural. Intravenous fentanyl and hydromorphone were titrated according to the presenting hemodynamic status and pain indicators, totaling 250 mcg and 1 mg respectively.

For infrarenal aortic aneurysms, an aortic clamp is placed below the renal arteries.[8] Aortic cross-clamping at the infrarenal level is associated with only minimal cardiovascular changes and no wall motion abnormalities compared to the supraceliac and/or suprarenal level.[6] During aortic cross clamping, hypertension occurs above the cross-clamp and hypotension occurs below the cross-clamp.[3] In addition, there is an increase in afterload and left ventricular end-diastolic volume, leading to increase in myocardial oxygen demand.[3] Anesthetic interventions to attenuate this response include the administration of vasodilators (sodium nitroprusside, nitroglycerin), opioids, and/or deepening the inhalation anesthetic.[4] Aortic unclamping is associated with a 70-80% decrease in peripheral vascular resistance, resulting in profound vasodilation.[4] Hypotension can also be caused by blood sequestration in the lower half of the body, ischemia-reperfusion injury, and the washout of anaerobic metabolites causing metabolic acidosis.[4] Anesthetic management consists of volume loading, vasoconstrictors (phenylephrine, ephedrine), and the use of positive inotropic drugs (calcium chloride, dobutamine).[4] Moderate augmenting of intravascular volume by administration of fluids (~500 mL) during the immediate prerelease period is indicated for infrarenal unclamping.[6] In addition, the surgeon can gradually release the aortic clamp, reducing the overall adverse effects associated with aortic unclamping.

Management of the patient with an abdominal aortic aneurysm can be very challenging, requiring vigilance with all aspects of anesthetic care. Appropriate perioperative interventions were taken, in accordance with current evidence to provide care for the patient with an infrarenal abdominal aortic aneurysm, as evidence of an uneventful AAA repair.

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Venous Air Embolus during a Rotational Scalp Flap Repair

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Key words: Venous air embolus, anesthesia, pediatric, scalp flap

Venous air embolism (VAE) is the entrainment of air or exogenously delivered gas from the operative field into venous or arterial vasculature.[1-3] The greater the gradient between the surgical site and right atrial pressure (RAP), the greater the susceptibility for air entrainment.[1,2] The critical gradient can be as low as 5 cm H_2O.[1] Air and blood interact inducing the production of fibrin with thrombosis formation which can further obstruct right ventricular and pulmonary outflow.[1] The severity of hypoxemia is variable and results in ventilation-perfusion (V/Q)

maldistribution.[1] Venous air emboli can produce a multitude of symptoms which can become lethal without early recognition and prompt treatment.

Case Report

A 15.1 kg, 6-year-old female presented for second stage scalp reconstruction with removal of tissue expanders. The patient's medical history included a burn occurring at 18 months of age that encompassed 45% of the total body surface area and involved the head, face, back, and right upper extremity. In addition, the patient underwent multiple reconstructive surgeries including placement of tissue expanders to correct a large area of right sided scalp alopecia and a full thickness skin graft to the right ear helix.

Preoperative vital signs included: blood pressure 102/54 mm Hg, pulse 110/min, SpO_2 100%, temperature 36.2º C. A complete blood count revealed a white blood cell count of 6.8 bil/L, hemoglobin 9.0 g/dL, hematocrit 28.4% and a platelet count of 425 bil/L. The patient was transferred to the operating room table and standard ASA monitors were applied. Anesthesia was induced via inhalation induction with a mixture of sevoflurane 8% and O2 8 L/min. A 22 gauge peripheral intravenous catheter was placed and an infusion of lactated ringers was initiated. Propofol 25 mg, rocuronium 5 mg, and fentanyl 15 mcg were administered intravenously to facilitate intubation. The airway was secured by direct laryngoscopy with a 4.5 mm microcuff endotracheal tube. Placement was verified by bilateral auscultation of the lungs and positive end tidal CO_2. Following intubation, anesthesia was maintained with isoflurane 1.6 -1.9 % in a mixture of O_2 1 L/min and air 1 L/min. The patient was positioned supine with the head and lower extremities in a neutral position and upper extremities tucked and secured at the patient's side. The patient's scalp was then prepared and draped and the tissue expanders were removed. Granulation tissue was curetted and the surgical site was irrigated with antibiotic solution.

Approximately 40 minutes into the case, the scalp was irrigated with sterile saline, during which a dramatic drop in $ETCO_2$ was noted. In addition, a manual blood pressure could not be obtained, the SpO_2 reading was undetectable and ST segment elevation was observed on the electrocardiogram. The inhalation agent was discontinued and the patient was placed on O_2 8 L/min. In rapid sequence, the surgeon was notified, compression of scalp was employed and the presence of a carotid pulse was verified. An infusion of lactated ringers was titrated to achieve a 10 mL/kg bolus while 5 mcg of epinephrine was administered. The heart rate began to decrease from 160/min to 110/min. Glycopyrrolate 0.15 mg along with and an additional 5 mcg of epinephrine were administered IV. Within minutes, a manual blood pressure reading of 50/30 mm Hg was obtained. At this time, another 22 gauge peripheral IV was placed and a 0.9% saline infusion was started at 50 mL/hr. Additionally, the patient received CaCl 150 mg and a transfusion of 150 mL of packed red blood cells was initiated. A left pedal arterial line was inserted for continuous blood pressure monitoring.

The patient's mean arterial pressures improved ranging from 50 – 70 mm Hg throughout the remainder of the case. Once hemodynamic stability was reestablished, the surgical procedure was reinitiated. The fasciocutaneous flap was then rotated by extending the incision along the area of the hairline. A second transfusion of 150 mL of packed red blood cells was given and a repeat complete blood count revealed the following: WBC 43.4 bil/L, Hgb 9.1 g/dl, Hct 26.9%

and Plt of 383 bil/L. Arterial blood gases and electrolytes were obtained and found to be within normal range. The remaining scalp tissue was then extended laterally on the left side. The total scalp rotational flap measured 210 cm^2. A drain was placed and skin closure was achieved with sutures and staples. The patient received a total IV intake of 400 mL of isotonic crystalloid and 300 mL of PRBCs. Estimated blood loss for the case was 150 mL. Neuromuscular blockade was antagonized with neostigmine 0.75 mg and glycopyrrolate 0.15 mg. Upon meeting criteria for extubation, the endotracheal tube was removed and the patient was transferred to the pediatric intensive care unit where she remained stable postoperatively.

Discussion

The detected incidence of VAE varies with the type of procedure, patient positioning, and sensitivity of methods to diagnose their occurrence.[1-7] The type of intraoperative monitoring employed for the detection of a VAE should be based on the likelihood of development, underlying illness, and invasiveness of surgery.[1] Monitoring devices for detection of a VAE include in order of most to least sensitive: transesophageal echocardiography (TEE), precordial ultrasound doppler, and pulmonary artery catheter. These devices were not employed in this particular case due to the low level of suspicion for developing a VAE in a patient positioned supine. Other forms of monitoring include end-tidal carbon dioxide and direct observation of slow continuous venous bleeding at the surgical site.[4] The absence of venous bleeding at the surgical site could indicate that venous pressure is greater than atmospheric, therefor increasing the probability of air entrainment.[1] Capnography is a moderately sensitive, semi-quantitative, non-invasive monitoring technique that can detect VAE prior to the onset of cardiovascular disturbances.[3] Although TEE is the most sensitive detection device of venous air, its use is not without risk.[1,3] One study revealed its use to be clinically insignificant while demonstrating that capnography is an efficient monitoring tool to detect VAE severe enough to cause hemodynamic disturbances.[3]

A sudden severe reduction in $ETCO_2$, as observed in this case, is usually accompanied by changes in the systemic hemodynamic and airway pressures with improvement only following active corrective measures.[3,4] Prevention of VAE is crucial and patient optimization prior to surgical intervention is important. Decreasing the pressure gradient between the surgical site and right atrial pressure is the ultimate goal, and can be achieved by maintaining hydration, mechanical ventilation with positive end-expiratory pressure (PEEP), and intermittent manual application of bilateral jugular compression during higher risk aspects of a procedure.[1] Intravenous volume loading prior to surgery to achieve a an RAP of 10 – 15 mm Hg can optimize risk reduction.[1,3] The utilization of PEEP can also be effective, however risks such as a decrease in cardiac output must be considered .[1]

Once VAE is detected, the primary treatment goals include elimination of air entrapment into the venous circulation and resuscitation to achieve hemodynamic stability.[1] The surgeon should be immediately notified to flood the surgical field with saline.[1] Other interventions include jugular vein compression, aspiration of a right atrial catheter if present, positioning to ensure the heart is above the surgical site, and administration of inotropic drugs such as calcium and epinephrine to minimize patient mortality and morbidity.[1-3] In this exampled case, air entrainment was

halted within minutes through compression of the scalp and hemodynamic stability achieved through inotropic medications and crystalloid and blood administration.

This case demonstrates that VAE can develop rapidly and because of this, early detection and monitoring are pertinent to achieve optimal outcomes. Oftentimes, VAE remains undiagnosed related to the fact that sensitive monitoring devices are not routinely employed in cases where there is a low level of suspicion of development as occurred in the case of the patient presented in this discussion.[2] Other case reports have also demonstrated the occurrence of VAE in neonates undergoing scalp vein cannulations.[2] To avoid adverse outcomes, vigilant identification and treatment including flooding the field with saline, placing the patient in Trendelenburg position, transient venous compression, and hemodynamic support must be employed immediately upon detection.[1,2,5,6,7] This case raises awareness of the possibility for VAE during highly vascularized flap procedures. The use of non-invasive and invasive devices capable of detecting a VAE may assist anesthesia professionals and surgical teams in early detection and intervention.

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Anesthesia Crises Resource Management: Anaphylaxis Simulation

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Keywords: anaphylaxis, Simulation, Education, Evaluation, Anesthesia, Student, Crises Resource Management.

Simulation scenarios are used extensively by educators worldwide to promote education and patient safety. Starting with Resusci Annie in the 1960s, the use of simulation has spanned the realms of anesthesia and the airline industry and has proven to be a valuable form of pedagogy for learners in both professions.[1-4] Over the years, a need for trainees to experience rarely occurring events with critical outcomes has evolved. A need for anesthesia crises resource management (ACRM) scenarios was identified.

Methods

This quasi experimental and qualitative research design used methods guided by the Kellogg Logic Model and ACRM literature.[5-8] Institutional review board approval was obtained and the proposal was deemed a quality improvement project. The study was performed in a high fidelity simulation laboratory. The target group was a convenience sample of student registered nurse anesthetists (SRNA). Quantitative evaluation was performed with pre-simulation and post-simulation tests. These tests were developed using the performance indicators from the World Allergy Organization guidelines.[8] The qualitative test was based on a 1-4 Likert scale. The questions included: (1) Was the experience positive, (2) Was the scenario believable, and (3) Do you feel more confident handling an anaphylaxis scenario in the operating room. A pre-brief paper and orientation was developed to orient the SRNAs to the clinical environment, stem of the scenario, and concepts of ACRM. A debriefing was developed to teach core concepts and provide time for reflection. The posttest followed the debriefing

Results

Twenty SRNAs attended the simulation scenario. The pretest results displayed an overall average of 5.55/11 (50.45%) questions correct. The posttest results displayed an overall average of 9.85/11 (89.54%) questions correct ($p<0.014$). The qualitative portion of the posttest received 17 student responses. On average the 17 students that responded agreed to strongly agreed that the experience was positive (avg. 3.76), believable (avg. 3.7), and they felt more confident handling an anaphylaxis situation in the operating room (avg. 3.65).

Discussion

The statistically significant results show that pre-test and post test scores improved after simulation and debriefing. The null hypothesis, which stated posttest simulation exam scores would not differ compared to pretest scores, was rejected. Of value, SRNAs felt more confident after handling the anaphylaxis scenario. The significance of this data reflects the efficacy of simulation to improve SRNAs confidence of handling a rarely occurring but deadly clinical

situation. Weaknesses of this study include size of sample in respect to global SRNA population and data collection regarding ACRM. Future recommendations include larger sample size, better blinding of students to the scenario, and assessment of resource management and/or time to identification of problem.

Anesthesia is a profession in which unexpected and critical situations arise without warning. Rarely occurring, but harmful events in anesthesia must be practiced and prepared for in a manner that leaves the practitioner able to intervene at any moment. This study suggests that simulation of rarely occurring but harmful events in anesthesia may be effectively taught to SRNAs while improving their confidence.

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Esophageal Doppler Monitoring during Colorectal Surgery

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Keywords: esophageal doppler monitoring, conventional hemodynamic monitoring, goal-directed fluid therapy, colorectal resection, length of hospital stay

Introduction

Intravascular volume status is highly subjective among anesthesia professionals and difficult to measure with standard monitoring equipment. In patients undergoing major abdominal surgery, strict management of intravascular volume is necessary to avoid both hypo- and hypervolemia,

and the associated adverse outcomes.[1] Reduced circulating volume may lead to splanchnic vasoconstriction resulting in impaired tissue perfusion and decreased oxygen.[1] Furthermore, prolonged preoperative fasting and bowel preparation may contribute to electrolyte disturbances.[1] Large quantities of crystalloid are often given during major abdominal surgeries to avoid complications of hypovolemia.[1] Gross tissue edema from fluid overload may result in pulmonary edema, cardiac compromise, impaired wound healing, and prolonged ileus.[2]

Controversy exists in the literature regarding optimal perioperative fluid administration.[2] Fluid management is diverse among anesthesia professionals. Lilot et al.[3] demonstrated the variability through a retrospective observational study of 5,912 patients who had uncomplicated, elective abdominal surgery with minimal estimated blood loss (EBL). The final regression model showed practitioner variability was a greater determinant of fluid administration than physiologic factors such as mean arterial pressure (MAP) and heart rate (HR). The volume of crystalloid fluid delivered ranged from 2.3 (SD 3.7) to 14 (SD 10) ml/kg/hr for similar patients.

Fluid management using conventional hemodynamic monitoring as a guide, including HR, blood pressure (BP), and urine output, poses a challenge to the anesthesia practitioner. Signs of hypovolemia are delayed, or absent, with conventional monitoring due to homeostatic mechanisms.[1] Stroke volume (SV) is an early indicator of fluid status.[1] However, traditional invasive monitoring with a pulmonary artery catheter (PAC) has been shown to increase complications.[1,2] Due to the inadequacies of PACs, non-invasive methods of monitoring SV to guide fluid administration, such as esophageal doppler monitoring (EDM), are gaining favor.

Esophageal doppler monitoring provides beat-to-beat analysis of SV and cardiac output (CO), allowing for early detection and modification of volume status.[1] EDM has been shown to correlate well with invasive methods, specifically thermodilution with a PAC.[1] On May 22, 2007, the Centers for Medicare and Medicaid Services stated EDM use for "…operative patients with a need for intra-operative fluid optimization is reasonable and necessary."[4] The statement is based on a meta-analysis conducted by the Agency for Healthcare Research and Quality.[5] Seven randomized studies, totaling 583 patients, were evaluated to assess whether the addition of EDM, or replacement of central venous pressure (CVP) with EDM, was associated with improved outcomes and reduced length of stay (LOS).[5] A 2 to 4 day reduction in hospital stay was found for colorectal surgery patients monitored with EDM.[5]

Significance

A retrospective analysis of LOS for 23,098 colorectal surgical patients between 2006 and 2007 was conducted utilizing data reported to the American College of Surgeon's National Surgical Quality Improvement Program.[6] The mean LOS for all patients was 7.4 days.[6] In contrast, the mean LOS for patients who experienced at least one postoperative complication increased to 16.1 days.[6] Another retrospective study analyzed University Health System Consortium data from 70,484 colorectal surgical patients who had surgery between 2008 and 2011.[7] A 30-day readmission rate of 13.7% was reported.[7] Of these, 13% required admission to the intensive care unit, 6% returned to the operating room, and 2% died during the readmission.[7]

Statement of Purpose
Patients undergoing colorectal surgery are at increased risk of complications related to fluid volume imbalances. The financial burden associated with complications and a prolonged LOS following colorectal surgery indicates a need for change in current practice. The purpose of this evidence-based practice analysis is to determine if implementing goal-directed fluid therapy (GDFT) with EDM reduces complications and LOS.

Methodology

Question Format
The population, intervention, comparison, and outcome (PICO) format was utilized to frame the posed question: In adult, non-emergent colorectal surgery patients (P), does goal-directed fluid resuscitation with esophageal doppler monitoring (I), compared with fluid therapy guided by conventional monitoring (C), reduce complications and the total length of hospital stay (O)?

Search Methods
A literature search was conducted using PubMed, CINAHL, and MEDLINE databases. Specific keywords utilized included: esophageal doppler, non-invasive monitoring, intraoperative fluid therapy, length of stay, goal-directed fluid, colorectal, and bowel. Studies evaluating EDM use for GDFT in colorectal surgical patients were reviewed. Studies involving children were excluded. Studies comparing conventional monitoring to GDFT using any device other than EDM (such as NICOM, LiDCO, and pulse pressure variation) were excluded. The studies included were published from 2006-2016.

Levels of Evidence
After the exclusion criteria above were considered, the database search yielded four recent original research articles meeting inclusion criteria. Of these, two were randomized control trials (Level II evidence) and two were cohort studies (Level IV evidence).[8-11]

Literature Review

In a double-blinded, randomized controlled trial, Noblett et al.[8] analyzed 108 elective colorectal resection patients. Exclusion criteria included: severe esophageal disease, recent esophageal or upper airway surgery, systemic steroid medication, moderate or severe aortic valve disease, bleeding diathesis, and patient choice. A power analysis from pilot data suggested a sample size of 108 patients to detect a 3-day reduction in LOS, with a power of 0.8 and significance level of 0.05. No significant differences between the two groups' demographics, risk indices, or duration and type of surgery. All patients received general anesthesia with volatile anesthetic agents. Conventional monitoring was used for all patients, including SpO2, end tidal CO2, electrocardiogram, and non-invasive BP. Patients in both groups may have had invasive monitoring at the discretion of anesthesia practitioner. An EDM probe was placed in all patients by a qualified researcher without any involvement in postoperative care or decision making.

Anesthesia professionals were blinded to hemodynamic values from the EDM. A researcher infused colloid boluses to the EDM group to obtain and maintain optimal SV and corrected flow time (FTc) greater than 0.35 seconds. A strict algorithm was followed: initial colloid bolus of 7

mL/kg was given if the initial FTc was less than 0.35 seconds; if FTc was at least 0.35 seconds, 3 mL/kg of colloid was given to evaluate SV response. Subsequent colloid boluses at 3 mL/kg were given if the FTc decreased to less than 0.35 seconds, if SV decreased greater than 10%, or if SV increased greater than 10% with the previous bolus in the absence of FTc greater than 0.4 seconds. The primary outcome was LOS, with secondary outcomes including morbidity, return of gastrointestinal function, and cytokine inflammatory markers. Clavien-Dindo complication classification was utilized: Grade 1- any deviation from normal without the need for treatment or intervention; Grade 2- pharmacologic treatment needed; Grade 3- surgical, endoscopic, or radiologic intervention required; Grade 4- life-threatening complication requiring high-dependency unit or intensive care unit care; Grade 5- death. Grade 4 complications were significantly lower in the treatment group, (0 versus 4, P= 0.042). Readmission rates were significantly lower in the treatment group (0 versus 6 patients, P=0.012). There was no significant difference in total fluid or crystalloid administration between the two groups. MAP and HR were similar throughout the surgery for both groups, and neither demonstrated prolonged systolic BPs below 90 mm Hg; however, at the end of surgery, SV and CO were considerably higher in intervention group (P= 0.039, P=0.031 respectively). Additionally, more vasopressors were used in the control group (26 versus 14 patients, P=0.015).

In a multi-center cohort study, Kuper et al.[9] compared 1,307 patient outcomes at three large hospitals before and after EDM implementation. Data was collected from various surgeries, including colorectal, urological, vascular, orthopedic, and organ transplant. At Royal Derby Hospital, only elective and urgent colorectal surgery patients were included, totaling 402 patients. Two limitations of this study are noted: 1. exclusion criteria are not described and 2. the study has a non-randomized, non-blinded design. Patient demographics between the groups were similar. A strict algorithm for fluid administration was followed in the EDM group. Following EDM probe insertion, initial SV measurement was noted, and 200-250 mL colloid bolus was infused over 5-10 minutes. If SV increased greater than 10%, additional boluses were given in the same manner until the increase in SV was 10% or less- the point at which SV was optimized. Throughout the surgery, additional colloid boluses were given if the SV decreased by 10% below the optimal value. The amount of crystalloid administration was not significantly different between the two groups. Complication rates were not compared between the control and treatment groups. The LOS for colorectal surgery patients was reduced by 2.5 days in the treatment group at Royal Derby Hospital (8.4 versus 10.9 days, P=0.007). Similarly, the overall LOS for all patient population types was reduced by 3.6 days in the treatment group (18.7 versus 15.1 days, P= 0.002).

A randomized controlled trial by Zakhaleva et al.[10] compared EDM outcomes in elective bowel resection patients. A pre-randomized, controlled pilot study suggested a need for 99 patients to detect a 1.5 day difference in LOS, with a power of 0.95 and significance of 0.05. Exclusion criteria included: admission as emergency surgery, failure to sign consent, patient choice, recent esophageal or upper airway surgery, moderate or severe aortic valve disease, or congestive heart failure. Additionally, some cases were excluded due to cancelled or failed surgery, or the inability to place the EDM probe, or collect required data. The final sample size for the control and treatment groups was 40 and 32, respectively. There was no difference between the groups' demographics or surgical factors, such as duration, incision length, and EBL. All surgeries were performed by one of four attending surgeons; the anesthesia practitioners were not blinded to the

study group. The EDM probe was placed by the anesthesia practitioner in the treatment group. The same fluid administration algorithm described by Noblett et al.[8] was utilized. There was no difference in total fluid administration between the two groups (P=0.15, P=0.53, respectively). Assessed complications included: ileus, acute renal failure, evisceration, urinary retention, bleeding, pulmonary embolism, superior mesenteric vein thrombus, anastomotic leakage, infection, and supraventricular tachycardia. The overall complication rate was significantly lower in the EDM group (7 versus 19 patients, P=0.022). There was an insufficient sample size to find a significant difference in LOS (treatment- 6 days, control- 5 days, P= 0.575).

A prospective cohort study by Srinivasa et al.[11] analyzed EDM outcomes in 81 patients, with 30-day complication rates as the primary outcome, and LOS and perioperative fluid administration as secondary outcomes. Exclusion criteria included: acute operations, multivisceral resections, patient refusal, severe bleeding diathesis, severe esophageal disease, recent esophageal surgery, and moderate to severe aortic valve disease. The sample size for the control group was 54 and for the treatment group was 27. No difference was found in patient demographics or surgical factors between the two groups. All surgeries were performed by one of three surgeons. All patients received general anesthesia with a volatile anesthetic agent and a thoracic epidural for analgesia, unless contraindicated. Conventional hemodynamic monitoring was used for all patients, with invasive BP monitoring used at the anesthesia practitioners' discretion. A trained research assistant, who provided no input for other perioperative care, placed an EDM probe for subjects in the EDM treatment group. The same fluid administration algorithm described by Noblett et al.[8] was utilized. Fluid was administered to control group patients per the anesthesia practitioners' preference. The Clavien-Dindo complication classification described in Noblett et al.[8] was utilized. Complication rates for grade 1, 2, 3, and 5 were higher in the control group, while grade 4 complications were higher in the treatment group; however, none of the differences were statistically significant. The LOS was decreased by one day in the EDM group, this was not enough to be significant (9 versus 10 days, P=0.92).

Table 1. Recent literature on esophageal Doppler monitoring during colorectal surgery

Author	Design	Outcomes	Sample	Results
Noblett et al.,[8] 2006	Randomized control trial, double blinded EDM: CardioQ- (Deltex Medical, Chichester, UK)	Primary: Length of postoperative stay Secondary: Morbidity, return of bowel function, cytokine markers	108 elective colorectal resection patients Control group: n=54 Treatment group: n=54	Major complications (grade 3-5): Control group: 15% Treatment group: 2% *(P= 0.043)* LOS: Control group: 9 days Treatment group: 7 days *(P= 0.005)* Reduction of both LOS and major complications were significant for EDM treatment group
Kuper et al.,[9] 2011	Multi-center cohort study EDM: CardioQ- (Deltex	Primary: Length of hospital stay Secondary: In-hospital	1,307 patients at 3 hospitals: colorectal, urological, vascular, orthopedic, organ transplant Control group: n=658 Treatment group: n= 649	Overall LOS: Control group: 18.7 days Treatment group: 15.1 days *(P= 0.002)*

	Medical, Chichester, UK)	mortality, readmission, and reoperation rates	**Royal Derby Hospital: colorectal only** **Control group: n=201** **Treatment group: n= 201** Whittington Hospital: colorectal and orthopedic Manchester Royal Infirmary: wide variety	**Royal Derby LOS:** **Control group: 10.9 days** **Treatment group: 8.4 days** ***(P= 0.007)*** Reduction in LOS was significant for the EDM treatment group overall and for colorectal patients specifically
Zakhaleva et al.,[10] 2013	Randomized control trial EDM: CardioQ- (Deltex Medical, Chichester, UK)	Primary: Complication rate	91 elective bowel resection with primary anastomosis patients Final inclusion: Control group: n= 40 Treatment group: n=32	Overall Complications: Control group: 19 (49%) Treatment group: 7 (22%) *(P= 0.022)* LOS: Control group: 5 days Treatment group: 6 days *(P= 0.575)* Significant reduction in complications. No significant difference in LOS
Srinivasa et al.,[11] 2014	Prospective cohort study EDM: CardioQ- (Pharmaco Inc, Auckland, NZ)	Primary: 30-day complication rate Secondary: Days to meet discharge criteria, LOS, and perioperative fluids	81 elective rectal resection patients Control group: n=54 Treatment group: n=27	Grade 4 Complications: Control group: 2 (4%) Treatment group: 4 (15%) *(P= 0.09)* LOS: Control group: 10 days Treatment group: 9 days *(P= 0.92)* No significant difference in LOS or complications

Conclusions

The goal of this evidence-based practice analysis was to determine if GDFT using EDM would reduce the LOS and complication rates for colorectal patients. Of the studies included, the two larger studies found a significant reduction in LOS by 2-2.5 days in the EDM group.[8,9] Another study showed a decreased LOS by 1 day.[11] However, this study had a small sample size with the treatment group comprising only about one-third of the total.[11] This study also was not randomized or blinded.[11] The final study had an insufficient sample size to accurately state LOS results, based on the power analysis.[10] Of the studies reviewed, three analyzed complication rates.[8,10,11] Two of these studies found a significant reduction in complications.[8,10] Noblett et al.[8] noted a 13% decrease in major complications (Clavien-Dindo grades 3-5). Zakhaleva et al.[10] found a 27% decrease in overall complications. One study also reported similar conventional hemodynamic values between the two groups throughout surgery, with an increased SV and reduced need for vasopressors in the EDM group.[8] This study supports that early signs of hypovolemia are absent with conventional monitoring, and are an insufficient assessment of circulatory status.[1, 8]

This evidence-based practice analysis is limited due to the small number of recent studies in this population. A meta-analysis of five earlier RCTs, published between 2002 and 2006, analyzed the effect of EDM versus conventional monitoring on average LOS for patients undergoing elective major abdominal surgery, including colorectal and gastro-intestinal procedures.[1] All of these trials found a significant reduction in LOS and complications.[1] The pooled analysis showed a weighted mean difference in LOS to be 1.6 days less for the EDM group, with an overall complication rate of 24%, versus 47% in the control group.[1] While additional, current research is desired, this analysis provides promising evidence that GDFT with EDM for colorectal resection patients will lead to decreased adverse outcomes and a shorter length of hospital stay.

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Mentor: Amber Davies, CRNA, DNP

Editorial

I'd like to wish everyone a Happy New Year! Reflecting back on 2016, one accomplishment that stood out in terms of the ISJNA was the Summer issue – our largest and most diverse issue yet with 24 total articles published, including 19 case reports, 1 abstract, and 4 evidence-based practice analysis reports. I'd like to thank my editorial board members and reviewers for all of their continued hard work – time is such a precious resource. I always wish I had more of it, so I am grateful for the contributions of my colleagues in supporting the ISJNA and furthering its mission.

We also had the opportunity to honor the founding editor of the ISJNA, Ronal Van Nest, CRNA, JD at the 2016 AANA Annual Congress in Washington, DC. His vision has led to a legacy that has benefited so many individuals and (I think :-) strengthened our profession.

Sincerely,

Vicki C. Coopmans, CRNA, PhD
Editor

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

To access prior issues of the ISJNA visit the following link:
www.aana.com/studentjournal

INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA
GUIDE FOR AUTHORS

MISSION STATEMENT

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

ITEMS ACCEPTED FOR PUBLICATION

Case reports, research abstracts, evidence-based practice (EBP) analysis reports, and letters to the editor may be submitted. These items must be authored by a student under the guidance of an anesthesia practitioner mentor (CRNA or physician). The mentor must submit the item for the student and serve as the contact person during the review process. Items submitted to this journal should not be under consideration with another journal. We encourage authors and mentors to critically evaluate the topic and the quality of the writing. If the topic and the written presentation are beyond the introductory publication level we strongly suggest that the article be submitted to a more prestigious publication such as the *AANA Journal.*

ITEM PREPARATION & SUBMISSION

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

The original intent of this journal was to publish items while the author is still a student. In order to consistently meet this goal, all submissions must be received by the editor at least **3 months prior** to the author's date of graduation.

PEER REVIEW

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

General guidelines

1. Items for publication must adhere to the *American Medical Association Manual of Style* (AMA, the same guide utilized by the *AANA Journal* and such prominent textbooks as *Nurse Anesthesia* by Nagelhout and Plaus). The review process will not be initiated on reports submitted with incorrect formatting and will be returned to the mentor for revision. Please note the following:
    a. Use of abbreviations is detailed in Section 14. Spell out acronyms/initialisms when first used. If you are using the phrase once, do not list the acronym/initialism at all.
    b. Instructions regarding units of measure can be found in Section 18. In most cases The International System of Units (SI) is used. Abbreviations for units of measure do not need to be spelled out with first use. Some examples: height/length should be reported in cm, weight in kg, temperature in ^{o}C, pressure in mm Hg or cm H_20.
    c. 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. Please use SpO_2 for oxygen saturation as measured by pulse oximetry.
    d. Use the nonproprietary (generic) name of drugs - avoid proprietary (brand) names. Type generic names in lowercase. When discussing dosages state the name of the drug, *then* the dosage (midazolam 2 mg).
    e. Use of descriptive terms for equipment and devices is preferred. If the use of a proprietary name is necessary (for clarity, or if more than one type is being discussed), give the name followed by the manufacturer and location in parenthesis:
        "A GlideScope (Verathon Inc., Bothell, WA) was used to . . . ."
        Please note, TM and ® symbols are not used per the AMA manual.

f. Examples of referencing are included later in this guide.

2. Report appropriate infusion rates and gas flow rates:
   a. When reporting infusion rates report them as mcg/kg/min or mg/kg/min. In some cases it may be appropriate to report dose or quantity/hr (i.e. insulin, hyperalimentation). If a mixture of drugs is being infused give the concentration of each drug and *report the infusion rate in mL/min*.
   b. Keep the gas laws in mind when reporting flow rates. Report the liter flows of oxygen and nitrous oxide and the percent of the volatile agent added to the gas mixture. Statements such as "40% oxygen, 60% nitrous oxide and 3% sevoflurane" do not = 100% and are thus incorrect. For example, "General anesthesia was maintained with sevoflurane 3% inspired concentration in a mixture of oxygen 1 L/min and air 1 L/min".
3. Only Microsoft Word file formats will be accepted with the following criteria:
   a. Font - 12 point, Times New Roman
   b. Single-spacing (except where indicated), paragraphs separated with a double space (do not indent)
   c. One-inch margins
   d. Place <u>one</u> space after the last punctuation of sentences. End the sentence with the period before placing the superscript number for the reference.
   e. Do not use columns, bolds (except where indicated), or unconventional lettering styles or fonts.
   f. Do not use endnote/footnote formats.
4. Do not use Endnotes or similar referencing software. Please remove all hyperlinks within the text.
5. Avoid jargon.
   a. *'The <u>patient</u> was reversed'* - Did you physically turn the patient around and point him in the opposite direction? "Neuromuscular blockade was antagonized."
   b. *The patient was put on oxygen.* "Oxygen was administered by face mask."
   c. *The <u>patient</u> was intubated and put on a ventilator*. "The trachea was intubated and respiration was controlled by a mechanical ventilator.
   d. *The patient had been on Motrin for three days.* "The patient had taken ibuprofen for three days."
   e. Avoid the term "MAC" when referring to a sedation technique - the term sedation (light, moderate, heavy, unconscious) sedation may be used. Since all anesthesia administration is monitored, the editors prefer to use specific pharmacology terminology rather than reimbursement terminology.
6. Use the words "anesthesia professionals" or "anesthesia practitioners" when discussing all persons who administer anesthesia (avoid the reimbursement term "anesthesia providers")
7. References
   a. Again, the **AMA Manual of Style** must be adhered to for reference formatting.
   b. All should be within the past 8 years, except for seminal works essential to the topic being presented.
   c. Primary sources are preferred.
   d. All items cited must be from peer-reviewed sources – use of internet sources must be carefully considered in this regard.
   e. Numbering should be positioned at the one-inch margin – text should begin at 1.25".
8. See each item for additional information.
9. **Heading** for each item (Case Report, Abstract, EBPA Report) must adhere to the following format:

Title (bold, centered, 70 characters or less)
[space]
Author Name (centered, include academic credentials only)
Name of Nurse Anesthesia Program (centered)
[space]
Anticipated date of graduation (italics, centered, will be removed prior to publication)
E-mail address (italics, centered, will be removed prior to publication)
[space, left-justify from this point forward]
Keywords: ('Keywords:' in bold, followed by keywords (normal font) that can be used to identify the report in an internet search.)

Case Reports

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

Heading (see #9 above in General Guidelines)

[space]

A brief introductory paragraph of less than 100 words to focus the reader's attention. This may include historical background, demographics or epidemiology (with appropriate references) of the problem about to be discussed. It is written in the *present tense*. Although it is introductory, the heading word '***Introduction'*** is not used. Be certain to cite references in this section, especially statistics and demographics pertaining to your topic.

[space]

Case Report (bold, 400-500 words)

[space]

This portion discusses the case performed in *400 words or less*, and is written in the *past tense*. Do not justify actions or behaviors in this section; simply report the events as they unfolded. Present the case in an orderly sequence. Some aspects need considerable elaboration and others only a cursory mention.

- Patient description: height, weight, age, gender.
- History of present illness
- Statement of co-existing conditions/diseases
- Mention the current medications, generic names **only**. (Give dosage and schedule only if that information is pertinent to the consequences of the case.)
- **Significant** laboratory values, x-rays or other diagnostic testing pertinent to the case. Give the units after the values (eg. Mmol/L or mg/dL).
- Physical examination/Pre-anesthesia evaluation - **significant** findings only. Include the ASA Physical Status and Mallampati Classification **only** if pertinent to the case.
- Anesthetic management (patient preparation, induction, maintenance, emergence, post-operative recovery).

Despite the detail presented here it is only to help the author organize the structure of the report. Under most circumstances if findings/actions are normal or not contributory to the case then they should not be described. Events significant to the focus of the report should be discussed in greater detail. The purpose of the case report is to set the stage (and 'hook' the reader) for the real point of your paper which is the discussion and teaching/learning derived from the case.

[space]

Discussion (bold, 600-800 words)

[space]

Describe the ***anesthesia*** implications of the focus of the case report citing current literature. Describe the rationale for your actions and risk/benefits of any options you may have had. This section is not merely a pathophysiology review that can be found in textbooks. *Relate the anesthesia literature with the conduct of your case noting how and why your case was the same or different from what is known in the literature.* Photographs are discouraged unless they are essential to the article. Photos with identifiable persons must have a signed consent by the person photographed forwarded to the editor via first class mail. Diag must have permission from original author. This is the most important part of the article. In terms of space and word count this should be longer than the case presentation. End the discussion with a summary lesson you learned from the case, perhaps what you would do differently if you had it to do over again.

[space]

References (bold)

[space]

A minimum of 5 references is recommended, with a maximum of 8 allowed. No more than 2 textbooks may be included in the reference list, and all references should be no older than 8 years, except for seminal works essential to the topic. This is also an exercise in evaluating and using current literature.

[space]

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

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

Research Abstracts

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

Heading (see #9 above in General Guidelines)
[space]
Introduction (bold)
[space]
A brief introductory paragraph including purpose and hypotheses.
[space]
Methods (bold)
[space]
Include research design and statistical analyses used
[space]
Results (bold)
[space]
Present results – do not justify or discuss here.
[space]
Discussion (bold)
[space]
Discuss results
[space]
References (bold)
[space]
Not required, but a maximum of 5 references is allowed.
[space]
Mentor: (bold, followed by mentor name and credentials in normal text)
E-mail address (italics, will be removed prior to publication)

EBP Analysis Reports

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

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

The same general format guidelines apply with the exception of the section headings as below. Please note that text books and non-peer reviewed internet sources should be avoided, and sources of reference should be less than 8 years old unless they are seminal works specifically related to your topic of inquiry:

Heading (see #9 above in General Guidelines)
[space]
Introduction (bold)
[space]
Briefly introduce the reader to the practice issue or controversy, describe the scope or significance or problem, and identify the purpose of your analysis. Describe the theoretical, conceptual, or scientific framework that supports your inquiry.
[space]
Methodology (bold)
[space]

Include the format used for formulating the specific question you seek to answer, search terms and methods used, and levels of evidence.
[space]
Literature Analysis (bold)
[space]
Review and critique the pertinent and current literature, determining scientific credibility and limitations of studies reviewed. Your synthesis table would be included in this section. Your review and discussion of the literature should logically lead to support a practice recommendation. Subheadings may be used if desired.
[space]
Conclusions (bold)
[space]
Summarize the salient points that support the practice recommendation and make research-supported recommendations that should improve the practice issue, while also acknowledging any limitations or weaknesses
[space]
References [bold]
[space]
A minimum of 8 references is recommended, with a maximum of 12 allowed.

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

AMA MANUAL OF STYLE

The following is brief introduction to the *AMA Manual of Style* reference format along with some links to basic, helpful guides on the internet. The website for the text is http://www.amamanualofstyle.com/oso/public/index.htmL. It is likely your institution's library has a copy on reserve.
http://www.docstyles.com/amastat.htm#Top
http://healthlinks.washington.edu/hsl/styleguides/ama.htmL

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

The International Student Journal of Nurse Anesthesia (ISJNA) is not listed in the PubMed Database. For the purpose of citing the ISJNA *in this Journal* use "**Int Student J Nurse Anesth**" as the abbreviation. The titles of text books are also printed in *italics*. Please pay close attention to ensure correct punctuation.

Journals
Note there is a comma after the first initials until the last author, which has a period. If there are six or less authors **cite all six**. If there are more than six authors **cite only the first three** followed by "et al." Only the first word of the title of the article is capitalized. The first letters of the major words of the journal title are capitalized. There is no space between the year, volume number, issue number, and page numbers. If there is no volume or issue number, use the month. If there is an issue number but no volume number use only the issue number (in parentheses). The pages are inclusive - **do not omit digits.**

Some journals (and books) may be available both as hard copies and online. When referencing a journal that has been accessed online, the DOI (digital object identifier) or PMID (PubMed identification number) should be included (see example below).

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

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

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

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

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

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

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

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

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

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

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

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

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

own words) or summary (a more concise restatement of another's ideas) must be properly cited." http://grad.georgetown.edu/pages/reg_7.cfm

HOW TO SUBMIT AN ITEM

Manuscripts must be submitted by the mentor of the student author via e-mail to **INTSJNA@aol.com** as an attachment. The subject line of the e-mail should be "Submission to Student Journal". The item should be saved in the following format – two-three word descriptor of the article_author's last name_school abbreviation_mentor's last name_date (e.g. PedsPain_Smyth_GU_Pearson_5.19.09)

REVIEW AND PUBLICATION

If the editor does not acknowledge receipt of the item within one week, assume that it was not received and please inquire. Upon receipt, the Editor will review the submission for compliance with the Guide to Authors. If proper format has not been following the item will be returned to the mentor for correction. This is very important as all reviewers serve on a volunteer basis. Their time should be spent ensuring appropriate content, not making format corrections. It is the mentor's responsibility to ensure formatting guidelines have been followed prior to submission.

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

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

Mentors of the papers may be asked to serve as reviewers of case reports by student authors from other prog and will be listed as contributing editors for the issue in which the item is published.

PHOTOS

Photos of students for the front cover of the Journal are welcome. Include a legend describing the activity and who is in the photo and identify the photographer. Only digital photos of high quality will be accepted via email to INTSJNA@aol.com. There must be a follow up hard copy signed by all present in the photo, as well as the photographer/ owner of the original photo, giving consent to publish the photo. Mail that consent to:

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

SUBMISSION CHECK LIST

___ **AMA Manual of Style and other format instructions are adhered to**.
___ Total word count not exceeded (1400 for case report, 500 for abstract, 3000 for EBPA).
___ The item is one continuous Word document without artificially created page breaks.
___ Verbatim phrases and sentences are quoted and referenced.
___ All matters that are not common knowledge to the author are referenced.
___ 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 is absent.

Heading
___ Concise title less than 70 characters long
___ Author name, credentials, nurse anesthesia program, graduation date and email are included.
___ 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 400-500 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.

Abstract
___ The 500 word count maximum is not exceeded.
___ Abstract reports the *outcome* of your study.
___ Includes Introduction, Methods, Results, and Conclusion sections.

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 and population is presented.
___ A focused foreground question following either the PICO or SPICE format is used.
___ Includes Introduction, Methodology, Literature Analysis, and Conclusion sections.

References
___ AMA Style for referencing is used correctly.
___ Reference numbers are sequenced beginning with one and superscripted.
___ References are from anesthesia and other current primary source literature.
___ All inclusive pages are cited, texts as well as journals.
___ Journal titles are abbreviated as they appear in the PubMed Journals Database.
___ Number of references adheres to specific item guidelines.
___ 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)
___ It is submitted by the mentor with cc to the student author
___ The words "Submission to Student Journal" are in the subject heading.