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TOPICS IN THIS ISSUE

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Front Cover:

Angela DeLaria, RN, BSN, a graduate student enrolled in the Webster University nurse anesthesia program (NAP), received the 2015 AANA Student Excellence Award for demonstrating outstanding leadership and professionalism during her matriculation. Ms. DeLaria also has a case report published in this issue of the ISJNA.

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Anesthetic Considerations of the Obese Parturient in Cesarean Delivery

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Keywords: obstetric anesthesia, morbid obesity, pregnancy, cesarean section, complications

Obesity and pregnancy pose significant anesthetic risks and increased morbidity and mortality, based on changes to the cardiovascular and pulmonary systems and higher incidence of coexisting diseases. The obesity epidemic in the United States continues to increase with over 30% classified as obese.[1] The significant physiologic changes of obesity place parturients at a higher risk for gestational diabetes, hypertension, pre-eclampsia, venous thromboembolism, and infection.[2] The obese parturient also poses greater technical challenges for the anesthesia professional when performing neuraxial anesthesia. Anesthetic management of the obese parturient benefits from a multidisciplinary approach with careful planning to reduce complications and improve outcomes.[3]

Case Report

A 38-year-old, 170 cm, 188 kg (body mass index = 65 kg/m^2) female presented for an elective cesarean section at 39 weeks and three days gestation. She had a history of morbid obesity, kidney stones with kidney stents, and gestational hypertension. Her medications included prenatal vitamins. The patient stated ability to walk flat and able to climb stairs slowly, equaling four metabolic equivalents (METs), limited by pelvic pain and dyspnea on exertion. Her airway exam revealed a Mallampati I, full range of motion of neck, and thyromental distance >6 cm. Spinous process and iliac crest landmarks for epidural placement were difficult to locate. A peripheral intravenous (IV) catheter was inserted in her right hand and right foot for medication and fluid access.

Once in the operating room, noninvasive monitors were placed on the patient and oxygen was administered via facemask at 10L/min. For epidural insertion the patient was placed in the sitting position, and tape was used to retract excess tissue to improve visualization of the vertebral midline. After sterile preparation and draping, a 17 gauge Tuohy needle was inserted between the spinous processes of $L_{3\text{-}4}$, and loss of resistance with saline occurred at 9 cm. The catheter was threaded into the epidural space 7 cm without difficulty. The epidural test dose of lidocaine 1.5% with epinephrine 1:200K was negative for venous/intrathecal injection. The patient was positioned supine with left uterine displacement and head slightly elevated. The epidural was dosed with lidocaine 2% in divided doses of 5 mL each, for a total of 20 mL administered. Twenty minutes later the sensory level was assessed at dermatome T_4 bilaterally. A phenylephrine infusion was available to treat decreases in blood pressure.

Prior to incision, the patient received cefazolin 4 g IV for infection prophylaxis, heparin 10,000 units SQ for prevention of clot formation, and citric acid/sodium citrate 30 mL PO to decrease stomach acidity. The patient's abdomen was retracted and secured with tape to improve access to the surgical site. A male infant was delivered 18 minutes after skin incision. The patient then became anxious and complained of pain. Ketamine 70 mg, midazolam 2 mg, and fentanyl 200

mcg IV were administered. After delivery of the placenta, 60 units of oxytocin in 1 L of normal saline was initiated. The patient also received lactated ringers 2,000 mL and ondansetron 4 mg IV. The estimated blood loss was 1,200 mL. The patient's abdomen was closed, a wound vacuum was applied, and an abdominal binder placed. The patient and neonate were transferred to recovery in stable condition, and discharged from hospital three days postoperatively with no complications.

Discussion

Anesthesia considerations of a patient who is morbidly obese and pregnant requires understanding the physiologic changes to the pulmonary, cardiovascular, and gastrointestinal systems, as well as preparing for the anatomical challenges obesity creates for neuraxial anesthesia.[2] Concerning the pulmonary system, obesity increases oxygen consumption, work of breathing, and decreases chest wall expansion leading to an increased demand on the lungs. Pregnancy weight gain further decreases chest wall expansion and increases work of breathing. Combined with placing a patient supine or trendelenburg, which is necessary for surgical exposure, this can further compromise the pulmonary system, leading to increased risk for hypoxemia. The patient in this case was positioned supine with left lateral tilt and blankets were used to build a ramp to help facilitate breathing and to optimize the patient if general anesthesia with endotracheal intubation was warranted. Most researchers reported supine position with left lateral tilt with slight head up position to be optimal for the morbidly obese parturient.[2,5]

Airway changes associated with pregnancy include edema to the upper airway, trachea, and vocal cords, which increases the risk of a difficult airway.[4] Obesity also creates a challenge to airway management due to the abundant soft tissue of the upper airway. Neuraxial anesthesia is the preferred method of anesthesia due to the higher risk of complications associated with general anesthesia in the morbidly obese parturient.[2,4,5] When comparing spinal and epidural blocks, spinal anesthesia has a higher risk of an amplified spread of local anesthetics with variable block levels, especially in the obese population.[3] In this case, an epidural was performed to minimize chance of manipulation of the airway associated with unpredictable block levels.

Major changes to the cardiovascular system in obesity and pregnancy include rises in cardiac output and blood volume, which can increase the risk for hypertension and left ventricular hypertrophy. Aorto-caval compression that commonly occurs with pregnancy can be exaggerated in the obese patient due to the large abdomen compressing the vasculature and further decreases blood flow to the uterus. This is why left uterine displacement is necessary for adequate blood flow.[2,4] In this case, the patient was positioned gradually to minimize changes in hemodynamics, and a phenylephrine infusion was available to treat changes in blood pressure. To avoid sudden decreases in blood pressure associated with spinal anesthesia, an epidural anesthetic was utilized, and dosed slowly to achieve the appropriate dermatome level for the surgical procedure, with close monitoring of blood pressure. Neuraxial anesthesia is the optimal method for analgesia as it is effective in minimizing oxygen consumption and changes in cardiac output associated with pregnancy.[3] Epidural anesthesia provides advantages over spinal anesthesia for Cesarean delivery in the obese parturient due to the unknown duration of surgery, ability to redose for effective analgesia, less chance for airway manipulation, and the gradual onset of anesthesia for

minimal changes in hemodynamics.[4] Despite choosing to place an epidural for this case, many researchers have reported success with spinal anesthesia, as well as combined spinal epidurals.[5]

Obese parturients are shown to have increased gastric contents and decreases stomach pH, increasing the risk of aspiration. [2] The patient in this case was given citric acid/sodium citrate 30 mL PO to increase stomach pH to decrease complications in case of pulmonary aspiration.[2,4] Compared to general anesthesia, neuraxial anesthesia is a safer and more commonly practiced method due to a decreased risk of pulmonary aspiration, as well as decreased manipulation of the airway.[6,7]

Despite the advantages of neuraxial over general anesthesia for Cesarean delivery, morbidly obese parturients present technical challenges for placement, which include difficulty in identifying anatomical landmarks, proper patient positioning, needle length to penetrate excess adipose tissue, and dose of local anesthetic.[6,8] Researchers have identified that enhancing patient position to improve the ability to identify anatomical landmarks increases chances for successful placement of the block.[7,8] Despite these increased challenges, researchers have determined little difference in failure rates of neuraxial anesthesia.[7] To facilitate success in this case, the patient was placed in the sitting position, and excess adipose tissue and skin was secured away from midline. Constant patient interaction also helped to identify midline and proper placement of the epidural needle.

Care for the morbidly obese parturients requires strategic planning, which involves understanding the physiological changes and challenges to provide a safe and appropriate anesthetic. Neuraxial techniques provide the obese parturient the safest mode of analgesia and anesthesia for Cesarean delivery. Despite the low failure rates for neuraxial placement in the obese patient, the anesthesia professional must be adequately prepared if block failure occurs and airway manipulation is required. Other options to consider include use of combined spinal epidural, for quicker onset of anesthesia and analgesia, as well as catheter placement for possible increased duration of surgery.

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Mentor: Victoria Goode, PhD, CRNA

IgA Nephropathy and Anesthetic Considerations

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Keywords: IgA Nephropathy, end-stage renal disease, corticosteroids, angiotensin-converting enzyme inhibitors, ketorolac

IgA nephropathy is the most common cause of idiopathic glomerulonephritis. Within 20 years of diagnosis, IgA nephropathy results in end-stage renal disease (ESRD) in 30-40% of affected patients.[1] Progression of IgA nephropathy can be predicted by elevated serum creatinine, hypertension, and persistent proteinuria. Intraoperative management of IgA nephropathy is dependent on the severity of kidney damage and medication regimen. Pharmacological treatment may include medications such as corticosteroids and inhibitors of the renin-angiotensin-aldosterone system (RAAS).[1,2] Intraoperative management must incorporate anesthetic considerations for patients with IgA nephropathy in conjunction with their current treatment regimen.

Case Report

A 44-year-old, 178 cm, 127 kg Caucasian male presented for right wrist hardware removal. His past medical history was significant for obesity, hypertension, psoriasis, and chronic kidney disease stage 3 secondary to IgA nephropathy. His surgical history included a left shoulder rotator cuff repair and an open reduction internal fixation of his right wrist. Both surgical procedures were without complications. His daily medication regimen included oral hydrochlorothiazide 25mg, lisinopril 40 mg, and a vitamin D supplement.

The patient's American Society of Anesthesiologists (ASA) physical status classification was designated to be III given his obesity and comorbidities of hypertension and IgA nephropathy. Notable laboratory values included a blood urea nitrogen level of 34 mg/dL, a creatinine level of 2.4 mg/dL, and a glomerular filtration rate (GFR) of 36 mL/min/1.73m^2.

Midazolam 5 mg was administered intravenously (IV) in the preoperative area. A time out verifying the patient's name, date of birth, and procedure was initiated upon his arrival to the operating room (OR). After transferring the patient onto the OR bed, a noninvasive blood pressure cuff, pulse oximeter, capnography, and an electrocardiogram were applied as standard ASA monitors. He was then preoxygenated with 100% oxygen (O2) at 8 liters per minute (L/min) via facemask. Induction of general anesthesia was initiated with the administration of fentanyl 50 mcg IV, lidocaine 100 mg IV, and propofol 200 mg IV. A #5 laryngeal mask airway (LMA) was inserted into the hypopharynx and placement was verified by end tidal CO_2, bilateral breath sounds, and positive chest rise. The LMA was connected to the breathing circuit and the patient maintained spontaneous ventilation with peak inspiratory pressures less than 20 cmH_20.

General anesthesia was maintained with desflurane at 6% inspired concentration and a mixture of O2 and air, both at 1 L/min. An additional fentanyl 50 mcg IV, ketorolac 30 mg IV, and ondansetron 4 mg IV were administered. An indwelling urinary catheter was not placed due to the short length of the procedure; urine output was not monitored. Estimated blood loss was 25 mL. Upon surgical completion, the LMA was removed once the patient was awake and emergence criteria was met. Emergence criteria included maintaining spontaneous ventilation on 100% O2 and the patient following commands. The patient was then transferred to the post-anesthesia care unit (PACU) on 100% O2 at 6 L/min via a simple facemask. Patient disposition was to home after three hours in the PACU.

Discussion

IgA nephropathy is the most common cause of primary glomerulonephritis in developing countries. Within the past fifteen years, research has focused on conducting randomized-controlled trials to discern optimal treatment modalities for this disease. Treatment goals include decreasing daily proteinuria and slowing the deterioration of renal function, most commonly measured by serum creatinine levels.[2] Current research focuses on two treatment modalities to manage this disease; corticosteroid therapy and angiotensin-converting enzyme inhibitors (ACE-I). Additionally, anesthesia providers must be judicious with intraoperative medication administration and potential deleterious effects on renal function. An example of one of those medications is ketorolac.

A meta-analysis conducted by Lv et al.[3] examined the literature regarding the use of steroid therapy in patients with IgA nephropathy. The authors concluded that immunosuppression with corticosteroid therapy could decrease the risk of developing ESRD by up to 66% in comparison to supportive measures or ACE-I therapy alone. Corticosteroid therapy also produced a statistically significant ($P<0.001$) reduction in proteinuria by -0.46g/d, which is indicative of kidney function preservation. However, steroid therapy is not a benign treatment option. An increase incidence of diabetes, weight gain, and cushingoid symptoms have been known to occur with steroid treatment.[3] In additiona, perioperative administration of supplemental exogenous steroids is highly debated within the anesthesia community. Since exogenously administered steroids suppress the response of the hypothalamic-pituitary axis, there may be an ineffective physiologic response to stressors like surgical stimulation. Without the physiologic rise in plasma cortisol levels, the risk of morbidity and mortality may increase. Recommendations from the ASA[4] suggest that if the patient receives routine corticosteroid therapy, their daily dose

should be administered on the day of surgery. Any additional stress-dose steroids should be based on the severity (minor, moderate, or major) of the surgical procedure. During the preoperative period, it is imperative to confirm if a patient with IgA nephropathy has received steroid therapy within one year of surgery. Correlating preoperative steroid use with the type of surgery will help determine the necessity for further steroid administration.

Researchers have also investigated the use of ACE-I for the treatment of IgA nephropathy. In comparison to the control groups, the ACE-I treatment groups showed a significant effect ($p<0.00001$) on protecting renal function and decreasing proteinuria without an increased risk for adverse events.[1] One major drawback of ACE-I therapy is hypotension during general anesthesia. If an ACE-I is administered within 8-24 hours of general anesthesia, intraoperative hypotension is more common.[5] Under normal conditions, the body recruits three endogenous vasopressor systems during periods of hypotension; the sympathetic nervous system (SNS), the RAAS, and the vasopressin system. When two or more of these systems are inhibited the body is unable to compensate during hypotension. For example, uncompensated hypotension may occur with blockade of the SNS under general anesthesia and ACE-I impedance of the RAAS. Patients who receive ACE-I preoperatively may only compensate via the vasopressin system. Therefore, hypotension may be unresponsive to first-line treatments such as fluid administration, ephedrine, and phenylephrine. Effective treatments may include IV vasopressin administration along with methylene blue infusion.[5]

The use of non-steroidal anti-inflammatory drugs (NSAID), such as ketorolac, is questionable in a patient with decreased kidney function. Prostaglandins dilate the afferent arteriole to sustain appropriate levels of renal perfusion. When this mechanism is blocked by the administration of NSAIDs, acute kidney injury can occur.[6] In patients with ESRD, decreasing renal perfusion with NSAIDs can increase the risk of further deterioration in renal function. A dose of ketorolac should be reduced by 50% when the GFR is 10-50 mL/min.[7] There is clear evidence that ketorolac can cause acute kidney injury, therefore creating the potential to compound the chronic injury in a patient with IgA nephropathy.

In this case study, the patient was not receiving a daily steroid regimen and his surgical procedure was classified as minor. Due to this criterion, it was not recommended that the patient receive any additional stress dose steroid coverage. The patient was currently receiving ACE-I therapy treatment. The risk of perioperative hypotension was increased as a result of his treatment regimen. Vasopressin or methylene blue could have been administered to treat intraoperative hypotension. Intraoperative ketorolac 30 mg IV was administered for additional pain control, which may have significantly decreased renal perfusion given his pre-existing disease. Due to his decreased GFR of 36 mLs/min/1.73m^2, a smaller dose of ketorolac 15 mg IV should have been administered. Monitoring urine output intraoperatively may have been beneficial in evaluating the status of his kidney function. There was no need for any further evaluation postoperatively. The patient successfully underwent surgical and anesthetic management. He was advised to follow up with his primary care physician should any concerns regarding his pre-existing renal function occur.

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Leiomyoma Removal: A Case Report

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Keywords: leiomyoma, uterine fibroid, gynecological surgery, abdominal mass, surgical blood loss

Leiomyoma's are the most common type of uterine fibroid occurring at a 20-25% incidence.[1] Considered a benign tumor, they are comprised of smooth muscle cells, fibrous tissue and collagen matrix. Leiomyoma's are the leading cause of primary hysterectomy and are the cause of several reproductive issues including, infertility, miscarriage, menorrhagia and abdominal pain.[1] In the United States, 200,000 out of the 600,000 hysterectomies performed are due to leiomyoma with healthcare costs estimated at over $2 billion annually.[2]

Case Report

A 38-year-old female presented with a large leiomyoma requiring surgical excision. She had limited access to healthcare and had delayed seeking care until the leiomyoma was quite large. She presented with abdominal pain and abnormal uterine bleeding persisting over the last two years, the fibroid had become so large that it was now impeding her activities of daily living. Preoperative evaluation showed an otherwise healthy 85 kg female with a large visible abdominal mass. She was not taking any medications, and her medical-surgical history included

a laparoscopic cholecystectomy and two uncomplicated pregnancies. Her hemoglobin (HGB) was 13.8 g/dL and hematocrit (HCT) 45%. A type and crossmatch was performed, and two units of packed red blood cells (PRBC) were placed on hold to the operating room. She had a negative human chorionic gonadotropin result, and all other labs were unremarkable.

Intravenous (IV) midazolam 2 mg and cefazolin 1 g were given and she was then brought to the operating room. Standard monitors were applied, and vital signs were normal. She was pre-oxygenated with O_2 10 L/min via facemask. General anesthesia was induced with fentanyl 150 mcg, propofol 200 mg, and rocuronium 30 mg. A 7.0 mm ID oral endotracheal tube was placed with a Macintosh 3 blade by direct laryngoscopy without event. The patient was placed on volume control ventilation with tidal volume 500 mL, respiratory rate of 10/min, positive end expiratory pressure of 5 cm H_2O, and in inspiratory to expiratory ratio of 1:2. Hydromorphone 1 mg was given for long-term pain control.

During dissection of the large leiomyoma, an artery supplying the fibroid was severed, resulting in the loss of approximately 3 L of blood in 30 minutes. During this time, blood was requested and an additional peripheral IV, as well as a right radial arterial line, were placed. During this time her blood pressure ranged in the 80's/40's mm Hg. Five liters of crystalloid and 250 mL of 5% albumin had been infused, followed by three units of PRBC's and one unit of fresh frozen plasma. A phenylephrine infusion was titrated to maintain mean arterial pressure (MAP) above 60 mm Hg.

The severed artery was cauterized and sutured controlling the bleeding and the leiomyoma was successfully removed, weighing in at over 3,500 g. Train of four count showed 4/4 twitches, and neuromuscular blockade was antagonized with neostigmine 3 mg and glycopyrrolate 0.6 mg. Spontaneous respirations were 16/min with tidal volumes of 200-300 mL. The trachea was extubated in the operating room and the patient was transferred to the post-anesthesia care unit. She remained hospitalized overnight. A repeat HGB/HCT were drawn and resulted at 7.8 g/dL and 24% respectively. The patient had an uneventful postoperative course and was discharged to home the following day.

Discussion

While obesity, hypertension, alcohol consumption and diets high in red meat are associated with leiomyoma occurrence, the precise pathophysiology behind their development remains unclear.[2] There are, however, a few theories regarding their etiology. There are two essential features of leiomyoma formation; an increase in smooth muscle proliferation, and an excess of extracellular deposition.[3]

A generally accepted theory of development is that cells of a leiomyoma originate from myoblasts in the uterine musculature, although clear progenitors have not been identified.[1] During the menstrual cycle, smooth muscle cells express both estrogen and progesterone. During the luteal phase of menstruation, myometrial smooth muscle cells demonstrate proliferative activity in the expectation of pregnancy.[1] If pregnancy does not occur, this activity normally ceases during menstruation; however, ischemic damage to these cells during menstruation could occur to the myometrial cells that remain in the proliferative phase resulting in damage to these

cells and possibly becoming the progenitor cells of leiomyoma.[1] The majority of injured cells should be eliminated as apoptatic cells or cycle arrested cells, but when injured cells survive they acquire a protective mechanism against oxidative stress and apoptosis, a similar response is seen when cellular injury occurs.[1] These injured smooth muscle cells respond to injury or ischemia with increased cell proliferation and production of extracellular matrix; both of which are critical to the pathogenesis of uterine leiomyoma.[1]

Several hormonal factors have also been implicated in the development of leiomyoma. Estrogen and progesterone have been considered to be the major hormonal factors in leiomyoma development, in addition to interactions between genes, growth factors, oxidative stress and cytokines.[3] Estrogen and estrogen metabolites are known pro-oxidants thus leading to their possible role in increasing genomic instability leading to tumor proliferation.[3]

Most leiomyomas occur during a woman's reproductive years, supporting progesterone's role in their development. They are rare in the postmenopausal period and existing leiomyomas regress after menopause occurs. Treatment of leiomyoma with progesterone results in an increase in cellularity and mitotic activity.[2] In postmenopausal women, treatment with estrogen also results in proliferation of leiomyoma.[2] Antiprogestins, such as mifepristone, are shown to effectively reduce uterine volume, bleeding and abdominal discomfort. Mifepristone is the only approved medical abortion pill in the United States, creating stigma towards its use in leiomyoma treatment. Compared to a normal myometrium, leiomyomas produce higher numbers of insulin receptors, insulin like growth factors, epidermal growth factors, and platelet derived growth factors inducing a rapid proliferation of leiomyoma cells.[2] Progesterone regulates expression of growth factor signaling proteins, increasing expression of anti-apoptotic genes and promoting transcription of leiomyoma cells.[2]

Another theory of leiomyoma development involves oxidative stress markers. Oxidation alters the structure and function of proteins.[3] A study conducted by Santulli et al. determined that women with leiomyomas have higher markers for oxidative stress measured by serum markers of thiols, advanced oxidative protein products (AOPP), carbonyls and nitrates than the control group. These findings support the hypothesis that oxidative stress can lead to development of leiomyomas.[3]

Thiols are the major organic compound that play a role in the body's defense of reactive oxygen species. In women with leiomyomas, thiol levels were found to be low. [3] Advanced oxidative protein products are carried by oxidized plasma proteins and are an indicator of the level of oxidative stress. The AOPP levels were found to be high. Carbonyl groups and nitrates are also markers of oxidative stress and both levels are also elevated in the leiomyoma group.[3]
In reference to this case, the factors making it unique were the sheer size of leiomyoma and the amount of blood loss that occurred during the dissection. The patient lost three liters of blood, which was replaced with 1,050 mL of PRBC's, 250 mL colloid and five liters of crystalloid.

Replacing blood loss is recommended when 10-20% of estimated blood volume has been lost.[4] According to the maximum allowable blood loss (MABL) formula, the MABL for a target HCT of 30 is 1,898 mL in this patient.[5] It is also recommended that estimated blood loss exceeding 1500 mL be treated with a combination of crystalloids and blood as was done in this case.[5]

Following the 3:1 rule for crystalloids and 1:1 rule of colloids, this patient got just over one liter of colloid and five liters of crystalloid which should have adequately replaced her blood volume. In retrospect the decision to wait on placement of an arterial line was perhaps remiss given the anticipated amount of dissection required to remove the tumor. The health care team was not anticipating such an extensive blood loss. It would have been prudent to place an arterial line preoperatively or immediately after induction.

In summary, the cause of leiomyomas are multifactorial and there are several risks inherent to their removal. The likelihood of extensive blood loss and the need for fluid resuscitation and close hemodynamic monitoring should be considered when formulating the preoperative plan. The anesthesia provider should have an individualized care plan in place for these women and be prepared to deal with possible complications that can arise in the perioperative period.

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Mentor: James Stimpson, CRNA, DNP

Left Frontotemporal Clipping of Middle Cerebral Artery Aneurysm

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Keywords: Cerebral aneurysm, hypothermia, hypocapnia, neuroprotection, brain relaxation, brain bulk, osmolality.

The four main arteries supplying the brain are the two internal carotid arteries and two vertebral arteries. The paired middle cerebral arteries arise from a bifurcation of the internal carotid arteries.[1] Intracranial aneurysms typically occur at branch points throughout the cerebral vasculature with aneurysms of the middle cerebral artery accounting for 25% of all those occurring in the brain.[2] Intracranial aneurysms are abnormal focal dilations that form as a result of weakening of the intima. Although most patients are asymptomatic until the aneurysm ruptures or become very large, causing pressure on adjacent structures, many aneurysms are discovered in time for therapeutic surgical clipping.[2] Despite new surgical techniques, patients undergoing neurovascular surgery are at risk for substantial mortality and morbidity. Sound

knowledge of cerebral vascular pathophysiology and appropriate choice of therapeutic intervention is essential for successful anesthetic management of these patients.

Case Report

A 58-year-old, 73.6 kg, 157.5 cm Caucasian female presented for left frontotemporal clipping of a 6 x 5 x 8 mm middle cerebral artery (MCA) aneurysm which was incidentally discovered during a diagnostic workup for multiple sclerosis. The patient had a 13 year history of experiencing headaches with a noticeable increase in pain intensity within the last year, accompanied by occasional nausea. The patient's past medical history was significant for type II diabetes, chronic obstructive pulmonary disease, anxiety, bipolar disorder, diverticulitis, recent colon resection, and multiple sclerosis. A preoperative neurological exam revealed that the cranial nerves were intact.

Preoperative airway examination revealed a Mallampati 2 airway classification, thyromental distance of 6 cm, a moderate overbite, and limited range of motion of the neck due to fusion of the C6-C7 cervical spine. An 18 gauge intravenous line was secured. The patient was premedicated with midazolam 2 mg intravenously and the left radial artery was cannulated with an arterial line in the preoperative holding area for invasive hemodynamic monitoring.
The patient was transferred to the operating room, and in addition to arterial monitoring, standard non-invasive monitors were applied. The patient was preoxygenated with 10 L/min O2 by mask for 3 minutes. General anesthesia was induced intravenously with lidocaine 50 mg, fentanyl 250 mcg, propofol 200 mg and vecuronium 5 mg respectively. Neuromuscular blockade was maintained with a train of four ratio of 0 out of 4. Direct visualization laryngoscopy was performed with a Macintosh 3 blade. A grade 2 glottic view was visualized and intubation of the trachea with a 7.5 mm endotracheal tube was successful upon use of the Eschmann stylet on the first attempt.

After induction of general anesthesia was completed, an additional #18 gauge intravenous line catheter was inserted, the patient was placed in the prone position, and the patient's anesthetic was deepened in preparation for pin insertion and head stabilization via the Mayfield frame and tongs. Before penetration of the dura ensued, mannitol 50 g and decadron 10 mg were administered intravenously. Anesthesia was maintained with 1.3-1.6 % inspired concentration of sevoflurane and a propofol infusion ranging between 50-75 mcg/kg/min. Fosphenytoin sodium 750 mg was administered intravenously prior to surgical replacement of the bone flap. The patient received 2 liters of normal saline and 1600 mL of Ringers Lactate intravenously throughout the case and urine output totaled 1800 mL.

Neuromuscular blockade was antagonized with glycopyrrolate 0.2 mg and neostigmine 2 mg. A train of four of 4/4 twitches, spontaneous tidal volumes greater than 300 mL and a respiratory rate of 12/min was observed. The patient was assessed for purposeful response to stimuli which was evident by appropriate responses to verbal command. The trachea was extubated and the patient was transported to the neurosurgical intensive care unit with O2 10 L/min via simple face mask and with stable vital signs.

Discussion

Anesthetic goals for management of patients undergoing intracranial aneurysm surgery include maintaining optimal cerebral perfusion pressure while avoiding increases in transmural aneurysm pressure. Additional considerations include careful attention to comorbidities, patient immobilization, facilitation of clear visualization and access to the aneurysm by decreasing intracranial tissue bulk, precise blood and intracranial pressure management, preparation for the possibility of intraoperative hemorrhage and rapid emergence from anesthesia.

The initial phase of surgical preparation involves induction of general anesthesia and the appropriate placement of the head in a pin fixation device. It is imperative that the anesthetist minimize hypertensive episodes, which could lead to aneurysm rupture or further bleeding in patients with known aneurysmal subarachnoid hemorrhage.[3] In an effort to blunt sympathetic responses to pin insertion, sevoflurane was increased to 2.3% expired and an additional dose of fentanyl 250 mcg was administered intravenously. In addition, esmolol 10 mg was administered to minimize the risk of sudden tachycardia and hypertension associated with laryngoscopy. Anticipation of and preparation for significant changes in blood pressure and heart rate is a key to maintaining hemodynamic stability. The anesthesia practitioner should individualize drug selection based upon clinical findings. Blood pressure control is maintained relative to the patient's baseline blood pressure. Autoregulation is maintained to a mean arterial pressure of 50-60 mm Hg and thus pressures below this limit are not recommended. Autoregulation is shifted to the right in patients with a history of hypertension and thus a lower limit of 60 mm Hg should be adopted.

During intracranial vessel surgery, blood flow to the cerebrum is reduced following vessel clipping leading to anaerobic cellular metabolism with reduction in adenosine triphosphate, instigation of inflammatory cascades, and later cellular injury and death. It has been shown that facilitating deliberate hypothermia impedes these destructive processes.[4] For this reason, forced air warming was not utilized as part of care for this patient and esophageal temperatures were maintained in the range of 35.8-36.3^{o}C during the surgery. No additional methods of cooling were utilized.

There are many instances during the perioperative period when cerebral oxygenation may be compromised. The selection of the anesthetic agent in the anesthetic plan plays a valuable role in benefitting efforts to maintain viability of brain tissue and providing neuroprotection. A combined anesthetic technique utilizing both an inhalational and intravenous agent was used to facilitate maximal brain protection for the patient. Sevoflurane has been proven to lessen glucose and oxygen deficient brain cell death and enhances preconditioning compared to other volatile agents.[5] A propofol infusion was also used which has been shown to decrease the cerebral metabolic rate of oxygen and decrease the damage threshold in periods of cerebral ischemia.[5] The patient was hyperventilated throughout the procedure, maintaining the end tidal CO_2 at 25 mmHg. The proposed benefits of hypocapnia include decreasing cerebral blood volume and subsequently intracranial pressure.[6] Hypocapnia was utilized to aid in brain relaxation, and to optimize the surgical field until the cranium was closed with the bone flap. Vigilant monitoring of end tidal CO_2 allows for effective titration of $PaCO_2$ leading to cerebral vasoconstriction in patients with intact CO_2 cerebrovascular activity.

Dexamethasone 10 mg was administered at the beginning of the procedure as well as mannitol 50 grams by infusion before penetration of the dura. Dexamethasone may decrease brain tissue edema and mannitol works to decrease brain water content, therefore reducing brain bulk. After infusion, mannitol causes an increase in plasma osmolality resulting in a fluid gradient that facilitates a fluid shift from brain tissue to the intravascular space. Research has demonstrated that mannitol's effectiveness in reducing intracranial pressure is associated with decreasing blood viscosity, decreasing CSF content, and decreasing brain water content.[7]

Successful intraoperative management of intracranial vessel surgery involves control of many complex factors. Careful management of hemodynamic response, use of optimal techniques and the safest evidence based methods are necessary for achieving optimal patient outcomes in this population.

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Uterine Rupture during Vaginal Delivery with no Previous Cesarean Delivery

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Keywords: uterine rupture, vaginal delivery, anesthesia, hemorrhage, cesarean delivery

Uterine rupture is a potentially catastrophic obstetric complication that may occur during the periods of antepartum, intrapartum or postpartum.[1] It is an obstetric emergency which threatens the life of both the mother and the newborn.[2] Previous cesarean section is recognized as the primary risk factor for uterine rupture in the United States and the rupture of an intact, unscarred uterus is rare.[3] Signs and symptoms depend on the extent of the rupture. Anesthetic management for laparotomy, uterine repair, hysterectomy, or hypogastric ligation is similar to that for an actively bleeding, acutely hypovolemic patient.[1]

Case Report

A 36-year-old, 72.1 kg, 154.9 cm, gravida 10, para 8, 39 week gestation female, presented for a scheduled induction for vaginal delivery. Her vital signs on arrival were: blood pressure (BP) 137/79 mm Hg, heart rate (HR) 76/min, respirations 20/min, temperature of 36.8ºC with a Body Mass Index was 30 kg/m^2. Laboratory values were hemoglobin 11.9 gm/dL, hematocrit 34.7%, and platelet count of 191,000 μL.

Approximately 4 hours after receiving an uneventful epidural, the patient had a spontaneous vaginal delivery of a 3,546g, 50.8 cm female with APGAR scores of 3 at one minute and 8 at five minutes. The placenta was expelled spontaneously and the patient began to hemorrhage. Estimated blood loss was 1000 mL. A vaginal exam was performed and a uterine rupture was suspected. Immediately following the exam, the patient's blood pressure was unobtainable, HR was 132/min, and respirations 26/min. Her hemoglobin was 8.6 gm/dL, hematocrit 24.9%, and platelets 152,000 μl. A rapid blood infusion protocol was initiated and anesthesia was contacted for immediate surgery.

Lactated Ringer's (LR) was administered and the patient was transferred to the operating room. Two units of packed red blood cells (PRBC) and one unit of fresh frozen plasma (FFP) was given via a fluid warmer. All standard ASA monitors were applied to the patient and oxygen was administered via mask at 10 L/min. The first vital signs were BP 41/23 mm Hg and HR 132 BPM. Ephedrine 25 mg and phenylephrine 300 mcg were given intravenously (IV). A rapid sequence induction was accomplished with propofol 200 mg and succinylcholine 100 mg IV, along with placement of a 6.5 mm endotracheal tube and surgery proceeded. An additional peripheral 16 gauge IV was placed.

Immediately after induction labs were drawn and the patient's hemoglobin was 8.7 gm/dL, hematocrit 25.5%, and platelets 104,000 μL. An additional two units of PRBC, one unit of FFP and a pack of platelets were transfused and follow-up labs were again drawn. A ruptured uterus was confirmed and an abdominal hysterectomy was performed. Vital signs remained stable after the transfusion and hysterectomy. Blood pressure was 96/42 mm Hg and HR 88/min.

The patient remained intubated and sedated and was transferred to the intensive care unit (ICU). Estimated blood loss for the case was 2500 mL. Fluid intake consisted of LR 2500 mL, normal saline 2200 mL, PRBC 1200 mL, FFP 600 mL, and platelets 230 mL. The vital signs on arrival to ICU were, BP 84/55 mm Hg and HR 97/min. The patient's lab values returned closer to normal limits, as hemoglobin was 10.8 gm/dL, hematocrit 31% and platelets 147,000 μl. Labs were drawn 3 hours after arriving to ICU and were hemoglobin 7.9 gm/dL, hematocrit 22.7% and platelet count of 168,000 μl and the patient received 2 more units of PRBC's after the subsequent lab values. The endotracheal tube was removed 4 hours after arriving in the ICU and the patient was discharged home on day 3 of her hospital stay.

Discussion

Although uterine rupture is a rare occurrence, knowledge of the risk factors, signs and symptoms are important for early detection and treatment.[3] Causes include (a) separation of the uterine scar, (b) rupture of the myomectomy scar, (c) previous difficult deliveries, (d) rapid, spontaneous, tumultuous labor, (e) prolonged labor in association with excessive oxytocin stimulation or cephalopelvic disproportion, (f) weak or stretched uterine muscles, such as might be found in the grand multipara, in multiple gestation, or in polyhydramnios, and (g) traumatic rupture (iatrogenic) occurring from intrauterine manipulations, difficult forcep applications, and excessive suprafundal pressure.[1,4]

Signs and symptoms of uterine rupture depend on the extent of the rupture and include vaginal bleeding, severe uterine or lower abdominal pain, shoulder pain from subdiaphragmatic irritation by blood, disappearance of fetal heart tones, and severe maternal hypotension and shock.[1] Regarding signs and symptoms of a uterine rupture, pain is a less reliable sign of uterine rupture than is a change in the fetal heart rate.[5] The clinical picture of uterine rupture is variable and depends on the time of occurrence, cause, degree and extent of rupture, amount of bleeding, and the general condition of the patient.[3]

Treatment of the uterine rupture during the prenatal period involves an emergency cesarean delivery in order to separate the fetus from the dysfunctional uterus and to control maternal bleeding. A rupture that is diagnosed after placental delivery is treated by laparotomy to repair or remove the damaged uterus.[5]

Maternal death from uterine rupture usually occurs because it goes undiagnosed, blood transfusions are inadequate or a laparotomy is delayed or not done.[1] Anesthetic management and considerations should include a thorough preanesthetic evaluation, including identifying and planning for a patient with increased risk factors. General anesthesia is the most frequently used anesthetic management for uterine rupture. Invasive hemodynamic monitoring may be appropriate in the acutely hypovolemic patient. Aggressive fluid volume resuscitation, maintenance of urine output, and the administration of blood and blood component replacements should be utilized.[3]

In a study conducted by M. Guiliano, et al 97,028 deliveries were evaluated and statistics identified. Of the complete uterine ruptures, 27 were women with no previous cesarean delivery.

Only six occurred in an unscarred uterus at a frequency estimated at 0.07 per 1000.[6] Of these six, the timing of the onset of the signs and symptoms for the complete ruptures occurred more frequently during labor as opposed to during the postpartum period.[6] The ruptures of an unscarred uterus were highly related to grand multiparas, as 66.7% had 4 previous deliveries.[6] The importance of this is rare uterine rupture is potentially catastrophic and most often occurs in women with a uterine scar (90% of cases).[6] The American College of Obstetricians and Gynecologists recommends that in the case of urgent or emergent cesarean delivery, the delivery should be accomplished within 30 minutes of the decision to do so ("decision-to-incision" time).[5] In the case of uterine rupture a time frame closer to 15 minutes is necessary, and availability of the surgical team is vitally important.[5]

Consideration of risk factors should always be an important aspect of a preanesthesia assessment. Moreover, documentation of these risk factors provides good communication to all anesthesia providers that may become involved. This woman presented with eight previous vaginal deliveries and there was no prior discussion of the possibility of uterine rupture. Due to her multiparas status and her weakened or stretched uterine muscles she should have been considered an increased risk of uterine rupture. Preparation is another vital component to the successful management of cases involving uterine rupture. Preparation includes making sure adequate blood products are available and laparotomy is not delayed, as time and blood replacement are crucial elements in this case.

This case could be better managed in the future by choosing ketamine 1 mg/kg or etomidate 0.3 mg/kg rather than propofol because her blood pressure being so low. Additionally, placement of an arterial line would have been beneficial for monitoring blood pressure and obtaining blood for needed lab values. However, several components which led to a positive outcome were in place including: the hemorrhage was identified immediately as an emergent situation, necessary blood products and a fluid warmer was readily accessible and the operating room and necessary staff were available.

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Anesthetic Management of a Carcinoid Tumor

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Keywords: carcinoid tumor, carcinoid syndrome, carcinoid crisis, anesthetic management, octreotide

Under anesthesia, although uncommon, carcinoid tumors can be deadly. Carcinoid tumors, which prove to be clinically significant, occur in 7 to 13:1,000,000 cases per year. Such tumors can occur in almost any tissue throughout the body.[1] Patients with a carcinoid tumor are at risk for developing an anesthetic challenge and should be anticipated throughout the perioperative period. Carcinoid syndrome occurs as a result of serotonin, kallikrein, and histamine being released from the tumor.[2] The secretion of such vasoactive substances can cause bronchospasm, labile arterial blood pressure, and supraventricular arrhythmias.[2] This case study examines a patient undergoing a colon resection for a rectal carcinoid tumor.

Case Report

A 47-year-old, 180 cm, 137 kg male presented for a laparoscopic resection of a primary rectal carcinoid tumor with coloanal anastomosis and loop ileostomy. His medical history was significant for hypertension, gastroesophageal reflux disorder (GERD), morbid obesity, obstructive sleep apnea, non-insulin dependent diabetes, stage 1 diastolic dysfunction, alcohol abuse, and liver metastasis from the rectal cancer. His current medications included; amlodipine, docusate sodium, losartan/ hydrochlorothiazide, metformin, omeprazole, fenofibrate, hydroxyzine hydrochloride, and hydrocodone. The patient was starting to exhibit signs of carcinoid syndrome as evidenced by frequent cutaneous flushing and occasional diarrhea. He was being treated with long acting intramuscular octreotide acetate 30 mg monthly. All laboratory testing and assessments were unremarkable, his electrocardiography (ECG) showed a normal sinus rhythm, and his stress echocardiogram was negative for signs of ischemia.

The patient was given midazolam 2 mg intravenously (IV) prior to placement of an epidural for intraoperative and postoperative pain management. The epidural catheter was dosed at the start of the case with fentanyl 100 mcg, and an infusion of bupivacaine 0.125% was initiated at 6 mL/hr towards the conclusion of the case. The anesthesia team confirmed the patient had received his monthly IM injection of octreotide to ensure a preoperative dose was not required. Due to the patient's history of alcohol abuse, the patient received lorazepam 2 mg IV and methadone 5 mg IV for premedication and was transferred to the operating room.

Upon arrival to the operating room, the patient was placed on ECG, pulse oximetry, and non-invasive blood pressure monitoring, and a rapid sequence induction was achieved with fentanyl 100 mcg IV, lidocaine 100 mg IV, propofol 150 mg IV, and succinylcholine 140 mg IV. Using a

Macintosh 4 blade, the trachea was intubated with an 8.0 oral endotracheal tube secured at 23 cm at the lip. Once correct tube placement was verified by the presence of consistent end-tidal carbon dioxide and bilaterally equal breath sounds on auscultation, cricoid pressure was released and respirations were controlled with volume control ventilation. General anesthesia was maintained with desflurane 6.6% inspired concentration in a mixture of oxygen 1 L/min and air 1 L/min. A 20 gauge, right radial arterial line and an 18 gauge, left forearm peripheral IV were placed prior to the start of the procedure, and neuromuscular blockade was maintained with rocuronium.

Over the course of the seven hour procedure, the patient remained hemodynamically stable. The mean arterial blood pressure was maintained within 20% of the patient's baseline without the use of vasoactive drugs. Octreotide 100 mcg, diluted into 10 mL of sodium chloride 0.9%, was readily available for administration and management of carcinoid crisis but was not required during the case. Prior to emergence, ondansetron 4 mg IV and acetaminophen 1 g IV were administered. Prior to extubation, neuromuscular blockade was antagonized with neostigmine 5 mg IV and glycopyrrolate 0.8 mg IV. Once the patient met all criteria, he was extubated without incident. The patient was transferred to the post anesthesia care unit (PACU) with 6 L/min of oxygen via simple face mask. The patient was recovered from surgery without any complications and discharged from the PACU.

Discussion

More than 75% of carcinoid tumors originate in the gastrointestinal tract, but they can also occur in the lungs, genitourinary tract, thyroid, pancreas, breast, thymus, and liver.[3] Only 25% of carcinoid tumors will actively secrete mediators capable of causing carcinoid syndrome, but less than 10% of patients will actually develop carcinoid syndrome.[3] This is due to the fact that metabolic byproducts are released into the portal circulation and destroyed by the liver before any symptoms arise.[2] The signs and symptoms of carcinoid syndrome often indicate the presence of pulmonary, hepatic, or other systemic metastases.

Carcinoid syndrome can create a potentially fatal situation if not recognized quickly. The release of serotonin and other vasoactive substances, such as histamine, are responsible for the clinical manifestations of carcinoid syndrome.[4] These clinical manifestations include: severe fluctuations of blood pressure (usually hypotension that is unresponsive to typical inotropic or vasopressor therapy), cutaneous flushing, bronchospasm, and carcinoid heart disease resulting in right or left sided heart failure.[3] Stressors such as general anesthesia and physical or chemical manipulation of the tumor can stimulate the release of the bioactive mediators which can mitigate carcinoid crisis.[3] Carcinoid crisis is a life-threatening form of carcinoid syndrome that results in exaggerated symptoms that mimic anaphylactic shock.

When planning for anesthetic management of a carcinoid tumor, there are two specific areas of concern: the presence of right or biventricular heart failure, and the potential of unpredictable, uncontrolled hemodynamic collapse.[4] A thorough preoperative work up is essential to identify cardiovascular risks that may complicate the anesthetic plan, and the availability of bioactive mediator antagonists are essential to prevent fatal outcomes. Even in asymptomatic patients, the stimulus to release vasoactive hormones will be much higher than in day to day life due to the

stress of anesthesia and surgery.[4] For this reason, it is paramount that tumor activity is minimized prior to the day of surgery with the use of octreotide. Care should be taken to avoid drugs that provoke mediator release (atracurium, morphine, epinephrine, dopamine, ketamine, and succinylcholine).[1] Succinylcholine was chosen in this case because of the risk for potential complications during induction associated with the patient's obesity, obstructive sleep apnea, and poorly controlled GERD.

Due to the uncommon occurrence of carcinoid syndrome, few prospective randomized clinical trials exist. A review of cases suggest somatostatin analogs, such as octreotide, are the preferred treatment to manage the symptoms of carcinoid syndrome.[3] It has been shown that perioperative boluses of octreotide up to 1 mg with the use of hydrocortisone can counteract carcinoid crisis induced hypotension.[3] Histamine receptor blockers (H_1 and $H_{2)}$, serotonin receptor blockers (5-HT_2 and 5-HT_3), and corticosteroids may also have a role as adjunct therapies in preventing carcinoid syndrome.[3] Octreotide doses of 50 to 200 mcg IV are effective in promptly reversing severe hypotension and bronchospasm, and a continuous infusion of 20 to 50 mcg/hr can be initiated to prevent further crisis.[3] A remifentanil infusion offers the advantage of adequate analgesia, little to no histamine release, good suppression of the intubation response, and rapid titratability, which may be beneficial in the management of a carcinoid patient.[5] The use of epidural anesthesia is only recommended in patients that have been adequately treated with preoperative octreotide to prevent adverse hemodynamic consequences.[5]

A balanced anesthetic technique appears to be the most common approach to managing a carcinoid patient. This incorporates an inhalation agent, a nondepolarizing neuromuscular blocking agent, positive pressure ventilation, and an opioid.[3] A stable induction, adequate anesthetic depth prior to intubation and throughout the operative course, and sufficient analgesia are key to preventing instability. Both total intravenous anesthesia and inhalation techniques have been used successfully with the choice depending on familiarity with the specific technique.[5] The anesthesia team utilized a balanced anesthetic approach with this patient by using both regional and general anesthesia.

Although the incidence of carcinoid syndrome is infrequent, anesthesia practitioners should be able to recognize and treat the syndrome as it occurs. There is not a standardized approach for the anesthetic management of a patient with a carcinoid tumor but adequate preparation is imperative for a safe outcome. Techniques focus on a thorough preoperative evaluation and assessment, avoidance of triggering agents throughout the perioperative course, and ample preparation and recognition in the event of a carcinoid crisis.

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Translaminar Epidural Steroid Injection for Chronic Low Back Pain

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Keywords: lumbar epidural steroid injection, low back pain, spinal stenosis, translaminar, transforaminal, caudal.

Low back pain affects almost 12% of the world's population and it is the leading cause of disability worldwide.[1] Epidural steroid injections (ESI) are the most common interventional technique utilized in U.S. interventional pain practices for the treatment of low back pain. Given that patients present with different symptoms and diagnosis, it is essential that anesthesia professionals understand the different approaches and indications for treatment of low back pain.

Case Report

A 50-year-old, 188 cm, 95 kg male was referred to the pain clinic by his neurosurgeon for an elective translaminar epidural steroid injection under fluoroscopy. His allergies included shellfish and chlorhexidine. His past medical history was significant for chronic low back pain, hypertension, and hyperlipidemia. Surgical history included rotator cuff repair, ACL repair, foot plantar fascia release, foot heel bone spur removal, and wisdom teeth extraction. His current low back pain was the result of a work related fall from 2008. A CT scan revealed a diagnosis of lumbar facet joint arthropathy, degenerative disk disease, neural foraminal stenosis of L3-L4, L4-L5, L5-S1 lumbar interspaces, and acquired spondylolisthesis. Upon physical assessment, radicular leg pain was generated with bilateral straight leg raising and sit up. The patient described the bilateral leg pain as sharp, shooting and stabbing and he rated it as a 6 on the 0 to 10 pain scale. He also felt numbness and tingling to his shins and at the top of his feet. Upon palpation of lumbar spine he complained of spinal tenderness. He had trialed physical therapy and medication management without relief. The neurosurgeon had recommended surgery; however, the patient wanted to trial injection therapy first.

Prior to the procedure a preoperative evaluation was performed. He reported no prior anesthesia problems. An airway evaluation revealed a Mallampati classification I with normal dentition. Pre-procedural vital signs included blood pressure 117/64, pulse 91, respirations 18 and SpO_2 97% on room air. His lungs were clear and heart rate was regular with S1 and S2. The procedure was explained to the patient and informed consent was obtained. The patient was brought back to

the procedure room, positioned prone and standard noninvasive monitors were applied. He was given two milligrams (mg) of midazolam and 100 micrograms (mcg) of fentanyl intravenous (IV) for conscious sedation. Oxygen was started per nasal canula. His lower back was prepped with betadine and draped utilizing sterile technique and a time out was performed. Three milliliters (mL) of 1% lidocaine was subcutaneously infiltrated at the L5-S1 level using a 25 gauge needle. An 18 gauge Tuohy needle was then placed using the loss of resistance technique through the L5-S1 interspace. Once loss of resistance was obtained, 2 mL of radiopaque contrast media was injected and needle placement was confirmed under fluoroscopy. Following confirmation of correct needle placement, 120 mg of methylprednisone and 20 mg of lidocaine was injected into epidural space.

The patient tolerated procedure well and his vital signs remained stable throughout the procedure. He denied any pain and/or nausea and was transferred to the PACU. At his three week follow up injection, the patient stated his pain was decreased while standing and walking and he rated it as a 4 on the 0 -10 pain scale.

Discussion

Chronic back pain is pain that lasts beyond normal healing after an injury, typically lasting longer than six months. It is a combination of nociceptive and neuropathic pain and includes both a mechanical and inflammatory process.[2] The patient in this case study presented with a multifactorial cause for his low back pain including spinal stenosis. Degenerative spinal stenosis develops as one ages and is the progressive narrowing of the spinal canal from a bulging disc, calcification, and hypertrophy of the ligamentum flavum and / or osteophyte formation that leads to compression of the spinal cord and nerve roots.[3] The pain presents with aching and burning pain in low back, and radicular pain in the leg. Compression of the nerves can lead to numbness, weakness and paresthesia in the legs, limiting a patient's ability to walk.

When treating low back pain, anesthesia practitioners must take into consideration patient diagnosis, patient preferences, current evidence and risk/ benefit of appropriate procedures. Benefits can be measured by pre and post procedural assessment of ability to perform activities of daily living (ADL's), hours of sleep, walking and pain. Less costly and less invasive therapeutic options should be considered first, followed by more invasive techniques and with a multimodal and multidisciplinary approach in mind.[4] The patient in the case study failed conservative measures prior to consulting with the neurosurgeon and interventional pain management specialist.

There are three types of ESI's that can be utilized to treat back pain: translaminar, caudal, and transforaminal. ESI's work by inhibiting nociceptive or A delta pain transmission and decreasing the sensitivity of C fibers to pain impulse.[5,6] Corticosteroids work by decreasing inflammation, suppression of dorsal horn sensitization and neuronal discharge, membrane stabilization, blockade of neural peptides, and inhibition of phospholipase A activity.[5] Complications of ESI's include infection, dural puncture, nerve damage, vascular injection, Cushing syndrome, osteoporosis, neuraxial suppression, and hyperglycemia.[5-7] Epidurals can be performed with or without sedation depending on the pain specialist preference. Sedation helps reduce anxiety but

it can also decrease a patient's ability to verbalize paresthesia during introduction of the Touhy needle.

Translaminar ESI's deliver the medication directly to site of pathology and they have shown to reduce pain and improve function in patients with chronic back pain.[5,6] There is a limitation however, in patients with spinal stenosis or post laminectomy syndrome, the pathology can limit the spread of the medication to the dorsal epidural space and not the ventral lateral space, leading to ineffective pain management.[5,8]

A caudal epidural steroid injection is an epidural steroid injection through the sacral hiatus. This can be performed with a needle advanced through the sacral hiatus into the epidural space. A catheter can also be advanced through the needle to the area of pathology. This approach is best utilized in elderly patients with tight interspaces, spinal stenosis, post-laminectomy syndrome or post spinal fusion with hardware.[5,7]

A transforaminal ESI targets the ventral lateral epidural space. Under fluoroscopic guidance a small amount of medication to be placed directly to the nerve root with pathology making transforaminal ESI's beneficial for radicular pain.[8] Transforaminal pose a higher risk of inadvertent arterial injection. Some practitioners recommend utilizing a non-particle steroid such as dexamethasone for transforaminal injection.[5]

A thorough review of the literature found that interlaminar, caudal, and transforaminal ESI's are all effective treatments for low back pain and there is no clear evidence that one approach is better than the other.[5] There is good evidence for the treatment of radiculopathy from herniated disc with caudal, interlaminar or transforaminal ESI.[5] When there is bilateral radiculopathy, translaminar and caudal are better choices than transforaminal injections because transforaminal injections are unilateral.[5,7] Due to the low number of high quality studies, all of the above steroid injections are deemed fair for the treatment of lumbar spinal stenosis.[5,6]
Since the patient received relief with the first epidural steroid injection, recommendations are to repeat the injection in two weeks to one month. If the patient has 50% relief in pain after his second injection a third injection can be performed two months from the second, with a grand total of 4 epidural steroid injections in a year.[5]

For this patient 120 mg of methylprednisone and 1% lidocaine 30 mg was utilized. It has been found that there is no significant difference between epidural injections performed with steroids and local anesthetics, compared to, local anesthetics alone.[5, 7] Methylprednisone 40 mg is as effective as 80 mg.[5] Based on these findings, it can be concluded higher doses of steroids do not necessarily produce greater efficacy.

With the complexity and high prevalence of chronic back pain in the U.S. it is essential for anesthesia practitioners to understand appropriate interventional treatment modalities for managing chronic low back pain. Epidural steroid injections have shown to be an effective intervention in the treatment of low back pain and/or improving patient function. Pain is an individualized and subjective patient experience and each patient should be treated as an individual with the appropriate guidelines in mind.

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Postoperative Nausea and Vomiting Prevention in High Risk Patients

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Keywords: Postoperative nausea and vomiting, PONV, total intravenous anesthesia, TIVA.

The development of postoperative nausea and vomiting (PONV) is associated with multiple risk factors including history of PONV, female gender, history of motion sickness, administration of opioids, use of nitrous oxide, use of volatile anesthetics, nonsmoker, duration/type of surgery, and patient's age.[1] With up to 75 million people or about one-third of surgical patients experiencing PONV and the possibility of patient dissatisfaction, discomfort, costly extended postoperative time, and/or unplanned hospital admission, the prevention of PONV is of paramount concern.[1] Strategies aimed towards PONV reduction and prevention may include the use of total intravenous anesthesia (TIVA), multimodal PONV prophylaxis, and avoidance of opioids perioperatively.[1]

Case Report

A 32-year-old, 175 cm, 100 kg male presented for endoscopic sinus surgery and septoplasty to treat chronic sinusitis and nasal airflow obstruction. His medical history included multiple occurrences of PONV following general anesthesia lasting up to 24 hours postoperatively, epidermolysis bullosa simplex, and gastroesophageal reflux disease well controlled with esomeprazole magnesium and diet. Based upon the patient's history, a PONV prevention strategy was devised. The anesthetic plan was for general anesthesia (GA) using TIVA, a scopolamine patch preoperatively, dexamethasone after intubation, and ondansetron and intravenous (IV) acetaminophen prior to anesthesia emergence.

The patient refused a scopolamine patch due to concern the patch would cause skin blistering related to the history of epidermolysis bullosa simplex. The patient was premedicated with midazolam 2 mg IV. On arrival in the OR, noninvasive monitors were applied and the patient was preoxygenated with O_2 8 L/min via anesthesia circuit facemask for 5 minutes. The IV induction consisted of lidocaine 100 mg, propofol 200 mg IV, and rocuronium 60 mg IV. The trachea was intubated with a Macintosh 3.5 laryngoscope blade and a 7.5 oral Ring, Adair, Elwyn endotracheal tube (ETT) was placed. Following intubation, dexamethasone 4 mg IV was administered. General anesthesia was maintained using IV propofol ranging from 75-150 mcg/kg/min and remifentanil at 0.2 mcg/kg/min continuous infusions.

The surgery was uneventful and lasted 4 hours. Ondansetron 4 mg and acetaminophen 1000 mg IV were administered 30 minutes prior to the conclusion of surgery. At the conclusion of surgery the surgeon injected 14 mL of 2% lidocaine at the surgical site. Neuromuscular blockade was antagonized with neostigmine 3 mg and glycopyrrolate 0.6 mg IV. Train of four monitoring identifying 4/4 twitches and sustained tetany was noted. The ETT was removed from the trachea following patient eye opening and responding to commands.

The patient's estimated blood loss was 100 mL with 2.2 liters of normal saline given intraoperatively. The patient was transported to the post anesthesia care unit (PACU) on room air. Pain was well controlled during the first hour of recovery and the patient received hydromorphone 0.4 mg IV in divided 0.2 mg doses during the PACU stay. Hydromorphone was administered based on a rating of pain at 2 and 3 on a Likert pain scale, the patient stated this was not a tolerable level. The patient did not experience PONV in the PACU, or on the first postoperative day.

Discussion

Attention to PONV prevention necessitates screening patients for PONV risk factors and tailoring the anesthetic and adjuvant agents. In this case, the type of surgery, nonsmoker, and a history of severe PONV were risk factors based on The American Society of PeriAnesthesia Nurses' (ASPAN) Evidence-Based Clinical Practice Guideline for the Prevention and/or Management of PONV/PDNV.[1] For the PONV prevention strategy, the anesthetic plan included TIVA, multimodal PONV prophylaxis, and opioid sparing where possible.[1] The result in this case revealed an absence of PONV in the 24 hour post anesthesia period.

According to ASPAN, volatile anesthetics are supported by strong evidence, class I and level A, as a PONV risk factor.[1] In this case, TIVA was administered to avoid volatile anesthetics.[1] The agents administered were propofol and remifentanil in continuous infusions. In 2014, Kumar et al conducted a meta-analysis of 18 randomized trials (n=1621) comparing propofol-based TIVA to desflurane or sevoflurane based GA in ambulatory surgical patients.[2] This study showed the advantages of TIVA over volatile anesthetics were not statistically significant. However, the results demonstrated the propofol-based TIVA group had a decrease in early PONV (13.8% vs. 29.2%) but similar post discharge nausea and vomiting (PDNV) results.[2]

The prevention of PONV via multimodal antiemetic prophylaxis was also employed in this case. Dexamethasone, a corticosteroid, and ondansetron, a 5-HT_3 receptor antagonist, were given after intubation and prior to anesthesia emergence respectively. A meta-analysis of 49 randomized control trials (n=12752) suggests dexamethasone and 5-HT_3 receptor antagonist combination therapy is more effective at preventing PONV than monotherapy with dexamethasone.[3] In a study by Subhi et al.[4], ondansetron, dexamethasone, and saline bolus placebo were administered to 3 separate groups following induction of propofol-based TIVA. The findings showed neither ondansetron nor dexamethasone separately were more effective than a saline bolus placebo in prevention of PONV over a 24 hour period postoperatively.[4] In this case, multimodal PONV prophylaxis consisted of dexamethasone IV after intubation and ondansetron IV prior to patient emergence.

An opioid sparing strategy was implemented for the anesthetic management. Remifentanil was co-administered with propofol, as a component of TIVA, for intraoperative pain control. Current literature suggests that remifentanil does not increase the risk of PONV.[5] In a retrospective observational study (n=1765), Hara et al[5] compared PONV occurrence in cases utilizing TIVA with remifentanil and cases utilizing TIVA without remifentanil. The remifentanil group experienced nausea or vomiting in 7.8% of cases, as compared to the non-remifentanil group, in which 10.1% of patients experience nausea or vomiting, indicating no significant difference between the two groups.[5] The patient in this case did receive remifentanil intraoperatively and required hydromorphone 0.4 mg IV during his recovery period, but the patient did not require rescue antiemetics and did not experience PONV or PDNV in the 24 hour post anesthesia period.

Complete avoidance of opioids may be necessary in patients at high risk for PONV. In a 2014 prospective randomized trial, patients received intraoperative opioids in combination with inhaled anesthetics or an opioid-free TIVA to include ketamine, propofol, and dexmedetomidine.[6] Both groups received acetaminophen and dexamethasone after induction of anesthesia and ondansetron and ketorolac prior to emergence from anesthesia. Oxycodone and hydromorphone were given for breakthrough pain postoperatively to both groups.[6] In the opioid-free TIVA group there was a 20% absolute risk reduction of PONV.[6] There was a 37.3% PONV occurrence in the opioid and volatile anesthetic group versus 17.3% in the opioid free TIVA group.[6]

Additional strategies employed to provide analgesia and reduce opioid requirements were the use of local anesthesia at the surgical site and administration of acetaminophen 1000 mg IV prior to emergence from anesthesia. Intraoperative and postoperative administration of acetaminophen IV (n=1464) has demonstrated improved pain control quantified by less opioid use and decreased

pain scores in patients.[7] In a meta-analysis of 30 randomized placebo-controlled trials (n=2364), intraoperative administration of acetaminophen IV resulted in a reduction in PONV comparable to antiemetic therapy.[8] The reduction of PONV due to acetaminophen's analgesic efficacy offers strong evidence for its use.[8]

This patient presented with 3 risk factors for PONV: undergoing ESS, nonsmoker, and a known high risk of PONV occurrence. The anesthetic plan was designed to balance surgical needs, patient comfort, and PONV prevention. A possible improvement may consist of an opioid-free anesthetic by the addition of ketamine and dexmedetomidine with propofol, however some patients may require postoperative pain control via opioid administration. Use of TIVA is justified because it demonstrates improvement in PONV prevention in high-risk patients experiencing PONV, such as in this case. Acetaminophen IV for postoperative pain prevention, dexamethasone and a 5-HT_3 receptor antagonist combination are all strongly recommended in these patients and in this case, employing this multimodal strategy, the patient did not experience PONV.

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Mentor: Michele E. Gold, CRNA, PhD

VATS Procedure for Pulmonary Sequestration Excision in a 13-month-old

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Keywords: pulmonary sequestration, pediatric VATS procedure, chronic respiratory infections, bronchial atresia, bronchopulmonary malformation, intralobar sequestration

Pulmonary sequestration is a rare congenital malformation of the respiratory tract. It is characterized by cystic, non-functioning embryonic lung tissue with vascularization of an abnormal systemic artery.[1]The location, size and vasculature involved is case specific, therefore so is its treatment. It often causes chronic respiratory infections and eventually, residual damage to the respiratory tract, if left untreated. Currently, there is limited research and case studies available due to the uncommon nature of this congenital anomaly. Prior to the use of video-assisted thoracoscopic surgery (VATS) approach for intra-thoracic procedures, an open thoracotomy was the only option for resection.

Case Report

A 13-month-old male, full term at birth, presented for VATS for pulmonary sequestration resection causing chronic respiratory infections. A fetal magnetic resonance imaging (MRI) demonstrated a left lower lobe bronchopulmonaryforgut malformation with systemic arterial supply. The patient's height and weight were 32 cm and 10 kg, respectively. The patient had allergies to penicillin and milk. The patient's past medical history was significant for asthma, reactive airway disease and chronic respiratory infections that resulted in numerous hospitalizations. This was the first surgical intervention. Vital signs pre-operatively were: blood pressure 90/55 mmHg, heart rate 116/min and temperature of 38°C. His medications included albuterol nebs as needed for wheezing. Pertinent laboratory values were as follows: white blood cell count 13,000x mm^3, hemoglobin 9.9 g/dL, hematocrit 30.1% and platelets 389,000/µL. Preoperative tests included a chest x-ray (CXR) that demonstrated opacity at the medial aspect of the left lower lobe, which corresponded with the patient's known left lower lobe pulmonary sequestration.

The patient presented the day of surgery with symptoms of an upper respiratory tract infection (URI), including rhinitis and a low grade temperature. After discussion with the surgeon, the case was cleared to proceed with knowledge of the URI and low grade temperature. After explaining risks and benefits of the procedure and obtaining informed consent from the patient's mother, an inhalational mask induction was performed and the airway was secured with a cuffed, single lumen 4.0 mm endotracheal tube. Two peripheral intravenous catheters were inserted and an arterial line established and secured in the right radial artery. The endotracheal tube was advanced into the right main bronchus to isolate the operative lung and ensure one lung ventilation (OLV) was achievable. The patient was prepped in the right lateral decubitus position and endobronchial placement of the endotracheal tube reconfirmed. Ventilation settings were adjusted to decrease tidal volumes and positive inspiratory pressure, while maintaining PEEP at 5cm of H_2O and increasing the respiratory rate to adapt the ventilation needs during OLV.

Due to the very compliant chest wall in children the operative lung was dropped by the surgeon upon entering the thoracic cavity and creating an intentional pneumothorax. The incision into the thoracic cavity resulted in the loss of the negative pleural pressure, normally seen. The surgically created pressure equilibrium in addition to the child's lax, cartilaginous rib cage resulted in collapse of the operative lung. Once the surgeons accessed the pleural cavity their initial surveillance yielded an even more abundant vascular supply than originally expected. The surgeon was unable to obtain a complete view of the vasculature involved and the procedure was converted to an open thoracotomy.

Maintenance of the patient's oxygen saturation was an intermittent concern but desaturations responded well to intervention that was specifically timed with the surgeon. Manual ventilation with breath holding was attempted as a recruitment measure to open atelectic regions of the right lung. This also offered opportunity to assess tactile changes in ventilatory compliance and resistance. If these interventions did not adequately improve the patient's oxygenation status, upon surgical release the endotracheal tube was withdrawn until the tip was in the trachea and temporary inflation of the collapsed lung was performed. Breaths were administered with increased volume, pressure and duration during these periodic recruitment attempts. The surgery was completed as planned with complete removal of the vascular supply and wedge resection. Anesthetic maintenance was achieved by intermittent dosing of morphine and rocuronium in addition to 0.8 MAC of sevoflurane. Estimated blood loss was 45mL and intravenous fluid administration totaled 300 mL of lactated ringers. Overall, the patient tolerated the procedure well and a chest tube was placed in the left pleural cavity, prior to closure of the surgical site.

Prior to extubation the patient was noted to have audible wheezing and was given an albuterol nebulizer though his ETT. He was successfully extubated without any respiratory compromise. Total surgical time was 3.5 hours after which the patient was transferred to the Pediatric Intensive Care Unit (PICU) for hemodynamic monitoring, close observation of respiratory status and pain management. Postoperative recovery was uneventful and the patient was discharged on the 7th day of admission.

Discussion

A patient presenting pre-operatively with URI symptoms and temperature may frequently be sent home and rescheduled. In this specific scenario all factors needed to be considered and the risk versus benefit of operating versus postponing thoroughly assessed. Open communication between all involved disciplines was key to making the best decision. This also allowed each provider to express concerns they may have had and provided a sense of team, prior to initiating the case. This patient had chronic URIs and the surgical resection of the non-functioning portion of lung was the ultimate treatment this patient needed. The concern of the surgeon was that this patient may never present without symptoms and may progressively get worse until the wedge resection was completed.

Pulmonary sequestration is classified into intralobar sequestration (ILS) and extralobar sequestration (ELS) according to the absence or presence of independent visceral pleura encased in abnormal lung tissues. The postoperative surgical diagnosis for this patient was bronchopulmonary malformation secondary to bronchial atresia with systemic vascular supply

(intralobar sequestration). Pulmonary sequestration results in an anatomically caused V/Q (ventilation/perfusion) mismatch. The affected portion of lung receives perfusion but no ventilation and is considered a shunt. It is taking from the systemic blood supply but serving no benefit to the oxygenation of the patient. Congenital lung malformations can be diagnosed at any age and present with a variety of symptoms.[3]

Due to a limited amount of data, there is no set standard of care or recommendations. The limited use of VATS as the surgical approach for pulmonary resection in pediatrics also restricts what is known and recommended regarding this surgical technique. Utilizing the cases and information available, has resulted in variations in interpretations and conclusive recommendations, such as: age when VATS technique can safely be used, absolute and relative contraindications to using VATS in pediatrics and preferred treatment in asymptomatic patients. Asymptomatic patients will tend to exhibit symptoms by adolescence. However, the published case reports display a large range of ages when symptoms begin. Cases have been reported in patients ranging in age from newborns to middle aged adults that were either asymptomatic or misdiagnosed.

In symptomatic patients, regardless if their presentation is acute or chronic most researchers recommend surgical resection as the primary treatment. The management of asymptomatic patients remains controversial as some authors advocate close observation and others favor elective resection due to long term risks.[4] Potential complications of leaving the sequestered tissue in place include chronic respiratory infections, commonly fungal or bacterial in nature. Other potential outcomes include cardiovascular disease, hemothorax, pulmonary disease, malignancy and/or hemoptysis that may be fatal due to the abnormal vasculature. Ballouhey et al state that early neonatal surgical intervention should be done for symptomatic patients, otherwise close follow up and surgical resection at about 12 months of age is the ideal treatment of choice.[4]Other researchers recommend resection at as young as 6 months of age. There is a lack of definitive guidelines. Resections can be done by thoracoscopy or by thoracotomy depending on the size, location, regional vascular supply and surgeon's preference and experience.

Regardless of surgical approach, the common post-operative complications that are seen after resection include pneumonia, hemothorax, plural empyema, and pleural effusion. As minimally invasive surgery continues to grow and offer our population new surgical options, the benefit to its utilization becomes more apparent. VATS approach has been utilized for chest disease in adults since 1994.[5] However, its use among the pediatric population is more recent and limited. VATS approach for pulmonary resection has been widely used in the adult population with good results. This approach uses smaller incisions, which results in decreased blood loss and leads to decreased complications, decreased post-operative pain and faster recovery and discharge.

The problems most frequently encountered with this approach is difficulty in handling aberrant arteries and pleural adhesions intraoperatively.[5] Attempting the resection thoracoscopically appears to be universally thought of as beneficial to the patient and overall outcome. It is important to recognize that patients of any age may require the procedure to be converted to an open approach but pediatric patients, statistically, have a much higher rate of conversion to an open thoracotomy. Lagausie et al. report a conversion rate as high as 25%.[1] It was the ultimate outcome of this case. It is reasonable to assume that limited amount of experience with VATs in

pediatric patients plays a significant role. It will be of interest to follow trends as both medical providers and facilities become more fluent in pediatric VATS.

After exploration and interpretation of the available clinical research it is safe to conclude that there is a clear lack of research and case reports utilizing the VATS approach for removal of pulmonary sequestration in the pediatric population. Due to the lack of literature and experience on this subject a clear and concise recommendation cannot be rendered. Researchers have come to similar interpretations, drawn from the limited available data. After reviewing the literature, it appears that VATS is not only safe but on the way to becoming the first line treatment. This trend will continue to grow as surgeons and facilities gain more experience and opportunities to utilize this surgical approach. Anesthesia professionals should remain aware that the potential for conversion to an open thoracotomy is possible among patients of any age but even more so in the pediatric population. Both the lack of documented cases of pulmonary sequestration and the minimal use of VATS for major pulmonary resections in pediatric patients are significant restrictions that limit the conclusions that can be drawn. More research is warranted in the use of the VATS approach for pediatric patients requiring thoracic surgery and pulmonary resections.

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Mentor: Anne Tierney, CRNA, MSN, MA

Anesthesia and Single Coronary Artery Anomaly during Non-cardiac Surgery

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Keywords: congenital coronary anomaly, single coronary artery, left coronary artery anomaly, Pete Maravich heart

A single coronary artery (SCA) is a congenital anomaly in the origin of the coronary arteries in which blood is supplied to the heart by one coronary artery arising from a single coronary ostium.[1] Coronary artery anomalies (CAAs) include a diverse group of congenital disorders with a high variability of pathophysiologic mechanisms of coronary circulation.[2] The majority of

CAAs affect approximately 1% of the population with a SCA occurring in approximately 0.024% of the population.[2] Coronary artery anomalies are usually found incidentally during either coronary angiography or autopsy as they are generally asymptomatic.[2] Some may cause myocardial ischemia and sudden cardiac death, especially in young adults.[2]

Case Report

A 56-year-old, 103 kg, 167 cm female presented for a cervical corpectomy for the treatment of cervical myelopathy. The patient's medical history was significant for hypertension, coronary artery disease, obesity, diabetes, a 20 pack-year history of smoking, and a left coronary artery anomaly. Prior coronary angiography revealed mild stenosis in the right coronary artery and an ejection fraction of 68% but no vessel arising from the left coronary sinus resulting in an absence of a left main coronary artery. Her surgical history included a tonsillectomy and cardiac catheterization without previous anesthetic complications. The patient reported no known allergies. Her home medications included norvasc, aspirin, neurontin, hydrochlorothiazide, metformin, metoprolol, zolpidem, and ibuprofen. Cardiology clearance for surgery was verified. Preoperative midazolam 2 mg intravenous (IV) was administered. A 20 gauge right radial arterial pressure catheter was placed prior to induction of general anesthesia.

Upon arrival to the operating room, the patient was preoxygenated with O_2 10 L/min for 5 minutes and blood pressure, pulse oximetry, and 5 lead electrocardiogram monitors were applied. Anesthesia was induced with fentanyl 100 mcg, lidocaine 100 mg, and propofol 200 mg IV. Manual ventilation was established and rocuronium 50 mg was administered IV. Direct laryngoscopy was performed, and the trachea was intubated with a 7.0 endotracheal tube (ETT). An esophageal stethoscope/temperature probe was inserted, and a lower body air warmer was placed over the lower extremities. An additional 18 gauge IV catheter was inserted, and connected to an inline fluid warmer. Neuromuscular blockade was assessed via peripheral nerve stimulator on the facial nerve, and no response was elicited to train-of-four or tetanic stimulation. Prior to incision, cefazolin 2 g and fentanyl 50 mcg IV were administered.

General anesthesia was maintained with sevoflurane with end-tidal concentrations between 2.0-2.3% in O_2 2 L/min. Phenylephrine was administered as needed to keep the mean arterial pressure greater than 80 mm Hg. The patient's vital signs remained stable throughout the case. At the conclusion of surgery, neuromuscular blockade was antagonized with neostigmine 5 mg and glycopyrrolate 0.8 mg IV. The patient returned to spontaneous ventilation. The oropharynx was suctioned, and the ETT was removed once the patient was awake and protective reflexes were intact. Supplemental O_2 via non rebreather mask was administered. The patient was transported to the post anesthesia care unit without incident. Total surgical time was 3 hours and 30 minutes. Her recovery was uneventful, and she was transferred to the floor on supplemental oxygen.

Discussion

Congenital coronary vessel anomalies are rare and usually present in young adults as sudden cardiac death, congestive heart failure, myocardial infarction, or syncope.[3] These anomalies are rarely identified during life.[4] Approximately 80% of congenital coronary anomalies are benign,

while the remaining 20% can produce life threatening symptoms.[4] Congenital coronary anomalies are the second most common cause of sudden cardiac death in young athletes.[4] Symptoms of CAAs are generally related to a pronounced left-to-right shunt and are frequently associated with other congenital heart anomalies such as transposition of the greater arteries, tetralogy of Fallot, and persistent truncus arteriosus.[2]

There are approximately 20 variants of SCA, according to a classification constructed by Shirani and Roberts.[3] In the more worrisome variants, the intraarterial course of the anomalous coronary artery lies between the aorta and pulmonary trunk; however, mortality does not appear to increase as a result of an intraarterial course.[6] One subtype of SCA, discussed in this case study, is known as a Pete Maravich heart.[5] This is a congenital cardiac condition resulting in the absence of a left main coronary artery, and both the left anterior descending coronary artery and the left circumflex coronary artery arise from the right coronary artery.[5]

Medical management of patients with a known SCA depends on clinical presentation; however, there is no consensus.[1] Asymptomatic patients are generally managed conservatively with observation and pharmaceuticals.[1] As in this present case, the patient had a SCA that had been discovered incidentally, and she had been asymptomatic throughout her life. However, patients who are symptomatic with angina, congestive heart failure, or acute coronary syndromes undergo coronary interventions.[7] Successful treatment has been documented with angioplasty, stent insertion, and coronary artery bypass graft surgery.[7] However, a conflicting study concluded there was no improvement in long term survival with surgical intervention.[6] In the aforementioned study, data collection over a 40-year period found survival to be similar, regardless of whether medical or surgical therapy was utilized.[6] Generally, a good prognosis with SCA has been documented unless one of the branches has an intra-arterial course.[3] With this configuration, compression of the coronary artery between the main arteries can occur during strenuous exercise with resultant coronary steal and ischemia.[3]

Anesthetic management of a patient with a known SCA or other coronary anomalies has never been formally outlined.[1] In the present case, anesthesia technique focused on maintaining hemodynamic stability, optimizing myocardial oxygen supply, preventing arrhythmias in response to noxious stimuli, and monitoring for ischemia. Prompt identification and treatment of ischemia is exceptionally important with the already present coronary circulation anomaly. However, this patient with a known SCA tolerated induction, maintenance, and emergence of anesthesia for a non-cardiac surgery without any difficulty.

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McArdle's Disease and the Risk for Malignant Hyperthermia

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Keywords: McArdle's Disease, Malignant Hyperthermia, TIVA, musculoskeletal disease, myophosphorylase

Malignant Hyperthermia (MH) is well known among anesthesia practitioners. While typically thought of as a familial disease of genetics, MH can occur in conjunction with other genetic musculoskeletal diseases such as McArdle's Disease. McArdle's Disease is an autosomal recessive metabolic disease of skeletal muscle. Patients lack myophosphorylase, preventing glycogenolysis. Patients tolerate light to moderate physical activity, but become easily fatigued with strenuous exercise, requiring rest periods to regain strength. More serious problems associated with McArdle's include rhabdomyolysis, renal failure, and higher risk for MH. Definitive diagnosis includes genetic testing and muscle biopsy.[1, 2]

Case Report

A 20-year-old, 113 kg Caucasian female presented for laparoscopic cholecystectomy following a diagnosis of acute cholecystitis. The patient had a positive medical history for gastro-esophageal reflux disease (GERD), negative past surgical history, and denied prescription medications. When questioned about her allergies, the patient stated her family was allergic to anesthesia. Further investigation revealed a familial history of McArdle's Disease; the patient's father and one sibling had positive muscle biopsy tests and subsequent diagnoses. The patient, however, had never been tested for the disease despite the family members' definitive diagnosis. Preoperative laboratory data revealed a negative urine human chorionic gonadotropin. Preoperative vital signs were blood pressure 131/75 mm Hg, heart rate 88/min, respiratory rate 16/min, and SpO_2 98%. Prior to arrival to the operating room, a new breathing circuit was placed on the anesthesia machine, vaporizers were removed, and the anesthesia machine was flushed with fresh O_2 for 15 minutes at 12 L/min. Once the anesthesia staff and equipment were ready, preoperative midazolam 2 mg intravenous (IV) was administered.

After arrival and transfer to the operating room table, the patient was preoxygenated with O_2 12 L/min for 5 minutes while the following monitors were applied; noninvasive blood pressure, pulse oximetry, electrocardiogram, temperature, and bispectral index. Anesthesia was induced via rapid sequence induction with propofol 200 mg, remifentanil 113 mcg, and rocuronium 70 mg IV. Mask ventilation was not attempted. Direct laryngoscopy was performed and a 7.0 endotracheal tube (ETT) was placed without difficulty. General anesthesia was maintained with total intravenous anesthesia; propofol 125-150 mcg/kg/min and remifentanil 0.05-0.5 mcg/kg/min. Controlled ventilation was achieved with a tidal volume of 550 mL, respiratory rate of 10/min, and an end tidal CO_2 of 30-35 mm Hg.

Approximately 30 minutes after induction, neuromuscular blockade was assessed via peripheral nerve stimulator on the facial nerve. No response was seen with a train of four or tetanic stimulation. Surgery concluded 60 minutes post induction, and neuromuscular blockade was reassessed via peripheral nerve stimulator on the facial and ulnar nerves. The patient exhibited a train-of-four count (TOFC) of 0/4 twitches, followed by 5 seconds of sustained tetany with 50 Hz stimulus and 5 post-tetanic twitches. The patient received neostigmine 5 mg and glycopyrrulate 0.6 mg IV. The patient regained spontaneous ventilation and a TOFC of 4/4 twitches and no fade with sustained tetany stimulus of 5 seconds. The propofol and remifentanil infusions were discontinued. The endotracheal tube was removed and the patient transported to the PACU on O_2 6 L/min via face mask. She remained in PACU for a full hour with temperature checks every 15 min, and a myoglobinuria screen prior to discharge.

Discussion

This case required special attention due to the patient's familial history of McArdle's Disease. McArdle's Disease is also known as glycogen storage disease type V due to the inability to breakdown glycogen and use and store glucose. It is caused by a mutation in the phosphorylase, glycogen, muscle gene, or PYGM, located on chromosome 11q12-q13.2. As previously stated, it is an autosomal recessive disease, meaning that two copies of the mutated gene must be present for the disease to be penetrant. The patient's family history indicated that her father carried two copies and her mother carried one copy putting her at a 50% chance of autosomal recessive inheritance and McArdle's Disease.[3]

Certain muscular diseases have a higher risk of developing MH when anesthetized with known triggering substances. Literature reviewing different diseases and the risk of MH could only verify a weak association of McArdle's Disease with MH. In a study of 8 McArdle patients with 23 general anesthetics, only two of the three patients were found to be MH susceptible. For definitive diagnosis, patients should undergo the in vitro contracture test (IVCT).[1]
Despite the weak association, most literature recommends that patients be treated as MH susceptible, especially due to the fact that there is also high risk for rhabdomyolysis and acute renal failure. Other factors should be taken into consideration for the McArdle's patient in addition to avoiding MH triggers. It has been suggested that patients receive a bolus of a 50% glucose solution before the conclusion of surgery to improve energy to the respiratory muscles. Patients can receive a continuous glucose infusion to ensure a steady energy supply. It is also recommended to prevent shivering by way of keeping the patient normothermic or administering non-shivering pharmacologic agents. Tourniquets should be avoided as well so as to prevent

muscular damage with subsequent myoglobinuria and renal failure. Anesthesia practitioners should carefully weigh their options prior to administering anesthesia to patients with McArdle's disease. They should have a complete understanding of MH and its treatment, should an episode be triggered.[1, 4]

Malignant hyperthermia is a rare, life-threatening disorder whereby patients undergo hypermetabolism in skeletal muscle upon exposure to certain anesthetic agents. The causes of MH remain uncertain but the triggers are well documented. It is known that inhaled volatile anesthetics and the depolarizing muscle relaxant, succinylcholine, trigger an episode. A defect in calcium regulation causes an uncontrollable rise in myoplasmic calcium via the ryanodine receptor (RYR1). When exposed to a triggering agent, calcium is released from the sarcoplasmic reticulum in myocytes. This release results in myocardial contraction. Reuptake mechanisms via RYR1 attempt to remove the calcium from the intracellular space causing an increase in muscle metabolism. This two to three fold increase in metabolism results in increased oxygen consumption, augmented carbon dioxide and heat production, depleted adenosine triphosphate stores (ATP), and generation of lactic acid. Metabolic acidosis, hyperthermia, and ATP stores depletion cause sarcolemic destruction resulting in an efflux of potassium, myoglobin, and creatinine kinase. The uncontrollable release of calcium and ensuing metabolic disaster cause a spectrum of clinical manifestations. Typically, an episode of MH presents while the patient is under general anesthesia. The clinical manifestations include an increase in end-tidal carbon dioxide ($ETCO_2$), tachycardia, tachypnea, skin mottling and cyanosis, muscle rigidity, and hyperthermia. Muscle rigidity is most pronounced in the masseter muscle and probably the most obvious clinical sign.[5, 6]

The Malignant Hyperthermia Association of the United States recommends the following during an MH crisis; notify the surgeon to stop the procedure immediately, call for help and dantrolene, remove the triggering agent, hyperventilate the patient with 100% oxygen at flows >10 L/min, and give IV dantrolene 2.5 mg/kg rapidly through a large bore IV. Dantrolene can be repeated as necessary until the patient responds with decreased $ETCO_2$, decreased muscle rigidity, or lowered heart rate.[6]

Even though the risk for MH in this patient was minimal, it was decided to proceed as though she was truly MH susceptible. In order to properly prepare for the patient, the anesthesia machine had to be readied. As such, all vaporizers were removed, a new breathing circuit was placed, and the machine was allowed to ventilate to a bag with fresh O_2 for 15 minutes to ensure that all volatile agent was removed from the system; a new circuit was then placed. The patient required a rapid sequence induction due to her history of GERD. Therefore, rocuronium was used to lieu of succinylcholine to avoid any possible trigger. She was maintained on a total intravenous anesthetic of propofol and remifentanil. Utilizing these precautions and vigilant care, the case was uneventful and the patient was without negative outcomes. She was discharged home later that same day.

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Myocardial Ischemic Preconditioning with the Use of Volatile Anesthetic Agents

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Keywords: Ischemic, Preconditioning, Coronary Artery Disease, Isoflurane, Sevoflurane

Introduction:

Coronary artery disease leads to 720,000 myocardial infarctions per year and kills nearly 380,000 people.[1] Preconditioning with volatile anesthetic agents has shown to have cardioprotective properties that aid in preventing myocardial ischemia and preserving cardiac function.[2] The proven benefits of anesthetic agents such as sevoflurane, isoflurane, and desflurane have prompted the American College of Cardiology/American Heart Association (ACC/AHA) to establish recommendations for the use of volatile anesthetics in non-cardiac surgical patients whom are at risk for myocardial ischemia.[2] In patients undergoing vascular surgery, the prevalence of perioperative myocardial ischemia is 14% to 47%.[2] Prevention of myocardial ischemia is essential in order to preserve cardiac function.

When the body experiences ischemia, anaerobic metabolism dominates on a cellular level, which causes a decrease in the production of adenosine triphosphate (ATP).[3] If ischemia continues over the course of fifteen minutes, the tissue will become necrotic and the damage is irreversible.[4] A decrease in ATP eventually leads to cell death if oxygen is not restored to the area. Once oxygen is restored to the area of ischemia, reactive oxygen species (ROS) form causing a release of kinases through the cytokine pathway.[3] The activation of the cytokine pathway is thought to preserve cardiac tissue.[4] A period of ischemic preconditioning also causes a decrease in activity of cardiac myocytes, which in turn leads to conservation of energy and ATP consumption, further protecting the tissue.[4]

Volatile anesthetic agents have been shown to have a similar effect to ischemic preconditioning by activating the ATP and ROS cytokine pathway. When cardiac myocytes are exposed to an anesthetic gas, similar to that of isoflurane or sevoflurane, myocardial ischemia is decreased.[4] With ischemic preconditioning, the exposure to a volatile agent readies the mitochondrial K_{ATP} channels.[4] The opening of the mitochondrial channels generates ROS. The activation of both the mitochondrial K_{ATP} channels and the release of ROS, both play an important role in protection from cardiac ischemia.[4]

Methodology

Evidence-based Practice Model
The PICO format was utilized to formulate the clinical question which guided the search criteria. From the research presented on ischemic preconditioning, the following PICO question was formulated: P (patient population) for patients with coronary artery disease, I (intervention) does preconditioning with a volatile anesthetic, C (comparison) compared to no anesthetic agent or total intravenous anesthesia, O (outcome) prevent myocardial ischemia and maintain cardiac function during surgery.

Purpose
The purpose of this review was to determine if preconditioning with volatile anesthetic gases has cardioprotective effects against myocardial ischemia during coronary artery bypass grafting surgery. The clinical questions that were used to guide this literature review included the following: For patients with coronary artery disease, does preconditioning with a volatile anesthetic, compared to no anesthetic agent or total intravenous anesthesia prevent myocardial ischemia and maintain cardiac function during surgery?

Search Models
A thorough search was conducted on several electronic databases including Cumulative Index to Nursing and Allied Health Literature (CINAHL), EBSCOhost, PubMed, ScienceDirect and the Cochrane Library. The search results were restricted to articles that were published in English from the year 2000 to 2015 to establish any gaps within the literature.

Search Terms
Coronary artery disease, ischemia, ischemic preconditioning, sevoflurane, isoflurane, cardioprotection, coronary artery bypass grafting

Levels of Evidence
The evidence utilized for this review was obtained from eight randomized control trails and one systematic review providing level I and level II based on the Joanna Briggs Institute levels of evidence hierarchy.

Literature Review

Activation of Reactive Oxygen Species (ROS)

The opening of mitochondrial K_{ATP} channels generates ROS, a cardioprotective agent that leads to oxygen conservation and protection of the myocardium.[4] Hanouz et al.[5] measured the effectiveness of pretreatment of sevoflurane and desflurane by studying the force of contraction and to what extent improvement was seen in atrial trabeculae in vitro. This study compared the utilization of volatile anesthetic to improve the force of myocardial contraction when compared to no anesthetic agent. The atrial appendages were removed and studied in 42 patients undergoing elective coronary artery bypass grafting (CABG) or aortic valve replacement surgery.[5] The atrial appendages in the control group were exposed to 30 minutes of hypoxia followed by a 60-minute reperfusion period with oxygen.[5] In the preconditioning groups, the atrial appendages were either exposed to 5 minutes of 2% sevoflurane or 6% desflurane followed by a 10-minute washout period.[5] The muscles received the same 30 minutes of hypoxia followed by reperfusion the control group received.[5] The control group saw a recovery in the force of contraction from 13% to 53% after reoxygenation was achieved.[5] When sevoflurane was used, recovery in the force of contraction after 60 minutes went from 53% to 85%.[5] Improvement was also seen in the desflurane group from 53% to 86%, but when ROS was removed from the tissue through the use of N-(2-mercaptopropionyl)-glycine, there was little to no improvement seen with either anesthetic gas only further proving that ROS along with a volatile agent must both be present to provide a cardioprotective effect.[5]

Julier et al.[6] obtained right atrial tissue samples from 72 patients undergoing CABG surgery. Within the tissue the researchers examined the translocation of Protein kinase C (PKC) in response to exposure to sevoflurane.[6] The PKC triggers the opening of the mitochondrial K_{ATP} channels placing into motion the cascade of events that produces a cardioprotective effect.[4] In the tissue samples that were exposed to sevoflurane, PKC had translocated into the sarcolemma and mitochondria, whereas, the samples without exposure to sevoflurane were void of this phenomenon.[6] Julier et al.[6] proved that preconditioning via sevoflurane activates PKC in order to provide a critical step in the process of myocardial protection.

Troponin I

Increased troponin I levels has been shown to be an independent predictor of mortality after surgery.[7] A study conducted by Levy et al.[7] demonstrated that patients with elevated troponin levels are at risk for mortality 6 times that of a patient without elevated troponin I levels up through the first year after surgery.[7] Cardiac troponin I has extreme sensitivity to even the smallest amount of myocardial necrosis.[8]

Amr and Yassin[9] studied the affect of ischemic preconditioning with the volatile anesthetic isoflurane. Fifteen patients undergoing CABG surgery received preconditioning with isoflurane 2.5% for a 10 minute period before aortic cross-clamping, the other 15 patients in the control group received pure oxygen during this same time.[9] Preoperative troponin I levels were measured with each patient and showed to be consistent across groups.[9] Postoperatively, the troponin I levels showed a significant decrease in the group of patients that received isoflurane when compared to the control group.[9] The control groups increase in troponin I levels remained elevated throughout the 36 hour mark of the hospitalization, whereas, the isoflurane treated

patients troponin I levels remained significantly lower.[9] Kottenberg et al.[10] also used troponin I as a measure of myocardial injury when they studied the effect of ischemic preconditioning with isoflurane as opposed to total intravenous anesthesia with propofol during CABG surgery. The researchers in this study, also showed a decreased in troponin I levels within the isoflurane group when compared to the propofol group.[10]

Guarracino et al.[8] showed similar results with desflurane when testing patients who underwent CABG surgery. In this study, 112 patients presenting for CABG surgery were placed into two groups both having similar baseline demographics.[8] The first group received total intravenous anesthesia with propofol 2-3 mg/kg/hr.[8] The second group received a volatile anesthetic of desflurane with a 0.5-2 minimum alveolar concentration.[8] The individuals who received desflurane showed a significant decrease in peak troponin I levels when compared to the group that only received propofol, 1.3 ng/dL vs. 2.8 ng/dL respectively.[8] The researchers continued to observe the troponin I levels at regular intervals starting upon admission to the Intensive Care Unit (ICU) at 4, 8, and 12 hours after surgery.[8] Though there was an increase in troponin levels in both groups after surgery, the patients who received desflurane had significantly lower troponin I levels throughout the 12-hour period after surgery was complete.[8]

Bassuoni and Amr[2] measured the cardioprotective effect of sevoflurane. Forty patients were placed into two groups, the first was induced with 8% sevoflurane and maintained at 1-1.5 minimal alveolar concentration.[2] The second group was induced with propofol 1-2 mg/kg and after intubation maintained on a continuous infusion of 2-3 mg/kg/hr of propofol.[2] Anesthetic concentration was titrated to keep blood pressure and heart rate within 20% of baseline.[2] Again, Bassuoni and Amr[2] showed significantly lower levels of troponin I throughout the 48 hour postoperative period in the patients that received sevoflurane when compared to the patients who received propofol.

Along with troponin I levels, ischemic changes in the form of ST-segment depression were also measured in each patient group.[2] Perioperative myocardial ischemia at the time of surgical stress is often a result of increased oxygen demand.[2] Ischemia due to increased oxygen requirements manifests as ST-segment depression rather than ST-segment elevation.[2] The occurrence of ST-segment depression was significantly less in the sevoflurane ischemic preconditioning group but when ST-segment depression did occur the ischemic preconditioning patients continued to have a lower troponin level even with electrocardiogram (EKG) changes.[2]

Guerrero Orriach et al.[11] took the basis of ischemic preconditioning with anesthetic gases further by introducing administration of sevoflurane in the early postoperative period. The previously mentioned study had 60 participants that were divided into three groups with similar demographics that were presenting for elective CABG surgery.[11] One group received sevoflurane in both the intraoperative and postoperative period after surgery, the second group received sevoflurane throughout the procedure but was then given propofol in the postoperative period, and the third group only received propofol both intraoperatively and postoperatively.[11] The peak level of troponin I was reached 24 hours postoperatively.[11] Guerrero Orriach et al.[11] showed there were significantly lower troponin I levels in the group who received sevoflurane throughout the postoperative period in comparison to the propofol and sevoflurane-propofol groups. The peak troponin I levels in the sevoflurane only group was 0.5 ± 0.4 ng/mL in

comparison to 1.61 ± 1.3 ng/mL and 2.27 ± 1.5 ng/mL in the sevoflurane-propofol group and propofol only group respectively.[11] This research only further confirms that ischemic preconditioning with sevoflurane provides cardioprotection by decreasing troponin I levels intraoperatively as well as postoperatively.[11]

Pro-Brain Natriuretic Peptide

N-terminal pro brain natriuretic peptide (NT-proBNP) is an amino acid that is secreted by the ventricles of the heart in response to the contractile state of the myocardium and the resulting myocardial damage.[6] NT-proBNP has an inverse relationship with left ventricular function, ejection fraction, and cardiac output.[6] NT-proBNP is a sensitive indicator of myocardial dysfunction and much like troponin I has a pivotal role in predicting cardiac risk of heart failure, infarction, and cardiac death.[6]

Julier et al.[6] studied 72 patients undergoing elective CABG surgery who were placed in either a placebo group that received pure oxygen or preconditioning group who received sevoflurane at a rate of 2 minimum alveolar concentration for 10 minutes. NT-proBNP levels were drawn at regular intervals in the preoperative and postoperative periods including arrival to the ICU, 24, 48, and 72 hours after surgery.[6] Though there was no difference between NT-proBNP levels preoperatively for the two groups, postoperatively the NT-proBNP levels remained significantly lower in the group who received sevoflurane.[6] Guerrero Orriach et al.[11] also looked at the effects of NT-proBNP in relation to myocardial dysfunction after CABG surgery. In their study of 60 cardiac patients, NT-proBNP levels was significantly lower in 24 and 48 hours postoperatively in the patients who received sevoflurane as opposed to the patients who received propofol.[11]

Hemodynamic Stability

Amr and Yassin[9] studied 45 patients who received ischemic preconditioning with isoflurane during CABG surgery. Hemodynamics where measured on each participant via EKG monitor, pulse oximetry, and pulmonary artery catheter.[9] Mean arterial pressure (MAP) showed significant improvement in the group that received isoflurane when compared to the control group who received pure oxygen 15 minutes prior to aortic cross clamping.[9] Inotropic support was required by eight of the patients in the control group as compared to four patients in the isoflurane group.[9] Cardiac index was decreased after cardiopulmonary bypass in the control group in comparison to the preoperative values and did not return to baseline values until three hours after the operation was complete.[9] The control group also experienced an increase in heart rate from the baseline values that continued to be elevated 24 hours postoperatively.[9] The isoflurane preconditioning group had an increased left ventricular stroke work index post cardiopulmonary bypass, showing an improvement in left ventricular function and contractility.[9]

Bassuoni and Amr[2] also examined hemodynamic changes in a study with 126 patients undergoing CABG surgery. The patients who received sevoflurane only had 19 of the 64 participants experience tachycardia in comparison to 28 of the 62 patients in the propofol group.[2] Seventy-one percent of the patients who received only propofol experienced hypotension which required inotropic support.[2] A study by Guarracino et al.[8] also showed an increase need for inotropic support in the group that received propofol. Fifty-six percent of the patients in the propofol group required inotropic support postoperatively when compared with 35% of the group who received desflurane.[8]

Reference	ROS	Troponin I	NT-proBNP	Hemodynamics
Hanouz et al., (2007). Reactive Oxygen Species Mediate Sevoflurane- and Desflurane-Induced Preconditioning in Isolated Right Atria In Vitro.	- 42 patients undergoing elective CABG or aortic valve replacement surgery - Appendage exposed to 5 minutes of 2% sevoflurane or 6% desflurane followed by a 10 minute washout period - sevoflurane was used, recovery in the force of contraction went from 53% to 85% - desflurane group from 53% to 86% - when ROS was removed from the tissue through the use MPG there was little improvement seen with either anesthetic gas			
Julier et al., (2003). Preconditioning by Sevoflurane Decreases Biochemical Markers for Myocardial and Renal Dysfunction in Coronary Artery Bypass Graft Surgery: A Double-blinded Placebo-controlled, Multicenter Study.	-right atrial tissue samples from 72 patients undergoing CABG surgery -examined the translocation of Protein kinase C (PKC) in response to exposure to sevoflurane -PKC triggers the opening of the mitochondrial K_{ATP} channels - With sevoflurane, PKC had translocated into the		-NT-proBNP levels were drawn at regular intervals in the preoperative and postoperative periods including arrival to the ICU, 24, 48, and 72 hours -no difference between NT-proBNP levels preoperatively -postoperatively the NT-proBNP levels	

	sarcolemma and mitochondria		remained significantly lower in the group who received sevoflurane	
Amr & Yassin, (2010). Cardiac Protection During On-Pump Coronary Artery Bypass Grafting: Ischemic Versus Isoflurane Preconditioning.		- Preoperative troponin I levels were measured with each patient in this study were consistent across groups - troponin I levels showed a significant decrease in the group of patients that received isoflurane when compared to the control group -control groups increase in troponin I levels remained elevated for 36 hours, -isoflurane treated patients troponin I levels remained significantly lower		-45 patients who received ischemic preconditioning with isoflurane during CABG surgery - measured EKG monitor, pulse oximetry, and pulmonary artery catheter - MAP showed significant improvement in the group that received isoflurane -CI was decreased after cardiopulmonary bypass in the control group in comparison to the preoperative values and did not return to baseline values until three hours after the operation -control group experienced an increase in heart rate from the baseline values that continued to be elevated 24 hours

		postoperatively -isoflurane group had an increased LVSWI post cardiopulmonary bypass
Kottenberg et al., (2012). Protection by remote ischemic preconditioning during coronary artery bypass graft surgery with isoflurane not propofol- a clinical trial.	- showed a decreased in troponin I levels within the isoflurane group when compared to the propofol group	
Guarracino et al., (2006). Myocardial Damage Prevented by Volatile Anesthetics: A Multicenter Randomized Controlled Study.	-112 patients for CABG -propofol 2-3 mg/kg/hr -desflurane with a 0.5-2 MAC - desflurane showed a significant decrease in peak troponin I levels when compared to the propofol group	-56% need for inotropic support in the group that received propofol postop -35% of the group who received desflurane inotropic support postop
Bassuoni & Amr, (2012). Cardioprotective effect of sevoflurane in patients with coronary arty disease undergoing vascular surgery.	-induced with 8% sevoflurane and maintained at 1-1.5 MAC - 2nd group received propofol 1-2 mg/kg and after intubation maintained on a continuous infusion of 2-3 mg/kg/hr -significantly lower levels of troponin I	-126 patients undergoing CABG surgery - sevoflurane only had 19 of 64 with tachycardia in comparison to 28 of 62 patients in the propofol group -71% of the patients who received only propofol experienced

	throughout the 48 hour postop period in the patients that received sevoflurane - ST depression was significantly less in the sevoflurane group but when ST depression did occur the sevoflurane group continued to have a lower troponin level even with (EKG) changes		hypotension that requires inotropic support
Guerrero et al., (2013). Prolonged sevoflurane administration in the off-pump coronary artery bypass graft surgery: Beneficial effects	-60 participants that were divided into three groups with similar demographics for elective CABG surgery - I: sevoflurane in both the intraoperative and postoperative period -II: sevoflurane throughout the procedure but was then given propofol in the postoperative period, -III: received propofol both intraoperatively and postoperatively -peak level of troponin I was reached 24 hours postop, were significantly lower	-lower levels of NT-proBNP in 24 and 48 hours postoperatively in the patients who received sevoflurane as opposed to the patients who received only propofol	

	troponin I levels in the group who received sevoflurane throughout	
Frabdorf et al. (2010). Sevoflurane-induced preconditioning. Impact of Protocol and Aprotinin Administration on Infarct Size and Endothelial Nitric-Oxide Synthase Phosphorylation in the Rat Heart In Vivo.		-ischemic preconditioning with sevoflurane in relation to infarct size in rats -observed an initial decreased in mean aortic pressure and heart rate during the initiation of three cycles of five minute period of sevoflurane delivered at one MAC by a 10 minute washout period with pure oxygen -after the washout was period was complete and before the start of left coronary artery occlusion, these hemodynamic changes disappeared -sevoflurane showed a reduced infarct size after a period of 25 minutes of left coronary artery occlusion and 2 hours of reperfusion

Frabdorf et al.[12] studied the effect of ischemic preconditioning with sevoflurane in relation to infarct size on the rat population. The researchers observed an initial decrease in mean aortic pressure and heart rate during the initiation of three cycles of sevoflurane for 5 minute increments delivered at one minimum alveolar concentration followed by a 10 minute washout period with pure oxygen.[12] However, after the washout was period was complete and before the start of left coronary artery occlusion, these hemodynamic changes disappeared.[12] The preconditioning of sevoflurane showed a reduced infarct size after a period of 25 minutes of left coronary artery occlusion and two hours of reperfusion, therefore, proving there are cardioprotective effects of sevoflurane in rat hearts in vivo.[12]

Conclusions

This literature review has shown volatile anesthetics play a pivotal role in protection of the myocardium during periods of ischemia in both animal and clinical studies. When undergoing the stress of surgery, 18-74% of patients with coronary artery disease will experience myocardial ischemia.[2] Activation of the kinase pathway on the cellular level decreases the effect of ischemia on the myocardium, therefore, preserving cardiac function.[1] Volatile anesthetic agents activate the complex cellular cascade to provide cardioprotection and preserve function in times of surgical induced stress.[5,6,9,11] The preservation of cardiac function, demonstrated by lower levels of troponin I and a decreased need for inotropic support, substantiates improvement in tissue perfusion.[6] Greater tissue perfusion leads to greater surgical outcomes, decreased recovery times, and therefore a reduction in hospital length of stay.[8]

Several limitations are presented in this literature review. Many of the study sample sizes were relatively small with low risk patient populations. No long-term follow up was established for the study participants. Though the patients were randomized into different clinical trial groups, it was difficult to blind the treatment modalities from the clinical practitioners due to the difference in drug administration devices.[11] Due to the variation of drug administration that occurs during surgical procedures, it is challenging to differentiate the cardioprotective affects that occur from the volatile anesthetic agent when inotropic medications and opioids are often utilized as multimodal therapy.[5-7]

Some weaknesses within the animal trials have been identified as a disparity in myocardial response to volatile anesthetic agents versus that of human clinical participants.[5] Premenopausal women have a decreased risk for cardiovascular disease compared to males of the same age, which has been attributed to estrogen secretion.[12] Animal studies have shown inconsistencies between infarct size in relation estrogen secretion.[12] Frabdorf et al.[12] revealed female rabbits had a smaller infarct size compared with male rabbits. Administration of isoflurane 24 hours before ischemia and reperfusion reduced infarct size in male rabbits.[12] The inconsistencies shown in animal studies may support human clinical trials.

Based on the information provided in this literature review, several recommendations can be made to advance the practice of preconditioning with volatile anesthetic agents. Further research into the mechanism of action in the kinase pathway could provide information to direct future clinical trials. A larger clinical trial with long term follow up of patient outcomes can provide insight to the optimal anesthetic agent that should be used to protect against myocardial

ischemia. The risk and/or benefits of preconditioning should be researched for high-risk cardiac patients. Amongst the current research studies, several different protocols were utilized regarding the administration of anesthetic gas for the purpose of preconditioning. It may prove beneficial to develop protocols in providing uniformity with the utilization of volatile anesthetics in regards to preconditioning in the prevention of myocardial ischemia.

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Mentor: Ann B. Miller, CRNA, DNP

Editorial

Happy New Year! 2016 is upon us, with all the hope and aspiration a new year brings with it. I have many items on my to-do list for this year, including an update to the Guide for Authors (which was on my 2015 list as well). Suggestions and feedback are welcome and can be sent to intsjna@aol.com.

We have a good variety of reports in this issue, all but two from different nurse anesthesia programs. At this time, about 28% of nurse anesthesia programs have had a graduate student publish in the ISJNA. I would love to receive submissions from more programs, both within and outside the United States – another item on my list. Toward that end, I hope to have a link to our journal posted on the International Federation of Nurse Anesthetists in the future.

Thank you for another wonderful year of the ISJNA, and for all of the hard work and time contributed by our editors, reviewers, authors, and mentors!

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 practitioners")
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.