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TOPICS IN THIS ISSUE

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Front Cover: Anad Thomas, BSN, a graduate student in the Northeastern University Nurse Anesthesia Program performing a preanesthetic assessment on a patient during her Global Health Rotation in Kigali, Rwanda.

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Table of Contents

Case Reports

Anesthetic Management of Necrotizing Fasciitis

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Keywords: Sepsis, anesthesia, necrotizing fasciitis.

Necrotizing fasciitis is a rarely seen infection of the subcutaneous tissues which creates a challenge for most health care practitioners in both diagnosis and treatment. Overall mortality with this process remains high at 25%. Increased mortality seen in patients with necrotizing fasciitis correlates to age, bacteremia, hypotension, and underlying disease processes.[1] Common co-existing factors such as diabetes mellitus, alcoholism, male gender and immune deficiency place patients at a higher risk of death. Necrotizing fasciitis is delineated into two types. Type one describes a polymicrobial infection and type two is a monomicrobial infection. Necrotizing fasciitis often has streptococcal M protein types, exotoxins A and B, and mitogenic factor causing a release of cytokines from immune cells, which result in shock, multisystem organ failure, myocardial depression, and immune suppression.[1] Patients may exhibit a high white blood cell count, thrombocytopenia, coagulopathy, electrolyte abnormalities, acidosis, hyperglycemia, elevated C reactive protein levels, and radiographic evidence of extensive necrotic inflammation with subcutaneous air .[2] The literature supports an immediate and extensive surgical debridement of the necrotic tissue This debridement, coupled with broad spectrum antibiotics, can reduce mortality rates.

Case Report

A 68 year-old, 55 kg, 162 cm, homeless male presented to the hospital with a necrotic skin infection. His medical history consisted of four years of untreated hypertension and twenty years of alcoholism. The patient's last drink was the previous day. Airway examination revealed a Mallampati grade III view with a limited cervical range of motion less than 70 degrees. On physical examination, the patient's lungs were clear to bilateral auscultation, heart sounds were normal and no edema or jugular venous distention were noted. Initial blood pressure was 89/48 mmHg with a heart rate of 101 beats per minute. Several labs were drawn in the emergency room; hemoglobin was 9 mg/dl, white blood cell count 25 per mm3, sodium 132 mmol/L, C reactive protein > 150 mg/L, the patient's platelet count was 150,000 u/L, and glucose 150 mg/dl. The remainder of the basic chemistry panel was normal. The first blood gas was drawn intraoperatively.

The patient was transferred to the operating room and preoxygenated with 100% FIO2 via facemask. A bolus of hetastarch 500 ml was infused before induction. A rapid sequence induction with propofol 50 mg, ketamine 40 mg, and rocuronium 60 mg was uneventful. A video laryngoscope was utilized to visualize the glottic opening. The trachea was intubated with a 7.0 cm endotracheal tube (ETT). Mechanical volume control ventilation was initiated after ETT placement was confirmed by the auscultation of bilateral breath sounds and end tidal carbon dioxide detection. Initial volume controlled ventilation settings were as follows: tidal volume of 480 ml and ventilation rate 14 breaths per minute, with plateau pressures maintained at 28 cm H_2O. Anesthesia was maintained with sevoflurane

at 0.7 minimum alveolar concentration with one liter flow each of oxygen and air. A radial arterial line and an internal jugular vein central venous catheter were inserted. The patient received clindamycin 900 mg, gentamycin 100 mg, metronidazole 500 mg and 2000 ml of crystalloid at the start of the case via the central venous catheter. The patient's blood pressure was 105/68 mmHg after the initial 2000 ml of crystalloid. The initial arterial blood gas showed a metabolic acidosis with a pH of 7.32 mm Hg, base deficit of 5mEq/L, $Paco_2$ 32 mm Hg.The ventilation rate was decreased to 12 breaths per minute and the tidal volume was maintained at 480 ml. The patient's urine output during the first hour of the case was 30 ml.

During the second hour of the case, the patient's blood pressure trended to 80/40 mmHg over a twenty minute period, and the urine output decreased to 15 ml. Albumin 1000 ml was given and the patient's urine output improved to 120-160 ml an hour. The arterial blood gas and electrolyte panel obtained during the second hour of the case showed a pH of 7.25, base deficit of 10 mEq/L, $Paco_2$ of 35 mmHg, calcium level of 3.43 mg/dl, hemoglobin 7 mg/dl, and lactate of 3 mmol/L. Based upon the results of this blood gas, calcium chloride 1 gm was administered. Ventilator parameters remained the same. The estimated blood loss after the second hour of the case was 800ml.

The patient received 4 units of packed red blood cells and 2 units of fresh frozen plasma. The arterial blood gas obtained during the third hour of the case revealed a worsening metabolic acidosis with a pH 7.21, base deficit 8 mEq/L, $Paco_2$ 32 mm Hg, bicarbonate level 17 meq/liter, , and a lactate level of 4. The central venous pressure (CVP) measured 12 mmHg after the completion of the blood product infusions; however, the blood pressure remained 79/40. A norepinephrine infusion was started at 0.4mcg/kg/min. Vasopressin 1 unit was given intravenously.

The patient's vital signs stabilized with blood pressure of 98/54 mm Hg and heart rate of 84 beats per minutes after administration of the blood products, vasopressin, and the norepinephrine infusion. At the end of the case the last arterial blood gas obtained was: pH of 7.31, base deficit -2, $Paco_2$ of 36 mmHg, bicarbonate level of 21 meq/liter, lactate of 2 mmol/L, Hgb 10g/dl, and oxygen saturation of 98% on an FiO_2 of 0.60 with a final Pao_2 of 150 mmHg. The patient was transferred to the intensive care unit intubated, ventilated, and sedated. He was subsequently discharged six weeks later after several additional debridement procedures.

Discussion

The necrotizing fasciitis mortality rate of 25-35% has not changed in the last 30 years and the likelihood of a health care practitioner encountering the disease is twice in his/her career.[3] The anesthesia practitioner should start fluid resuscitation in the preoperative period to counter hypovolemia, acidosis and hypotension. Hypotension is a sign of progression to septic shock with the potential for multi-system organ failure. Anesthesia practitioners should anticipate this cascade of events, noting the importance of starting preoperative, goal directed therapy. The goals should include improving the overall oxygen delivery to the tissues and resolution of lactic acidosis. This can be accomplished by administering intravenous fluids, such as colloids and blood products. Hemorrhage is not uncommon after debridement, and in cases of disseminated intravascular coagulation (DIC) such

bleeding may be difficult to control.[4] The patient in this case did not develop DIC. It is essential that an arterial line and central venous catheter be placed pre- or intraoperatively for volume resuscitation, frequent lab draws, and continuous hemodynamic measuring in order to achieve the resuscitative goals.

In the hypotensive septic patient with lactate levels >3 mmol/L, the initial resuscitation should include the use of crystalloids, colloids, or blood products with the goal of reaching endpoints such as: CVP 8-12 mmHg, mean arterial pressure > 65 mm Hg, urine output > 0.5 ml/kg, and SVO_2 > 70%.[5] Norepinephrine 0.05-0.5 mcg/kg/min and vasopressin 0.01-0.04 units/min are the recommended vasopressors in sepsis management.[2] Norepinephrine activates adrenergic receptors causing signal transduction which increases intracellular calcium in the sarcoplasmic reticulum through inositol triphosphate mediated calcium release. This leads to vascular smooth muscle contraction.[6] According to Dellinger et al. norepinephrine is more potent than dopamine and may be more effective in reversing hypotension in patients with septic shock.[7] Norepinephrine causes only a slight change in heart rate along with a moderate increase in stroke volume. The patient in this case study was resuscitated with crystalloids, colloids and blood products, and ultimately vasopressor support, in order to reach the optimal endpoints discussed. The Surviving Sepsis Campaign by Dellinger et al. recommends that dobutamine be used as the first line inotrope along with vasopressor support in patients that remain hypotensive after adequate fluid resuscitation.[7] Patients that maintain adequate left ventricular filling pressures and demonstrate continued low cardiac output states can benefit from both inotropic and vasopressor support. The patient did not require a dobutamine infusion but did benefit from inotropic support in the form of calcium chloride.

Monitoring of blood gases, urine output, vital signs, fluid shifts, blood loss, and ventilation is essential to the overall success of the case. Volume replacement with crystalloids, colloids, and blood products coupled with vasopressor support improved the patient's hemodynamic profile. Maintaining electrolyte balance as well as fluid resuscitation and vasopressor support resulted in the improvement of the patient's blood pressure. The sepsis guidelines determined by Dellinger et al [7] indicate maintaining the hemoglobin between 7.0-9.0 mg/dl. Lastly, one should optimize ventilation in order to avoid any ventilation or perfusion mismatch. The patient was maintained throughout the case on volume controlled ventilation with a tidal volume of 450-500 ml, respiratory rate of 12 breaths per minute along with plateau pressures of less than 30 cm H_2O and was subsequently transported to the ICU at the end of the procedure.

The mainstay of treatment is immediate fluid resuscitation, acid/base balance and hemodynamic stability, followed by aggressive surgical debridement.[4] A sound anesthetic plan should be carefully devised to meet the challenges faced with a septic necrotizing fasciitis patient. This will help minimize the incidence of increased morbidity or mortality.

References

1. Leitch HA, Palepu A, Fernandes CM. Necrotizing fasciitis secondary to group a streptococcus. Can Fam Physician. 2000;46:1460-1466.
2. Avidan MS. Infectious disease. In Hines RL, Marshall KE, eds. Stoelting's

anesthesia and co-existing disease. 5th ed. Philadelphia: Churchill Livingstone; 2008:479-482.
3. Sarani B, Strong M, Pascual J, Schwab WC. Necrotizing fasciitis: current concepts and review of literature. J Am Coll Surg. 2009;208:279-287.
4. Hasham S, Matteumli P, Stanley PR, Hart NB. Necrotising fasciitis. BM J. 2005;330:830-833.
5. Eissa D, Carton EG, Buggy DJ. Anaesthetic management of patients with severe sepsis. Br J Anaesth. 2010; 105:734-743.
6. Karlet MC. The endocrine system and anesthesia. In Nagelhout JJ, Plaus KL, eds. Nurse anesthesia, 4th ed. St Louis: Elsevier; 2010:803-804.
7. Dellinger RP, Carlet JM, Masur H, et al: Surviving Sepsis Campaign guidelines for management of severe sepsis and septic shock. Crit Care Med. 2004;32:858-873.

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Small Cell Lung Cancer

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Keywords: small cell lung cancer, prolonged muscular weakness, succinylcholine, paraneoplastic syndrome, Lambert-Eaton myasthenic syndrome

In the United States lung cancer is the leading cause of cancer-related deaths.[1] Small cell lung cancer (SCLC) accounts for 15% of newly diagnosed lung cancers.[2] Arising from malignant epithelial cells, these tumors are rapidly growing and are quick to spread to distant areas.[2] The occurrence of paraneoplastic syndromes are hallmark features of SCLC. The most common paraneoplastic syndrome is Lambert-Eaton myasthenic syndrome (LEMS). Fifty percent of patients diagnosed with LEMS have a concurrent SCLC.[2] The following case report discusses the effect of the neuromuscular blocking drug succinylcholine on the patient with possible SCLC without prior symptoms of LEMS.

Case Report

A 71-year-old, 66 kg, 165 cm Caucasian female presented for laparoscopic cholecystectomy following a diagnosis of acute cholecystitis. The patient had a medical history of ventricular tachycardia, coronary artery disease, chronic obstructive pulmonary disease, small cell lung cancer, hypothyroidism and ischemic cardiomyopathy. The patient's past surgical history included coronary artery bypass grafting and pacemaker/automated implantable cardioverter-defibrillator (AICD) placement. The patient denied a personal or family history of anesthetic complications. Current medications included zolpidem, hydralazine, ipatropium, rosuvastatin, levothyroxine, hydrocodone, trandolapril, nortriptyline and oxycodone. The patient had no known drug allergies. Preoperative vital signs were blood pressure 146/81 mm Hg, heart rate 88 beats/min, respiratory rate 16 breaths/min, and SpO2 98%. Preoperative midazolam 2 mg IV was administered. An arterial pressure catheter

was placed prior to induction of general anesthesia, and the AICD was deactivated as per protocol.

Upon arrival to the operating room the patient was preoxygenated with 10 L/min O_2 for 5 minutes and blood pressure, pulse oximetry and electrocardiogram monitors were applied. Anesthesia was induced with a defasciculating dose of rocuronium 5 mg, fentanyl 100 mcg, lidocaine 100 mg and etomidate 10 mg IV. Mask ventilation was verified and succinylcholine 100 mg was administered. Direct laryngoscopy was performed and a 6.5 endotracheal tube (ETT) was placed without difficulty. General anesthesia was maintained with isoflurane with end-tidal concentrations between 1.1-1.3%. Controlled ventilation was achieved with a tidal volume of 400 ml, respiratory rate of 10 breaths/min, and an end tidal CO_2 of 38-39 mmHg.

Immediately after induction, muscle relaxation was assessed via peripheral nerve stimulator on the facial nerve and no response was elicited to train-of-four (TOF) or tetanic stimulation. At 20 and 40 minutes post-induction, neuromuscular function was reassessed utilizing the facial and ulnar nerves however, both remained absent. At 60 minutes post-induction and the conclusion of the surgery, the patient's neuromuscular function still remained absent. The patient was placed on 10 L/min of oxygen via ambu bag and transported to the post anesthesia care unit (PACU) with an ETT in place. The patient was then mechanically ventilated. One hour after admission to PACU the patient's neuromuscular function remained absent. After three hours in the PACU and four hours post-induction the endotracheal tube was removed and the patient was placed on 2 L/min of oxygen via nasal cannula. The patient was monitored in the PACU for an additional hour and subsequently admitted to the intensive care unit.

Discussion

In this case, a number of putative causes of the prolonged muscle paralysis following succinylcholine were considered. The most commonly recognized reason this occurs is due to pseudocholinesterase deficiency. This disorder represents an inability to hydrolyze succinylcholine, which leads to an overabundance of the molecules that reach the acetylcholine receptors at the neuromuscular junction.[3] The paralytic effect of succinycholine, that in a normal circumstance would only last 3-5 min due to rapid hydrolysis, may now be extended for up to 8 hr.[3]

For this situation, a less common cause was considered. SCLC, also called oat cell carcinoma, is an aggressive cancer that metastasizes more rapidly than non-small cell carcinoma.[4] This metastasis often causes paraneoplastic syndromes, which are defined as signs and symptoms that occur as a result of organ or tissue damage at a site that is remote from the area of primary tumor origin.[4] Paraneoplastic syndromes are triggered by an altered immune system response to the neoplasm.[1] There are several classifications of paraneoplastic syndromes including nonspecific, rheumatologic, renal, gastrointestinal, hematological, cutaneous, endocrine, and neurological.[1] Specific to this case are paraneoplastic neurologic disorders, which are almost exclusive to SCLC. The paraneoplastic neurological disorder of interest in this case is LEMS. Immunoglobin G autoantibodies attack the P/Q type voltage–gated calcium channels at the presynaptic membrane of the neuromuscular junction.[4] As a result, there is a decrease in calcium influx and a decreased amount of acetylcholine released at the neuromuscular

junction causing prolonged muscular weakness.[1] The time to recovery from muscle relaxants is prolonged after the administration of depolarizing and nondepolarizing paralytics.[5] The patient in this case report took approximately four hours to recover full train of four via peripheral nerve stimulation and spontaneous ventilation with a tidal volume of greater than 200 ml with only an intubating dose of succinylcholine.

Typically, paraneoplastic syndrome symptoms appear prior to the diagnosis of SCLC. These symptoms are weakness to the lower extremities, fatigue, diminished or absent deep tendon reflexes, respiratory weakness, blurred vision, diplopia, ptosis, dry mouth and constipation.[1] Electromyography and serum levels for P/Q type voltage-gated calcium channel antibodies are the diagnostic tests used to determine if a patient has LEMS.[1] However, other cases have been reported of patients with LEMS who suffered prolonged recovery from muscle relaxants with no preoperative symptoms or diagnosis.[5,6,7] This patient had no prior diagnosis of LEMS, and therefore the reaction to neuromuscular blockers was not anticipated.

The primary treatment of LEMS in patients with SCLC is eradication of the tumor with a combination of chemotherapy and radiation therapy. Surgical removal is rarely an option due to SCLC being a systemic disease with early spread.[4] The patient described in this case study was currently receiving both chemotherapy and radiation. Additional treatment for LEMS is 3,4-diaminopyridine, an agent that blocks potassium channels and prevents potassium release from the cell. This prolongs the action potential, leaving the voltage-gated calcium channels open longer.[5] The result is increased calcium release, which enhances presynaptic acetylcholine release and muscular strength.[5] Other treatments include plasma replacement and immunosuppressive therapies. However, immunosuppressive therapies are only useful for LEMS treatment in the presence of malignancy.[1] Even with treatment, the survival rate of patients diagnosed with SCLC over 5 years is 5.6% due to relapse and decreased responsiveness to chemotherapy and radiation.[2]

Management of patients with SCLC and concurrent prolonged muscle weakness caused by LEMS requires mechanical ventilation via ETT and continued monitoring of neuromuscular function with a nerve stimulator. Once the patient has demonstrated four out of four twitches and demonstrates full recovery of muscle strength, extubation may be attempted. The patient should be monitored immediately post-extubation and overnight for any extended signs of respiratory difficulty.

In summary, this case presents a unique report of suspected subclinical LEMS perceived only after neuromuscular blocking agents had been administered to the patient with SCLC. Though it does not occur often, anesthesia professionals should be aware that patients diagnosed with SCLC may have undiagnosed LEMS, which only becomes apparent after the administration of paralytic agents. Vigilant monitoring of TOF is paramount in the detection of an altered response to neuromuscular blocking agents and an extended need for respiratory support.

References

1. McClelland M. Paraneoplastic syndromes related to lung cancer. *Clin J of Oncol Nurs*. 2010;14(3):357-364.
2. Kazarian M, Laird-Offringa IA. Small-cell lung cancer-associated autoantibodies:

potential applications to cancer diagnosis, early detection, and therapy. *Mol Cancer*. 2011;10:33.
3. Ama T, Bounmythavong S, Blaze J, Weismann M, Marienau MS, Nicholson WT. Implications of pharmacogenomics for anesthesia providers. *AANA J*. 2010;78(5):393-399.
4. Sher T, Dy GK, Adjei AA. Small cell lung cancer. *Mayo Clin Proc*. 2008;83(3):355-367.
5. Lee CJ, Lim SH, Shin CM, et al. Lambert-Eaton myasthenic syndrome as a cause of persistent neuromuscular weakness after a mediastinoscopic biopsy: A case report. *Korean J Anesthesiol.* 2010;59(1):45-48.
6. Leonovicz BM, Gordon EA, Wass CT. Paraneoplastic syndromes associated with lung cancer: a unique case of concomitant subacute cerebellar degeneration and Lambert-Eaton myasthenic syndrome. *Anesth Analg.* 2001;93(6):1557-1559.
7. Small S, Ali HH, Lennon VA, Brown RH Jr, Carr DB, de Armendi A. Anesthesia for an unsuspected Lambert-Eaton myasthenic syndrome with autoantibodies and occult small cell lung carcinoma. *Anesthesiology.* 1992;76(1):142-145.

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Wolff-Parkinson-White Syndrome

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Keywords: Wolff-Parkinson-White, PSVT, paroxysmal supraventricular tachycardia, ventricular pre-excitation, Bundle of Kent

Wolff-Parkinson-White (WPW) syndrome is a congenital cardiac condition resulting in a ventricular pre-excitation syndrome affecting approximately 0.1 – 0.3 percent of the general population.[1] WPW can result in paroxysmal supraventricular tachycardia (PSVT) due to abnormal electrical communication through an accessory tract pathway traveling between the atria and ventricles known as the Bundle of Kent.[2] Paroxysmal supraventricular tachycardia is a type of symptomatic tachycardia that can be difficult to treat, particularly while the patient is under anesthesia.[2] The cardiac arrhythmias associated with WPW syndrome can lead to sudden death, therefore it is essential to be prepared to appropriately treat arrhythmias in a patient with WPW syndrome.

Case Report

A 31 year-old, 31 kg, 91 cm male presented for a transjugular intrahepatic portosystemic shunt (TIPS) placement for the treatment of portal hypertension and esophageal varices. The patient's medical history was significant for mental retardation, Bruton's aggamaglobulinemia, gastrointestinal bleeding secondary to esophageal varices, idiopathic liver cirrhosis, scoliosis, and WPW syndrome. His surgical history included percutaneous endoscopic gastrostomy, Nissen fundoplication, and rod placement for scoliosis. The patient's mother stated he had a severe fever following scoliosis surgery, however was unable to give details if it was associated with the anesthetic. The patient had allergies to clindamycin, sulfamethoxazole, and paper tape. The medications he was taking at home included nadolol and pantoprazole. He did not have meaningful speech and was dependent on his parents to aid with communication. Significant laboratory

studies revealed hemoglobin 10.4 grams/deciliter (g/dl) and platelets 44,000 per microliter.

The patient was taken to the interventional radiology procedure room and standard monitors were placed. An existing 22g intravenous line was patent in his right wrist. Due to scoliosis deformities, the patient remained upright on the stretcher and was induced with fentanyl 50 mcg, lidocaine 50 mg, and propofol 70 mg. Mask ventilation was easily accomplished with the aid of a 9 cm oral airway, after which rocuronium 30 mg was administered to facilitate successful endotracheal intubation. A mechanical ventilator controlled respirations. Anesthesia was maintained with infusions of propofol at 15mcg/kg/min and remifentanil at 0.03mcg/kg/min. Prior to incision, rocephin 1gm and platelets (268 mls) were administered.

Premature ventricular beats were noted with increasing frequency as the case progressed. Paroxysmal supraventricular tachycardia occurred for 45 sec with TIPS catheter manipulation. A lidocaine 30 mg bolus was given and normal sinus rhythm returned. The radiologist confirmed two stents were unsecured and had come to rest in the right ventricle and pulmonary artery. The patient again experienced PSVT, which progressed to ventricular tachycardia without a pulse. The radiologist performed chest compressions while the defibrillating pads were placed. The patient was defibrillated at 200 joules and received a bolus of epinephrine 1 mg, after which, spontaneous circulation returned. Amiodarone 150 mg was administered intravenously over 10 min and a phenylephrine infusion was titrated for blood pressure support.

A transesophageal echocardiogram was performed which ruled out a patent foreamen ovale. The procedure was abandoned due to the risks associated with retrieving the displaced stents. The patient's respirations continued to be supported by mechanical ventilation and he was transferred to the intensive care unit on phenylephrine and propofol infusions. The patient was extubated three days later. Once the patient's mentation returned to baseline and blood pressure stabilized 11 days following the procedure he was discharged home.

Discussion

The electrical conduction pathway normally originates in the upper right atrium of the heart in an area called the sinoatrial (SA) node. The impulse spreads throughout the atria causing atrial contraction and continues through a tract between the atria and ventricles, the atrioventricular (AV) node. The impulse is delayed slightly while crossing this node to allow the ventricles to fill with blood during atrial contraction. The electrical impulse then distributes throughout the ventricles resulting in ventricular contraction.

In the syndrome of WPW, the Bundle of Kent can manifest itself during periods of stress or noxious stimuli. This accessory tract pathway also located between the atria and ventricle, can bypass the AV node, conducting impulses much faster with no delay across the AV node, allowing for a faster ventricular rate and rapid atrial fibrillation.[3] On the electrocardiogram, a short PR interval or a delta wave (slurring of the QRS complex) produced by early depolarization of the ventricles through the accessory tract is suggestive of WPW.[4]

The Bundle of Kent can conduct electrical impulses both downward through the AV node to the ventricles and return upwards

through the node creating a loop circuit known as a reentry tachycardia, or PSVT.[4] Once this loop circuit is activated, first line responses such as vagal maneuvers or carotid massage can be attempted in an effort to slow the impulse. If these cannot be performed, or are ineffective, amiodarone or cardioversion may be required to achieve a normal rhythm.[5,6]

During this case, a bolus of lidocaine 30 mg was administered to treat the increasing frequency of premature ventricular contractions (PVC). When the patient developed pulseless VT, defibrillation and epinephrine were the most appropriate treatments based on the American Heart Association Advanced Cardiac Life Support guidelines.[7] The patient received an amiodarone bolus after return of circulation to prolong the refractory period of the myocardial tissue and the accessory pathways in the heart.[1] Amiodarone, a class III anti-arrhythmic medication has shown to be successful for a chemically induced cardioversion of PSVT in many patients with WPW.[8] Amiodarone slows the heart rate and prolongs AV nodal, intracardiac, and Bundle of Kent conduction.[9]

Medications that are contraindicated in patients with WPW experiencing acute episodes of tachycardia include calcium channel blockers, beta blockers, and digoxin. These medications may paradoxically increase the ventricular rate by prolonging the conduction time through the AV node. This allows the Bundle of Kent to take over, which can then lead to ventricular fibrillation.[1,10] Many deaths from digoxin have resulted from the direct and indirect inotropic effects on the cardiovascular system.[1] Adenosine is another drug that is not an appropriate selection in this case. While adenosine can cause profound changes in AV node conduction, it does not affect the conduction of accessory pathways. [1]

Inducing anesthesia for the patient with WPW is focused primarily on maintaining hemodynamic stability and preventing arrhythmias in response to the noxious stimuli of laryngoscopy. Several very reliable drugs can be used to blunt this response.[5,6] Propofol has little effect on the refractory period or accessory pathways and is an appropriate choice for the hemodynamically stable patient. Propofol has been shown to decrease the appearance of the delta wave on ECG tracing after its use on patients with WPW.[9] Cisatracurium can be used safely for muscle relaxation. It is metabolized by nonspecific esterases and does not have to be chemically reversed. It is not recommended to use atropine, glycopyrrolate, or ketamine because of the potential to produce tachycardia, which in turn can result in PSVT.[5]

Propofol, lidocaine, fentanyl, and rocuronium were all used with success and little change in hemodynamic stability for this patient. Isoflurane is an agent of choice because it suppresses the accessory pathway.[5] However, volatile anesthetics were avoided due to the patient's history of high fever after scoliosis surgery which may be suggestive of malignant hyperthermia. In conclusion, extra preparation for a patient with WPW is prudent. Documentation of a thorough medical history during the pre-operative assessment, including in depth questioning regarding significant cardiac events and a cardiac evaluation is necessary. A manual external defibrillator and adhesive electrode pads, amiodarone, and emergency cardiac resuscitation medications should be in the room before the start of the case. Careful selection of cardiac medications is imperative and should be well thought out ahead of time to promote positive outcomes.

References

1. Ellis, CR.Wolff-parkinson-white-syndrome. Emedicine. http://emedicine.medscape.com/article/159222-overview#a0101. Updated April, 5, 2010. Accessed March 6, 2011.
2. Hermosura T, Bradshaw W. Wolff-parkinson-white syndrome in infants. *Neonatal Netw*. 2010;29(4):215-223.
3. Kulig J, Koplan B. Wolff-parkinson-white syndrome and accessory pathways. *Circulation*. 2010;122:480-483.
4. Evans-Murray, A. Wolff parkinson white syndrome: what the critical care nurse needs to consider when administering antiarrhythmics. *Aust Crit Care*. 2001;14(1):5-9.
5. Rahul A, Patel RD, Dewoolkar. *Internet Journal of Anesthesiology*. 2007;11(2). http://www.ispub.com/ostia/index.php?xmlFilePath=journals/ija/vol11n2/wpw.xml. Accessed February 12, 2011.
6. Hillel, Z, Landesberg, G. Electrocardiography. In Miller R, Eriksson L, Fleisher LA, et al. *Miller's Anesthesia*, 7th ed. Philadelphia:Churchill Livingstone, Elsevier; 2009;1367-1372.
7. American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2005;112:I70.
8. Kuga, K, Yamaguchi I, Sugishita Y. Effect of intravenous amiodarone on electrophysiologic variables and on the modes of termination of atrioventricular reciprocating tachycardia in wolff-parkinson-white syndrome. *Jpn Circ J*. 1999;63:189-195.
9. Siddoway, L. Amiodarone: guidelines for use and monitoring. *Am Fam Physician*. 2003;68:2189-2197.
10. Knott, L. Wolff-parkinson-white syndrome. Patient.co.uk. http://www.patient.co.uk/doctor/Wolff-Parkinson-White-Syndrome.htm. Updated April 28, 2010. Accessed April 15, 2011.

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Placement of Labor Epidural in Hemophilia A Carrier

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Keywords: labor, epidural, hemophilia A, carrier, factor VIII

Administration of epidural anesthesia is not risk free. The incidence of spinal hematoma following lumbar epidural insertion is less than 1 in 150,000.[1] Coagulopathies increase the risk of this devastating complication during insertion and removal of epidural catheters.[1] Hemophilia affects 1 in 10,000 persons in varying degrees. Specifically, Hemophilia A contributes to 80% of the disease and is characterized by a factor VIII deficiency. Women who are carriers of hemophilia typically have normal levels of factor VIII. Some, however, may have a reduction in the baseline level of circulating factors and are at an increased risk of bleeding during invasive procedures.[2]

Case Report

During the late evening, a 23-year-old gravida 2, para 0 patient was admitted to the

obstetrical unit in active labor at 39 weeks and 1 day gestation. The patient was 170.2 cm tall and weighed 71.8 kg. On admission her cervix was 1 cm dilated and 75% effaced. The patient was a known hemophilia A (factor VIII deficiency) carrier reporting no prior bleeding complications. With appropriate prenatal care, it was discovered she had a succenturiate lobe of placenta, increasing her risk for having a retained placenta. Baseline coagulation studies performed at 35 weeks gestation showed a 42% factor VIII baseline level. Admission laboratory values were as follows: PTT 28.7 seconds, WBC 7.1x103/mm^3, RBC 3.74 million/mm^3, hemoglobin 11.7g/dL, hematocrit 34%, and platelets 178,000/mm^3. Intravaginal misoprostal gel was administered and the patient's membranes were ruptured artificially.

The following morning, oxytocin IV was started at 2 milliunits/min to augment labor. A thorough discussion with the patient regarding the risks and benefits of epidural placement was conducted, focusing specifically on the potential for bleeding complications and hematoma formation. The patient consented and agreed to proceed. One hour prior to labor epidural placement, the patient received an IV infusion of antihemophilic recombinant factor VIII. Labor analgesia was provided by continuous lumbar epidural when she was 3 cm dilated and 95% effaced, with the fetus at station 0. Successful catheter placement was achieved with an 18 gauge Tuohy needle inserted midline at the L3-4 interspace after 2 attempts. The epidural space was encountered at 6 cm by loss of resistance technique with an air and saline mixture. The catheter was easily inserted 5cm into the epidural space. Following a negative aspiration of cerebrospinal fluid or blood, a 3ml test dose of lidocaine 1.5% with epinephrine 1:200,000 was administered. After confirming a negative test dose, the patient was given a 6ml bolus of ropivacaine 0.2% with fentanyl 100mcg via the epidural catheter. A continuous epidural infusion of ropivacaine 0.2% at a rate of 14 ml/hr was initiated.

The patient was hemodynamically stable both prior to and after labor epidural catheter insertion. The patient continued laboring and reported satisfactory pain control. The patient vaginally delivered a 7 pound 8 ounce infant without complication. One minute and five minute Apgar scores were 9 and 9 respectively. Placental delivery followed 25 minutes after delivery. Total estimated blood loss was 350ml. The epidural catheter was discontinued shortly after delivery without complications. The insertion site was dry without visible hematoma. The patient was discharged 2 days post-delivery.

Discussion

There are no definitive guidelines for placing labor epidurals in patients with inherited bleeding disorders despite neuraxial anesthesia techniques being used in approximately 60% of women for control of labor pain.[4] Because hemophilia is a relative contraindication for epidural placement, anesthesia practitioners should proceed with caution when caring for a patient with a known deficiency. Most women who are carriers of Hemophilia A have clotting factors that are within the normal range by the end of their 3rd trimester.[5] Although clotting factors in pregnant women with inherited bleeding disorders may increase to normal non-pregnancy levels, they do not necessarily reach normal levels found in patients without a bleeding disorder. Pregnancy causes the epidural veins to become

engorged and distended leading to increased risk of venous puncture and bleeding in the epidural space. This may lead to hematoma formation and subsequent compression of the spinal cord with the possibility of permanent neurologic consequences.[4] The consensus in the literature is that factors should be replaced specific to the patient's deficiency with a known hemophilia diagnosis.[3] Labor epidural placement is considered safe if coagulation factors are > 0.5 IU ml^{-1} or 50%.[3] Levels of factor VIII should sustain this level for insertion and removal of the epidural catheter.[6] In addition, the patient should have a normal coagulation profile as an abnormal aPTT can be an indication of a deficiency in factor VIII levels.[8]

In the case presented above, the patient's functioning factor VIII levels were less than 50% despite her admission coagulation studies being within normal limits. Replacement of factor VIII at the recommended dose of 40 units/kg was administered as requested by the patient's hematologist. This treatment and course of action is consistent with what is found in the literature. In this case review, the anesthesia practitioners were aware of the patient's status prior to her admission and were able to have replacement factors readily available. This patient was given antihemophilic recombinant factor VIII. The half-life of this factor VIII replacement is 9-14 hours. It was not necessary to re-dose the patient prior to removal of her epidural catheter as she delivered 7 ½ hours after epidural catheter placement. Should the patient have labored for a period of time exceeding the ½ life of antihemophilic recombinant factor VIII, she would have required an additional dose before removing the epidural catheter. Epidural catheter removal is also a time of increased risk. It has been shown that 50% of epidural hematomas are associated with removal of the catheter.[5]

In a scenario where providers are not alerted prior to patient presentation and replacement of factor VIII is necessary, multiple alternative treatment options are available. Cryoprecipitate or fresh frozen plasma can be used as an alternate treatment as they both contain factor VIII. Additionally, desmopressin is an alternative treatment option for patients with mild hemophilia A that exhibit a decrease in factor VIII levels. Treatment with this medication will increase the release of both von Willebrand factor and factor VIII.[7] This patient did not receive desmopressin because she did not exhibit a decrease in von Willebrand factor levels. As long as these factors remain at normal levels from the time of insertion through the time of catheter removal, patients are not an increased risk for the development of an epidural hematoma.[6] A midline approach to insertion is favored to avoid the epidural veins.[8] A review of literature relays that patients with bleeding disorders, such as hemophilia A, can benefit from a labor epidural without an increased risk for epidural hematoma subsequent to proper planning. Collaborative involvement from the patient's obstetrician, hematologist, and anesthetist is necessary for the comprehensive care of this obstetrical patient population.[5]

References

1. Barash PG, Cullen BF, Stoelting RK, Cahalan MK, Stock MC. *Clinical Anesthesia*. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009.
2. Giangrande P. Pregnancy in women with inherited bleeding disorders. *Treatment of Hemophilia*. 2003;29:1-9.
3. Choi S, Brull R. Neuraxial techniques in obstetric and non-obstetric patients

with common bleeding diatheses. *International Anesthesia Research Society*. 2009;109:(2):648-660.
4. Chow L, Farber M, Camann W. Anesthesia in the pregnant patient with hematologic disorders. *Hematol Oncol Clin North Am*. 2011;25:425-443.
5. Chi C, Lee CA, England A, Hingorani J, Paintsil J, Kadir RA. Obstetric analgesia and anaesthesia in women with inherited bleeding disorders. *J Thromb Haemost*. 2009;101:1104-1111.
6. Hara K; Kishi, Nobuyuki K, Sata T. Considerations for epidural anesthesia in a patient with type 1 von willebrand disease. *J Anesth*. 2009;23(4):597-600.
7. National Heart lung and Blood institute. Hemophilia. National Institutes of Health. http://www.nhlbi.nih.gov/health/dci/Diseases/hemophilia/hemophilia_what.html. Updated July 2009. Accessed April 13, 2011.
8. Dhar P, Abramovitz D, DiMichele D, Gibb CB, Gadalla F. Management of pregnancy in a patient with severe haemophilia a. *Br J Anaesth*. 2003;91(3):432-435.

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Ossification of the Posterior Longitudinal Ligament

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Keywords: cervical, spinal cord injury, elderly, OPLL, positioning

The geriatric population, older than 65 years, is expected to reach 69 million by the year 2030 in North America.[1] This age group has a higher risk for cervical spine injuries than does other age groups.[2] During the aging process, the cervical spine undergoes degenerative changes making it more vulnerable to spinal cord injuries (SCI).[3] There is also a propensity towards spinal canal stenosis with aging, that increases the risk of a SCI from minor trauma or even from the positioning during a surgical procedure.[1,3]

Case Report

A 57 year old, 150 cm, 93 kg, female presented for posterior cervical laminectomy and decompression of her cervical vertebra 2 through 5. Four days earlier she had sustained a fall and had struck her forehead on a wall causing acute cervical hyperextension. She had immediately noticed loss of sensation below the neck level, weakness to all extremities, rectal incontinence, followed by severe neck pain. Her past medical history included type II diabetes mellitus and hypertension. Current medications included regular and glargine insulin, pantoprazole, simvastatin, methocarbamol and methylprednisolone. A complete spine computerized axial tomography (CT) scan showed “no acute cervical fracture, but extensive ossification of the posterior longitudinal ligament causing severe multilevel central stenosis, thoracic posterior disk osteophtye at T6 and 7, causing moderate central stenosis, and no acute thoracic fracture”. The lumbar spine had “no acute lumbar fracture but severe central stenosis at L4 and 5.” Magnetic resonance imaging (MRI) of the cervical spine, with and without contrast, showed

"severe central stenosis and cord compression from C2-5 and a large area of abnormal intramedullary cord signal consistent with myelomalacia." MRI of the thoracic spine revealed a "left paracentral disk osteophyte at T6-7."

Preoperative exam revealed a patient who was alert and oriented to person, place and time, had spontaneous respirations, and appeared in no acute distress. A cervical collar was in place and the patient had no voluntary movement of extremities and no sensation below the C2-3 dermatome level. The patient, surgeon, and anesthesia team discussed the plan of an awake intubation utilizing a video laryngoscope, general anesthesia, and surgery in a sitting position using Mayfield tongs.

The patient was given glycopyrrolate 0.1 mg, and ketamine 10 mg intravenously (IV) and transported to the operating room where basic monitors meeting the anesthesia standard of care were applied. Oxygen was administered at 10 L/min via face mask for five minutes. Ketamine 20 mg, fentanyl 50 mcg, and propofol 50 mg were administered IV prior to careful video laryngoscope insertion and intubation of her trachea. Her cervical spine was maintained in neutral alignment with rigid cervical collar in place. General anesthesia was maintained with sevoflurane at an end tidal concentration of 1.8% and vecuronium 6 mg IV.

The patient was then transferred to the surgical table maintaining neutral cervical alignment with minimal neck movement and her head positioned in the Mayfield headrest per the surgeon after he removed her cervical collar. Phenylephrine and ephedrine IV were used to maintain the mean arterial pressure greater than 90 mmHg throughout the procedure. Once the surgery was completed, neuromuscular blockade was antagonized using neostigmine 5.0 mg and glycopyrrolate 1.0 mg IV. The patient had 4 of 4 twitches and sustained tetanus for five seconds and was taken to the post anesthesia care unit (PACU) where she was extubated once fully awake, responsive, and able to achieve a negative inspiratory force of -20 mmHg. The patient has had no improvement to her tetraplegia and remains hospitalized four months later.

Discussion

This patient presented with severe cord stenosis and cord compression resulting in tetraplegia. She had fallen and struck her forehead on a wall, hyperextending her neck causing impingement on a pre-existing stenotic cervical cord canal. She had a history of falls and intermittent numbness and tingling in her hands. She had sought treatment 1 month prior to the current admission and a CT scan revealed cervical stenosis, for which she had been referred to see a neurosurgeon. This patient was diagnosed with ossification of the posterior longitudinal ligament (OPLL), which originates with degeneration of ligamentous fibers followed by endochondral ossification. Seventy percent of the cases involve the cervical spine resulting in progressive degeneration, increasing spinal stenosis, and severe neurologic dysfunction.[5]

The incidence of OPLL is mostly reported in Japanese literature because of the high rate of occurrence in Asian populations.[4,5] It has been reported to affect 1.9% to 4.3% of the population over 30 years old, with men being affected more than twice as often as women. The occurrence rate increases 80% for every 10 years of age, suggesting a slow, degenerative nature. Patients can be asymptomatic for years, but the disease severely affects spinal cord function and often leads to surgical intervention.[4,6] OPLL

is a significant health problem in Japan and has been designated by the Japanese Ministry of Public Health and Welfare as an intractable disease.[5] Although OPLL occurs more frequently in the Asian populations, it has also been documented to occur in the non-Asian populations as well.[4,5,6]

Cervical spine degeneration impacts care for patients. According to Hindeman et al., most cervical cord injuries are not due to trauma, instability or airway management.[3] Risk factors for a SCI are having a cervical spine procedure, being in the sitting position, and/or having cervical spondylosis.[1] In this country, cervical spondylosis is the most common factor associated with spinal cord injury in closed claim analysis.[3] Cervical spondylosis is a degenerative disease process that causes disc space collapse, osteophyte development and ligament buckling, resulting in canal stenosis and cord compression.[1,7] Disc changes, intervertebral narrowing and/or disc herniation occur in twenty to thirty percent of patients by age forty.[3] Geriatric patients with spondylosis are at a higher risk of having a SCI due to minor trauma, brief hyperextension, positioning of the head and neck during surgical procedures, and hypotension.[1,3] Patients with severe chronic stenosis have limited perfusion reserves and appear to be more susceptible to ischemia.[3]

When providing care to the older population, great care should be taken in manipulating the head and neck. One should maintain neutral alignment as much as possible and avoid severe hyperextension of the neck. Many of the degenerative disease processes that can affect the elderly cannot be diagnosed with simple radiographic films, and often require CT scans or MRI.[1,2,8] These degenerative processes occur slowly over time and patients may not exhibit any symptoms.[3,6] Careful preoperative evaluation for any subtle symptoms of cervical canal stenosis, such as weakness or paresthesias in the upper extremities, may suggest a possible underlying problem. Cervical cord injury frequently results in alterations in respiratory function and cardiovascular instability making the anesthesia professionals goal of minimizing secondary injury by optimizing cord perfusion and oxygenation even harder to achieve. Aggressive prevention and treatment of hypotension, hypothermia, and maintaining arterial oxygen content at a normal level are essential elements of care.[3,7]

In this case study, the patient was managed according to the literature. Several aspects of this case required increased vigilance to reduce the risk to her already compromised spinal cord. Although the most appropriate technique for tracheal intubation in a patient with cervical spine injury continues to be debated, authors agree that maintenance of alignment and cervical immobilization are a crucial aspect of care.[3,7] Direct laryngoscopy and tracheal intubation can cause pathologic cervical motion that can be detrimental in a patient with a cervical cord injury.[3] Although several techniques (lighted stylet, fiberoptic bronchoscopes, nasal intubation, tracheostomy) could have been employed, this patient remained in a rigid cervical collar to limit neck motion and was intubated, while awake, using a video laryngoscope. Sedation and induction of anesthesia could have been carried out with different combinations of medications with efficacy. Alternative sedatives include midazolam, diazepam, lorazepam, dexmetomidine, or propofol. According to closed claim analysis, the sitting position carries a disproportionate, twofold risk of cervical cord injury related to malpositioning and decreased cord perfusion.[3] Neutral alignment of the cervical

spine was carefully maintained throughout positioning. Although an arterial line might have facilitated more precise blood pressure monitoring, the mean arterial blood pressure was monitored closely and treated prudently with vasoactive medications to maintain cord perfusion. The anesthesia team developed and followed a plan of care that effectively managed this patient from the preoperative holding room, throughout the surgical procedure, and into the PACU.

References

1. Jabbour P, Fehlings M, Vaccaro AR, Harrop JS. Traumatic spine injuries in the geriatric population. *Neurosurg Focus.* 2008;25(5):E16.
2. Ong AW, Rodriguez A, Kelly R, Cortes V, Protech J, Daffner RH. Detection of cervical spine injuries in alert, asymptomatic geriatric blunt trauma patients: Who benefits from radiologic imaging? *Amer Surg.* 2006;72:773-777.
3. Hindman BJ, Palecek JP, Posner KL, et al. Cervical spinal cord, root, and bony spine injuries. A closed claims analysis. *Anesthesiol.* 2011;114(4):782-795.
4. Sugrue PA, McClendon J, Halpin RJ, Liu JC, Koski TR, Ganju AG. Surgical management of cervical ossification of the posterior longitudinal ligament: natural history and the role of surgical decompression and stabilization. *Neurosurg Focus.* 2011;30(3):E3.
5. Wang MY, Thambuswamy M. Ossification of the posterior longitudinal ligament in non-Asians; demographic, clinical, and radiographic findings in 43 patients. *Neurosurg Focus.* 2011;30(3):E4.
6. Wu JC, Liu L, Chen YC, Huang WC, Chen TJ, Cheng H. Ossification of the posterior longitudinal ligament in the cervical spine: an 11-year comprehensive national epidemiology study. *Neurosurg Focus.* 2011;30(3):E5.
7. Kumar R, Taylor C. Cervical spine disease and anaesthesia. *Neurosurg Anaesth.* 2011;12(6):225-228.
8. Cain G, Shepherdson J, Elliott V, Svensson J, Brennan P. Imaging suspected cervical spine injury: Plain radiography or computed tomography? Systematic review. *Radiol.* 2010;16:68-77.

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The Lightwand: A Valuable Adjunct to Airway Management

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Keywords: difficult intubation, lightwand, limited atlanto-occipital joint extension, transillumination, lighted stylet

While direct laryngoscopy remains the standard method for tracheal intubation, anesthesia practitioners should be skilled with various alternative airway devices. Tracheal intubation under direct vision is difficult in 4.5% to 7.5% of the general surgical population;[1] therefore, knowledge and utilization of various intubation techniques is imperative for patient safety.[1] A difficult intubation, whether anticipated or unanticipated, may be aided with a simple and cost-effective light-guided airway device. Devices utilizing transillumination may be clinically useful for the patient with

congenital or acquired abnormalities of the upper airway, short thyromental distance, short neck, high Mallampati grade, and/or secretions in the oropharynx.[2]

Case Report

A 45-year old, 197 cm, 124 kg Caucasian male presented as a recipient for a living donor kidney transplant. The patient had no known drug allergies. The patient's history included immunoglobulin A nephropathy, controlled hypertension, hyperlipidemia, rheumatoid arthritis (RA), gout, and hyperparathyroidism. The patient was hemodialyzed three days per week, with the last treatment two days before surgery. His medications included allopurinol, multivitamin, calcium carbonate, nifedipine, and simvastatin all taken daily, and carvedilol, taken twice daily. The patient quit smoking 1 year ago after a five pack-year smoking history. Previous surgeries included a left radiocephalic arteriovenous fistula in December 2009, a ventral hernia repair in August 2010, and an appendectomy in August 2010. He reported no anesthetic complications. Physical examination revealed a Mallampati Class III airway, thyromental distance of one fingerbreadth, a 2 cm oral aperture, short neck, and normal Atlanto-occipital joint extension. The patient had large facial bones and natural dentition. A 16 gauge intravenous catheter was inserted into the right antecubital vein without difficulty and midazolam 2 mg was administered intravenously.

In the operating room with standard monitors applied, the patient was pre-oxygenated with 100% oxygen for approximately 5 minutes. Anesthesia was induced intravenously with fentanyl 50 mcg, lidocaine 100 mg, and propofol 225 mg. The patient was easy to mask ventilate and was given cisatricurium 12.5 mg intravenously. A lightwand technique was selected for tracheal intubation based on the patient's physical exam. The lightwand was medially inserted into the airway without difficulty. Transillumination of the trachea was obtained following advancement of a 7.5 mm oral endotracheal tube into the trachea. The tube was secured at 23 cm at the lip. Endotracheal tube placement was confirmed with positive end-tidal CO_2 tracing, bilateral breath sounds, and equal chest rise with positive pressure ventilation. Intravascular volume was monitored and replaced based on central venous pressure goal of 10-15 mm Hg to maintain hemodynamic stability. At the conclusion of the procedure, the patient's neuromuscular blockade was antagonized with neostigmine 5 mg and glycopyrrolate 0.8 mg intravenously. The patient demonstrated return of spontaneous ventilation at a rate of 13 breaths per minute with exhaled tidal volumes of 500 ml and was able to follow commands. The patient was orally suctioned and the endotracheal tube was removed with positive pressure without difficulty. The patient's transfer to the intensive care unit with O_2 10 L/min via facemask was without incident with stable vital signs and a patent airway.

Discussion

According to the American Society of Anesthesiologists (ASA), the definition of a difficult airway is a situation in which the "conventionally trained anesthesiologist experiences difficulty with intubation, mask ventilation or both."[1] The ASA first established the difficult airway algorithm in 1993 as a concise guide to prevent adverse outcomes and later updated the algorithm in 2003.[1] Although the most commonly utilized alternative airway device for the difficult airway remains the flexible fiberoptic bronchoscope, the light-guided airway device is identified in the ASA's difficult airway algorithm as an alternative

approach to intubation.[1,3] Completion of a thorough preoperative evaluation of the patient's airway by the anesthesia practitioner will provide insight regarding the potential difficulty of the airway. With an anticipated difficult airway, the anesthesia practitioner has time to consider strategy, optimize the situation, and obtain all necessary equipment and resources for airway management.

There is evidence to support the lighted stylet as an effective and safe method for tracheal intubation for those patients that demonstrate a difficult airway or failed standard intubation.[4] Various versions of the lightwand have been in existence since 1959, with necessary updates to the device more recently.[5] The device is portable, cost effective, and most importantly, has been identified as atraumatic.[6] Securing an airway utilizing a lightwand is a blind, light-guided technique that does not provide direct visualization of the laryngeal structures.[4] Rather, this type of airway device is dependent upon transillumination of the tissue over the trachea and larynx.[1] The distal end of the wand contains a bright lightbulb, which, once placed inside the glottis, demonstrates a bright glow visible in the tissue over the anterior neck.[5] Should the device be inserted into the esophagus, no transillumination of the tissue will be noted.[5] For successful placement of the lighted airway device, the tip of the wand must be bent at a 70-90 degree angle at 6.5 cm.[6] The practitioner must utilize a jaw lift for medial oral insertion, and the wand must be adequately lubricated prior to placing the endotracheal tube over the device.[4,6] Once the anterior neck is transilluminated, the anesthesia practitioner continues advancement blindly into the trachea. The wand is then removed from the endotracheal tube.[4] Confirmation of endotracheal tube placement is validated with auscultation of bilateral breath sounds, symmetrical chest rise, and end-tidal CO_2.

While the lightwand is an appropriate airway device selection for many patients, the skill and experience of the anesthesia practitioner must be considered for the success of such a device.[5] The lighted stylet remains a safe choice for the patient with minimal movement of the cervical spine, and is also described as being a safer alternative when compared with direct laryngoscopy.[7] When using a light-guided airway device, the patient is able to maintain a neutral head position. RA, as identified in this case report, may be associated with cervical spine immobility, as well as atlantoaxial instability.[8] Patients with atlantoaxial subluxation have a greater risk of mortality, due to the possibility of spinal cord compression.[8] For this reason, in addition to the elevated Mallampati score and decreased oral aperture, the lightwand was a safe and effective method for intubation in the presented case. The lightwand has also been associated with reduced incidence of dysphagia, hoarseness, and throat discomfort postoperatively when compared with direct laryngoscopy.[6] In comparison with a glidescope, or video-assisted intubation device, the lighted stylet demonstrates a milder hemodynamic response and quicker intubation time.[7] With this case report, the hemodynamic response associated with intubation was attenuated with use of the lightwand. It was also noted that there was very little deviation in hemodynamic status post-intubation when compared with vital signs during the pre-induction period. Intubation of the patient's trachea with the lighted stylet was successful with one attempt. While the lightwand was the chosen airway device for the patient's anticipated difficult airway, all anesthesia practitioners involved in this case thoroughly discussed the anesthetic plan.

Additional airway equipment (i.e. gum elastic bougie, fiberscope, laryngoscope with Macintosh and Miller blades) was readily accessible, providing alternate airway management options in accordance with the ASA's difficult airway algorithm.

Since a difficult airway is not a rare occurrence, it is imperative that anesthesia practitioners remain proficient with the ASA's difficult airway algorithm, as well as skilled with various advanced airway devices. As evidenced with the presented case report, the light-guided technique is an economical and valuable tool for managing a challenging airway. At approximately $69 for a durable kit, the lightwand is a more cost-effective option than other devices costing hundreds of dollars.[6] Demonstrating skill with multiple airway modalities allows anesthesia professionals to safely render care in the most urgent of situations.

References

1. Rosenblatt WH, Sukhupragarn W. Airway management. In Barash PG, Cullen BF, Stoelting RK, Cahalan MK, Stock MC eds. *Clinical Anesthesia*. 5th ed. Philadelphia, PA: Lippincott,Williams and Wilkins; 2009: 771-773, 787.
2. Rhee K, Lee J, Kim J, Park S, Kwon W, Han S. A comparison of lighted stylet and direct laryngoscopic intubation in patients with high Mallampati scores. *Anesth Analg*. 2009;108:1215-1219.
3. Hung O, Law A. Continuing medical education: advances in airway management. *Can J Anaesth*. 2006;53:628-631.
4. Park EY, Kim JY, Lee JS. Tracheal intubation using the Airtraq: a comparison with the lightwand. *Anaesthesia*. 2010;65:729-732.
5. Agro F, Hung OR, Cataldo R, Carassiti M, Gherardi S. Lightwand intubation using the Trachlight: a brief review of current knowledge. *Can J Anaesth.* 2001;48:592-599.
6. Bamgbade OA. Lightwand intubation for difficult airway patients. *Int J Anesth*. 2007;11:1-4.
7. Huang W, Huang C, Chung, Y. Clinical comparisons between the Glidescope video laryngoscope and Trachlight in simulated cervical spine instability. *J Clin Anesth*. 2007;19:110-114.
8. Imagama S, Oishi Y, Miura Y, et al. Predictors of aggravation of cervical spine instability in rheumatoid arthritis patients: the large joint index. *J Orthop Sci*. 2010;15:540-546.

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Laryngoscopic Techniques for Obese Patients

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Keywords: obesity, airway management, difficult airway, reduced functional residual capacity, pre-oxygenation in obese

Obese patients pose a unique challenge to anesthesia providers because they possess several anatomical qualities which can impede intubation. The combination of anatomically small mouth opening, short thyromental distance, as well as excessive redundant tissue, can impair the

larygoscopic view. Obese patients have increased airway hyper-responsiveness due to accelerated airway remodeling and reduced functional residual capacity which creates a higher risk of hypoxia.[1] The case reported will examine the care of an obese male, the preoperative evaluation of his airway, and steps taken to ensure an optimal situation for laryngoscopy and intubation.

Case Report

A 33 year old, 178 cm,166 kg Hispanic male with a history of obstructive sleep apnea and obesity was admitted through the emergency department for an emergency appendectomy. The patient spoke adequate English and denied taking any daily medications, though he stated that he had not received any medical attention in years. It had been recommended that he use a bipap machine at home several years prior, but he never purchased one. The patient stated that he often woke up during the night short of breath and had difficulty walking up a flight of stairs without experiencing shortness of breath. He also stated that he did experience gastric reflux frequently during the night. He denied having any chest pain or cardiac conditions, seizures, liver disease, smoking, diabetes, thyroid disease or any other pulmonary diseases and denied ever using any inhalers. Upon auscultation, his lungs were clear bilaterally and his heart had a regular rate and rhythm. The patient's admission bloodwork was positive for cocaine and marijuana. The patient stated that he had used both drugs the day of his admission.

Upon examination of the patient's airway, he had a Mallampati class III score with head raised off of the bed and head tilted back as far as comfortably possible. The patient had no limitations to range of motion of his neck. He had a thyromental distance (TMD) of approximately 1.5 cm, and a mouth opening measurement of approximately 4 cm. The patient had a full set of teeth, however with very poor condition and several crowns. The patient denied having any loose teeth or removable dental work.

The anesthesia plan included a rapid sequence induction and would utilize ramping and the Backwards, Upwards, Right Pressure (BURP) maneuver. Ramping is positioning a patient using pillows, blankets, or inflatable devices underneath the patient's shoulders to assist in aligning the airway axes. The patient was ramped using blankets and was preoxygenated for more than 2 minutes with 100% oxygen by mask which was held firmly by the circulating nurse while the patient was instructed to take deep breaths. The patient was premedicated with midazolam 2 mg prior to being moved and positioned on the operating room (OR) table. The induction medications were fentanyl 100 mcg, propofol 200 mg, and succinylcholine 140 mg. A rapid sequence induction was performed with no attempted manual ventilation. Cricoid cartilage pressure was applied using the BURP maneuver. The trachea was intubated with a 7.5 endotracheal tube via direct laryngoscopy using a miller 4 blade on the first attempt with a grade II glottic view.

Discussion

The preoperative evaluation of the airway is accomplished through history, physical exam, anatomic airway measurement, and non-invasive clinical tests. Important conditions in the patient's history include diabetes, obstructive sleep apnea, trauma, surgery to the airway and a previous history of a known difficult airway. Prior operative notes can be helpful if they are available. A

thick, short neck, obesity and obstructive sleep apnea are significant predictors for difficult intubation and are often seen together.[2]

The Upper-Lip-Bite Test (ULBT) has three grades that are useful in evaluating the ability of the patient to cover the upper lip with their lower incisors and are as follows: Grade I - lower incisors can fully cover upper lip; Grade II - upper lip is touched by the lower incisors but not covered; Grade III - inability to bite the upper lip. A Grade III ULBT has been associated with difficult intubations.[3] The advantage of the ULBT is its ease of applicability, reliability, and accuracy of prediction. Original reports using the ULBT showed more accuracy of prediction than the use of the Mallampati classification system.[4]

Other methods to test the mandibular space are the evaluation of TMD and hyomental distance. Thyromental distance is defined as the distance between the tip of the mandible to the thyroid cartilage and is greater than or equal to 6.5 cm in the normal adult with a good airway.[5] Sternomental distance (SMD) is another test that measures from the tip of the chin to the sterna notch and should be greater than 12.5 cm and essentially takes into account the amount of neck extension. Measurement of the incisor gap, the distance between the upper and lower teeth during mouth opening, is another useful measure. An incisor gap less than 4-5 cm is defined as restricted, and there is relative risk of difficult intubation.[5]. In practical clinical use, 4-5 cm usually equates to the width of 2-3 fingers

It has also been shown that adequate preoxygenation in obese patients by providing eight deep breaths over a period of 60 s with 100% oxygen in the head-elevated position can increase the apnea tolerance by up to one minute longer than in patients preoxygenated in the supine position.[3] However, other characteristics of preoxygenation have to be taken into consideration, such as fresh gas flows of at least 10 L/min which reduces the amount of exhaled nitrogen being rebreathed, type of ventilation and the specific characteristics of the patients being preoxygenated.[6] Preoxygenation is key because it increases oxygen reserve in all compartments, arterial, venous, alveolar, and tissue. These patients should be induced the moment they are placed supine, if they are to be induced supine, since FRC drops quickly after induction and neuromuscular blockade.[7]

During attempted laryngoscopy, manipulation of the cricoid cartilage using pressure can help facilitate view of the vocal cords. There are two maneuvers commonly used for this, Sellick's maneuver and the BURP maneuver. Sellick's maneuver was designed to have the cervical spine extended with pressure on the cricoid cartilage in the classic "sniffing" position. It is classically used to occlude the esophagus for prevention of passive regurgitation of stomach contents during intubation.[8] Sometimes this can be useful, however Sellick's maneuver has been shown to sometimes distort the glottic view, which can become detrimental in patients at high risk for hypoxia and aspiration.[9] Studies examining Sellick's maneuver have shown conflicting results. The use of cricoid pressure alone can actually hamper results of laryngoscopy when used with excessive force.[8] The BURP maneuver consists of the backward, upward and right-sided pressure on the thyroid and cricoid cartilages and was first introduced in 1993 to improve glottis view during intubation. Research has since validated this maneuver to improve glottic view.[9]

In conclusion, several steps must be taken by the anesthesia professional to optimize the safe airway management of the obese patient. These steps begin in the preoperative state with the careful evaluation of the patient and making sure adequate equipment is available in case of emergency. This attention to safety must continue into induction utilizing adequate preoxygenation and techniques such as the BURP maneuver to have the most favorable conditions possible for laryngoscopy and intubation attempt. If appropriate measures are taken this ensures the safest induction possible for this vulnerable patient population.

References

1. Shore SA, Fredberg JJ. Obesity, smooth muscle and airway hyperresponsiveness. *J Allergy Clin Immunol*. 2005;115(5):925-7.
2. Lavi R, Segal D, Ziser A. Predicting difficult airways using the intubation difficult scale: a study comparing obese and non-obese patients. *J Clin Anesth*. 2009;21(4):264-7.
3. Eberhart LHJ, Arndt C, Cierpka T, Schwanekamp J, Wulf H, Putzke C. The reliability and validity of the upper lip bite test compared with the Mallampati classification to predict difficult laryngoscopy: an external prospective evaluation. *Anesth Analg*. 2005;101(1):284-9.
4. Khan ZH, Kashfi A, Ebrahimkhani E. A comparison of the upper lip bite test (a simple new technique) with modified Mallampati classification in predicting difficulty in endotracheal intubation: a prospective blind study. *Anesth Analg.* 2003 Feb;96(2):595-9.
5. Kovac AL. Evaluation of the airway. *Curr Rev Clin Anesth.* 2008;28(17):19.
6. Tang L, Huang S, Ma H, Wang Z. Desaturation following rapid sequence induction using succinylcholine vs. rocuronium in overweight patients. *Acta Anaesthesiol Scand*. 2011;55(2):203-8.
7. Altermatt FR. Pre-oxygenation in the obese patient: effects of position on tolerance to apnoea. *Br J Anaesth.* 2005;95(5):706-9.
8. Levitan RM, Kinkle WC, Levin WJ, Everett WW. Laryngeal view during laryngoscopy: a randomized trial comparing cricoid pressure, backward-upward-rightward pressure and bimanual laryngoscopy. *Ann Emerg Med.* 2006;47(6):548-55.
9. Snider DD, Clarke D, Finucane BT. The "BURP" maneuver worsens the glottic view when applied in combination with cricoid pressure. *Can J Anaesth.* 2005;52(1):100-4.

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Protamine Reaction in a Patient with Previous Vasectomy

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Keywords: Protamine Sulfate, allergic reaction, vasectomy

Protamine is a low molecular weight protein, rich in arginine, obtained from salmon sperm and the sperm of other fish.[1] Packaged as a sterile isotonic solution, protamine sulfate is administered intravenously for the neutralization of heparin anticoagulation. Protamine sulfate is alkaline and when combined with acidic heparin, it creates a stable salt reversing anticoagulant activity.[1] It is well documented that patients with a history of vasectomy possess increased risk for an adverse reaction to protamine sulfate.[1,2,3,4] This case report explores the management of an allergic reaction to protamine sulfate in a patient with a previous vasectomy.

Case Report

A 71 year old Caucasian male, with carotid artery stenosis > 50%, presented for a right carotid endarterectomy (CEA) to be performed with sedation and a cervical plexus block. The patient weighed 97 kg with a height of 180 cm, and reported no drug allergies. His history included dyslipidemia, peripheral vascular disease, hypertention, degenerative joint disease, and a 41 pack-year history of smoking with cessation 9 years prior. Past surgical history included right hip arthroplasty, bilateral knee arthroscopy and arthroplasy, and lumbar fusion. The patient did not report that he had a vasectomy during preoperative assessment. His medications included simvastatin, metoprolol, and lisinopril. Preoperative vital signs and laboratory test were within normal limits.

Preoperatively, the patient was given midazolam 1 mg intravenously (IV). Oxygen was administered via facemask and standard monitors were applied. The patient received a cervical plexus block with 30 ml of 0.75% ropivacaine without complications.

Once in the operating room, standard monitors were applied. As surgery commenced, midazolam 1 mg IV and fentanyl 50 mcg IV were used for patient sedation and pain management. The patient remained awake during the procedure with no changes in neurological assessment and minimal hemodynamic shifts. Heparin 5,000 units IV was administered prior to cross clamp per surgeon request. Surgery was nearing completion when protamine sulfate 35 mg IV was administered over 3 minutes. Approximately 2-5 minutes after protamine sulfate administration, the patient began to have stridor with dyspnea. Mild angioedema of the face and oropharyngeal tissues quickly followed. Systolic blood pressure decreased from 120-130 mm Hg to 80-90 mm Hg, SpO2 remained 92-96% on facemask oxygen, heart rate increased from 82 to 115-125 beats per minute (bpm). Upon questioning, the patient denied an allergy to fish or protamine, as well as, any previous exposure to protamine sulfate. The patient validated a history of a vasectomy.

Treatment included albuterol inhaler 2 puffs, diphenhydramine 25 mg IV and famotidine 20 mg IV. The patient's stridor improved, however dyspnea and angioedema persisted. Upon surgical wound closure, the patient was transferred to the stretcher with head of bed elevated to 45 degrees. The patient

transferred to the post anesthesia care unit, and a nebulized albuterol breathing treatment was initiated. Hydrocortisone 125 mg IV was administered with oxygen changed to humidified oxygen via facemask. Airway examination of oropharynx revealed engorged and erythematous tissue with gelatinous appearance. Patient symptoms improved over time with complete recovery to baseline in 2.5 hours.

Discussion

Adverse reactions to protamine sulfate have been described for years and are reported to occur at a rate varying from 0.06 to 10.6%.[2] Protamine is a non-human protein that can be antigenic, resulting in the production of specific antiprotamine immunoglobulin (IgM). Anaphylactic reactions to protamine require a previous exposure to protamine or protamine like agents to produce sensitization.[4] Mediation of anaphylactic reactions are through IgE and IgG antibodies.[3]

Anaphylaxis is a type I hypersensitivity reaction involving antigen cross-linking with specific IgE located on the surface of basophils or mast cells. This cross-linking produces degranulation of mast cells or basophils causing release of histamine, tryptase, carboxypeptidase A, and proteoglycans.[5] Arachidonic acid metabolites, kinins, and cytokines are synthesized and released due to cellular activation.[4] It is release of these mediators that produces cardiovascular (hypotension and arrhythmias), respiratory (bronchospasm and upper airway obstruction), cutaneous (urticaria and angioedema) and gastrointestinal (abdominal cramps, nausea, and vomiting) symptoms.[5] It is important to note that protamine may also activate the complement pathway and cause release of thromboxane, leading to bronchoconstriction and pulmonary hypertension.[6]

There is an increased risk of anaphylactic reactions to protamine sulfate in patients with past exposure to protamine sulfate, those who use protamine containing medications (such as insulin), patients allergic to fish, and male patients who have undergone a vasectomy. This procedure blocks ejaculatory pathways causing systemic reabsorption of sperm and can lead to antibody production. There is protamine in human sperm that can cause a cross reactivity, with 50-60% of vasectomized men producing spermatozoa antibodies and 22-30% developing human protamine antibodies.[3] When presented with a protamine reaction it is vital the anesthesia practitioner react quickly to provide treatment that could be life saving in the event of anaphylaxis.

Treatment of adverse responses to protamine is aimed at supporting the affected organs and reducing the effects of histamine release.[4] Respiratory changes were the first symptoms evident in our awake patient. Treatment consisted of oxygen therapy to maintain oxygen saturation, diphenhydramine and famotidine to mediate adverse effect of histamine, hydrocortisone to treat bronchospasm and hypotension, and albuterol to bronchodilate the airways.[4] The patient was able to maintain an oxygen saturation of 92%-96% without airway intervention. The anesthesia providers felt the erythematic and engorged appearance of the oropharynx increased the risk associated with obtaining an advanced airway. Due to these risks and the ability of the patient to maintain his oxygen saturation, the decision was made to medically manage him unless his respiratory status worsened.

The patient's systolic blood pressure started at 124 mm Hg with lowest measurement at 80 mm Hg. Fluid replacement with a normal saline bolus of 1000 ml was employed to treat mild hypotension and IV depletion. The patient's systolic blood pressure increased to >90 mm Hg with a mean arterial pressure >60 following the fluid bolus.

An additional treatment of subcutaneous epinephrine to relax bronchial smooth muscle was not utilized in this patient. This treatment was omitted at the time due to an increase in heart rate to 115 to 125 bpm. Upon re-evaluation of the event, epinephrine would have been a good therapeutic option despite the tachycardia. It is considered the drug of choice for anaphylaxis due to its ability to vasoconstrict, bronchodilate, and inhibit mediator release.[4]

When the protamine reaction began, the anesthesia professionals believed it to be of an anaphylactic nature. When the awake patient was asked about risk factors that could cause protamine anaphylaxis, he revealed he had undergone a vasectomy. Anesthesia professionals made the diagnosis of anaphylaxis based on clinically gathered information. A tryptase blood level could have been utilized to corroborate the anesthesia provider's diagnosis of anaphylaxis. Increase in tryptase blood levels would suggest an IgE mediated anaphylaxis due to degranulation of mast cells. Increases in tryptase levels are the preferred assay for detection of mast cell-mediated anaphylaxis.[7]

During the preoperative interview, the patient failed to inform the anesthesia practitioners of his previous vasectomy when queried about previous surgeries. In retrospect, when any patient is having a procedure where the use of protamine sulfate to reverse heparin is anticipated, perhaps the practitioner should directly ask, "have you ever had a vasectomy?" It is reasonable to accept that a patient may forget or not consider a vasectomy a surgical procedure. The knowledge of a previous vasectomy would heighten the practitioner's awareness that an anaphylactic reaction could occur upon administration of protamine sulfate, thus speeding recognition and initiation of therapy.

Protamine sulfate is the only medication currently approved by the Food and Drug Administration to reverse the anticoagulant effects of heparin.[6] It is imperative that anesthesia practitioners are aware of the potential for anaphylaxis with this medication. There is no evidence that pretreatment with corticosteroids and antihistamines reduce the severity of anaphylactic reactions; however, identifying a prior vasectomy as a risk factor will allow anesthesia providers to better prepare should a reaction occur.

References:

1. Pharmaceutical Partners of Canada Inc. Protamine Sulfate Drug Insert. PPC. http://www.ppcdrugs.com/en/products/product_inserts/EN_WebInsert_ProtaminSulf.pdf. Accessed October 16, 2011.
2. Nybo, M, Madsen JS. Serious anaphylactic reactions due to protamine sulfate: a systemic literature review. *Basic & Clinical Pharmacology & Tocicology*. 2008;103:192-196.
3. Collins C, O'Donnell A. Does an allergy to fish pre-empt an adverse protamine reaction? A case report and literature review. *Perfusion.* 2008;23:369-372.
4. Levy JH. Immune Function and Allergic Response. In Barash PG, Cullen BF,

Stoelting RK, Cahalan MK, Stock MC, eds.: *Clinical Anesthesia* 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009:256-270.
5. Nel L, Eren E. Peri-operative anaphylaxis. *British Journal of Clinical Pharmacology.* 2011;71:647-658.
6. Stoelting R, Dierdorf S. *Anesthesia and Co-Existing Disease.* 5th ed. Philadelphia: Churchill Livingstone; 2008:529.
7. Khodoun MV, Strait R, Armstrong L, Yanase N, Finkelman FD. Identification of markers that distinguish IgE- from IgG-mediated anaphylaxis. *PNAS.* 2011;108:12413-12418.

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Tetracaine Spinal for Large Ovarian Mass in Rwanda

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Keywords: tetracaine, spinal anesthesia, pelvic mass, global anesthesia, Rwanda

Anesthesia and surgery in a country with limited resources takes ingenuity and flexibility. Rwanda is a small, densely populated country that has only 0.02 doctors per 1000 people, according to the World Health Orangization.[1] Non-physician anesthetists independently perform almost all anesthetics. Anesthetic choice is influenced by resource and safety concerns making subarachnoid block (SAB) the technique of choice for many cases that would routinely be performed under general anesthesia or a regional-general combined technique in the US. The following case was conducted while on a global health rotation with the International Organization for Women and Development (IOWD) in Rwanda.

Case Report

A 45-year-old, 65 kg, 167cm non pregnant healthy female presented to district hospital in Kigali, Rwanda with a term 40-week pregnancy size pelvis. The pre-operative assessment was performed using a Kinyarwanda speaking translator. The patient reported having a large, non tender "mass" extending to her xyphoid for over three years. She had been denied surgery at three district hospitals in Rwanda. Past medical history was unremarkable with no prior abdominal surgeries and no known allergies.

The patient was brought to the operating suite, and EKG, non-invasive blood pressure (BP), and pulse oximeter monitors were applied. She was premedicated with diphenhydramine 50 mg via and 18 gauge peripheral intravenous (IV) catheter. The patient was placed in the sitting position on the operating room (OR) table, prepped, and draped in sterile fashion in preparation for spinal anesthesia administration. After identifying the proper landmarks, spinal anesthesia was performed at the L4-5 level, using a 25 gauge pencil point needle with 1.0% tetracaine 15 mg, epinephrine "wash", and fentanyl 10 mcg without difficulty. The patient was then placed in the supine position. The surgeons used 10 ml of 0.25% marcaine prior to pfannenstiel incision. The intraoperative period was uneventful, no pharmacological support or sedation was required. The surgeons were able to successfully drain 7,500cc of clear fluid

from the ovarian mass. The shell of the mass was dissected and removed along with the involved ovary. The incision was closed and an additional 20cc of 0.25% marcaine was injected around the incision. The patient remained comfortable throughout the procedure and the postoperative period. She received a total of 3,500 ml of crystalloid, as well as ketorolac 30 mg upon arrival in the post anesthesia care area. She began taking oral analgesics that evening. Her recovery was uneventful.

Discussion

Ovarian cysts are common in premenopausal women and cause a host of symptoms, most frequently pain, ranging from slight to debilitating. In the United States, ovarian cysts have been found in 14% of the postmenopausal population.[2] Ovarian cysts requiring surgery are typically removed via laparoscopic approach in the United States.

At our district hospital in Rwanda the laparoscopic approach was not an option. In a country with 10 million people, access to health care, surgical, and anesthesia services is limited and particularly difficult for a young woman living in a rural village. There are only 17 surgeons and 9 anesthesiologists practicing in the country, and most of these physicians practice at the larger referral hospitals.[3,4] General practice doctors and nurses staff district hospitals. Our patient had been denied surgery at several district hospitals due to the size and unknown composition of the mass. Lack of diagnostic imaging, experienced specialists, and specialized equipment present a challenge for surgeons and anesthetists.

Neuraxial anesthesia is routinely the first choice. Considerations when choosing which local anesthetic to use include length and type of surgery, as well as facility and provider capabilities. Tetracaine is an ester local anesthetic, which undergoes hydrolysis by pseudocholinesterase.[5] There have been many studies comparing local anesthetics for SAB. Tetracaine and bupivicaine have been compared in a number of these investigations. Tetracaine, although very similar to bupivicaine with regard to hemodynamic instability, shows significant increases in duration for sensory and motor return.[5,6] One study was performed evaluating orthopedic patients, looking at time until return of knee bend. The mean time for bupivicaine with epinephrine was 225.7 minutes, while in the tetracaine with epinephrine group it was 307.7 minutes.[5] This suggests to providers when the need for a prolonged block exists, tetracaine is the superior choice. In contrast, a comparison of tetracaine and bupivicane spinals for cesarean sections (c-sections) concluded that bupivicaine is the superior local anesthetic.[6] This study looked at analgesic supplementation during surgery. Twenty-two percent of patients with tetracaine and morphine spinal required additional analgesics during surgery while just 4.2% of the patients who had a spinal block with bupivicaine and morphine needed parenteral analgesic supplement. This suggests that bupivicaine provides a more intense visceral blockade than tetracaine.[6] This denser block coupled with the prolonged length of the tetracaine blockade, explains why most providers today choose bupivicaine over tetracaine for spinal anesthesia for procedures such as cesarean sections and knee arthroplasty.

Many factors were considered in selecting spinal anesthesia for this patient. Tetracaine was chosen as the agent, most importantly because of the unknown length of the anticipated surgery and the unavailability of large quantities of short acting sedation

agents. Without modern imaging, such as ultrasound and CT scan, it was difficult for the surgeons to be certain of exactly what they would encounter. An epinephrine wash was performed prior to the addition of the tetracaine to the syringe to prolong the block. For additional analgesic effect, fentanyl 10 mcg was added to the mixture. Using this combination of agent, vasoconstrictor, and narcotic allowed for maximum block duration. Secondly, tetracaine was chosen for its safe profile. Lidocaine has been shown to have an array of unwanted side effects including transient neurological symptoms, depressed respiratory drive, and depressed myocardial automaticity. In the case of unintentional intravascular injection, bupivicaine can cause severe cardiotoxicity. Tetracaine is less toxic. Additionally, the surgeons supplemented the neuraxial blockade analgesia with maximum infiltration of local bupivicaine.

With the availability of a long acting local anesthetic, the choice of neuraxial anesthesia over general was deemed the best option for this patient. General anesthesia for long cases at district hospitals in Rwanda presents several problems. First, the only inhalation agent available to anesthetists is halothane, which is not often used in the US because of its known risks, such as toxic hepatic necrosis, higher solubility, and metabolism[7]. Secondly, anesthesia machines in the district hospitals are not maintained and calibrated on a regular basis. Vaporizers are not regularly calibrated, therefore making an overdose or awareness a possibility.[3] In addition, no gas monitoring is available. Intravenous drugs for total intravenous techniques are in short supply, and there is no carbon dioxide absorption on the anesthesia machines which makes open system inhalation anesthesia the only choice for prolonged general anesthesia. Lastly, the patient was amendable to SAB and was informed through a native speaking interpreter that conversion to a general anesthetic was a possibility. An interpreter was available throughout the case to communicate with the awake patient. This made the choice of spinal anesthesia reasonable and appropriate for this case.

When working in countries with limited resources, anesthetists must become innovative and flexible with their anesthetic plans without compromising patient safety. Despite imperfect conditions, anesthesia can be safely administered by a variety of methods and the lives of those who may have otherwise not received the benefits of surgery can be improved.

References

1. World Health Organization. http://www.who.int/countries/rwa/en/ Updated 2009. Accessed September 17, 2011.
2. Greenlee R, Kessel B, Reding D, et al. Prevalence, incidence, and natural history of simple ovarian cysts among women > 55 years old in a large cancer screening trial. *Am J Obstet Gynecol*. n.d.;202(4) Accessed September 17, 2011
3. Enright, A. Anesthesia Training in Rwanda. *Can J Anaesth.* November 2007;54 (11):935-939
4. Notrica M, Evans F, Knowlton L, McQueen K. Rwandan Surgical and Anesthesia Infrastructure: A Survey of District Hospitals. *World J Surg* . n.d.;35(8):1770-1780. Accessed October 2, 2011
5. Leonard M, Moore L, Algozzine R, Gregorino J, Giles B. Recovery times from subarachnoid blocks using bupivacaine hydrochloride and tetracaine hydrochloride with and

without epinephrine. *AANA Journal*. June 1997;65(3):260-264. Accessed October 2, 2011
6. Nakagawa M, Kinouchi K, Miyagawa Y, Iura A, Shimizu T, Kitamura S. 7-year survey of anesthesia for cesarean section--comparison of tetracaine and bupivacaine as intrathecal anesthetic agents. Masui. *Jpn J Anesthesiol*. January 2007;56(1):61-68.. Accessed October 15, 2011
7. McKay,R., Inhaled Anesthetics, in Miller,R., Pardo, M. *Basics of Anesthesia*, *sixth ed*., Elsevier,2011:78-97.

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Sickle Cell Anemia with Hereditary Persistence of Fetal Hemoglobin

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Keywords: sickle cell disease, sickle cell anemia, hereditary persistence of fetal hemoglobin, HbS/HPFH

Sickle cell anemia is present in 0.3-1% of West African and American blacks.[1] It is one of the more common hemoglobinopathies, and is the most common genetic disorder in the United States.[2,3,4] The autosomal recessive trait results in abnormalities in hemoglobin chains.[1,2] Hemoglobin F (HbF) production is switched off at birth or soon after,[5] but the predominance of HbF continues to provide protection in the first few months of life.[6] Transition from HbF to adult hemoglobin (HbA) increases the production and percentage of sickle hemoglobin (HbS).[6] Persistent hereditary fetal hemoglobin (PHFH) results when HbF levels remain high in adults with sickle cell disease.[5]

Case Report

A 6 year old, 31kg male with a left inguinal hernia and Blount's disease presented for hernia repair and epiphyseodesis of bilateral tibias. Past medical history consisted of asthma, Blount's disease, sickle cell anemia with hereditary persistence of fetal hemoglobin, acute chest syndrome, and delayed development and speech. He had no allergies, surgical history, or daily medications and his immunizations were up to date.

Preoperative laboratory values included: Hb 12.4 g/dl, hematacrit (Hct) 37.6%, white blood cell count 10.3 x1000/mm^3, platelet count 281 x1000/mm^3, HbA estimate 2.5%, HbF 43.9%, and HbS 53.6%. Bone length scanogram about 1.5 months prior to surgery confirmed bilateral Blount's disease.

The patient was transported to the operating room with his father at his side and a pulse oximeter was applied. An under body warming blanket was placed on the bed prior to the patient entering the room and utilized during the case. He was anesthetized via inhalation induction with 8% sevoflurane and oxygen at 6L/min. Non-invasive blood pressure cuff, ECG leads and precordial stethoscope were applied. A 22 gauge IV was established in the right hand and a ringer's lactate infusion started at 50 ml/hr through a hot line fluid warmer. Rocuronium 20 mg IV was administered followed by a smooth, atraumatic intubation with a cuffed 5.0 mm oral endotracheal tube

(ETT) via direct laryngoscopy with a 1.5 Wis-Hipple blade. He was then placed on volume control ventilation and anesthesia was maintained with sevoflurane 2.4-3%.

An orogastric tube was placed and initially suctioned, then drained to gravity and removed prior to extubation. A warming blanket was set at 43°C, warm blankets were placed on top of the patient and the room temperature was raised. He remained normothermic, as indicated by the esophageal temperature probe. Postoperative nausea and vomiting prophylaxis included dexamethasone 4mg IV after induction, and ondansetron 4mg IV at the end of the case. Cefazolin 750 mg and morphine 2 mg was administered prior to incision, with additional morphine titrated during the case for a total of 4.5 mg. A Foley catheter was inserted.

The first surgery, inguinal hernia repair, proceeded uneventfully, followed by the left and right epiphyseodesis. Pressure support ventilation was provided once the patient began making respiratory effort with a backup respiratory rate of 14 breaths per minute. The oropharynx was suctioned prior to extubation. The ETT was removed once the patient was awake and protective reflexes intact. Supplemental oxygen via non rebreather mask was administered after removal of the ETT. Total surgical time was 4 hours and 2 minutes. He received a total of 1.4 L ringer's lactate solution. Urine output was measured at 205 ml and estimated blood loss at 35 ml. His recovery was uneventful and he was transferred to the floor on supplemental oxygen.

Discussion

It is important to understand the mechanism of sickle cell disease when planning a safe anesthetic for patients with that diagnosis. It is a combination of erythrocyte "sickling" and "sticking" that lead to this acute disruption of vascular dysfunction.[7] Patients with sickle cell disease have a high incidence of perioperative complications. Though minor procedures such as inguinal hernia repair and extremity surgery are considered low risk, it is important to consider the intrinsic risk of the type of surgery along with other factors such as age, frequency of hospitalizations, presence of organ damage, history of central nervous system events, and concurrent infection.[8]

Sickle cell disease is characterized by the presence of HbS, which is an inherited consequence of a substitution of the amino acid valine for the glutamic acid at one point in each of two beta chains.[1,6] Exposure of this molecule to low levels of oxygen causes formation of elongated crystals inside the red blood cells (RBCs) making it almost impossible for the cells to pass through small capillaries.[1] Precipitated HbS damages the RBC membranes, making them fragile and distorted. When the hydrophobic regions aggregate, it results in oxidative damage to the membrane, impaired deformability and shortened cell life.[8] Low oxygen tension causes sickling, which leads to RBC rupture, causing further decrease in oxygen tension and still more sickling and red cell destruction. This vicious cycle of events is known as sickle cells crisis, and once it starts progresses rapidly to a state of critical decrease in RBCs within a few hours, and often death.[1]

Persistent HbF appears to be a built-in protective mechanism. Lab studies demonstrated interference of polymerization of deoxygenated HbS by HbF.[7] Elevated HbF levels in sicklers has been observed as a protective mechanism, decreasing the incidence and severity of crises.[5] HbF allows for a higher level of oxygen carrying

capacity. This is understood by discussing P50, which describes the affinity of a given Hb for oxygen, and is defined as the PO_2 level that hemoglobin becomes 50% saturated with oxygen. There is an inverse relationship between P50 and PO_2 levels, that is, as the P50 decreases, oxygen affinity increases. Compare HbA (P50 26-27 mm Hg) with that of HbF, which has a higher affinity for oxygen (P50 value 20 mm Hg), and HbS has a tendency to release oxygen (P50 value 34 mm Hg).[9] One therapeutic strategy at increasing oxygen carrying capacity is red blood cell transfusion which can theoretically be avoided in patients with PHFH, who have a "natural" enhanced ability to carry oxygen.

Preoperative transfusion management goals have changed in recent years owing to studies that revealed no benefit to aggressive transfusion strategies, which aimed at increasing the ratio of normal Hb to HbS. A more conservative goal of achieving a preoperative Hct of 30% is generally recommended, recognizing that complications related to blood transfusions outweigh their benefit in this particular population, as the aggressive strategy necessitated significantly more transfusions.[6,8] Low-risk procedures rarely require any preoperative transfusion.[8] Patients undergoing moderate- to high-risk operations, intra-abdominal, intracranial or intrathoracic procedures may require a correction of preoperative anemia to a target Hct of 30%.[8]

Anesthetic technique choice is a controversial topic. A previous study revealed a higher rate of sickle cell disease specific complications with regional versus general anesthesia in a review of 1,079 anesthetics.[6] This study, however, did not control for the effects of obstetric cases, commonly utilizing regional techniques, and the impact obstetric interventions has on sickle cell risks alone.[6] Hines and Marschall[8] state that anesthetic technique does not seem to impact the complication risks associated with sickle cell disease. They did note that a regional technique might provide better pain relief than intravenous analgesics, preventing the vasoconstriction associated with pain crises.[8]

The goals of our anesthetic focused on avoiding dehydration, acidosis, hypothermia, and hypoxia, theoretically reducing the risk of perioperative sickling events. Dehydration may cause perioperative complications if we assume that hemoglobin concentration and sickling occur in the presence of intracellular dehydration.[6] We aimed to maintain euvolemia, though clinical evidence confirming this assumed causal relationship is lacking. Hypothermia causes a left shift in the oxyhemoglobin dissociation curve and is often identified as a precipitating event of perioperative sickle cell complications.[1,6] Warming methods were utilized in attempt to maintain normothermia, a basic objective in the general patient population, and basis of care for patients with sickle cell disease. Acidosis in the presence of hypoxia hastens erythrocyte deformation, is widely suspected as a trigger to sickle cell disease complications and should be avoided in the perioperative period.[6] Supplemental oxygen should be provided during and after surgery to maintain oxygen saturations above 95%, or that above the child's baseline.[3]

Though occlusive orthopedic tourniquets are not absolutely contraindicated, use of them for the bilateral epiphyseodesis was avoided. Morphine was titrated during the case to prevent postoperative pain, as pain at the operative site and that due to vasoocclusive events can exacerbate disease complications.

[8] This patient also had a history of acute chest syndrome (ACS), a pneumonia-like complication characterized by a new pulmonary infiltrate involving at lease one complete lung segment and accompanied by at least one of the following: chest pain, fever > 38.5°C, tachypnea, wheezing, or cough. ACS may develop 2-3 days postoperatively and demands aggressive focus on oxygenation, adequate analgesia and frequent blood transfusion to correct anemia and improve oxygenation.[4,8]

This case serves to review the anesthetic implications for patients with sickle cell disease. Anesthesia practitioners may find themselves in a fast paced environment that does not allow much time for research prior to starting their case. It is essential to be able to individualize the anesthetic and to plan with an understanding of the hemoglobinopathy.

References

1. Guyton AC, Hall JE. *Textbook of Medical Physiology*. 11th ed. Philadelphia:Elsevier Inc.; 2006:421-427.
2. Anim SO, Strunk RC, DeBaun MR. Asthma morbidity and treatment in children with sickle cell disease. *Expert Rev of Respir Med.* 2011;October:635-652.
3. Cartwright R, Howard J, Rawle H, Thomas VJ, Westerdale N. Quality of life and pain management in sickle-cell disease. *Ped Health*. 2008;June:377-397.
4. Pack-Mabien A, Haynes J. A primary care provider's guide to preventive and acute care management of adults and children with sickle cell disease. *J Am Acad Nurse Pract*. 2009;21:250-257.
5. Edoh D, Antwi-Bosaiko C, Amuzu D. Fetal hemoglobin during infancy and in sickle cell adults. *Afr Health Sci.* 2006;6:51-54.
6. Kim, TW. Perioperative care of the patient with sickle cell disease. *ASA Refresher Courses in Anesthesiology.* 2008;36:59-74.
7. Frenette PS, Atweh GF. Sickle cell disease: old discoveries, new concepts, and future promise. *J Clin Invest*. 2007:117:850-859.
8. Rinder CS. Hematologic Disorders. In Hines RL, Marschall KE, eds. *Stoelting's Anesthesia and Co-existing Disease*. 5th ed. Philadelphia:Churchill Livingstone; 2008:411-412.
9. Elton D. Oxygen – Hemoglobin affinity. *Lexington Pulmonary and Critical Care.* 2011. http://www.lexingtonpulmonary.com/edu/oxyhb/oxyhb.html. Accessed October 29, 2011.

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Pain Management in a Patient Undergoing a Tonsillectomy

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Keywords: tonsillectomy, pain management, nonsteroidal anti-inflammatory medication, postoperative bleeding, postoperative nausea and vomiting

Tonsillectomy is the second most common surgery performed on pediatric patients in the United States. Patients undergoing tonsillectomy experience moderate to severe pain postoperatively, and are at high risk for

postoperative nausea and vomiting (PONV).[1] Effective pain management is essential to prevent poor outcomes, yet studies document that after tonsillectomy, pain control is not always adequate.[1,2] Pain management post-tonsillectomy commonly involves opioids, acetaminophen, and non-steroidal anti-inflammatory drugs (NSAIDs). [3,4]

Case Report

A 16 year old, 75kg, 157cm, female with a diagnosis of recurrent tonsillitis presented for bilateral tonsillectomy. The patient had approximately eight episodes of tonsillitis over the past year. The patient had no other significant past medical history. Recent home medications included acetaminophen as needed for pain and fever, and a multivitamin daily. Her physical exam was remarkable for grade three tonsillar hypertrophy. Her preoperative heart rate was 78 beats per minute (bpm), respirations 12 breaths/min, and blood pressure 108/68 mm Hg.
The patient was administered midazolam 2 mg intravenously (IV) in the preoperative area, and then transported to the operating room.

In the operating room, a pulse oximeter, blood pressure cuff, 5-lead electrocardiogram, and skin temperature probe were placed. The patient was preoxygenated with oxygen, 10 liters/minute (l/min), via face mask prior to induction. IV induction was performed with fentanyl 100 micrograms (mcg), lidocaine 70 mg, propofol 150 mg, and rocuronium 40 mg. Direct laryngoscopy using a Macintosh 3 blade was performed and a grade 1 view of the airway was noted. A 7.0 endotracheal tube was inserted into the trachea without difficulty. Immediately after intubation, dexamethasone 12 mg IV, and ondansetron 4 mg IV were administered. Anesthesia was maintained with desflurane 6-7 percent, oxygen 1 L/min, and air 1 L/min.

Prior to incision, the surgeon inserted a throat pack into the pharynx, and injected lidocaine 100 mg into the peritonsillar mucosa. Morphine 8 mg IV was titrated for analgesia throughout the case. After the procedure, the surgeon removed the throat pack, and used an orogastric tube to empty the patient's stomach contents. The neuromuscular blockade was antagonized with glycopyrrolate 0.6 mg and neostigmine 3 mg. A total of 600 mL of lactated ringers was administered, and there was 15 mL of blood loss. The trachea was extubated without incident.

After arriving in the postanesthesia care unit (PACU) the patient complained of throat pain, 8 out of 10 on a numeric scale, with 10 being the most severe pain rating, and 0 no pain. Her heart rate was 98 bpm, respiratory rate was 24 breaths per minute, and blood pressure was 130/79 mmHg. The patient was given hydromorphone 3 mg IV in divided doses over 10 minutes. The patient's pain rating decreased to 4 out of 10. After 45 minutes in the PACU, the patient complained of nausea and worsening throat pain. Her PACU stay was extended 1.5 hours past the expected length of stay, due to her need for additional IV narcotics, and antiemetics.

Discussion

After a tonsillectomy, patients have moderate to severe pain, and are at high risk for PONV and dehydration. [5] Significant pain and PONV can lead to a negative anesthetic experience, delayed discharge from the PACU, or an unplanned admission.[1]

Tonsillectomy surgeries are being performed on an outpatient basis. To prevent delays in discharging patients after outpatient surgery, they must have satisfactory pain control that minimizes adverse side effects. Opioids provide potent analgesia by binding to receptors in the central nervous system, inhibiting ascending pain pathways, and causing central nervous system depression. Opioids are also associated with undesirable effects such as nausea, vomiting, sedation, and respiratory depression. [2] NSAIDs are a potential adjunct therapy for the treatment of severe pain. NSAIDs work by reversibly inhibiting the cyclooxygenase-1 and 2 enzymes, thereby decreasing the production of prostaglandin. By decreasing prostaglandin production, NSAIDs cause a decrease in pain signal transmission, and have an anti-inflammatory effect. NSAIDs relieve moderate pain, have anti-inflammatory properties, and do not have the adverse central nervous system respiratory effects of opioids.[5]

The use of NSAIDs in tonsillectomy surgery may lessen the need for opioids, thereby reducing their potentially adverse effects. NSAID use postoperatively has been shown to successfully manage moderate pain, and decrease the incidence of postoperative nausea and vomiting. The use of NSAIDs is associated with patients tolerating oral intake earlier and discharge earlier, than patients who are managed with opioids alone.[4] According to studies, patients experience a significant improvement in pain control when NSAIDs are used in addition to opioids. When NSAIDs are used alone after a tonsillectomy they are shown to have equal pain control efficacy as opioids, but a significant decrease in the incidence of PONV.[6] However, there is concern that NSAIDs contribute to rebleeding and hemorrhage as they inhibit platelet aggregation. The actual clinical effect of NSAIDs on bleeding in the postoperative period is not clear.[5]

Hemorrhage after tonsillectomy is a relatively rare, but serious, complication. Approximately 2 to 3 percent of patients experience hemorrhage during or after tonsillectomy surgery.[3] The rate of reoperation for hemorrhage in patients who did not receive NSAIDs was reported to be approximately 0.3 to 1.9 percent, whereas patients receiving NSAIDs had an incidence of approximately 2 percent. [6] However, the literature on NSAID use and hemorrhage is controversial, as many studies incorporated in the reviews lack the power necessary to draw statistically significant conclusions.[6] According to a recent large-scale systematic review, there was no evidence of NSAIDs causing a statistically significant increase in bleeding that required surgical intervention after tonsillectomy.[4]

In the case described, the patient was given only opioids for analgesia. The patient experienced significant post-operative pain and nausea, despite prophylactic administration of ondansetron and dexamethasone. The patient was unable to tolerate oral intake and her discharge was delayed. The use of a NSAID, such as ketorolac 30 mg IV, administered at the conclusion of the procedure, may have improved the patient's pain level. In addition, it may have decreased her need for opioid analgesia, thereby potentially decreasing her postoperative nausea and preventing her extended stay in PACU. The literature does not conclusively state that administering a NSAID can significantly increase the risk of post-tonsillectomy hemorrhage.[4] However, even if the use of a NSAID potentially causes a slight increase in the risk of hemorrhage, the benefits of an additive analgesic effect and decreased adverse effects of opioid administration may

outweigh the risk.[6] In cases of tonsillectomy the risk benefit ratio between the use of opioids or the use of opioids in combination with NSAIDs should be evaluated on a case by case basis to provide optimal patient safety and pain management.

References

1. Hadden SM, Burke CN, Skotcher S, Voepel-Lewis T. Early postoperative outcomes in children after adenotonsillectomy. *J Perianesth Nurse.* 2011;26:89-95.
2. Hamunena K, Kontinenb V. Systematic review on analgesics given for pain following tonsillectomy in children. *Pain.* 2005;117:40-50.
3. Jevakumar A, Brickman TM, Williamson ME, et al. Nonsteroidal anti-inflammatory drugs and postoperative bleeding following adenotonsillectomy in pediatric patients. *Arch Otolaryngol Head Neck Surg*. 2008;134:24-27.
4. Cardwell ME, Siviter G, Smith AF. Nonsteroidal anti-inflammatory drugs and perioperative bleeding in the paediatric tonsillectomy. *Cochrane Database Syst Rev.* 2010;2.doi: 10.1002/14651858.
5. Krishna S, Hughes LF, Lin SY. Postoperative hemorrhage with nonsteroidal anti-inflammatory drug use after tonsillectomy: a meta-analysis. *Arch Otolaryngol Head Neck Surg.* 2003;129:1086-1089.
6. Moiniche S, Romsing J, Dahl JB, Tramer MR. Nonsteroidal antiinflammatory drugs and the risk of operative site bleeding after tonsillectomy: a quantitative systematic review. *Anesth Analg.* 2003;96:68-77.

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Magnesium Sulfate in the Management of Pheochromocytoma

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Keywords: pheochromocytoma, magnesium sulfate, anesthesia, adrenal tumor, hemodynamics

Pheochromocytoma is a rare, catecholamine-producing tumor that presents with paroxysmal manifestations of hypertension, headache, and diaphoresis. They are often referred to as "10% tumors" because 10% of the tumors are bilateral, extraadrenal, familial, malignant, and occur in the pediatric population.[1] The incidence rate is 1 to 2 per 100,000 adults annually while the prevalence rate in the hypertensive population range from 0.1 to 0.6%. [2] It is associated with increased morbidity and mortality if left untreated. The most definitive measure to cure pheochromocytoma is surgical resection. The use of magnesium sulfate may help stabilize hemodynamics through catecholamine release inhibition.

Case Report

A 50 year old, 85 kg female patient was scheduled for a laparoscopic adrenalectomy of left adrenal tumor on CT scan. An elevated serum metanephrine confirmed the diagnosis of pheochromocytoma. Her signs and symptoms included headaches, hypertension, lightheadedness, and flushing.

She has no known drug or food allergies. Her past medical history includes hypertension, hypercholesteremia, gastroesophageal reflux disease, goiter (with normal thyroid function tests), and diabetes mellitus (type 2). Her past surgical history includes cesarean section, cholecystectomy, hysterectomy, and left heart cardiac catheterization.
Her preoperative vital signs were the following: temperature 36.7° Celsius, heart rate 90, blood pressure 135/85, respiratory rate of 14, SpO_2 99%. Her preoperative laboratory values were unremarkable and a type and screen for transfusion was completed. Her electrocardiogram showed normal sinus rhythm. Her current medications included phenoxybenzamine, metoprolol, simvastatin, pantoprazole, and metformin. She took all her medications the day of surgery except for metformin, which was stopped 3 days prior to surgery.

In the preoperative area, the patient was evaluated for surgery. A general anesthetic with intravenous induction was planned. An 18G IV catheter was started. Midazolam 2 mg IV was administered and magnesium sulfate 35 mg/kg IV infusion over one hour.

In the operating room, IV induction was achieved with fentanyl 100 mcg, propofol 250 mg, rocuronium 50 mg after preoxygenation. The trachea was intubated and respiration was controlled by a mechanical ventilator. An arterial line and central venous catheter were placed to monitor hemodynamics and provide IV access. Maintenance of anesthesia was done with oxygen 1L/min and air 1L/min and isoflurane 0.6-1.5%. Additional magnesium sulfate 2g/hr IV was administered. A nitroprusside infusion (0.3-2 mcg/kg/min) was titrated to keep systolic blood pressures (SBP) less than 140 mm Hg and mean arterial pressures (MAP) less than 90 during tumor devascularization. After tumor resection, a phenylephrine infusion (20-40 mcg/min) was titrated to keep SBP greater than 90 mm Hg and MAP greater than 70 mm Hg.
Prior to emergence, the patient was given hydromorphone 1 mg IV for analgesia.

Neuromuscular blockade was antagonized with neostigmine 3 mg IV and glycopyrrolate 0.6 mg IV after return of train of four ratio (TOF ratio) of at least 2 out of 4 twitches. Ondansetron 4 mg IV was given for antiemetic. Isoflurane and air were turned off and fresh gas flow of O_2 increased to 10 L/min. After meeting extubation criteria of adequate minute ventilation and return of TOF ratio to greater than 0.9, patient extubated and oxygen administered by non-rebreather face mask. The patient was transferred to the post anesthesia care unit (PACU). Initial vital signs were stable. Patient had an uneventful recovery and was discharged to home with nursing services on postoperative day 2.

Discussion

The literature on the use of magnesium in the anesthetic management of pheochromocytoma is limited to case reports and small scale studies. Because of the low incidence rate of pheochromocytoma, prospective randomized clinical trials (RCT) are difficult to achieve. A few case reports and RCTs will be discussed to evaluate the safety and efficacy of magnesium.
Magnesium can help maintain hemodynamic stability by inhibiting the release of catecholamines during induction, tumor manipulation, creation of pneumoperitoneum, and tumor resection.[3] Magnesium inhibits release of catecholamines from the adrenal medulla and peripheral nerve endings, directly antagonizes peripheral adrenergic receptors

leading to vasodilatation, and antagonizes L-type calcium channels leading to antiarrhythmic properties.

From a historical perspective, James published a paper in 1989 demonstrating the effectiveness of magnesium in 17 patients with pheochromocytoma.[4] Recently in 2004, James and Cronje described the potential benefits of magnesium when nitroprusside and phentolamine failed to achieve stable hemodynamics in 3 patients.[5] These authors concluded that magnesium provided adequate hemodynamic control including during the period when the tumors were resected.

The preoperative medical management of pheochromocytoma has become standardized.[6] It involves first administering α receptor antagonists to achieve α blockade, then if necessary due to tachycardia or cardiac arrhythmias, β receptor antagonists are administered. It is important to achieve adequate α blockade before β blockers are introduced because β blockade may worsen epinephrine-induced vasocontriction through inhibition of β adrenergic, vasodilator properties. Two commonly used α receptor antagonists for the preoperative treatment of pheochromocytoma are phenoxybenzamine (a non-selective $\alpha 1$ and $\alpha 2$ receptor antagonist) and $\alpha 1$ specific antagonists such as terazosin and prazosin. Common β receptors blockers include propranolol, atenolol, metoprolol, and labetalol. Calcium channel blockers have also been used to treat pheochromocytoma in the preoperative period. In this case report, the patient received phenoxybenzamine 2 weeks prior to surgery then metoprolol was added only after adequate α blockade.

Magnesium can help attenuate the sympathetic response due to direct laryngoscopy and tracheal intubation.[7] In this RCT study of 200 randomized patients, magnesium, given at 10 to 20 mg/kg prior to intubation, minimized increases in blood pressure but not heart rate. In this case report, direct laryngoscopy was performed easily and no significant changes to blood pressure were observed but the heart rate did increase from 65 to 90 beats/min.

Magnesium significantly attenuated the arterial pressure increase before pneumoperitoneum during laparoscopic cholecystectomy in 32 randomized patients.[8] This RCT also showed that plasma levels of catecholamines and vasopressin were increased significantly in the control group but not in the magnesium group. With regards to the patient mentioned in this case report, there were no significant increase in the arterial blood pressure noted when pneumoperitoneum was established.

Magnesium was not the sole vasoactive therapy utilized during this case and this is consistent with some of the literature.[4-6,9] During manipulation, devascularization, and eventual resection of the tumor in this case report, significant changes in hemodynamics were seen in a five minute period. There was an increase in arterial blood pressure from 120/70 to 180/90 mm Hg with increases in heart rate from 72 to 110 beats/min. Esmolol 20 mg IV was administered and nitroprusside infusion was started to stabilize hemodynamics. Bryskin and Weldon suggested that it is possible to avoid the use of other vasoactive agents i.e. esmolol and nicardipine if the loading dose and subsequent infusion of magnesium were increased.[9] James and his study of 17 cases suggested a loading dose of magnesium 40-60 mg/kg followed by a continuous infusion of 2g/h.[4] Only 4 patients required additional nitroprusside infusion. In this case report, the patient received a lower loading dose

(35mg/kg) while maintained on 2g/hr. It is possible that with larger loading dose, the need for esmolol and nitroprusside during this case would either be decreased or unnecessary.

There are potential side effects of magnesium that clinicians must consider.[3] Magnesium can cause pain on injection, which can be mitigated by decreasing the rate of infusion. It can enhance neuromuscular blocking drugs and prolong paralysis. It can potentiate calcium channel blockers, resulting in hypotension. During this case, pain on injection was not observed with the loading dose of magnesium. Acceleromyography was monitored throughout the case and train of four ratio greater than 0.90 was achieved after administration of neostigmine with glycopyrrolate. This patient was extubated after meeting criteria. Hypotension is commonly seen after adrenal tumor resection and a phenylephrine infusion was titrated to keep SBPs greater than 90 mm Hg and MAPs greater than 70 mm Hg. This infusion was discontinued prior to this patient's transfer from the PACU to the surgical floor.

In summary, this case report illustrates the role of magnesium in the anesthetic management of a patient undergoing laparoscopic left adrenalectomy of an adrenal tumor. Magnesium can help attenuate the catecholamine release encountered within the intraoperative period. Magnesium is a safe, inexpensive, and simple yet effective option in the management of pheochromocytoma.

References

1. Guller U, Turek J, Eubanks S, DeLong ER, Oertli D, Feldman JM. Detecting pheochromocytoma: defining the most sensitive test. *Ann Sur*. 2006;243(1):103-107.
2. Bhansali A, Behera A, Singh SK, et al. Pheochromocytoma: a study of 95 patients. *The Endocrinologist*. 2009;19(1):19-23.
3. Herroder S, Schonherr ME, De Hert SG, Hollmann MW. Magnesium- essentials for anesthesiologists. *Anesthesiology*. 2011;114(4):971-993.
4. James MF. Use of magnesium sulphate in the anaesthetic management of phaechromocytoma: a review of 17 anaesthetics. *Brit J Anaesth*. 1989;62:616-623.
5. James MF, Cronje L. Pheochromocytoma crisis: the use of magnesium sulfate. *Anesth & Analg*. 2004;99:680-686.
6. Lentschener C, Gaujoux S, Tesniere A, Dousset B. Point of controversy: perioperative care of patients undergoing pheochromocytoma removal- time for reappraisal? *Eur J Endocrinol*. 2011;165:365-373.
7. Shin YH, Choi SJ, Jeong HY, Kim MH. Evaluation of dose effects of magnesium sulfate on rocuronium injection pain and hemodynamic changes by laryngoscopy and endotracheal intubation. *Korean J Anesthesiol*. 2011;60(5):329-333.
8. Jee D, Lee D, Yun S, Lee C. Magnesium sulphate attenuates arterial pressure increase during laparoscopic cholecystectomy. *Brit J Anaesth*. 2009;103(4):484-489.
9. Bryskin B, Weldon BC. Dexmedetomidine and magnesium sulfate in the perioperativemanagement of a child undergoing laparoscopic resection of bilateral pheochromocytomas. *J Clin Anesth*. 2010;22:126-129.

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Causes of Failed Neuraxial Anesthetic

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Keywords: regional anesthesia, failed block, neuraxial, epidural anesthesia, subdural injection

Regional anesthesia may offer potential advantages over general anesthesia. In addition to being less costly, regional anesthesia can offer post-operative pain relief and less residual sedation. Numerous factors may make a regional anesthetic ineffective. Just as important as understanding the technique of performing the procedure, the anesthetist must also be familiar with causes of a failed block and have a plan for management of the patient in whom a regional anesthetic fails.

Case Report

A 61 year old male, 188 cm tall and 80 kg, presented for a transurethral resection of the prostate (TURP). His past medical history was significant for hyperlipidemia, gastro-esophageal reflux disease and chronic prostatitis. The patient consented to both regional and general anesthesia, understanding that general anesthesia would only be employed in the event of a failed or insufficient regional block.

At 0720, the patient was transported to the operating room and positioned sitting for subarachnoid block (SAB) placement. Immediately prior to positioning, the patient was given Midazolam 4 mg IV. Assessment revealed significant lordosis of the lumbar spine. A mid-line approach with 25 g Whitacre needle was attempted at the L3-L4 level. The anesthesiologist was unable to locate the subarachnoid space. A paramedian approach was then attempted at the L2-L3 level without success. At 0730 a SAB was placed in the L2-3 level using a mid-line approach with clear cerebrospinal fluid (CSF) noted. No parasthesias were elicited, and 0.75% bupivacaine 1.5 mL was injected. The patient was then positioned supine.

At 0740, the block was assessed using a blunt tip needle. The patient did not report sensation below the dermatome level of L2-L3, and surgical preparation ensued. Upon commencement of the procedure at 0742, the patient reported significant discomfort upon insertion of the urethral dilator. The decision to convert to general anesthesia was made. At 0746, a “modified” rapid sequence induction was performed utilizing fentanyl 150 mcg, lidocaine 100 mg, propofol 200 mg and rocuronium 30 mg IV. Laryngoscopy and intubation were completed without complication.

Following induction and intubation, the TURP procedure was completed under general anesthesia with sevoflurane 2%. Following completion of the procedure, neuromuscular blockade was antagonized, the patient regained spontaneous ventilation, and the endotracheal tube was removed without complication before transfer to PACU.

Discussion

In successful neuraxial anesthetics, pain transmission and sympathetic nervous response are significantly dampened, if not altogether blocked. Placement of local anesthetics with or without adjunct medications in the subarachnoid or epidural

space opposes neural signaling to varying degrees, depending on factors such as concentration and volume of medication utilized. In addition to unforeseen surgical complications, abandoning a neuraxial anesthetic may be required due to improper placement of anesthetic, inadequate depth or duration of blockade, and improper selection of technique or medication.[1]

Improper placement occurs when medication is deposited into an anatomical location other than the epidural or subarachnoid space. A false loss-of-resistance can be encountered within the subcutaneous tissue, and is likely the most common cause of complete epidural failure.[2] The incidence of complication or failed blockade can be associated with increasing patient body-mass-index, extremes of age, and lack of clinical experience by the anesthetic practitioner.[1] Of note, failed subarachnoid block due to improper placement occurs at a lower rate in combined spinal epidural (CSE) technique.3 In between the epidural and subarachnoid space lies the anatomical space known as the subdural space. Despite the effectiveness of neuraxial anesthetic in both the epidural and subarachnoid space, subdural placement of local anesthetic frequently manifests as incomplete and/or problematic blockade. Clinical findings such as an absence of CSF and blood on aspiration, extensive sympathectomy, negative test dose and widespread, patchy block are characteristic of subdural injection.[4]

While improper placement plays a prominent role in instances of failed neuraxial blockade, medication injection into the desired location can still lead to an inadequate block. In a majority of cases requiring conversion from neuraxial block to general anesthesia, failed and/or insufficient onset and duration of block were noted as contributory.[1] Improper medication choice (delayed onset, short duration) and unpredictably fast or extended labor are significant culprits of inadequate neuraxial technique in the laboring patient.[2] Despite the valuable pain control elicited by proper neuraxial block, the nurse anesthetist must take care to match the duration and onset of a particular drug with the clinical situation. Furthermore, in epidural blockade, varying concentrations and volumes can be utilized to manipulate the depth and extent of blockade. Use of insufficient volume or concentration can result in an inadequate block. Additionally, the distance of catheter insertion is also related to adequacy of epidural block.[2] Catheter insertion of less than 3 cm. is associated with epidural failure, while insertion greater than 5 cm. is linked to intravenous placement or unilateral blockade.[4]

Despite the usefulness of neuraxial technique for pain management, care must be taken to ensure both the patient and procedure are appropriately matched with the desired technique. Conditions such as severe aortic stenosis, or procedures requiring muscle paralysis exclude spinal or epidural as a viable lone anesthetic. Although not always absolute contraindications, problematic anatomy, such as excessive subcutaneous tissue and altered spinal anatomy (stenosis, lumbar lordosis, scoliosis, etc.) are frequent causes of ineffective epidural placement.[2] Additionally, deciding between an epidural and spinal technique requires careful consideration by the anesthetist. Although neither adequate nor ideal for all procedures completed under neuraxial technique, subarachnoid anesthesia is associated with a lower risk of complications and is more commonly efficacious than the epidural route of administration.[5] Moreover, the loss of resistance technique utilized in the

epidural anesthetic may not be as easily perceived by the novice practitioner.[4]

In addition to recognizing common causes of neuraxial failure, it is important to consider new ways of improving clinical practice. Ultrasound is being examined as an adjunct to improve examination of patient anatomy in neuraxial anesthesia. While ultrasonography is utilized in many institutions to visualize nerve bundles during the placement of non-neuraxial regional anesthetics, recent studies examining its usefulness in neuraxial techniques have shown promise. Data regarding the efficacy of ultrasonography as an aid in placement of neuraxial anesthesia is somewhat limited, and current findings point toward ultrasonography as a helpful addition to the traditional examination performed before needle placement.[6]

References

1. Guglielmo, L., Pignataro, A., DiFiore, G., Lanza, V., Mercadante S. Conversion of spinal anesthesia into general anesthesia: An evaluation of more than 35,000 spinal anesthetics. *Minerva Anestesiol.* 2010;76:688-689.
2. Arendt, K., Segal, S. Why epidurals do not always work. *Rev Obstet Gynecol.*2008; 2:49-55.
3. Pan, P., Bogard, T., Owen, M. Incidence and characteristics of failures in obstetric neuraxial analgesia and anesthesia: a retrospective analysis of 19,259 deliveries. *Int'l J Obstet Anesth.* 2004; 13(4):227-233.
4. Nagelhout, J., Plaus, K. *Nurse Anesthesia*. 4th ed. St, Louis: Saunders Elsevier: 2010:1065-1072.
5. McIntyre, P., Deer, T., Hayek, S. Complications of spinal infusion therapies. *Tech Reg Anesth Pain Mgmt* 2007; 11: 183-192.
6. Perlas, A. Evidence for the use of ultrasound in neuraxial blocks. *Reg Anesth Pain Med* 2010; 35: 43-46.

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Anesthesia and Triple X Syndrome

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Keywords: triple X syndrome, 47 XXX, sex chromosomal disorder, difficult airway, anesthesia for inherited chromosome anomaly

One in every 1,000 females born has an extra X chromosome and has the genetic designation of Triple X, or XXX, Syndrome. However, many of these infants grow up undiagnosed.[1] Triple X Syndrome was first documented in 1959, with girls displaying early growth patterns, especially in years 4-8, with notably longer legs.[1] Triple X Syndrome females tend to have delayed language and motor skill development and difficulty forming interpersonal relationships.[1] A lower IQ than siblings is reported by some authors.[2] Reference to smaller brain size and epilepsy are also documented,[3] as well as mandibular prognathism or retrognathism.[1] The condition, however, is often over-looked unless discovered during chromosomal testing secondary to language delay.[2]

Case Report

A 16 year-old, 72kg, 167 cm female presented for umbilical hernia repair (UHR). This patient had a positive diagnosis for XXX Syndrome. The patient was tall for family size, slightly delayed in language skills, and the mother reported the patient's IQ was significantly lower than that of her siblings. The patient had retrognathism, with a thyromental distance of 5cm and a Mallampati score of III. The mother reported that the girl had exhibited muscle weakness of unknown etiology when emerging from anesthesia after a tonsillectomy at age five. The patient took no medications and denied allergies. Lab work, which included a negative urine pregnancy test, was within normal limits.

Rapport was established with the patient. A 20 gauge IV was started. Because of the possible need for neuro-muscular blockade to perform the surgical procedure, concerns were discussed with the surgeon related to airway difficulty and the patient's history of post-operative muscle weakness. The collaborative plan included initial insertion of a fastrach laryngeal mask airway (LMA Fastrach, San Diego, CA). If paralysis was required as the surgery progressed, an intermediate-acting neuromuscular blocker would be given and an endotracheal tube would be placed. The patient and mother received thorough pre-operative teaching.

Ondansetron 4mg and decadron 4mg were administered intravenously (IV) for prevention of nausea and vomiting. The patient also received midazolam 2 mg IV as an anti-anxiolytic before being transported to the operating room. Monitors and a face mask delivering O_2 at 10 L/min were secured on the patient. Lidocaine 40 mg IV was injected, followed by propofol 140 mg IV. Once eyelid responses were no longer present, the intubating laryngeal mask airway was placed without difficulty. Bilateral breaths sounds were auscultated, and $ETCO_2$ was observed on the monitor. Sevoflurane was initiated in O2 1 L/min and air 1 L/min. The patient's breathing was assisted until spontaneous respirations resumed at adequate tidal volumes and rate to maintain $ETCO_2$ between 35-45 mmHg. General anesthesia was maintained with sevoflurane and fentanyl 150 mcg total, administered in incremental doses. An upper body air warmer was secured on the patient. The patient breathed spontaneously for the duration of surgery.

Once the patient followed commands, the LMA was removed. Oxygen saturation, ventilation, and vital signs remained good throughout the recovery room stay. The patient was discharged the following day without incident.

Discussion

Characteristics of Triple X Syndrome related to anesthesia care, including an increased likelihood of difficult airway, strained interpersonal relations, and a lower than expected IQ, along with this patient's history of post-operative muscle weakness, will be addressed in this discussion.

Diligent observation, evaluation, and the awareness of specific characteristics related to specific conditions which may affect anesthesia choices and outcomes are crucial for anesthesia practitioners. This is especially necessary when dealing with a disorder that is often under-diagnosed, as is sometimes the case with XXX Syndrome.[1] The review of the literature indicates that patients with Triple X Syndrome are tall, often uncoordinated females with difficulty in language skills and interpersonal relationships.[1] A smaller head circumference

and an IQ significantly lower than their family members have been documented in XXX females.[1] Cardiac anomalies are of no greater frequency than found within the general population,[1] but a higher incidence of seizure activity is reported.[3] Short necks and mandibular prognathism or retrognathism are also documented.[1] Irregularities in menstruation are common, as is premature ovarian syndrome.[2] Many XXX females, however, produce chromosomally normal children.[3]

This patient displayed many of the above symptoms. For her age, this patient was slow in language development and motor skills. Her mother reported the patient had significant difficulty developing and maintaining inter-personal relationships outside of their immediate family. Physically, the patient was tall for her family size and had a wide, short neck, with notable retrognathism.

Information was directed to both the patient and her mother in order to build a relationship of trust with both. The anesthesia plan was explained at a level that the patient could understand in order to decrease her anxiety and consequential responses. Gentle touch, simple humor, and an unrushed approach were employed to ease the apprehension caused by the IV start. Midazolam 2mg IV was slowly titrated in order to decrease the patient's anxiety. Propofol was the induction drug of choice secondary to the higher incidence of seizure activity in XXX females, although this patient had no seizure history.

Because of the patient's retrognathism, a thyromental distance of 5cm, and a Mallampati score of III, there was an increased likelihood of difficult airway.[4] Given this patient's history of reported post-operative weakness, the surgeon was consulted to discuss the need for neuro-muscular blockade and airway options. During UHR, neuro-muscular blockers (NMBs) may or may not be required depending on the patient's condition, the surgeon's preference, and anesthesia practitioner preference. Since this patient had no contraindications for LMA placement, it was decided that an LMA fastrach would initially be inserted, the surgeon would examine the wound, and if paralysis was necessary, a NMB would be administered. An endotracheal tube could be placed through the LMA fastrach if needed.

Use of an LMA requires fewer medications to attenuate the sympathetic nervous system response secondary to airway placement and there is a decrease in coughing and incisional stress at the time of airway removal when compared to extubation.[5] While both spinal and epidural anesthesia are possible anesthesia choices for UHR, the mother reported that this patient would not be able to sit still for either procedure, so these options were ruled out.

As noted during the pre-operative evaluation, the patient's mother reported post-operative muscle weakness following the patient's tonsillectomy. While the patient was not reintubated post-operatively, she was retained in the recovery room for four hours secondary to poor respiratory efforts. The report of the patient's anesthetic care during her previous surgery would have been extremely helpful, but was not available for review. Because of this patient's reported post-operative weakness, however, collaboration with the surgeon was especially important in order to avoid unnecessary polypharmacy. The surgeon was not certain that paralysis was necessary, and after discussion, it was decided to initially use fentanyl, propofol, and sevoflorane. Had complete paralysis become

necessary, a low-dose of intermediate-acting NMB would have been administered.

Patients with Triple X Syndrome show an array of documented characteristics which must be considered by the anesthesia practitioner, as outlined above. The high incidence of undiagnosed disease, difficulties with language processing and interpersonal interaction, difficult airway, and increased incidence of seizures are serious concerns with Triple X Syndrome.[1] This patient's history of post-operative muscle weakness was isolated, but still of concern and was included in our anesthesia plan. As always, ongoing research and conscientious evaluation, planning, documentation, and follow-up for each specific patient will improve both individual and general care practices in the provision of effective anesthesia.

References

1. Otter M, Schrander-Stumpel C, Curfs L. Triple X syndrome: A review of the literature. *Eur J Hum Genet.* 2010;18:265-271.
2. Lashley F. *Clincal Genetics in Nursing Practice.* 3rd ed. New York, NY: Springer Publishing Company; 2006: 114.
3. Gross S, Faretani M, DiBartolo R, et al. Electroencephalographic and epileptic patterns in X chromosomal anomalies. *J Clin Neurophysiol.* 2004;21:249-253
4. Lavery G, McCloskey B. The difficult airway in adult critical care. *Crit Care Med.* 2008;36:2163-2173.
5. Yu S. Laryngeal mask airways have a lower risk of airway complications compared with entotracheal intubation: A systematic review. *Maxillofacial Surgery* 2010;68:2359-2376.

Mentor: Christine Langer**,** CRNA, MS, MSEd, MSN

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

To access prior issues of the ISJNA visit the following link:

www.aana.com/studentjournal

INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA
GUIDE FOR AUTHORS

MISSION STATEMENT

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

ITEMS ACCEPTED FOR PUBLICATION

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

ITEM PREPARATION & SUBMISSION

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

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

PEER REVIEW

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

General guidelines

1. Items for publication must adhere to the *American Medical Association Manual of Style* (AMA, the same guide utilized by the *AANA Journal* and such prominent textbooks as *Nurse Anesthesia* by Nagelhout and Plaus). The review process will not be initiated on reports submitted with incorrect formatting and will be returned to the mentor for revision. Please note the following:
    a. Use of abbreviations is detailed in Section 14. Spell out acronyms/initialisms when first used. If you are using the phrase once, do not list the acronym/initialism at all.
    b. Instructions regarding units of measure can be found in Section 18. In most cases The International System of Units (SI) is used. Abbreviations for units of measure do not need to be spelled out with first use. Some examples: height/length should be reported in cm, weight in kg, temperature in ^{o}C, pressure in mm Hg or cm H_20.
    c. In general, first use of pulmonary/respiratory abbreviations should be expanded, with the following exceptions: O_2, CO_2, PCO_2, $PaCO_2$, PO_2, PaO_2. Please use SpO_2 for oxygen saturation as measured by pulse oximetry.
    d. Use the nonproprietary (generic) name of drugs - avoid proprietary (brand) names. Type generic names in lowercase. When discussing dosages state the name of the drug, *then* the dosage (midazolam 2 mg).
    e. Use of descriptive terms for equipment and devices is preferred. If the use of a proprietary name is necessary (for clarity, or if more than one type is being discussed), give the name followed by the manufacturer and location in parenthesis:
        "A GlideScope (Verathon Inc., Bothell, WA) was used to . . . ."
        Please note, TM and ® symbols are not used per the AMA manual.
    f. Examples of referencing are included later in this guide.

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

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

<u>Case Reports</u>

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. Diagrams must have permission from original author. This is the most important part of the article. In terms of space and word count this should be longer than the case presentation. End the discussion with a summary lesson you learned from the case, perhaps what you would do differently if you had it to do over again.

[space]

References (bold)

[space]

A minimum of 5 references is recommended, with a maximum of 8 allowed. 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 programs 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
Goldfarb School of Nursing at Barnes-Jewish College
4483 Duncan Ave., Mailstop 90-36-697
St. Louis, MO 63110

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.