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The Vortex

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Front Cover:

Amy Bell, RN, BSN, a graduate student enrolled in the Truman Medical Center Nurse Anesthesia Program in Kansas City, Missouri, places an arterial catheter.

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Prevention of Coagulopathy Associated with Massive Transfusion

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Keywords: massive transfusion, coagulopathy, thrombocytopenia, fresh frozen plasma, acidosis

Individuals suffering from massive hemorrhage are at risk for consumptive coagulopathy as well as dilutional coagulopathy after massive transfusion of packed red blood cells (PRBCs).[1] The cause of death from severe hemorrhage that requires transfusion of large volumes of blood products is generally due not to lack of hemostasis, but from acidosis, hypothermia, and the accompanying coagulopathy.[2] Adequate replacement of blood volume by crystalloids, colloids, and PRBCs may improve hemodynamics and oxygen-carrying capacity of the vasculature, but does not provide clotting factors, and can subsequently lead to dilutional coagulopathy.

Case Report

A 58-year-old, 59 kg, 173 cm male presented for an open bilateral retroperitoneal lymph node dissection. His medical history included testicular cancer involving the lymphatics. Treatment included chemotherapy sessions which ended six months prior. Other medical history included left ischemic optic neuropathy with vision loss and spontaneous subdural hematoma with no residual effects. Current medications included aspirin 81 mg per day for optic neuropathy prevention. Pre-operative laboratory results revealed a hemoglobin of 12.6 g/dL, hematocrit of 37.9%, and platelet count of 229 K/cumm.

A peripheral intravenous (IV) catheter was established and midazolam 2 mg was administered. The patient was placed supine on the operating table and general anesthesia was induced with lidocaine 60 mg, fentanyl 100 mcg, propofol 120 mg, and neuromuscular blockade was initiated with rocuronium 60 mg. The trachea was successfully intubated with an 8.0 mm cuffed endotracheal tube and an esophageal doppler probe inserted for stroke volume (SV) assessment. An additional peripheral IV catheter and an arterial catheter were placed, fluid was administered through a fluid warmer, and an upper body forced air warming blanket applied. General anesthesia was maintained with desflurane 6% inspired concentration in a mixture of O_2 1 L/min and air 1 L/min.

The patient's baseline SV measured 60 mL/beat. Two hours into the case, SV fell to 30 mL/beat followed by the surgeon reporting a tear in the inferior vena cava (IVC). Arterial blood pressure subsequently decreased to 60/30 mm Hg. Phenylephrine 300 mcg was administered and an intravenous infusion of phenylephrine 0.5 mcg/kg/min was initiated. Intravascular volume was resuscitated with 2 L of crystalloids, 500 mL of 5% albumin, and 2 units of PRBCs while the IVC was repaired. An arterial blood gas analysis drawn at the beginning of resuscitation revealed pH of 7.31, PO_2 206 mm Hg, PCO_2 51 mm Hg, bicarbonate concentration of 23 mmol/L, and hematocrit of 37%.

In anticipation of further blood loss, two additional PIV catheters were placed. An hour further into the case, a second IVC tear occurred. Additional resuscitation included 1900 mL of

crystalloid, 500 mL of 5% albumin, 6 units of PRBCs, 4 units of fresh frozen plasma (FFP), and 2 units of platelets were transfused. Calcium chloride 2 g and sodium bicarbonate 12.6 g were administered. Total estimated blood loss exceeded 3300 mL and mid-transfusion laboratory results revealed pH of 7.22, PO_2 246 mm Hg, PCO_2 57 mm Hg, hemoglobin of 10.3 g/dL, hematocrit of 31.4%, platelet count of 110 K/cumm, and INR of 1.31.

Due to the above complications, continuation of surgery was aborted. The phenylephrine infusion was titrated off, and a propofol infusion initiated. Divided doses of hydromorphone 0.2 mg were administered. The patient remained intubated and was transferred to the intensive care unit. Post-operative laboratory results revealed pH of 7.33, PO_2 96 mm Hg, PCO_2 51 mm Hg, hemoglobin of 13.9 g/dL, hematocrit of 41.7%, platelet count of 149 K/cumm, and INR of 1.12. The following morning the endotracheal tube was successfully removed and the patient experienced no further complications.

Discussion

The administration of PRBCs without additional clotting factors or platelets may result in dilutional coagulopathy and thrombocytopenia, further inhibiting hemostasis. In the past, hemorrhaging patients were initially resuscitated with crystalloid infusions, only administered blood products based on laboratory results, with the goal hemoglobin above 10 g/dL, platelet count above 50 K/cumm, and INR less than or equal to 1.5.[3] However, with these practices blood loss continued due to laboratory result delays, decreasing blood viscosity, and diluted clotting factors and platelets,[3] while large crystalloid infusions resulted in intracellular edema and disruption of biochemical processes.[4]

Furthermore, an acute endogenous coagulopathy associated with systemic hypoperfusion, anticoagulation, and hyperfibrinolysis can occur before clotting factor depletion or dilution after massive hemorrhage.[1] The mechanism involves alteration in the thrombomodulin protein C pathway with activated protein C and systemic anticoagulation.[1]

Recent literature recommends restriction of crystalloid infusions, with the use of plasma as the primary means of volume expansion.[4] Early transfusion of high ratios of FFP to PRBCs, such as 1:1 or 1:3, to treat postinjury coagulopathy has been shown to reduce mortality and replenish clotting factors.[2] Furthermore, current literature supports the use of 1:1:1 ratios of FFP to PRBCs to platelets for transfusion protocols, as it leads to restoration of intravascular volume, avoidance of dilution of platelets, and replenishment of clotting factors.[5]

Acidosis, hypothermia, and coagulopathy exacerbate each other, even leading to death.[1] Although stored blood products have an acidic pH, the metabolic product of citrate is bicarbonate, thereby rarely affecting the patient's pH balance.[1] Instead, the cause of acidosis with hemorrhage and massive transfusion is generally due to hypoperfusion of the tissues. Clotting factors are impaired by acidemia, as a decrease from 7.4 to 7.0 reduces clotting factor activity by 55-90%.[1] Treatment for acidosis should involve sodium bicarbonate administration as well as resolution of the causative condition and restoration of tissue perfusion.[1]

Hypothermia may cause a coagulopathy as well via reduction in clotting factor activity. Prolonging clotting times are present at temperatures below 33°C with a 10% reduction in coagulation factor activity with each 1°C drop in temperature.[1] Treatment includes elevation of room temperature, applying heating blankets, utilizing heated and humidified gases for ventilators, and administering blood and fluids through warmers.[1]

Citrate intoxication due to very rapid infusion of banked blood reduces the level of ionized calcium, causing complications such as hypotension, a prolonged QT interval, or decreased myocardial contractility.[1] However, normothermic patients with normal kidney and liver function are at a low risk.[1] Therefore, ionized calcium should be monitored and maintained within normal range.

Throughout resuscitation, the patient's vascular oxygen-carrying capacity, coagulation profile, and metabolic derangements should be closely assessed.[3] At this time, these are most often monitored by hemoglobin, hematocrit, prothrombin, INR, pH, and calcium levels. However, many of these laboratory assays are not likely to be available in real time, therefore transfusions should continue while laboratory analysis of samples is in progress.[3]

Other hemostatic assays include thromboelastography (TEG) and rotational thromboelastometry (ROTEM) which may offer coagulation assessment in a timely fashion.[3] Such assays provide a graphical representation of the coagulation process and provide a quantitative measure of individual clotting factors.[3] Advantages of these point-of-care tests include fast turnaround time, ability to detect hyperfibrinolysis, and assessment of phases of coagulation. The can be performed at the patient's true temperature, thereby being sensitive to coagulopathy due to hypothermia.[3] Furthermore, ROTEM has been shown to decrease the amount of blood products administered during massive transfusion, improve patient survival, and promote accurate diagnosis of coagulopathy.[5]

In this presented case, coagulopathy was successfully avoided, although a large volume of crystalloids was administered. A high ratio of FFP to PRBCs of 1:2.5 was transfused along with platelets. Citrate toxicity was avoided due to calcium chloride administration. Furthermore, arterial pH was monitored and acidosis was corrected with sodium bicarbonate and maintenance of tissue perfusion with vasopressors and blood volume resuscitation. Although warming blankets, fluid and blood warmers, and a humidified and heated ventilator circuit was utilized, the patient's core temperature averaged 34.5°C, thus warming of the operating room should also have been utilized. Laboratory assays were sent during mid-transfusion of products, but utilization of TEG and ROTEM assays should have considered, potentially leading to administration of cryoprecipitate, fibrinogen, or tranexamic acid. The post-operative laboratory results revealed normal hemoglobin, hematocrit, platelet, and INR, thereby verifying successful replacement of intravascular volume and oxygen-carrying capacity of the blood, without induction of a dilutional coagulopathy.

Risks of bleeding are inevitable with many surgical cases, therefore it is important for anesthesia practitioners to be aware of interventions to avoid coagulopathies associated with massive blood product administration. By transfusing high FFP to PRBC ratios, administering platelets as

clinically indicated, avoiding acidosis, preventing citrate toxicity, and maintaining normothermia, the incidence of coagulopathies can be reduced.

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Anesthetic Management and Considerations for Venous Air Embolism

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Keywords: Venous air embolism, vascular air embolism, gas embolism, carbon dioxide embolism, and management

A venous air embolism (VAE) is a potentially fatal condition that occurs when air enters into the vasculature from an existing pressure gradient. Studies show patients undergoing neurosurgery, especially posterior fossa surgeries, in the sitting position have the highest incidence of VAE. Other high-risk surgeries include laparoscopic, orthopedic, obstetric-gynecological, and cervical laminectomies.[1] Mortality rates of a VAE show to be as high as 28%.[2]

Case Report

A 45-year-old, 70 kg, 168 cm female presented for a total laparoscopic hysterectomy indicated for dysmenorrhea due to uterine fibroids. Her medical history included migraine headaches, dyslipidemia, uterine fibroids, intrauterine fetal death, and disseminated intravascular coagulation. Surgical history included three cesarean sections and two umbilical hernia repairs. Her medications included norgestimate/ethinylestradiol, iron, acetaminophen, and a multivitamin.

The patient was given midazolam 2 mg intravenously (IV) in the holding room and then transported to the operating room (OR). She was transferred to the OR table where non-invasive monitors were applied including a bispectral index monitor (BIS), non-invasive blood pressure cuff, electrocardiogram, and finger pulse oximetry. Her vital signs prior to induction were: heart rate 89/min, blood pressure 168/71 mm Hg, and a SpO_2 of 100%. The patient was pre-oxygenated via facemask with O_2 6 L/min. Induction medications were administered IV and included: fentanyl 50 mcg, lidocaine 50 mg, propofol 180 mg, rocuronium 5 mg, and succinylcholine 140 mg. A 7.0 endotracheal tube was placed, bilateral breath sounds were auscultated, and she was placed on a mechanical ventilator with a respiratory rate of 10/min, a tidal volume of 500 mL, and positive end-expiratory pressure of 4 cm H_2O. Inspired oxygen was delivered at 70% and general anesthesia was maintained with sevoflurane with end-tidal concentrations of 1.7-2.6% throughout the case.

The patient was repositioned in the lithotomy position and fentanyl 50 mcg and rocuronium 30 mg was given IV just prior to incision. Insufflation of the abdomen began and there was an immediate drop in $ETCO_2$ from 32 to 15 mm Hg and then to 0 mm Hg. Additionally, there was a decrease in blood pressure, heart rate, and oxygen saturation of 44/30 mm Hg, 32/min, and 76% respectively. The surgeon was notified and the abdomen was desufflated. Ephedrine 25 mg was administered IV promptly. The patient was found to be in asystole and chest compressions were initiated. Chest compressions ceased after 2 minutes at which point a femoral pulse was detected. A total of epinephrine 2 mg and atropine 2 mg were given in 0.5 mg doses IV to support the patient's hemodynamics during the period of resuscitation.

A decision was made to cancel the surgery, however, blood was found to be pooling from the patient's vagina and an emergent exploratory laparotomy was performed. Two additional intravenous catheters, an arterial line, and central line were placed. Throughout the case, the patient's BIS read 0, $ETCO_2$ remained in the mid 20's mm Hg, and SpO_2 90-92%. The patient's uterus was removed as this was the suspected source of air entrainment due to perforation related to trochar placement. At the completion of the surgery, the patient was transferred to the surgical critical care unit, intubated and mechanically ventilated. Over the following 36 hours, the patient was weaned from the ventilator and extubated. She showed evidence of left-sided weakness and was diagnosed as having a cerebral vascular accident by computed tomography. The patient was discharged from the hospital to her home on postoperative day six.

Discussion

The primary factors that make up the pathophysiology of a VAE and determine its severity are the volume of air entering the vasculature and the air entrainment rate.[3] In adults, the lethal volume of air remains unknown, but it is estimated to be from 200 to 300 mL or 3 to 5 mL/kg.[1] Overall, death can occur from a VAE due to circulatory collapse from gas lock in the pulmonary circulation. Right ventricular outflow is impeded resulting in a decreased left atrial preload. Consequently, decreased cardiac output and hypotension occur.[4] In this case, the patient experience immediate hemodynamic instability and cardiac collapse during insufflation, most likely due to the uterus being perforated creating an avenue for air entrainment.

The presenting signs and symptoms of a VAE include electrocardiogram changes, hypotension, changes in respiratory pattern and heart rate, increased peak airway pressure and pulmonary artery pressure, and decreased $ETCO_2$ and oxygen saturation.[2,4] This information correlates with the clinical picture the patient displayed from the case study.

Detection sensitivity of a VAE varies between monitoring methods. Currently, transesophageal echocardiogram is the most sensitive method of detection as it has been shown to detect as little as 0.02 mL/kg of air. Of the noninvasive methods of detecting a VAE, precordial doppler ultrasound is the most sensitive. It has the ability to detect as little as 0.05 mL/kg of air.[3] However, its effectiveness is questionable. One study has shown some degree of VAE to occur in 100% of patients undergoing laparoscopic surgeries.[5] Yet, in a study of 61 patients undergoing laparoscopic surgery, VAE could not be detected using a precordial doppler ultrasound.[6] Another method for detecting a VAE is through the use of $ETCO_2$ monitoring. A decrease in $ETCO_2$ can be seen with a VAE related to blood flow obstruction and the increase in pulmonary dead space.[2] This occurrence has been demonstrated to be the initial response to a VAE through a case report where a 69-year-old female who underwent a laparoscopic cholecystectomy developed a VAE. Her $ETCO_2$ decreased from 40 mm Hg to 7 mm Hg and then to 0 mm Hg.[7] This is very similar to the pattern the patient from the case study presented. Lastly, using a precordial stethoscope to detect a "mill-wheel" murmur has been demonstrated as a late sign, and unreliable.[1] A recent review revealed that a "mill-wheel" murmur could only be detected by less than half of patients who had a VAE.[2] In this case, no monitors were used specifically for the detection of VAE, however, $ETCO_2$ played a key role. A decrease in $ETCO_2$ was the first indication of circulatory collapse because the patient could still be adequately ventilated. Given the clinical picture of a decrease in ETCO2, hemodynamic instability, and asystole upon insufflation, a diagnosis of VAE could be made.

Recognizing high-risk patients and surgeries is vital to prevent a VAE. Any surgery in which the surgical site is above the level of the heart creates a pressure gradient and places the patient at an increased risk for a VAE. Also, laparoscopic surgeries and patients with decreased central venous pressure (CVP) have also been shown to have an increased incidence of VAE.[3,5] If these surgical conditions occur, increasing venous return to the right side of the heart by elevating the patient's legs, administering a fluid bolus, and ensuring adequate hydration prior to surgery to achieve a CVP of 10-15 mm Hg will decrease the pressure gradient.[3,4] The patient's fluid status from the case study was not optimized as she had been fasting throughout the night. This placed her at an increased risk for a VAE, as did the nature of the laparoscopic surgery.

Specific interventions should be immediately implemented to help improve patient outcomes when a VAE occurs. Goals are to prevent further air entry, reduce the amount of air entrained, and provide hemodynamic support.[3] These interventions include ceasing insufflation, covering routes of entry with saline soaked gauze, rapid fluid volume expansion to elevate venous pressure, and ceasing of any nitrous oxide administration.[1,2,4] Air retrieval through a central venous catheter in order to reduce the obstruction is a controversial intervention. One case study found it to be successful where several mL of air could be retrieved and the patient survived.[8] However, many animal and lab studies indicate unreliable amounts of air can be aspirated which may not be significant enough for survival.[2-4] Because evidence is variable, recommendations are to not place a central venous catheter for aspirating air during a VAE as a first line treatment. If a

central venous catheter is already present, then its use is warranted. Air retrieval was not attempted in the case study patient. However, a central line was placed emergently for rapid fluid administration.

Hemodynamic support, which may include cardiopulmonary resuscitation, and defibrillation is required to maintain oxygenation and perfusion to vital organs during the event of a VAE. With cardiac outflow obstruction, chest compressions and positive inotropic agents have been shown to be effective in relieving the gas lock because they force the air into smaller vessels allowing the continuity of blood flow.[3,4] Treatment for the case study patient included providing hemodynamic support with ACLS resuscitation and cardiac compressions. Implementation of evidence-based recommendations for VAE management increases the probability of optimal patient outcomes.

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Blood Loss during Cesarean Delivery

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Keywords: spinal anesthesia, subarachnoid block, cesarean delivery, blood loss, intrapartum hemorrhage

Hemorrhage is the leading cause of maternal death.[1] Intrapartum and postpartum hemorrhage can occur in otherwise healthy parturients. Many methods to control hemorrhage have been studied in surgical patients but their usefulness in obstetric patients has recently been discovered.[2]

Case Report

The patient was a 28-year-old gravida 2, para 1 female scheduled for an elective cesarean delivery at 38 5/7 weeks gestation. Her surgical history included a cesarean delivery for breech presentation in 2013 and an appendectomy in 1998. Her medical history only included nephrolithiasis. Her daily medications included a prenatal vitamin. She was 167 cm and 72 kg after gaining 14 kg with the pregnancy. Her baseline blood pressure (BP) was 112/68 mm Hg. Preoperative laboratory analysis included a hematocrit of 36%, hemoglobin (Hgb) of 11.8 gm/dL and platelet count of 212,000 platelets per microliter of blood. A type and screen was ordered and collected.

The patient was transported into the operative suite with a 20-gauge peripheral intravenous (IV) catheter infusing lactated ringer's (LR). Standard monitoring was applied and the patient was placed in the sitting position. A subarachnoid block was placed using 0.75% bupivicaine 12 mg with fentanyl 20 mcg. The patient was repositioned supine with a left uterine tilt of 15 degrees. She became hypotensive with a BP of 82/58 mm Hg and received phenylephrine 80 mcg and an increased rate of fluid administration.

The fetus was delivered from the uterus and oxytocin 20 units was added to 1000 mL LR. As the placenta was being removed the patient became tachycardic and hypotensive with a heart rate of 120-130/min and BP of 72/38 mm Hg. Blood samples for a complete blood count and crossmatching were sent after the obstetrician stated she had encountered unexpected bleeding into the retroperitoneal space. An 18-gauge peripheral IV was placed in the patient's left forearm and LR 1000 mL was infused rapidly using a pressurized bag. The patient's BP was treated with both phenylephrine and ephedrine but experienced a decreased level of consciousness and paleness of her face. Another LR 1000 mL was infused as well as 5% albumin 500 mL. The obstetricians reported they had located and controlled a hemorrhaging uterine artery. Estimated blood loss was 1600 mL. The obstetricians indicated that there was additional blood loss in the retroperitoneal space that they were unable to quantify or evacuate. The patient's BP normalized to her preoperative level and her level of consciousness returned to baseline. The patient's intraoperative Hgb was 10 gm/dL so transfusion was not required.

Discussion

Hemorrhage is responsible for 25% of maternal deaths worldwide, making it the most common cause of maternal death.[1] Complications of maternal hemorrhage include myocardial ischemia, infarction, cerebrovascular accident, and other organ dysfunction.[1] Hemodynamic shock is the most common cause of repeat laparotomy after cesarean delivery.[3] Obstetricians and anesthesia practitioners frequently underestimate intrapartum blood loss causing delays in treatment.[1] The Advanced Trauma Life Support publication classifies hemorrhagic shock into four categories. Hypotension and tachycardia greater than 120/min are not evident until Class III hemorrhage. Hemorrhage is categorized as Class III when the patient has lost 30-40% of total blood volume and is moderately anxious and confused.[1]

Retroperitoneal hematomas during cesarean section are the most dangerous hematoma as they are rapidly enlarging and often concealed.[1] Unanticipated tachycardia, hypotension and

decreased hematocrit should lead the anesthesia practitioner to consider a concealed retroperitoneal hematoma. In a study of 39 women with a broad ligament hematoma leading to a retroperitoneal bleed, 38 required a blood transfusion and a hysterectomy was performed for 8 of the women.[4] Restrictive transfusion practices should be utilized due to the associated risks, including transfusion associated circulatory overload, transfusion related acute lung injury, viral transmission, electrolyte disturbances, and transfusion reactions. Current recommendations support transfusion when hgb is less than 7 gm/dL in asymptomatic patients. The American Association of Blood Banks and the American College of Obstetrics and Gynecology do not have published guidelines for transfusion practices in obstetric patients.[1] In the previously described case study, the patient's repeat hgb was 10 gm/dL, which led to the decision not to transfuse. Her hypotension was controlled with crystalloid, vasopressors and colloid, and her level of consciousness improved once she became normotensive.

The majority of planned cesarean sections are performed under spinal anesthesia. Subarachnoid blocks lead to hypotension through decreased arteriolar tone and maternal sensitivity to local anesthetics causing a greater sympathectomy.[5] These factors further complicate the hypotension patients exhibit from blood loss. Formerly, patients were routinely pre-loaded with crystalloid before placement of a subarachnoid block.[5] The patient in this case study was not pre-loaded with crystalloid but had received approximately 1000 mL of LR before the bleeding was noted.

Evidence suggests that crystalloid rapidly redistributes from the intravascular space into the interstitial space. Pregnant patients have decreased plasma proteins, negating the effects of pre-loading. Pre-loading with colloid, such as hydroxyethylstarch or albumin, is now recommended instead of crystalloid due to its less rapid redistribution to the interstitial space.[5] The practice of crystalloid co-loading, in which crystalloid is rapidly infused during placement of the intrathecal local anesthetic, is also described in the literature. [5] The rapid transfer of crystalloid into the interstitial space is minimized in co-loading due to the simultaneous vasodilation from the autonomic blockade.[5] In this case study, when the hemorrhage was noted the patient was initially given a bolus of LR. When her hemodynamic parameters did not improve after crystalloid was rapidly administered, her blood pressure was treated with vasopressors. Albumin was also infused within minutes with an improvement in BP. It may be inferred that had the anesthesia practitioner intervened with colloid sooner, the patient may have experienced less drastic hypotension and potentially could have maintained her level of consciousness.

Tranexamic acid (TXA) is an antifibrinolytic that inhibits fibrin from binding to plasmin and plasminogen.[2] Its use has been studied extensively in general and trauma surgery but the efficacy of TXA in intrapartum and postpartum hemorrhage has only been evident in the literature recently. Infusing TXA before a cesarean section has significant benefits, including less blood loss and decrease in hemoglobin, postpartum hemorrhage, and need for pharmacologic methods to increase uterine tone.[2] Antifibrinolytic agents may cause thrombosis, which is of concern in pregnant patients who are pro-thrombotic. The meta-analysis showed no difference in the risk of thromboembolus between patients who received TXA and those who did not.[2] TXA could have been a viable option to promote clotting and assure that the patient was able to maintain her hemoglobin level in the postpartum period.

During cesarean deliveries, anesthesia professionals should be aware of the risks associated with intrapartum hemorrhage, particularly in otherwise healthy patients. Crystalloid pre-loading before initiation of a spinal anesthetic should be abandoned for either a co-loading technique, during the placement of the spinal, or pre-loading with colloid.[5] In addition, the use of TXA should be further investigated in parturients as it may be a safer and more effective method for control of hemorrhage than blood product transfusion.[2]

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Perioperative Median Nerve Injury

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Keywords: peripheral nerve injuries, median nerve injury, prone position, nephrolithotrispy

Perioperative peripheral nerve injuries occur with a reported incidence of 0.03 % to 0.1 % and represent 15 % to 16 % of anesthesia professionals malpractice claims.[1,2] The cause of perioperative nerve injuries is often multifaceted including preoperative identifiable risk factors, intraoperative position, type of surgery and intraoperative equipment.[1,2] The slowing or change in impulse conduction that manifests in a peripheral nerve injury is generally identified in the postoperative period, increasing the difficulty in pinpointing its exact intraoperative cause.[1,2]

Case Report

A 53-year-old, 160 cm, 96 kg, female with a body mass index of 37.6 kg/m^2 presented for a left percutaneous nephrolithotripsy. Past medical history was significant for obesity, hypertension, non-insulin dependent diabetes complicated by neuropathy, osteoarthritis, and kidney stones. Home medications consisted of januvia, lisinopril, metoprolol, premarin, metformin, mirena, and nitrofurantoin. The patient's allergies were listed as penicillin, bactrim, and morphine. Previous

surgical history included lithotripsy, neck cyst removal, right thumb repair, and a cesarean section with no reported complications. Current laboratory values were reviewed as normal except for A1C of 7.1%.

In the preoperative area two 20 gauge peripheral intravenous (IV) lines were inserted into her right antecubital and right hand. Midazolam 2 mg IV was administered. In the operating room noninvasive monitors were placed; the blood pressure cuff was placed on her left upper extremity and set to cycle every 3 minutes. General anesthesia was initiated with fentanyl 100 mcg, lidocaine 60 mg, propofol 150 mg, and rocuronium 50 mg administered IV. A 7.0 mm endotracheal tube was placed without complication and ventilation was established. The patient was then repositioned to a prone position, full monitoring and volume control ventilation was reestablished. General anesthesia was maintained with sevoflurane 2% inspired concentration in a mixture of O_2 1 L/min and air 1 L/min. Her position was further inspected and she was found to have a neutral head position, shoulders were abducted less than 90°, the patient's forearms were secured on padded arm boards with velcro paddled straps while maintaining a neutral wrist position. In addition padded bolsters were placed laterally without pressure on her breasts, and padding was positioned at the knees.

Intraoperative narcotics, totaling fentanyl 200 mcg and hydromorphone 1.4 mg, were administered during the case. Prior to the completion of surgery train of four (TOF) was documented as 3 out of 4 twitches. Neuromuscular blockade was antagonized with glycopyrrolate 0.4 mg and neostigmine 2 mg, with TOF returning to 4 out of 4 twenty minutes after administration. At the conclusion of surgery patient was repositioned supine onto the hospital bed. The endotracheal tube was removed once the patient demonstrated 8 mL/kg spontaneous tidal volumes, sustained head lift, and could follow commands. She was transferred to post-anesthesia care unit (PACU) in stable condition.

After arrival to PACU patient reported severe numbness and tingling on all aspects of her left arm and hand beginning two inches below the elbow and extending through the hand and fingers. The patient also noted loss of left hand coordination, and was unable to hold a cup without spilling. A neurological consult was initiated. There was no obvious injury, edema, or erythema to the left arm. Twenty-seven-hours after anesthesia the patient's symptoms had improved with numbness and tingling localized to the palmer surface of the hand as well as the thumb, index, and middle fingers. The patient was diagnosed with a left arm median nerve injury and followed by neurology while inpatient. On day of discharge, post-operative day four, the patient's symptoms had resolved and no further follow up was required.

Discussion

The PACU team quickly identified a nerve injury in this patient. Initially a more severe generalized brachial plexus injury was hypothesized but quickly ruled out as the signs and symptoms clearly pointed to a median nerve injury, such as her weak grasp, loss of sensation in the first three fingers of the left hand, and inability to hold a cup.[3] The median nerve "supplies motor innervation to most of the flexor muscle in the forearm" as well as supplying "sensory innervation to the radial aspect of the palm and the thumb, index, long and radial half of the ring finger". [3, p.95] The injury to her median nerve resolved in 4 days thus suggesting a neuropraxia

injury, which does not affect the axon of the nerve but instead results in decreased nerve conduction resulting from myelin damage. [3] The cause of this injury is more difficult to pinpoint than the diagnosis. However, preoperative risk factors, the type of surgery, the patient's position and equipment utilized intraoperatively could all increase the risk of nerve injury.[2,4-8]

Observational studies have reported perioperative nerve injuries in patients with preoperative diagnoses of diabetes, hypertension, extreme body habitus, vascular disease, alcohol dependence and arthritis.[2,4] The patient in this case study suffered from chronic hypertension, large body habitus, and non-insulin dependent diabetes poorly controlled and complicated by pre-existing peripheral neuropathy. Identifying conditions which may place the patient at an increased risk for developing peripheral nerve injury is an important aspect of the preoperative assessment and can alert the anesthesia professional to an increased risk of complications.[4]

The surgery type and position can also increase the risk of peripheral nerve injury. In a study involving 380,680 cases over a ten-year-period, 112 peripheral nerve injuries were reported, of this number 15% involved urologic procedures.[5] The need to access the retroperitoneal and pelvic organs involves positions that have been shown to increase the peripheral nerve injury.[6] The prone position utilized in this three-and-a-half hour case has demonstrated an increase in the occurrence of upper extremity neurapraxia.[6] The increased incidence of injury is primarily attributed to stretch or compression of the peripheral nerves. Stretching the nerve 5-15% beyond the normal resting length can cause ischemia resulting in injury.[2] But, as described above, the patient's arms were not abducted greater than 90^{o} and the forearms were in a neutral position. The practice advisory for the prevention of perioperative peripheral neuropathies states that there is insufficient literature to evaluate perioperative positioning strategies to reduce the occurrence of median nerve injury.[4] The lack of evidence makes it difficult to determine if positioning contributed to the injury, in this case the injury was unilateral, and there was no difference in positioning between the two extremities. Positioning could have been a contributing factor, but it is likely than compression was also a factor in this injury.

Intraoperatively every three minutes on the left extremity the blood pressure cuff inflated and recorded a blood pressure for evaluation. In a review of the literature five cases reports were found that described ulnar and radial nerve injuries from the use of an automated blood pressure cuff .[4] No case studies were identified describing isolated median nerve injury associated with the use of automated blood pressure cuffs, but this may have been a contributing factor in this injury. A compression injury could have occurred where the median nerve passed down the medial arm between the biceps brachii and the brachialis muscles parallel to the brachial artery.[7] The compression injury would have led to an increase in intraneural and extraneural pressure, this would reduce the perfusion pressure to the median nerve resulting in ischemia.[8]

There are many lessons to be learned from this event. Rounding on this patient in the PACU allowed for quick identification of the injury and follow up with neurology was initiated.[6] The patient's concerns were also address and she communicated with both of her anesthesia practitioners before discharge to the floor. A neurologist saw the patient twenty-seven hours after the initial report of injury and followed up every day until discharge when the patient reported resolution of symptoms.

Peripheral nerve injuries occur even when optimal patient positioning is utilized and documented. Anesthesia professionals can only attempt to mitigate the occurrence and document adherence to professional standards. In this case, it may be advisable to alternate the arm for blood pressure measurement for patients in the prone position, or any patient at increased risk of nerve injury, such as those with underlying neuropathies. In summary, it is often hard to pinpoint the cause of peripheral nerve injuries, but, by identifying a patient's specific *comorbidities* preoperatively, limiting intraoperative factors, and initiating prompt treatment upon recognition of injury we can better protect and care for our patients.

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Kartagener's Syndrome and Video Assisted Thoracoscopy Surgery

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Keywords: Kartagener's syndrome, primary ciliary dyskinesia, situs inversus totalis, video assisted thoracoscopy surgery, dextrocardia

Kartagener's syndrome (KS), a form of primary ciliary dyskinesia (PCD) is a rare inherited autosomal recessive disorder that is characterized by deficient ciliary motility, situs inversus totalis (SIT), and defects in fertility.[1] Structurally and functionally defective cilia leads to a host

of clinical manifestations that are relevant to the anesthesia practitioner, including recurrent upper and lower respiratory tract infections, paranasal sinusitis, and bronchiectasis.[1]

Case Report

A 35-year-old, 47 kg, 165 cm Caucasian female was scheduled for video assisted thoracoscopy surgery (VATS) and decortication of the left lung for a loculated pleural effusion. Her medical history included a diagnosis of KS, with secondary recurrent bouts of sinusitis and pneumonia. The patient was admitted for treatment of pneumonia and bronchiectasis. A left sided chest tube was placed 5 days prior to surgery to decrease fluid accumulation around the lung. An additional history of methamphetamine abuse was noted, with last ingestion being 2 weeks prior to surgery date. Inpatient medications included vancomycin 1 g every 8 hours, albuterol/ipratroprium bromide nebulizer treatment every 4 hours, piperacillin/tazobactam 3.375 Gm every 8 hours, and subcutaneous heparin 5,000 units, every 8 hours.

Laboratory values included a white blood cell count of 11.4 k/mm^3, which was decreased from 22 k/mm^3 on admission. Hemoglobin and hematocrit were 8.8 g/dL and 27.7%, respectively. An arterial blood gas assessment revealed a partially compensated respiratory acidosis with a pH of 7.34, pCO_2 53.7 mm Hg, PO_2 of 136.2 mm Hg, HCO_3 of 29.2 mEq/L, and SpO_2 of 99.4%. Preadmission medications included an albuterol inhaler and ibuprofen. A 12-lead electrocardiogram (ECG) showed sinus tachycardia with right axis deviation, and the chest x-ray revealed SIT, dextrocardia, and left pleural effusion. Preoperative vital signs were normal. Auscultation revealed distant precordial heart sounds and diminished breath sounds in the left lung fields. The patient's SpO_2 was 95% on 2L/min via nasal cannula with no complaints of shortness of breath.

Upon arrival to the operating room, preoxygenation via the anesthesia circuit with O_2 at 10 L/min commenced. Standard monitors were applied; however, ECG leads were applied in the opposite direction due to the patient's diagnosis of dextrocardia. Ancef 2 g intravenous (IV) was administered followed by an IV induction with fentanyl 100 mcg, 1% lidocaine 50 mg, propofol 120 mg, and rocuronium 50 mg. Due to the diagnosis of SIT, the trachea was intubated using a 37 french left sided endotracheal tube. After the tube was advanced through the glottis, the bronchial lumen was placed in the right mainstem bronchus due to the mirror image reversal of pulmonary anatomy that occurs with SIT. Correct placement was verified via auscultation and fiberoptic bronchoscopy. An arterial line was placed in the right radial artery and a triple lumen central line was placed in the left subclavian vein using ultrasound guidance. The patient was placed in right lateral position, and placement of the double lumen tube was verified a second time with the fiberoptic bronchoscope after positioning. General anesthesia was maintained with 2% sevoflurane inspired concentration in O_2 2 L/min. Dexamethasone 4 mg and ondansetron 4 mg were administered IV for prevention of postoperative nausea and vomiting. The patient remained hemodynamically stable during the procedure. Emergence, awake extubation, and the postoperative period were uneventful.

Discussion

Kartagener's Syndrome is a disease that originates during the embryonic development phase, and is a result of defects in certain proteins that are responsible for rotation and siting of the internal organs.[4] Ciliary function in the mouse node, a structure that is responsible for the symmetric positioning of vital organs is what influences the flow of these proteins to the correct side of the embryo.[4] Since the genes that code for normal ciliary function are defective, the proteins cannot migrate to the correct side of the embryo to exert their function of correct positioning and rotation of the organs.[4] As such, defective ciliary function and transposition of the vital organs are linked.[4]

In regards to thoracic surgery in a patient with KS, there are many anesthetic implications that the practitioner must consider. The preoperative chest x-ray of a patient with KS with SIT will yield an anatomical right lung in the left hemithorax, and the left lung in the right hemithorax, as evidenced by the acute angle of the right main bronchus situated on the opposite side of the chest as well as dextrocardia with a rightward pointing cardiac apex.[5] As such, apical heart sounds are best heard to the right of the sternum, a fact that is imperative when assessing the presence of murmurs.[4] The ECG of a patient with Kartagener's syndrome will characteristically show marked right axis deviation, negative p-wave (because the right atrium is located on the left) in lead I and aVL, and low voltage QRS complexes in the precordial leads.[3,5,6] In order to obtain an accurate ECG, the limb leads should be reversed and the precordial leads should be placed on the right chest.[3] Furthermore, in the event that advanced cardiovascular life support is needed, the chest pads should be placed on the right side of the chest.[3,7] Additionally, any respiratory infections should be treated with antibiotics in the preoperative period and chest physiotherapy should be instituted.[6] Perioperative lung mobilization is an important component in maintaining pulmonary integrity in patients with KS.[6] Bronchodilators, incentive spirometry, postural drainage, and a steroid regimen have also shown to be helpful in prevention and management of respiratory complications.[3,6]

Careful selection of the correct double lumen endotracheal tube is also an important consideration for the nurse anesthetist. In patients with SIT undergoing VATS on the left lung, which anatomically is the tri-lobed lung, the double lumen endotracheal tube should be rotated to the right, placing the bronchial lumen in the right bronchus of the bi-lobed lung.[6] However, a patient undergoing surgery on the right lung will require a right sided double lumen tube that is rotated to the left.[6] A univent tube with a bronchial blocker is another consideration for management of one lung ventilation in patients with KS.[6]

The transposition of the thoracic organs also creates alterations in the vasculature, posing a great challenge to the anesthesia practitioner attempting to place a central line.[3,6] Placement of a right subclavian central line may be difficult in a patient with KS as the catheter must cross midline to reach the intended location above the right atrium, which is located on the left side in these patients.[3] As a result the most direct route for subclavian line placement is from the left side of the thorax.[3] A left internal jugular central venous catheter was placed in this case as it is the preferred route for central venous cannulation, because it is more direct and decreases incidence of injury to the thoracic duct.[6] Nonetheless, ultrasound guidance should be used for the

placement of not only central venous catheters, but also arterial and central venous catheters due to unpredictable vascular course.[3,6]

Finally, patients with KS require humidification of the anesthesia circuit, and repeated suctioning of the endotracheal tube may be essential.[3] Although a heat and moisture exchanger (HME), active humidifier or coaxial circuit would have been a good option to maintain pulmonary integrity, one was not used in this case. Due to chronic sinusitis, instrumentation of the nasal passages with nasal airways or nasal intubation should be avoided. Due to the high risk for pulmonary complications and prolonged intubation, regional anesthesia should be considered whenever possible. A thoracic epidural, although not employed for this patient, is a viable option to consider for anesthesia and pain management in patients with Kartagener's who are undergoing thoracic surgery.[3]

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Anesthetic Management in a Patient with Malignant Hyperthermia Susceptibility

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Keywords: Malignant hyperthermia, triggering agents, MH, anesthesia machine flushing

Malignant Hyperthemia (MH) is a rare inherited autosomal dominant disorder of calcium regulation in muscle tissue.[1] Triggering events are usually related to exposure to halogenated anesthetic gases or a depolarizing neuromuscular blocker. Persons exhibiting MH will go into a hypermetabolic state often exhibiting increased end tidal CO_2 ($ETCO_2$), tachycardia, hyperthermia and metabolic acidosis. The condition can be fatal, and the mortality of MH is estimated to be 5%.[2,3] The incidence is thought to be between 1:10,000 to 1:250,000 anesthetics.[3]

Case Report

A 69-year-old, 93.5 kg, 181 cm male with hydrocephalus presented for an endoscopic third ventriculostomy via right frontal approach. He had a known history of ventriculomegaly consistent with aquaductal stenosis, previously diagnosed in 1997 from an MRI after a motor vehicle accident (MVA). He also suffered a herniated disk from the MVA and subsequently underwent a C5/6 fusion. The patient had recently exhibited progressive worsening of symptoms including gait instability, irritable moods and memory dysfunction. Other past medical history included seasonal allergies, erectile dysfunction, Lyme disease, babesiosis, prostate cancer status post prostatectomy, and obstructive sleep apnea, for which he used a bilevel positive airway pressure machine. Current medications were cetirizine, montelukast, memantine, naproxen, tadalafil, and triamcinolone inhaler.

The patient had a family history of a sibling with an MH event. In 1996 the patient had a MH muscle biopsy test, which displayed susceptibility for the condition. In preparation for his procedure the MH guidelines for the Brigham and Women's Hospital Anesthesia Department were implemented[4]. These actions included removing the vaporizers from the anesthesia machine, changing the breathing circuit and soda lime container, and installing Vapor-Clean (Dynasthetics, Salt Lake City, UT) activated charcoal filters on the expiratory and inspiratory outlets of the ventilator. The reservoir bag was removed from the manual ventilation hose and placed on the breathing circuit Y. The ventilator was placed into volume control (VC) mode, with oxygen at 10 L/min, tidal volume 600 mL and a rate of 10/min. The machine cycled with these settings for approximately 45 minutes prior to the start of the case.

The patient was preoxygenated via facemask with oxygen at 10 L/min. Intravenous induction of anesthesia was achieved with the administration of fentanyl 100 mcg, propofol 200 mg, and rocuronium 50 mg. The trachea was uneventfully intubated with a Macintosh 3 blade, and an 8.0 endotracheal tube was positioned at 22cm at the lip. The patient was placed on a ventilator in VC mode, with a mixture of O_2 1 L/min and air 1 L/min. General anesthesia was maintained with propofol 100 mcg/kg/min and remifentanyl 0.125 mcg/kg/min. He received levetiracetam 500 mg intravenously per the surgical team's request. A temperature probe was placed in the patient's nasopharynx, and he was warmed by a forced-air warming device during the case. The procedure went as planned and the patient was uneventfully extubated 78 minutes after intubation. His pain was well controlled in the recovery area. He was transferred to the patient unit for overnight observation and was discharged the next day.

Discussion

MH susceptible patients who are exposed to trigger substances can develop skeletal muscle hypermetabolism in which Ca^{2+} ions are released from the cell's sarcoplasmic reticulum (SR) in an uncontrolled fashion. This causes myoplasmic levels of Ca^{2+} to rise rapidly and activate prolonged myofibril contractions.[1] Prolonged contraction of the muscle fibers causes increased use of oxygen and glucose, depletion of adenosine triphosphate, and increased production of carbon dioxide and heat.[1] Eventually the sustained cellular stress leads to breakdown of the cell membrane and rhabdomyolysis, leading to subsequent release of cellular contents into the bloodstream, creating hyperkalemia and myoglobinuria.[1-3] Disseminated intravascular

coagulation occurs frequently if the patient is hyperthermic above 40°C. The main gene responsible for MH is the ryanodine receptor type 1. However, the exact process in which the triggering agent exposure causes uncontrolled release of Ca^{2+} from the SR remains elusive.[1] Commonly recognized trigger substances include halogenated inhalation agents, ether, and the depolarizing neuromuscular blocking agent succinylcholine.[5] There are case reports implicating statins, tetracaine, ondansetron and methylene blue with MH, but the evidence is minimal.[5] In view of the fact that some MH susceptible patients have multiple exposures to trigger agents before their first MH episode, it has been suggested that certain agents administered around the same time as the trigger agents had a protective effect decreasing the probability of an MH event.[4] These drugs include thiopental and non-depolarizing neuromuscular blocking agents.[5]
MH can be difficult for clinicians to recognize. The clinical presentation is often variable: early signs often include sinus tachycardia, an unexplained increase in $ETCO_2$ and masseter rigidity.[1] Other signs include hyperthermia, metabolic acidosis and rhabdomyolysis.[1] MH is more prevalent in males and has been reported most frequently in those under 18 years old. Many patients who are MH susceptible had non-eventful exposures to triggering agents in the past.[3,5,6] In fact, it takes an average of three exposures before MH occurs.[3]
MH is diagnosed either by lab testing or clinical diagnosis. The most commonly used lab test to diagnose MH is the caffeine halothane contracture test where a biopsied muscle fiber is exposed to halothane and caffeine and its contractions are measured.[1-3] A person is considered susceptible to MH if there is a contraction reaction to halothane or caffeine; they are considered not susceptible if there is no reaction to either.[1]
The cornerstone of successful treatment of MH is early recognition. Once recognized, all triggering agents should be discontinued immediately, vaporizers should be removed from the anesthesia machine, the patient should be placed on 100% oxygen with high gas flows and hyperventilated.[3,5-7] Active cooling should be initiated if indicated and acid/base or electrolyte disturbances should be corrected.[3,5-7] Dantrolene therapy should be started immediately as dantrolene is the only approved drug to treat MH. It should be dosed at 2.5 mg/kg every 5 minutes up to a maximum dose of 10mg/kg.[1-3,5-7] Dantrolene works as a ryanodine receptor antagonist, which slows the abnormal release of Ca^{2+} from the SR.[1] If surgery is to continue the patient should be managed on non-triggering agents.[1]

For our patient, the anesthesia plan was to only use non-triggering agents and to follow hospital protocols regarding preparing the anesthesia machine for use with an MH susceptible patient. The goal for the preparation of the anesthesia machine is to washout any residual halogenated agents to a concentration of <5 parts per million, which is achieved by the flushing procedure and adding the activated charcoal filters to the expiratory and inspiratory outlets of the ventilator. Our patient displayed no clinical signs of MH. His $ETCO_2$ never went above 40, his HR was about 70 for much of the case, only rising around the time of emergence, he displayed no muscle rigidity and his temperature only increased from 36.0 °C to 36.2 °C during the case. Having an MH protocol in place and taking every precaution is vital to minimizing any risk of triggering an MH episode.

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Limiting Opiates during Colorectal Surgery under an ERAS Protocol

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Keywords: ERAS protocol, ketamine infusion, idocaine infusion, transversus abdominis plane block, TAP block, abdominal surgery, multimodal analgesia

The Enhanced Recovery after Surgery (ERAS) protocol is a multimodal perioperative regimen developed to reduce perioperative complications which may lead to postoperative morbidity and increased hospitalization.[1] Under the ERAS protocol, consideration for alternative pain management is imperative. Perioperative opioid administration can lead to undesired outcomes that prevent discharge such as; nausea and vomiting (N/V), postoperative ileus, and respiratory depression.[2] It is important to consider a multimodal pain regimen that targets different pain pathways to limit perioperative opioid use such as intraoperative ketamine and lidocaine infusion, or a transversus abdominis plane (TAP) block.

Case Report

A 47-year-old, 64 kg, 183 cm male presented for an exploratory laparotomy with a Roux-en-Y hepaticojejunostomy and a loop gastrojejunostomy for the treatment of biliary and gastric outlet obstruction. The patient's medical history included gastroesophageal reflux disease, chronic alcoholic pancreatitis, alcohol abuse, hypertension, and tobacco use. His surgical history included a cholecystectomy. Recent laboratory values for this patient were unremarkable. Recurring episodes of pancreatitis, due to his gastric obstruction, had led to multiple and recent hospitalizations.

The ERAS protocol, used for specific open abdominal procedures, was being trialed at the facility where the patient's surgery was to take place. Keeping the ERAS protocol in mind, the patient was instructed to stop any oral intake of food eight hours prior to surgery. He was then instructed to drink 12 ounces of Gatorade 2 hours before the start of his procedure. The morning of his surgery, the patient received oral pregabalin 75 mg, celecoxib 400 mg, and acetaminophen 1000 mg.

Once in the operating room, electrocardiogram , noninvasive blood pressure, and pulse oximetry monitors were applied. A subarachnoid block (SAB) was administered using isobaric bupivicaine 5 mg, and preservative free morphine sulfate 100 mcg. After administration of the SAB, the patient was positioned supine and preoxygenated with O_2 10L/min via bag mask. General anesthesia was induced intravenously (IV) and endotracheal intubation was achieved via a rapid sequence induction. Fentanyl 50 mcg, ketamine 30 mg, propofol 200 mg, and succinylcholine 160 mg IV were administered to induce general anesthesia and to provide neuromuscular blockade. A direct laryngoscopy was performed with a Miller II blade exposing a grade I view. A 7.5 mm oral endotracheal tube (ETT) was placed without difficulty and secured at 22 cm at the teeth once breath sounds were auscultated evenly, bilaterally, and end tidal CO_2 was confirmed. Rocuronium 50 mg IV was administered after peripheral nerve stimulation demonstrated recovery of four out of four twitches from the succinylcholine. Rocuronium was administered throughout the case to maintain neuromuscular blockade at 1 to 2 out of 4 twitches.

A right radial arterial line and right internal jugular central line were placed under ultrasound guidance. A baseline blood pressure of 98/65 mm Hg was obtained and correlated with the Cheetah noninvasive cardiac output monitor (NICOM) noninvasive stroke volume management system. Albumin 250 mL was administered IV to assist in the calibration of the stroke volume management system. A change in stroke volume index (SVI) after the bolus test was 11% implying that the patient would be responsive to fluid if needed. A ketamine infusion was started and maintained at 10 mcg/kg/min IV. A lidocaine infusion was also started and maintained at 33 mcg/kg/min IV. These drugs were chosen as a multimodal approach to provide analgesia for the duration of the surgery. General anesthesia was maintained with expired desflurane 6% in O_2 1 L/min, and air at 1 L/min.

Intraoperatively the patient became briefly hypotensive. The Cheetah NICOM, when correlated with a blood pressure of 77/58 mm Hg, gave a total peripheral resistance value of 720. The patient's CVP was 5 mm Hg. Stroke volume based on these values was deemed to be adequate so a phenylephrine infusion of 30 mcg/min IV was initiated in order to increase afterload. The phenylephrine infusion was discontinued once the patient was hemodynamically stable.

Over the 6-hour duration of the procedure, the patient received 850 mL of lactated ringer's solution IV and his estimated blood loss (EBL) was 700 mL. Thirty minutes prior to surgical closure, the lidocaine and ketamine infusions were discontinued. Once the procedure was complete and the surgical dressing was applied, bilateral TAP blocks were administered under ultrasound guidance using bupivacaine 0.25% 20 mL divided equally on each side. A transverse abdominis plane peripheral nerve block catheter was also placed to provide continuous pain

management throughout the postoperative period. Once the blocks were placed, neuromuscular blockade was reversed with neostigmine 3 mg and glycopyrrolate 0.4 mg IV. Ondansetron 4 mg IV was also given to prevent postoperative nausea and vomiting. The desflurane was discontinued and the O_2 was increased to 10L/min. Train of four was assessed and the patient had 4 out of 4 twitches without fade. Once the patient was awake, following commands, and initiating regular tidal volume breaths greater than 400 mL, the ETT was removed without complication. He was then transferred to the postoperative care area on O_2 3 L/min via nasal cannula.

Discussion

The anesthetic management of patients undergoing open abdominal colorectal surgery can be challenging. Depending on the complexity of the case, these procedures have the potential to be longer than 4 hours which challenges the anesthetist to consider appropriate pain management. Excessive opioid administration during these procedures may not be uncommon. Excessive administration of opioids during these procedures can lead to postoperative respiratory depression, delayed emergence, and long term effects such as postoperative ileus. The ERAS protocol is a clinical pathway used to avoid these potential negative outcomes. ERAS protocols may vary among institutions.[3] Some institutions incorporate an epidural anesthetic whereas others, like the institution in this case report, utilize intrathecal anesthesia.[3] There are benefits to both pathways. Given that this patient did not have an epidural catheter, it was beneficial to incorporate other ways of providing analgesia that would not affect emergence from general anesthesia or delay postoperative progression. Given these variables, the addition of a ketamine and lidocaine infusion was helpful in providing a steady level of multimodal analgesia that did not seem to affect progression of recovery and would have benefited pain control in the early postoperative period. The bilateral TAP blocks were helpful in providing pain relief postoperatively because the SAB of intrathecal morphine placed preoperatively may have been dissipating given the long duration of the procedure. The peripheral nerve catheter was also connected to a patient controlled analgesia (PCA) pump once in the postoperative care area to provide extra localized pain relief to the segmental nerves within the plane of the surgical site.

Ketamine interacts with many receptors in the body but is mainly known by its antagonistic effects at the N-methyl-D-aspartate (NMDA) receptor which can cause profound analgesia and amnesia. Used in a multimodal perioperative pain regimen, it can significantly reduce postoperative analgesia requirements.[4] The infusion dose of intraoperative ketamine can vary. One review defined low dose ketamine infusion as being less than 1.2 mg/kg/hr but found that most drips were infused at a rate of 0.18 mg/kg/hr or less.[5] The IV infusion rate for the patient in this case report was 0.01 mg/kg/min which is consistent with those findings. Studies suggest that an intraoperative ketamine infusion can reduce postoperative opioid consumption by as much as 40%.[5] Furthermore, the intraoperative administration of ketamine has been shown to reduce the need for opioids in chronic opioid consuming patients within the first 24 to 48 hours postoperatively.[6] The use of ketamine would arguably help promote early patient mobility and reduce the incidence of postoperative ileus.

Intravenous lidocaine infusions have shown to provide perioperative analgesia in patients undergoing intraabdominal surgery. Lidocaine blocks pain transmission along neuronal pathways, particularly in the dorsal horn.[7] It also demonstrates anti-inflammatory effects by inhibiting the release of inflammatory mediators and free radicals.[7] Intraoperative IV lidocaine infusions may also help decrease the incidence of postoperative ileus formation by blunting the inflammatory stress response, promoting earlier hospital discharge. Lidocaine has also been shown to have a positive effect on gastrointestinal motility.[7] Therefore, IV lidocaine infusions may be a good anesthetic adjunct when weighing acute pain management options under ERAS protocol guidelines.

A transversus abdominis plane (TAP) block is achieved when a local anesthetic agent is injected into the lateral abdominal wall between the internal oblique and the transversus abdominis muscles. This is meant to block the segmental nerves (T9-12 and L1) within this plane.[8] This can effectively anesthetize the upper abdomen and provide pain relief. One systematic review compared nine studies that included 413 patients. Results indicated that patients who received a TAP block had a significant reduction in morphine consumption within the first 24 hours postoperatively.[8] The patient in the case report received a continuous TAP block via a peripheral nerve catheter which provided analgesia beyond the first 24 hours postoperatively. Opioid induced side effects were avoided within the first 48 hours using this technique.

The multimodal approach of using IV ketamine and lidocaine infusions for this patient was successful in reducing the amount of perioperative opioid administration. The patient received a single dose of fentanyl 50 mcg IV which was administered during induction. He emerged at the end of the case without incident and did not have any postoperative complications. Most of the studies that have reviewed the benefits of multimodal analgesia methods, such as IV ketamine/lidocaine infusions and the administration of TAP blocks, are compared to the postoperative administration of morphine. Those studies are focused on the first 24 to 48 hours postoperatively. The facility in which this surgery was performed utilizes hydromorphone more frequently than morphine. The patient did require hydromorphone 2.5 mg IV for pain within the first 24 hours postoperatively. Although the patient did require additional IV opioid administration for pain, the amount required was insignificant in comparison to those patients who did not have a multimodal approach to pain management.

Both IV ketamine and lidocaine infusions have proven beneficial during colorectal surgery. They provide a proven clinical pathway for acute postoperative pain management when administering anesthesia to a challenging patient in a complex case. When compared to the use of opioids alone, there is a decreased incidence of respiratory depression and postoperative ileus. TAP blocks are also a good alternative to postoperative opioid administration in this patient population. They can provide analgesia without the undesirable side effects of opioid administration. Therefore, these multimodal analgesia pathways are invaluable when implementing an ERAS protocol.

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Management of Morquio Syndrome in a Repeat Caesarean Section

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Keywords: Morquio syndrome, mucopolysaccharidosis type IV, caesarean section

Mucopolysaccharidosis type IV, known as Morquio syndrome, is an autosomal recessive lysosomal storage disorder that occurs in 1:76,000 to 1:640,000 live births.[1] People with Morquio syndrome lack enzymes to break down long-strand sugar molecules, resulting in a build-up of glycosaminoglycans (GAG) that can lead to organ damage.[1] The most prominent features of Morquio syndrome are musculoskeletal abnormalities, including short stature, short neck, kyphoscoliosis, genu valgum, hypermobile joints, and pectus carinatum.[1] Atlanto-occipital instability and restrictive lung disease, two of the most serious repercussions of the disorder, can create a significant challenge for the anesthetist. This case report summarizes the care of a patient with Morquio syndrome during a repeat cesarean section (c-section).

Case Report

A 24-year-old, 147 cm, 78 kg female presented for a repeat c-section. The medical history included Morquio Syndrome but was otherwise unremarkable. Medications included prenatal vitamins. Surgical history included a previous c-section that was complicated with pulmonary edema due to fluid overload, which required fiberoptic tracheal reintubatation postoperatively on the labor and delivery unit.

Airway assessment revealed widely spaced teeth with a thyromental distance of two fingerbreadths. A Mallampati Classification II was assigned. Limited neck mobility was noted. Lung sounds were clear to auscultation bilaterally and the spine revealed kyphoscoliosis. The patient expressed anxiety related to awake airway management and requested an asleep intubation and general anesthesia.

The anesthetic plan was general anesthesia based on the current recommendations to avoid neuraxial anesthesia in Morquio patients.[1] Midazolam 2 mg intravenous (IV) was administered preoperatively. On arrival to the operating room, standard monitors were applied, including pulse oximetry, non-invasive blood pressure, and EKG monitors. Room air SpO_2 was 98%, pre-oxygenation was accomplished via face mask with O_2 10 L/min for an SpO_2 of 100% until an end tidal O_2 concentration of 90% was achieved. Propofol 200 mg IV and succinylcholine 100 mg IV were administered with application of cricoid pressure. To decrease manipulation to the cervical spine, a Glidescope (Verathon Inc., Seattle, WA) was used for laryngoscopy. The student nurse anesthetist noted an edematous and narrowed glottis, and atraumatically intubated the patient with a 6.5 endotracheal tube (ETT) after two attempts.

Anesthesia was maintained with sevoflurane 2% inspired concentration in O_2 2 L/min. During the procedure, the SpO_2 was 94% on volume controlled ventilation with an end tidal carbon dioxide between 35 and 40 mm Hg, tidal volumes of 560 mL, a rate of 12/min, and positive end expiratory pressure of 5 cm H_2O.

Once the fetus was successfully delivered, fentanyl 150 mcg IV was administered. . Sevoflurane was titrated to off during closure of the abdomen. Spontaneous ventilation occurred, and the trachea was extubated once the patient demonstrated movement of all four extremities and extubation criteria were met. She received O_2 8 L/min via simple face mask, and transfer to the high risk labor post anesthesia care unit (PACU) was achieved successfully, as evidenced by stable vital signs.

Discussion

The most important anesthesia related implications of Morquio syndrome involve restrictive airway disease related to kyphoscoliosis, as well as tortuous and narrowed trachea and bronchi, and atlanto-occipital instability. Patients with Morquio syndrome are known to have difficult airways, and their presence in the operating room merits a careful and thorough airway examination. This should include a physical examination, consultation with a physician who follows the patient, and recent radiograph or magnetic resonance imaging (MRI). Recent imaging is essential because patients with Morquio syndrome tend to experience worsened

airways as they get older. In a retrospective study examining the MRIs of 28 patients diagnosed with Morquio A syndrome, 19 had tracheal narrowing.[2] MRIs Tracheal narrowing progressively worsened with age, as all 8 subjects over the age of 15 had greater than 50% tracheal narrowing.[2]

The anesthesia team should plan for a difficult airway, and ensure surgery is scheduled with adequate support. Preoperative pulmonary and otolaryngologic consultation is highly recommended. [3] Theroux et al[3] suggest video laryngoscopes, such as the Glidescope (Verathon Inc, Bothell, WA) are the most effective difficult airway adjuncts to intubate patients with Morquio syndrome. Manually displacing the tongue with a gauze or Magill forceps prior to insertion of the Glidescope will significantly enhance larynx visualization.[3] Video laryngoscopy is considered more appropriate for patients who have had cervical spine stabilization surgery. For patients with an unstable cervical spine due to occipitocervical instability, an awake fiberoptic intubation should be the first choice.[3] Our patient, who had been followed closely since being diagnosed with Morquio syndrome at a children's hospital, had been cleared for surgery after radiographic evidence revealed only a mildly hypoplastic dens with no previous occipitocervical stabilization. The anesthesia team agreed that video laryngoscopy was an acceptable approach to intubation to make certain that the patient's neck was not manipulated.

The video laryngoscopy revealed a narrowed, edematous glottis. The patient's airway narrowing was complicated by pregnancy, and therefore a 6.5 internal diameter (ID) ETT was chosen with a 6.0 and 5.5 ID ETT available as a back up. It is imperative with this patient population to have multiple ETTs with two half-size internal diameters below the chosen ETT. The anesthetist should also have multiple airway adjuncts available, and if possible, an anesthesia practitioner who is experienced in the management of patients with Morquio syndrome.[4] A preoperative assessment of the patient's cricothyroid membrane will allow the anesthetist to prepare for a cricothyrotomy in the event that the patient cannot be ventilated or oxygenated.

Regional anesthesia might appear to be a better choice during caesarean section, due to the multiple airway considerations among patients with Morquio syndrome. However, Drummond et al[5] concluded epidural anesthesia as relatively contraindicated for patients with Morquio syndrome due to their vulnerability to spinal cord ischemia. Drummond et al[5] described a case report of paraplegia in a patient with Morquio syndrome after an epidural-general anesthetic was administered. Authors advised against epidural anesthesia due to potential difficulty in maintaining adequate blood pressure control and possible delay in diagnosis. Since the mechanism behind the paraplegia related to epidural anesthesia has not been causally linked, and since evidence on the use of spinal anesthesia in Morquio syndrome is insufficient in general, our anesthesia team decided to avoid spinal anesthesia.

Morquio patients are living longer and experiencing improved quality of life due to advances in symptom management and enzyme replacement therapy.[1] As a result, they increasingly present for procedures beyond pediatric health systems and medical and surgical management of Morquio syndrome is evolving. Consultation with their long-term managing physicians is helpful in planning a successful anesthetic. Careful preoperative assessment must include an up-to-date cervical spine radiograph or MRI, a thorough airway assessment including the position of the cricothyroid membrane, and the availability of multiple airway adjuncts. By understanding

Morquio syndrome, the anesthetist will be better able to plan an anesthetic that avoids dire airway emergency and ensures a seamless intraoperative course.

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General Anesthesia for a Pediatric Patient with History of Preterm Delivery

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Keywords: pediatric anesthesia, former preterm infant, reactive airway, laryngospasm

Preterm infants are defined as those born before 37 completed weeks of gestation. Increased rates of long-term neurodevelopment impairment and chronic respiratory problems result in the potential for increased intervention when providing anesthesia.[1] This case report describes the events of a former preterm (FPT) 6-year-old female who presented for bilateral tympanostomy tube placement, tonsillectomy and adenoidectomy. Upon arousal, after extubation, she began coughing to the extent of an oxygen desaturation event lasting >45 seconds. This patient demonstrated indicators of a reactive airway, in connection to being a FPT infant, prior to surgery.

Case Report

The patient was a 6-year-old female, weighing 22.7 kg and 113 cm tall, who presented for bilateral tympanostomy tube placement, tonsillectomy, and adenoidectomy. Her mother reported significant history of frequent ear infections, up to 6-7 in a year. Chronic nasal obstruction, significant sleep-disordered breathing at night, frequent snoring, constant mouth breathing, and frequent dysphagia were also reported. The patient was born at 32 weeks gestation and spent 6 weeks in the neonatal intensive care unit (NICU). She did not require intubation at that time. The mother reported frequent upper respiratory illnesses with a croupy cough requiring emergency

room treatment with IV steroids. Hospital admission has not been required. The patient was otherwise healthy, denied drug allergies, and took no medications at home.

General anesthesia was induced by mask with a mixture of oxygen at 3 L/min and nitrous oxide at 7 L/min, and sevoflurane inspired concentration of 6% without adverse event. Immediately after induction the nitrous oxide was discontinued and total flow was decreased to 2 L/min. A peripheral IV line was started in the left wrist and the IV medications propofol and fentanyl were administered at doses of 100 mg and 40 mcg respectively to prepare the patient for endotracheal intubation. A cuffed 4.5 mm inner diameter endotracheal tube (ETT) was then easily placed under direct laryngoscopy and an air leak was confirmed while holding 20 cm H_20 positive pressure after inflation of the cuff with 1.75 mL of air. The inspired concentration of sevoflurane was adjusted to maintain an age-adjusted minimum alveolar concentration of 1.4 until emergence. The surgeon proceeded with the bilateral tympanostomy tube placement and then moved quickly to the tonsillectomy and adenoidectomy. The patient tolerated the entire procedure well, was mechanically ventilated with pressure support ventilation at a rate of 14-22/min and only required fentanyl 10 mcg during maintenance of anesthesia.

After the surgeon had completed all procedures, the patient was allowed to breathe spontaneously with O_2 13 L/min. She was gently suctioned to remove any secretions from the oropharyngeal cavity. While breathing spontaneously the patient drew tidal volumes of about 150-160 mL with a respiratory rate of 15/min. When her end-tidal concentration of sevoflurane fell to 0.3%, she responded to voice commands and when lightly touched, she opened her eyes and began to cough and reach for the ETT. She was then extubated and a mask connected to the anesthesia circuit delivering 12 L/min of oxygen was placed on her face. She continued to cough and despite taking tidal volumes in the low 100's in between coughing, her SpO_2 decreased to 84%. The anesthetist began assisting the patient's spontaneous ventilation with the bag on the anesthesia circuit. After about 45-60 seconds her SpO_2 rose to 97% and the patient's coughing episode subsided. She was transitioned to a simple oxygen mask and monitored for adequate ventilation and oxygen saturation while being transferred to the post-anesthesia care unit. No further interventions were required for her airway or pulmonary status.

Discussion

The Centers for Disease Control and Prevention report that in the United States about 450,000 children are born prematurely each year.[2] Complex medical issues such as neurologic disabilities, respiratory issues, feeding dysfunction, and developmental delays are shown to be common in this population.[2] Prematurity itself is a dominant risk factor for lung injury and long-term impairment of pulmonary function. Children born prematurely are at higher risk than those born at term for bronchopulmonary dysplasia, reactive airway disease, pulmonary artery hypertension, chronic obstructive lung disease, and associated complications.[1] These medical issues frequently result in the need for diagnostic tests and surgical procedures that require sedation or general anesthesia.[2] The extent of classic research states that this population of preterm infants and children up to the age of 3 years more commonly experiences perioperative respiratory adverse events (PRAE) when receiving sedation or general anesthesia.[1,3]

One goal of induction of anesthesia in the patient at risk for PRAE is to minimize the possibility of a bronchoconstrictive response when providing airway management.[4] It is generally accepted that induction should provide a level of anesthesia deep enough to prevent any physiologic response to airway manipulation. When possible and tolerated by the child, IV induction is recommended rather than inhalation induction for children at risk for PRAE.[4] In a single-center randomized controlled trial, children with risk factors for reactive airway disease who were induced with sevoflurane had a higher incidence of PRAE compared with children who received IV induction with propofol.[5] Nagelhout attests that "nitrous oxide and sevoflurane are considered the standards because of the low incidence of breath holding, coughing, secretions, and laryngospasm encountered during inhalational induction."[6] Sevoflurane is generally preferred during maintenance of anesthesia because it is considered to be the most effective bronchodilator.[4] When formulating a plan for emergence and extubation, it should be noted that in a single-center randomized study it was discovered that, among term children, awake removal of the ETT has been shown to increase the incidence of persistent coughing and oxygen desaturation, while deep removal of the ETT increases the incidence of partial airway obstruction.[7]

The age at which former preterm infants are no longer considered high risk is difficult to determine.[2] One recent study examined the records of 57,628 patients ranging in age from birth to 22 years who had undergone sedation or general anesthesia.[2] Findings indicated that 14.7% of preterm and FPT children experienced adverse events compared with 8.5% of children born at term.[2] Oxygen desaturation (4.5%), coughing (3.4%), and airway obstruction (2.9%) were the most common adverse sedation or general anesthesia events reported in the FPT group.[2] Amid FPT patients the highest percentage of adverse events were seen between 10 and 13 years of age, but with adverse effects occurring up to the age of 22 years.[2]

The longitudinal risk analysis of patients from birth to 22 years of age suggests that the aforementioned patient's episode of coughing and resulting period of oxygen desaturation may have been related to her having been an FPT infant. Her frequent upper respiratory illnesses and chronic "croupy cough" were history enough to warrant increased vigilance for PRAE.

General anesthesia and sedation in preterm infants and children place the patient at an increased risk for PRAE.[3] Close attention to airway and pulmonary details, collaboration between the surgeon and anesthesia team, and obtaining a complete health history will help ensure successful outcomes.[3] Careful consideration to the patient's airway status prior to emergence will guide the decision to extubate in a deep plane of anesthesia or awake, based on susceptibility to coughing and desaturation or partial airway obstruction. The uniqueness of the claims submitted by the referenced recent publication lies in the singularity of its conclusion.[2] That is, that there exists a much broader range of age than previously established during which patients who were born prematurely remain at risk for adverse anesthesia related events.[2] Additional research and case reports are needed to corroborate the suggested increased vulnerability of FPT patients into their early adulthood years.

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The Vortex Approach: A Novel Cognitive Aid for Use in Difficult Airway Management

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Keywords: difficult airway, algorithm, cognitive, vortex

The difficult airway is a life threatening situation requiring established practice guidelines to ensure efficient effective action, especially in the face of inexperience.[1] To date, the American Society of Anesthesiologists' (ASA) difficult airway algorithm is the standard of care for difficult airway management in the United States.[2] It has been critiqued as complex and inflexible, both significantly detrimental factors in a high stakes airway crisis.[2,6] The Vortex approach is a newly-developed cognitive aid whose simplicity allows for easier implementation and recall.[1] Its goal-oriented approach allows for proficient use not only by inexperienced practitioners, but also multiple disciplines.[1]

Case Report

A 74-year-old, 160 cm, 101 kg Caucasian female arrived for cystoscopy and fulguration for treatment of interstitial cystitis. Her past medical history was significant for hypertension, hyperlipidemia, hypothyroidism, and arthritis. Previous surgeries included a right total hip replacement, tonsillectomy, and colonoscopy. Preoperatively, the patient stated she had a "difficult airway". She was unsure of further details, but was told this after her hip replacement in 2015. No previous anesthesia records were available. Her airway assessment revealed a thyromental distance of approximately 4cm, Mallampati classification of 3, small mouth opening, and full cervical range of motion. Laboratory values included a hemoglobin level of 13.4 g/dL, hematocrit of 45%, and a platelet count of 245 x 10^9/L. A comprehensive metabolic panel showed sodium levels at 142 mEq/L, potassium at 4.2 mEq/L, glucose at 125 mg/dL, and creatinine at 0.98 g/day.

Prior to induction, the difficult airway cart and GlideScope (Verathon Inc., Bothell, WA) were obtained. The patient was ramped with two blankets to facilitate ventilation and intubation. An anesthesiologist, certified registered nurse anesthetist (CRNA), student registered nurse anesthetist (SRNA), and operating room (OR) staff were at the bedside. The patient was pre-oxygenated using a mask strap and O_2 10 L/min for 3 minutes. Intravenous induction was accomplished with fentanyl 50 mcg, 2% lidocaine 60 mg, propofol 150 mg and succinylcholine 120 mg. Ventilation was not attempted prior to the administration of succinylcholine.
The anesthesia practitioner's first intubation attempt using the GlideScope showed a grade IV view and was unsuccessful. The anesthesia practitioner's second intubation attempt, also with the GlideScope, revealed a bloody airway with minimal visualization. The patient's SpO_2 was 85%, as measured by pulse oximetry. Mask ventilation was attempted without success. An oral airway was inserted before the anesthesia practitioner assumed a two-hand mask hold, with manual bag assistance by the CRNA. This technique yielded tidal volumes of no more than 50 mL, resulting in a decline in SpO_2 to 70%. Simultaneously, the fiberoptic scope was prepared for use by the anesthesia staff and circulating OR nurses.

At this stage, versed 4 mg and an additional bolus of propofol 50 mg were given. The CRNA again assumed the two-hand mask hold, with bag assistance by the SRNA. Tidal volumes of approximately 150 mL were achieved, while the patient's SpO_2 remained at 70%. The CRNA then inserted a size 3 laryngeal mask airway (LMA), achieving airway patency as evidenced by tidal volumes of 300 mL and the presence of end tidal CO_2. The patient's SpO_2 increased to 90%. The anesthesia practitioner elected to remove the LMA and perform fiberoptic intubation. After two fiberoptic intubation attempts, the patient's vocal cords were visualized and an endotracheal tube (ETT) was inserted. An end tidal CO_2 waveform was visualized and adequate ventilation confirmed. Cystoscopy and fulguration were performed without complication. The ETT was removed once the patient was fully awake, with tidal volumes of greater than 300 mL and a head lift lasting 5 seconds. She underwent an uneventful recovery.

Discussion

Difficult airways, defined as "cannot intubate, cannot ventilate" situations, are rare and unpredictable, thus research is lacking.[3] The current body of knowledge revolves around case

reports and expert opinions, both of which fall into the lowest levels of evidence-based practice.[3] The action taken is often dictated more so by clinical context, expertise, and facility culture[3] than existing difficult airway algorithms, presumably due to poor recall in high stress situations.[5] As such, a goal-oriented cognitive tool that is both easily executed and universally-applicable becomes invaluable.[1,3] Cognitive aids demonstrate increased performance during anesthetic emergencies.[4] The Vortex is one such aid, offering a pictorial representation of difficult airway management designed for easy recall and application.[1]

During emergency airway situations, the primary goal is alveolar oxygen delivery (AOD), accomplished through both a patent airway and means of oxygen delivery.[1] This is represented by the green zone in Figure 1.[1] Patency is established via surgical or non-surgical routes.[1] Non-surgical airways (NSAs) include a face mask (extraglottic), LMA (supraglottic), or endotracheal tube (transglottic).[1,5] Surgical routes include emergency surgical airways (needle cricothyroidotomy or tracheotomy) and definitive surgical airways (percutaneous, surgical cricothyroidotomy, or tracheotomy).[1]

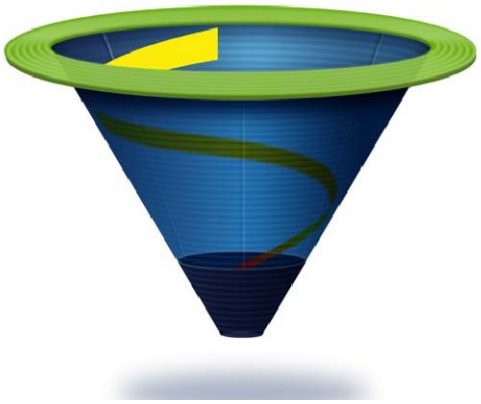

Figure 1. Vortex lateral view[1]

The focal point of the Vortex approach is a funnel-shaped cognitive aid (Figure 1) comprised of three primary sections representing each NSA, as shown in Figure 2.[1,5] This shape is particularly notable as it is nonprescriptive in terms of which NSA the practitioner chooses to initiate.[1]

Faced with a difficult airway, a practitioner's goal is to stay within or return to the green zone.[1] This is accomplished through NSA establishment or alternatively, waking the patient up.[1] Efforts at establishing an NSA must be declared optimal before moving to an alternate NSA.[1] Factors that improve NSA success (Figure 2) should be utilized with each successive try, up to a maximum of three attempts.[1] In the event of NSA failure, practitioners are directed to an emergency surgical airway (ESA), regardless of oxygen saturation.[1] Oxygen desaturation is a late sign of airway compromise.[1] If the patient is already desaturating during NSA attempts, simultaneous preparation for an ESA should be considered.[1] Once an ESA is established, the healthcare team is able to assess whether to wake the patient or convert to a definitive surgical or nonsurgical airway.[1]

The Vortex approach addresses three major concerns found in difficult airway scenarios and case reports.[1] First is the failure to make optimal attempts at all three NSAs as described above.[1] Additional factors to consider when utilizing any NSA are listed alongside the Vortex funnel (Figure 2).[1] These include head and neck manipulations, adjuncts, the size/type of device, the use of suction, and pharyngeal muscle tone.[1]

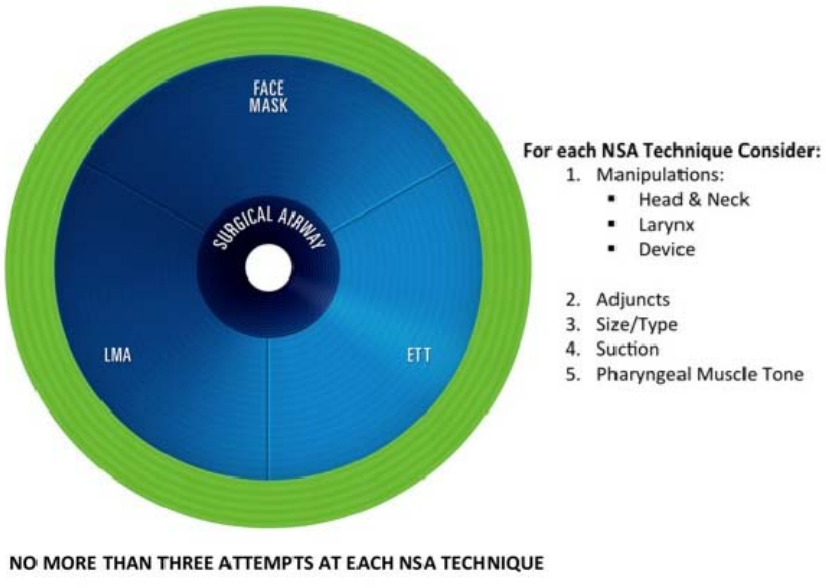

Figure 2. Vortex cognitive aid[1]

By altering subsequent tries, NSA attempts are purposeful, increasing the likelihood of success.[1] A second concern is excessive airway manipulation, leading to compromised AOD.[1] Research shows anesthesia practitioners often attempt the same intervention multiple times.[3] The Vortex addresses this practice by allowing no more than three tries before an effort is declared optimal, with the least amount of tries being ideal.[1] A third concern is the failure to initiate an ESA in time, if at all.[1] The sloping nature of the Vortex emphasizes the rapidity of events and their final outcome.[1]

Existing difficult airway algorithms, such as the ASA and DAS algorithms, are criticized for their complexity, making them difficult to apply under stress.[2,5] They are also solely anesthetic-driven, creating a barrier to team oriented decision making.[1,5] The Vortex approach offers a standardized protocol, much like the Advanced Cardiac Life Support guidelines, meant to prompt staff to anticipate treatment priorities.[1,5] Its inherent simplicity is beneficial to both seasoned caregivers, as well as newer practitioners and students.[6] In a study conducted by researchers at the University of Texas Southwestern Medical Center, 81.8% of anesthesia residents trained in the Vortex approach were better prepared for airway management in "cannot intubate, cannot ventilate" scenarios than those trained in the ASA difficult airway algorithm.[6]

In the aforementioned case study, the healthcare team found themselves in a "cannot intubate, cannot ventilate" situation. The first NSA attempted – the endotracheal tube – was given two identical, unsuccessful tries with the GlideScope, inconsistent with either existing algorithms or the Vortex approach. To make this an optimal attempt, an adjunct (i.e. bougie) could have been used. The second NSA attempt – the face mask – may have been successful with further head and neck manipulations, an alternately sized oral airway or earlier substitution of a different practitioner. Once the LMA – the last NSA attempt – was in place with confirmed AOD, the team could have elected to continue with surgery or to wake the patient up, neither of which was discussed. Lastly, the OR staff was not educated in difficult airway management, in terms of both equipment and course of action. Retrospectively, application of the Vortex approach may have led to team oriented decision making, as well as a safer, more rapid procurement of a definitive airway.

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Placental Increta: A Case Study

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Keywords: placenta, increta, accreta, hysterectomy, uterus

Placental increta, sometimes misunderstood as placental accreta, is the second most common type of abnormal implantation of the placenta to the myometrium, accounting for 17% of all cases.[1] The normal placenta of a parturient implants itself on endometrium, but when implantation occurs on, in, or through the myometrium, it is termed placental accreta, increta, or percreta, respectively.[1] Surgical removal of the placenta or hysterectomy is indicated when any of these abnormal placental implantations occur. Massive hemorrhage is the number one concern for this disease process in the parturient.[1]

Case Study

A 31-year-old, gravida 4 para 3 female presented to the labor and delivery unit with pelvic pain and vaginal bleeding, stating that she was 34 weeks pregnant. Vital signs were as follows: blood pressure 134/79 mm Hg, heart rate 85/min, SpO_2 96%, respiratory rate 20/min, and fetal heart rate 144/min. The patient stated that she was receiving care in Mexico, although no medical records were available. The patient stated that she had no medical history or drug allergies and

her previous three deliveries were by cesarean section via spinal anesthesia. The patient denied any anesthetic or obstetric complications.

After formal sonography, an abnormal placenta was visualized and placental increta was diagnosed. The fetus was normal for gestational age and did not show any signs of distress or reason for concern related to the diagnosis of placental increta. Due to the pelvic pain and spontaneous vaginal bleeding, the decision was made by the attending obstetrician that the patient would be scheduled for cesarean section followed by hysterectomy the following morning.

Leading up to the procedure, the status of both the patient and fetus did not change. The patient's hemoglobin and hematocrit were 11.2 g/dL and 29%, respectively, with a platelet count of 292/mcL. Prior to arriving in the operating room, a lumbar epidural was placed and the patient was taken to interventional radiology for bilateral balloon catheter placement into the uterine arteries. The patient and fetus tolerated this procedure with no complications. The care plan included use of the lumbar epidural as the primary anesthetic for the cesarean section, and then administer general anesthesia for the hysterectomy once the newborn was delivered.

The cesarean section was performed with the help of a sterile ultrasound, which assisted in identifying and avoiding the increta. Less than two minutes after surgical incision, the preterm neonate was delivered and the patient stated readiness for general anesthesia. We proceeded with our plan of general anesthesia which included a routine induction. The direct laryngoscopy was performed with a Macintosh-3 blade and a 7.0 mm endotracheal tube was placed at a distance of 20cm from the end of the endotracheal tube to the teeth, end-tidal carbon dioxide was confirmed and bilateral breath sounds were present. Prior to further manipulation of the uterus, an arterial catheter was placed in the right radial artery, a 9-french central line catheter was placed in the right internal jugular vein, and preexisting intravenous catheters were in place. On hold were; six units of packed red blood cells, three units of fresh frozen plasma, and one ten pack of platelets.

In addition to the anesthesia team, there were approximately twenty-five personnel involved in this procedure. Again, the obstetrician used a sterile ultrasound on the uterus to avoid any dissection in the area of the abnormally implanted placenta. The uterus was carefully dissected and eventually vessels were tied off. The uterus was removed from the abdominal cavity with no signs of massive hemorrhage. No blood products were administered and the hysterectomy did not involve any complications.

Discussion

The abnormal attachment of the placenta to the myometrium is uncommon and holds life-threatening features. With most patients presenting asymptomatic, the only way to diagnose the abnormality is through an ultrasound or an MRI, with ultrasound being the modality of choice.[2] The literature states that previous cesarean section is the underlying cause of developing a morbidly adherent placenta such as a placental accreta.[2]

The pathophysiology involved with this disease remains with question, although the leading theory involves abnormal revascularization of the endometrium after abdominal surgery, most

commonly a cesarean section.[2] This answers the question as to why a parturient is categorized at a higher risk for accreta when previous cesarean section is present in the medical history. Although not common, any pregnant female is at risk for developing a morbidly adherent placenta. According to the literature, two hallmark signs for this disease process include severe lower abdominal pain and vaginal bleeding[1]. These two symptoms are common during pregnancy, which make the diagnosis challenging. Some cases have also reported uterine rupture from an accreta, which is considered a medical emergency.

Hemorrhage will be the primary anesthesia concern with a stable diagnosed patient, such as the one presented in the earlier case study. Although the patient was stable leading up to the procedure, at any point during the surgery the patient could have developed massive hemorrhage. It is important to remember that the placenta normally detaches from the endometrium with ease once the fetus is delivered, but instead stays attached to the myometrium in the case of an increta. Careful manipulation of the uterus is extremely important when handling the fetus and cord after delivery, because this can cause a disruption in the tissue and vasculature of the placenta that's abnormally attached to the myometrium, which can result is hemorrhage.

Changes in uterine blood flow during pregnancy make for a better understanding of the significance of hemorrhage. The uterine blood flow before pregnancy is approximately 95mL/min, and during late gestation the flow increases to as much as 340mL/min.[3] This is a significant change that warrants caution for the anesthesia practitioner to be cognizant of. The risk of hemorrhage lies within the abnormal implantation of the placenta; if bleeding is detected due to a dissection or severing of vessels, then the balloons placed during interventional radiology can be inflated, which will stop blood flow to the uterus.[4] Keep in mind, no study has demonstrated that this technique is always helpful. Mixed results have come from uterine artery balloon pumps used for hemorrhage in the accreta patient.[4] No study has proven advantageous at providing enough evidence to make the balloon pump occlusion during this surgery a standard of care.

Hysterectomy following the delivery of the fetus by cesarean section is proven to decrease mortality and morbidity of the female patient.[5] Reduced manipulation of the placenta is said to alleviate the amount of bleeding following delivery of the fetus.[5] Hemorrhage is the number one concern when developing an anesthetic plan, which warrants hemodynamic stability.[1] The anesthesia care plan should have an emphasis on maintaining normovolemia and anticipating major blood loss.[1] The preoperative interview is important for the patient to understand the anesthetic plan for this procedure. The patient should be able to verbalize full understanding of the procedure and have knowledge of what to expect.

Overall, the anesthesia plan for this patient was adequate and the anesthesia team was ready to handle massive hemorrhage. All participants in the care of this patient had the same goal in mind which was patient safety, and worked collaboratively to achieve the best outcome for the patient. The patient was discharged home from the hospital two days after surgery, and the infant was discharged from the neonatal intensive care unit approximately three weeks later.

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Unilateral Tongue Neuropraxia following Laryngeal Mask Airway Placement

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Keywords: laryngeal mask airway, unilateral tongue numbness, manometry, lingual ischemia, hypoglossal nerve palsy, neuropraxia

Laryngeal mask airways (LMAs) are supraglottic airway devices that were developed in 1981 and have been used in clinical practice since 1988. They are an integral part of the ASA Difficult Airway Algorithm.[1] A study of 15,795 patients showed that only 1.1% of attempted LMA placements failed to the point of requiring endotracheal intubation.[2] LMAs have been shown to successfully facilitate ventilation in 94.1% of patients that cannot be mask ventilated or intubated.[1] Reported complications from LMA placement include but are not limited to aspiration, bronchospasm, laryngospasm, trauma, neurovascular injury, sore throat, dysphagia, pulmonary edema, and dysrhythmias.[3]

Case Report

A 163 cm, 52 kg, 61-year-old female presented for left-sided carpal tunnel release and ligament reconstruction and tendon interposition (LRTI). Her comorbidities included carpal tunnel syndrome, obstructive sleep apnea, fibromyalgia, gastro esophageal reflux disease, thyroid nodule, chronic fatigue syndrome, and chronic opioid use. Surgical history included correction of jaw fracture, tonsillectomy, lumbar fusion, cholecystectomy, bilateral tubal ligation, vein stripping and Harrington rod placement. Her medications included lorazepam, quetiapine, trazodone, morphine, fentanyl patch, gabapentin, and hydroxyzine. The patient had stopped taking meloxicam 7 days prior. There were no irregularities in her laboratory workup. She had a mallampati score of 2 and no abnormalities with her dentition. Her jaw surgery did not limit range of motion and the patient was not classified as a difficult airway.

A pre-operative single-shot supraclavicular nerve block was administered prior to transporting the patient to the operating room. She was pre-medicated with midazolam 2 mg and injected with ropivicaine 0.2% with 1:200,000 epinephrine 20 mL. Location of the injection was confirmed by ultrasound guidance. Once in the operating room, a pre-induction timeout was performed and noninvasive monitors were applied. Oxygen 10 L/min was administered by face mask for 3 minutes prior to induction. Induction was facilitated with propofol 150 mg, fentanyl 100 mcg, and lidocaine 50 mg. After general anesthesia was achieved a number 4 LMA (Medline Industries, Mansfield, MA) was lubricated and inserted without deflation of the cuff. No additional air was introduced to the LMA after insertion and the LMA has a built in bite block. No resistance was met during insertion and alignment of the tongue was confirmed visually and via digital examination. Placement was confirmed via positive end tidal carbon dioxide ($ETCO_2$) readings and auscultation of the patient's neck and bilateral lungs. Mechanical ventilation was maintained using pressure support with tidal volumes of 301-410 mL and a respiratory rate of 9 to 15/min. Peak inspiratory pressures had a range of 11 to 14 cm H_2O. General anesthesia was maintained with sevoflurane 1.5% inspired concentration in a mixture of O_2 1 L/min and air 1 L/min. Cefazolin 1 g was administered 20 minutes prior to the application of a mechanical orthopedic tourniquet to the patient's left upper extremity (inflated to 230 mm Hg).

Approximately 2 hours after inflation of the tourniquet/incision, the patient's heart rate increased from 70 to 100/min, her blood pressure increased from 90/60 to 120/80 mm Hg, and her head temporarily rotated slightly to the left. Sevoflurane was increased to 2% inspired concentration and the patient was given propofol 50 mg, esmolol 10 mg, and fentanyl 100 mcg. Her vitals returned to baseline and the rest of the procedure continued without issue. The patient was transitioned to spontaneous ventilation for the final 2 hours of her case. There was no change in respiratory rate, peak inspiratory pressures, or tidal volumes and the LMA was removed after being in place for 3 hours 23 minutes. While in the PACU the patient verbalized having unilateral tongue numbness. A neurology consult was placed and the surgical team was notified. Her assessment was negative for dysphagia, dysphonia, but positive for sore throat.

Discussion

Although there are documented cases of transient lingual neuropraxia following general anesthesia with an artificial airway, they are exceptionally rare. A 2009 review of the literature[4] found a total of 13 reported cases of lingual nerve neuropraxia dating back to 1971. This case report was limited in scope however, because its search was limited to journals written in English. Six cases were performed with an endotracheal tube (ETT), 1 was performed with a cuffed oropharyngeal airway (COPA), and 6 were performed with LMAs. Patient age ranged from 20-61 years, female to male ratio was 1.2:1, surgical duration was 20-150 minutes and duration of symptoms was 7 – 120 days with an average of 28 days. The patient matches well with these demographics with the exception of surgery duration. A surgical time of 191 minutes places her outside of the average surgical duration. We also know that the patient's tongue numbness resolved within four weeks from the day of surgery because weekly follow up telephone calls were made to track her progress. The patient was also seen by neurology as an outpatient as an added precaution. The neurology report also believed the symptoms to be indicative of unilateral tongue neuropraxia but failed to make a final diagnosis.

A Scandinavian case report[5] published in 2012 focused on two incidents of tongue numbness following LMA placement. During their literature review, they found 6 additional cases. Patient age ranged from 20-73 years, female to male ratio was 1:1, surgical duration was 45-140 minutes, and duration of symptoms was 3 to 24 weeks with an average of 10.5 weeks. There was no correlation found between duration of surgery and duration of symptoms or weight of the patient and duration of symptoms in either literature review.[4, 5] Sixty one percent of the patients in their literature review who experienced tongue numbness received N_2O as part of their anesthetic.[5]

Being able to discern which nerve has been damaged is important in ensuring patient safety. Cardinal signs of lingual injury include unilateral tongue numbness and loss of taste on the anterior tongue.[1] Injury to the hypoglossal nerve can prove to be much more problematic as it can result in dysarthria and dysphagia. As of 2014 there were nine recorded incidents of hypoglossal nerve palsy following the use of an LMA.[6] Eight of the nine cases were performed with LMAs that had silicone cuffs. The ninth case was performed with an LMA supreme (The Laryngeal Mask Company Ltd., St. Helier, Jersey, UK) which is made with polyvinyl chloride. The patient (a 67-year-old, 55 kg female) did not exhibit symptoms until the following day when she noticed she was slurring her speech. She did not exhibit dysphagia and her gag reflex remained intact.[6] This remains a possibility following hypoglossal injury though and would place the patient at significant risk for aspiration.

Although the number of reported cases of nerve injury following LMA placement are very rare, it is possible that cases have been under-reported in the literature and the incidence is actually much higher. The following place a patient at risk of injury or failed LMA placement: a mallampati score of 3-4, inability to protrude lower incisors to upper incisors, a qualitatively thick neck, having a beard, poor cervical spine mobility, poor dentition, thyromental distance less than 6cm, and reduced mouth opening less than 3 cm.[1] It has also been reported that use of nitrous may precipitate nerve injury during general anesthesia with an LMA.[5,6] Monitoring the intracuff pressure of LMAs has been shown to reduce the incidence of postoperative pharyngolaryngeal adverse events. Limiting the intracuff pressure to less than 44mmHg or 60 cm H20 reduced adverse events in ambulatory surgical patients by 70%.[7] The sample size of this study was 200 patients and there were no reported incidents of nerve injury.

There are steps that could have been taken that may have prevented lingual ischemia in this case report. Although there was no change in the patient's ability to ventilate after she moved during the case, digital manipulation of the mouth could have been helpful in confirming that there was no change in position of the LMA. Manometry would also have aided in making sure that the cuff was inflated to an appropriate pressure and therefore not compressing the lingual vasculature and nerves. It is thought that the tortuosity of the lingual nerve is what makes it prone to injury[4] and therefore attention to detail is paramount in ensuring patient safety. Reassuring the patient that the effects of the injury are most likely temporary[4, 5] and will resolve on their own can be very therapeutic. More research is needed in this area and better reporting will aid in illuminating its true prevalence.

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Anesthetic Implications of Buprenorphine Therapy

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Keywords: agonist-antagonist, buprenorphine, ketamine

Buprenorphine is a semisynthetic opioid agonist-antagonist that exhibits partial agonism at mu receptors and antagonism at kappa receptors.[1] Due to the pharmacodynamic characteristics of these drugs, patients receiving agonist-antagonist therapy presenting for surgery pose a challenge to the anesthetist in the management of surgical pain.[2] The features of this drug make it an attractive option for the management of chronic pain and it is being prescribed with increasing regularity.[3] Coupled with the Institute of Medicine's finding that 116 million Americans experience chronic non-cancer pain, strategies for the perioperative management of patients on buprenorphine therapy are critical tools for the anesthetist.[4]

Case Report

A 60-year-old, 182 cm, 101 kg male presented emergently for fusion of the first to the third cervical spine vertebrae (C1-C3) and an intermedullary nailing of his right femur following a motor vehicle collision. Computed tomography scans revealed a comminuted fracture of the

odontoid process of C2, a fractured left clavicle, and a closed intertrochanteric fracture of the right femur. No focal deficits were present. His medical history was significant for an orthotopic liver transplant secondary to hepatitis C cirrhosis, hypertension, hypothyroidism, and a 20 pack-year history of tobacco use. His home medications included amlodipine, everolimus, levothyroxine, sulfamethoxazole-trimethoprim, tacrolimus, and buprenorphine-naloxone.

In the preoperative area, the patient was identified with his cervical spine stabilized by a hard collar, an 18-gauge peripheral intravenous line (IV) in his forearm, and a urinary catheter present in his bladder. He received lactated ringer's 1 L and glycopyrrolate 0.2 mg IV prior to transport. In the operating room midazolam 2 mg, fentanyl 50 mcg, and ketamine 20 mg were titrated IV to facilitate the placement of airway blocks to permit safe endotracheal intubation. The superior laryngeal, glossopharyngeal, and recurrent laryngeal nerves were targeted with field blocks utilizing 2 and 4% lidocaine for a total of 4 and 5 mL respectively. The patient's gag reflex was ablated and a 5.7 mm fiberscope was introduced through the vocal cords to facilitate the passage of a 7.0 mm endotracheal tube. Following an unchanged neurological examination and confirmation of tracheal intubation, propofol 100 mg and rocuronium 50 mg were administered IV with isoflurane 0.8% expired concentration. A radial arterial catheter was placed in the patient's wrist along with a 16-gauge peripheral IV catheter in his hand. The patient was then repositioned prone on the operating table with cervical stabilization. Fentanyl 100 mcg and labetalol 10 mg were administered during placement of the cranial fixation device.

General anesthesia was maintained with isoflurane 0.8% expired concentration in O_2 0.8 L/min and air 0.8 L/min in addition to IV infusions of sufentanil 0.5 mcg/kg/hr and ketamine 30 mg/hr. Phenylephrine boluses were administered as needed and the patient was briefly maintained on a phenylephrine infusion. Rocuronium was titrated per peripheral neuromuscular monitoring. Surgical antibiotic prophylaxis was provided with cefazolin 2 g. The patient's laboratory values were closely monitored via arterial blood gas analysis and a metabolic acidosis with hemoglobin of 7.4 g/dL was corrected with sodium bicarbonate 50 milliequivalents, packed red blood cells 2 units, and calcium chloride 500 mg. The patient was removed from the cranial fixation device and turned supine at the culmination of the spinal fusion procedure and the orthopedic segment was begun with the patient remaining hemodynamically stable throughout. In total, the patient received crystalloid 4,200 mL, packed red blood cells 600 mL, made 850 mL of urine, and had 400 mL of estimated blood loss. Due to a prolonged emergence and the presence of multiple comorbidities, the patient was taken to the intensive care unit with the endotracheal tube in place.

Discussion

Buprenorphine is commonly prescribed as part of a 4:1 combination with the short-acting opioid antagonist naloxone in a sublingual preparation.[5] This unique composition slightly alters the pharmacokinetics of buprenorphine when administered by the sublingual route as the bioavailability of buprenorphine increases while the immediate effects of the drug are slightly attenuated by the relatively small addition of naloxone.[5] Naloxone is not well-absorbed in sublingual dosing and is added to buprenorphine in order to discourage opioid misuse should the drug be altered or administered by an unintended route.[1,5]

Patients on chronic opioid agonist-antagonist therapy provide several unique considerations for the anesthesia care team. The management of this patient was further complicated by an emergent presentation allowing few options for the perioperative optimization of their agonist-antagonist regimen. Options for the anesthesia practitioner encountering a patient on buprenorphine therapy in the perioperative setting generally include abruptly discontinuing the agent or continuing the drug and supplementing perioperative analgesia with potent opioid agonists and adjunct therapies.[2] Additionally, it may be feasible to wean the patient off buprenorphine therapy in preparation for surgery. Buprenorphine exhibits a half-life of 37 hours attributed to its slow dissociation from opioid receptors.[1,2] For this reason, it is recommended that the drug be completely discontinued 3 days prior to the procedure.[2] A common method by which to achieve this goal is to gradually taper buprenorphine by 2mg every 2-3 days over a period of 2-3 weeks until the drug is discontinued within an appropriate window for the surgical procedure to take place.[2] Withdrawal symptoms may occur, however withdrawal from buprenorphine is comparatively mild to that of agents such as morphine and symptoms may be treated with opioid agonists.[1] Withdrawal symptoms may be avoided altogether if methadone is substituted for buprenorphine preoperatively and continued throughout the perioperative period.[2] The substitution for methadone, a pure opioid agonist, will allow for more predictable control of analgesia as the antagonistic effects on the kappa receptors provided by buprenorphine are obviated.[1, 2]

Conversely, a buprenorphine taper or preoperative switch to methadone is inappropriate for patients presenting for emergent surgery and the anesthesia team must be prepared for complex pain management. One option that exists in this scenario is maintaining the patient on buprenorphine therapy throughout the perioperative period.[2] However, a ceiling effect exists and an escalating dose of buprenorphine may cease to provide adequate analgesia at a certain point.[1,2] In the absence of randomized controlled studies, current practices are based on case reports and opinions regarding surgical pain management for patients on chronic opioid agonist-antagonist regimens.[2] Common strategies employed to achieve adequate pain control in this patient population include nonsteroidal anti-inflammatory agents, acetaminophen, gabapentin or related analogues, intravenous lidocaine, alpha-2 agonists, NMDA antagonists, and regional anesthesia.[6] A ketamine infusion combined with a potent pure opioid agonist, sufentanil, was chosen for the maintenance phase of the aforementioned procedure.

Ketamine, an NMDA antagonist, has a unique role in the management of the patient on chronic opioid therapy. In addition to providing analgesia, ketamine acts to reduce and reverse opioid-induced hyperalgesia and opioid tolerance.[6] While the amount of opiate required to engender hyperalgesia and tolerance is unknown, ketamine has been shown to be beneficial to both opioid-naïve and opioid-dependent patients.[7] A fundamental tenet of chronic pain transmission involves the sensitization of neurons in the dorsal horn of the spinal cord which may occur by multiple mechanisms.[1] It is proposed that NMDA activation and the resultant excitatory transmission of nociceptive impulses play an integral role in the sensitization process.[1] The inhibition of these receptors in the perioperative environment has been shown to reduce pain scores and reduce opiate use up to 2 days postoperatively.[6] The addition of intravenous ketamine as part of a multimodal approach to pain management should be considered in this unique patient population.[7]

The anesthetic management of this patient was complex given his unstable cervical spine and presence of various traumatic injuries. A significant additional challenge posed to the anesthesia team was management of pain control during two lengthy and stimulating procedures. This aspect of care was further complicated by the patient's opioid agonist-antagonist maintenance therapy and the emergent nature of the patient's presentation allowing few options for the optimization of his opioid regimen. Thus, a thorough understanding of the pharmacokinetic and pharmacodynamic principles governing buprenorphine therapy and the use of non-opioid adjuncts for the management of acute surgical pain are critical to safely care for these patients in the perioperative period as the implementation of multi-modal therapy can facilitate a reduction in both postoperative pain and opioid use.[2] Effective pain control may contribute to better postoperative outcomes, reduced morbidity complications, and fewer days in the hospital for surgical patients and this can be facilitated through preemptive analgesia and the skillful and knowledgeable application of multi-modal therapy by the anesthetist.[2,6]

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The Incidence and Risk Factors of Airway Compromise during Endoscopy

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Keywords: sedation-related complications, endoscopy, airway compromise

Introduction

Millions of endoscopies occur each year under anesthesia. The current risk of airway compromise during endoscopy is 1.24%.[1] Anesthesia practitioners hypothesized the incidence at a large urban tertiary center would be higher. A follow-up study confirmed an increase in airway compromise at this facility with an incidence of 6.57%, five times greater than the national average. The increase is attributed to increased age, body mass index (BMI), as well as type and length of the procedure which were statistically significant risk factors in this study using logistic regression analysis. Neuman's Systems Model was used as the guiding theoretical framework for primary prevention of airway compromise.

Methods

International Review Board (IRB) approval was first obtained. Subsequently, a retrospective chart review was conducted on all patients undergoing both upper and lower endoscopy at a large, tertiary hospital in an urban setting. Medical records for 700 patients were included in the study, with no exclusion criteria. Airway compromise was defined as desaturation <90%, two or more airway maneuvers, nasal/oral airway insertion, bag-mask ventilation, or endotracheal intubation. Age, sex, race, ASA class, BMI, smoking status, and type and length of the procedure were also collected. Data were de-identified and stored in a password-protected Excel document. Data analysis was performed using logistic regression analysis via SPSS in coordination with a statistician from the affiliated university.

Results

Airway compromise occurred in 46 of 700 patients (6.57%). The rate of desaturation was 3.14%, two or more airway maneuvers 2.86%, and nasal/oral airway insertion 2.29%. Even more staggering is the increased rate of endotracheal intubation in 0.86% of patients compared to 0% in many published studies. Logistic regression determined age ($p=0.015$), type of procedure ($p=0.036$), BMI ($p=0.025$), and length of the procedure ($p=0.021$) as statistically significant patient risk factors for airway compromise. 44% of patients who experienced airway compromise were 51-60 years old, and 24% of patients were 61-70 years old. Patients who undergo colonoscopy have a 25% higher incidence of airway compromise (Ex(B)=1.250) compared to EGD. The incidence of airway compromise increases 41.7% (Ex(B)=1.417) as procedure time increases. Ultimately, 24% of events occurred between 11-20 minutes and 37% occurred between 21-30 minutes. As BMI increases, the risk of airway compromise increases; patients with a BMI of 31-35 are 41% (Ex(B)=1.409) more likely to have an airway compromise event than patients with a BMI of 20-30. ASA class, smoking status, sex, and race were not deemed statistically significant risk factors.

Discussion

At this study site, an urban tertiary facility, the rate of airway compromise is five times higher compared to the national average. Neuman's system model was utilized to determine which stressors, or patient specific risk factors, would disrupt homeostasis and lead to airway compromise. This study determined those stressors include age, BMI, and type and length of procedure. A sedation risk assessment tool has been designed and implemented in the endoscopy clinic with the goal of decreasing the rate of airway compromise from 6.57% to the national average of 1.24%.

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Effect of Cricoid Pressure on Laryngeal View, Airway Obstruction, and Aspiration

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Keywords: cricoid pressure, laryngoscopic view, airway obstruction, aspiration, rapid sequence induction

Introduction

According to the Centers for Disease Control and Prevention, over 100 million patients in the United States undergo surgical procedures each year with many requiring tracheal intubation.[1] In an attempt to prevent pulmonary aspiration of gastric contents during tracheal intubation, cricoid pressure (CP) is applied in thoughts of occluding the esophagus with compression.[2] This maneuver is mainly utilized when the possibility of a patient with a full stomach is suspected or uncontrolled gastric reflux is present.[2] Aspiration may lead to severe pulmonary complications including infection and/or death.[2] Studies composed over the last decade have revealed that not only was cricoid pressure performed incorrectly by various healthcare personnel, including physicians and anesthesia professionals, but also applied the incorrect amount of force.[2] Recommended CP force in the awake patient is 10 Newtons (N) and increased to 30 N once the patient becomes unconscious, 1 kg is equivalent to 9.8 N of force.[3]

In 2010, the American Heart Association (AHA) changed their position by no longer recommending use of CP during Rapid Sequence Induction (RSI) and cardiac arrest.[4] In attempt to prevent hypoxia and minimize time between induction of anesthesia and the act of laryngoscopy, RSI is performed while utilizing methods to decrease potential pulmonary

aspiration of gastric contents.[4] The basis of the recommendation included that CP may prevent correct placement of laryngeal tubes and laryngeal mask airways, worsen laryngoscopic view, impair ventilation, and cause complete airway obstruction in up to 50% of patients.[4] Injuries and complications related to inadequate or inaccurate CP include cartilage fracture, esophageal rupture, airway obstruction, laryngeal and cervical trauma.[3]

The purpose of this evidence-based practice analysis is to evaluate proper application of cricoid pressure, its effect on laryngeal view, esophageal displacement preventing complete occlusion, obstruction of airway, and prevention of aspiration. The rational for the research is to obtain greater knowledge about possible harm and ineffective outcomes that can result from various methods and forces of CP. The conceptual framework of the limited observational studies that focused on the effects CP has on laryngeal view, airway occlusion, and prevention of aspiration in the past ten years was sufficient evidence for the AHA to remove CP from the guidelines and recommendations.[4]

Roughly 0.15% of adults will aspirate upon induction of anesthesia.[3] Cricoid pressure was originally studied in 1961 by Brian Arthur Sellick, an anesthetist, on cadavers by applying pressure on the cricoid cartilage using a one-handed technique to occlude the esophagus against the cervical vertebrae.[5] Sellick applied his theory in 26 patients at high risk for aspiration during induction of anesthesia. The result was 100% prevention of gastric regurgitation while the Sellick's maneuver (CP) was applied and 12% had immediate gastric reflux once CP was released after laryngoscopy.[5] Cricoid pressure became a standard of care in RSI and during induction of anesthesia in patients at high risk for aspiration.[3,6] Cricoid pressure remains widely practiced despite the lack of randomized clinical trials (RCT) and scientific evidence that validates Sellick's research.[3] The lack of evidence to validate the original report by Sellick is a major barrier requiring additional research and randomized studies.

Methodology

Evidence-based Practice Model

Utilizing the PICO format developed the clinical question which initiated a specific search criteria. The research formulated the following PICO question: In adult surgical patients at high risk for aspiration (P), does the utilization of cricoid pressure (I) compared to no cricoid pressure (C) obscure laryngoscopic view, obstruct the airway, and prevent aspiration (O).

Search Models

Cumulative Index to Nursing & Allied Health Literature (CINAHL), MEDLINE, Ovid, ScienceDirect, Cochrane Library, PubMed, Wiley Online Journal Library, and Google Scholar were utilized to conduct a thorough search of the most recent and significant data. Eligibility criteria included full-text, studies with a level III evidence or above, peer reviewed studies and articles published since 2005. One background article and one study that was published prior to 2005 was selected for purposes of historical significance and lack of relevant RCTs. The exclusion criteria included pediatric studies, studies in progress, and languages other than English.

Search Terms
Keywords cricoid pressure, laryngoscopic view, airway obstruction, aspiration, and rapid sequence induction produced numerous results. Exclusions and limitations were utilized to narrow down the search for pertinent studies and background articles.

Levels of Evidence
Final studies selected for the literature review consisted of six observational studies, including quantitative and prospective, as well as one RCT. Five background articles were included to build the foundation for the literature review. Studies selected provided level I, II, and III on the Joanna Briggs Institute levels of evidence hierarchy.

Literature Review

Obscured Laryngoscopic View
Since the Sellick's maneuver was introduced in 1961, CP has been used during RSI with an objective to prevent gastric aspiration. Since then, many studies have been conducted using various methods to provide evidence that CP can obscure laryngeal view. Studies that fulfilled the search criteria were included in this literature review.

An observational study was conducted using 40 ASA I or II patients requiring endotracheal intubation for elective surgery.[7] Exclusion criteria include ASA III or above, under 18 years of age, current pregnancy, and risk of regurgitation. A rigid endoscope with a 100˚ view angle was used to record digital images of laryngeal view. After pre-oxygenation with 100% O2, the endoscope was placed along the anesthetist's line of view during normal laryngoscopy. Exact images were captured without CP. Once images were recorded, CP was applied and exact force was measured based on the person's weight applying pressure while standing on a scale. For every 9.81 N of pressure applied, the assistants weight would decrease by 1 kg. Cricoid pressure was increased by increments of 10 N, up to 60 N, with continued recording of digital images. The study concluded that views deteriorated with forces of 30 N or greater, which is usually recommended for RSI.[7]

A prospective observational study conducted on 402 patients compared laryngeal view and intubation success rate with CP versus laryngeal manipulation.[8] Laryngeal manipulation was required in 61 intubations on 55 patients or 13.6%. In 22 subjects, removal of CP improved laryngeal view in 50% of cases. There was a 60% improvement in 25 intubations that utilized bimanual laryngeal manipulation. Backward-upward-rightward pressure (BURP) method improved the view in 64% of 14 intubations.[8]

A prospective controlled randomized double-blind, cross-over study was conducted on 50 patients undergoing spinal surgery under general anesthesia to evaluate effects of CP on duration of fiber-optic intubation (FOI) and glottic view.[9] Exclusion criteria included ASA >2, indication for RSI, age <18 years, and BMI >35 kg/m2.[9] All 50 subjects, 21 males and 29 females, were fiber-optically intubated by one anesthetist with both 30 N of CP and no CP in randomized order.[9] Duration of intubation without CP was 59 seconds (s), 53-79 median interquartile, with CP was 75 s, 53-79 median interquartile, (P<0.001).[9] Unsuccessful intubations >180 s were

considered as a failed attempt.[9] Failed intubations were recorded in three patients without CP and 13 with CP (P=0.017).[9] Cormach-Lehane scores were as followed: five grade 1 with CP and 10 without, three grade 2 with CP and 14 without, 29 grade 3 with CP and 19 without, seven with CP were failed intubations, 13 grade 4 with CP and 7 without, six with CP were failed intubations.[9] It was concluded that CP prolongs duration of FOI and impairs glottic view.[9]

A prospective crossover study involving 100 patients undergoing elective surgery requiring general anesthesia.[10] A Wilcoxon signed-rank test was used, P-values ≤0.025 were considered statistically significant. Tracheal intubation was performed utilizing videolaryngoscopy followed by immediate digital image capture.[10] Jaw thrust and CP were performed separately in randomized order with images captured after each maneuver.[10] Images were randomly presented to a blinded investigator to grade the laryngoscopic views using the modified Cormack and Lehane system.[10] Glottic grade was significantly improved with jaw thrust compared to videolaryngoscopy alone (P<0.001) in 31% of subjects and worsened in 4%.[10] Cricoid pressure did not show a difference in glottic grade when compared to videolaryngoscopy alone (P=0.19).[10] Glottic grade improved in 39% of subjects but worsened in 20%.[10] The glottic area was measured by a second blinded investigator in pixels squared.[10] Glottic opening area was significantly improved with jaw thrust compared to videolaryngoscopy alone.[10]

Obstruction of Airway

Obstruction of airway has also been studied as a negative result from application of CP. Many studies found during the search were conducted greater than 10 years. Inconsistent education, technique or cricoid force may contribute to airway obstruction.

An observational study was conducted on 52 ASA 1 and 2 female patients aged 21-67 weighing 44-108 kg undergoing gynecological surgery with general anesthesia.[11] Exclusion criteria included history of gastroesophageal reflux disease.[11] After induction, expired tidal volume and inflation pressures during ventilation via an oral pharyngeal airway and facemask were recorded.[11] Four different CPs: none, 30 N backwards, 30 N upwards and backwards, and 44 N backwards were applied in random order by the same anesthetist.[11] After tracheal intubation with an 8.0 mm endotracheal tube, the expired tidal volume, peak inspiratory pressure, Mallampati, and Cormack & Lehane scores were recorded using the same methods of CP.[11] Airway obstruction was classified with expired tidal volumes <200 mL as evidenced by lack of chest movement and absence of capnography tracing.[11] A Chi-square test was used for statistical analysis with a P-value of 0.05 being considered as significant.[11] There was zero complete airway obstruction without CP, one subject or 2% had airway obstruction when 30 N of CP was applied, 29 or 56% when 30 N of CP was applied using upward and backward direction, and 18 or 35% when 44 N of CP was applied.[11] Airway obstruction was observed more often with 44 N of CP than 30 N (P=0.0001) or no CP (P<0.0001).[11] Airway obstruction occurred more frequently with 30 N of CP using upward and backward direction than 30 N of CP using backward direction alone (P<0.0001).[11] No correlation between airway obstruction and intubation grade or Mallampati score was noted.[11]

In a previous study, Arenkiel at el.[9] noted eight of 13 failed intubations were due to resistance during endotracheal tube insertion or impingement complications. Ten intubations that failed

with CP were successful without CP on either the first or second attempt suggesting that CP causes airway occlusion.[9]

A blinded prospective observational study conducted using 110 critical care staff, 74 nursing and 36 medical, who were instructed to apply 30-40 N of pressure on a plastic human larynx with cricoid model in supine position fixed on a validated scale and was recorded.[6] After initial attempt, the subjects were given three minutes of uncoached practice, followed by a second attempt.[6] The range of CP force applied during the first attempt was 3.7-98.1 N.[6] Following the intervention, the range of force was 23.7-59.8 N.[6] Only 22 or 20% of subjects correctly applied target range force of 30-40 N on the first attempt.[6] This improved to 57 or 52% post intervention (P<0.01).[6] Difference in force was significantly evident in both nursing and medical subgroups.[6]

Prevention of Aspiration and Esophageal Displacement

Aspiration prevention during RSI has included the use of CP over the past several decades. Several studies have been conducted using radiological imaging to challenge whether CP effectively occludes the esophagus.

A quantitative descriptive study was conducted on 20 conscious volunteers, 10 males and 10 females, to examine the anatomical effects caused by CP on esophageal lumen occlusion using magnetic resonance imaging (MRI) for 12 seconds by one fully trained anesthesiologist.[12] Esophageal patency was also assessed and recorded with MRI prior to CP.[12] A radiologist confirmed accurate CP by assessing appropriate finger placement on the cricoid cartilage, alignment with the vertebral body, and amount of distance reduction between the ventral surface of vertebral body and posterior area of cricoid ring.[12] Any visible esophageal lumen noted at the level of the cricoid during CP was classified as incomplete esophageal occlusion.[12] A reduction in cricovertebral distance of 43%, with range of 25-70%, was achieved using target CP in 16 out of 20 subjects.[12] Of these 16 individuals, esophageal occlusion was inadequate in 10, or 62% using a 95% confidence interval of 35-85%.[12] Images shown in the study displayed evidence of lateral deviation of the esophagus in these 10 subjects.[12] Overall, of the 20 subjects involved, 14 or 70%, had incomplete esophageal occlusion with CP.[12]

Further concern of the effectiveness of CP is the lack of adequate esophageal occlusion.[12] In an observational cohort study, 20 conscious volunteers were examined using magnetic resonance imaging (MRI) to determine the degree of esophageal occlusion during CP.[12] The reduction of cricovertebral distance ranged from 25-80%, as well as 20% of subjects had CP applied incorrectly.[12] Even when the appropriate cricoid pressure was applied, there was a 62.5% incomplete occlusion of the esophagus with lateral deviation pronounced.[12] The final result was a 70% failed attempt of digestive conduit occlusion at the level of the cricoid cartilage.[12]

Conclusion

Originally, Sellick's research was performed in patients placed in Trendelenburg with the neck fully extended or "tonsil" position.[5] Sellick's study has not been repeated in patients placed in the supine position. Patients today are either placed in a neutral or "sniffing" position instead of the neck fully extended creating even more doubt in Sellick's original study. Boet et al.[12]

hypothesized that the hyperextension of the neck Sellick (1961) placed on cadavers would have caused stretching of the esophagus tightly over the midline allowing greater occlusion with CP.

Additional risks of inadequate or inaccurate CP include further injury of an unstable cervical spine, decreased tone in lower esophageal sphincter, esophageal rupture, cricoid injury, laryngeal trauma, impaired laryngeal view during laryngoscopy, airway obstruction, and difficult ventilation.[3,12] Lack of RCT's to support ongoing use of CP is significant. Fear of litigation to withhold CP during RSI or in patients with uncontrolled gastric reflux may be an indicator for the lack of new randomized trials.[3] The level I, II, and III studies presented in the literature review, provided a strong basis on negative outcomes that result from CP. Various methods were utilized in a variety of samples which provided a wide range of validation. The research studies suggest that CP lacks effectiveness due to inconsistent technique and force utilized, as well as airway obstruction and incomplete esophageal occlusion.

The AHA is the gold standard for emergency resuscitation guidelines, which are based on empirical evidence. As healthcare practitioners, following evidence-based practice provides safe care. Suggestions for further research on the effects of CP remains highly recommended.

Literature Review Matrix

Resource	Obscure Laryngoscopic View	Obstruction of Airway	Aspiration Prevention and Esophageal Displacement	Misc.
Haslam at el., 2005	The study concluded that views deteriorate with forces of 30 N or more, which are usually recommended for RSI. Results are as follows: **Force** **% View deteriorated** 0-10 N 30% 10-20 N 40% 20-30 N 48% 30-40 N 63% 40-50 N 55% 50-60 N 53%			
Boet at el., 2012			No change in cricovertebral distance was considered inaccurate CP application and was noted in 20% of subjects. A reduction in cricovertebral distance of 43%, with range of 25-70%, was achieved using target CP in 16 out of 20 subjects. Of these 16 individuals, esophageal occlusion was inadequate in 62%. Study images displayed evidence of lateral esophageal deviation in these subjects. Overall, of the 20 subjects in this study, 70% had incomplete esophageal occlusion with CP.	Additional risk of CP include further injury of an unstable cervical spine, decreased lower esophageal sphincter tone, esophageal rupture, cricoid injury, impaired laryngeal view during laryngoscopy, and difficult ventilation.

Harris at el., 2010	Laryngeal manipulation was required in 61 intubations. In 22 subjects, removal of CP improved laryngeal view in 50% of the cases. There was an improvement in 60% of 25 intubations that utilized bimanual laryngeal manipulation. BURP improved the view in 64% of 14 intubations.			CP causes less effective bag-mask ventilation.
Hartsilver at el., 2000		There was zero complete airway obstruction without CP, 2% had airway obstruction when 30 N of CP was applied, 56% with 30 N of CP using upward and backward direction, and 35% when 44 N of CP. Airway obstruction was observed more often with 44 N of CP than 30 N (P=0.0001) or no CP (P<0.0001). Airway obstruction occurred more frequently with 30 N of CP using upward and backward direction than 30 N of CP using backward direction alone (P<0.0001). No correlation between airway obstruction and intubation grade or Mallampati score was noted.		
Arenkiel at el., 2013	Duration of intubation without CP was 59 seconds (s), and 75 s with CP, (P<0.001). Failed intubations were recorded in three patients without CP, 13 with CP (P=0.017). Cormach-Lehane scores were as followed: Five Grade 1 with CP, 10 without. Three Grade 2 with CP, 14 without. Twenty-nine Grade 3 with CP, 19 without, seven with CP were failed intubations. Thirteen Grade 4 with CP and 7 without CP, six with CP were failed intubations.			

May at el., 2007				Range of CP force applied during first attempt was 3.7-98.1 N. Following the intervention, range of force was 23.7-59.8 N. Only 20% of subjects correctly applied target range force of 30-40 N on first attempt. This improved to 52% post intervention.
Corda at el., 2012	Glottic grade significantly improved with jaw thrust compared to videolaryngoscopy alone (P<0.001) in 31% of subjects and worsened in 4%. Cricoid pressure did not show a difference in glottic grade when compared to videolaryngoscopy alone (P=0.19). Glottic grade improved in 39% of subjects but worsened in 20%. Glottic opening area significantly improved with jaw thrust compared to videolaryngoscopy alone. (P<0.001). Median increase in pixels squared was 507, and the median percentage change was 12%. Glottic opening area significantly decreased with CP compared to videolaryngoscopy alone (P<0.001). Median decrease in pixels squared was -1,042, median percentage change was -27%.			

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Cuffed verses Uncuffed Endotracheal Tubes for Pediatric Patients

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Keywords: cuffed, uncuffed, endotracheal tubes, stridor, re-intubation, pediatric,

Introduction

Pediatric airway management can present many challenges for anesthetists. One such challenge is whether to use a cuffed or uncuffed endotracheal tube (ETT) for pediatric patients younger than eight years. Practitioners have used uncuffed ETTs for smaller children due to the risk of cuffed ETTs causing airway mucosal injury, which may lead to subglottic stenosis and stridor.[1] In recent years, cuffed ETTs have been used more frequently in order to reduce tube exchanges, ventilation leak around the tube, unreliable end-tidal carbon dioxide (ETCO2) monitoring, and the risk of aspiration.[1] The purpose of this evidence based practice analysis is to compare the

incidence of common airway complications in pediatric patients intubated with a cuffed or uncuffed ETT.

Methodology

A population, intervention, comparison, and outcome (PICO) question was developed to aid in the research and literature review process. The PICO question posed was "Do pediatric patients less than eighteen years of age intubated with a cuffed ETT have a reduced instance of tube exchanges and post-extubation stridor compared to patients with an uncuffed ETT ?"

Data was obtained by searching the following databases: CINAHL, Cochrane Collection, EBSCO, and PubMed. Keywords used included: cuffed, uncuffed, endotracheal tubes, airway, post-extubation, stridor, re-intubation, endotracheal tube exchange, pediatric, and infant. All studies published in 2010 or later, written in English, and including the keywords were retrieved and analyzed. The inclusion criteria were studies published in a peer review journal which evaluated pediatric endotracheal intubation and reported post-extubation stridor and/or ETT exchange data. Five studies met inclusion criteria with varying levels of evidence including: meta-analysis, randomized control trial (RCT), prospective observational, and retrospective review.

Literature Review

A meta-analysis by Shi et al. assessed current evidence regarding post-extubation morbidity and tracheal tube exchanges in pediatric patients. Four RCTs and prospective cohort studies which compared outcomes in pediatric patients intubated with either cuffed or uncuffed ETTs were included. Post-extubation stridor was the primary outcome analyzed with stridor found in 94 of 1979 (4.75%) in the cuffed ETT group, and 99 of 1803 (5.49%) in the uncuffed group. Shi et al. found no statistically significant difference in the incidence of stridor ($p=0.36$).[1] Secondary outcomes analyzed included the frequency of ETT exchange, rate of re-intubation following planned extubation, and the duration of intubation. Twenty-seven of 1370 (1.97%) in the cuffed ETT group compared to 401 of 1364 (29.40%) in the uncuffed ETT group required an ETT exchange ($p=0.00001$).[1] Two RCTs in the meta-analysis compared the incidence of ETT exchange and both found that uncuffed ETTs were associated with a clinically and statistically significant increase in the frequency of ETT exchanges. Multiple reasons for the exchanges were identified including: resistance to passing the ETT, lack of leak at 20cm H_2O positive airway pressure, and excessive leak during positive pressure ventialation.[1] Shi et al. concluded cuffed ETTs are associated with a reduced incidence of ETT exchanges, and the rate of post-extubation stridor did not significantly differ between the cuffed and uncuffed ETT groups. Two of the studies analyzed in the meta-analysis were published in 1997 and 1994, and the findings may not reflect current practice or ETT design.

A RCT performed by Mukhopadhyay et al. evaluated post-extubation stridor, laryngospasm, sore throat and other complications in 110 pediatric patients undergoing a cleft palate repair.[3] Patients between the ages of 2 and 12 years were randomly assigned to a cuffed or uncuffed group. Patients assigned to the cuffed group were intubated with a preformed, south facing ETT with a

high volume, low-pressure cuff. A leak test was performed with no air in the cuff, and the ETT was replaced with a 0.5 mm smaller ETT if a leak was not present with $\leq$ 20 cm H_2O positive pressure. The cuffed ETT was replaced with a larger size if the cuff pressure required to seal the trachea was > 20 cm H_2O. A minimal sealing pressure was determined by slowly deflating the cuff until a leak occurred, and then slowly reinflating the cuff until the leak disappeared. Cuff pressures were monitored with a monometer and limited to 20 cm H_2O. A preformed uncuffed ETT was placed in those assigned to the uncuffed group. The uncuffed ETT was replaced with a 0.5 mm smaller ETT if a leak was not present with $\leq$ 20 cm H_2O positive pressure. A wet gauze throat pack or placement of a cuffed ETT were utilized in cases of excessive leak. The anesthetic protocol included dexamethasone and paracetamol. There was no statistically significant difference in the rate of post-extubation stridor between the two groups (p= .15). Five of 53 (9.4%) patients in the cuffed ETT group developed post-extubation stridor while 5 of 57 (8.8%) in the uncuffed ETT group developed post-extubation stridor.[3] Mukhopadhyay et al. included dexamethasone and paracetamol in the anesthetic protocol which may have influenced the occurrence of stridor. Cuff pressures were also carefully established and monitored which may be difficult to reproduce in many clinical settings.

Nascimento et al., examined the risk factors for the development of post-extubation stridor in pediatric intensive care unit (ICU) patients in a prospective observational study. Factors analyzed included sex, age, weight, duration of intubation, duration of mechanical ventilation, admission diagnosis, cuffed or uncuffed ETT, and size of ETT. All patients included in the study were intubated and mechanically ventilated for more than 24 hours. The study design was prospective but the attending clinician determined the choice of an uncuffed or cuffed ETT. Seventy-four patients (54%) had a cuffed ETT, but 19 of the cuffed ETTs did not have the cuffed inflated. Cuff pressures in inflated cuffed ETTs were monitored and maintained at pressures < 20 cm H2O.

Nascimento et al. studied 136 patients between 3 days and 17 years of age admitted to the pediatric ICU between January 2008 and May 2011. An uncuffed ETT was placed more often in infants less than 1 year of age than in older children. A cuffed ETT was placed in 11 of 45 infants (24%), in 14 of 24 children between 1 and 2 years of age (60%), and in 23 of 31 children between 2 and 6 years (73%). Almost all children >6 years of age had a cuffed ETT placed with only 1 of 36 with an uncuffed ETT. The authors defined stridor as any noisy breathing, specifically high-pitched in nature, which was associated with obstruction after intubation.[2] Patients were followed for 72 hours post extubation and monitored for any instances of post-extubation stridor. It was found that 21 of 55 patients (38.2%) in the inflated cuffed ETT, 11 of 19 (57.9%) in the deflated cuffed ETT, and 24 of 62 (38.7%) in the uncuffed ETT groups developed stridor. Nascimento et al. reported no statistically significant difference between the cuffed and uncuffed ETT groups and the incidence of stridor. It was found that the two risk factors for post-extubation stridor were the duration of mechanical ventilation and the duration of intubation.[2] Uncuffed ETTs were placed more often in younger children with presumably smaller airways which could have an effect on the incidence of stridor in the uncuffed group.

A prospective observational study of 100 pediatric patients undergoing laparoscopic surgery under general endotracheal anesthesia with a Microcuff® cuffed ETT was performed by Mhamane et al.. Goals of the study included evaluation of variations in intracuff pressures and

post-extubation airway complications. The Microcuff® ETT was designed specifically for pediatrics with an ultrathin cuff located near the tip to avoid pressure in the subglottic area.[4] The size of the ETT was determined by a standard protocol based on the patient's age. A positive pressure leak test following intubation was used to verify an appropriate size. An ETT with no leak present at 20 cm H2O pressure was replaced with a 0.5 mm smaller ETT. An ETT with an excessive leak interfering with ventilation was replaced with a 0.5 mm larger ETT. Cuff pressure was monitored during inflation and continuously during the anesthetic. Cuff pressure was limited to 20 cm H_2O or less by a pressure release valve connected to the pilot balloon. A minimal sealing pressure was determined by reducing the cuff pressure until a leak was detected and then increasing the pressure until the leak stopped.[4] The mean intracuff pressure was 11.72 cm H_2O in the supine position, 12.48 cm H_2O during pneumoperitoneum, and 13.32 cm H_2O in a head down position. Mhamane et al. determined the cuff pressure in the Microcuff® cuffed ETT will increase with creation of a pneumoperitoneum and repositioning in a head down position but remained below 20 cm H_2O. No patients in the study had signs and symptoms of stridor after extubation.[4] The ETT is an alternative to be considered for pediatric patients as long as the cuff pressure is carefully monitored.

Lastly, Dorsey et al., retrospectively reviewed operating room intubations performed on patients in a pediatric burn ICU. The incident of adverse events including aspiration, post-extubation stridor, leak around the ETT, and ETT exchanges were compared between cuffed and uncuffed ETT placement. The authors of the study note that the airway of pediatric burn patients presents many challenges. One challenge is these patients typically require multiple intubations due to multiple surgeries for skin grafting.[5] If the rate of ETT exchanges is high, combined with multiple procedures requiring intubation, an increased chance for damage to the trachea presents itself.

The Dorsey study included 228 patients with ages ranging from 0 to 10 years. Data was examined for the 10 year time period January 1998 to December 2007. Of the 228 patients 52 patients needed re-intubation for any reason including an ETT exchange for a leak. In the uncuffed ETT group a leak occurred in 23% (27/117) while in the cuffed ETT group 2 of 111 (1.8%) had a leak around the ETT (<0.001). In the cuffed ETT group, 8 of the 111 (7.2%) required a re-intubation.[5] The reintubation rate was significantly higher in the uncuffed ETT group with 44 of 117 (37.6%) ($p<0.001$).[5] For the second outcome of post-extubation stridor a significant difference was not found. In the cuffed ETT group, 8 of 111 patients (7.2%) developed stridor and in the uncuffed ETT group, 5 of 117 patients (4.3%) developed stridor ($p=0.4$).[5] The choice of ETT size and design was based on clinical judgement of the anesthesia practitioner. Younger children were more likely to have an uncuffed ETT placed which may influence the findings.[5] It is unknown how many reintubations were done due to an air leak as all re-intubations for any reason were reported in a single category. The cuffed ETT group did experience a statistically significant lower incidence of air leak around the ETT.

Author, Date	Design	Sample	Tube Exchanges	Stridor
Shi[1] et al, 2015	Meta-analysis	4 studies • 2 RCTs • 2 Prospective cohort studies	n= 2734 *Cuffed* • 1.97% • n=27/1370 *Uncuffed* • 29.4% • 401/1364 p<0.00001	n= 3782 *Cuffed* • 4.75% • n= 94/1979 *Uncuffed* • 5.49% • n= 99/1803 p= 0.36
Mukhopadhyay[3] et al, 2016	RCT	Ages 2-12yrs with cleft lip n=110	No data reported	*Cuffed* • 9.4% • n= 5/53 *Uncuffed* • 8.8% • n= 5/57 p= 0.15
Nascimento[2] et al, 2015	Prospective cohort study	Ages 3 days- 17yrs ICU admission with mechanical ventilation for >24 hours n= 136	No data reported	*Cuffed* • 43.2% • n= 32/74 p= 0.953 *Uncuffed* • 38.7% • n= 24/62 p= 0.605
Mhamane[4] et al, 2015	Prospective observational study	Ages 8mo- 9yrs Laparoscopic surgery with Microcuff® cuffed ETT n= 100	No data reported	*Cuffed* • 0% • n=0/100
Dorsey[5] et al, 2010	Retrospective review	Age 0-10yrs Burn patient operating room intubations n= 228	*Cuffed* • 7.2% • n= 8/111 *Uncuffed* • 37.6% • n= 44/117 p<0.001	*Cuffed* • 7.2% • n= 8/111 *Uncuffed* • 4.3% • n= 5/117 p= 0.4001

Conclusions

A wide range of ages of pediatric patients was included in the study. The airway anatomy of an adolescent has matured and differs from the airway anatomy of the younger child. This limitation must be kept in mind when considering the conclusions.

Cuffed ETTs were not associated with an increased risk of post-extubation stridor in the studies reviewed. Four of the studies compared the incidence of stridor and none found a statistically significant difference between cuffed and uncuffed ETT groups. The incidence of stridor in the ICU patients was increased in both cuffed and uncuffed ETTs compared to the three studies of surgical patients with shorter durations of intubation. Stridor was reported in more than 40% of the ICU patients who were intubated for > 24 hours. The incidence of stridor in the surgical patients with a cuffed ETT ranged from 4.75% to 9.4% compared to a range of 4.3% to 8.8% in the uncuffed groups. The available data found no statistically significant difference between cuffed and uncuffed ETTs with post-extubation stridor occurring with both types of ETTs.

There was a significant difference found between cuffed and uncuffed ETTs and the incidence of reintubation. It has been found that cuffed ETTs have a lower incidence of ETT exchange in contrast to uncuffed ETTs that more frequently need to be exchanged. Two studies reported reintubation rates with 1.97% to 7.2% of cuffed ETTs requiring replacement compared to 29.4% to 37.6% of uncuffed ETTs. One of the studies reporting reintubation rates involved pediatric burn patients. Burn patients in general are subject to multiple ETT exchanges due to airway edema related to their disease process. The disease process could be implicated in the statistically significant difference reported.

Implications for future practice include the need for vigilant monitoring of cuff pressures when using cuffed ETTs. It was found that over 60% of pediatric practitioners surveyed do not monitor cuff pressures in a recent survey by Sathyamoorthy et al..[6] Monitoring cuff pressures is one important factor in maintaining adequate blood flow to tracheal mucosa. Maintaining adequate blood flow is ensured when cuff pressures are below the capillary perfusion pressure of the tracheal mucosa. It has been noted via endoscopic examination that obstruction to mucosal blood flow occurred at a lateral pressure above 30cm H_2O. Intracuff pressure should therefore be maintained between 20 and 30 cm H_2O.[7] Blood flow to the tracheal mucosa can be compromised due to pressure with either cuffed or uncuffed ETTs. Cuffed ETTs allow measurement of the pressure exerted by the inflated cuff. Gopalakrishnan et al. proposed a novel method to continuously monitor ETT cuff pressure by attaching the cuff pilot balloon to an invasive pressure monitoring setup. Anesthesia practitioners can reduce damage to the trachea by continuously monitoring cuff pressures and maintaining adequate blood flow to the mucosa.[8]

Future research needs include studies with greater numbers of participants as well as studies performed in multiple clinical settings which will increase the generalizability of the results. The neonatal anatomy is unique and research in this population is lacking. Future research to determine a reliable and cost effective method to monitor ETT cuff pressures would be helpful to increase the utilization of cuff monitoring by the clinical practitioner.[6] Cuff pressure monitoring will become especially important if the trend continues to move towards the widespread use of cuffed ETTs in the pediatric population.[6,7]

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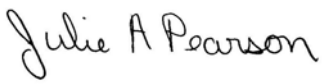
Mentor: Sharon Hadenfeldt, CRNA, PhD

Editorial

One of the things I really like about the International Student Journal of Nurse Anesthesia (ISJNA) is our ability to publish a wide range of reports, both in subject matter and complexity. A major purpose of the ISJNA is to introduce the nurse anesthesia graduate student to the peer review process in an encouraging, supportive manner. As such, we accept reports with a focus on basic, introductory concepts, as well as those covering rare or more complex content. We are also able to accept reports on cases that cover similar topics, sometimes even publishing them in the same issue. As we can attest to, every patient, and therefore every case, is different and offers an opportunity for learning. For that, I am grateful to every author who has submitted a report for publication.

Sincerely,

Vicki C. Coopmans, CRNA, PhD
Editor



Julie A. Pearson, CRNA, PhD
Associate Editor

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

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INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA
GUIDE FOR AUTHORS

MISSION STATEMENT

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

ITEMS ACCEPTED FOR PUBLICATION

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

ITEM PREPARATION & SUBMISSION

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

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

PEER REVIEW

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

General guidelines

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

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

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

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

Case Reports

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

Heading (see #9 above in General Guidelines)

[space]

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

[space]

Case Report (bold, 400-500 words)

[space]

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

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

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

[space]

Discussion (bold, 600-800 words)

[space]

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

[space]

References (bold)

[space]

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

[space]

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

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

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

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

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

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

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

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

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

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

AMA MANUAL OF STYLE

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

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

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

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

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

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

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

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

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

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

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

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

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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.

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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.**

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

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

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

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

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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:

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Webster University
470 E. Lockwood Ave. Suite 15
St. Louis, MO 63119

SUBMISSION CHECK LIST

___ **AMA Manual of Style and other format instructions are adhered to**.
___ Total word count not exceeded (1400 for case report, 500 for abstract, 3000 for EBPA).
___ The item is one continuous Word document without artificially created page breaks.
___ Verbatim phrases and sentences are quoted and referenced.
___ All matters that are not common knowledge to the author are referenced.
___ Generic names for drugs and products are used throughout and spelled correctly in lower-case.
___ Units are designated for all dosages, physical findings, and laboratory results.
___ Endnotes, footnotes not used.
___ Jargon is absent.

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

Case Report
___ Introduction is less than 100 words.
___ Case Report section states only those facts vital to the account (no opinions or rationale)
___ Case report section is 400-500 words and not longer than the discussion.
___ Discussion section is 600-800 words.
___ Discussion of the case management is based on a review of current literature
___ Discussion concludes with lessons learned and how the case might be better managed in the future.

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___ 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.
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