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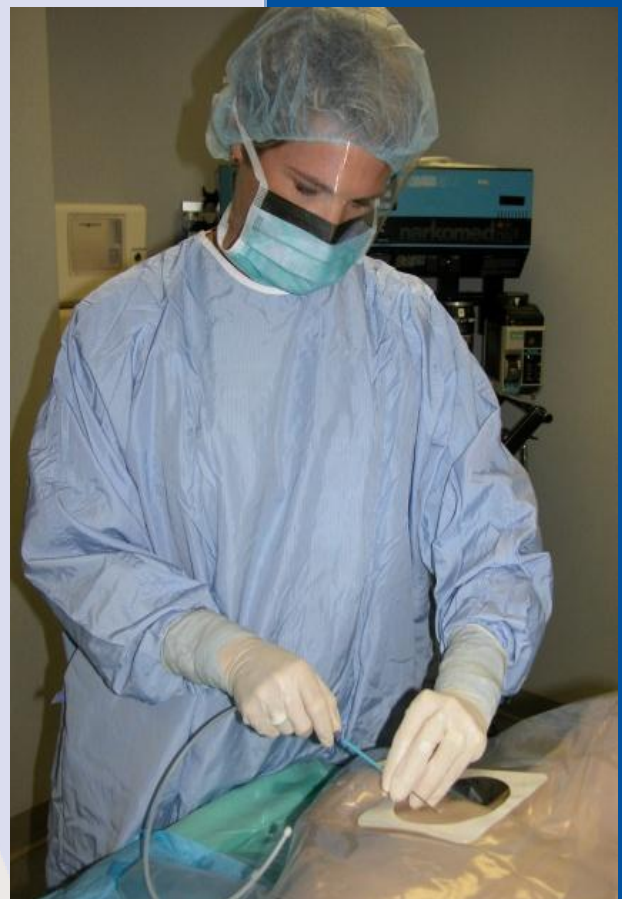
von Willebrand Disease

Emergence Agitation - Peds

Beach Chair Hypotension

Awake Fiberoptic Intubation

Delirium in Elderly Patients



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Front Cover: Lynne Edwards, BSN, student at the Wake Forest University Baptist Medical Center Nurse Anesthesia Program practices insertion of a central line.

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Heparin Resistance during Cardiopulmonary Bypass

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Key Words: Heparin Resistance, cardiopulmonary bypass, Antithrombin III, human AT III concentrate, coagulopathy

Systemic anticoagulation is utilized before cannulation in cardiopulmonary bypass (CPB) to prevent thrombosis. Heparin is the anticoagulant of choice in CPB due to its rapid onset of action and the ability to reverse its actions with protamine sulfate.¹ Activated clotting time (ACT) is the most widely used test to assess systemic anticoagulation after heparin administration in the setting of CPB. In most institutions, an ACT of 400 seconds or greater is required before cannulation and throughout the CPB period to ensure adequate anticoagulation.¹ Initial pre-CPB heparinization is achieved with an intravenous (IV) dose of 300-400 IU/kg.² Patients who require upwards of 500 IU/kg to prolong the ACT to > 400 seconds are referred to as having heparin resistance. Heparin resistance has been reported in as many as 20% of patients presenting for coronary artery bypass graft (CABG) surgery.¹

Case Report

A 67-year-old, obese male patient with a history of hypertension, hyperlipidemia, and peripheral vascular disease was diagnosed

with a non-ST-elevation myocardial infarction after presenting with complaints of chest pain and shortness of breath. Chest radiography revealed diastolic congestive heart failure. The patient underwent cardiac catheterization which showed severe 3-vessel coronary artery disease, with 90% occlusion of the left anterior descending artery and an ejection fraction of 35%. Upon admission, the patient was started on aspirin, beta blocker, and IV nitroglycerin therapies. Additionally, the patient was given continuous IV heparin infusion, which continued for six days and was discontinued on the morning of surgery. After proper diuresis and improvement of his pulmonary status, the patient presented for CABG surgery. Preoperative hemoglobin was 10.2g/dl and hematocrit was 33%. Platelet count was 285,000 mm³, and prothrombin and activated partial thromboplastin times were 14.5 seconds and 44 seconds, respectively. Baseline ACT was 154 seconds.

The patient underwent a smooth IV induction of general anesthesia, and oral endotracheal intubation was uneventful. Aminocaproic acid infusion was started at 40ml/hr for thirty minutes, and then continued at 4ml/hr throughout the case. Prior to CPB cannulation, 30,000 IU (300

IU/kg) of heparin was given into the right atrium by the surgeon. Three minutes later an ACT value of 352 seconds was reported. An additional 10,000 IU heparin IV was given, and repeat ACT value was 354 seconds. At this time, acquired Antithrombin (AT) III deficiency was suspected in this patient and 645 units of human AT III concentrate was administered by the perfusionist. Subsequent ACT reading after three minutes was 552 seconds. CPB commenced and continued for three hours with ACT values ranging from 407-512 seconds. The perfusionist utilized a dose-response curve for successive heparin dosing during CPB, and he reported administration of an additional 50,000 IU of heparin over the course of the three-hour CPB period.

Quadruple bypass grafting was completed, and the patient was successfully weaned from CPB. Heparinization was reversed with protamine sulfate 500mg IV, and post-reversal ACT value was 147 seconds. The patient was transported, intubated and sedated, to the cardiovascular intensive care unit. He was extubated the following morning and the remainder of his postoperative course was uneventful. He was discharged home on postoperative day six.

Discussion

Heparin works by binding to AT III, a naturally occurring thrombin inhibitor. The Heparin-AT III (H-AT) complex acts by irreversibly binding to thrombin, and thereby neutralizes the effect of thrombin on

fibrinogen and prevents thrombus formation.³ Heparin increases the rate of reaction between AT III and factors IIa, Xa, XIa, XIIa, and XIIIa; however, the anticoagulant activity of unfractionated heparin varies among commercially-prepared samples and patient response to weight-based dosing varies as well.² During CPB, there is unique activation of the coagulation cascade, and adequate anticoagulation is essential to prevent catastrophic thrombus formation.⁴ In the presence of known or suspected heparin resistance, adequate anticoagulation with the use of heparin alone may be unachievable.¹ When standard bolus dosing of heparin fails to produce adequate anticoagulation in a patient, heparin resistance should be suspected.

Experts utilize multiple theories to explain the causes of heparin resistance: heparin-induced thrombocytopenia, primary or secondary AT III deficiency, and enhancement of factor VIII activity.¹ The incidence of primary or congenital AT III deficiency has been reported to be as high as 0.05%;⁵ however, the majority of cases of heparin resistance are a result of secondary or acquired AT III deficiency. Occurrences of heparin resistance are often encountered in acute illness, cases of malignancy, during peri- and postpartum periods, and in the setting of drug-induced causes such as aprotinin and nitroglycerin therapies.⁴ Heparin therapy itself is also known to produce a decrease in circulating AT III levels which is independent of the initial dose and is detectable after one day.⁴ According to Tanaka et al., plasma AT III

levels decrease by 17-33% during IV or subcutaneous heparin therapy, and when AT III levels drop below 60% of normal values, the anticoagulant effects of heparin become insufficient.⁶

In the case of this patient, it is likely that the preoperative heparin infusion produced an acquired AT III deficiency; and, the baseline acquired deficiency was further compounded by hemodilution when CPB was instituted intraoperatively. As previously mentioned, nitroglycerin infusions have also been linked to the development of heparin resistance; however, the exact mechanism by which nitroglycerin produces resistance is still unknown. In this case, the concurrent nitroglycerin therapy may have also played a role in the patient's development of heparin resistance. In cases of known or suspected AT III deficiency, AT III supplementation is recommended to manage heparin resistance during CPB in patients undergoing open heart surgery.¹ Like all blood products, human AT III concentrate carries a risk of transfusion transmitted infection because it is made from human plasma; however, statistics have shown that AT III concentrate is safer for human administration than normal serum albumin.⁵ While, fresh frozen plasma (FFP) may also be given to restore AT III levels, a preparation of AT III concentrate requires simple reconstitution and can be ready for administration within minutes, thereby eliminating the thawing time associated with FFP administration.⁵ Additionally, the use of AT III concentrate, in place of FFP, significantly reduces the volume transfused to the patient.

This case serves to remind anesthesia practitioners that the possibility of heparin resistance exists in patients who have received preoperative heparin and/or nitroglycerin therapy. Patients with coagulopathies present many challenges to the anesthetist; and, although anticoagulation management of the patient on CPB is the primary responsibility of the perfusionist, anesthesia practitioners should understand the mechanisms involved in heparin resistance and the management of AT III deficiency.

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Laryngospasm Following Vocal Cord Injection

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Keywords: laryngospasm, negative pressure pulmonary edema (NPPE), vocal cord injection

Postoperative laryngospasm is a recognized complication related to anesthesia care. Laryngospasm is described as airway obstruction caused by a forceful involuntary spasm of laryngeal musculature due to sensory stimulation of the superior laryngeal nerve. This event can be caused by such stimuli as respiratory secretions, vomitus or blood in the airway.¹ Although laryngospasm occurs relatively frequently, early recognition and treatment decrease the potential for morbidity and mortality.² The following is a case of awake recurrent partial laryngospasm and its management in an adult male after general endotracheal anesthesia for a vocal cord injection.

Case Report

A 65-year-old, 72-kilogram, 5'9" male presented for direct laryngoscopy with vocal cord injection for left vocal cord paralysis related to recently diagnosed lung cancer. The patient reported that he had quit smoking 7 months prior, after a 15 pack-

year smoking history. He also stated that he suffered from gastroesophageal reflux disease that was well controlled with daily esomeprazole. Preoperative evaluation revealed Mallampati Class I pharyngeal visualization, full range-of-motion, thyromental distance of two fingerbreadths equaling approximately 4 cm, vocal hoarseness and clear breath sounds.

The patient was medicated with midazolam 2 milligrams (mg) and fentanyl 100 micrograms (mcg) upon entering the operating room. Standard monitors were placed and the patient was pre-oxygenated. Intravenous induction with lidocaine 80 mg, propofol 140 mg and rocuronium 50 mg was performed. Direct laryngoscopy was performed and a 5.5 mm ID endotracheal tube (ETT) was placed. The ETT placement was verified and positive pressure ventilation was initiated at a rate of 12 breaths per minute (BPM) with tidal volumes of 450 ml. Desflurane 8% with oxygen 2.0 L/min flow was used to maintain anesthesia. The procedure was completed without complication in 35 minutes.

Upon completion of the procedure, train-of-four (TOF) revealed 1/4 twitches. Neostigmine 4 mg and glycopyrrolate 0.8 mg were administered. The desflurane was discontinued and oxygen flows were increased. The patient maintained regular respiratory patterns with 16-18 BPM and 300-400 ml tidal volumes. The TOF was repeated and revealed 4/4 twitches with sustained tetany. The patient was slow to open his eyes but able to follow commands with a strong hand grasp and sustained head lift. Additional neostigmine 1 mg and glycopyrrolate 0.2 mg were given. The oropharynx was suctioned, removing large amounts of bloody secretions and the ETT was removed.

Following extubation, there was audible stridor and decreased air exchange. The patient was agitated and attempting to sit up. After approximately 60 seconds the patient was noted to be tachycardic with oxygen saturations decreasing to 90%. The patient was given propofol 100 mg and midazolam 2 mg and a 6.0 mm ID ETT was inserted. The patient was transferred to the post anesthesia care unit (PACU). Upon arrival he was attempting to sit up, with oxygen saturations of 100%. The ETT cuff was deflated with air exchanged noted. The ETT was removed after oral and pharyngeal suctioning. Stridor and respiratory distress were observed immediately with oxygen saturations 85-90%. Propofol 200 mg and succinylcholine 140 mg were given and a 6.0 mm ID ETT was inserted. Tachycardia was noted after intubation and resolved with esmolol 40 mg.

The patient was discharged from the PACU to the intensive care unit (ICU) for mechanical ventilation. Removal of the ETT was attempted in the ICU with recurring incidents of desaturation, resulting in reinsertion of an ETT. Two days following the procedure, the patient was taken to the operating room for vocal cord examination and removal of the ETT. The vocal cords were found to be mildly edematous, however the ETT was removed without complication. The patient was observed overnight and subsequently discharged home the following day.

Discussion

Laryngospasm is an event that occurs relatively frequently in the field of anesthesia.² A laryngospasm is described as a forceful and involuntary spasm of the laryngeal musculature that causes a closure of the vocal cords leading to an inability to ventilate.³ The risk of laryngospasm is increased by factors such as difficult intubations; nasal, oral or pharyngeal surgical site; and obesity with obstructive sleep apnea. It has also been found that laryngospasm is most likely to occur in the first 90 minutes after extubation for those cases involving intubation and mechanical ventilation.² Because laryngospasm is often caused by respiratory secretion, vomitus, or blood in the airway, it is very important to suction the oral and pharyngeal cavity prior to extubation.¹ It is important to note that while this patient was suctioned prior to

extubation, symptoms consistent with laryngospasm were still observed.

The most important aspect of dealing with a laryngospasm is early recognition and treatment. When recognition or treatment is delayed there is a very high risk for morbidity and mortality. Untreated or poorly managed laryngospasm has the ability to produce serious complications such as pulmonary aspiration and negative pressure pulmonary edema.² In this case early recognition of airway compromise and reinsertion of the ETT was crucial. The patient was given less than 120 seconds after extubation before the decision to reinsert the ETT was made.

This patient demonstrated the most common sign and symptom associated with a laryngospasm, which is desaturation. Some other conditions to be cautious of when laryngospasm is suspected include bradycardia, pulmonary edema and pulmonary aspiration.² While this patient did not experience all of the associated problems, he did exhibit audible stridor and an obvious decrease in air exchange. Aside from the symptoms accompanying this patient's respiratory distress, diagnosis and treatment was aided by the fact that the procedure involves manipulation of laryngeal tissue and therefore, produces a high suspicion of laryngospasm and surgical related airway trauma and edema.

According to literature, there are other associated complications of vocal cord injection that may present similarly to a laryngospasm. These most commonly

include transient airway compromise and laryngeal edema.³ Mild edema was noted on laryngeal exam on the third post-operative day. However, the vocal cords were not directly examined immediately post-operatively. Thus, it is difficult to determine if the cause of the edema was related to surgical factors or the multiple endotracheal intubations required. It is quite possible that surgical complications such as transient airway compromise and laryngeal edema contributed to the stridor and respiratory distress symptoms observed on post-operative extubation attempts.

The anesthesia related literature dedicated to laryngospasm and airway obstruction heavily discusses the complication of negative pressure pulmonary edema (NPPE).^{2,4,5} Post anesthetic laryngospasm has been identified as the most common cause of the development of NPPE. The development of hypoxemia up to 90 minutes after a laryngospasm usually indicates the occurrence of NPPE. The correct diagnosis must be made ruling out cardiogenic pulmonary edema and aspiration. A chest radiograph and serial arterial blood gases (ABG's) should be included in the work-up for suspected NPPE. Correction consists of supplemental oxygen, and in some cases prolonged intubation and mechanical ventilation.⁵

This patient had three attempts of ETT removal with development of airway obstruction and ETT reinsertion before successful extubation, producing a high risk for development of NPPE. His care included serial ABG's, an electrocardiogram (EKG),

a chest radiograph and a computed tomography (CT) of the thorax. The ABG consistently revealed a PaO₂ ranging from 179-503 mmHg. The EKG, chest radiograph and CT were not indicative of NPPE and showed minimal changes compared to previous examinations, causing little suspicion of NPPE.

This case had many similarities and many differences when compared to the literature. The ETT was removed appropriately after oral suctioning with the patient following commands, exhibiting full strength and regular respirations with adequate tidal volumes. Early recognition and intervention may have prevented severe complications. This case report demonstrates the significance of a laryngospasm and airway compromise and how important the diligence of an anesthesia professional can be in recognizing the event and providing prompt treatment.

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Use of LMA and Positive Pressure Ventilation

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Keywords: Laryngeal mask airway, positive pressure ventilation, difficult airway, difficult intubation, airway devices

An unanticipated difficult airway is a frequent cause of major anesthesia-related

morbidity.¹ The American Society of Anesthesiologists (ASA)-with the assistance of expert opinions and a consensus conference- published a difficult airway management algorithm to assist practitioners in managing this stressful event.^{2,3} In the

“cannot intubate and cannot ventilate” (CICV) scenarios, strategies for airway management require the use of airway devices conceived to facilitate tracheal intubation, to create a patent airway, or both. The laryngeal mask airway (LMA) is one such device and its use is discussed in the following case study.

Case Report

A 61 year-old male 68 inches tall, weighing 73.2 kilograms presented for an elective open left inguinal hernia repair. History of present illness was significant for inguinal hernia, without obstruction. Past medical history was significant for hypercholesterolemia, allergic rhinitis and cataracts. The patient had no known drug allergies and the only current medication was simvastatin for lowering cholesterol. He was evaluated as an ASA physical status II. Preoperative 12 lead EKG and lab values were unremarkable. Preoperative vital signs were blood pressure of 142/83, heart rate of 67, respiratory rate of 18 and oxygen saturation of 100% on room air. Physical exam revealed the patient had a Mallampati class II airway with good dentition and adequate neck flexion and extension. A 20 gauge peripheral intravenous catheter was inserted and midazolam 2 mg was administered.

When the patient arrived in the operating room, standard monitors were placed. The patient was oxygenated with 100% O₂ via a face mask with EtCO₂ monitoring capability. Anesthesia was induced with lidocaine, fentanyl, propofol and

rocuronium. Mask ventilation was easily achieved following induction. Direct laryngoscopy (DL) was performed with a Macintosh 3.5 blade by the student registered nurse anesthetist with a resulting, grade 3 visualization of the airway. The patient was repositioned with the head hyperextended upon which a second DL was attempted, still revealing a grade 3 view. The patient was then mask ventilated with 100% oxygen and the oxygen saturation remained 98%. A third DL was performed by the attending anesthesiologist, once again revealing a grade 3 view. An attempt to pass a gum elastic bougie was unsuccessful. The patient was once again mask ventilated with 100% oxygen with oxygen saturation remaining greater than 98% and a size 4 LMA was inserted successfully with positive EtCO₂, and equal bilateral breath sounds. The patient's respirations were controlled with positive-pressure ventilation (PPV) secondary to muscle relaxation.

The remainder of the case was uneventful. General anesthesia was maintained with sevoflurane and oxygen. Ventilation was maintained using PPV via the LMA throughout the case until a return of spontaneous ventilation near the conclusion of the procedure. Neuromuscular blockade was reversed with neostigmine 5 mg and glycopyrrolate 1 mg at the termination of the surgical procedure 1.5 hours after induction following the initial rocuronium dose of 50 mg. The patient was spontaneously ventilating tidal volumes of greater than 350 ml. on 100% oxygen, neuromuscular blockade was reversed with train of four 4/4, sustained tetanus > 5 seconds, and the LMA

was removed without incident. The patient was slow to follow commands and respond to verbal stimulus. While in the post anesthesia care unit physostigmine was given to reverse anesthesia with the patient subsequently responding appropriately to verbal stimulation, following commands, and remaining lucid. The patient was informed of his difficult intubation and given instructions to notify future anesthesia care givers.

Discussion

Fundamental to the safe conduction of anesthesia are airway management and endotracheal intubation skills. Difficult intubation may arise unexpectedly, despite the most thorough preoperative airway assessment. Difficult intubation can be anticipated in a number of circumstances including a previous history of difficult intubation, pathoanatomical states that involve the head and neck region as well as certain syndromes known to be associated with difficult intubation.⁴ The true incidence of difficult intubation is unknown but is estimated to be 1-3% while the reported incidence of CICV scenario is 0.01-0.07%.^{5, 6}

The traditional “gold standard” for establishment of a secure airway in a patient with a suspected or proven difficult airway has been an awake fiberoptic intubation.⁷ The safety and effectiveness of awake fiberoptic intubation has been illustrated by numerous authors.⁷⁻¹¹ However, there are many drawbacks of an awake fiberoptic intubation, including the need for a

cooperative patient. In addition, tachycardia, hypertension and oxygen desaturation have all been associated with an awake fiberoptic intubation.¹² Finally, a 55% incidence of patient discomfort has been reported with an awake fiberoptic intubation.¹²⁻¹³

Several devices and techniques are available to assist with a difficult intubation, one such being the LMA. The LMA is a supraglottic device intended for either spontaneous or positive pressure ventilation, designed specifically to aid in blind intubation and for use during general anesthesia.¹⁴ A clinical trial by G. Caponas, has confirmed the ease of its insertion as well as the reliable ventilation of almost all patients with its use.¹⁵ The advantages of the LMA over tracheal intubation include a more rapid insertion and an increased success rate.¹⁶⁻¹⁹ H. Joo et al. demonstrated that ventilation with the intubating LMA was successful in all patients within two attempts by the nominally experienced operator, an added advantage over tracheal intubation.⁷ Finally, the LMA may facilitate airway management when conventional tracheal intubation has been deemed difficult or impossible as in the CICV situation.^{20,21,22}

Inflation of the LMA cuff results in a low-pressure seal surrounding the larynx, thus enabling positive pressure ventilation with inspiratory pressures of 15-20 cm H₂O.^{23,24} The efficacy of the seal depends on the fit between the oval-shaped cuff of the LMA and the oval-shaped groove surrounding the glottic inlet.²⁵ Factors influencing the seal include selecting the appropriate size LMA, correct placement, avoiding over or under

inflation of the cuff, ensuring optimal fixation, and maintaining low compliance by monitoring neuromuscular blockade and the depth of anesthesia.^{25, 26} The proper and optimal cuff inflation for each patient should always be checked by anterior neck auscultation and ballottement of the pilot balloon. Appropriate size selection in combination with optimal cuff inflation, will produce a near-perfect fit, and allow for PPV.²⁷

Multiple studies support the use of the LMA with PPV.^{28,29,30} Devitt et al. demonstrated that ventilation through the LMA is adequate at ventilation pressures ranging from 15-30 cm of H₂O and is comparable to tracheal tube ventilation.²⁸ Berry and Verghese reported no leaks with tidal volumes of 10 ml/kg.²⁹ The effects of PPV on gastric insufflation using a nasogastric tube and the aspiration techniques were studied by Graziotti, who found no difference between the LMA and the tracheal tube.³⁰ All patients were ventilated to a mean peak airway pressure above 17 cm H₂O.³⁰ Recently, using size 4 and size 5 LMAs, Heinrichs et al. demonstrated the mean ventilation pressure which led to gastric insufflation was 28 cm H₂O (range of 19-33), and that the mean oropharyngeal leak was 31 cm H₂O (range 19-41). They imply that the margin of safety with regard to gastric insufflation was essentially comparable to that of the face mask, however, inherently inferior to that of a cuffed tracheal tube.³¹ Fascinatingly, the incidence of overall complications with PPV via LMA technique may be less than PPV via tracheal intubation technique.^{31,32} Haden

et al. used the PPV via LMA technique on 593 occasions and report only two significant clinical problems (0.3%).³² Interestingly, during that same period, there were three serious problems with the PPV via tracheal intubation technique in 187 uses (1.6%).³²

A considerable amount of experience and large scale studies support the practice of using positive pressure ventilation with the LMA in adults as well as children.^{23,24,28,33,34,35} In a prospective study of 11,910 LMA anesthetics, 5,236 patients underwent PPV with no cases of aspiration or morbidity reported.³⁵ Positive pressure ventilation using the LMA is a safe and effective technique with a low failure and problem rate when using modest tidal volumes and low peak inspiratory pressure to minimize leakage of gas around the LMA cuff in patients with normal pulmonary function. Although gastric insufflation is unlikely at modest tidal volumes, epigastric auscultation, and monitoring of the airway integrity, gas leak, and abdominal distention should be performed in all patients to ensure that gastric dilatation does not occur.^{25,34}

The sequelae of a difficult/failed intubation range from the trivial to loss of life; therefore every anesthesia practitioner should have a pre-learned strategy for dealing with this situation. This patient demonstrated an unanticipated difficult intubation and a tragedy was averted by the insertion of an LMA. When difficult intubation occurs, the problem should be clearly documented in the case notes indicating the precipitating events and

actions that were useful in resolving the crisis. In addition, the patient should be given notification to warn future anesthesia providers, as was done in this case. Finally, one might also consider the use of a Medic-alert bracelet for this serious event.

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Management of Patients with von Willebrand Disease in Elective Surgery

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Keywords: von Willebrand disease, von Willebrand factor, desmopressin, clotting factor concentrates, elective surgery

Von Willebrand disease (vWD) is the most common inherited bleeding disorder and is characterized by either quantitative or qualitative abnormalities of von Willebrand factor (vWF).^{1,2} The vWF plays a role in both primary and secondary hemostasis.^{2,3} Von Willebrand disease has a prevalence of about 1% to 3% of the general population often manifesting as a lifelong history of bruising and epistaxis.¹⁻³ Because patients with vWD are at an increased risk for bleeding, any surgical or invasive procedure has the potential to be hazardous and anesthesia providers must be knowledgeable in the management of these patients.

Case Report

A 50 year-old, 45-kilogram, ASA 2 female was scheduled for a biopsy and excision of axillary lymph nodes. The patient's past medical history was significant for small lymphocytic leukemia for two years, vWD, mild aortic regurgitation (AR), mild asthma, gastroesophageal reflux disease (GERD), and anxiety. Past surgical history included cesarean section, hysterectomy, neck biopsy, and tonsillectomy in which she required a transfusion for postoperative bleeding. The patient had no known history of complications with general anesthesia and

her social history was non-contributory. Current medications included omeprazole and albuterol.

The patient had never received chemotherapy or radiation for her small lymphocytic leukemia. She denied syncope or shortness of breath associated with her mild AR. The patient's last asthma attack was one year ago, where she received a nebulizer treatment in the emergency room. She reported using the albuterol inhaler only as needed. The patient reported her GERD symptoms were well controlled with taking omeprazole regularly.

The patient has been diagnosed with vWD since 2001. At age 21 years of age, she underwent a tonsillectomy which was complicated by postoperative bleeding. She reported receiving a blood transfusion without complications and denied any spontaneous bleeding since her diagnosis. Patient has been followed regularly by a hematologist, who recommended preoperative desmopressin (DDAVP).

Preoperative electrocardiogram and chest radiograph were unremarkable. Laboratory values were significant for slightly increased coagulation values including a PT of 14.5 seconds and PTT of 36.4 seconds, and a decreased platelet count of 129,000.

Physical examination the day of surgery was significant for a grade I/VI diastolic murmur heard loudest at the right upper sternal border; heart sounds were of regular rate and rhythm. Lung fields were clear on auscultation. Preoperative vital signs included a blood pressure of 130/81 mm of Hg, heart rate of 99 beats per minute, respiratory rate of 20 breaths per minute, and afebrile.

Preoperatively, the patient received desmopressin 15 mcg intravenously (IV), 0.3 mcg/kg,^{1,4} over 15 minutes prior to entering the operating room. The patient also received 2 puffs of albuterol via inhalation and midazolam 2 mg IV.

The patient was brought to the operating room, standard monitors applied, and oxygen at 10 L/min via mask was administered. Induction proceeded with fentanyl 150 mcg, ketamine 20 mg, lidocaine 100 mg, and propofol 100 mg. Mask ventilation was easily achieved, and rocuronium 50 mg was given IV for muscle relaxation. The trachea was successfully intubated, with confirmation via positive end-tidal CO₂ and bilateral breath sounds.

General anesthesia was maintained with sevoflurane, oxygen, and air. No further muscle relaxation was warranted for the type of surgical procedure. The patient remained stable throughout the procedure. Blood loss was monitored closely, with a total estimated blood loss at the end of the procedure of less than 5 ml. After the axillary biopsy was completed, neuromuscular blockade was reversed with

glycopyrrolate 0.4 mg and neostigmine 3 mg, extubation criteria were met, the trachea was extubated, and oxygen was delivered via face mask at 8 L/min. Patient was taken to the post anesthesia recovery area and eventually transferred to the ward.

The patient was seen on postoperative day one. Vital signs were stable overnight, no anesthetic complications and no signs of hematoma formation at the incision site were noted.

Discussion

Von Willebrand disease is the most common inherited bleeding disorder and affects both genders. Patients with vWD either are deficient or produce defective vWF. The vWF is responsible in primary hemostasis by adhering platelets to exposed endothelium.^{1,2} It also has a role in secondary hemostasis by protecting factor VIII against cleavage from proteolytic enzymes; without vWF only 10% of circulating factor VIII exists.² A 10-year retrospective review studied the management of patients with vWD in elective surgery.⁵ The review recommended that the management of these patients should be guided by the type and severity of vWD and the choice of treatment.⁵

There are three phenotypes of vWD that are recognized: types 1, 2, and 3, which is the most severe.^{3,4,6} Type 1 is a mild to moderate quantitative deficiency of vWF accounting for 60% to 80% of cases, resulting in decreased factor VIII levels from 5% to 30%.^{2-4,6} Treatment for these patients includes either intranasal or IV DDAVP.

Patients with type I vWD typically do not require exogenous vWF replacement.³ Type 2 accounts for 20% to 30% of cases and is a qualitative vWF deficiency which includes several subtypes.^{2-4,6} DDAVP is ineffective in most patients with type 2 vWD.³ Patients with the subtype, type 2B, may develop severe thrombocytopenia after receiving DDAVP.³ Type 3 is a severe, quantitative deficiency of vWF, with very low to undetectable levels of vWF and factor VIII.^{2-4,6} As a result, these patients have a significant defect in platelet plug formation, thrombin generation, and fibrin formation.³

Researchers identify two main forms of prophylactic treatment of patients with vWD for elective surgery: DDAVP and exogenous vWF contained in clotting factor concentrates (CFCs).^{4,5,7} DDAVP is a vasopressin analogue that releases vWF from endothelial cells; the recommended dose is 0.3 mcg/kg IV over 15 to 30 minutes with maximum effect occurring within 30 minutes.¹⁻⁸ An individualized response to DDAVP exists depending on the type of vWD; factor VIII and vWF levels can normalize in certain types of vWD and other patients may have an inadequate response to DDAVP.²⁻³ Therefore, a patient's response to DDAVP must be assessed prior to the invasive procedure.³ The specific type of vWD in our patient was not known at the time of surgery. However, her hematologist prescribed DDAVP as a prophylactic treatment, which implied that she had a type of vWD that was responsive to DDAVP administration.

The second form of prophylactic treatment is with CFCs, or vWF/factor VIII concentrates.^{3-5,7} CFCs are manufactured from human plasma and contain large amounts of vWF, antifibrinolytics, and platelets.⁵⁻⁶ In several studies, CFCs were used as prophylactic treatment prior to surgical or invasive procedures and the effectiveness of hemostasis was rated excellent in 82% to 100% of cases.^{3-5,7} Dosing and dosing intervals of CFCs were guided by factor VIII:C levels, vWF:Ristocetin cofactor activity, and clinical response.^{3-5,7}

The majority of case reports used DDAVP as prophylactic treatment since it is less expensive and has a higher safety profile in comparison to CFCs which is derived from pooled plasma.³ However, studies have shown that CFCs used as prophylactic treatment for patients with vWD have resulted in no serious drug-related adverse events or thrombotic events.^{3,4,7} Recombinant factor VIII should not be used as treatment because it does not contain vWF.⁶ The literature also recommends fresh frozen plasma, 20 to 25 ml/kg IV, as treatment of perioperative bleeding.⁶ DDAVP has been found to increase factor VIII and vWF levels 3 to 5 times above baseline.³ Also, in one retrospective review, DDAVP was given as prophylactic treatment in 38% of elective cases and the hemostasis was rated excellent in 91% of cases.³ Although this patient underwent a surgical procedure with relatively minimal blood loss risk, she appeared to have respond well to prophylactic DDAVP administration.

The management of patients with vWD can be challenging. Understanding the pathophysiology and performing a thorough preoperative assessment can aid in choosing the best prophylactic treatment. Both DDAVP and clotting factor concentrates have been found to achieve excellent hemostasis when used as prophylactic treatment prior to elective surgery.^{3-5,7}

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Emergence Agitation in a Pediatric Patient

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Keywords: pediatric emergence agitation, pediatric postoperative agitation, low solubility inhalational agents, pediatric delirium, pediatric surgery

Emergence agitation (EA) or postoperative agitation can be described as a dissociated state of consciousness with a condition of uncontrollable behavior or confusion upon emergence from general anesthesia.^{1,2} It is

estimated that EA after general anesthesia with inhalational agents ranges from 2% to 55% with a higher incidence in children between ages two to five.²

Postoperative agitation in children is characterized by inconsolable crying, incoherence, agitation, and an increased length of stay in the recovery room. Typically, these children do not recognize familiar objects or people.² It is thought that EA is more frequently related with the use of agents with a low blood gas solubility profile, such as sevoflurane or desflurane.^{3,4}

The greatest incidence of EA occurs during the first 30 minutes after emergence.⁵ It typically lasts 5-15 minutes and generally resolves spontaneously.⁵ Although brief in duration, the management of EA in the postoperative care unit (PACU) can be challenging.

Case Report

A 6 year-old, 23-kilogram (kg) male, ASA physical status II, with a medical history significant for primary iridocyclitis and vitreous degeneration, presented for bilateral eye examination under general anesthesia. His past medical history was otherwise negative. Past surgical history was negative. The patient was premedicated with oral midazolam at 0.5 milligram per kilogram (mg/kg), which produced a sedated, calm and cooperative patient, who separated well from his parents. He appeared calm during monitor placement and the application of a facemask. Smooth inhalational induction was initiated with nitrous oxide, oxygen, and gradual incremental flow of sevoflurane up

to 8%. An intravenous catheter was then placed without difficulty. A laryngeal mask airway (LMA) was inserted to secure his airway. In addition, rectal acetaminophen was administered for preemptive analgesia.

General anesthesia was maintained with nitrous oxide, oxygen, and sevoflurane. A total dosage of 30 micrograms (mcg) of fentanyl was administered during the procedure. His vital signs remained stable throughout the procedure. After an uneventful procedure, the LMA was removed while the patient was deeply anesthetized. The patient was then transported to the PACU in stable condition.

Upon awakening in the PACU, the patient was extremely agitated, inconsolable, combative, and repeatedly attempted to get out of bed. He was sedated with a bolus of propofol 20 mg IV for the safety of the patient and staff. An oxygen facemask at 6 liters per minute was then placed and the patient's airway was supported manually. His vital signs remained stable, and the parents were brought back to the PACU. Upon reawakening, he was calm and oriented. No further medications were required.

Discussion

Although the underlying cause of EA has not been determined, the incidence in the pediatric population has been attributed to multiple factors.⁶ These factors include: rapid return of consciousness, the type of surgical procedure, anesthetic technique, postoperative pain, child's age, and personality.⁶

A rapid recovery from sevoflurane has been suggested to play a potential role in increasing the incidence of EA.⁷ One possible explanation is that this agent depresses the central nervous systems inhibitory centers, resulting in alterations of neurotransmitters including serotonin, dopamine, and acetylcholine.⁶ This imbalance exerts an irritating effect on the central nervous system.⁶ Other studies suggest that the occurrence of epileptiform activity may be related to the use of sevoflurane, isoflurane, or desflurane but has not been associated with halothane.⁶ This may explain the low incidence of EA in patients receiving halothane intraoperatively.⁶

Certain anesthetic techniques are associated with an increased incidence of EA. A study done by Uezona, et al. demonstrated that preschool children receiving sevoflurane for maintenance of anesthesia had a 38% incidence of emergence agitation versus 0% with propofol alone.⁷ Additionally, rapid emergence from inhalational anesthesia to consciousness may increase apprehension in children experiencing unfamiliar surroundings.⁷

Specific surgical procedures in children have also been associated with an increased incidence of postoperative agitation. Although there are no scientific data to support this phenomenon, there appears to be an increased incidence of EA in procedures such as tonsillectomy, thyroidectomy, and eye examination under anesthesia.⁶ It has been suggested that a

sense of suffocation in these procedures may contribute EA.⁶

Inadequate postoperative analgesia has been postulated as a contributing factor in EA.⁴ The incidence of EA in patients receiving sevoflurane was significantly decreased by preemptive analgesia.⁴ A study done by Cravero et al. showed that patients receiving fentanyl intravenously at 1 mcg/kg, 10 minutes prior to the discontinuation of anesthesia, experienced a 44% decrease in EA compared to placebo.⁴ Intraoperative administration of intravenous ketorolac at 1 mg/kg was also effective in decreasing incidence of EA in patients undergoing otolaryngology procedures under sevoflurane anesthesia.⁸ Acetaminophen administered after induction of anesthesia has been found to be effective in decreasing agitation postoperatively.⁹ Other pharmacological agents, such as clonidine, have been shown to decrease agitation upon emergence from anesthesia in children undergoing circumcision.¹⁰ Although the use of clonidine at 2 mcg/kg following induction has been shown to be effective, its use has been associated with delayed awakening and an increased level of sedation following discharge.¹¹

Emergence agitation has also been observed in nonpainful procedures, such as magnetic resonance imaging (MRI) scanning.⁶ Additionally, children who had caudal blocks and adequate post operative pain relief showed no significant difference in the incidence of EA.⁶ From these findings, postoperative pain in children does not

appear to be an independent causative factor for EA.⁶

The incidence of EA in children undergoing anesthesia with sevoflurane is more common in preschool boys three to five years of age.¹² It is thought the preschool child's psychological immaturity, coupled with an unfamiliar environment, contributes to EA.¹² The physiologic immaturity of the hippocampus in the pediatric brain may also add to an increased susceptibility of younger children to EA.¹² In addition, younger children with separation anxiety or impulsiveness are more prone to this phenomenon.^{12,13} These predisposing factors of a child's temperament may increase the likelihood of postoperative agitation.^{12,13}

For this case, one or more factors may have contributed to the patient's postoperative agitation. The type of procedure itself posed a known risk factor for EA. Although preoperatively the child's demeanor did not indicate excessive anxiety, measures to prevent EA were taken to decrease the likelihood of EA. Preoperatively, the child was medicated with oral midazolam. In addition, preemptive analgesia with an acetaminophen suppository was administered after induction.

Intraoperatively, fentanyl was titrated during the procedure. Additionally, the patient was allowed to wake up slowly from anesthesia at the conclusion of the procedure. Despite all these measures, the child still experienced a period of agitation that required a rescue medication postoperatively. Propofol was selected as a rescue medication. In this case, the use of

propofol was effective, and the patient responded well. No further intervention was necessary, and the parents were reunited with the patient in the postoperative area.

The act of reuniting the patient with his family may also serve as a factor in decreasing EA. In a study done by Voepel-Lewis et al., 194 children who experienced EA were observed in the PACU to determine the efficacy of decreasing agitation based on nursing intervention.¹⁴ Voepel-Lewis et al. demonstrated that 80% of the nurses caring for agitated children would most often select analgesics or sedatives as their first line of intervention.¹⁴ However, they determined that the most effective intervention was reuniting the child with their parents.¹⁴ According to the study, agitation was alleviated in 57% of cases after reuniting the patients with their parents, as compared to a 42% decrease when analgesics or sedatives were selected as an initial treatment.¹⁴

The exact etiology of EA remains unknown. It does appear that multiple factors play a part in this condition. The child's temperament, specific surgical procedure, use of preoperative medication, type and technique of anesthesia, all play a role in the incidence of EA. Other factors, such as reuniting patient and family in a calm environment, are also important measures to consider in the pediatric population. Finally, postoperative rescue medications should be administered when patient and staff safety becomes an issue.

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Hypotension and Cerebral Perfusion in Beach Chair Position

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Keywords: Beach chair position, postural hypotension, cerebral perfusion, shoulder surgery.

The beach chair position was created over two decades ago specifically for shoulder arthroscopy surgeries. This position provides excellent surgical exposure, facilitates ventilation and provides easier access to the airway. The beach chair position is associated with a lower incidence of brachial plexus injuries compared to the lateral decubitus position used for shoulder surgery.¹ However, placing an anesthetized patient in an upright position causes significant hemodynamic changes, notably postural hypotension, which compromises cerebral perfusion pressure (CPP). Decreased CPP can lead to hypoperfusion and ischemia of the brain and spinal cord, resulting in devastating outcomes such as stroke, visual loss, quadriplegia and brain death.²

Case Report

A 55 year old male, ASA III, presented to the ambulatory surgery center for left shoulder arthroscopy secondary to rotator cuff injury. His medical history was significant for hypertension (HTN), non-insulin dependent diabetes mellitus (DM), gastroesophageal reflux disease (GERD), hyperlipidemia (HL) and obesity. The patient was taking pioglitazone HCL,

benazepril, ezetimibe/simvastatin, and olmesartan. He had not taken any medications the morning of surgery. His preoperative laboratory values and electrocardiogram were within normal limits. The anesthetic plan was general anesthesia with endotracheal tube (ETT) placement, and possible postoperative placement of an interscalene block. The patient's baseline vital signs were blood pressure(mean arterial pressure) [BP(MAP)] 133/83(99) mmHg, heart rate (HR) 71 beats per minute (bpm), and oxygen (O₂) saturation of 96% on room air.

The patient was medicated preoperatively with midazolam 2 mg intravenous (IV). After 5 minutes of 100% O₂ by mask, a rapid sequence induction with cricoid pressure was initiated. Propofol 200 mg, succinylcholine 100 mg, esmolol 50 mg, and fentanyl 50 mcg were given IV for induction of anesthesia. After confirmation of end-tidal carbon dioxide (ETCO₂) and bilateral breath sounds, the ETT was secured and sevoflurane was initiated at 4%. The patient had received 350 mls of crystalloid solution.

The patient was placed in the beach chair position, his head was secured to the head rest, and his pressure points were padded. His BP dropped to 106/72(83) mmHg and the sevoflurane was decreased to 2%. A subsequent BP reading was unobtainable.

Immediately, the BP cuff was assessed for proper functioning. Again, a BP reading was unobtainable while the HR remained 78-82 bpm. Sevoflurane was discontinued and ephedrine 10 mg IV was given. A BP of 78/39(52) mmHg was measured. Subsequent BP readings were: 83/44(57) mmHg, 100/54(69) mmHg, 120/78(92) mmHg. Sevoflurane was restarted at 2%. Approximately 90 seconds lapsed between the initial unobtainable BP reading and the BP reading of 78/39 mmHg. The patient remained in the beach chair position throughout.

The patient's vital signs remained stable throughout the surgery with the BP maintained between 100/60(73) mmHg and 142/90(107) mmHg. The BP cuff was located on the right upper extremity at all times. At the end of surgery, sevoflurane was discontinued, O₂ was delivered at 10 L/min and muscle relaxant antagonist was given. Upon meeting extubation criteria, the patient's trachea was extubated without incident. The patient was transferred to the post-anesthesia care unit awake, alert, oriented and moving all of his extremities. The patient was discharged home later that day with no anesthetic complication noted.

Discussion

The case report presents a patient who fortunately emerged from anesthesia without incident. The potential for a devastating outcome existed with the occurrence of significant postural hypotension when placed in the beach chair position.

Gravity attributes to the normal physiological changes that accompany the sitting position, and these changes are exaggerated under general anesthesia. Volatile anesthetics produce vasodilation and myocardial depression, causing a reduction in venous return (VR), cardiac output (CO) and stroke volume (SV). Normally, the body compensates by increasing the systemic vascular resistance (SVR), by up to 60%, to maintain a normal BP. In the anesthetized state, the body's autonomic response is blocked and the reduction in CO, VR, and BP are exaggerated.²

Normal cerebral blood flow (CBF) is 40-50 ml/100gm/min. Anesthetic drugs, CPP and autoregulation are determining factors of CBF. Cerebral perfusion pressure is calculated by the difference between MAP and intracranial pressure (ICP) (or central venous pressure (CVP) whichever is higher). Mean arterial pressure is the sum of diastolic blood pressure and one third the pulse pressure.³

Autoregulation is the intrinsic myogenic regulation of vascular tone, which maintains a constant cerebral blood flow (CBF) in the presence of changing CPP. When the MAP is between 50 to 150 mmHg, cerebral autoregulation is considered to be functioning adequately. Autoregulation is abolished when minimal alveolar concentration (MAC) is greater than 1, at which time CBF becomes directly proportional to MAP.^{2,3,4}

The lower limit of autoregulation (LLA) should be calculated per patient depending on cardiovascular and cerebrovascular risk factors.² Patients with essential HTN, especially those who are poorly controlled, will have a rightward shift of the autoregulation curve making them more vulnerable to cerebral ischemia when CPP decreases.³ These factors indicate the need for a higher MAP to ensure adequate cerebral perfusion. The LLA for non-anesthetized patients is 25% of their resting MAP. When MAP decreases greater than 40% of the resting value, symptoms of cerebral hypoperfusion may appear.²

In the sitting position, MAP at the brain is very different compared to the MAP at the arm or leg and is critical in determining cerebral perfusion. The difference is equal to the hydrostatic pressure gradient between the brain and the heart/arm or leg. To calculate the hydrostatic gradient, there is a 0.77 mmHg decrease for every centimeter gradient. For example, if the external auditory meatus (EAM) represents the base of the brain, the difference in height between the brain and the heart/arm is 20 cm. This represents a 15 mmHg difference between the MAP at the heart and the MAP at the brain.¹ Unfortunately, these calculations are not performed regularly on patients in the upright position and the significant hypoperfusion that results can potentially lead to visual loss and catastrophic neurological outcomes.^{1,5}

Pohl and Cullen discuss a case series of cerebral ischemia during shoulder surgery in the upright position. Four cases with

devastating outcomes are presented, that most likely resulted from inadequate cerebral perfusion secondary to postural hypotension. Major brain or spinal cord ischemia occurred in relatively healthy, middle aged individuals with extremely low to no risk factors for cerebrovascular events.²

One case describes a healthy 47 year old female who underwent shoulder arthroscopy in the beach chair position. Her baseline BP was 125/83 mmHg. An intraoperative SBP of 80 to 90 mmHg was maintained. Postoperative, the patient did not awaken and remained in a vegetative state with a Glasgow coma scale of 3. An MRI was suggestive of cortical infarcts in bilateral cerebral hemispheres.²

Another case presented by Pohl and Cullen, describes a 54 year old female who underwent a similar procedure. Two years prior, she ruled out for a myocardial infarction. Her cardiac evaluation was normal. The BP cuff was placed on the lower extremity secondary to a history of mastectomy. Deliberate hypotension was attained with nitroglycerin and labetalol boluses, maintaining a SBP of 70-100 mmHg. A drop in SBP to 50 mmHg was treated with phenylephrine. Postoperatively, the patient was unarousable and apneic. She was declared brain dead. An autopsy revealed upper spinal cord and medullary infarcts.² The use of deliberate hypotension should be weighed carefully and avoided in high risk patients who require a normal or higher perfusion pressure.²

There is little known about the potential adverse outcomes for sitting position in shoulder surgery compared to the more studied neurosurgical procedures. Shoulder surgeries are less complex and typically shorter in duration. Despite the low incidence of reported complications with the beach chair position for shoulder surgery, catastrophic outcomes may arise, as evidenced by compelling case reports.²

In the presented case report, there are many factors that contributed to the significant hypotension that occurred when the patient was placed in the beach chair position. The patient had received only 350 ml's of IV fluid before positioning. Due to the patient's NPO status, estimated fluid deficit, and chronic relative hypovolemia secondary to essential HTN, 500 to 1000 ml's of crystalloid solution should have been administered before induction of anesthesia and positioning. For less dramatic hemodynamic changes, more careful titration of inhaled anesthetic should have occurred along with a more gradual incline to the beach chair position.⁴

In summary, always consider baseline MAP, past medical history and LLA. Calculating the hydrostatic pressure gradient in BP cuff placement will allow for adequate assessment of cerebral perfusion. Maintaining a neutral head alignment will

promote CBF and avoid mechanical obstruction of vessels. To avoid perioperative complications related to cerebral hypoperfusion, one should carefully titrate inhaled anesthetics, maintain an adequate intravascular volume, aggressively treat hypotension with vasopressors, avoid deliberate hypotension and always maintain vigilance.^{1,2}

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Awake Fiberoptic Intubation Using Dexmedetomidine and a Mucosal Atomization Device

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Keywords: Fiberoptic Intubation, Dexmedetomidine, Atomization Device, Awake Intubation

A difficult airway is defined as when a skilled anesthesia provider experiences difficulty with face mask ventilation, difficulty with tracheal intubation, or both. The principal adverse outcomes associated with the difficult airway include death, brain injury, cardiopulmonary arrest, unnecessary tracheostomy, airway trauma, and damage to teeth.¹ In the general population the incidence of difficult tracheal intubation in anesthesiology is approximately 6%.² To avoid the problems associated with an unrecognized difficult airway, an airway history and physical examination should be conducted, whenever feasible, prior to the initiation of an anesthetic care and airway management in all patients. Any description of a difficult airway on a previous anesthesia record is suggestive that difficulty may reoccur. Therefore, examination of previous anesthetic records may yield useful information about airway management.¹ Armed with this information, the anesthesia provider can then formulate a primary and secondary anesthetic plan ensuring availability of specialty equipment, supplies and medications. The American Society of Anesthesiologists (ASA)

Difficult Airway Algorithm is an invaluable tool used to guide the anesthesia provider in making such plans. In a cooperative patient with a previous history of an awake fiberoptic intubation, it would seem reasonable to perform an awake fiberoptic intubation for any future procedures if time permits.

Case Report

A 23-year old active duty white male, weighing 66 kg, height of 69 inches, ASA physical status 1E, with no significant medical history, presented to the main operating room for an exploratory laparotomy to rule out a possible small bowel obstruction. His medications included antibiotics, a multivitamin, stool softeners, and a morphine patient controlled analgesia (PCA) pump that was discontinued upon his arrival in the preoperative holding area. The patient had no known drug allergies. His surgical history included a laparoscopic cholecystectomy two days prior for which he had an awake fiberoptic oral intubation which was noted on the previous anesthetic record. No anesthetic complications were noted. Preoperative physical exam for the second procedure revealed: a Mallampati class III airway, thyromental distance of less than 3 cm, interincisor distance of less

than 3 cm, and full range of motion of the atlanto-occipital joint.

In the preoperative holding area, midazolam 1mg IV for anxiolysis and glycopyrrolate 0.4mg IV as an antisialagogue were administered. The patient was given a tongue depressor with approximately 2 ml of 5% lidocaine ointment and instructed to place ointment as far back on his tongue as possible and work the ointment back by using a swallowing motion. A dexmedetomidine bolus of 66mcg IV was administered over 10 minutes and then a continuous infusion at 13.2mcg/ hour was initiated. The patient was transferred to the operating room (OR) and moved to the OR table and placed in semifowlers position. Standard monitors were applied and baseline vital signs were obtained. The larynx was then topicalized with 2 ml of 4% lidocaine using a Mucosal Atomization Device (MAD). The trachea was then topicalized using a transtracheal block. First, a small skin wheel was placed over the cricothyroid membrane (CTM) using 0.5 ml of 1% lidocaine. Subsequently, a 20 gauge IV angiocatheter, attached to a syringe, was then inserted through the CTM in a slightly caudad direction while aspirating to confirm placement in the trachea. Once air bubbles were visualized in the syringe, the needle was removed and 3 ml of 4% lidocaine was injected via the angiocatheter on end exhalation. Once it was determined that the gag reflex was blunted, a flexible fiberoptic scope with a pre-positioned 7.5 endotracheal tube (ETT) was inserted into the oropharynx and advanced until the

vocal cords were identified. After passing the fiberoptic scope through the glottic opening, tracheal rings were visualized and the ETT was advanced over the scope. After the cuff was inflated, ETT placement was confirmed by the presence of end tidal carbon dioxide, equal chest rise and fall, bilateral breath sounds and condensation in the ETT. Once the patient's airway was successfully secured, the dexmedetomidine infusion was discontinued and propofol 100mg and rocuronium 40mg IV were administered to induce general anesthesia. The remainder of the case proceeded without incident. During post-operative follow up the next day the patient had no recall of the events and was discharged to home four days later.

Discussion

Awake fiberoptic intubation can be achieved using a conservative sedation plan that along with proper topicalization blunts airway reflexes, maintains spontaneous ventilation, and provides conscious sedation. Short-acting and easily-titratable medications are excellent choices for this intensely stimulating, but usually brief airway manipulation during fiberoptic tracheal intubation.³ The success rate for a fiberoptic guided intubation is 87-100% when used on either a known difficult airway, or a routine airway.¹ After performing an airway exam and reviewing the previous anesthetic record it was determined that the patient was at high risk for a difficult airway and a fiberoptic intubation would provide the safest method of securing his airway. The decision to

proceed with an awake versus asleep fiberoptic intubation was made because the patient was deemed an aspiration risk related to his current condition of possible small bowel obstruction following his previous surgery.

The use of airway blocks to anesthetize the upper airway structures allows for a more comfortable passage of the flexible fiberoptic scope into the trachea. With the introduction of disposable products such as the MAD, the need for more invasive blocks such as the superior laryngeal and glossopharyngeal nerve blocks are decreasing. The use of atomizers provides an excellent method for localizing the airway rapidly and safely in patients who are at high risk for a difficult airway.⁴

However, to prevent toxicity, the anesthetist must calculate the total amount of local anesthetic that a patient can receive when performing several different airway blocks. The patient in this case received approximately 305mg of lidocaine which is below the maximum recommended dose of 5mg/kg or 330mg⁵. Lidocaine is rapidly absorbed from the upper airway, tracheobronchial tree, and alveoli into the bloodstream, with peak blood concentrations usually reached in 20 to 40 minutes after application.⁶ By the time lidocaine peaks the patient is usually anesthetized and cannot inform the anesthetist of common signs of toxicity such as metallic taste in mouth and ringing in ears; therefore the anesthetist must remain vigilant for the signs of CNS toxicity, such as tachycardia, muscle twitching, convulsions and respiratory

arrest throughout the procedure. However, this may prove difficult since these signs and symptoms can be blurred or rendered nonexistent by other pharmacologic adjuncts. In addition to topical anesthetics, sedation can be administered and the use of dexmedetomidine, an alpha-2 adrenoreceptor agonist is an excellent choice because it is free of significant effects on respiratory function, it reduces anesthetic requirements, and it has analgesic, sedative and sympatholytic properties.^{7,8}

In patients who have a known or suspected difficult airway, successful endotracheal intubation can be performed with a flexible fiberoptic scope when suitable conditions are provided. Dexmedetomidine is one useful option for sedation. Adequate airway anesthesia may be provided with airway blocks or topical anesthesia through mucosal atomizers and transtracheal lidocaine.

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Anesthesia Related Delirium in Elderly Patients

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Keywords: elderly, geriatric, postoperative delirium, anesthesia, hip fracture

Management of geriatric patients is becoming a major topic in anesthesia because of the rate at which the population is aging. The number of people over the age of 60 years worldwide is estimated to triple from 606 million to 2 billion by the year 2050.¹ It is essential that clinicians understand the age-related changes that occur in order to provide safe and effective treatment to this growing population. Delirium is one of the most common postoperative complications in the elderly. It leads to increased hospitalizations, placements in skilled-care facilities, poor recoveries and higher costs.²

Case Report

The patient was an alert, oriented, self sufficient 89 year old male who weighted 72 kilograms. He had sustained a hip fracture and was scheduled to have a total hip

replacement. His past medical history included hypertension, chronic renal insufficiency, mild aortic stenosis, and anemia. Labs were within normal limits except for the following: blood urea nitrogen 55mg/dl, creatinine 2.9mg/dl, hemoglobin 9.1g/dl, hematocrit 27%. The electrocardiogram showed a heart rate of 70 with a first degree atrioventricular block. His medication regimen included hydrochlorothiazide, lisinopril, prazosin, verapamil and metoprolol.

The morning of surgery, the patient was given ketamine 40mg IV and fentanyl 12.5mg intravenously before receiving a subarachnoid block (SAB) in the holding room. A continuous spinal catheter was placed and dosed with bupivacaine 12.5mg, epinephrine 200mcg and fentanyl 20mg. After receiving sedation for the SAB, assessment by the anesthesia practitioners revealed an extremely confused and combative patient. The subarachnoid block

had taken effect and the patient's systolic blood pressure was 160mm/Hg. The plan was to re-dose the spinal as needed, give ketamine for sedation and keep his systolic blood pressure greater than 140mm/Hg. Ketamine 10mg intravenously was given twice. His blood pressure remained stable, but he continued to be agitated. A propofol infusion was started at 10mcg/kg/min with a 0.1% mixture of ketamine. The patient slept comfortably and remained stable throughout the surgery. He received a total of 4mg ketamine and 40mg propofol via the infusion.

On the third postoperative day, the patient was seen in his room. His vital signs were stable, but the patient remained confused. His son reported the patient had been delirious over the past 2 days, but was improving. The patient was going to be transferred to a rehabilitation facility later in the week.

Discussion

Delirium is a temporary change in mental status developing over a short period of time associated with a reduced level of consciousness, memory impairment and perceptual disturbances.³ The incidence of elderly patients with hip fractures developing delirium after surgery is over 20 percent.³ Although delirium is usually reversible, it is associated with increased hospital stays and nursing home placements.⁴ It is important to recognize patients who are at risk for developing delirium such as those who are male, immobile, malnourished, have more than three medications added in a hospital stay, or experience a general anesthesia.³

The patient's age and past medical history placed him at a high risk for developing postoperative delirium. The anesthetic plan

to provide regional anesthesia via a spinal catheter in hopes of providing hemodynamic stability and decreasing chances for developing delirium is supported by the literature. A study by Edelstein et al.⁴ found a lower incidence of postoperative delirium in patients who received spinal anesthesia than those who received general anesthesia. General anesthesia is more likely to cause substantial hemodynamic changes and cerebral hypoxia. A continuous spinal catheter in an elderly patient allows slow titration of local anesthetics to achieve a desired level of anesthesia with minimal hemodynamic changes; therefore, decreasing the likelihood of cerebral ischemia.⁵

Opioids have been shown to enhance a subarachnoid block without dramatically decreasing the patient's blood pressure.⁵ Tsui et al.⁵ suggests the addition of 20 mcg of fentanyl to low-dose bupivacaine to provide analgesia for elderly patients having total hip surgery. Alternatively, clonidine has shown to increase the duration of blockade, provide analgesia, and decrease the amount of local anesthetic used without causing great hemodynamic changes or an increase in the incidence of delirium.⁵ As was done in this case, starting with low-dose local anesthetics, adding an opioid, and giving doses incrementally via a spinal catheter is a very effective and safe way of managing a subarachnoid block in the elderly patient and will help to decrease the risk of postoperative delirium.⁵

The risk of developing delirium in the postoperative patient is more influenced by the medications given during the perioperative period than the patient's age.¹ When preparing patients for regional anesthesia, the judicious use of sedation is essential. The elderly patient is more sensitive to drugs acting on the central

nervous system than younger patients due to the alterations in physiology that occur with age, such as a decrease in number and function of receptors, decreased hepatic metabolism and decreased renal function.⁶ Sedatives used in the elderly should be short-acting, have a high margin of safety, and no active metabolites. Morphine is metabolized to morphine-6-glucuronide which may cause prolonged respiratory depression.⁶ Meperidine also has an active metabolite that possesses central nervous system toxicity and may cause seizures.⁶ Long-acting benzodiazepines such as diazepam should be avoided.⁵ It is recognized in the literature that propofol, midazolam, and ketamine are the best choices for sedation during regional anesthesia in the elderly population as long as dosages are decreased and titrated carefully with each patient.⁵ Low-dose propofol is often tolerated well due to its rapid metabolism; however, numerous studies have shown a decreased clearance and increased sensitivity of propofol in the elderly population so careful titration is essential.⁶ Ketamine is a nonbarbiturate agent that has dissociative and analgesic properties, but it does not cause respiratory or cardiac depression at subanesthetic doses.⁷ Ketamine is known to produce emergence delirium in a large population, but it is recommended for patients with aortic stenosis to maintain cardiac stability.⁷ When ketamine is used to supplement propofol, as used in this case study, it provides a more rapid onset and improved level of sedation for surgery while helping to maintain cardiac and respiratory stability.⁵

Due to the patient's age, decreased renal function, immobilization after surgery and the dissociative effects of ketamine, the patient suffered three days of delirium. Unfortunately, there is no single anesthetic

technique has shown to be perfect for the elderly patient. The selection of medications should be based on the risks and benefits for each patient.⁶ No changes would be made to this case. The benefit of hemodynamic stability ketamine provides outweighs the increased risk of delirium in this patient. Overall, it was a challenging anesthetic. The research supports the drugs and dosages administered throughout the case; however, in the future more judicious doses of ketamine will be administered to elderly patients. Educating the patient's family on the increased risks of postoperative delirium in this patient before surgery would have been beneficial for their understanding of his mental state in the postoperative period.

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