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Front Cover: D.Julie Medori, BSN (left) and Mary Hudson, BSN (center), graduate students enrolled in the University of Pennsylvania Nurse Anesthesia Program, participate in a simulated operating room fire scenario with instruction by Donna Wargo, MS (right).

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Nasoendotracheal Tube Obstruction

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Keywords: nasal intubation, nasoendotracheal obstruction

Nasoendotracheal intubation is the preferred method of securing an airway for many surgical procedures, and the practitioner must be aware of the potential complications associated with this technique. Several complications have been attributed to nasoendotracheal intubation such as submucosal or retropharyngeal dissection by the endotracheal tube, nasopharyngeal abrasions and lacerations resulting in epistaxis, bacteremia, and maxillary sinusitis.[1-4] It is imperative that the practitioner recognize the problem and intervene in a timely manner.

Case Report

A 49 year old, 147 cm, 76 kg female with Down's syndrome was scheduled for dental restoration. The patient's history included congestive heart failure, mitral regurgitation, pneumonia, hypothyroidism, gastric reflux, hiatal hernia, cholecystectomy, and recent urinary tract infection. Medications included levothyroxine 125 mcg, esomeprazole 20 mg, paroxetine 20 mg, and risperidone 2 mg. The patient had taken amoxicillin 500 mg orally as directed by the surgeon prior to arrival at the hospital. The patient had no known allergies.

Airway assessment revealed limited mouth opening, thyromental distance of 4 cm, with a Mallampati 3 class airway. Caregivers of the patient reported no recent upper respiratory tract infections, nasal drainage, cough, or fever. Lungs were clear to auscultation. The patient was transported to the operating room where standard monitors were applied. Propofol 150 mg was administered intravenous to get the patient positioned for surgery safely. The patient was preoxygenated with 100% oxygen; phenylephrine nasal spray was instilled in the right naris. Fentanyl 100 mcg, propofol 50 mg, and succinylcholine 100 mg were administered intravenously for induction of anesthesia. A well lubricated 5.5 nasal Ring-Adair-Elwyn endotracheal tube that was presoaked in warm sterile saline solution was inserted without difficulty with direct visualization of the vocal cords. No secretions were visualized, and the patient did not require suctioning of the airway. The cuff was inflated on the nasoendotracheal tube; bilateral breath sounds were auscultated, positive end tidal waveform was noted on the monitor, pulse oximetry was 100%. The tube was securely taped in place.

Anesthesia was maintained with an end tidal desflurane concentration of 5% with oxygen and air at a two liter total flow. The patient's vital signs remained stable. Within five minutes of intubation, the peak airway pressures increased from 24 to 45 mmHg. The end tidal carbon dioxide waveform was disappearing intermittently. Pulse oximetry decreased from 100% to 96%. At this time the patient was removed from the ventilator and manually ventilated with some difficulty with every other breath. Breath sounds were audible and clear bilaterally. The nasotracheal tube was removed without prior suctioning. A mucus plug was noted at the end of the nasotracheal tube. The patient's oxygen saturation remained greater than 92%, and all other vital signs remained stable. The patient was mask ventilated with 100% Oxygen, and a 7.0 mm endotracheal tube was placed orally without incident.

Surgery proceeded and was uneventful. The patient was transported to the recovery room on 4 liters nasal cannula, and discharged to home after meeting discharge criteria.

Discussion

Nasal intubation carries additional risks: hemorrhage, turbinate fractures, laceration of nasal mucosa, and dissection of the adenoids. These risks can be minimized by using a smaller endotracheal tube, preferably a Ring-Adair-Elwyn, that is well lubricated and softened in warm sterile water, instilling a vasoconstrictor intranasal prior to nasal intubation, and never forcing a nasotracheal tube during insertion.[5, 6] Mechanical dilation using different sized nasopharyngeal airways has also been advocated prior to nasotracheal intubation to reduce trauma and hemorrhage; however, others have shown this technique to be detrimental, causing more hemorrhage.[4] We did utilize all of these precautionary measures, except mechanical dilation. We choose a 5.5 nasal Ring-Adair-Elwyn in this patient rather than a size 6.0 because the patient had Down syndrome.[7] Patients with Down syndrome have smaller craniofacial and airway proportions, narrow nasopharynx, and an increased incidence of laryngotracheal and subglottic stenosis. Craniofacial and other anomalies in Down syndrome patients make them more susceptible to anesthesia related complications.

Increased airway pressures and elevated end expired carbon dioxide levels are signs of a partial airway obstruction. A rise in end tidal carbon dioxide without change in the waveform morphology may be associated with hypoventilation, a leak in the ventilator circuit, partial airway obstruction, rising body temperature, or absorption of carbon dioxide from an exogenous source.[2,3] In this case a partial airway obstruction, which was rapidly diagnosed and treated with reintubation, was secondary to the mucous plug acting like a valve on the end of the endotracheal tube. Upper and lower respiratory problems are common in the Down syndrome population. Factors affecting the airways include obesity, hypotonia, glossoptosis, increased secretions, midface hypoplasia, cardiac disease, small upper airway volume, and large tonsils and adenoids. These findings place this population at increased risk of pulmonary problems. A thorough history and physical is of the utmost importance. Caregivers in this case denied recent upper respiratory infection, cough, or nasal discharge, and the patient's lungs were clear to auscultation. During intubation there was no mucus visualized.

When faced with an airway obstruction in the endotracheal tube, whether it is a partial or total obstruction, a decision needs to be made promptly. We choose to remove the endotracheal tube without prior suctioning. The feeling was that we may dislodge the obstruction, advancing it further down the trachea or bronchial tree, and waste valuable time thus compromising the patient. Bronchial suction is not always effective in removal of endotracheal tube obstruction. Park et al. reported repeated suction attempts without success, followed by flexible fiberoptic bronchoscopy which dislodged a clot causing an endotracheal tube obstruction. Foreign body obstruction must also be taken into consideration when dealing with mentally handicapped adults or children. These two populations are prone to inserting foreign bodies into their nose and ears. When ventilation is impossible immediately after nasotracheal intubation, the only method to improve the situation is to remove the tube, keeping in mind that an airway obstruction may be the result of a dislodged foreign body.[8]

Increasing peak airway pressures and end tidal carbon dioxide concentration serve as early warning signs of airway obstruction. In this case it rapidly detected potentially disastrous obstruction of the airway system prior to the patient experiencing respiratory or cardiac arrest.

Mucoid impaction is a well described phenomenon that most commonly results from the inspissations of mucus and other secretions within a bronchus.[2] This is most often seen in patients with bronchogenic carcinoma, asthma, chronic bronchitis, or cystic fibrosis, which results in excessive or abnormally viscous mucous production. This patient did not have any of these comorbidities. Mucoid impaction is not uncommon in the perioperative period.[1] Differential diagnosis to be considered includes pneumothorax, perforation of the trachea or bronchus with an intubating stylet, barotrauma caused by over distention of the pulmonary tissue, large pulmonary embolism, or a mainstem bronchus intubation.[1] An intermittent capnography waveform and increasing end tidal carbon dioxide concentration, with increasing peak airway pressures, clear equal breath sounds bilaterally, without hemodynamic compromise ruled out these differential diagnoses.

A mucus plug is a possibility despite the patient having no documentation of reactive airway disease or recent upper respiratory tract infection, or mucus present in the airway during intubation. The key here is the early recognition of increasing peak airway pressures and increasing or intermittent capnography waveform.[1-3, 9] With early detection catastrophes can be avoided.

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Ehlers-Danlos Syndrome

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Keywords: Ehlers-Danlos Syndrome, connective tissue disorder, hypermobility, skin extensibility, tissue fragility

Ehlers-Danlos Syndrome (EDS) is a rare, heterogeneous group of inheritable connective tissue disorders first described in the early 1900's by two physicians named Edward Ehlers and Henri-Alexandre Danlos. EDS affects males and females of all races and ethnicities at a rate of 1 in 5000. The condition is characterized by easy bruising, bleeding diathesis, joint hypermobility, skin extensibility, and tissue fragility. EDS results from a genetic alteration in collagen and can affect skin, ligaments, tendons, bones, fascia, eyes, blood vessels, arteries, and hollow organs. There are six primary types of EDS; the Classical, Hypermobility, Vascular, Kyphoscoliosis, Arthrochalasia, and Dermatosparaxis.[1] Presentation of Ehlers-Danlos syndrome in the preoperative setting challenges anesthesia professionals to consider the systemic implications of the disease and how it will affect the patient's anesthetic.

Case Report

A 54 year old male presented for left spermatocelectomy. He weighed 75 kilograms and his height was 69 inches. He was diagnosed with EDS two months prior. His past medical history consisted of hypertension, hypoglycemia, diverticulitis, and skin cancer. ASA physical status was II and his past surgical history consisted of left total hip replacement and a cervical laminectomy, both of which were uneventful. He had a known drug allergy to intravenous iodine. His daily medication regimen included lisinopril, trazadone, clonidine, and propecia. The day prior to surgery, the patient was prescribed to take e-aminocaproic acid in two, 5 gram doses, orally. Laboratory study results included hemoglobin of 15.9 g/dl, hematocrit of 45.1%, and platelet count of 288 mm^3.

The anesthesia practitioners met the patient for the first time on the day of surgery. A brief physical exam was performed. Joint mobility and appearance of tissue were within normal limits with no evidence of bruising. DDAVP, 22.5 milligrams, was given per an 18-gauge intravenous catheter over 30 minutes. The patient entered the operating room 30 minutes post infusion. Midazolam, 2 milligrams (mg), and fentanyl, 50 micrograms (mcg), were administered intravenously (IV). A pulse oximeter, non-invasive blood pressure cuff, and a 5-lead electrocardiogram were applied. He was preoxygenated per face mask. Intravenous induction followed with lidocaine, 100 mg IV, propofol, 400 mg IV, and fentanyl, 50 mcg IV. A size 3.5 laryngeal mask airway (LMA) was inserted without difficulty. Sevoflurane was initiated at 3% and titrated throughout the procedure to maintain hemodynamic stability. Oxygen flow was reduced to one liter and air was added at one liter flow. Prior to surgical incision the patient received cefazolin, 1 gram IV. Intraoperatively fentanyl was administered at 25-50 mcg IV (total 150 mcg) and titrated according to patient response. Ondansetron 4 mg IV was given 30 minutes prior to completion of the procedure. At the end of procedure when the patient was responsive to verbal commands, the LMA was removed. The patient was

transported to the post anesthesia care unit by stretcher and was discharged to home later that day.

Discussion

This case study illustrates three areas of concern for the anesthesia practitioner. Positioning of these patients for induction, laryngoscopy, and during the surgical procedure is a primary concern for the anesthesia professional. These patients experience musculoskeletal discomfort, increased incidence of osteoarthritis, and joint hypermobility, instability, dislocation, and subluxation in the shoulders, spine, elbows, wrists, fingers, knees, and ankles. This patient was assessed preoperatively for potential problems with body positioning and joint intactness. Positional changes can lead to tachycardia, bradycardia, hypotension, or vasovagal syncope secondary to autonomic neuropathies.[2]

EDS patients may have characteristic soft velvet-like skin with fragile skin prone to bruising and tearing. IV catheter patency and extravasation may be difficult to monitor secondary to extreme skin distensibility. Multiple attempts at laryngoscopy should be avoided to avoid pharyngeal and esophageal trauma. Pulmonary tissue fragility is related to an increased incidence of pneumothorax, and high ventilatory pressures should be avoided.[3] Airway manipulation and tissue trauma were minimized by use of an LMA and pressure controlled mechanical ventilation in this patient.

Ehlers-Danlos patients have a high frequency of bleeding inconsistent with normal hematologic studies. This clinical feature is generally explained by defects in the structural integrity of vascular and perivascular tissues and reduced attraction of platelets to injured subendothelium, not coagulopathy. However, coagulation measures and platelet levels should be assessed preoperatively. Even with normal bleeding times, clotting times, and platelet count some patients may still require blood transfusions and intravenous alimentation. Preoperative laboratory studies for this patient were within normal limits. While the surgical procedure was associated with low risks of bleeding, the patient's hematologist elected to treat the patient preoperatively with hemostatic agents.

Studies for EDS showing DDAVP to help control bleeding are relatively new.[4] DDAVP is an antidiuretic hormone known to increase endothelial cell release of Von Willebrand factor, factor VIII, and plasminogen activator and its effects are rapid and last several hours. Dosage is 0.3 micrograms per kilogram intravenously. This drug should be given slowly as hypotension or hypertension may result. Wait 30-60 minutes after administration before surgical incision.[5] Another therapeutic modality includes e-aminocaproic acid which is an antifibrinolytic that produces a structural change in both plasminogen and plasmin. The conversion of plasminogen to plasmin is inhibited and plasmin is prevented from degrading fibrinogen and fibrin. Aminocaproic acid can be administered intravenously or orally at a dosage of 5 grams for the initial loading dose and 1.25 grams per hour. Maximum daily dosage is limited to 30 grams. The dual action of these drugs decreases fibrinolysis and the formation of degradation products of fibrinogen and fibrin.[6] Anesthetic practitioners should avoid regional anesthesia and should be prepared for excessive surgical bleeding.[4]

Due to varying classifications and degrees of presenting symptoms in Ehlers-Danlos syndrome each patient needs to be evaluated and treated with individualized care. This patient was well educated on his syndrome and facilitated discussion between the surgical team, anesthesia practitioners, his diagnosing physician, and a hematologist. This level of communication and understanding of the syndrome is important for all patients when planning for surgery. Preoperatively the patients' coagulation and bleeding should be evaluated and optimized as necessary. All care providers should be aware of these patients' tendencies to bleed and close assessment is required both intraoperatively and postoperatively. This patient did not have complications with his joints and skin, but thorough assessment should be completed in all patients so that proper precautions can be made in the operating room prior to induction and surgery. Ehlers-Danlos syndrome, while rare, holds implications for anesthesia care and planning. Through knowledge and planning anesthesia practitioners can optimize outcomes in this unique population.

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Urgent Appendectomy in the Patient with Hemophilia and HIV

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Keywords: appendectomy, Factor VIII, hemophilia, human immunodeficiency virus

Approximately 7% of the United States' population will undergo appendectomy for appendicitis, and an additional 10% undergo appendectomy for incidental reasons in their lifetime.[1,2] In contrast, hemophilia afflicts 0.0001% of viable male births.[3] Although appendectomy has become routine, the

hemophilia patient offers particular challenges. In this case, preemptive treatment with antihemophilic factor VIII enabled a satisfactory surgical course without complications. The pathophysiology of hemophilia is reviewed, as are considerations for the immunocompromised patient. The roles of factor VIII replacement and hematologic management are also described.

Case Report

A 51 year old Caucasian male, 73 inches tall and 75.7 kilograms (kg), was admitted through the emergency department with abdominal tenderness radiating from the lower right abdominal quadrant, McBurney's Point. Past medical history was significant for hemophilia A, hepatitis C (HCV), Human immunodeficiency virus (HIV) diagnosed in 1986, systemic hypertension, idiopathic cardiomyopathy, gastroesophageal reflux disease, hiatal hernia, esophageal strictures, and alcohol abuse. The surgical history was negative for anesthetic complications.

Current medications included activated recombinant factor VIII (rFVIIIa), abacavir, amlodipine, benazepril, bupron, carvedilol, esomperazole, eszopiclone, lamivudine, spironolactone, tenofovir, and zidovudine. A complete blood count and metabolic panel revealed low platelets (143,000/mm^3) and elevated aspartate aminotransferase (AST) of 51 IU L^{-1}, consistent with mild liver damage. Blood pressure was 141/92, temperature 99.0 F; other vital signs were within normal limits. An electrocardiogram suggested prior inferior infarct, as evidenced by the presence of Q waves, and a prolonged QT interval. An echocardiography study obtained 6 months prior to presentation to the OR displayed an ejection fraction of ~ 45%. The patient stated that he has required frequent rFVIIIa infusions in the past due to physical trauma inherent to his occupation. Prior to rFVIIIa availability, blood transfusions were routine, and, therefore, the likely source of his HCV and HIV infections. An American Society of Anesthesiologists' Physical Status Classification of 4 was assigned to this patient.

Two 18 gauge peripheral intravenous (IV) lines were started and rFVIIIa 50 units/kg was given as a bolus in the emergency department. Upon transport to the operating room an rFVIIIa continuous infusion was started. Standard monitors were applied and the patient was pre-oxygenated with FiO_2 1.0. Cricoid pressure was applied and rapid sequence intravenous induction was accomplished with fentanyl 100 mcg, propofol 160 mg, and succinylcholine 100 mg. An 8.0 mm cuffed endotracheal tube was successfully placed under direct laryngoscopy. Anesthesia was maintained with 5% end-tidal desflurane with 2 liters per minute oxygen fresh gas flow. The patient was warmed by forced air blanket and temperature was monitored intranasally. Cisatricurium 6 mg was given IV for muscle relaxation maintenance. Lactated ringers 1500 ml was given for intravascular volume repletion and ephedrine 15 mg IV was given for hemodynamic maintenance. Upon closure of the surgical incision, muscle relaxation was antagonized with neostigmine 4 mg and glycopyrrolate 0.6 mg IV. The patient was extubated when awake and with the head elevated. No anesthetic complications were noted. The patient was discharged to home on post-operative day 2.

Discussion

Hemophilias A and B are X chromosome linked recessive traits, which result in deficiencies of clotting factors VIII and IX

respectively.[3] Factor VIII is necessary for the conversion of factor X to Xa by the intrinsic clotting mechanism.[3,4] Severity of Hemophilia A is described by three primary forms according to the level of factor VIII deficiency: severe, moderate and mild. Those with severe factor VIII deficiency suffer from spontaneous bleeding starting in childhood.[3,5] Those with moderate deficiency, 1%-5% normal factor VIII activity, have significant risk of bleeding in association with surgery or trauma.[3,4,5] Bleeding associated with severe and moderate forms may result in hematomas which can create airway, circulatory, or neurologic complications.[5] Those with mild forms, 6%-30% normal factor VIII activity, rarely suffer life threatening spontaneous bleeding, and might initially present with prolonged bleeding after surgery.[3,4,5]

Redwine and Bold recently described a case involving the perioperative care of a patient with hemophilia A having undergone an urgent appendectomy.[4] Of paramount importance is the risk of intractable perioperative bleeding in the patient with hemophilia A. In the case of major surgery, a therapeutic factor VIII plasma level of 0.5 U/ml must be maintained at all times and throughout the postoperative recovery period.[4,5] One unit of rFVIIIa/kg body weight will increase factor VIII activity by 2%, or the plasma level by 0.02 U/ml.[4,5,6] Repeated or continuous dosing is required due to the 8 hour half life of rFVIIIa.[5] In the presented case, a moderate hemophilia may be improved to normal levels pre-operatively by infusing 40-50 U/kg of rFVIIIa product. Factor VIII levels are subsequently maintained with a continuous infusion of rFVIIIa at 3-4 U/kg/hr. If intra-operative hemostasis is easily achieved, continuous rFVIIIa infusion may be discontinued in lieu of intermittent IV injection of rFVIIIa every 8 to 12 hours.[4,6] It is possible, however, to develop inhibitory antibodies to exogenous FVIII and the proteins used in its delivery. As a result rFVIIIa may be ineffective in up to 33% of cases.[4] It is incumbent upon the anesthesia practitioner to recognize the limitations of rFVIIIa in some patients, which is often manifest as uncontrolled bleeding or anaphylaxis after rFVIIIa administration. Appropriate treatment may include FVIII concentrate or recombinant activated factor VII (rFVIIa) for heavy bleeding.[6,7] Desmopressin acetate (DDAVP), aminocaproic acid, and cryoprecipitate may be used for less severe bleeding.[5,6] Intermittent boluses of rFVIIa with a dose of 90mg/kg should be given every 2-6 hours until hemorrhage subsides.[4] DDAVP 0.3 mcg/kg may be used to further increase factor VIII levels.[5] The initial dose of aminocaproic acid is 100-200 mg/kg IV bolus, followed by continuous infusion of 33.3mg/kg/hr for 5-7 days.[6]
Persons with hemophilia have been found to have an 11% incidence of HIV infection.[8]

While HIV and associated immunocompromise are worthy concerns in the patient undergoing elective surgery, their importance is limited for the patient undergoing urgent procedures. HIV is an infective retrovirus which destroys T lymphocytes. Reduced serum levels of T lymphocytes results in cell mediated immunodeficiency and proliferation of opportunistic infections and cancer.[9] The patient should be protected from nosocomial pathogens as best as possible.

Prophylaxis for pneumocystis carinii pneumonia should be considered in the immunocompromised patient receiving tracheal intubation, and is accomplished with the administration of dapsone, a

bacteriostatic oral agent.[9] An open surgical technique may be preferred as development of an intra-abdominal abscess is nearly three times more common after laparoscopic appendectomy; procedure times average 14 minutes longer when compared to open appendectomy.[10] Universal precautions for body substance isolation should be carefully observed. In the presence of active AIDS, a terminal cleaning of the operating theater and ventilator may be necessary after the case.[9]

The HIV infected patient may develop immune thromcocytopenic purpura, further compromising coagulability.[3] Thrombocytopenia secondary to HIV infection places the surgical hemophilic patient at further risk of bleeding complications. Platelet replacement should be considered when platelet count falls below 100,000 x 10^9/L.

Intraoperative invasive temperature monitoring must be placed with care in the patient at risk for nasal hemorrhage or esophageal varices. Pre-operative medications should be administered by the intravenous route to avoid unnecessary intramuscular injection and the associated risk of bleeding.[5] Regional anesthesia is relatively contraindicated due to an inherent risk of bleeding.[5] The choice of drugs, such as muscle relaxants, should be considered in light of co-existing hepatic disease due to viral infection or alcoholic cirrhosis. Precise dosing of rFVIIIa is essential for optimizing outcomes in the hemophilia patient.

Therefore, consultation with a pharmacist is important concerning a medication that may not be often used in a community hospital. Major surgery is safe for persons affected by hemophilia A through the proper use of rFVIIIa infusion. The surgical course of this patient with hemophilia A was optimized with an rFVIIIa bolus dose and subsequent continuous infusion. Debate persists regarding the optimal mode of postoperative rFVIIIa delivery. Intermittent bolus dosing can be done at home, which allows early discharge from the hospital. Continuous infusion maintains both a steady therapeutic level and conserves this valuable resource. The primary consideration for the hemophilia patient undergoing surgery for acute appendicitis is control of bleeding.

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Anesthetic Management of Hypoplastic Left Heart Syndrome

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Keywords: anesthesia, Glenn procedure, hypoplastic left heart syndrome, surgery

Hypoplastic left heart syndrome (HLHS) and other complex single ventricles refer to congenital abnormalities of the left-sided heart structures including the left ventricle, mitral valve and aortic arch.[1,2,3] These changes result in a univentricular heart and is unvaryingly fatal in infancy without surgical intervention.[1,2,3] The incidence of HLHS is reported to be between 0.016% to 0.036% of all live births, but until the advent of new surgical techniques in the 1970s, these children few options.[1,2] Heart transplantation and staged palliative surgery have become the standards of care raising the 15 and 25-year survival rates to 75% and 60%, respectively with the survival rate improving with each passing decade.[3,4] It is essential that the anesthesia professional understand the anatomical and physiological changes unique to HLHS in order to develop an anesthetic plan for these individuals as they present for non-cardiac surgeries.[3,4,5]

Case Report

A 6 year-old, 17kg, ASA physical status IV, female presented for dental extractions and restorations under general anesthesia. Her past medical history was significant for HLHS status post Damus-Kaye-Stansel procedure as a newborn and bi-directional Glenn procedure at 8 months of age. Her preoperative exam was notable for a room air oxygen saturation of 85% with periods of desaturation into the 70s noted with activity. The patient's medication included captopril and blood pressure on the day of surgery was 98/51 mmHg. The most recent echocardiogram results indicated a single ventricle of right ventricular morphology, an ejection fraction of 50%, mild tricuspid regurgitation and trace semilunar valve regurgitation. The most recent cardiac catheterization report indicated normal pulmonary arterial pressures. A cardiac anesthesiologist was consulted preoperatively for recommendations about specific anesthetic management techniques; these included antibiotic therapy for subacute bacterial endocarditis (SBE) prophylaxis, maintenance of ventilation near the patient's baseline and avoidance of myocardial depressants such as propofol.

Following premedication with oral midazolam, the patient was taken to the operating room and monitors were placed for pulse oximetry, blood pressure and electrocardiogram (EKG). The patient was

observed to be in sinus rhythm with an oxygen saturation of 86%. Inhalation induction was initiated by increasing the sevoflurane concentration up to 8% in 30% oxygen. Oxygen saturation upon induction fell as low as 77% and oxygen concentration was briefly increased to 60%. Peripheral intravenous access was secured and a 10 ml/kg bolus of Lactated Ringers was infused over the next 10 minutes. The patient's nose was prepared for nasal intubation using phenylephrine spray and dilating nasal airways. Prior to intubation, vecuronium 0.1mg/kg and fentanyl 1mcg/kg were given. Upon direct laryngoscopy, a moderate amount of blood was noted in the oropharynx. Successful nasotracheal intubation was achieved and proper placement of the nasotracheal tube was confirmed. SBE prophylaxis was provided with ampicillin and gentamicin. The patient's lungs were mechanically ventilated and end-tidal carbon dioxide levels were maintained between 36-40 mmHg. Sevoflurane 2.5% was provided in an oxygen concentration that was titrated between 21-27% in order to maintain oxygen saturations between 85-91%. Vecuronium 0.5 mg and additional doses of fentanyl (3mcg/kg total) were given. The patient received a total of 350ml of Lactated Ringers throughout the case. An hour and half into the anesthetic the muscle relaxant was reversed and spontaneous ventilation resumed. Morphine 0.5 mg was given, as well as ondansetron 2mg. The trachea was extubated without incident and the patient was taken to the recovery room with oxygen on standby. Following an uneventful stay in the recovery room, the patient was discharged to home without complications.

Discussion

Neonates with HLHS will appear healthy as long as there is a patent ductus arteriosus to provide systemic circulation.[2] Once the ductus closes, these patients present with hypoxemia, acidosis and shock.[2] Cardiac transplantation is an option, but organ procurement is limited in this age group.[2] The more common treatment plan involves a series of three, staged, palliative surgeries that change the cardiac anatomy in order to provide balance between the systemic and pulmonary flows.[2,3,4] The timeline for the surgeries is typically as follows-- Norwood procedure is performed at birth, birdirectional Glenn procedure at 6-8 months of age, and Fontan procedure at 18 months to 4 years.[2]

The patient in this scenario did not have a Norwood procedure, but instead had a Damus-Kaye-Stansel procedure at birth which entails an anastomosis of the end of the proximal pulmonary artery to the side of the ascending aorta.[6] Blood flow to the pulmonary arteries is then reestablished by a graft from the subclavian artery or from the thoracic aorta.[6] The patient went on to have a bi-directional Glenn procedure which channels blood from the superior vena cava (SVC) to the main pulmonary artery and removes the previous systemic to pulmonary graft.[2,3,4] This procedure improves exercise tolerance, however cyanosis and right to left shunting persist because blood from the inferior vena cava (IVC) still drains directly into the right atrium and bypasses the lungs.[2,3,4] For this reason, patients typically go on to have the Fontan procedure in which circulation from the IVC is also routed to the pulmonary arteries through a conduit.[2,4] This will result in oxygen saturations near 95% while breathing room air and a commensurate increase in activity tolerance.[2,3,4] The patient in this case was unique in that she was 6 years old and had yet to undergo the Fontan procedure. Clinical notes indicated that the patient's

family was hesitant to proceed with another operation.

Goals of anesthetic management for patients with cavopulmonary anastamoses rely on an understanding of the surgical changes to the anatomy in order to balance the pulmonary (Qp) and systemic (Qs) circulations.[3-7] A Qp:Qs ratio of slightly <1 is considered optimal in order to promote systemic tissue oxygenation.[3,7] Since pulmonary circulation is entirely passive, it is paramount that the anesthetist understand what factors can effect PVR and how these can alter blood flow through the heart.[3,4] The anesthetist's manipulation of ventilation is pivotal to the exploitation of PVR; hypoxia or hypercarbia induce pulmonary vasoconstriction while hyperoxia or hypocarbia induce pulmonary vasodilation.[3,4,7] Other factors that increase PVR are positive pressure ventilation (PPV), positive end-expiratory pressure (PEEP), acidemia, pain, and sympathetic stimulation.[4] Indeed, the negative intrathoracic pressure during spontaneous ventilation is the principle driving force for blood flow through the cavopulmonary anastamosis.[3]

Other considerations for the patient with a cavopulmonary anastomsis include a thorough preoperative evaluation, adequate premedication, and appropriate intraoperative monitoring.[4-8] Historical information obtained from the patient should elucidate the status of the cardiorespiratory system and the anesthetist should be alert for any symptoms of congestive heart failure, cyanosis or activity intolerance.[5] The most important risk factors of negative outcomes for children with congenital heart disease following non-cardiac surgery were found to be treatment for congestive heart failure, the presence of cyanosis or pulmonary hypertension, age less than 2 years, and procedures involving the respiratory or nervous systems.[8] Cyanosis, as observed in this patient, is to be expected from pulmonary hypoperfusion and right to left shunting.[4] When cyanosis is present, goals include maintaining balance over the PVR and systemic vascular resistance (SVR), avoiding dehydration, minimizing increases in oxygen consumption, and maintaining adequate hematocrit for oxygen delivery.[3,4,5]

Premedication with an anxiolytic is recommended in order to decrease the oxygen consumption that accompanies a child crying and struggling during anesthetic induction.[5,9] This has been shown to be safe in children with congenital heart disease and does not worsen hypoxia.[9] Antibiotic therapy for endocarditis prophylaxis was indicated in this case scenario because complex congenital heart disease is associated with a high risk for infection, and dental procedures remain the most frequent portal of entry.[3,4,5] Notably, end-tidal carbon dioxide monitoring has been found to demonstrate large individual variations from Pa_{CO2} in the presence of right to left shunting and should be monitored for trends.[4,5,10] Pulse oximetry has been shown to be inaccurate at oxygen saturations below 80% and to overestimate the true value.[5,11] EKG monitoring is essential in order to monitor the patient's heart rate and rhythm, and arrhythmias are poorly tolerated in these patients.[4,5]

Sevoflurane was chosen in this instance and has been shown to produce fewer dysrhythmias, cause less myocardial depression and be more easily titrated than halothane in children with congenital heart disease.[12] Blood pressure should not be monitored on the side of a previous Blalock-Taussig (B-T) shunt due to the distortion of the subclavian arterial anatomy and although the patient in this scenario did not have a previous B-T shunt, the blood pressure cuff

was placed on her leg during the case.[5] Transesophageal echocardiography (TEE) is usually only indicated during cardiac surgeries and was therefore not used in this case.[13]

Laboratory studies were not indicated for this outpatient procedure, however there are certain abnormalities that may be associated with HLHS. Following the bi-directional Glenn procedure, hepatic venous blood drains to the IVC and bypasses the lungs.[4] This is the proposed mechanism of injury responsible for pulmonary arteriovenous malformations that frequently form 5-15 years following the procedure.[4] Hepatic dysfunction and chronic cholestasis is commonly associated with changes in both the anticoagulant and procoagulant systems; the patient in this scenario bled easily following nasal instrumentation of her airway.[4] The adaptive mechanism to chronic hypoxia is polycythemia, which can predispose these patients to thrombosis while hypovolemic.[3,4,5]
As more patients with HLHS survive longer, anesthetists can expect to see them present more frequently for non-cardiac surgeries. It is important for the anesthesia professional to take the lead in order to provide the appropriate management and safe outcomes for these patients.

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Pediatric Laminectomy for Tethered Spinal Cord

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Keywords: tethered spinal cord, occult spinal dysraphism, somatosensory evoked potentials (SSEP), motor evoked potentials (MEP), TIVA

Occult spinal dysraphism includes various congenital abnormalities involving the spinal cord.[1] Tethered spinal cord is due to a "stretch induced functional disorder," that results in the caudal spinal cord being fixed to "inelastic structures".[2] A "thick rope-like filum terminale", a threadlike spinal cord prolongation, occurs at, or distal to, the L2 level.[3] It results in both motor and sensory problems of the lower extremities, as well as urologic, musculoskeletal and orthopedic problems.[2] Surgical intervention, via lumbar laminectomy and detethering, is the treatment of choice.[2] The goal of this case study is to discuss the anesthetic management of tethered cord surgery in pediatric patients.

Case Report

A 19 month old female, 10.5 kg, 75 cm, American Society of Anesthesiologists (ASA) physical status class 2, presented for a 2-level lumbar laminectomy and release of a tethered spinal cord with somatosensory evoked potential (SSEP) and motor evoked potential (MEP) monitoring. The patient was born full term without complications. The patient's past medical history included neonatal hyperthyroidism, for which the child was treated from birth to 6 months and has currently been resolved, iron deficiency anemia, constipation and lower extremity motor delay. During evaluation of the lower extremity motor delay, the current diagnosis was made via magnetic resonance imaging (MRI). The patient had no known drug or food allergies, nor any prior surgical history. The current medications included iron. On physical exam the patient was unable to ambulate without assistance, and had asymmetrical gluteal creases. The remainder of the physical exam, laboratory values, and vital signs were unremarkable. The patient's airway classification was a Mallampati 2.

Fifteen minutes prior to surgery midazolam 5 mg was given orally. Standard monitors were applied and general anesthesia was induced by facemask with 4 L/min oxygen, 7L/min nitrous oxide, and sevoflurane titrated to 8%. After an intravenous (IV) line was secured, propofol 20 mg and fentanyl 10 mcg were given. No neuromuscular blockade was administered. Using a Wisconsin #1 laryngoscope blade, the trachea was intubated with a 3.5 cuffed endotracheal tube (ETT). An orogastric tube was then placed and a second IV line secured. Nitrous oxide and sevoflurane were discontinued and propofol 200 mcg/kg/min and remifentanil 0.05 mcg/kg/min infusions were initiated for maintenance. A bispectral index (BIS) monitor was placed, with readings maintained between 40 and 60. The patient was then positioned prone with no pressure on the eyes, ears, nose, or

genitalia. Endotracheal tube placement was again confirmed with bilateral breath sounds and positive end-tidal carbon dioxide ($EtCO_2$). The remifentanil infusion was then increased to 0.1 mcg/kg/min for the remainder of the case. Gentamycin 25 mg, ampicillin 250 mg and dexamethasone 5 mg were administered prior to incision.

There were no surgical complications and blood loss was approximately 50 mL. The propofol and remifentanil infusions were discontinued thirty minutes prior to extubation. A total of propofol 220 mg and remifentanil 110 mcg were infused. The patient was awake for tracheal extubation which occurred without complications. The patient was stable and transported to the post-anesthesia care unit (PACU) with oxygen via facemask. She experienced no postoperative complications and was discharged to home on post-operative day two.

Discussion

Preoperative considerations in this patient population must include assessing for any congenital problems, especially those involving the respiratory, cardiovascular, or neurologic systems, along with any allergy problems.[3] These patients can be at increased risk of developing latex allergies after frequent exposure to latex gloves worn by the surgical team.[3] This case was performed in a designated latex-free neurosurgery room.

Induction of anesthesia can be accomplished by either inhalational or intravenous routes.[3] A nondepolarizing neuromuscular blocking agent such as rocuronium (0.6 mg/kg) or vecuronium (0.1 mg/kg) can be used.[3,4] Succinylcholine (1.5-2 mg/kg) can be administered if SSEP or MEP monitoring is utilized by the surgeons.[4] A large bore IV should be placed as blood loss can range from 50 to 1000 mL.[3,5]
Cardiovascular changes in the prone position include a decrease in cardiac index (CI), and mean arterial pressure (MAP) due to an increase in systemic vascular pressure (SVR) and pulmonary vascular resistance (PVR).[6] Compression of the inferior vena cava (IVC) leads to an overall decrease in cardiac output (CO).[6] This can be further complicated by a compressed abdomen, which can result in increased bleeding due to the diverting of venous drainage back to the heart by small, thin-walled veins that become distended from the increased pressure.[6] Chest rolls help to prevent abdominal compression. Respiratory changes include an increase in functional residual capacity (FRC), and redistribution in ventilation and perfusion throughout all lung fields.[6] Complications include carotid or vertebral artery occlusion, which can be prevented by maintaining neutral neck alignment.[6] Brachial plexus injuries can be avoided by proper positioning of the upper extremities.[4] Dependant areas, such as the ears, eyes, nose, and genitalia should be well padded.[3]

In SSEP monitoring, electrical potentials are evoked from the sensory system.[3] Motor evoked potential monitoring is achieved through stimulating motor areas of the brain or spinal cord via direct root stimulation.[3] Therefore, maintenance of anesthesia must be conducive to accurate SSEP and MEP readings so the surgeons can differentiate between neurologic dysfunction and normal stimulation during the surgery.[3] Increased intracranial pressure decreases SSEPs, while causing a small increase in MEPs.[7] Anemia suppresses SSEPs, while there is no evidence on its effects in MEPs.[7] Hypotension and spinal cord ischemia, hypoxemia, hypercarbia, and hypothermia all decrease SSEPs and MEPs.[3,7] The

halogenated inhalational agents all cause suppression of SSEPs and can completely obliterate MEP recordings.[7] However, SSEP and MEP recordings can tolerate up to 0.5 to 0.75 minimum alveolar concentration (MAC) of the volatile agents.[8] Nitrous oxide also suppresses SSEPs and MEPs, but nitrous oxide, up to 60%, is tolerable.[8] If no inhalational agent is desired, a total intravenous anesthetic (TIVA) technique can be utilized.

Neuromuscular blockers have minimal effect on SSEPs, but have a great effect on MEPs, as discussed by Sloan and Heyer.[7] Ketamine increases SSEP and MEP amplitude, whereas barbiturates and benzodiazepines have the opposite effect.[7] Opioid analgesics, including alfentanil, fentanyl, remifentanil, and sufentanil have a minimal effect on SSEPs and MEPs and are optimal for this type of surgery.[7] Propofol suppresses SSEPs, but has no effect when the SSEP is stimulated in the epidural space.[7] Because it is rapidly metabolized, it produces desirable anesthetic conditions for SSEP and MEP monitoring during TIVA.[7]

Advantages of TIVA include hemodynamic stability, consistent depth of anesthesia, decreased total dose of anesthetics used, decreased pollution and toxicity within the OR, and a rapid recovery from anesthesia.[9] Two agents used effectively with TIVA are propofol and remifentanil. Propofol can be used to maintain general anesthesia at a rate of 100 to 200 mcg/kg/min.[8] It has a rapid onset, high plasma clearance, and short context-sensitive half-time.[8] It is also advantageous due to its fast emergence and antiemetic properties.[9] Remifentanil can be used in conjunction with a propofol infusion at a rate of 0.05 to 2.0 mcg/kg/min.[8] It is titrateable and has a rapid onset and offset due to its metabolism by methyl ester hydrolysis and tissue esterases within the blood.[8] Unfortunately, it does not provide adequate postoperative analgesia.[9]
A study done by Pechstein et al,[10] compared two different anesthetic techniques for their effects on MEP readings. In the first group, TIVA was utilized with propofol, and alfentanil. Anesthesia in the second group was maintained with oxygen, nitrous oxide, and isoflurane.[10] The researchers found that propofol provided a more conducive environment for monitoring MEPs intraoperatively than a balanced anesthetic with nitrous oxide and isoflurane.[10]

Prior to extubation, a long-acting opioid, such as morphine, should be titrated before the discontinuation of the remifentanil infusion.[5,8] Local anesthetics injected by the surgeon will also aid with postoperative analgesia.[3] Barring any contraindication, non-steroidal anti-inflammatory medications should be used.[3] For this case, both propofol and remifentanil infusions were discontinued approximately thirty minutes prior to the end of the case, and no other opioids were titrated. Morphine was prescribed postoperatively.

The literature review supports our anesthetic management of the patient presented in this case study. As anesthesia practitioners it is important to be familiar with the surgical procedure, positioning, intraoperative neurologic monitoring techniques, and available anesthetic options. Open communication between the anesthesia team, nursing and surgical team, the surgeons, and the neurophysiology technicians will ensure patient safety during any type of surgery.

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Blood Loss and Anesthetic Technique in Prostatectomy

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Keywords: Radical retropubic prostatectomy, neuraxial anesthesia, general anesthesia, blood loss, induced hypotension

Prostate cancer accounts for 33% of all noncutaneous male malignancies, making it the most frequently diagnosed malignancy in men.[1] Beginning at age 50, Caucasian males in the United States have a 10% risk of developing prostate carcinoma. This phenomenon occurs twice as often in African-Americans.[2] Treatment includes pharmacologic and chemotherapeutic modalities, radiation and surgical excision. When surgical intervention is warranted, prostatectomy has a strong potential for significant intraoperative blood loss. Attempts at decreasing blood loss in prostatectomy with the use of neuraxial anesthesia have been extensively studied, often with conflicting results.[3-10]

Case Report

A 60 year old, 76kg, 175cm, African-American male presented for radical retropubic prostatectomy (RRP). He had no known allergies. His social/medical history were significant for a 10 pack year history of tobacco use (cessation 2 years prior), hyperlipidemia, asymptomatic bradycardia with a resting heart rate in the mid 50's and carpal tunnel syndrome of the right extremity. Medications included

hydrocodone, simvastatin and vardenafil. The patient self administered a bowel prep of oral magnesium citrate and phosphates enema the night prior to admission. The patient's preoperative labs and chest radiograph were within normal limits, including a hemoglobin (Hgb) of 14.6 g/dl and hematocrit (Hct) of 42.8 %. Four units of packed red cells were available the morning of surgery in the event transfusions were needed.

The patient's physical assessment was grossly unremarkable. He reported that his only oral intake the day before surgery had been two cups of broth. He walked 2 to 3 miles a day and was assigned an American Society of Anesthesiologists physical status of II. Vital signs were as follows: blood pressure 116/70 mmHg, heart rate 55 beats per minute, respiratory rate 12 breaths per minute, oral temperature 98.9 F, and 99% oxygen saturation on room air. In the preoperative holding area, a 20 gauge angiocatheter was inserted in the left hand and an arterial line was placed in the left radial artery.

The patient was transported to the operating room and standard monitors were applied. Vital signs remained in the preoperative range. Oxygen was administered by face mask while the patient received fentanyl boluses of 50 mcg titrated to moderate sedation. The patient received a total of 1000 mcg of fentanyl up to the time of induction and approximately 500 ml plasmalyte intravenously. General anesthesia was induced with lidocaine 100 mg, propofol 60 mg and vecuronium 7 mg. The blood pressure was noted to be 84/48 mmHg after induction. For this reason, the trachea was intubated before the onset of action of the muscle relaxant. Respirations were controlled with a mechanical ventilator. The patient's blood pressure ranged from 114/62 mmHg to 108/64 mmHg after intubation of the trachea and isoflurane was slowly titrated up to 0.8% by end tidal measurement prior to surgical incision.

The patient's blood pressure was maintained at approximately 100-110/60-80 mmHg with the exception of an isolated reading of 75/58 mmHg. The blood pressure measurement correlated with a non invasive blood pressure cuff reading on the contralateral arm. These readings coincided with excision of the dorsal venous complex. Estimated blood loss (EBL) had been minimal to that point, but quickly reached 375 ml. The blood pressure increased to 112/72 mmHg after administration of a single phenylephrine bolus of 200mcg. Hetastarch 500 ml was then administered over fifteen minutes. Blood loss tapered after that point and the procedure was finished one hour later with a final EBL of 400 ml. An arterial blood gas was drawn at the end of the case, which revealed a final Hgb of 11.4 g/dl and Hct of 35 %. The patient's muscle relaxant was antagonized, isoflurane was titrated off, extubation criteria were met and the patient's trachea was extubated while he was awake with no difficulty or complications.

Discussion

The methods employed to minimize blood loss during prostatectomy usually consist of either induced hypotension or acute normovolemic hemodilution (ANV). Induced hypotension, according to Degoute, entails lowering the systolic blood pressure to 80-90 mmHg, lowering the mean arterial pressure (MAP) to 50-65 mmHg or effecting a 30% reduction of the MAP from the baseline.[11] Weiskopf found that an HCT of 25-30% provides tissue oxygen delivery comparable to an HCT of 30-35% in a patient receiving ANV, providing that

cardiac output can compensate for the decreased HCT.[12] Boldt et al., however, found that there is less blood loss in prostatectomy with controlled hypotension than with ANV.[4]

Spinal or epidural anesthesia can be utilized to achieve controlled hypotension. Neuraxial anesthesia induces hypotension via a sympathectomy which ensues after the anesthetic administration.[5] An advantage of neuraxial induced hypotension includes dilation of both arterial and venous vessels in the desired region, which generally occurs without reflex tachycardia.[5] Neuraxial induced hypotension, however, may be unpredictable and prolonged, requiring vasoconstrictor infusions and/or fluid boluses to maintain an acceptable blood pressure.

The anesthesia practitioner may otherwise induce hypotension, for example, via a sodium nitroprusside or nitroglycerine infusion during a general anesthetic. An advantage of vasodilator infusions includes the ability to titrate the medication quickly to obtain the desired effect. Adverse drug reactions or complications notwithstanding, and strictly in terms of their hypotensive characteristics, this technique has disadvantages as well. Vasodilator infusion effects are systemic, the drugs target either arterial or venous vessels, and a reflex tachycardia may result.

This patient did not receive a neuraxial anesthetic. Considering this patient's general good physical condition, normal Hgb and Hct, and the invasiveness of the cancer, blood loss was not expected to be substantial.[6,13] An allowable blood loss was calculated for this patient to be 3300 ml, which would correspond to a decrease in the Hct to 25%. Vasodilator infusions were not utilized either, as the patient's blood pressure remained in the range noted earlier. As blood loss was minimal for most of the procedure, decreasing the blood pressure further as an intervention was not implemented.

The blood loss for this procedure totaled 400 ml. According to a study by Barré et al, epidural anesthesia may have resulted in a 27% decrease in blood loss, which for this patient, would be equivalent to approximately 100 ml[7]. Neither the loss of an additional 100 ml of blood nor the decision on whether or not to transfuse blood products had a significant impact on this patient's hemodynamic status.

A study by Guay concluded that regional anesthesia for RRP resulted in decreased blood loss, but did not necessarily lead to a reduction in the number of patients requiring transfusion.[6] This meta-analysis focused solely on blood loss and did not mention eventual patient outcomes. This suggests an almost negligible difference and coincides with the findings of Barré et al.[7] Salonia et al concluded that blood loss for RRP with spinal anesthesia was approximately 250-300 ml less than with a general anesthetic.[8] The subjects in this study, however, had a higher preoperative cancer stage than the patient of this case study. They also experienced a blood loss of greater than 850ml, even with the use of spinal anesthesia. This implies that neuraxial anesthesia may impart the most benefit when the tumor burden is high and the surgery more complicated. However, a retrospective study at Johns Hopkins University by Wong et al. found no significant differences in blood loss between either technique.[9] Barré et al also concluded that it was patient position, namely supine jackknife with a relative Trendelenburg orientation, and an experienced surgical team that had the greatest impact on blood

loss.[7] Barré et al purports that the surgeons of this study were each responsible for carrying out specific tasks and maneuvers during the procedure which resulted in improved skill and efficiency.[7] This simple intervention had an effect on decreasing blood loss that exceeded any one technique commonly utilized by an anesthesia practitioner.[7]

Robotic assisted radical prostatectomy (RARP) is a new surgical option that promises to decrease blood loss. While there are no randomized studies on outcomes post RARP as compared to RRP, there are studies that imply RARP may be superior to RRP in regards to blood loss.[14] Dasgupta and Kirby performed RALP on four patients with an identical cancer stage as the patient of this case study and their mean blood loss was 117 ml.[14]

Induced hypotension, either by a neuraxial anesthetic technique or through the use of a vasoactive agent, would likely have yielded marginal benefit for this patient. Had his preoperative cancer stage been higher or starting Hgb and Hct lower, the use of regional anesthesia or a vasoactive adjuvant may have been considered of greater value. In light of the ambiguity demonstrated by the research available on this subject, one can better appreciate the different approaches adopted by practitioners. Despite our best efforts, appreciable blood loss may be an unavoidable characteristic of undergoing RRP.

Blood loss in prostatectomy can vary widely. This may be related more to the severity of the tumor, the difficulty of the procedure and the skill of the surgeons rather than the anesthetic technique.[7,9,13] It remains pertinent, however, for the anesthesia practitioner to plan for the anesthetic which attempts to limit blood loss. Anesthetic considerations for each patient vary as their cancer stage and comorbidities dictate. An individualized assessment of the risks and benefits of the anesthetic approach itself, and the potential for blood product transfusion, is necessary in order to provide the safest anesthetic possible for any given patient.

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Myasthenia Gravis Complicated by Malignant Hyperthermia

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Keywords: myasthenia gravis, malignant hyperthermia, neuromuscular blockade sensitivity

Myasthenia gravis is an autoimmune, neuromuscular disorder characterized by muscle weakness and increased fatigue. Compromised neuromuscular transmission renders myasthenic patients sensitive to non-depolarizing muscle relaxants and resistant to depolarizing muscle relaxants.[1] Malignant Hyperthermia (MH) manifests as a hypermetabolic disorder when genetically susceptible individuals are exposed to potent inhalation agents and depolarizing muscle relaxants.[2] Delivering anesthesia care to a patient with a history significant for myasthenia gravis, malignant hyperthermia and potential difficult airway presents a unique anesthetic challenge. The following case demonstrates the importance of adequate preparation and communication when delivering anesthesia care to a patient with multiple, significant co-morbidities.

Case Report

A 61-year old, 78 kilogram, 62 inch female presented for laparoscopic cholecystectomy and liver biopsy. History of present illness was significant for multiple episodes of symptomatic cholelithiasis and a year long history of abnormal liver function tests (elevated liver enzymes and increasing number of hepatic auto antibodies). Her past medical history included diagnoses of class I myasthenia gravis, diabetes mellitus type II, asthma, gastro esophageal reflux disease (GERD), auto immune hepatitis, hypothyroidism, obesity (body mass index:

32) and paternal history of malignant hyperthermia (MH). She reported shortness of breath after climbing one flight of stairs and lead a sedentary lifestyle. She was medically managed with synthroid, fluticasone, montelukast, metformin, lisinopril, and omeprazole.

On the day of surgery, the patient fasted for 8 hours and self administered her fluticasone nebulizer and montelukast. Preoperative electrocardiogram was assessed as sinus rhythm and blood glucose measured 167mg/dl. Airway examination revealed Mallampati Class II pharyngeal visualization, full cervical range of motion, short thick neck with anterior and posterior redundant tissue, small mouth opening, small chin, and a thyroid mental distance of 2.5 fingerbreadths. Metoclopramide 10mg IV, ranitidine 50mg IV, and midazolam 2mg IV was titrated through a 20g peripheral intravenous catheter and citric acid, sodium citrate 30ml oral solution was administered in the preoperative holding area.

Upon entering the operating room suite, standard monitors were applied, 100% oxygen administered via face mask, and a pre-induction right radial arterial line inserted. A propofol drip was initiated at 75mcg/kg/min, and an additional midazolam 2 mg IV administered. The patient was then induced sequentially with fentanyl 50 mcg IV, lidocaine 80 mg IV, application of cricoid pressure, and propofol 100mg IV. After the patient became apneic, one breath was administered via face mask and train of four (TOF) stimulation of the orbicularis oculi revealed 4 strong twitches. Following administration of cisatracurium 0.075 mg/kg (6 mg), train of four (TOF) was assessed every 30seconds while mask ventilating patient with continuous cricoid pressure. After approximately 90seconds, TOF stimulation elicited 0/4 twitches. Direct laryngoscopy was performed, grade II view achieved, and a 7.0 endotracheal tube (ETT) placed in the trachea without complication. After ETT placement was confirmed, cricoid pressure was released.

Anesthesia was maintained with a continuous infusion of remifentanil at 0.125 mcg/kg/hr, and a continuous infusion of propofol at 150 mcg/kg/min. TOF monitoring was performed every five minutes. Four twitches and sustained tetany returned at approximately 75 minutes after initial induction dose. Neuromuscular blockade was antagonized with neostigmine 4mg, and glycopyrrolate 0.6mg. The surgical procedure was uneventful.

When the patient was fully awake and responding to commands (able to sustain head lift, strong hand grasp) her trachea was extubated. The patient subsequently developed tachypnea (respiratory rate: 25) and complained of abdominal pain. Morphine 4 mg IV was administered and provided good pain relief.

The patient's postoperative course was uneventful. She was able to maintain oxygen saturation >95% during the night while on room air and denied any weakness or shortness of breath. She was subsequently discharged from the hospital on postoperative day two.

Discussion

Myasthenia gravis is an autoimmune disorder which alters neuromuscular junction transmission by producing antibodies directed against the nicotinic acetylcholine receptor.[3] This disorder is characterized by weakness and easy fatigability. The degree of muscle impairment determines the classification of

myasthenia gravis, Class I-V. Class I is the most mild form of the disease, presenting as ocular weakness only, while the most severe form (Class V) necessitates intubation.[4] The ocular muscles are most commonly affected resulting in ptosis and diplopia. Bulbar involvement results in laryngeal and pharyngeal muscle weakness leading to dysarthria, difficulty chewing, swallowing, clearing secretions, and pulmonary aspiration. As the disease progresses, acetylcholine receptors may be reduced by 70%, which greatly diminishes respiratory muscle function and the patient ultimately requires ventilatory support.[5] Anticholinesterase drugs are administered to treat muscle weakness, while corticosteroids and immunosuppressive drugs are utilized to suppress the autoimmune response.

Malignant hyperthermia, a pharmacogenetic disorder, produces episodes of rapid sustained rise in myoplasmic Ca^{2+}. This abnormal rise in calcium levels is caused by alterations in the skeletal muscle ryanodine receptor, located in the sarcoplasmic reticulum.[6] Malignant hyperthermia may occur when a patient with an inherited MH-susceptible mutation is exposed to one or both types of anesthetic triggering agents, such as volatile inhalational agents and succinylcholine.[7] Clinical signs reflect a hypermetabolic state and include increased end-tidal CO_2, tachycardia, hypertension, dysrhythmia, cyanosis, muscle rigidity, masseter spasm, hyperthermia (late sign), and dark brown urine from rhabdomyolysis.[8] Malignant hyperthermia may manifest throughout the perioperative period and treatment includes intravenous dantrolene combined with supportive care. Larach et al[8] reported a 1.4% mortality rate for malignant hyperthermia when promptly treated with Dantrolene. Prior to the use of Dantrolene, the mortality rate for MH approached 70%.[9]

This patient presented with several challenging anesthesia related problems which were potentially life-threatening. Our principal challenge was to determine the safest method of securing the airway, as her multiple co-morbidities precluded the use of several medications. A rapid sequence induction due to GERD, obesity, and presence of diabetes was indicated. However, paternal history of MH negated the use of succinylcholine. High dose rocuronium (1.2 mg/kg) was not utilized for rapid sequence induction because of possible prolonged metabolism due to her autoimmune hepatitis disorder and her myasthenia gravis. Liver disease increases the volume of distribution of rocuronium, which can result in a longer elimination half-life and subsequent longer duration of action.[10] In addition, patients diagnosed with myasthenia gravis demonstrate increased sensitivity to nondepolarizing muscle relaxants.[11] If we were to administer a high dose of rocuronium to a myasthenic gravis patient, drug induced paralysis would most likely outlast the surgical procedure, thus necessitating prolonged intubation and intensive care unit admission. We decided to administer cisatracurium since this neuromuscular blocking agent does not undergo hepatic metabolism. In consideration of her myasthenic disease, we decreased the standard induction dose by 50% (1.5 x ED_{95}). Baraka et al[11] reported a rapid onset of a complete neuromuscular block in the Class II myasthenic patient, after administration of cisatracurium 0.05 mg/kg (1 x ED_{95}). The patient's symptomatic reflux disease was addressed with a modified rapid sequence induction technique, administering continuous cricoid pressure during manual ventilations.

Complicating this patient's presentation was her increased risk factors for a difficult airway. There is a significant association

between difficult intubation and a short neck.[12] According to the ASA Difficult Airway Algorithm, a strategic plan needs to be in place to manage the patient's airway.[13] In lieu of having a difficult airway cart in the operating suite, we had a Glidescope® Video Laryngoscope, an Eschmann Tracheal Tube Introducer, a Laryngeal Mask Airway, and a Surch-Lite® lightwand present in the suite. In a review of 1541 cases from the ASA Closed Claims Database found that failure to achieve a patent airway resulted from unanticipated difficult tracheal intubation (17%); such failures can lead to hypoxic brain injury or death.[14] To further decrease the risk of a potential difficult airway, the patient was placed in a head-elevated laryngoscopic position with the placement of blankets under the patient's head and shoulders. A ramped position improves laryngeal view in the obese patient.[15]

In addition to limiting choice of paralytic agents on induction, the presence of a familial history of malignant hyperthermia impacted intraoperative management and operating room preparation. As potent inhalation agents have been identified as triggering agents, a propofol infusion provided anesthesia. Due to the possibility of residual potent inhalation agent in the circuit, the anesthesia machine was prepared by placing a new circuit, removing the vaporizers, placing new CO_2 absorbers, and flushing the machine with a fresh gas flow for ten minutes. A malignant hyperthermia cart was inspected and immediately available as well.

A history significant for myasthenia gravis affected our choice of intraoperative narcotics and postoperative care. As myasthenic gravis patients are more sensitive to the muscle relaxant effects of nondepolarizing and potent inhalation agents, our primary concern was depressed ventilation postoperatively. Long acting opioids can further depress a patient's ability to maintain a respiratory minute volume capable of maintaining adequate ventilation. We chose to utilize remifentanil, a short acting, and selective mu agonist with a context-sensitive half-life of approximately 4 minutes. Remifentanil's brevity of action is due to its small volume of distribution, rapid clearance rate, and metabolism by nonspecific plasma and tissue esterases.[16] Due to the increased risk for respiratory insufficiency, the need for additional postoperative monitoring was identified prior to surgery. Admission to a monitored setting postoperatively was coordinated and scheduled with the surgical team. In addition, we counseled the patient regarding the potential requirement of controlled ventilation after surgery.

The increased risk of morbidity and mortality due to the presence of MG, MH and other co-morbidities can only be modified by diligent preparation and communication with the surgeon and those responsible for postoperative monitoring. A review of malpractice claims indicated that 35% of postoperative miscommunication led to errors that could have been prevented.[17] The value of communication in patient safety cannot be underestimated.

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Prior Bleomycin Exposure in a Patient with a Difficult Airway

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Keywords: Bleomycin, ABVD chemotherapy, general anesthesia, difficult airway, pulmonary edema

Bleomycin is a chemotherapeutic antibiotic effective in the treatment of squamous cell, testicular, and lymphomatous cancers. The most serious complication of bleomycin is pulmonary fibrosis and impaired lung function. Hyperoxia in patients previously exposed to bleomycin may cause severe

pulmonary oxygen toxicity in the hours to days following surgery [1,2]. This case report presents the management of a post bleomycin chemotherapy patient who presented with an anatomically and physiologically difficult airway.

Case Report

A 46 year old male with a history of stage IIIB Hodgkin's disease who had completed six cycles of ABVD (adriamycin, bleomycin, vinblastine, dacarbazine) chemotherapy presented for emergency surgery. According to Rueda Dominguez et al[4], ABVD chemotherapy is the most common and effective treatment for Hodgkin's Lymphoma which consists of a combination of chemotherapeutic agents, including bleomycin.

The patient, who was 72 inches tall and 102 kilograms in weight, presented to the hospital with a large second degree burn encompassing his left ankle and foot. He was immediately scheduled for an irrigation, debridement, and a skin graft procedure. His past medical history included schizo-affective disorder, treated stage IIIB Hodgkin's Disease, untreated chronic lymphocytic leukemia and severe, untreated gastroesophageal reflux disease (GERD). His preoperative medications included clozapine, iron supplements, escitalopram and quetiapine

His blood pressure was 126/90 mm Hg, pulse was 100 beats per minute, a regular respiration rate of 16 breaths per minute, temperature was 96.2 degrees and his oxygen saturation was 96% on room air. He was classified with a Mallampati Classification III airway, a thyromental distance of two finger breaths and a mouth opening of three finger breaths. He was designated an ASA classification III E. In reviewing the history, the patient had received general anesthesia in the past with a laryngeal mask airway. No prior intubation reports were noted in the chart, and the patient denied a history of previous endotracheal intubation.

The preoperative evaluation of the patient included an electrocardiogram (ECG), transthoracic echocardiogram and a pulmonary function testing. His ECG revealed normal sinus rhythm. A transthoracic echocardiogram ultrasound revealed a calculated ejection fraction of 60%. His pulmonary function testing revealed a reduced forced expiratory volume in 1 second (FEV1), forced vital capacity (FVC), total lung capacity and residual volume leading to a diagnosis of moderate to severe restrictive ventilatory deficit.

In the preoperative area, an 18 gauge intravenous (IV) was placed and the patient was started on lactated ringers solution. Midazolam 2mg IV was administered for anxiolysis. Additionally, sodium citrate 30 ml PO, ranitidine 50 mg IV, and metoclopramide 10 mg IV were administered for aspiration prophylaxis. In the operating room, standard monitors were applied and 8 L/min of air was administered to the patient via a face mask. His baseline oxygen saturation was 97%. A rapid sequence induction of anesthesia was performed with the administration of 100 mcg of fentanyl, 20 mg of lidocaine, 200 mg of propofol and 60 mg of succinylcholine.

Upon laryngoscopy the laryngeal view was determined to be a grade 4 and the vocal cords were not visualized. The patient's oxygen saturation decreased to 80%. The laryngoscope was removed and the patient was hand ventilated with air by mask and oxygen saturation improved to 97% . On the second laryngoscopy the patient again

rapidly desaturated to 85%. The laryngoscope was removed, the face mask reapplied and the patient was mask ventilated and oxygen saturation improved to 97%. .

The third intubation effort by a skilled provider was attempted utilizing a gum elastic bougie for guidance. The larynx was not visualized and the introducer was not passed due to the fact the saturation decreased to 78%. A laryngeal mask airway (LMA) #5 was then successfully positioned and the patient was manually supported on 21% oxygen and the oxygen saturation increased to 95%.. Additional propofol was administered and a 5.5 mm endotracheal tube (ETT) was placed through the LMA under fiberoptic guidance. The LMA was removed and endotracheal tube position was confirmed by auscultation of bilateral breath sounds and the presence of continuous end tidal CO_2. An endotracheal tube exchanger device was utilized to remove the 5.5 mm ETT and place a 7.5 mm ETT. ETT placement was reconfirmed by fiberoptic visualization, auscultation of bilateral breath sounds, and presence of continuous end tidal CO_2. The patient was then placed on volume control ventilation with air and sevoflurane at 1.3%.

The patient remained hemodynamically stable throughout the remainder of the case. His oxygenation did not decrease below 94%. The trachea was extubated without incident. The remainder of the patient's hospital stay was unremarkable, and he was discharged to home on post operative day three.

Discussion

Given the fact that a known serious complication of bleomycin administration is pulmonary fibrosis and impaired lung function, a patient with a history of treatment with bleomycin and documented impaired lung function should alert an anesthesia practitioner to the risk of the potential complication of pulmonary oxygen toxicity. Hay et al[5] report in their study involving rats that lung injury was not observed when exposure to hyperoxia preceded bleomycin treatment. However, severe lung injury was noted with hyperoxia following bleomycin treatment. Their findings implicate oxygen in the pathways leading to acute lung injury during and after bleomycin treatment in the post surgical setting. As a result, Hay suggests oxygen therapy should be kept to a minimum in this patient population preoperatively, intraoperatively and postoperatively[5].

Goldiner reported a study of 5 patients who received bleomycin within a year preceding surgery and developed terminal pulmonary changes consistent with oxygen toxicity following their surgical procedures. An additional 12 patients who also received bleomycin within a year preceding surgery were maintained on oxygen levels at or near 21%. These patients suffered no respiratory complications following anesthesia[1].

The overall risk of a patient with a past exposure to bleomycin and their relative risk for pulmonary oxygen toxicity following surgery appear to be multi-factorial. Bleomycin is 60-70% excreted in the kidney; therefore, renal insufficiency will prolong the lungs' exposure to this drug. Likewise, Davies and Shock[6] demonstrated that with age greater than 40, the glomerular filtration rate begins to decrease. For those treated with this chemotherapy agent beyond the age of 40, a greater risk for lung damage exists. According to O'Sullivan, bleomycin exposure within 4 months of surgery is associated with an even greater risk of pulmonary oxygen toxicity postoperatively[3].

Patients with a history of bleomycin exposure should be examined on an individual basis as to the degree of caution they should receive intraoperatively. Pre-existing pulmonary damage, time of exposure, dose of bleomycin, age and renal function should all be considered when formulating an effective and safe anesthesia care plan. One or more existing risk factors warrants caution when administering oxygen to these individuals perioperatively[3].

The patient in this case report had both a past exposure to bleomycin during his treatment for lymphoma at age 42 and had impaired lung function as confirmed by his pulmonary function testing prior to surgery. For his irrigation, debridement, and skin graft, we chose to manage him on 21% oxygen throughout the procedure in order to avoid the potential lethal complication of pulmonary oxygen toxicity as reported by Hay and Goldiner[5] and others. Management of his anticipated difficult intubation followed the ASA difficult airway algorithm.

Delaying our patient's surgery until his GERD was better managed, thus allowing the use of an LMA, was not an option because of the high risk for infection that the burn on his foot presented. A spinal anesthetic with light sedation was a viable option given the patient's respiratory status, however, he declined a regional technique prior to induction.

Despite the fact that our patient presented with an unanticipated difficult intubation, he was able to be easily mask ventilated. Maintaining our patient on 21% oxygen was prudent given his high risk for oxygen toxic changes. The comparison of the risk of the situation in comparison to the related benefits should always be considered.

Individuals with one or more risk factors associated with pulmonary fibrosis and impaired lung function secondary to bleomycin exposure should be managed at the lowest fraction of inspired oxygen to maintain an oxygen saturation level of greater than 90% and to avoid the potentially lethal effect of pulmonary oxygen toxicity.

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Anesthesia in a Parturient with Syringomyelia

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Key words: syringomyelia, regional anesthesia, general anesthesia

Syringomyelia is a rare condition in which a tubular cavitation, or syrinx, accumulates cerebrospinal fluid within the spinal cord that can impinge on local nerve fibers. Syringomyelias develop as a result of congenital anomalies, spinal injury, infections, tumors, and lesions, particularly the Arnold-Chiari malformation. Pregnant women with syringomyelia are at risk of spinal cord compression from physiologic changes that occur during labor. Pushing and straining during labor compresses the syrinx, resulting in a wide array of neurological symptoms. Regional anesthesia and endotracheal intubation may result in the same complication.[1] The management of these obstetric patients presents a challenge to anesthesia professionals.

Case Report

A 31 year old, 120 kg, 66 inch tall, ASA II female patient presented for scheduled Cesarean delivery at 39 weeks gestation. Her medical history consisted of a 2 year history of syringomyelia with bilateral arm paresthesias. Imaging from the prior year demonstrated a thin cervical-thoracic syrinx extending from C4 through T10, with no tumor or Chiari malformation. A neurology consult from the prior year stated that the cause of the syrinx was most likely a developmental anomaly. No surgical intervention was recommended. The patient returned to her neurologist during the second trimester of pregnancy to discuss options for childbirth. At that time, general anesthesia was recommended. Her only medication was daily prenatal vitamins. No known drug allergies were reported. Hemoglobin, hematocrit, platelets, and the chemistry profile were all within normal limits. Her airway classification was Mallampati III with a thyromental distance of 6 centimeters. Full atlanto-occipital extension, interincisor distance greater than 3 cm, and good dentition were noted. The patient's lungs were clear, and no heart murmur was appreciated.

An 18 gauge peripheral intravenous line was placed and 1000 ml of lactated ringers was infused in the holding area. Sodium citrate, 30 ml, was administered five minutes prior to entering the operating room. The patient was positioned supine with left uterine displacement. Monitors included a pulse oximeter, non-invasive blood pressure cuff, and EKG. The patient was pre-oxygenated with 100% oxygen via facemask for several minutes while her abdomen was prepped and draped. The head of the patient's bed was elevated 30 degrees prior to anesthetic induction. After application of cricoid pressure, general anesthesia was induced with thiopental 400 mg and succinylcholine 200 mg. Laryngoscopy was performed with a Macintosh 3 blade, and the trachea was intubated with a 7.0 endotracheal tube (ETT). The oropharynx was clear and there was no evidence of aspiration. Endotracheal tube placement was verified by end tidal CO2 (ETCO2) and bilateral breath sounds. Anesthesia was maintained with sevoflurane 1.4%, nitrous oxide 2L/min, and oxygen 2L/min. End tidal CO2 was maintained between 32-36 mmHg. After delivery of the fetus and placenta, oxytocin 10 units, midazolam 5 mg, morphine 10 mg, ondansetron 4 mg, metoclopramide 10 mg, and clindamycin 600 mg were administered

intravenously. Oxytocin 10 units were also added to the remaining 500 ml of lactated ringers and infused rapidly. When the patient demonstrated regular spontaneous respirations with tidal volumes of 5 ml/kg, the oropharynx was suctioned an oropharyngeal airway placed. With the patient moving all extremities and following commands, the trachea was extubated. The patient was transported to the recovery area awake with oxygen via nasal cannula. Morphine 5 mg was administered intravenously for pain. The patient's vital signs were stable throughout the case and the neonate's 1 and 5 minute apgar scores were 8 and 9 respectively. The patient's recovery was uneventful and her neurological status remained unchanged.

Discussion

Syringomyelia is a chronic progressive disease characterized by an expanding cystic cavity within the spinal cord, filled with cerebrospinal fluid (CSF), which elongates over time destroying the center of the spinal cord. This rare disorder leads to spinal cord damage and may result in pain, weakness, stiffness of the back, shoulders, arms, neck, or legs, paresthesias of the extremities, and referred chest pain.[2] Surgery is sometimes recommended to drain the syrinx. Whether treated or not, many patients are advised to avoid straining or other activities that cause CSF pressure to fluctuate. Active labor and delivery significantly increase CSF pressure, and could result in compression of the syrinx in these patients. However, regional anesthesia and endotracheal intubation may result in the same complication.[1] One of the most important goals of anesthesia for these patients is to avoid aggravating the already disturbed CSF pressure relationship. This is particularly applicable to the use of general anesthesia: there are reports of deterioration after general anesthesia for non-cranial surgery, when no specific attempt was made to control intracranial pressure (ICP).[3]

A review of the literature revealed that historically, cesarean delivery with general anesthesia has been preferred for women with syringomyelia to avoid possible neurological deterioration due to bearing down, and regional anesthesia was ruled out for medico-legal reasons. However, the presence of active neurological disease is no longer considered an absolute contraindication to regional anesthesia.[4] There are very few documented cases of anesthesia for parturients with syringomyelia in the current literature. To illustrate current perspectives on anesthesia management of these women, five published case studies are presented: an epidural block for spontaneous vaginal delivery, an epidural block for operative forceps delivery, two epidural blocks for cesarean deliveries, and a general anesthetic for a cesarean delivery.

In 2002, Chantigan et al[5] presented a case of a parturient with a syrinx extending from the cervical through the thoracic spine, with clinically significant symptoms, who developed preterm labor at 32 weeks. She labored for 9 hours with an epidural block. The patient reported no significant increase in symptoms during labor, spontaneous vaginal delivery, or during the postpartum period. Additionally, no complications were reported related to the epidural block.[5]

Broberg, Napolitano, and Parker[1] presented two cases, each managed differently, but with equally effective outcomes. Their first case involved a parturient with syringomyelia previously treated with a cervico-thoracic shunt. She presented in labor with headaches, parasthesias, and weakness. A neurology consult concluded that these symptoms were consistent with

spinal cord/syrinx compression due to increased ICP.[1] This patient underwent urgent cesarean delivery using a slowly dosed epidural. Within twenty four hours of delivery, the patient had resolution of her symptoms and her postpartum course was uncomplicated. The authors presented a second case in which a pregnant woman with syringomyelia presented in labor at 39 weeks gestation. She had no new neurological symptoms and an uneventful pregnancy. An epidural was placed and slowly dosed without complications. After a passive second stage of labor (no maternal pushing), the patient underwent a low forceps delivery and had an uncomplicated postpartum course.[1]

Nel, Robson, and Robinson[3] presented a case in 1998 of a parturient with syringomyelia and a related Chiari type I anomaly, admitted for elective Cesarean delivery at 38 weeks gestation. Her neurological symptoms had developed three months prior to becoming pregnant. Urgent foramen magnum decompression was recommended; however, the patient became pregnant and declined surgery. During her second and third trimesters, her symptoms intensified, and the neurologist and neurosurgeon agreed that elective Cesarean delivery would be the method least likely to aggravate the syrinx.[3] The patient received a slowly dosed epidural. The Cesarean delivery proceeded uneventfully and seven weeks after delivery there had been no neurological deterioration. The authors cited the advantages of epidural anesthesia as the avoidance of potential hazards in securing the airway, less compromise of respiratory function, and better preservation of the existing craniospinal CSF pressure relationship.

The last case, presented by Murayama et al[6], describes the safe use of general anesthesia for Cesarean delivery for a parturient with syringomyelia, with precautions taken to avoid increases in ICP to reduce risks of untoward neurologic events. A priming dose of a non-depolarizing muscle relaxant was administered, followed by an intubating dose. The patient was intubated, remained hemodynamically stable, and was extubated upon full reversal of neuromuscular blockade. The patient experienced no neurological changes post-operatively. The authors chose general anesthesia rather than a spinal or epidural anesthesia for medico-legal reasons. They stressed that one of the most important considerations in the anesthetic management of syringomyelia is the avoidance of increased ICP, especially during intubation, and recommend using nondepolarizing muscle relaxants.

In May, 2008, our patient's neurosurgeon recommended to avoid spinal anesthesia even though the syrinx and neurological exams had not changed. A cesarean delivery was also recommended as it would impose the least stress on the patient's syrinx. We decided to use general anesthesia for our patient's cesarean delivery because historically, general anesthesia has been used for almost all parturients of syringomyelia, with regional anesthesia being ruled out presumably for medico-legal reasons. We also felt that with an epidural, there was an unknown amount of risk of fluctuation in CSF pressure, as well as a risk of inadvertent subarachnoid puncture. Additionally, compressing the subarachnoid space with local anesthetic may induce untoward neurologic sequellae. Moreover, since syringomyelia is a progressive disease, it would be difficult to identify the cause of neurological symptoms that appear following regional anesthesia. We agreed, however, that epidural anesthesia could indeed be used in this population.

Whichever anesthesia technique is chosen, prevention of a rise in CSF pressure and ICP are paramount. During our case, no specific attempt was made to control ICP. In retrospect, we could have used a non-depolarizing muscle relaxant to avoid the slight increase in ICP that may occur with succinylcholine. Mild hyperventilation during mechanical ventilation could have been utilized to avoid increased ICP as well. Optimal outcomes require a multidisciplinary team approach including anesthesiology, obstetrics, neurology, neurosurgery, with the full agreement of the patient. Anesthesia professionals appear to be accepting anecdotal reports that regional anesthesia by a slowly administered epidural is a safe option for this population. General anesthesia should probably be avoided given the inherent risks of airway management and the possibility of hypertension and increased ICP during intubation. However, if general anesthesia is absolutely necessary, then measures must be taken to control ICP.
In the case of vaginal delivery, a key strategy is to initiate pain control early in labor with a slowly dosed epidural and to avoid all maternal pushing during the second stage of labor. Delivery then can be accomplished with forceps or vacuum when safe application is possible.[1] Clearly, the true risk of increased intracranial or intraspinal pressure with subarachnoid, epidural, and general anesthesia remains unknown; however, the presence of active neurological disease is no longer considered an absolute contraindication to regional anesthesia.[4]

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Intranasal Fentanyl in Pediatric Myringotomy Tube Placement

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Keywords*:* myringotomy, BMT, intranasal fentanyl, transnasal fentanyl, transnasal butorphanol

Bilateral myringotomy and tympanosotomy tube placement (BMT) is one of the most common pediatric surgical procedures. BMT is a surgical procedure that is often

performed as a treatment for chronic or recurrent otitis media. Due to the brevity of the procedure, BMT is generally performed with the patient spontaneously breathing inhalational anesthetics via a facemask, often without intravenous access. It is reported that up to 76% of patients having BMT require postoperative pain management when inhaled anesthesia is not supplemented with analgesia[1]. The absence of an intravenous access limits the choice of analgesia for pain management. Frequently, acetaminophen is administered orally or rectally to supplement inhaled anesthesia. However, it is reported that intranasal fentanyl is effective in providing post-operative pain relief in patients who had BMT surgery[2,3].

Case Report

A 17-month-old, 9.2 kg Caucasian male was scheduled for BMT for recurrent otitis media. Save for recurrent otitis media, the patient had no past medical history, birth or neonatal complications, or recent upper respiratory infection. The mother reported that her son was not taking any medication. Allergies included eggs, amoxicillin, and ranitidine. Immunizations were up to date. There are no smokers in the household.

On the day of surgery, physical examination revealed an awake and alert patient. Preoperative vital signs included a temperature of 36.4°C. via tympanic thermometer, 98% pulse-oximetry saturation, and a pulse rate of 125. Physical assessment was unremarkable. Patient had a clear bilateral breath sounds and no signs of upper respiratory infection. The mother confirmed that patient consumed nothing by mouth for six hours. The patient was given an American Society of Anesthesiologists physical status classification of 2. Assessment for Mallampati classification was not performed.

After a brief discussion, the anesthesia practitioners decided that general anesthesia using sevoflurane without intravenous access was a safe choice for this patient. No preoperative medication was administered. The patient was escorted to the operating room with his mother.

Prior to mask induction, the pulse-oximeter was applied and the anesthesia circuit was primed with 8% sevoflurane and a flow rate of 5 liters of nitrous oxide and 5 liters of oxygen per minute. With the patient sitting on the mother's lap, the facemask was applied to the patient. The patient was adequately anesthetized after a few spontaneous breaths, and there were no complications or agitation during the mask induction. Once anesthetized, the patient was transferred to the bed, a warm blanket and monitors (EKG, precordial stethoscope, and blood pressure cuff) were applied. Anesthesia was maintained on 3% sevoflurane and 3 liters of oxygen per minute.

BMT was performed in 25 minutes without any complications. Vital signs were stable throughout the case, and assisted ventilation was not given. Prior to emergence from anesthesia, acetaminophen 240 mg PR and fentanyl 18 mcg intranasal were administered. The patient was transferred to the post-anesthesia care unit with spontaneous respirations, unsupported airway, and 12 liters of oxygen blow-by using a simple mask. After we gave report to the postoperative care unit nurse, the patient awakened and cried. Crying ceased when the mother arrived to hold the patient.

After one hour in the PACU, the patient was discharged to home. The nurse who

discharged the patient stated that there were no post-operative complications and no other medications were given. Furthermore, the patient's mother was very satisfied with the surgery and anesthesia.

Discussion

According to Finkel et al., pain after BMT procedure often occurs during emergence and diminishes within 45-60 minutes[2]. Thus, it is appropriate to manage pain prior to emergence. Since BMT surgery is often performed without intravenous access, there are limited choices for analgesia. Acetaminophen administered orally or rectally is one analgesic option for BMT surgery. The onset of acetaminophen is between 60-90 minutes and its action peaks in 2-3 hours[1]. Since pain with BMT surgery occurs on awakening, acetaminophen is not an effective emergence analgesic choice. In addition, absorption of drugs when given orally or rectally may be variable and slow.

Another analgesic option that does not require IV access is intranasal butorphanol and it has been used for BMT post op discomfort. Butorphanol is a synthetic opioid agonist-antagonist analgesic agent that can be given intranasally for moderate to severe pain. Butorphanol 25 mcg/kg intranasal is an effective analgesic for BMT surgery due to its pharmacokinetics. Beenie et al. reported that intranasal butorphanol reduced the need additional analgesia after BMT in children.[4] The onset of intranasal butorphanol is within 15 minutes, however peak effect occurs within 1-2 hours, after most of the painful stimulus has manifested.

Numerous studies have examined the efficacy of acetaminophen and butorphanol in preventing postoperative pain after BMT.[1,4] However, the previous drugs mentioned have limitations. The pharmacokinetics of acetaminophen and butorphanol do not provide optimum timing for pain relief after BMT. In addition, butorphanol is a relatively expensive drug.[5] Fentanyl is a synthetic opioid that is commonly administered as an adjunct to anesthesia to reduce the amount of pain during and after surgery. Fentanyl has an onset time of 15-30 minutes and a duration time of approximately one hour.[2] In addition, fentanyl is highly lipohilic and has a low molecular weight. Intramuscular injection of fentanyl in children with BMT is not usually used because of pain, bleeding, and swelling at injection site.

Studies have reported that intranasal fentanyl in conjunction with sevoflurane decreases the incidence of agitation and postoperative pain, without increasing postoperative respiratory depression, emesis, or discharge time after BMT surgery.[2,3] The extensive vasculature and large surface area of nasal cavity allows for rapid absorption of intranasal fentanyl.[2] Galitkin et al. reported that intranasal fentanyl at a dose of 2 μg/kg yields serum concentrations similar to intravenous administration of fentanyl.[3] The study by Galitkin et al. has limitations. Given that the study consisted of only 26 children, larger trials are needed to determine if intranasal fentanyl increases the risk of laryngospasm, chest wall rigidity, and upper airway obstruction with BMT surgery.

In this case study, our patient experienced an uncomplicated minimally invasive anesthetic and comfortable, rapid recovery with the use of intranasal fentanyl after an inhalation anesthetic. Several studies have demonstrated the efficacy of intranasal fentanyl during BMT surgery.[2,3] Intranasal fentanyl decreases postoperative agitation and the need for additional rescue analgesia without requiring IV access. It is an

alternative or adjunct to oral or rectal acetaminophen. However, the use of intranasal fentanyl has its complications in addition to those associated with BMT surgery, such as, laryngospasm, bronchospasm, chest-wall rigidity, and cardiovascular collapse.[6] Anesthesia practitioners should be aware of these potential complications and prepare to treat them. Dosages of intramuscular injections of atropine, succinylcholine, and other emergency drugs should be known and must be prepared in case of emergency for any case performed without intravenous access.

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Total Intravenous Anesthesia for Bilateral Breast Reduction in an Adolescent

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Keywords: total intravenous anesthesia (TIVA), postoperative nausea and vomiting (PONV), Propofol, Ketamine, breast surgery

Postoperative nausea and vomiting (PONV) is an unfortunate, yet common complication after anesthesia. There are several measures that may reduce the degree of this mulitfactoral dilemma. Studies demonstrate that total intravenous anesthesia (TIVA) with propofol and ketamine reduces the incidence of PONV. The following is a case report describing a patient with multiple risk factors for PONV and the anesthetic technique used to minimize this complication.

Case Report

A seventeen-year-old female presented for an elective bilateral breast reduction for macromastia. This healthy, 5 foot 9 inch, 80-kilogram teenager had no prior surgical history. She did not smoke and was otherwise in good health. She took no daily medications. Physical and airway exam were within normal limits for this ASA classification I patient. The only lab data available was a negative HCG. Preoperative vital signs were within normal limits and there were no known drug allergies. After all identifications and consents were confirmed, a 20 gauge peripheral intravenous (IV) line was started in the right hand. Premedication of midazolam 2 mg intravenous (IV) was administered. A scopolamine patch was applied behind the right ear.

Upon arrival to the operating room, the bispectral Index (BIS) and standard monitors were applied. Preoxygenation was administered for five minutes. A smooth IV induction occurred with propofol 200 mg and ketamine 40 mg IV. A laryngeal mask airway (LMA), size 4, was inserted and seated appropriately. Bilateral breath sounds and end tidal CO_2 were confirmed before the LMA was secured. Spontaneous ventilation returned and was maintained throughout. No inhalation anesthetics were delivered. Oxygen and air were provided at 1 liter/minute each via the anesthesia circuit. A continuous infusion of propofol (10 mg/ml) with added ketamine (0.5 mg ketamine/ml propofol) was initiated through a micro-infusion pump. This infusion was titrated to maintain general anesthesia and hypnosis throughout the operation.

Prior to surgical incision, dexamethasone 8 mg, 50 mcg of fentanyl, and cefazolin 1 gram were delivered. An additional 50 mcg of fentanyl was administered when the surgeon infiltrated 40 ml of 0.25% bupivicaine to each breast. Midazolam 3 mg was administered in divided doses during the procedure for a case total of 5 mg. A final 50 mcg of fentanyl was delivered when the nipple was sutured, totaling 150 mcg. Prior to completion, ketorolac 30 mg and ondansetron 4 mg were administered IV. A total of 1500 ml of lactated ringers was infused. There were no intraoperative complications and vital signs remained within normal limits.

The propofol-ketamine infusion was discontinued at the end of surgery. A total of 1900 mg of propofol and 95 mg of ketamine were delivered through the continuous infusion. The LMA was removed once all bandages were in place. The patient maintained a patent airway with spontaneous respirations. Supplemental oxygen was applied and remained in place during the transfer to the post anesthesia care unit (PACU).

At the postoperative visit the patient denied any nausea, vomiting, pain, intraoperative awareness, disturbing dreams or hallucinations. Nystagmus of the right eye, noted in the immediate postoperative period, resolved within two hours. The patient spent one night on a surgical floor for observation and was discharged to home the following morning with no postoperative complications.

Discussion

PONV is one of the most frequent complaints after surgery and anesthesia.[1] There are multiple features that have been shown to predispose to this complication. Factors associated with an increased risk include younger age, female gender, nonsmokers, lower physical status (ASA I), no preoperative medical conditions, and no

prior surgical history. Intraoperative risk factors for PONV include elective procedures, long duration of anesthesia, inhaled anesthetic technique compared to regional anesthesia, and administration of opioids.[2] There is a high incidence of PONV associated with some surgical procedures including breast surgery. PONV negatively impacts the consumption of healthcare time, resources, and finances.[1] The patient detailed in the case report above demonstrated most risk factors. For this reason, the she was considered to be at high-risk for PONV while creating the anesthetic plan. Many treatment measures were taken to reduce the possibility of PONV. These include the preoperative scopolamine patch, propofol-ketamine TIVA, avoidance of anesthetic gases, minimizing the administration of opioids, multimodal analgesia, adequate intravenous hydration, and delivery of IV antiemetics ondansetron and dexamethasone.

Propofol and ketamine total intravenous anesthetic (TIVA) is a technique that has been associated with a markedly low incidence of PONV. This opioid and inhalation agent sparing approach allows for the maintenance of spontaneous ventilation. The literature supports its success in various settings. Friedberg (1999) published the results of a propofol-ketamine, room air spontaneous ventilation technique with 1264 patients in the office setting. These anesthetics were performed during a variety of surgical interventions that often included local anesthetic from the surgeon. There was a 0.6% incidence of PONV and zero hospital admissions for uncontrolled nausea, vomiting, or pain. Less than 1% of the patients reported dreaming. None of these individuals reported that they were bothered by these dreams.[3] These results are reflected in the case study above. This anesthetic technique used in the case example resulted in no postoperative nausea, vomiting, or pain. The patient reported having no recollection of dreams or intraoperative awareness.

In 2000, Badrinath et al. evaluated the sedative and analgesic role of the propofol-ketamine TIVA under monitored anesthesia care. This randomized, double-blinded, placebo-controlled study consisted of one hundred female outpatients undergoing breast biopsy procedures. Patients were randomized to one of four subject groups. Groups included one saline placebo arm and three groups of propofol diluted with increasing concentrations of ketamine. Research results revealed that the group with subhypnotic doses of ketamine delivered with a propofol infusion provided significant analgesia without negative psychotomimetic, respiratory, or hemodynamic side effects. The arm with the largest dose of ketamine did reveal significant increases in PONV and hallucinogenic effects. More than 90% of the study subjects reported satisfaction with their anesthetic and were willing to receive the same technique again.[4] The individual in this case report received a low dose of ketamine (0.5 mg/ml of propofol). This dose along with the lack of PONV, pain, and hallucinations correlates with the data suggested by Badrinath et al (2000).

Some anesthesia practitioners avoid the use of ketamine because of its potential for delirium. However, there is evidence highlighting its usefulness in anesthetic techniques when delivered at low doses, like the case study described. This sub hypnotic dose, along with premedication of benzodiazepines, helps to minimize the likelihood of negative psychic effects.[3,4] Even when administering smaller doses, ketamine maintains analgesic properties.[4] This feature, in conjunction with adequate

local anesthetic administration to the surgical site may explain the minimal opioid requirement in this case report. Additional advantages to using ketamine in this situation include its sympathomimetic actions to counteract the hemodynamic depression of propofol, lack of respiratory depression, and increased upper airway and laryngeal reflexes.[3] The last two properties back the use of the LMA in this case.

As noted, studies suggest that a TIVA with propofol aids in reducing the incidence of PONV.[5] Unfortunately, this anesthetic technique is currently more expensive than inhalation anesthesia. Visser et al. (2001) performed a randomized control trial evaluating the PONV and economic results of a propofol TIVA versus inhalation anesthesia in 2,010 surgical patients. The TIVA resulted in a significant reduction in PONV compared to the inhalation anesthetic. Nonetheless, the anesthetic costs were three times greater in the propofol TIVA group than the inhalation agent group. An economic gain was not demonstrated, as the PACU times were similar in both arms. [6] With rising medical costs and other economic constraints, it might be prudent today to consider costs along with patient outcomes when developing an anesthetic plan. The literature suggests a decreased incidence of PONV with the use of TIVA techniques. However, in efforts to contain cost, it may be more sensible to carefully reserve this approach for those at greatest risk for PONV.

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Arginase Deficiency and Anesthetic Implications

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Keywords: arginase deficiency, amino acids and anesthesia, urea cycle disorders, hyperammonemia, metabolic disorders

Arginine is an essential amino acid required for mammalian growth. It is an intermediate in the urea cycle, and a precursor in the formation of nitric oxide, creatinine and glutamate. Furthermore, arginine stimulates the secretion of insulin, growth hormone, glucagon, and prolactin[1]. Arginase Deficiency Syndrome is a rare autosomal recessive disorder that results in impaired breakdown of arginine in the urea cycle, producing a state of hyperammonemia. Children who are born with this disorder commonly exhibit spastic tetraplegia, seizures, ataxia, psychomotor deterioration, hyperactivity, hepatomegaly, and growth failure. Treatment focuses on maintaining a low protein diet and avoiding prolonged fasting which would result in a catabolic state. Patients with Arginase Deficiency Syndrome who present for surgery provide many challenges for the anesthetist.[2]

Case Report

A 17 year-old male, ASA III, 46 kg, 177 cm, presented to our day surgery unit for odontectomies of impacted teeth. The patient also had a history of Arginase Deficiency complicated by mental retardation, spastic diplegia, seizures, and multiple hospital admissions for hyperammonemia. Prior surgeries included a liver biopsy at 6 months of age, an inguinal hernia repair at 12 months of age, and dental rehabilitation under general anesthesia. Medications included amoxicillin 6 ml every 8 hours via J-tube, sodium phenylbutyrate ¾ teaspoon four times daily, and a formula consisting of 1 ¼ cups of cyclinex, 4 2/3 cups of pro-phree added to 72 ounces of water via his J-tube.

On physical exam, the patient was with his mother, smiling, nonverbal, and showed repetitive upper extremity movements. His heart rate was 84 bpm and blood pressure was 108/58. He was breathing comfortably on room air with an oxygen saturation of 99%. His lung sounds were diminished at the bases but otherwise clear. He had a J-tube that was clean and intact. Due to his mental retardation, he was unable to follow commands to open his mouth for airway assessment, but had good ROM of his neck and a three finger thyromental distance.

After surgical and anesthesia consents were obtained, a #22 gauge IV was placed in the right antecubital vein and 2 mg of midazolam was administered in the preoperative holding area. The patient was continuously monitored via an oximeter probe. He was transferred to the operating room table, placed supine, and standard monitors were applied. Intravenous induction of general anesthesia was obtained with propofol 200mg. After ease of mask ventilation was confirmed, vecuronium 3mg was administered. The patient received 1 spray in each nare of phenyleprine ¼% and a # 6.0 nasal RAE cuffed endotracheal tube was advanced without difficulty. A Grade I view of the vocal cords was seen with a MAC 3 blade and McGill forceps were used to advance the ETT into the trachea. After confirmation of ETT placement, general anesthesia was maintained with isoflurane 1.5%, 1 L/min oxygen and 1 L/min of air. Additional medications of morphine 4mg,

dexamethasone 8mg, ampicillin 2 gm, and ondansetron 4mg were administered throughout the course of the surgery. The patient's maintenance fluid was dextrose 10% in water at 125 ml/hr with a KVO of 0.45% NaCl. The patient was hemodynamically stable throughout the procedure and removal of the ETT occurred after return of spontaneous respirations and opening of the eyes. The patient was transferred to the recovery room in stable condition. He was discharged to home later that day.

Discussion

The diagnosis of Arginase Deficiency Syndrome and associated metabolic and neurologic impairments pose several potential anesthetic problems that require careful perioperative anesthetic management. First, the duration of the fasting period prior to surgery is critical in patients with Arginase Deficiency Syndrome and other urea cycle disorders. To avoid risk of hyperammonemia, we ensured that this patient did not fast longer than 8 hours. Also, patients with high ammonia levels typically demonstrate disorientation, asterixis, lethargy, vomiting, and potential coma, all of which can be masked under general anesthesia[7]. The genetics and metabolism service professional leading this patient's medical care was consulted and stated that fasting and stress associated with surgery carries a high risk of episodic hyperammonemia due to the breakdown of endogenous protein and accumulation of nitrogen as ammonia. In order to prevent this catabolic state from occurring, the patient was prioritized to the first case of the day.

Another measure we took to avoid a perioperative hyperammonemic episode focused on the careful selection of maintenance fluids. The perioperative intravenous administration of a glucose-containing solution was integral in preventing a catabolic and hyperammonemic state. We chose a high glucose containing solution and infused $D_{10}W$ at 1.5 times his maintenance rate after IV access was obtained. The administration of glucose-containing fluids prevents the body from utilizing protein breakdown for energy. Attention was also paid to avoid over-hydrating the patient, which could result in cerebral edema due to the disrupting effects of ammonia on the blood brain barrier. Lastly, it was imperative that these fluids continue until the patient resumed his home feeding routine [5].

Seizures tend to be common in these patients due to high ammonia levels and, as such, anesthetic drugs that have anticonvulsive properties should be administered during the perioperative period. General anesthesia may mask seizures. The patient was given 2mg midazolam preoperatively and sodium thiopental was readily available should a perioperative or postoperative seizure occur.

Providing anesthesia for this patient carried some cardiovascular risks. Nitric oxide is a potent vasodilator involving several cell types, including endothelial, vascular and neuronal cells, and production of cellular nitric oxide is regulated by the enzyme arginase, which was deficient in this patient [2]. Patients with urea cycle disorders can have difficulty autoregulating blood pressure, although there are no current research studies surrounding arginase deficiency and nitric oxide production. Further, a defect in the production of nitric oxide due to arginase deficiency can lead to the development of early atherosclerosis, placing the patient at higher risk during anesthesia for myocardial infarction[6]. We

ensured the patient had adequate oxygenation and maintained his systolic blood pressure between 100-140 mmHg.

Urea cycle disorders can also adversely affect the liver, and many of the patients with these conditions eventually progress to overt hepatic failure. In the patient with a failing liver, medications metabolized by the CYP 450 enzyme system should be avoided. Cisatracurium relies upon Hoffman elimination instead of metabolism by the liver, and can therefore be used for neuromuscular relaxation. Liver dysfunction can also lead to coagulation disorders and so proper coagulation studies should be obtained prior to the placement of any regional anesthetic in patients with urea cycle disorders [6]. Although the patient in this case study tolerated the general anesthetic without difficulties, the serum ammonia level and clotting values should have been obtained and reviewed prior to surgery to ensure optimal preoperative preparation.

Patients with urea cycle disorders, although uncommon, could present to any hospital and require surgery. Medications that raise the seizure threshold should be administered during induction and anesthetic management should focus on avoiding hyperammonemic states by ensuring a short fasting period, administration of dextrose containing fluids, and careful titration of anesthesia to avoid hyper or hypotension. Further research focused on the anesthetic management of a patient with urea cycle disorders is necessary to provide safe surgical environments for these complex patients.

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Regional Anesthesia in the Parturient with Idiopathic Thrombocytopenia Purpura

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Keywords: Idiopathic thrombocytopenic purpura, ITP, Thrombocytopenia

Idiopathic thrombocytopenic purpura (ITP) is an autoimmune disease in which antiplatelet immunoglobulins bind to platelet membranes, resulting in platelet destruction and ultimately thrombocytopenia.[1] ITP is commonly seen in young women and is cited to affect one to two of every 1,000 pregnancies.[2] The management of ITP during pregnancy is complex, but morbidity and mortality are rare, even in the presence of severely low platelet counts. The decision to use a regional anesthetic in a parturient with thrombocytopenia is controversial and requires a careful risk-benefit analysis that should be made on an individual patient basis. [3]

Case Report

A 21 year-old, 60 inch, 124 kg, 39-week nulliparious parturient presented to labor and delivery for induction of labor according to patient request. She received prenatal care throughout her pregnancy. She had a large uterine fibroid but her pregnancy had otherwise been uncomplicated. The patient reported that her medical and surgical histories were negative. She denied having any drug allergies and her current daily medications included prenatal vitamins only. Baseline vital signs were blood pressure (BP) 144/72 mm Hg, heart rate (HR) 74 beats per minute (bpm), respiratory rate (RR) 18, oxygen saturation (SpO_2) 95%, and temperature (T) 98.1 °F. Fetal HR was 130-165 bpm with good variability. Serum electrolytes were within acceptable limits; however, the complete blood count (CBC) revealed a platelet count of 14,000 mm/μL. Airway assessment revealed a Mallampati III classification, mouth opening of 4 centimeters, and thyromental distance greater than 6 centimeters.

Physical assessment was without petechiae or ecchymoses. The patient denied gingival bleeding, epistaxis, and excessive bruising or bleeding. When re-questioned about her history, the patient reported that she was diagnosed with chronic ITP as a teenager but forgot to report it to her obstetrician during her prenatal care.

Labor induction was postponed and hematology was consulted. Corticosteroid and intravenous immunoglobulin therapies were initiated while serial platelet counts were monitored. After three days of treatment, the platelet count increased to 46,000 mm/μL and natural labor began to progress. Ten units of platelets were given, increasing the platelet count to 53,000 mm/μL. After careful collaboration between the obstetrician and nurse anesthetist, the decision was made to immediately proceed with a caesarean section using a spinal anesthetic.

The patient was taken to the operating room and appropriate monitors were applied. Initial vital signs were BP 148/76 mm Hg, HR 93 bpm, RR 19, SpO_2 94%, and T 97° F. Four liters per minute of oxygen were administered per nasal cannula. With the patient in the sitting position, a subarachnoid block was performed. The skin was anesthetized with 3 ml of 1% lidocaine. One pass was made with a 25-guage Whitacre needle through a 20-gauge introducer.

Cerebrospinal fluid was clear and free flowing. 5% lidocaine 100 mg, epinephrine 100 mcg, and fentanyl 20 mcg were injected.

Sensory anesthesia was achieved to a T6 level. Two 16-gauge peripheral IV catheters were inserted prior to incision. Ten additional units of platelets and two units of fresh frozen plasma were given. A viable female infant was delivered 16 minutes after skin incision. Oxytocin 40 units was immediately added to one liter of intravenous fluid. The uterus contracted appropriately and bleeding was well controlled. Estimated blood loss was 800 ml. Vital signs remained stable throughout the case: BP 86-152/52-88 mm Hg, HR 83-110 bpm, RR 16-22, SpO_2 94-99%, skin T 97° F. Post-operative CBC revealed a platelet count of 56,000 mm/μL and a hematocrit of 33%.

Corticosteroid therapy was continued for 24 hours after delivery and then weaned appropriately. Post-operative anesthetic and neurologic evaluations were done every two hours until the anesthetic subsided. The patient and her infant were discharged to home 72 hours after delivery without obstetric or anesthetic complications.

Discussion

While the specific etiology of ITP is unclear, current theory suggests it is an autoimmune disease in which antiplatelet antibodies bind to the antigens on platelet surfaces, resulting in premature platelet destruction. It is defined as a platelet count less than 100,000 mm/μL with normal bone marrow function and no other cause of thrombocytopenia.[4] Corticosteroids are the first line treatment for ITP, as they suppress the immune system and interfere with autoantibody attack on platelets. If corticosteroid treatment is inadequate, intravenous immunoglobulin is used to bind to platelets and block the attachment of antiplatelet antibodies. If platelet counts remain severely low (<10,000 mm/μL), a splenectomy is indicated in order to eliminate the platelet-trapping role of the spleen and its function in producing antiplatelet antibodies.[5]

ITP can present at any age, but it tends to occur in young women and is therefore a common cause of thrombocytopenia during pregnancy.[2] The management of ITP in a pregnant woman is complex and carries with it the added responsibility of also caring for the fetus. Maternal mortality is exceptionally rare and morbidity is most often associated with postpartum hemorrhage.[3] The risks to the neonate are also associated with thrombocytopenia, as antiplatelet antibodies can cross the placenta and induce fetal thrombocytopenia. Retrospective studies have shown that up to 15% of neonates born to mothers with ITP have platelet counts below 50,000 mm/μL, with severe intracranial hemorrhage occurring in less than 1% of these neonates.[6] Vaginal delivery has historically been believed to increase the risk of intracranial hemorrhage in the neonate with thrombocytopenia; however, recent research is inconclusive and indicates that the mode of delivery should be determined by standard obstetric criteria.[5]

Thrombocytopenia is considered to be a relative contraindication to the use of regional anesthesia. These patients are theoretically at an increased risk of developing a rare but serious complication in which localized bleeding forms a hematoma which could potentially compress and damage the spinal cord.[2] Despite this concern, there are no published reports of a spinal hematoma occurring in a patient with ITP.[6]

Although the absolute minimum platelet count below which it is unsafe to use a regional anesthetic is unknown, it has historically been recommended that regional anesthetic techniques be withheld if platelet counts are less than 100,000 mm/μL.[3] Recent research, however, has shown that epidural or spinal anesthetics can be safely performed with platelet counts much lower than 100,00 mm/μL. Rasmus et al. report a study of 5 parturients with ITP who had platelet counts less than 100,000 mm/μL and received epidural anesthetics without sequelae.[2] Moeller et al. reports safely placing an epidural anesthetic in a parturient with ITP who had only 26,000 mm/μL platelets.[7] Chang reports safely performing a spinal anesthetic in a patient with a platelet count of 50,000 mm/μL.[8] There is also a case report of a patient who safely received an epidural anesthetic with a platelet count of only 2,000 mm/μL.[4]

The use of regional anesthesia in parturients with ITP is based on an individual risk-benefit assessment. The clinician must consider the history, physical exam, and evaluate the trend and rate of decline of the platelet count. If the decision is made to proceed with a regional anesthetic, a local anesthetic with a short duration of action and the lowest concentration necessary to produce anesthesia is recommended.[2] Assessment of sensory and motor anesthesia should be conducted every two hours until the anesthetic has subsided, allowing the anesthetist to be acutely aware and take appropriate actions if complications occur. If a hematoma develops, neurosurgery should be consulted and an emergent laminectomy and decompression performed within 6-12 hours to preserve neurologic function.[4]

This case is presented to educate anesthetists of recent research indicating that regional anesthesia can safely be performed in parturients with ITP with platelet counts less than 100,000 mm/μL. A history of excessive bleeding, the presence of ecchymoses or petechiae, or a rapid decline in the platelet count should be considered warning signs that regional anesthesia should be avoided. Considering each patient individually, obstetricians and anesthetists must coordinate their approaches to provide optimal care for the mother and her fetus.

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Dextrocardia and Coronary Artery Bypass Graft Surgery

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Key Words: dextrocardia, situs inversus, coronary artery bypass graft, primary ciliary dyskinesia, Kartagener syndrome

Dextrocardia with situs inversus totalis is a rare congenital abnormality that develops in 1-2 per 10,000 births.[1-3] During fetal development, the majority of the heart arises in the right hemithorax in patients with dextrocardia. Situs inversus totalis is an abnormal anatomic arrangement whereby the great vessels and viscera are in reversed location within the body.[4] Dextrocardia with situs inversus presents unique challenges to the anesthesia professional and the surgeon during coronary artery bypass graft surgery.

Case Report

A 78 year-old male with a history of severe coronary artery disease presented for coronary artery bypass graft surgery because of fatigue and escalating shortness of breath. He also had a history of hypertension, hyperlipidemia, and obesity but denied any chronic respiratory problems. He was 70 inches tall and weighed 98.4 kg. The patient reported that he was diagnosed with dextrocardia with situs inversus as a child. He denied any allergies. His medications included pravastatin, amlodipine, benazepril, metoprolol, aspirin, magnesium, hydrochlorothiazide, and meloxicam. He had a history of numerous non-cardiac surgeries without any anesthesia problems. His electrocardiogram showed a sinus rhythm and a tracing that was consistent with dextrocardia. The CT scan documented complete situs inversus. His cardiac catheterization report showed atherosclerotic vascular disease, dextrocardia, and blockage of the left anterior descending artery, the first diagonal branch, and the right coronary artery. It reported normal left ventricular function with excellent contractility and normal ejection fraction. The cardiac catheterization report was significant for difficulty in placing a pulmonary artery catheter through the vena cava due to irregularities of the inferior vena cava with a dual system. His preoperative hemoglobin and hematocrit were 12.5 g/dl and 38.4% respectively. His coagulation studies, blood urea nitrogen, electrolytes and liver function tests were within normal limits.

The patient was transferred to the operating room and standard monitors were applied. His electrocardiogram leads were reversed with the brown, white and green leads on the left and the black and red leads on the right. A 20-gauge arterial line was placed in the right radial artery. He was given 100% oxygen by facemask, and general anesthesia was induced. The central venous line and pulmonary artery catheter were placed through the left internal jugular vein without difficulty.

A median sternotomy provided surgical access. The surgeon stood on the left side of the patient and harvested the right internal mammary artery for anastomosis to the anterior descending artery. The right saphenous vein was harvested for anastomosis to the right coronary artery and the diagonal branch. Cardiopulmonary bypass proceeded without difficulty. The patient required one unit of packed red blood cells. He was transported to the intensive care unit in stable condition. His postoperative echocardiogram revealed an ejection fraction of 55% with excellent biventricular performance. On postoperative day one, the patient was stable with spontaneous respirations. He was discharged to a skilled nursing facility on postoperative day 11.

Discussion

It is believed that an autosomal recessive gene causes dextrocardia, but the exact etiology has not been determined.[5,6] Stoelting and Dierdorf[7] state that during fetal development, normal ciliary movement is necessary for the typical placement of asymmetric organs. Primary ciliary dyskinesia is a congenital condition that impairs ciliary motility. In fetuses with this disorder, the placement of organs is random and may be an etiology for situs inversus.

Kartagener syndrome is a disorder of situs inversus with primary ciliary dyskinesia. Impairment of cilia results in retained respiratory mucus secretions secondary to loss of ciliary clearance. This can lead to bronchiectasis and chronic sinusitis. Additionally infertility in males is due to weakened spermatic flagella and decreased fertility in females is due to nonfunctioning ciliated epithelium of the oviducts. 50% of patients with nonfunctioning cilia have situs inversus.[7] Approximately 20% of patients with situs inversus have Kartagener syndrome.[8] It is therefore crucial to elicit a thorough respiratory history for patients with situs inversus. This patient was in the 80% who do not display this syndrome.

Patients with dextrocardia are classified by the arrangement of the viscera and great vessels. In situs solitus, the viscera and vessels are in their normal anatomic locations. With situs inversus the superior and inferior vena cava are located on the left side of the body and are connected to the morphologic right atrium on the left side of the heart. The liver is on the left and the stomach is on the right. This is referred to as a mirror image of the normal anatomy. Patients with situs ambiguous, also known as isomerism, have an abnormal inferior vena cava and/or descending aorta with the liver located equally in the right and left sides of the abdomen and an indefinite stomach locale.[4]

Patients with dextrocardia should have their electrocardiogram leads reversed in order to produce a heart rhythm pattern that will allow correct analysis.[7]We reversed the leads for our patient and it revealed a sinus rhythm. Furthermore, left internal jugular vein cannulation provides the most direct route to the right atrium and avoidance of the thoracic duct. We found this to be true for our patient. Cannulation of the left internal jugular vein allowed the catheter to be advanced into the pulmonary artery without difficulty.

Bohun et al.[4] conducted a retrospective chart review for dextrocardia in Vancouver, Canada from January 1, 1985 to December 31, 2001. They discovered a marked difference in the incidence and severity of associated anomalies based on situs. In patients with situs solitus, 96% had cardiac malformations, 37% had noncardiac

anomalies, and of those who received treatment 50% were well at the time of follow-up. In the isomerism group, 100% had cardiac malformations, 58% had noncardiac anomalies, and of those who received treatment 30% were well at the time of follow-up. The isomerism patients had the highest incidence and most complex abnormalities. However, in the patients with situs inversus 23% had cardiac malformations, 20% had noncardiac anomalies, and of those who received treatment 100% were well at follow-up. There were a variety of cardiac malformations found in the patients in this study including abnormal pulmonary veins; bilateral superior or inferior venae cavae; a common atrium; atrial or ventricular septal defect; a common atrioventricular valve; atrioventricular discordance; abnormal tricuspid, pulmonic, mitral, or aortic valves, solitary ventricle, double-outlet right or left ventricle, pulmonary atresia, coarctation of the aorta, and/or abnormal pulmonary or coronary arteries. The non-cardiac anomalies displayed in this population consist of gastrointestinal malrotation; Meckel's diverticulum; hypoplastic pancreas; abnormal lung, urogenital system, rib, or foot; thoracoscoliosis; dysmorphic features; hydrocephalus; eye tags; absent left ear; developmental delay; neck webbing; congenital hip dislocation; and neuroblastoma. Gastrointestinal malrotation was the most common non-cardiac anomaly with an incidence of 18.5%.

Bonde and Campalani[9] assert that of patients with dextrocardia, only those with situs inversus totalis will live a long enough life to develop atherosclerosis leading to coronary artery disease. In patients with dextrocardia with situs inversus, the incidence of coronary artery disease is similar to that of the general population.[1,2,5,6]

There is disagreement regarding the degree of increased complexity of the surgical procedure secondary to dextrocardia with situs inversus. Some purport that only minor modifications are needed in the surgical strategy to compensate for the anatomical variation,[2,5,8,9] but Chakravarthy et al[1] contend that the technical difficulty of the procedure in these patients is increased and that cannulation of the aorta and vena cava could be especially problematic. Typically the left internal mammary artery is utilized for anastomosis to the left anterior descending artery for coronary artery bypass graft surgery.[10] Conversely, in patients with dextrocardia with situs inversus, the right internal mammary artery is frequently chosen for anastomosis to the anterior descending artery.[1,2,5,6] The surgeon may choose to stand to the left of the patient instead of on the right for easier access to the surgical field.[1,2,6,9] During our patient's surgery, the surgeon stood on the left to harvest the right internal mammary artery. No technical difficulty was noted.

When assessing for cardiac symptoms it is important to note that frequently these patients will have right-sided anginal pain.[5] Careful attention to the preoperative history and physical exam with an emphasis on associated anomalies and pulmonary function will prepare the anesthesia professional to develop an appropriate plan of care. Adjustments to electrocardiogram lead placement and cannulating the left internal jugular vein instead of the right will tailor the anesthesia plan of care specifically for patients with dextrocardia with situs inversus totalis.

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Respiratory Complications Following Laparoscopic Surgery

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Key Words: laparoscopy, subcutaneous emphysema, pneumothorax, hypercarbia, fundoplication.

Laparoscopic surgery has been performed since 1937[1] and is being performed with increasing frequency because of its cost savings and faster recovery time.[2,3] During laparoscopic surgery a pneumoperitoneum is created using carbon dioxide (CO_2) insufflation to help provide surgical visualization. Creation of the pneumoperitoneum is done mainly with CO_2 because of its rapid diffusion ability, low cost, and it does not support combustion. The use of CO_2 during insufflation, however, can cause it to diffuse into subcutaneous tissue leading to subcutaneous emphysema, hypercarbia, and pneumothorax. Anesthesia professionals need to be aware of the complications of laparoscopic surgery in order to prevent unfavorable outcomes.[4]

Case Report

A 57 year old male, 91 kg, 74 inches tall, presented for a laparoscopic Nissen Fundoplication with endoscopy related to a ten year history of gastroesophageal reflux disease and hiatal hernia. The patient's other past medical history included arthritis. Past surgical history included endoscopy, hemorrhoidectomy, vein stripping, and ankle surgery. He stated no family history of anesthetic complications and a personal history of post operative nausea and vomiting. He denied any drug allergies and preoperative medications included pantoprazole. Physical and airway exams as well as baseline vital signs and complete blood count were unremarkable.

In the preoperative holding area the patient received midazolam 2 mg intravenously (IV) and sodium citrate 30 ml by mouth and was brought back to the operating room. Preoxygenation was administered via face mask using 100% FiO_2 and standard monitors were placed. A rapid sequence induction with cricoid pressure was performed using fentanyl 250 micrograms, lidocaine 100 mg, propofol 300 mg, midazolam 3 mg, succinylcholine 120 mg, and rocuronium 10 mg. A 7.5 mm endotracheal tube was placed atraumatically. Anesthesia was maintained with desflurane 6%, one liter of oxygen flow, and rocuronium for muscle relaxation. The patient was positioned in reverse trendelenburg and ventilation remained controlled throughout the procedure with a tidal volume of 600 ml and initial respiratory rate of 12.

Initial end tidal carbon dioxide ($ETCO_2$) was 32mmHg. Approximately 30 minutes after CO_2 insufflation, the $ETCO_2$ increased to mid 60's.Subcutaneous emphysema was noted on bilateral chest, neck, and face. The surgeon was notified, and measures taken to decrease $ETCO_2$ included hyperventilation, decreasing insufflation pressure, and intermittent abdominal desufflation of CO_2. $ETCO_2$ remained in the 50's throughout the case. Peak airway pressures ranged from 22-26 mm Hg, oxygen saturation (SpO_2) remained greater than 98% on 100% FiO_2, and vital signs remained stable with no dysrhythmias. Throughout the anesthetic hydromorphone 1mg was titrated for postoperative analgesia. A total dose of 70 mg of rocuronium was administered throughout the 165 minute procedure. The patient also received dexamethasone 4 mg, ondansetron 4 mg, metoclopramide 10 mg, and ranitidine 50 mg for nausea prophylaxis.

After surgery was completed and hemostasis achieved with minimal blood loss, neuromuscular blockade was antagonized with neostigmine 5 mg and glycopyrrolate 0.8 mg. The patient shortly resumed spontaneous ventilation and revealed a final $ETCO_2$ of 37 before extubation. It was determined that the patient would tolerate extubation because tidal volumes were around 500 ml and $ETCO_2$ had normalized. The oropharynx was suctioned and patient was extubated after he met extubation criteria including eye opening to command, hand grip, and spontaneous ventilation with adequate tidal volumes. 100% O_2 via nonrebreather mask was placed on the patient and he was then taken to the post anesthesia care unit (PACU). In the PACU the patient's vital signs were: blood pressure 140/70, heart rate 91, respirations 18, SpO_2 97% on 8 liters (L) O_2 via nonrebreather mask. A portable chest x-ray was obtained in PACU to determine the extent of subcutaneous emphysema and evaluate for possible pneumothorax. The x-ray revealed a small left apical pneumothorax. An oxygen saturation of 92-98% was maintained by the patient, he did not

complain of any shortness of breath, and oxygen flow was weaned down to 2 L O_2 over the next couple hours. The patient was sent to telemetry on continuous pulse oximetry and subsequently discharged the next day without any further problems.

Discussion

Laparoscopic surgery is minimally invasive and has the advantages of reduced adverse events, shorter hospital stay, and faster recovery.[5] In order for visceral structures to be identified, the abdomen is insufflated with CO_2 at pressures not greater than 12 to 15 mm Hg.[6] Other gases such as nitrous oxide and helium have been used for insufflation, but CO_2 is the safest because it does not support combustion and it is rapidly absorbed.[1] Excess CO_2 introduced during insufflation must be absorbed from the tissue, transported in the blood, cross the alveolar membrane, and be exhaled into the atmosphere mirroring the body's normal physiologic process. Therefore, without an increase in minute ventilation, hypercapnia will ensue.[1] Consequences of laparoscopic surgery using CO_2 include many physiologic changes such as increase in mean arterial pressure, systemic vascular resistance, and cardiac filling pressures, and a reduction in cardiac index.[3,7] It can also cause increased cerebral blood flow and intracranial pressure while decreasing total hepatic and renal flow. Respiratory changes include reduction in functional residual capacity, increased peak airway pressures, and ventilation perfusion mismatch.[1,3] Subcutaneous emphysema and pneumothorax are known complications associated with CO_2 insufflation.[4]

Subcutaneous emphysema following the creation of a pneumoperitoneum in laparoscopic surgery is a common complication.[2,6] Subcutaneous emphysema is caused by leakage of gas within the tissue below the skin[1] and is usually benign.[2] It is diagnosed by palpation that produces a crackling sensation and an increase in $ETCO_2$, which is typically the first sign.[4,8] To decrease $ETCO_2$, minute ventilation must be increased as needed to overcome the acidosis. To maintain eucapnia, an increase in minute ventilation by 20% to 30% may be needed.[1] The increase in $ETCO_2$ also causes stimulation of the sympathetic nervous system and catecholamine release leading to tachycardia, decrease in myocardial contractility and cardiac output, decreased venous return, and lower arrhythmia threshold. Mild subcutaneous CO_2 is usually self-limiting, without long term complications, and resolves after the abdomen is deflated.[3-4,8] Subcutaneous emphysema is not a contraindication for extubation[3] and usually resolves in one to four days.[4] However, the CO_2 can track to the thorax and mediastinum resulting in pneumothorax, especially if emphysema is noted on the chest, neck, or face.[5]

Pneumothorax can develop during laparoscopy and is common during laparoscopic esophageal surgery.[9,10] Dissection of the esophageal hiatus exposes the parietal pleura and puts it at risk, particularly on the left side. This is due to the fact that the esophagus is above the left crux of the diaphragm and the left lung lies in close proximity to this. The most frequent signs of intraoperative pneumothorax include hypoxemia, decrease in lung compliance, decreased breath sounds, hypercarbia, and an increase in airway pressures.[9,10] Treatment of pneumothorax depends on the severity of symptoms and hemodynamic stability. Conservative treatment is warranted if minimal physiologic compromise is noted because CO_2 is rapidly absorbed. This includes close observation, monitoring, and supplemental

oxygen. However, with severe physiologic compromise actions such as ventilatory support and/or placement of an intercostal cannula or chest tube may be needed. [5]

Laparoscopic surgery has many benefits including smaller incisions, faster recovery time, reduced postoperative pain, and cost savings.[3,5] The anesthesia professional should understand and anticipate the physiologic changes and risks that accompany laparoscopic surgery in order to maintain patient safety.[5] Subcutaneous emphysema and pneumothorax are known complications of laparoscopic surgery.[2,6,9,10] In order to avoid negative or potentially life-threatening complications, the professional needs to be vigilant and understand the risk factors, symptoms, and treatment of complications from laparoscopic surgery. The anesthetized patient relies on the anesthesia professional to manually implement the minute ventilation when a rise in CO_2 is noted.[4] Prompt recognition and treatment of increased $ETCO_2$ and subcutaneous emphysema in this patient led to a favorable outcome with conservative treatment.

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Managing Oxidative Stress and G6PD Deficiency

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Keywords: Glucose-6-phosphate dehydrogenase (G6PD); general anesthesia; pediatrics; oxidative stress; TIVA

Glucose-6-phosphate dehydrogenase (G6PD) deficiency, an inherited disorder of red blood cell metabolism, is the most common enzyme deficiency worldwide affecting persons of African, Asian, Mediterranean or Middle-Eastern descent.[1-4] The G6PD enzyme is responsible for the production of nicotinamide adenine dinucleotide phosphate (NADPH) via the pentose phosphate pathway.[1-4] NADPH is critical in the protection of cells from oxidative damage. Erythrocytes, unlike other cells, can only produce NADPH by the action of G6PD, causing the erythrocytes to be more susceptible to oxidative stress destruction.[1,2,4] Anesthesia considerations for the G6PD deficient patient should focus on avoidance of oxidative stress conditions and the use of anti-oxidative anesthetic drugs.

Case report

A 3 year old African American boy, 36 inches, 12 kg, ASA physical status II diagnosed with dental caries presented for oral rehab. His past medical history was significant for G6PD deficiency and asthma. The G6PD deficiency was diagnosed at birth via newborn screening after a positive family history. The patient had remained free of hemolytic events and hospitalizations. His asthma was well controlled and his only medication was albuterol as needed. His health history was negative for a recent upper respiratory illness within the last two months. He had no previous anesthetic exposure. He was premedicated with oral midazolam 9 mg producing a calm, sedated, cooperative patient who separated easily from his parents. In the operating room a pulse oximeter, electrocardiograph, cutaneous temperature and a non-invasive blood pressure monitor were applied. An inhalational induction was performed with sevoflurane (8% reduced to 3%) in 6 liters/min nitrous oxide and 4 liters/min oxygen via face mask and IV access was secured. Nasotracheal intubation was facilitated with phenylephrine nasal spray, rocuronium 6 mg, and morphine 1 mg. A 5.0 mm uncuffed nasal right angle endotracheal tube was inserted without difficulty. Anesthesia was maintained with desflurane at approximately 6.5% end tidal in 1 liter/ min nitrous oxide and 1 liter/min oxygen. Ventilation was mechanically controlled to maintain normocapnia (end tidal carbon dioxide 35-40mm Hg). Hemodynamic parameters remained stable. Dexamethasone 4 mg and ondansetron 1.2 mg were also administered.

The patient's hemodynamic and other vital parameters remained stable for the seventy five minute procedure. The blood loss for the surgery was less than 10 ml. At the conclusion of the procedure, neuromuscular blockade was antagonized with glycopyrrolate 0.12 mg and neostigmine 0.8 mg. Sevoflurane and nitrous oxide were discontinued, and the patient's trachea was extubated. The patient was transported to the recovery room where he remained for 60 minutes and was discharged later that day.

Discussion

Glucose-6-phosphate dehydrogenase (G6PD) is an enzyme found in all tissues responsible for initiating the first (oxidative) phase of the pentose phosphate pathway generating nicotinamide adenine dinucleotide phosphate (NADPH).[2,4] NADPH provides the reductive biosynthesis and oxidation-reduction reactions involved in the protection of cells against the toxicity of reactive oxygen species (free radicals).[2,4] Free radicals are a byproduct of aerobic metabolism that plays an important role in cell signaling and immune function. However, when free radical levels increase, significant damage to cell structures occurs resulting in oxidative stress.

The G6PD- NADPH complex system is responsible for neutralizing excess reactive oxygen species that can lead to cell modification and cell death. Unlike other tissues, the erythrocyte can only produce NADPH via the oxidative phase of the pentose phosphate pathway, making the erythrocyte more susceptible to oxidative hemolytic destruction.[1-4]

G6PD deficiency is a hereditary X-linked disorder[1-4] that can be the result of decreased enzyme molecules, a structural defect in the enzyme causing instability or both.[2] It is the most common enzymopathy in man.[1-3] Between 1966 and 1986 over 400 biochemical variants were characterized resulting in different levels of enzyme activity.[1-3] Currently, approximately 400 million people worldwide are affected; with an increased regional concentration per capita found throughout Africa, Asia, the Mediterranean, and the Middle East.[1-4] In the United States, approximately 10% of black males are affected.[1]

Of the 400 G6PD genetic variants that have been identified, only a few evoke clinical hemolysis.[1,2] The degree of enzyme deficiency or instability correlates with NADPH production and the resultant oxidative protection, with total deficiency being incompatible with life.[1] The World Heath Organization has classified the G6PD variants in five groups, class I-V,[2] based on the degree of enzyme activity and hemolytic damage. Class I deficiency corresponds to severe deficiency associated with chronic non-spherocytic hemolytic anemia resulting in spontaneous hemolysis without the presence of oxidative stress.[2] Class II deficiency is characterized by severe deficiency (1-10% residual activity) associated with acute hemolytic anemia.[2] Included in this class is the second most recognized variant, the Mediterranean form, which results in severe hemolysis with exposure to oxidative stress.[1] Class III deficiency is defined as moderate deficiency (10-60% residual activity) resulting in mild hemolysis following exposure to oxidative drugs.[2] The most common enzyme mutation, the African variant, is an example of the Class III deficiency.[1] Class IV and V deficiencies are rare, representing normal enzymatic activity (60-150%) and increased activity (>150%) respectively.[2]

It is well known that a large number of drugs, substances and clinical disorders have been reported as being high risk for provoking hemolysis in G6PD deficient patients. The genetic diversity of G6PD deficiency, the duration of action and dose of the oxidative drug administered and the presence of additional oxidant stressors all factor in to the risk and severity of the resulting hemolysis.[2] Clinically, acute hemolysis can manifest as back or abdominal pain[1], jaundice[1,2] and hemoglobinuria[2] with symptoms appearing

in 24-72 hours and resolution within 8-10 days.[2,3]

Surgery and concurrent infections are known to enhance the generation of free radicals potentially leading to an imbalance between free radical formation and free radical scavenging in the erythrocyte. Moreover, commonly used anesthetic drugs and their effects on G6PD enzymatic activity has not been fully investigated.[5] Therefore, anesthetic management in G6PD deficiency should utilize known medications that possess anti-oxidant activity and avoidance of suspected G6PD inhibitor drugs and known oxidative drugs.

Propofol, a highly lipid soluble anesthetic used widely during induction and maintenance of anesthesia, has been proven to possess antioxidant activity in vitro and in vivo.[6,7] In a study performed by Tsuchiya et al, propofol was found to have potent antioxidant activity and was shown to decrease the physical stress on the erythrocyte, resulting in a greater preservation of red blood cell counts after surgery compared with sevoflurane anesthesia.[8] Additionally, in one animal study, desflurane was shown to decrease the antioxidant enzyme activity as well as increase the levels of oxidative stress in the circulation and in the lungs, suggesting that desflurane could contribute to increased levels of oxidative stress in the G6PD deficient patient.[9]

In two recent in vivo studies, propofol was again shown to decrease free radical production and modulation of oxidative stress when compared with sevoflurane.[6, 9, 10] In contrast, an in vitro study reported isoflurane, sevoflurane, diazepam and midazolam had an inhibitory effect on the G6PD enzyme, while halothane, ketamine and prilocaine had no effects on the enzymatic activity of G6PD.[5]

According to the limited research available, propofol total intravenous anesthesia (TIVA) technique is a practical alternative to volatile anesthesia for the G6PD deficient population over 3 years of age.[11] In this case report, midazolam and sevoflurane, both shown to have inhibitory effects on the G6PD enzyme, was administered to the G6PD deficient patient. Even though there were no hemolytic symptoms noted during the perioperative period, it is not clear that this combination of drugs provided the safest anesthesia for this patient. With the ambulatory nature of the surgery, the patient was discharged to home before classical symptoms of hemolysis would appear.

There is no doubt that inhalational anesthesia can be used in this patient population. However, there is convincing data suggesting that TIVA as opposed to inhalational anesthesia would be a safer alternative. Understanding of the pathophysiology involved in G6PD deficiency as well as the theoretical advantages, unique antioxidant ability and free radical scavenger properties of propofol, TIVA should be considered as the general anesthetic technique for the G6PD deficient patient.

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Ultrasound-guided Pediatric Ilioinguinal/iliohypogastric Nerve Block

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Keywords: ilioinguinal/iliohypogastric nerves, inguinal hernia repair, ultrasound guidance, nerve block, pediatric

The ilioinguinal/iliohypogastric nerves provide sensation to the superficial tissues overlying the inguinal ligament and proximal scrotum[1]. These nerves originate from the lower thoracic and upper lumbar plexus and continue caudally along the lateral portion of the abdomen between the transverses abdominis and internal oblique muscles.[1]

Traditionally, anatomical landmarks are identified and a single injection of local anesthetic is administered to the ilioinguinal/iliohypogastric nerves to provide anesthesia and analgesia for inguinal hernia repair. Ultrasonographic guidance for ilioinguinal/iliohypogastric nerve blocks in children allows for direct visualization and injection of the local anesthetic as well as a reduction in the volume of local anesthetic administered.[2] The following case report describes the administration of an ilioinguinal/iliohypogastric nerve block

using ultrasound guidance and subsequent intraoperative management of a patient undergoing inguinal hernia repair.

Case Report

A 2 year-old male patient presented the morning of surgery for right inguinal hernia repair. A right reducible non-tender inguinal hernia was discovered at a regularly scheduled visit by the child's primary pediatrician. Past medical and surgical history was insignificant. The infant measured in the 50th percentile for height and weight, had all immunizations up-to-date, and met age-appropriate mile stones. The child had no allergies to food, drugs, or latex products. Home medications included multivitamins once per day. Perioperative evaluation revealed an 88 cm long, 14 kilogram well-nourished ambulating male. Vitals signs were age-appropriate. Dentition revealed no loose or chipped teeth. The child was free from any illnesses within the last month. Last clear fluid intake was 1 ounce of apple juice 4 hours prior to arrival.

Prior to surgery the child was given a weight-based mixture of oral midazolam and acetaminophen to provide anxiolysis with parental separation. The child was transported via stretcher to the operating room (OR) without any distress. Routine ASA monitors were applied and an inhalational induction was initiated. Inhalation induction included 7L nitrous oxide and 3L oxygen mixture with incremental adjustments of sevoflurane titrated to hemodynamics. Once unconsciousness was obtained, nitrous oxide was discontinued and a mixture of 100% oxygen with sevoflurane was titrated to maintain unconsciousness and stable vital signs. A 22 gauge peripheral intravenous (IV) catheter line was placed. Propofol 30mg was administered IV with subsequent placement of a size #2 laryngeal mask airway (LMA). Cefazolin 350mg and dexamethasone 7mg was administered IV. Anesthesia was maintained with 2% end-tidal sevoflurane in 1L oxygen and 1L air mixture. The child's right groin area was prepped sterilely for ilioinguinal/iliohypogastric nerve block. Utilizing sterile technique, ultrasound guidance was used to identify the ilioinguinal/iliohypogastric nerves and surrounding landmarks. The nerve block was performed with a 22 gauge 1 ½ inch short-beveled needle with an injection line. Once the needle tip had been visualized by ultrasound in the appropriate position relative to the nerves and confirmation with a negative aspiration test obtained, a single injection of 3ml of 0.25% bupivicaine with 1:200,000 epinephrine was administered in 1ml increments.

The child remained in the supine position for surgery. The heart rate and blood pressure changed less than 15% with incision and throughout the procedure. No rescue analgesia was required during the procedure or in the recovery unit. Ondansetron 4mg IV was administered approximately 30 minutes prior to procedure completion. Spontaneous ventilation with pressure-support via LMA supported throughout the surgery. Upon completion of the procedure, a mixture of oxygen with sevoflurane 3% was administered. The child's respiratory pattern was regular with adequate tidal volumes and the LMA was removed. A patent airway was maintained with jaw-lift and 100% oxygen was administered by face mask. The child was transported to the recovery area for emergence and continued monitoring of vital signs and comfort level.

Discussion

The ilioinguinal/iliohypogastric nerve block is a popular regional anesthetic technique for children undergoing inguinal hernia surgery. It has a failure rate as high as 30% when using the conventional 'blind' technique.[2] The conventional technique includes identifying a puncture site 1cm medial to the superior anterior iliac spine and a fascial 'click' or 'pop' is noted after needle puncture upon which the local anesthetic is injected[2]. The depth of the nerve is variable, particularly in children of different ages or different body habitus making the conventional technique a challenge for even an experienced anesthesia practitioner.[3] Complications when using the conventional technique can include colonic puncture, small bowel puncture, and hematoma.[2] The conventional method administers a larger volume of local anesthetic compared to the ultrasound guidance technique.[3] In addition, due to the unpredictable spread of larger volumes of local anesthetic, the potential to cause motor blocks and urinary retention is greater.[3] A femoral nerve block secondary to an ilioinguinal/iliohypogastric nerve block is believed to be volume-related.[2] This occurs due to the spread of local anesthetic between the muscle layers to the nerve.[2] Blocking the femoral nerve can delay ambulation and possibly be disturbing to the child and parents.[2] The risk for local anesthetic toxicity is also increased when using higher anesthetic volumes.[3] This is especially important with regards to neonates and infant due to their lower plasma concentration of alpha-1 acid glycoprotein which allows higher free plasma concentrations of local anesthetic.[3]

Incorrect placement of the local anesthetic is the most probable reason for the conventional technique high failure rate. Research has shown that in the majority of patients, landmark-based techniques inaccurately place the local in adjacent anatomical structures causing unpredictable block results.[4] There have been successful ilioinguinal/iliohypogastric nerve blocks reported despite incorrect injection of the local anesthetic to surround the nerves.[4] This is thought to be related to the local anesthetic placed less than one tissue plane away from the nerves.[4]

A success rate of nearly 100% can be achieved when using ultrasonographic guidance to visualize the targeted nerves.[5] The ilioinguinal/iliohypogastric nerves are relatively superficial and easily identified with high resolution portable ultrasound[3]. Ultrasound guidance allows for identification of the needle tip between the ilioinguinal and iliohypogastric nerves in the correct fascial plane between the interval oblique and transverses abdominis.[3] Observation of the spread of local anesthetic around the nerves allows for significantly smaller amount of local anesthetic to be injected and have clinically effective blocks.[3] Additionally, studies using ultrasound guidance have suggested that more accurate placement of smaller amounts of local anesthetic does not reduce the effectiveness of the block.[4] The amount of local anesthetic is reduced six- to eightfold when using ultrasonographic guidance compared with dosages recommended in pediatric textbooks.[2] The risk of intraperitoneal, intravascular, and intraneuronal injection are reduced greatly when injection of the local anesthetic is done using direct visualization to identify the nerves and surrounding structures.[3] In addition, an improved block greatly decreases the likelihood of additional analgesia requirements or the need for rescue analgesia.

The use of ultrasonographic guidance in

ilioinguinal/iliohypogastric nerve block results in more frequent success using smaller volumes of local anesthetic, with fewer side effects when compared with the conventional 'blind' technique.[5] The risk of toxicity is diminished as well as side effects related to local anesthetic volume.[2] The requirement for additional analgesia is significantly lower.[5] Reducing the need to administer any additional analgesia, particularly narcotics, reduces the likelihood of untoward side effects for the child and possible delay of discharge. In conclusion, current research supports the use of ultrasonography for pediatric anesthesia and recommends its use for ilioinguinal/iliohypogastric nerve blocks.

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Blood Pressure Interpretation in the Beach Chair Position

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Keywords: Anesthesia, Blood Pressure, Beach Chair, Sitting, Shoulder Surgery

The beach chair or sitting position places patients at angles varying from 30-90° above the horizontal plane and was developed for use during shoulder arthroscopy procedures.[1] Shoulder surgery in beach chair position presents unique intraoperative risks to the patient and challenges to the anesthesia practitioner.[2] Although infrequent, catastrophic events associated with upright positioning, including stroke, cerebral ischemia, spinal cord injury, and death have occurred despite vigilant anesthesia care.[2]Venous air embolism is another well-known risk associated with the sitting position due to the negative pressure gradient between the right atrium and operative site.[3]Specific physiological changes associated with beach chair positioning require special consideration for medical history, preoperative blood pressure (BP), and intraoperative interpretation of BP.[2]

Case Report

A 31-year-old, 70 in, 124 kg male presented to the holding area with a failed left shoulder

hemi-arthroplasty. He was scheduled for removal of prosthesis with revision to total arthroplasty. His surgical history included several procedures on the left shoulder secondary to an injury. His medical history included type II diabetes mellitus, obesity, hypertension, dyslipidemia, asthma, and osteoarthritis. Preoperative vital signs and lab values were within normal limits except for a glucose of 156 mg/dl and white blood cell count of 21,000/ml. While in holding, IV access was obtained with a 20-gauge catheter in the right hand. The patient and operative site were identified, risks of general and regional anesthesia discussed, and consent was obtained. Pulse oximetry, EKG, and NIBP monitoring were applied and midazolam 2 mg was administered IV.

With emergency equipment at hand, an interscalene block was performed under sterile technique with a 22-gauge, insulated needle and nerve stimulator. Following proper needle placement, 40 ml of 0.5% Bupivicaine with epinephrine was injected in 3-5 ml increments with intermittent aspiration. The patient denied sharp pain during injection. The patient received an additional 1 mg of midazolam IV in holding. Following standard induction and confirmation of endotracheal tube (ETT) placement, mechanical ventilation was initiated and general anesthesia was maintained with oxygen 1L/min, nitrous oxide 1L/min, and sevoflurane 2%. The patient's head was padded and secured to a horseshoe headrest and he was placed in the beach chair position at 90°.

Following repositioning, the patient's head was verified to be in neutral position to prevent impedance of venous flow and decreased cerebral blood flow (CBF).[2] Proper ETT placement was also reconfirmed. Sequential compression device stockings remained on both legs throughout the case to reduce pooling of blood and increase venous return.[4]

The arthroplasty was well tolerated by the patient and was without complication. The patient's vital signs remained stable throughout the case and he did not require pharmacologic intervention for BP management. His BP ranged from 130/62 mmHg (MAP 84 mmHg) to 99/58 mmHg (MAP 71 mmHg). Neuromuscular blockade was reversed and the ETT was removed without incident. A facemask was placed on the patient at 6L O_2 and he was taken to PACU.

The patient was seen postoperatively one day later with vital signs at baseline values. He showed no neurological or neuromuscular deficits. He was discharged to home the following day minus surgical or anesthetic complications.

Discussion

Beach chair or sitting position is preferred by surgeons during shoulder procedures for four reasons: 1) unnatural traction on the shoulder is not necessary; 2) an arthroscopic procedure could be converted to an open surgery without patient repositioning; 3) incidence of traction related nerve damage is significantly decreased as brachial plexus stretch is reduced; and 4) upright positioning aids surgical exposure and manipulation of the arm and shoulder.[5,6]While upright positioning is ideal for surgical performance, many physiological changes occur that require increased awareness and understanding from the anesthesia practitioner.
Normal physiological changes that occur when a patient positioned upright are mainly due to gravity.[2] Upright positioning under non-anesthetized conditions causes a series of related cardiovascular phenomena:

venous pooling, decreased venous return, up to 20% decrease in stroke volume (SV) and cardiac output (CO), and roughly 15% decrease in cerebral perfusion pressure (CPP).[2] These changes are normally accompanied by a compensatory increase in systemic vascular resistance (SVR) of between 50% to 80% .[2] General anesthesia blocks the compensatory increase in SVR through the vasodilating and myocardial depressing effects of anesthetic agents.[2]Venous return from cerebral circulation is increased by negative intrathoracic pressure during spontaneous respiration.[2] This too is negated during general anesthesia because of positive pressure ventilation.[2] Obstruction of internal jugular veins from head misalignment in beach chair position can also contribute to poor cerebral venous drainage.[2]

Another complication associated with upright positioning is activation of the Bezold-Jarisch reflex.[5]Profound hypotension and bradycardia frequently occur from activation of the Bezold-Jarisch reflex during shoulder surgery when an interscalene block has been used.[5]
The anesthesia practitioner must consider that BP in the brain differs from the site where BP is actually measured.[1] The difference in BP is equal to the hydrostatic pressure gradient between sites.[1] The base of the brain is approximately 20 cm higher than the heart in average adults with an additional 9 cm difference to the Circle of Willis.[1] This equates to a 15 mmHg variation from the heart to the base of the brain.[1]Some anesthesia practitioners prefer calf cuff placement to avoid interference with IV flow during cuff inflation. The hydrostatic discrepancy between the calf and brain has been documented as high as 94 mmHg.[7]The anesthesia practitioner may interpret this high reading as hypertension caused by pain or inadequate anesthesia. Not realizing this measurement correlates with a significantly lower BP in the brain could be dangerous. The consideration for BP variance becomes especially crucial when induced hypotension is requested.[7] Induced hypotension may have advantages for the patient and surgeon, but negligence or ignorance regarding compensation for BP measurement location can be disastrous.[7]

With a thorough understanding of physiological changes associated with upright positioning, anesthesia practitioners can avoid catastrophic neurologic outcomes resultant from positioning during should surgery.[2] The following recommendations should be followed: 1) place the cuff on the non-surgical arm to avoid the larger discrepancy associated with calf cuff placement; 2) use the level of heart as the gold standard reference for non-invasive BP monitoring during upright positioning; 3) treat perioperative BP lower than 80% of preoperative baseline values aggressively to increase the margin of safety and avoid potential ischemia; 4) if invasive BP monitoring is used, the transducer should be leveled at the external meatus rather than the heart; 5) never induce hypotension to a specific value (e.g. 100 mmHg) without first considering MAP, patient position, BP cuff location, and pre-existing conditions that require increased intraoperative BP (e.g. carotid stenosis).[2,7]

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Perioperative Anesthetic Management of Myasthenia Gravis

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Keywords: myasthenia gravis, epidural, sevoflurane, cystectomy, ropivacaine

We report the case of a patient with myasthenia gravis (MG) that presented for a radical cystectomy with extracorporeal ileal conduit urinary diversion for treatment of bladder cancer. Delivery of anesthesia for the MG patient poses special challenges and risks including careful pre-operative optimization of medical therapy, management of interactions between drugs used to treat MG and muscle relaxants, and preparation of the patient for possible post-operative intubation and mechanical ventilation.

This case report highlights the importance of the anesthesia practitioner having an in-depth knowledge of MG. Additionally, this report illustrates the utility of neuraxial anesthesia and an inhalational anesthetic approach in conjunction with train-of-four monitoring as an alternative to conventional anesthesia involving muscle relaxants in the setting of MG.

Case Report

A 52-year old female (weighing 85 kg with a height of 162.6 cm) with MG Type IIA Ossermann and Genkins classification presented to an urban cancer center for a radical cystectomy with extracorporeal ileal conduit urinary diversion for the treatment of bladder cancer. The patient had a past medical history of bladder cancer and MG. She was taking oral pyridostigmine, 60mg, four times daily. On physical exam, the patient was afebrile and had stable vital signs. Further exam revealed moderate muscle weakness and ocular symptoms including ptosis. The rest of her exam was normal. Her pulmonary function tests were within normal limits. Airway exam revealed a Mallampati classification I airway and ability to subluxate the mandible.

The patient received her morning dose of oral pyridostigmine 60 mg preoperatively. Before transport to the operating room, the patient received midazolam 1 mg

intravenously (IV) via a 14 gauge catheter. She positioned herself on the operating table and standard noninvasive monitors were applied. After baseline vital signs were recorded, the patient was assisted into the sitting position in preparation for epidural insertion. Ketamine 20 mg was administered to the patient prior to epidural insertion. The epidural catheter was placed at T8-9 level and a test dose of 0.25 percent bupivacaine with 1:200,000 epinephrine was administered through the catheter. The patient was assisted to the supine position.

After adequate preoxygenation with oxygen 100 %, anesthesia was induced via sevoflurane 8%, propofol 100 mg IV and fentanyl 150 mcg IV. After the patient became deeply anesthetized and laryngeal structures relaxed, laryngoscopy was performed with a resulting grade I view. The trachea was intubated with a cuffed 7.5 endotracheal tube (ETT). After correct placement of the ETT was confirmed, sevoflurane in oxygen 1L/min and air 1L/min was administered and depth of anesthesia was titrated to keep a train of four (TOF) count of 3 out of 4 twitches. Tidal volumes were maintained at 5 to 7 ml per kg to maintain an end tidal carbon dioxide concentration between 30 and 35 mmHg and an oxygen saturation of greater than 97 %.

Prior to surgical incision, an additional 14 gauge IV catheter was placed; the radial artery was cannulated; and the epidural catheter was bolused with 0.2% ropivacaine 10ml. An epidural infusion of 0.2% ropivacaine was started at 10ml per hour and titrated to a heart rate greater than 60 beats per minute and systolic blood pressure greater than 100mmHg. Neuromuscular blockers were not administered at any time during the surgery. Neuromuscular function was monitored with TOF peripheral nerve stimulation. Perioperatively, TOF monitoring was always equal to or greater than 3 out of 4 twitches at any time during anesthesia. Total operating time was 360 minutes. Prior to extubation, ondansetron 4mg IV was administered.

Following the surgical procedure, the patient was extubated in the operating room after demonstration of sustained head lift, the ability to follow commands, spontaneous ventilation with tidal volumes greater than 500ml and a respiratory rate greater than 12 breaths per minute. The patient was extubated with no sequellae. Oral pyridostigmine was resumed 4 hours postoperatively. The patient was observed overnight in the Intensive Care Unit and made an uneventful recovery.

Discussion

Anesthesia practitioners must have a thorough understanding of the pathophysiology of the specific disease process to provide safe and effective care. This is especially important in the context of neuromuscular diseases because of implications in the perioperative and postoperative period.

MG is an autoimmune disease characterized by weakness and fatigability of skeletal muscles, which improves following rest[1]. It may be localized to specific muscle groups or it may be generalized. The incidence of MG is 50 to 142 cases per million[2]. MG is caused by a decrease in the numbers of postsynaptic acetylcholine receptors at the neuromuscular junction, which decreases the capacity of the neuromuscular end-plate to transmit the nerve signal[1,2]. Initially, in response to a stimulus resulting in depolarization, acetylcholine is released presynaptically. In MG, the number of activated postsynaptic receptors may be insufficient to trigger a muscle action

potential. Further, with repeated stimulation, the decline in release of acetylcholine correlates with the characteristic fatigability[1-3]. The Ossermann and Genkins classification system was originally developed to prognosticate outcomes in MG patients following thymectomy, with Stages I and IIa having the best remission rates[4]. The classification system is also used in MG patients undergoing other types of surgeries to anticipate and manage the peri- and post-operative course.

Inhaled anesthetics may cause muscle relaxation in normal patients, but profound relaxation in MG patients. Isoflurane increases TOF fade in the myasthenic patient[5]. It depresses twitch height twice as much as equipotent concentrations of halothane. TOF responses are also decreased to varying degrees in myasthenic patients receiving enflurane[5]. Sevoflurane at a concentration of 2.5 percent depresses electromyography responses[6]. A recent report found sevoflurane was suitable as a sole anesthetic for a myasthenic patient undergoing sternal split thymectomy, implying that sevoflurane alone provided adequate muscle relaxation[7]. Sevoflurane appears to depress neuromuscular transmission to the same degree as isoflurane. After discontinuation of sevoflurane, TOF ratios return to values not significantly different from baseline[5-7]. Low blood-gas and tissue-gas solubility might be partly responsible for this rapid recovery from neuromuscular depression by sevoflurane. It has been demonstrated that sevoflurane has an inhibitory effect on neuromuscular transmission in both MG and healthy patients. The inhibitory effects of sevoflurane were more prominent in MG patients than healthy patients[7,8].

Potentiation of neuromuscular blocking drugs by local anesthetics has been reported; therefore, regional and local anesthesia should be performed using reduced doses of local anesthetics to avoid high blood levels[5]. These agents decrease the sensitivity of the postsynaptic membrane to acetylcholine. This could cause weakness in MG patients if blood levels are high. Ester anesthetics, which are metabolized by cholinesterase, may present particular problems in patients taking anticholinesterases[5,9]. The safe and successful use of thoracic epidural blockade with bupivacaine for intraoperative anesthesia and postoperative analgesia has been reported[10]. Epidural techniques facilitate tight control of blockade level and may obviate the need for opioids in postoperative pain management. Other studies have shown that the combination of a thoracic epidural and total IV anesthesia with propofol and remifentanil is an effective technique for providing hemodynamic stability during surgery followed by rapid awakening, a quick transition to spontaneous breathing, excellent postoperative analgesia and an uneventful recovery[10].

The safe use of general anesthesia in this setting requires attention to monitoring the patient and understanding the variable responses that the MG patient may have to different drugs. The use of potent inhalational agents facilitates tracheal intubation and provides relaxation for surgery and in turn helps the anesthesia professional avoid neuromuscular blocking agents altogether[11]. Compared with muscle relaxants, inhalational agents allow earlier recovery of neuromuscular transmission, with rapid elimination of these agents at the end of surgery. In addition, regional techniques may reduce or eliminate the need for muscle relaxants in abdominal surgery[5,10]. Epidural techniques offer the advantage of postoperative pain control with minimal or no opioid use.

Patients with bulbar involvement are at increased risk for respiratory depression and pulmonary aspiration during the perioperative period. In turn, this increases the likelihood of the need for continued post-operative mechanical ventilation. There have been several attempts including the use of pulmonary function tests to predict the need for postoperative ventilation in MG patients[11]. Based on the preoperative condition of the patient, the surgical procedure, and the residual anesthetic effects, a carefully planned extubation may be carried out in most patients[12]. Adequate postoperative pain control and the avoidance of drugs that interfere with neuromuscular transmission will facilitate tracheal extubation and early recovery.

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EDITORIAL

Greetings

I was honored when Ron Van Nest, CRNA, JD asked if I would be interested in assuming the role of Editor in Chief of ***The International Student Journal of Nurse Anesthesia***. My involvement with the journal began over four years ago, when, at the suggestion of Associate Editor Julie Pearson, CRNA, PhD, I submitted a case report for one of my graduate students for the first time. It was not long before Ron recruited me to serve as a reviewer, and then Associate Editor. What a great pleasure it has been to grow with this journal, both in witnessing the sense of accomplishment felt by the student authors I have mentored and gaining the experience necessary to take the reigns as editor.

I cannot thank Ron and Julie enough for their guidance and support during this transition, but as Ron has said on many occasions, this journal would not be possible without the hard work and dedication of the Section Editors, Reviewers, and Mentors who graciously volunteer their time and effort toward this worthwhile endeavor. The success of this journal is a testament to these individuals in promoting scholarship and advancing knowledge in our profession.

As I must learn to walk before I can run, my immediate goal for this, my first, issue has been the on time publication of a product worthy of the readership. I hope you will find I have fulfilled this goal. Future plans of the Editorial Board include adding evidence-based analysis reports and increasing the visibility of the journal. The addition of the journal to EBSCO Publishing's title list and the Cumulative Index to Nursing and Allied Health Literature (CINAHL), one of Ron's many accomplishments during his tenure as Editor, will help serve the latter. To realize the former, and increase in the number of Section Editors and Reviewers will be necessary. If you have served as a Mentor in the past, please do not be surprised if you receive an invitation from the Editorial Board to serve in one of these roles! And to all the CRNAs who published as graduate students and now serve as clinical instructors or faculty, carry on the tradition by serving as mentor to a new author.

So, it is with excitement that I move forward in leading this journal. Ron could not have made this easier, with his vision and the path he has laid. I look forward to building on his legacy.

Vicki C. Coopmans, CRNA, PhD
Editor

He who would learn to fly one day must first learn
to stand and walk and run and climb and dance;
one cannot fly into flying.

Friedrich Nietzsche