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TOPICS IN THIS ISSUE

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Front Cover:
Jen Phelan, RN, BSN, a senior graduate student enrolled in the Nurse Anesthesia Program at Northeastern University and Rwandan nurse anesthetist Marie Ange Nyirankundabekize work together to secure intravenous access for a patient undergoing fistula repair at Kibagabaga Hospital (Kigali, Rwanda) during a global health clinical experience with the International Organization for Women and Development.

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Unanticipated Difficult Airway and Lingual Tonsillar Hypertrophy

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Keywords: lingual tonsillar hypertrophy, unanticipated difficult airway, difficult intubation, fiberoptic intubation, light wand stylet, retrograde intubation

Lingual tonsillar hypertrophy (LTH) can cause an unanticipated obstructed view of the epiglottis and the vocal cords. Lingual tonsils exist at the base of the tongue and are bounded by the epiglottis posteriorly, vallate papillae anteriorly, and tonsillar pillars bilaterally. Following induction of anesthesia and muscle relaxation, lingual tonsils may act as a "ball valve" that prohibits antegrade ventilation or insertion of an endotracheal tube.[1] Sarcoidosis is an inflammatory disease that affects multiple organs. Abnormal masses of inflamed tissue, called granulomas, form in certain organs and tissues.[2] Upper respiratory tract involvement with sarcoidosis is well recognized.[3] On visualization, there is fullness in the rim of the epiglottis with diffuse enlargement and a granular appearance along supraglottic structures. This appearance is consistent with laryngeal sarcoidosis, and can prevent visualization of the true vocal cords.[3] This case report involves an African-American female with a history of sarcoidosis. She presented for surgery with asymptomatic airway involvement of her sarcoidosis. A difficult tracheal intubation was encountered.

Case Report

A 58-year-old, 87 kg, 165 cm African-American female presented for resection of a posterior thigh mass. The planned operative position was prone. She had a past medical history significant for sarcoidosis, and obesity (BMI 32 kg/m²). Review of past anesthetic records revealed no airway difficulties.

Examination of her airway revealed a Mallampati I classification, thyromental distance of 4 cm, mouth opening 4 cm, large tongue, and neck movement with full range of motion. She was premedicated with intravenous midazolam 2 mg, glycopyrrolate 0.2 mg, and a transdermal scopolamine patch (1.5 mg) was applied topically. Direct laryngoscopy (DL) with general anesthesia was planned due to prone positioning.

Upon arrival to the operating room standard monitors were applied, and the patient was preoxygenated with 10 L/min of 1.0 FiO_2 oxygen. The patient's anesthetic was induced intravenously with fentanyl 150 mcg , lidocaine 50 mg , propofol 200 mg and after confirmation of successful mask ventilation, rocuronium 30 mg was administered. Direct laryngoscopy with a Macintosh 3 blade revealed a grade 3 glottic view. A second attempt was made with a Miller 2 blade also revealing a grade 3 glottic view, and an attempt to pass a 7.0 ETT was unsuccessful. There was no visualization of hypertrophied tissue with direct laryngoscopy. A light wand intubation was then attempted without success. The patient's SpO_2 remained 100%, and mask ventilation was resumed and with inhalation of isoflurane 1% while a fiberoptic scope was obtained.

Fiberoptic laryngoscopy exposed a significant amount of pink hypertrophied tissue anterior to the epiglottis which obstructed the view of the epiglottis. The

carina was visualized, and a 7.0-mm ID endotracheal tube was passed through the vocal cords and secured at 23 cm at the lip. Endotracheal tube position was confirmed by auscultation of bilateral breath sounds and end tidal carbon dioxide. The patient developed bilateral wheezing after intubation and was treated with 12 puffs of albuterol via the ETT, and dexamethasone 10 mg IV was also administered. The posterior thigh mass was then successfully removed. No further complications occurred throughout the procedure.

The patient was transported to the post anesthesia care unit (PACU) with an endotracheal tube in place due to the concern of airway edema secondary to the multiple intubation attempts, and noted facial edema. The patient remained stable and was extubated within 45 minutes of admission to 40% oxygen via face shield. The patients SpO_2 remained consistent at 100%. She was observed in the PACU for an additional 1 hour following extubation, and was then transferred to a surgical, non-monitored bed. Discharge occurred the following day. A comprehensive progress note was written by the attending anesthesiologist, and a detailed report of the difficult intubation was noted in her anesthesia record for future reference.

Discussion

Lingual tonsillar hypertrophy (LTH) is a documented cause of difficult mask ventilation and tracheal intubation. LTH is often asymptomatic, although some patients complain of alteration in voice, cough, snoring, or obstructive sleep apnea.[4] This patient's preoperative airway assessment was normal and previous anesthesia records did not reveal any difficulty with prior endotracheal intubation. Lingual tonsils consist of lymphoid tissue located at the base of the tongue which are bound by the epiglottis posteriorly, and can become inflamed and hypertrophied. LTH displaces the epiglottis posteriorly, and with the presence of redundant pharyngeal tissue, a view of the vocal cords may be significantly obscured leading to failure of rigid laryngoscopic intubation. The most common factor that limits successful tracheal intubation is the inability to visualize the vocal cords during direct laryngoscopy.[4]

Tonsillar hypertrophy and other airway disease processes should alert the anesthetist to the possibility of difficult laryngeal visualization. For example, sarcoidosis of the upper airway is also associated with the inability to visualize the true vocal cords. This usually occurs with supraglottic involvement. Although relatively rare, sarcoid granuloma with laryngeal infiltration has been reported.[3] The possibility of airway involvement must be considered in patients with known granulomatous disease. There have also been numerous reports supporting a majority of sarcoid cases within the African-American ethnicity. The mortality rate for sarcoidosis among African-Americans has been reported as much higher than that of Caucasian Americans.[3]

In this patient, unsuccessful attempts at endotracheal intubation with both the Macintosh 3 blade and the Miller 2 blade were encountered, face mask ventilation was resumed, and adequate oxygenation was maintained. According to the American Society of Anesthesiologists' (ASA) Difficult Airway Algorithm, if face mask ventilation is adequate, a non-emergency pathway can be taken. Acceptable alternative non-invasive approaches at this point include using a different laryngoscope blades, laryngeal mask airway (LMA) as an intubation conduit, fiberoptic intubation,

intubating stylet, light wand, retrograde intubation, and blind or nasal intubation.[5]

Lighted stylets (light wands) consist of a malleable stylet with a light at the distal tip. The ETT is mounted on the stylet, and a "hockey stick" curve is placed in the distal third of the tube. The assembled device is advanced in the midline of the airway until a well-circumscribed glow is seen through the anterior surface of the neck. As the device is advanced further, the light should remain distinct as it is seen to travel down the neck and disappear under the sternal notch.[6] Because it is a blind technique, success of light wand intubation depends on predictable anatomy. In this patient, the light wand was unable to be passed down the larynx and through the vocal cords into the trachea, probably due to distortion caused by the hypertrophied lingual tonsils.

Fiberoptic intubation (FOI) is useful in a patient with an anticipated difficult airway. It is also useful when the difficult airway is unanticipated, provided there is enough time to prepare and carry out the technique. With a known difficult airway, awake FOI remains the "gold standard".[7]
An important difference in performing fiberoptic laryngoscopy in an anesthetized versus the awake patient is that the soft tissues of the pharynx tend to relax and limit space for visualization with the fiberoptic bronchoscope.[6] In addition to the relaxed soft tissue, the optics of the fiberoptic scope can become easily obscured with blood and secretions. This patient had received glycopyrrolate 0.2 mg IV preoperatively as an antisialogogue for the prone position, and this proved beneficial for the FOI. Fortunately, FOI in this already anesthetized patient was quickly achieved, and the presence of LTH was unexpectedly discovered.

Lingual tonsillar hypertrophy is life-threatening, and can rapidly progress to a "cannot intubate, cannot ventilate" situation. When this occurs, the difficult airway algorithm takes the emergency pathway. The first action is to call for help, and with failure of a non-invasive airway, to immediately obtain invasive airway access.[5] Invasive airway access includes surgical or percutaneous tracheostomy or cricothyrotomy. Also reported in the literature is rationale supporting the early use of wire-guided retrograde intubation.[7]

Pre-operative airway assessment techniques are not 100% sensitive for predicting a difficult tracheal intubation. In patients with a history of sarcoidosis, a prior history of an easy intubation does not guarantee subsequent intubation attempts may not be difficult. Patients with sarcoidosis may develop lingual tonsillar hypertrophy, which can contribute to difficulty with intubation. When preparing to provide anesthesia for a patient with sarcoidosis anesthesia providers should ensure they have additional difficult airway equipment available and remember to call for help early if an unanticipated difficult airway is encountered.

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Cytoreductive Intraperitoneal Hyperthermic Chemotherapy

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Keywords: intraperitoneal hyperthermic chemotherapy, fluid management, temperature management

Peritoneal carcinomatosis (PC) is widespread metastasis of cancerous tumors from non-gynecological malignancies to the inside of abdominal surfaces. Patients with PC have a mean survival of six months, with staging at the time of diagnosis providing important prognostic information.[1] Combining cytoreductive surgery (CS) with intraperitoneal hyperthermic chemotherapy (IPHC), treatment has been shown to increase median survival time to approximately 8.4 months.[1] This procedure is challenging due to alterations in patients' temperature, fluid status, electrolyte balance, and pain level. It is imperative that anesthesia practitioners be familiar with these challenges during this surgical procedure.

Case Report

A 60-year old, 166 cm, 68 kg male was scheduled to have IPHC following a diagnosis of PC. He had a past medical and surgical history significant for kidney stones and an appendectomy. Laboratory values were within normal limits with the exception of a low hemoglobin at 12 g/L and hematocrit of 39%. All pertinent documentation was verified and the patient was given versed 2 mg intravenously (IV) before proceeding to the operating room (OR). The patient was then transferred to the surgical table and standard noninvasive monitors were applied.

The patient was pre-oxygenated with 10 L/min of oxygen via face mask. General anesthesia was induced using IV fentanyl 100 mcg, lidocaine 100 mg, propofol 175 mg, and rocuronium 50 mg. One attempt at direct laryngoscopy with a macintosh 3

blade revealed a grade I view, and the trachea was intubated atraumatically. The endotracheal tube was secured at 23 cm at the lips after equal bilateral breath sounds and positive end tidal CO_2 were both confirmed. The patient's right radial artery was cannulated to monitor blood pressure, and an additional 16 gauge IV catheter placed in the left forearm. Isoflurane at 1%, oxygen and air each at 0.5 liters/minute were administered. Neuromuscular blockade was maintained with vecuronium, and fentanyl boluses were given for analgesia and additional analgesia through a continuous thoracic epidural catheter with 0.125% bupivicaine and 2 mcg/ml fentanyl.

After induction, a two-part procedure began. The first procedure included two biliary stents placed by urology in the lithotomy position. The patient was repositioned supine and a midline abdominal incision was made and visible cancerous tissue was excised. After resection of all gross intraperioneal disease, hyperthermic peritoneal cytotoxic fluid of mitomycin C was administered. The patient's temperature ranged between 35^0C to 38.5^oC. A forced air warmer and an inline fluid warmer were discontinued with initiation of the warmed peritoneal fluid irrigation.

Total blood loss was 1,200ml, and the urine output 1,600 ml. Intravenous fluid totals were as follows: lactated ringer's 3L, albumin 5% 750 ml, normal saline 3L, and packed red blood cells 4 units were administered. Arterial blood gases were monitored hourly during the case. The procedure was completed and neuromuscular blockade was antagonized using neostigmine 3.5 mg and glycopyrrolate 0.6 mg. The oropharynx was suctioned and following the return of spontaneous respiration, adequate tidal volume, and ability of the patient to follow commands the trachea was extubated. Supplemental oxygen was provided. The patient was transported to the intensive care unit with supplemental oxygen and hemodynamic monitoring in stable condition.

Discussion

Cytoreductive intraperitoneal hyperthermia chemotherapy involves direct administration of heated cytotoxic chemothrepeautic agent such as mitomycin C into the peritoneal cavity following cytoreductive surgery. There are two techniques for IPHC, the "closed technique" and the "coliseum technique", also known as the "open abdomen technique." The open abdominal technique, which was performed in this case, involves placing a Tenckhoff catheter and closed suction drains through the abdominal wall, and creating a watertight seal with a purse-string suture at the skin. Temperature probes are placed on the skin edge and secured to a retractor and a plastic sheet cover. The surgeon, through a slit in the plastic cover, is able to access the abdomen and manipulate viscera to keep adherence of peritoneal surfaces to a minimum. Roller pumps force the chemotherapy solution into the abdomen through the Tenckhoff catheter and pull it out through the drains. A heat exchanger keeps the fluid being infused at temperatures of 44°C to 46°C, so that the intraperitoneal fluid is maintained at 42°C to 43°C.

After the intraoperative perfusion is complete, the abdomen is suctioned dry of fluid. The abdomen is then reopened, and reconstructive surgery is performed.[2] This technique is advantageous as it provides access to the entire abdomen and pelvis during the intraperitoneal perfusion, allowing the surgeon to ensure adequate treatment with heat and chemotherapy to

every abdomino-pelvic region while preventing excessive heating of normal tissue. It also provides an access to sample the perfusate and measurement of chemotherapeutic solution concentrations while enabling identification and containment of bleeding.[2]A drawback of this technique is that heat dissipation may make it harder to achieve uniform hyperthermia during the 90-120 minutes of IPHC.[2]

The cytoreductive stage (CS) involves surgical removal of all macroscopic disease in the involved organs, peritoneum, and associated tissues.[3]This phase is associated with significant blood loss. Hypovolemia due to extended bowel preparation for this surgery, coupled with tissue resection during the CS phase presents a considerable challenge for volume resuscitation and hemodynamic shifts. Reduced intravascular volume may cause a decrease in preload leading to splanchnic hypoperfusion, and postoperative complications such as cholecystitis and intestinal ischemia and increased morbidity and mortality.[4] Adequate intraoperative volume resuscitation optimizes cardiac preload, blood viscosity, and peripheral perfusion.[4] Impairment of coagulopathy has been attributed to large volume shifts and protein loss with high fluid turnover and possibly the hyperthermic chemotherapy solution. Laboratory analysis during the case may reveal an increase in International normalized ratio (INR) a fall in Antithrombin III values (AT III), Prolonged partial thromboplastin time (PTT) and a reduced number of platelets.[5]

During the first stage of the surgery patients may require large amounts of volume due to NPO status, bowel preparation, blood loss and third-space deficit. Fluid status can be evaluated using central venous pressure monitoring and/or an esophageal doppler. Arterial blood gases can be used to determine perfusion adequacy and electrolyte imbalances attributable to volume shifts. It is also imperative to maintain urine output. In some cases, low dose furosemide therapy may be used. On the contrary, volume overload can lead to alterations in organ function and postoperative implications.[4] In this case; we used an arterial line and monitored urine output. Volume resuscitation was achieved using blood, crystalloids, and colloids.

The second stage involves infusion of the heated cytotoxic agent mitomycin C. Hemodynamic stability must be carefully assessed and normovolemia restored before starting IPHC to avoid hypotension. Hyperthermia increases the penetration of chemotherapy into tissues. Heat is also cytotoxic to cancer cells and increases the cytotoxicity of selected antineoplastic agents. This synergism occurs only at the interface of heat and body tissue at the peritoneal surface.[1-2] Before commencement of this stage, all IV fluid warmers and forced warm air blankets must be discontinued to prevent hyperthermia. Patients develop an increased metabolic rate, a rise in end tidal carbon dioxide values; metabolic acidosis and elevated arterial lactate values are mild and transient. An increase in oxygen consumption and extraction due to hyperthermic metabolic conditions during IPHC has been documented. The body's initial compensation to heat stress is dilation of the peripheral vasculature, thus increasing heat loss from the core to the environment. Maintaining normothermia presents an anesthesia concern during this procedure, a forced warm air blanket and fluid warmer are needed to prevent hypothermia during the first stage however during the second stage, it is imperative to turn these devices off before this stage is underway. The heart rate increases to maintain cardiac output in

the face of decreasing peripheral vascular resistance. This normalizes as chemotherapeutic lavage is completed, although the heart rate may remain elevated.[5]

This surgery is painful due to the extensive abdominal resection. Supplementary epidural analgesia during both intra- and postoperative period has been shown to reduce the necessity and duration of postoperative ventilation and also the need for opioids, which decreases the length of hospital stay.[5] The literature also suggests that epidural provides better post operative analgesia, early recovery of bowel function, fewer side effects and higher patient satisfaction.[5] On the contrary, some reports say that pain control can also be achieved without epidural infusions of local anesthetic, and seldom impacts the need for postoperative mechanical ventilation.[6] In our case, analgesia was provided via intravenous fentanyl and through a continuous thoracic epidural catheter with 0.125% bupivicaine and fentanyl 2 mcg/ml.

In conclusion, understanding the dynamics of this lengthy and challenging procedure should not be underestimated. Fluid management, temperature regulation, hemodynamic changes, and pain management are key areas anesthesia practitioners must address in order to improve patient outcome and satisfaction in a population that is faced with a life-threatening terminal disease.

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Anesthetic Management of a Patient with Ludwig's Angina

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Keywords: Ludwig's Angina, inflammation, submandibular, sublingual, dental infections

Ludwig's Angina is a non-localized inflammation of the submandibular, submental, and sublingual areas that is capable of becoming life-threatening.[1] Ludwig's Angina may expand rapidly and usually occurs in young children and adults with dental infections or previous dental procedures.[2] Warning signs include oral swelling, fever, dysphagia, toothache, severe neck pain, drooling, bad breath, and malaise.[2] Patients presenting with stridor are at high risk for future airway emergencies.[3] Streptococci and staphylococci are the usual causes of the bacterial infection. Other factors that contribute to this infection are sickle cell disease, a compromised immune system, and trauma.[4] The mortality rate for patients with this diagnosis is 0-10%.[3]

Case Report

A 22-year-old female, 170 cm in height, weighing 59 kg was admitted to the emergency department with the chief complaint of swelling and tenderness to the left side of her face after having a dental procedure, and noticed the edema was increasing over the next few days. Ludwig's Angina was the diagnosis upon admission. She admitted to having a recent dental extraction. Her past medical history includes smoking 1 pack of cigarettes per week for 3 years. Physical assessment on the day of admission showed a healthy, young female, with trismus. A large, left submandibular abscess and sublingual abscess was noted on initial assessment. The patient appeared extremely nervous and confused. Her airway was assessed and determined to be a Mallampati class IV. The patient received solumedrol 125mg IV, morphine 3mg IV, vancomycin, clindamycin, and hydromorphone 0.4mg IV in the emergency room. The patient was not currently on any home medications.

Labs were drawn and were as follows: WBC 20.7 x 10^3 /μL, Hbg 13.7 g/dL, Hct 42.3%, platelets 281x 10^3/μL, and a urine pregnancy test which was negative. Electrolytes and coagulation studies were within normal range. The patient noted an allergy to penicillin. The patient was to undergo incision and drainage of the sublingual, submental, and submandibular space, with possible dental extraction. The patient had been NPO for 9 hours. The patient received midazolam 5 mg intravenous (IV) pre-operatively. A right nasal trumpet was placed in the pre-operative holding area and lidocaine 4% topical was added to the left nare and pharynx using a laryngeal tracheal anesthesia (LTA) kit.

The patient was transferred to the operating room in the sitting position. She moved herself to the table and remained in the sitting position during induction. Noninvasive monitors were applied and the patient was preoxygenated with O_2 10 L/min for 5 min. In preparation for a possible tracheotomy an otolaryngology surgeon was present with a tracheostomy kit at the bedside. propofol 100 mg IV and fentanyl 50 mcg IV were administered, and FOB was attempted unsuccessfully. After a failed second attempt at FOB positive pressure mask ventilation was initiated successfully

and an additional dose of propofol 100 mg IV was administered. Direct laryngoscopy was then attempted and the trachea was successfully intubated without neuromuscular blockade. The anesthesia practitioner assessed positive $ETCO_2$ and bilateral breath sounds.

The 7.5 mm endotracheal tube (ETT) was secured and general anesthesia was maintained. Vecuronium 3 mg intravenous was given to maintain neuromuscular blockade, with sevoflurane at 2%. A throat pack was added and removed per the oral maxillofacial surgeon (OMFS). The OMFS also wired the ETT to the patient's molar tooth and placed an orogastric tube at the end of the procedure. Midazolam 5 mg and hydromorphone 1 mg were given at end of the procedure. The patient remained intubated, was transferred to the intensive care unit with stable vital signs, and eventually extubated and discharged.

Discussion

Ludwig's Angina is a rare, cellutitis in the floor of the mouth that can expand quickly.[3] The criteria for diagnosis of Ludwig's Angina is dyspnea, fever, neck pain and swelling, malaise, difficulty swallowing, and drooling.[2] A physical examination of the neck and head will show swelling and redness of chin and upper neck. The tongue may also be edematous.[3] A CT scan is the most widely used tool to localize abscesses in the head and neck and direct surgical planning, although this was not performed in this case.[1]

This infection can be life-threatening if the airway is compromised at any time. Formerly this diagnosis was fatal, but since the development of antibiotics and aggressive surgical approaches in the 1940s, the mortality rate has been greatly reduced.[5] Early recognition of the microorganism causing the infection will indicate the need to initiate antibiotics, which is a crucial step in preventing morbidity and mortality.[5] The most common pathogens in Ludwig's Angina are streptococci and staphylococci.[4] The patient in the current case study was allergic to penicillin, so the alternative clindamycin was used. Penicillin is used in these cases because it targets gram positive organisms and kills streptocci.[6] The rationale for the use of steroids is to decrease soft tissue edema by decreasing the activity of the immune system. Dexamethasone (4-8 mg IV) or solu-medrol (125 mg IV), both glucocorticoids, can be administered. [7]

Constant assessment and maintenance of the airway is the most imperative concern for anesthesia practitioners.[2] Submandibular edema and an edematous, elevated tongue are signs that immediate airway intervention is necessary. [3] This patient was cooperative, but apprehensive. This diagnosis is considered an emergency because at any moment the airway could become compromised. Fiberoptic bronchoscopy to intubate the trachea was the initial plan because it can provide direct visualization of the vocal cords and improved placement of the ETT.[5] In this case, pre-operative use of the LTA kit was performed to assist the anesthesia practitioner with awake fiberoptic bronchoscopy (FOB), even though infected tissue is not easily anesthetized. Oral secretions were obstructing the view during FOB, so direct laryngoscopy was performed. A Fast-Track LMA was available if the alternative plan failed. The most serious complication would be airway compromise and inability to establish an airway, so being prepared for a tracheostomy is a top priority.[2] Anterior neck edema is common with this diagnosis, which could make a tracheotomy technically difficult.

The decision was made to administer benzodiazepines to provide anxiolysis for the patient in the pre-operative holding area while the operating room was prepared for this emergency procedure, and necessary hospital personnel were available at the bedside. Upon transfer to the operating room the patient was cooperative and responsive while the propofol and fentanyl were administered for the awake intubation attempt. Neuromuscular blockers were avoided to allow for spontaneous ventilation, and decrease the risk of being unable to ventilate and intubate. Neuromuscular blockers may lead to occlusion of the airway because of loss of tone of the pharyngeal muscles.[2]

Glycopyrrolate, a quaternary ammonium compound, could have been given on induction to decrease secretions and possibly improve the view during FOB. Once intubated, the patient was turned away from the anesthesia provider. This would make an emergency re-intubation or tracheotomy difficult if the airway became compromised. Vigilant monitoring and securing of the airway is necessary.

Anesthesia practitioners must be aware of the signs of Ludwig's Angina and be able to make quick decisions regarding airway management. To ensure the safety of all patients with Ludwig's Angina, preparation for a difficult airway is critical. Good communication between anesthesia and the surgery team regarding airway management, taking into consideration all possible complications, will lead to the best outcome for this potentially lethal diagnosis.[3]

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Epiglottitis: A Case Study

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Keywords: epiglottitis, supraglottitis, airway obstruction, pediatric epiglottitis, adult epiglottitis

Epiglottitis is an acute life-threatening emergency. This is an infection of the epiglottis, aryepiglottic folds, and arytenoids, which can lead to complete airway obstruction very quickly.
This infection mainly affects children due to *Haemophilus* (*H*) *influenzae* type B, but since the vaccine for this pathogen there is a decrease in cases of pediatric epiglottitis.[1] Anesthesia practitioners must be wary of symptoms for epiglottitis and know appropriate actions to support a deteriorating airway. The importance of early recognition, preparation, and treatment is crucial for patient survival.

Case Report

A four- year-old, 24 kg, female presented emergently to the operating room for direct laryngoscopy and possible endotracheal tube placement (ETT) or possible tracheostomy. She was a patient in the pediatric intensive care unit (PICU) when the surgeon diagnosed her with suspected epiglottitis. Her past medical history consisted of asthma, gastro esophageal reflux, and myelodysplastic syndrome, with allergies to codeine and methadone. Her anesthetic history consisted of gastric tube placement and magnetic resonance imaging, with no complications from anesthesia. No laboratory values or diagnostic tests were necessary for this surgery. Her mallampati airway assessment was 3. The patient had edema noted around her cheeks, jaw, and neck regions, and her cheeks were flushed. She had no loose teeth and increased clear secretions. The patient was up-to-date with childhood immunizations. Her last gastric tube feeding had been over 9 hours.

She was transferred from the PICU to the operating room on a stretcher in the sitting position. The patient's parents accompanied her down to the surgery department, but did not proceed with her into the operating room. No sedation was given prior to transferring her to the operating room. She had excessive drool, inspiratory stridor, anxiety, and her crying was muffled. The patient was positioned in the sitting position on the operating bed. The pulse oximetry monitor was connected to the patient and a reading of 95% was obtained. Inhalational induction using sevoflurane, nitrous oxide, and oxygen was performed while in the sitting position. After induction, the patient was placed in the supine position and standard ASA monitors applied while bag mask ventilation was performed. A 22 gauge peripheral intravenous (IV) catheter was inserted into the patient's right forearm. Manual ventilation with sevoflurane and oxygen continued until an expired concentration of 2.3 was noted.

The bed was turned 90 degrees and the Ear Nose and Throat surgeon performed direct laryngoscopy and ETT placement without complication. A 4.5 uncuffed ETT was secured and the operating bed was turned back 90 degrees. The ETT was connected to the breathing circuit and ventilation continued with oxygen at 2 L/min flow and sevoflurane at an expired concentration of 2%. The patient was given fentanyl 30 mcg, propofol 50 mg, and vecuronium 4 mg IV.

The sevoflurane was discontinued, the ETT disconnected from the circuit, and the patient was transferred to a stretcher. The patient was connected to a pediatric ambu bag with oxygen at 8 L/min. Transport monitors were placed and the patient was manually ventilated during transfer to the PICU. The patient was placed on a ventilator in the PICU and report was given to the PICU nurse.

Discussion

The epiglottis is a small portion of tissue in the airway that helps prevent aspiration during swallowing by covering the glottis, the opening of the larynx. Epiglottitis should be considered a supraglottic disorder, as the base of the tongue, vallecula, aryepiglottic folds, arytenoids soft tissue, lingular tonsils, and epiglottis are all affected.[2] This infection can occur within hours with symptoms progressing quickly. Any suspected epiglottitis case needs to be closely monitored and personnel must be prepared for a rapid response, as complete airway obstruction can quickly develop due to involvement of multiple airway tissues.[3] The main cause of epiglottitis is *H influenza* type B, accounting for 25% of the cases. Current literature provides other causes of epiglottitis: *H parainfluenzae*, *Streptococcus pneumonae*, *Staphylococcus* aureus, and *B*-Hemolytic streptococci groups A, B, and C.[2] There is evidence that children who receive all recommended vaccinations may still be vulnerable to *H influenza* epiglottitis.[4] Epiglottitis can also occur due to noninfectious causes, such as a chemical or thermal burn, foreign object trauma, simulation by hereditary angioedema, or haemophagocytic lymphohistiocytosis.[5] The incidence of epiglottitis occurs in children between the ages of 2-7 with a mortality rate less than 1%.[2] Since the *H influenza* type B vaccine was developed the incidence of epiglottitis in children decreased from 3.47 per 100,000 to 0.63 per 100,000 from 1980 to 1990.[3] Research has shown that since the *H influenza* vaccine there has been an increase in adult epiglottitis cases.[1] Adults more often present between the ages of 20-40 with a mortality rate of 7%. The incidence of adult acute epiglottitis fluctuates from 0.97 to 3.1 per 100,000.[1] Further evidence, recorded from 1996-2005, supports a decrease in the incidence of pediatric cases and a steady incidence among adults during the *H influenza* vaccine era. During that time period, the pediatric incidence of epiglottitis was 0.2 cases per 100,000 per year. The adult incidence stayed consistently around 1.9 cases per 100,000 per year.[6]

A review of the current literature confirmed symptoms observed during this case. Patients with mild symptoms are managed differently from those with severe symptoms. Many patients present with symptoms, which do not distinctly specify epiglottitis. They can present with a sore throat, high fever, or a mild cough. This is a critical time period as symptoms can rapidly progress to signs of immediate airway obstruction. Signs that are indicative of epiglottitis are drooling, dyspnea, dysphagia, and dysphonia. The patient will also prefer the sitting position to improve respiratory effort. The patient will also have respiratory and inspiratory distress.[7] Epiglottitis mainly affects the airway, but can also cause a generalized toxemia. Patients may present with tachycardia, flushed face, high fever, and prostration. [3] Radiologic examination of the lateral neck may show the "thumb sign."[5] The literature indicates that radiologic examination is not always reliable and the only way a definitive diagnosis can be made is by direct laryngoscopy.[1] No radiologic examination was used to confirm epiglottitis in this case.

This patient had an increased risk for epiglottitis due to her medical history of myelodysplastic syndrome. This syndrome is a hematologic disorder that causes decreased white blood cell counts which increases the risk for infection. Current literature provides evidence that children vaccinated with *H* influenza type B can still acquire epiglottitis, though the incidence of this is low. Vaccinated children and non-vaccinated children present with comparable symptoms, and management depends on the patient's symptoms.[4]

The management of a patient with epiglottitis is the same for children and adults, except for maintaining the child calm. Children are afraid of hospitals and situations they do not understand. Children suspected of epiglottitis are facing both, and their anxiety is higher than normal. It is extremely important to keep a child suspected of epiglottitis calm. Anxiety and crying can exacerbate the symptoms of epiglottitis. Having the parents remain during induction can be helpful if separation is stressful for the child. Sedation is not appropriate for a child with possible airway complications.[7]

The severity of the patient's symptoms and airway obstruction depict what management the anesthesia professional will provide. In this case immediate action was taken due to the severity of the airway obstruction. The only treatment modality for epiglottitis severely occluding the airway is to place an ETT in the trachea, maintain adequate respiratory function spontaneously or mechanically, and administer IV antibiotics. Intravenous steroids were not always given, but research showed patients that received IV steroids had shorter ICU and hospital stays.[8] Racemic epinephrine remains controversial amongst researchers and medical professionals.[2] Research showed that 80% of the children with epiglottitis who received racemic epinephrine showed no improving effect.[9]

All appropriate personnel must be in the operating room to intervene and perform a tracheostomy if the airway becomes compromised.[4] All airway equipment should be immediately available in the operating room. The patient needs to be in the operating room, not the emergency room for direct laryngoscopy. Attempting this procedure without proper equipment or personnel can increase the risk of a laryngeal spasm or dynamic airway collapse, resulting in complete obstruction.[6]

For patients that present with mild symptoms of epiglottitis, immediate intubation may not be required. The patient can be monitored in the intensive care unit while receiving intravenous antibiotics.[2] For intubated patients, it is preferred for the patient to spontaneously ventilate to assess their respiratory function. Sedation may be needed to maintain calmness while the patient remains intubated, and prevent self extubation.[3] Another option noted in the literature to keep the intubated patient calm is to place a nasopharynx ETT, which is less stimulating than the oropharynx.[1]

In retrospect, allowing the patient to spontaneously ventilate and assist during induction rather than completely bag-mask ventilate for the patient. Some research suggests this technique to avoid possibly causing airway obstruction. The patient was at increased risk for obstruction due to dynamic airway closure or from a laryngospasm. Another different recommendation seen in the research was to place a nasopharynx ETT instead of an oropharynx ETT, to decrease accidental self extubation later in the ICU.[1]

Anesthesia professionals need to be knowledgeable of the symptoms and management of epiglottitis as they are responsible for airway management and emergencies. They must be attentive for patients at risk for epiglottitis and equipped to manage the airway risks associated with epiglottitis.

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Laparoscopic Appendectomy in the Patient with Prader-Willi Syndrome

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Keywords: Prader-Willi Syndrome; anesthesia; pediatric; chromosome abnormality; perioperative respiratory compromise

Patients with Prader-Willi Syndrome (PWS) can present many challenges to anesthesia practitioners. PWS has a prevalence rate of 1 per 10,000 to 1 per 30,000 people and is due to an abnormality on chromosome 15.[1] Patients with PWS presenting for surgery are considered high risk for pulmonary aspiration, difficult airway, perioperative respiratory complications due to severe hypotonia, and hypothalamic alteration.[2] This case report will discuss a patient with PWS undergoing a laparoscopic appendectomy.

Case Report

An 11-year-old, 43 kg, 142.5 cm male presented with appendicitis, which subsequently required an emergent laparoscopic appendectomy. The patient's medical history consisted of PWS and an

unremarkable surgical history. An allergy to amoxicillin was documented. The patient's only medication was a scheduled growth hormone regimen. Physical examination revealed clear bilateral breath sounds throughout all lung fields and regular cardiac rhythm and rate. Airway evaluation revealed a Mallampati Classification II with normal dentition. No laboratory data was obtained. The patient was NPO for 13 hours prior to evaluation.

Upon arrival to the operating room, standard monitors were applied including pulse oximetry, noninvasive blood pressure cuff, and 3-lead electrocardiogram. An inhalation induction was achieved with oxygen 3 L/min and nitrous oxide 7 L/min being administered for 1 minute prior to sevoflurane introduction at 8% inspired concentration. Intravenous (IV) access was obtained via the left hand with a 20 gauge IV catheter and propofol 50 mg IV was administered. Direct laryngoscopy was performed using a Miller 2 blade. A yankauer suction tip catheter was used to remove thick secretions in the oropharynx prior to the placement of a 6.0 mm cuffed endotracheal tube (ETT). The ETT was placed without complication and secured at 17 cm. Correct placement of ETT was verified via auscultation of bilateral breath sounds and an appropriate capnography waveform. An upper body Bair Hugger (Arizant Healthcare Inc., Eden Prairie, MN) was applied and patient temperature was monitored via an esophageal temperature probe.

General anesthesia was maintained with 3% sevoflurane in oxygen 3 L/min. Shortly after tracheal intubation, spontaneous ventilation returned, and the patient was placed on pressure support ventilation of 10 cm H_2O with positive end expiratory pressure (PEEP) measuring 4 cm H_2O. A pneumoperitoneum was established with carbon dioxide at a pressure of 15 mm Hg. Spontaneous ventilation with pressure support assistance was maintained throughout the surgical procedure. The surgical procedure was uneventful. At the conclusion of the surgical procedure; tidal volume was observed at 5 ml/kg, the oropharynx was suctioned with a yankauer suction tip catheter, and the ETT was removed after the patient exhibited the ability to lift his head for five seconds.

The patient was then transported to the post anesthesia care unit (PACU) with oxygen delivery via face mask at 6 L/min. Upon arrival to the PACU, the patient's vital signs were stable and SpO_2 was 100%. Oxygen support was progressively weaned in PACU, while the patient's respiratory status and effort was continuously monitored. Postoperative pain was also observed, as a conservative approach to pain control was taken. No problems were noted after a PACU stay of approximately two hours, and the patient was subsequently transferred to an observational unit overnight and then discharged home.

Discussion

PWS can present many challenges to anesthesia practitioners. Abnormalities in hypothalamic regulation lead to an insatiable appetite. This may lead to obesity, obstructive airway disease, and a Pickwickian syndrome.[2] PWS patients presenting for surgery should be considered a full stomach with a risk of pulmonary aspiration. These children often sneak meals and may need strict supervision with food. In this case the patient's body mass index was 20.5 kg/m^2, so parental vigilance and patient cooperation was evident. The decision was made to proceed with an inhalation induction versus a rapid sequence

induction because the parents were able to ensure that the patient was appropriately NPO and food control was obvious in this case. If the patient had presented at a heavier weight where food monitoring was not as evident, a rapid sequence induction may have been the prudent choice. The hypothalamic alterations of PWS patients make temperature control particularly important to anesthesia practitioners. The patient's body temperature was carefully maintained between 37.0 and 37.5 degrees Celsius via the use of a Bair Hugger (Arizant Healthcare Inc., Eden Prairie, MN).

Airway difficulties may be present in the patient with PWS. The presence of a narrow, high arched palate coupled with morbid obesity can make laryngoscopy difficult. Thick saliva is common in the patient with PWS and may lead to the formation of dental caries and poor dentition.[3] Parents may need education regarding proper oral hygiene and the importance of regular dental health visits. In the case described, the patient was found to have tenacious secretions in the oropharynx, despite his good oral health. As with any type of anesthetic, the availability of suction is of utmost importance.

A special concern to anesthesia practitioners is the hypotonic development frequently seen in patients with PWS. This can present respiratory difficulties perioperatively. These patients are at a high risk for the development of atelectasis and postoperative respiratory arrest due to poor respiratory effort and hypotonia often complicated by anesthesia. In the case described, no preoperative sedation was administered and anesthetic medications were given with caution. Although there have been cases of patients with PWS undergoing general anesthesia and receiving neuromuscular blockade, the decision was made not to administer any muscle relaxants because of the hypotonia present.[4] The surgical procedure was not expected to be lengthy, so spontaneous ventilation was maintained throughout and the volatile agent was sufficient for muscle relaxation.

Laparoscopic procedures require a pneumoperitoneum to enhance the visualization of anatomic structures. Insufflation of the abdomen displaces the diaphragm cephalad secondary to increased intra-abdominal pressure. In doing so, the functional residual capacity is greatly reduced and can lead to the development of atelectasis intraoperatively.[5] In the case described, the patient was spontaneously breathing with mechanical pressure support and PEEP. Applying PEEP intraoperatively can decrease atelectasis while improving oxygenation.[5] Ventilation of the lungs with PEEP during pneumoperitoneum has been shown to improve the arterial partial pressure of oxygen and can thus result in better gas exchange during laparoscopy.[6] During the intraoperative phase, the patient did not exhibit a decrease in SpO_2. The augmented spontaneous tidal volumes consistently averaged 5-6 ml/kg and end tidal CO_2 levels were maintained between 37-44 mm Hg. After completion of the procedure, appropriate extubation criteria were met and the patient had an uneventful postoperative outcome.

Prior case studies have focused on patients with PWS undergoing general anesthesia without the use of narcotics or muscle relaxants. It was found that patients with a normal body weight did not suffer any problems perioperatively, while those who were obese experienced respiratory compromise.[7] In the case described, the patient was not overweight which may have been the reason for an uneventful perioperative course. Close monitored

observation, with particular attention to the patient's respiratory status, is suggested in patients with PWS.

The systemic problems associated with PWS are of great concern to the anesthesia practitioner. Respiratory compromise can be regarded as the most significant complication; however, with conservative anesthetic delivery and respiratory support, positive anesthesia outcomes can certainly be achieved in a patient with PWS. This case report demonstrated that with the cautious use of drug administration and recognition of possible adverse outcomes, safe anesthetics can be delivered despite the presence of multiple health risk factors in this patient population.

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Minimally Invasive Hemodynamic Monitoring

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Keywords: Minimally, Invasive, Hemodynamic, Monitoring, CABG, Flotrac, Arterial, Coronary, ESRD, Jehovah's Witness.

Accurate hemodynamic monitoring is essential in the anesthetic management of patients undergoing coronary surgery. Perioperative hemodynamic optimization has been shown to improve patient outcomes by reducing morbidity and the length of stay on the intensive care unit.[1] Technically difficult invasive techniques such as right heart catherization and femoral/radial access have been the gold standard for cardiac optimization with hemodynamic monitoring in the coronary surgery patient. In this context, less invasive techniques that utilize pulse contour analysis such as the FloTrac/Vigileo system (Edwards Lifesciences LLC, Irvine, CA, USA) may be

implemented when central access cannot be obtained.

Case Report

A 55-year-old, 65 kg, 167 cm female presented for a coronary artery bypass graft (CABG) of two vessels. The patient had an extensive history including chronic end stage renal disease (ESRD), peripheral vascular disease (PVD), hypertension (HTN), hypothyroid, coronary artery disease (CAD) with stents, Type I diabetes mellitus (DM), neuropathy of hands and feet, anemia, gastroesophageal reflux (GERD). She was a Jehovah's Witness; therefore precautions were taken for expected blood loss, risk/benefits discussed, and a plan developed for volume resuscitation. The patient agreed to accept hetastarch expander and auto cell salvage as part of the resuscitation plan.

The patient was preoxygenated with oxygen at 8 L/min. After a smooth rapid sequence induction and a return of train of four response to peripheral nerve stimulation, vecuronium 7 mg was administered. At this time right radial arterial catheterization with 20 gauge catheter using ultrasound guidance was attempted. After two unsuccessful attempts left femoral artery access was obtained on the first attempt.

The patient was prepped and draped using strict sterile technique for right internal jugular (RIJ) central line access. After two failed attempts to thread the J wire into the RIJ, the left internal jugular was attempted twice unsuccessfully. An 8 fr triple lumen to the femoral vein on first attempt. Vital signs remained stable throughout catherization attempts.

The cardiac surgeon was made aware of the inability to obtain jugular and pulmonary artery access and it was decided to apply the Flotrac/Vigileo monitoring system to the left femoral arterial transducer. The Flotrac was calibrated and stroke volume variance (SVV), cardiac output (CO), and stroke volume (SV) were monitored throughout the case. Cardiac output remained stable ranging from 4.0-5.1 L/min.

Upon cessation of cardiopulmonary bypass a dobutamine infusion was intiated at 5 mcg/kg/min. In addition, a norepinephrine infusion was titrated to maintain systolic blood pressure above 110 mmHg. The patient was transported to the intensive care unit (ICU) with the endotracheal tube in place. Blood loss was an estimated 750 mL, and total of 1000 mL of hetastarch and 2200 mL of crystalloid were administered. Blood salvage volume was 150 mL. Stroke volume variance was utilized via the Flotrac and fluid responsiveness was attributed to a 5-10% increase in SVV. Crystalloid and colloid therapies were therefore considered optimized if SVV was greater than 5% but less than 15%. A 24 hour follow up revealed a stable patient with a hematocrit of 23.9% and absence of ventilator or vasopressor support.

Discussion

Estimation of hemodynamic variables such as CO, SV, and SVV in patients with ESRD undergoing coronary surgery is essential for performing goal directed therapy and reducing overall morbidity and mortality. These results are usually obtained via transesophageal echo (TEE) and pulmonary artery catherization (PAC). Auler Jr. et al. state that in most patients in the surgical setting, thermodilution using a pulmonary artery catheter is still the most frequently applied technique for measuring CO and has generally been accepted as the clinical gold standard.[3] End stage renal disease causes

many unique physiological anomalies that result in a higher risk of perioperative death. As a result, these patients often have associated disorders which predispose them to increased operative morbidity and mortality, such as inability to excrete certain medications, platelet dysfunction and susceptibility to infection.[2]

A study conducted by Gharsallah et al. reveals that patients who have ESRD have accelerated atherosclerosis and calcification of cardiac structures including coronary arteries, valves and conduction tissue.[2] Atherosclerosis and calcification is likely due to increased calcium phosphate secondary to hyperparathyroidism.[2] Vascular and arterial access is therefore more difficult to achieve in these patients. As a result of these pathophysiological changes, the anesthesia plan should take into account for instances in which central venous access and a PAC may be more difficult or of higher risk to the patient.

The FloTrac/Vigileo system allows for minimally invasive CO, SVV, and SV determination based on the arterial pressure waveform derived from any standard arterial catheter. The algorithm underlying CO calculation was recently modified to allow a more precise estimate of aortic compliance.[3] The device does not require external calibration. The system obtains the pressure wave signal from any standard peripheral arterial line. Standard deviation of the pulse pressure is empirically correlated with the stroke volume based on patient demographic characteristics (age, gender, body height, and weight) after automatic adjustment for actual vascular compliance.[3]

Although the Flotrac/Vigileo system is not a definitive alternative to the standard PAC, some studies suggest it has severe limitations in the coronary surgery setting. Compton et al. concluded that its limited accuracy might be acceptable, though, in situations where invasive CO monitoring is not an option and tracking of individual CO changes is more important than absolute CO values such as in the case where central access cannot be obtained.[4] Fluid administration in the ESRD patient undergoing coronary bypass requires meticulous monitoring and precise administrative requirements. Without central venous access, fluid shifts and hypovolemia are more difficult to manage. As a result, fluid overload can become a severe perioperative complication. The Flotrac/Vigileo system provides an alternative solution to this potentially dangerous situation.

SVV has repeatedly been shown to be a reliable predictor of fluid responsiveness.[5] Although there is only sparse data available with the recently introduced Flotrac/Vigileo system; modification to the software now allows for one minute adjustments in the vascular window which results in improved overall measurements. SVV is calculated from percentage changes in SV during the ventilatory cycle. The Flotrac/Vigileo uses proprietary algorithms that utilize the contribution of pulse pressure to SV being proportional to the standard deviation of arterial pulse pressure.[5] The SV is determined by the influences of vascular resistance and compliance. These values are interpreted based off the manually entered patient data previously discussed and pulse wave analysis.

In conclusion, ESRD patients undergoing a CABG have co-morbidities making them high risk patients. Eilers et al. state that the presence of ESRD is associated with increased perioperative mortality, even when adjusted for other variables, such as hypertension or diabetes.[6]. Chronic hypertension and renal disease, diabetes, or

high protein intake all disturb autoregulatory mechanisms.[6] Therefore, the disease process provides significant challenges both pathophysilogically and hemodynamically. The anesthetic plan should include alternatives to compensate for the complexities of ESRD as central venous and pulmonary artery access can be more challenging and carry an increased risk of complications. In this instance, a minimally invasive arterial monitoring system provided an alternative approach to hemodynamic management by utilizing the standard arterial monitoring system normally used with the operative procedure. While the data remains controversial in terms of the accuracy of measurement pertaining to CO, SVV, and SV with this device, this tool can be used as a substitute to determine fluid optimization and the mechanical efficiency of the heart.

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Pseudocholinesterase Activity in Patients with Human Immunodeficiency Virus

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Keywords: pseudocholinesterase deficiency, succinylcholine, HIV, liver disease, hepatotoxicity

Pseudocholinesterase, butyrylcholinesterase, plasma cholinesterase, and serum cholinesterase is an enzyme synthesized in the liver. In plasma, it is responsible for the metabolism of succinylcholine and ester local anesthetics.[1] Patients with a deficiency or alteration of pseudocholinesterase have a decreased metabolism of succinylcholine manifested by prolonged paralysis and apnea following administration of the drug.[2]

Since the enzyme is produced in the liver, hepatocellular dysfunction of any cause can lead to significant deficiency.[3] This case report will examine a case of pseudocholinesterase deficiency in a patient with hepatic impairment due to human immunodeficiency virus (HIV).

Case Report

A 50-year-old, 80 kg male presented with cholelithiasis and was admitted for laparoscopic cholecystectomy. The patient reported a past medical history of gastroesophageal reflux, hypertension, and HIV. Current medications included efavirenz/emtricitabine/tenofovir, dapsone, hydrocodone/acetaminophen, amlodipine, esomeprazole, morphine sulfate, and ondansetron. No previous anesthetic complications were reported. Drug allergies included sulfa drugs. Physical examination revealed regular cardiac rate and rhythm and clear breath sounds bilaterally. Pre-operative vital signs were stable, and no laboratory tests were ordered. Abdominal ultrasound revealed the following results: gallbladder adenomyomatosis, hepatic cysts, hepatic calcifications, hepatic fatty infiltration, and mild renal disease. The patient's last oral intake was 6 hours prior to assessment.

In the operating room, monitors were applied, and pre-oxygenation was administered. Considering NPO status and history of reflux, a standard rapid sequence induction was performed using the following drugs: midazolam 2 mg IV, fentanyl 100 mcg IV, lidocaine 80 mg IV, rocuronium 5 mg IV, propofol 150 mg IV, and succinylcholine 100 mg IV. Laryngoscopy was performed, and the patient's trachea was intubated with a 7.5 endotracheal tube without complication. Sevoflurane was used for maintenance of general anesthesia.

Prior to surgical incision, neuromuscular blockade was checked with a peripheral nerve stimulator 25 minutes after induction. No train of four (TOF) twitches or tetany was noted at that time. The first TOF twitch was noted 80 min after succinylcholine administration. Near the end of the procedure, 4 twitches and tetany were elicited. Spontaneous respirations resumed with a respiratory rate of 15-20 breaths/min and tidal volumes of 300-400 mL. The possibility of a pseudocholinesterase deficiency was overlooked at this time, due to the patient's report of no previous anesthetic complications. Instead, it was assumed that the patient's hepatic impairment had possibly caused a delayed metabolism of rocuronium, despite the minimal dose given. Therefore, a full antagonizing dose of neostigmine 5mg and glycopyrrolate 0.8 mg was administered. Neuromuscular blockade was not re-tested at this point. The trachea was then extubated after the patient opened his eyes to command. Upon extubation ventilatory drive became significantly decreased with tidal volumes of 50 mL, and TOF monitoring elicited no twitches. After 60 minutes of assisted ventilation, the patient's trachea was re-intubated. Transportation was provided to the intensive care unit where ventilatory support was required overnight. A serum cholinesterase level was measured and found to be 3.2 U/mL (reference range 8-18 U/mL). It was subsequently determined that the patient had pseudocholinesterase deficiency.

Discussion

Pseudocholinesterase activity can be decreased by many physiologic, pharmacologic, and pathologic factors. Inherited forms of pseudocholinesterase deficiency follow many genotypes and include homozygous and heterozygous

variations.[3] Causes of acquired decreases in pseudocholinesterase activity include: liver disease, renal disease, malnutrition, pregnancy, malignancy, and burns. Many drugs have been linked to the disorder including: anticholinesterases, pancuronium, oral contraceptives, and monoamine oxidase inhibitors.[2] Variations of the disorder account for differences in the decreased activity of the enzyme. When a >75% decrease in levels of normal pseudocholinesterase exists, clinical manifestations of prolongation of succinylcholine emerge.[3] Whether acquired or inherited, understanding the disorder is essential for those administering succinycholine. In order to provide safe care, anesthesia practitioners must appreciate the genetic factors, diseases, and medications that can contribute to this disorder.[2] Failure to recognize pseudocholinesterase deficiency can lead to insufficient respiratory function, delayed awakening, and prolonged hospitalization for the patient.

Of particular importance in this case is the patient's past medical history and current medications. Liver disease has become a main cause of morbidity and mortality in HIV patients on antiretroviral therapy (ART). HIV infection itself, immunodeficiency, and long-term ART are associated with the development of liver disease in this population. In a recent study, HIV-monoinfected patients with a high HIV-1 load were found to have chronic elevated alanine amino-transferease (ALT) and steatosis. In addition, these persons were also at risk for an aspirate aminotransferase (AST)-to-platelet ratio index greater than 1.5, indicating significant hepatic fibrosis. It is thought that the effects of HIV on hepatocytes are caused by immune-activating and pro-apoptotic effects. HIV has been found to enter and replicate in hepatic stellate cells, promoting hepatic fibrosis. Immunodeficiency among these patients has been found to be associated with increased liver injury; however, the mechanism of action is unknown.

A recent study also found that prolonged exposure to ART was associated with increased liver fibrosis. It has been proposed that nucleoside reverse transcriptase inhibitors (NRTI) lead to chronic ART-related hepatotoxicity by causing lactic acidosis and steatosis. Immune-mediated hypersensitivity reactions to these drugs are thought to contribute to their toxicity, as well.[4] Due to its synthesis in the liver, pseudocholinesterase production is impaired when liver function is weakened. Pseudocholinesterase activity may decrease by 30%-50% in acute hepatitis and by 50% in cirrhosis and chronic malignancies.[2] The patient detailed in this case report was currently taking efavirenz/emtricitabine/tenofovir, an ART consisting of two NRTIs and one non-mucleoside reverse transcriptase inhibitors (NNRT) with a black box warning for hepatotoxicity. The patient's abdominal ultrasound showed evidence of hepatic impairment including: hepatic cysts, hepatic calcifications, and hepatic fatty infiltration. Mild renal disease was also noted in the study. Like liver disease, renal impairment has been linked to decreased pseudocholinesterase levels. However, the cause of this association is unknown.[2] Further laboratory studies, including a chemistry panel and liver function enzymes, would have been useful values for pre-operative evaluation.

Another factor that must be considered in this case is the administration of neostigmine. The prolonged duration of this patient's neuromuscular block until the next morning indicates that factors other than

decreased pseudocholinesterase activity are responsible. Researchers feel that the effect of neostigmine on succinylcholine neuromuscular blockade depends on the balance between depolarizing block and phase II block. If the depolarizing block predominates, neostigmine has been found to inhibit pseudocholinesterase, prolong the presence of succinylcholine, and potentiate the block.[5] Therefore the administration of neostigmine in the presented case may have caused a phase II block and prolonged the duration of succinycholine further.

In summary, one must consider hepatic impairment when providing anesthesia for a patient with HIV. Further consideration should be given to the possibility of decreased pseudocholinesterase synthesis. When administering neostigmine after neuromuscular function has returned it may contribute to further blockade and prolongation of succinylcholine. Laboratory studies, including liver function tests, prior to administration of anesthesia may assist in determining presence and extent of hepatic impairment. If pseudocholinesterase deficiency is suspected, a dibucaine number may guide in choice of neuromuscular blocking agents. Detection of pseudocholinesterase deficiency will also guide administration of neostigmine and assist in avoiding prolonged neuromuscular blockade.

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Optimal Fluid Resuscitation for Operative Burn Patients

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Keywords: Burn injury, fluid resuscitation, blood transfusion, total body surface area, skin graft

Calculating the initial intra-operative fluid requirements for burn patients is dependent upon time elapsed since the burn injury along with the total body surface area burned. However, anesthesia professionals should anticipate the need for active fluid and blood resuscitation during the intra-operative period as these patients often have decreased albumin levels and increased capillary permeability which may lead to

hypovolemia.[1] This case report focuses on the challenges of fluid management for burn-injured patients.

Case Report

A 67-year-old, 80 kg male presented for allograft transplantation to the face, neck, scalp, chest, back, bilateral upper extremities and right lower extremity, with possible wound vacuum placement, four days after sustaining burns to 39% of his body surface area. The patient had no significant medical or surgical history. Reported significant preoperative vital signs included heart rate 99 beats per minute, blood pressure 154/69 mmHg, and SpO_2 95% on 40% oxygen administered via high-flow nasal cannula. Significant preoperative labs included sodium of 153 mEq/L, potassium of 3.3 mmol/L, fibrinogen >1000 mg/dL and albumin of 1.2 g/dL.

The patient had a previously placed triple lumen femoral venous catheter for intravenous access which was used during the surgical procedure. The patient also had a femoral arterial line. The patient was given intravenous doses of ketamine 40mg, lidocaine 80mg, and propofol 160mg to induce general anesthesia. Ability to successfully mask ventilate the patient was verified before administering rocuronium 50mg. Utilizing a Miller 2 blade for direct laryngoscopy the anesthesia practitioner obtained a grade two view, and the trachea was intubated with a 7.5 mm oral endotracheal tube (ETT). The ETT was sutured at 22cm at the gums by the surgeon. The patient was then moved to the operating room table and placed in a prone view device. A five lead electrocardiogram was applied, along with a pulse oximeter. A bladder temperature probe from the Foley catheter was utilized for assessment of core body temperature. Ambient room temperature was increased to assist with thermoregulation.

General anesthesia was maintained with 0.6% isoflurane and intravenous boluses of fentanyl 100 mcg and vecuronium 1mg as needed to keep train of four at one to two twitches. During grafting to the posterior side of the body, the patient remained in the prone position for 4.5 hours. The patient was then placed in supine position for grafting of the anterior chest, neck, and face. The operation lasted for more than eight hours and the patient's hemodynamics were monitored closely throughout the case utilizing blood pressure readings from a femoral arterial line. The estimated blood loss was greater than 850mL and the patient received 12 units of blood components, 7500 mL crystalloid, and 1750 mL colloid for fluid replacement. The patient remained sedated following the case and was transported back to the burn intensive care unit with an ETT in place. He was placed on mechanical ventilation upon return to the intensive care unit.

Discussion

Providing adequate anesthesia and managing hemodynamic fluctuations are essential for the anesthesia professional in all cases but prove especially challenging when caring for burn injured patients undergoing skin grafting procedures. When determining fluid requirements for this population, the anesthesia practitioner must be mindful of multiple variables. Time elapsed since burn injury, the patient's current hemodynamic stability, amount of fluid resuscitation provided in the intensive care setting, length of surgical procedure, anticipated blood loss and fluid shifts, and the impact of anesthetic gases on hemodynamics are all essential considerations. Post operative disposition and the need for mechanical ventilation must also be considered. In this report, variables

such as prone positioning and extended length of surgery further complicated the choice of fluid and extent of fluid resuscitation.

Various formulas outline early fluid resuscitation strategies for burn patients. These include the well-known Parkland formula, which was developed by Charles Baxter, MD in the 1960s.[2] This formula gives guidelines for volume resuscitation using LR solution at 4ml/kg body weight per percentage burn and recommends administering half this volume in the first eight hours post burn and administering the remaining volume over 16 hours.[2] Burn patients bring unique challenges to anesthesia professionals as thermal injuries can lead to irritated airways, significant inflammatory responses, cardiac instability, and significant electrolyte abnormalities. These concerns often present in stages of the burn's evolution.[3]

Current studies are debating the choice of crystalloid versus colloid resuscitation in peri-operative care. Studies have linked peri-operative fluid handling to anesthesia concerns including post-operative nausea and vomiting, pain, tissue oxygenation, cardiopulmonary disorders, need for revision of surgery, and duration of hospital stay. However, these concerns are dependent upon the type and extent of the surgery.[4] Recognizing the potential dangers from crystalloid overload, some research now recommends optimizing rather than maximizing fluid administration.[4] The complications of edema formation and coagulation abnormalities could be of particular concern to patients with burn injury undergoing skin graft procedures. In fact, research has supported that burn patients who receive the largest volumes of fluid resuscitation are actually at greater risk for complications and death secondary to their injuries.[5]

Other researchers have studied the unique effects of resuscitation techniques on burn patients, including the potentially negative effect of fluids on inflammatory response and capillary leak in burn patients. Current research indicates that over-resuscitation is not without consequence; rather, it can lead to conversion of superficial into deep burns and even the development of compartment syndrome.[4] The need for appropriate fluid resuscitation and the unique risks associated with fluid overload lead to a challenging predicament for anesthesia professionals when making decisions for fluid administration to burn patients during the intraoperative period.

To better guide us in this dilemma, researchers suggest utilizing patient markers for an individualized treatment approach. A study by Foldi et al. analyzed the effects of two resuscitation methods on inflammatory response as measured by cytokine production and the expression of the leukocyte surface markers on patients with burn injury.[6] Either hourly urine output or intrathoracic blood volume index (ITBVI) was used to guide fluid resuscitation. Intrathoracic blood volume index was measured utilizing a specialized arterial catheter and central venous pressure to calculate this transcardiopulmonary hemodynamic measurement. Researchers also found that ITBVI guided resuscitation suppressed the shift toward anti-inflammatory imbalance as indicated by blood levels of leukocyte blood marker expressions up to five days postoperatively.[6]

It is well known that surgical excision of burn wounds can lead to significant intra-operative bleeding requiring transfusion of blood components and coagulation factors.

These transfusions come with risks, and researchers have studied their impact on critically ill patients, linking use of the products to disadvantages such as increased infectious complications, multiple organ failure, and respiratory distress syndrome.[7] Schaden et al. studied the effects of timely correction of coagulopathies in bleeding burn patients utilizing a specific treatment algorithm. In the study, one patient group received transfusions based on the anesthesia practitioner's discretion, expertise and impression of bleeding in the surgical field. The other treatment group received transfusions based upon a standardized algorithm using point-of-care coagulopathy values and recommendations of a task force for perioperative coagulation. Results showed a significantly reduced use of allogenic blood products in the patient group being transfused based on the coagulation algorithm.[7]

This patient received a total of 6 L plasmalyte and 1500 LR of crystalloid, 1750 mL of 5% albumin of colloid, 5 units of packed red blood cells, 6 units of fresh frozen plasma, and 1 unit of platelets. The anesthesia professionals utilized hemodynamic markers of systolic blood pressure and urine output for indicators of the need for fluid resuscitation. Arterial blood gas measurements with hemoglobin and hematocrit values along with practitioner estimation of active bleeding on the surgical field guided the decision to administer blood products. During this case, use of a specific coagulation algorithm may have been beneficial had the professionals been adequately trained in its use, as would utilizing central venous pressure measurements. More research aimed at the specific challenges of managing fluid status during anesthesia care for burn patients along with focused training on treatment algorithms could improve our care for this population.

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Anesthesia for the Patient with Rheumatoid Arthritis

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Keywords: rheumatoid arthritis, autoimmune, systemic disease, atlantoaxial subluxation, methotrexate

Rheumatoid arthritis (RA) is a chronic autoimmune inflammatory disorder, which targets the synovium-lined joints and presents as painful symmetric polyarthropathy.[1-4] The etiology of RA remains unknown. The disorder affects nearly 69 million persons worldwide and is significantly correlated with increased rates of morbidity and mortality compared with the general population.[1,5,6] Rheumatoid arthritis patients frequently require anesthesia for orthopedic surgery. These patients are of particular concern due to systemic involvement of the disease, most notably cardiovascular and airway changes.[1,4,6] The side effects of the drugs routinely used in the treatment of RA also impact the perioperative management of rheumatoid arthritis patients.

Case Report

A 78-year old, 170 cm, 66 kg female presented for a left total elbow revision. Her past medical history was significant for RA, uncontrolled hypertension, diabetes mellitus type II, renal insufficiency, and chronic anemia. Prior surgical history included a left total knee arthroplasty and left total elbow arthroplasty, with no prior anesthesia complications. Current medications included furosemide, amlodipine, carvedilol, hydralazine, methotrexate, adalimumab, acetaminophen/codeine, sertraline, zolpidem, folic acid, and novolog. The patient reported taking an unknown dose of prednisone for an unknown duration of time prior to surgery.

The patient was edentulous and demonstrated good mouth opening with a Mallampati classification I airway. She exhibited full occipitoatlantoaxial mobility devoid of neurologic symptoms. The patient reported pain in her left elbow as 6 out of 10 on the numeric pain scale.

The patient's blood pressure was 166/82 mm Hg with a heart rate of 67 beats per minute (bpm). She took carvedilol 25 mg by mouth the morning of surgery. A recent echocardiogram revealed an ejection fraction of 45% and a cardiac stress test proved to be within normal limits. An electrocardiogram (ECG) displayed normal sinus rhythm with left ventricular hypertrophy. Significant laboratory values included: hemoglobin of 9.9 g/dl, hematocrit of 28.9%, and creatinine of 1.09 mg/dL. The patient was designated as an American Society of Anesthesiologist's Physical Status Classification of 3.

In the preoperative area, midazolam 2 mg and fentanyl 50 mcg were administered prior to placement of a left single-shot infraclavicular nerve block. Ropivicaine 0.5% 20 mL was injected. The patient received hydrocortisone 50 mg and was transported to the operating room. The patient was positioned while awake and range of motion of the left shoulder joint was assessed in anticipation of intraoperative surgical positioning. Preoxygenation per facemask with FiO_2 1.0 preceded an IV induction of fentanyl 100 mcg, lidocaine 80 mg, and propofol 120 mg.

A defasciculating dose of rocuronium was given and successful mask ventilation was confirmed before administering succinylcholine 100 mg. A GlideScope (Verathon Inc., Bothwell, WA) was used for direct visual laryngoscopy (DVL) and a 7.0 endotracheal tube (ETT) was inserted without difficulty. Anesthesia was maintained with sevoflurane 1.4% inspired concentration in a mixture of O_2 0.4 L/min and air 0.4 L/min. Vecuronium was administered for neuromuscular blockade. In addition to the peripheral nerve block, pain control was achieved with hydromorphone 1 mg, administered in divided doses. During the final steps of surgical incision closure, neuromuscular blockade was reversed and zofran 4 mg was given. The patient's heart rate increased to over 80 bpm during emergence from anesthesia and esmolol 10 mg was administered twice. The trachea was extubated and O_2 6 L/min was administered by facemask. Metoprolol 3 mg was given for heart rate and blood pressure control and the patient was taken to the post anesthesia care unit.

Discussion

Rheumatoid arthritis targets the cervical spine in nearly half of RA patients, presenting a risk of subluxation and spinal cord injury during airway manipulation. The atlantoaxial joint is most commonly involved and anterior cervical flexion accounts for 80% of subluxation incidents.[1,4,7] Symptoms of atlantoaxial involvement may include neck pain, parasthesias, and numbness; however, most patients are asymptomatic. Silent disease makes detection difficult and there is no consensus for obtaining imaging in asymptomatic RA patients.[1,4,5,7] Disagreement exists on the diagnostic value of imaging and its influence on anesthetic management.[1] The presented patient was asymptomatic and imaging was not performed. With the exception of awake fiberoptic-assisted tracheal intubation, there are currently no evidence-based guidelines for minimizing cervical flexion during DVL.[1,2,4] Samanta et al recommend manual inline stabilization for all RA patients as a best practice technique.[1] Anesthesia practitioners should employ this strategy in their management of RA patients. For the presented patient, a GlideScope (Verathon Inc., Bothwell, WA) was used to minimize cervical flexion. The patient did not experience neurologic symptoms prior to discharge from the hospital 3 days post-operation. Further research evaluating the efficacy of the GlideScope (Verathon Inc., Bothwell, WA) in maintaining cervical spine stability may benefit RA patients.

Limited occipitoatlantoaxial extension, decreased tempomandibular joint mobility, and cricoarytenitis are known manifestations of the RA process.[1,2,4] There should be high suspicion for a difficult airway in patients with rheumatoid arthritis, necessitating careful airway assessment.[1,2,4,5] No obvious predictors of difficult airway were present in this patient, yet succinylcholine was chosen for neuromuscular blockade during the induction of anesthesia to maximize safety. A 7.0 ETT was used in the case that glottic narrowing was encountered.

Rheumatoid arthritis commonly infiltrates the cardiovascular (CV) system. CV involvement accounts for half of all deaths and profoundly contributes to increased morbidity in RA patients.[1,6] Risk factors and symptoms of RA-associated cardiovascular disease markedly differ from those typical of the general population; for this reason, CV involvement often goes undetected.[6] Samanta et al explain "unlike heart failure in non-RA patients, there is often no history of preceding ischaemic heart disease.

…[S]tudies have shown that the ejection fraction is often normal in RA patients with failure, but there is left ventricular hypertrophy and diastolic dysfunction".[1] The echocardiogram and ECG of this patient mimicked these findings. Volatile agents were judiciously titrated and mean arterial pressure (MAP) was maintained within 20% of the patient's baseline MAP. During emergence from anesthesia, increases in heart rate were promptly treated with beta-antagonists to prevent exacerbation of underlying coronary artery disease.

Rheumatoid arthritis patients frequently present with pain and limited joint movement. Extra care is required in padding pressure points and with positioning.[1,2,4,7] Opioids and non-steroidal anti-inflammatory drugs are commonly used for symptom management in RA and a perioperative analgesic plan is imperative.[1,4] Regional anesthesia provides many benefits to RA patients, but can be met with anatomical challenges due to joint deformities.[1,4,5] In the presented patient, an infraclavicular nerve block proved to be an effective pain management strategy coupled with intraoperative IV opioids.

Disease-modifying pharmacotherapy for RA patients typically includes: biologic therapy agents, methotrexate, and corticosteroids. These medications pose risks in the perioperative period, most notably the risk of profound immunosuppression.[2,3,5,7,8] Biologic therapy agents are fairly novel medications and controversy exists over perioperative continuation; it is currently advised to discontinue therapy 1 to 4 weeks prior to surgery.[4,7,8] Adalimumab was held seven days in advance for this patient. Methotrexate therapy is complicated by toxic side effects such as interstitial lung disease, hepatotoxicity, and anemia.[1,4] Practice guidelines advise continuation of methotrexate perioperatively; however, selected literature discusses the benefit of its discontinuation in the elderly, diabetic patients, and individuals with renal impairment.[7,8] The presented patient met the criteria of the latter category, yet methotrexate was continued. Normocytic anemia, as a result of chronic disease and pharmacotherapy, puts RA patients at a higher risk of requiring intraoperative tranfusions.[1,2,7] The patient's laboratory values revealed an anemia and a blood type and screen was performed the morning of surgery.

Supplemental doses of corticosteroids are administered perioperatively to prevent hypotension, disease exacerbation, and adrenocortical crisis.[3,8] Goodman and Paget compared a group of corticosteroid-dependent RA patients with a corticosteroid-naïve RA population and found no difference in intraoperative blood pressure.[8] The authors recommend avoiding supraphysiologic corticosteroid dosing, unless hypotension is encountered; however, practice guidelines maintain that glucocorticoid continuation and supplementation is vital to maintaining perioperative hemodynamic stability.[3,7] The extent of the discussed patient's preoperative corticosteroid therapy was unclear, yet the benefit of supplementation was believed to outweigh the risks; she received hydrocortisone 50 mg preoperatively. No intraoperative hypotension was encountered.

The anesthetic course for the presented patient was uncomplicated and supported by current literature. To safely care for rheumatoid arthritis patients, anesthesia professionals must be knowledgeable about the systemic involvement of RA and properly optimize these patients in the perioperative period.

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Total Intravenous Anesthesia for Patient at Risk for Postoperative Nausea and Vomiting

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Keywords: postoperative nausea and vomiting, total intravenous anesthesia, propofol ketamine infusion, anesthesia for partial mastectomy

The most common adverse effect of anesthesia in the postoperative period is nausea and vomiting.[1] Postoperative nausea and vomiting (PONV) occurs in up to 70% of patients considered high risk, and 30% of

those not considered high risk.[2] The use of volatile anesthetics for breast surgery is among the highest prevalence of PONV with 60-80% of patients experiencing the adverse effect.[1] Though PONV is often non-life-threatening and short-term, it can lead to significant morbidity such as dehydration, electrolyte imbalance, suture dehiscence, and aspiration. Also, from the patients' perspective, the avoidance of PONV can be more desirable than pain control.[3]

Case Report

A 50-year-old, 163 cm, 68 kg female diagnosed with breast cancer presented for left partial mastectomy with sentinel lymph node biopsy. The procedure would also include a 25-minute intraoperative radiation treatment. Her past medical history was significant for breast cancer, rosacea and motion sickness, negative for cigarette smoking. Her breast cancer was diagnosed after routine mammogram and ultrasound guided breast biopsy. A peripheral intravenous (IV) line was started in her right hand and midazolam 2mg IV was administered as premedication. In the operating room (OR), monitors including a pulse oximeter, electrocardiograph, noninvasive blood pressure and cutaneous temperature probe were applied, as she was positioned supine. Oxygen via nasal cannula was administered at 2L/min. Induction medications included lidocaine 20 mg IV bolus and propofol 70mg IV bolus. A continuous infusion containing a 43mL mixture of propofol 400 mg (10 mg/mL), ketamine 50 mg (50 mg/mL) and fentanyl 100 mcg (50 mcg/mL) was started at a rate of 0.5 mL/min to maintain deep sedation (final concentration of 43 mL mixture was approximately: propofol 9 mg/mL, ketamine 1.2 mg/mL and fentanyl 2 mcg/mL). A total of 40 mL 0.25% bupivacaine was administered locally by the surgeon to produce anesthesia to the breast tissue. Dexamethasone 4mg IV was administered after induction for prevention of PONV.

The patient maintained spontaneous ventilation and hemodynamic stability throughout the procedure only requiring small adjustments in the rate of propofol/ketamine/fentanyl infusion. The propofol/ketamine/fentanyl infusion was stopped after approximately 90minutes once propofol 400 mg, ketamine 50 mg and fentanyl 100 mcg IV had been infused (the contents of the original 43 mL mixture). For the remainder of the procedure, sedation was maintained with a propofol infusion of 50mcg/kg/min until the initiation of incision closure. During the 25-minute intraoperative radiation treatment, occurring approximately 105 min into the procedure, the patient required no anesthetic intervention. The patient received 1400 mL of Lactated Ringers throughout the procedure and estimated blood loss was 10 mL. Upon surgical closure, the propofol infusion was discontinued and ondansetron 4 mg IV was administered. In total the patient received propofol 826 mg, ketamine 50 mg and fentanyl 100 mcg IV throughout the entire 180-minute case. Once the procedure was complete the patient was transferred to the recovery room where she remained for 60 min without requiring opioid for pain or antiemetic for PONV.

Discussion

Defined as nausea and/or vomiting within 24hours after surgery, PONV can take place in a single occurrence or multiple episodes lasting minutes, hours or days.[2] PONV is multifactorial in etiology with factors such as patient characteristics, anesthetic technique and surgical procedure all contributing.[3] The pathophysiology of nausea and vomiting is complex and poorly

understood; numerous neurotransmitters are involved, which supports the finding that monotherapy in high risk patients will not suffice.[2] However, recognition of risk factors has dramatically decreased the incidence of PONV and is essential to its prevention.[2]

Well-established risk factors for PONV can be divided into patient, surgical and anesthesia related factors. Individual patient factors that increase the probability of PONV are female gender, non-smoking status, history of PONV or motion sickness, and childhood or young adulthood.[3] Female, non-smoking patients, with a history of motion sickness are among the highest risk patients to suffer from PONV. Anesthesia-related risk factors include the use of volatile anesthetics, large doses of perioperative opioids, nitrous oxide and large doses of neostigmine.[3] All of these anesthetic techniques may significantly contribute to the occurrence of PONV, particularly in high risk patients. Type of surgery, including gynecological, otolaryngological and laparoscopic procedures, and duration of surgical procedure are surgical-related factors that increase the risk of PONV. Each 30-minute increase in procedure time increases the risk of PONV by 60%.[3]

Based on the well-established risk factors, McCracken et al, recommends that anesthesia professionals survey patients carefully and baseline risks reduced whenever possible. In high risk patients, regional anesthesia should be considered, as general anesthesia is associated with an 11 fold increased risk of PONV.[3] When general anesthesia is indicated, propofol should be considered as an induction agent.[3] The antiemetic effects of propofol are poorly understood, however, the incidence of PONV is decreased when propofol is administered, potentially more effectively than the use of ondansetron.[4]

Also, McCracken et al recommends the limited use of intraoperative opioids in high risk PONV patients. Opioids directly stimulate the chemoreceptor trigger zone therefore, they are profound emetics.[4] The use of regional anesthesia can significantly reduce the need for opioid but if not possible, non-opioid analgesics should be used. Ketamine is a phencyclidine derivative that produces dissociative anesthesia.[4] In subanesthetic doses, ketamine produces profound analgesia and decreases the total required dose of propofol and opioid when given simultaneously.[5] Also, the addition of ketamine to a propofol infusion promotes hemodynamic stability and a lesser incidence of apnea, which is a common complication of continuous propofol infusion.[5] Hemodynamic and respiratory stability were particularly desirable in this case due to the need to monitor the patient from outside the OR during the intraoperative radiation treatment. Apnea or hypotension during this time would have required interruption of the radiation treatment. Though not used in this case the inclusion of an antisialagogue, like glycopyrrolate, should be considered as ketamine can induce increased salivary secretions, causing cough or laryngospasm.[4]

The novel drug combination of propofol and ketamine has gained popularity in many emergency departments across North America; often referred to as "Ketofol."[5-7] Because propofol is an IV emulsion, its mixing compatibility is questionable. Research on this topic is limited and according to the U.S. Food and Drug Administration, propofol should not be mixed with other therapeutic agents.[8] Based on high performance liquid chromatography and physical evaluation, Donnelly et al and

Calimaran et al, found a ketamine-propofol mixture to be physically compatible and chemically stable for use in a syringe for 3 hours.[6,7] Researchers concluded that both ketamine and propofol retained 97% of their original concentrations with no visible layering or oil droplet formation when mixed in a syringe and were therefore stable.[6,7] However the evidence of compatibility is still uncertain and before a ketamine-propofol mixture can be accepted as nurse anesthesia standard of care, further research should be conducted to conclude safety and establish combination guidelines.

As an anesthesia team, avoiding PONV while maintaining a sedated, hemodynamically stable patient without apnea, was our goal. Recognizing this patient was at high risk for PONV because of her non-smoking status, history of motion sickness, female gender and prolonged duration of surgery, an anesthetic with low PONV risk was necessary. Total intravenous anesthesia (TIVA) with propofol, ketamine and low dose opioid along with local infiltration of bupivacaine was our choice to diminish the risk of anesthetic complication, as it avoided volatile anesthetics and minimized opioids. Low dose ketamine also helped maintain respiratory drive and avoid hypotension. These effects of ketamine were of particular importance during the intraoperative radiation treatment, but also contributed to a smooth anesthetic with minimal use of opioid. Ultimately, the patient's post anesthesia course, both without pain or PONV, confirmed the anesthetic management was a success. In future cases, separate infusions of ketamine and propofol should be used until protocols for mixing have been established and safety of a ketamine-propofol combination supported.

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Anesthetic and Hemodynamic Management for Pheochromocytoma

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Keywords: pheochromocytoma, catecholamines, hypertension, adrenalectomy, norepinephrine

Pheochromocytomas are catecholamine secreting tumors most commonly found on the adrenal medulla. These chromaffin tissue neoplasms produce high levels of epinephrine, norepinephrine and dopamine, presenting considerable hemodynamic challenges to the anesthetist.[1] Despite an incidence of only 1.55-2.1 per 1 million patients each year, it is imperative to have a strong understanding of the pathophysiology and hemodynamic management of the pheochromocytoma patient.[2]

Case Report

A 63-year-old female presented for a laparoscopic adrenalectomy secondary to a pheochromocytoma. Diagnosis was made six weeks prior, following a non-embolic cerebrovascular accident (CVA). Past medical history included hypertension, coronary artery disease (CAD), myocardial infarction (MI) with stent placement, obstructive sleep apnea, hypothyroidism, hyperlipidemia and CVA. The patient was obese based on a BMI of 32.5, calculated from height (157 cm) and weight (80 kg). Recent cardiac catheterization showed nonobstructive CAD and a left ventricular ejection fraction of 75%. Home medications included metoprolol 12.5 mg, amlodipine 5 mg, atorvastatin 80 mg, levothyroxine 88 mcg, and aspirin 81 mg. A phenoxybenzamine regimen of 10 mg, twice daily (BID), had been in place during the six weeks between diagnosis and surgery. All prescribed medications, including phenoxybenzamine, were taken the morning of surgery. Complete blood count and electrolytes were within normal limits. Preoperative blood pressure (BP) was 160/93 mmHg and heart rate (HR) was 57 beats/min.

Standard monitors were applied and a thoracic epidural was inserted at the T9-T10 level for postoperative analgesia and hemodynamic control. A femoral arterial line was placed with ultrasound after unsuccessful radial artery cannulation. The patient received four 1 mg doses of midazolam, three 25 mcg doses of fentanyl, and 500 mL of normal saline during these procedures. Non-invasive BP ranged from 140/75 – 180/90 mmHg. The patient was given intravenous (IV) 2% lidocaine 100 mg followed by propofol 200 mg for induction. Rocuronium 50 mg was administered and the trachea was intubated with minimal BP change.

Anesthesia was maintained with sevoflurane. Following induction, a central venous catheter was placed in the right internal jugular vein. The patient exhibited hemodynamic instability during two hypertensive episodes with systolic blood pressures (SBP) greater than 180 mmHg requiring intervention. 2% lidocaine 10 mL was given through the epidural to lower BP via analgesia and sympathectomy. Following the administration of regional anesthesia there were brief reductions of BP to 120/65 – 140/75 mmHg, requiring no intervention. A third exhibition of hemodynamic instability occurred related to direct surgical manipulation of the pheochromocytoma. BP elevated to 220/110

mmHg and was sustained for approximately 7 minutes. Due to the severity of the hypertension and the direct tumor manipulation, IV hypotensive agents were chosen over epidural management. The patient received IV hydralazine 20 mg and IV nitroglycerine 120 mcg for hemodynamic control. After surgical clamping of the adrenal vein, the patient became hypotensive. BP was unresponsive to phenylephrine or ephedrine, and an SBP of 80 mmHg was sustained. An IV norepinephrine infusion was started at 6 mcg/min and titrated to a BP of 115/55 mmHg. Due to inadequate hemodynamics, the epidural was not re-dosed for post-operative analgesia. The patient was extubated in the operating room and taken to the post anesthesia care unit with norepinephrine infusing at 4 mcg/min.

Discussion

The goal of pheochromocytoma treatment is to reduce the hemodynamic response to catecholamines; however, the only definitive treatment is surgical removal of the tumor.[1,2] Over 90% of pheochromocytomas are located on the adrenal gland, therefore tumor eradication frequently requires adrenalectomy.[1] The laparoscopic approach to adrenalectomy is preferred due to the advantages of smaller incisions, decreased postoperative pain, scar tissue minimization, fewer postoperative complications, and greater patient satisfaction.[3] Despite the advantages of the laparoscopic approach, the pheochromocytoma patient provides a myriad of challenges to the anesthesia practitioner.

Anesthetic management should begin at least two weeks prior to adrenalectomy with the institution of an alpha (α) blockade, followed by introduction of beta (β) blockade.[1,2,4] Current literature stresses withholding β-blockade until α-activity has been suppressed to avoid unopposed α-activation and subsequent myocardial distress evidenced by angina, dyspnea, and/or electrocardiography (ECG) changes.[1,2,4] Preoperative treatment targets normalization of blood pressure, heart rate, and organ function, as well as restoration of intravascular volume.[5] Preoperative treatment goals vary institutionally, however most research suggests an average blood pressure target of 130/80 mmHg or less and a heart rate of 60-70.[5] In the event of uncontrollable preoperative hypertension or tachydysrhythmias, it is at the discretion of the anesthesia practitioner to postpone surgery until hemodynamic status is optimized.[5]

The use of phenoxybenzamine, a nonselective α-adrenergic antagonist, is a cornerstone for pre-operative optimization.[6] With greater effects at the α-1 receptors, phenoxybenzamine provides adequate relief of peripheral vasoconstriction mediated by the pheochromocytoma.[6] A 2012 review by Domi and Laho strongly recommends discontinuing phenoxybenzamine 24-48 hours prior to surgery to avoid refractory hypotension following tumor removal.[4] The continuation of α-antagonism contributes to the sustained hypotension and required hemodynamic support postoperatively. An ideal alternative to continuation or cessation of phenoxybenzamine would be to bridge the gap with a short-acting α1-selective-antagonist like doxazosin or terazosin.[2,3]

Intraoperative management of anesthesia should be focused on the periods of highest risk for hemodynamic instability: laryngoscopy, intubation, and surgical manipulation of the pheochromocytoma.[2,3] The most efficient hemodynamic control occurs with a combination of general and regional anesthesia with invasive BP

monitoring.[3,7] Regional anesthesia, specifically epidural, has been shown to considerably reduce catecholamine release during periods of physiologic stress through blockade of sympathetic nerve fibers.[2,3] The thoracic epidural provides remarkable control over SBP and HR during times of physiologic stress, however, it is incapable of blunting the sympathetic response to surgical pheochromocytoma manipulation.[2] Tumor manipulation is a period of extreme hemodynamic stress which requires use of intravenous hypotensive agents.

In addition to epidural anesthesia, intravenous agents are required to circumvent the hemodynamic effects of laryngoscopy and intubation. Opioids are highly effective in blunting the sympathetic response, particularly remifentanil, a valuable alternative agent in this case.[4] Propofol is appropriate for induction due to its associated reduction of systemic vascular resistance.[2,7] Ketamine is contraindicated due to its sympathomimetic properties.[1] For neuromuscular blockade, it is imperative to avoid histamine releasing agents (mivacurium, atracurium, and tubocurare).[1,2] Succinylcholine should be used sparingly due to its ability to increase intra abdominal pressure leading to inadvertent pheochromocytoma manipulation.[2] Rocuronium does not promote histamine release, nor does it possess vagolytic effects, and is therefore used for initial relaxation. Vecuronium has an identical cardiovascular profile and subsequently provides maintenance for this case.

General anesthesia should be maintained with the volatile anesthetic sevoflurane or isoflurane.[1,2] The ability to rapidly titrate desflurane is desirable, but it is typically avoided due to the potential tachycardia response.[1,2] Older agents such as enflurane and halothane are contraindicated due to predisposition to ventricular dysrhythmias in the presence of epinephrine.[2] The use of sevoflurane in this case is appropriate; however isoflurane is an acceptable alternative.

Surgical manipulation of the pheochromocytoma causes severe hemodynamic changes requiring rapid acting hypotensive agents.[1,3,4,7,8] Communication between the surgeon and anesthesia professional is paramount at this stage.[7] In the event of hypertensive emergency, there are multiple acceptable agents to restore homeostasis. Nitroprusside, nicardipine, nitroglycerine, magnesium, esmolol, phentolamine, and increasing the depth of anesthetic agents have all been successful in hypertensive emergencies.[1,2,4,7,8] Increasing anesthetic depth and short acting hypotensive agents such as esmolol provide only short term relief, and more significant hypotensive agents are necessary. The agent of choice in this case is nitroglycerine, an ideal drug due to SBP decrease and preload supplementation.[6] Nitroglycerine also provides coronary artery vasodilation which is beneficial with the CAD patient.[6] Avoiding nitroprusside is prudent due to the coronary steal effect.[6] The addition of a calcium channel blocker could prove to be an ample adjunct.

After excision of the pheochromocytoma, there is a high risk of hypotension due to the sudden decrease in circulating catecholamines and decreased blood volume related to vasodilation.[1,2,4,8] This effect is enhanced by phenoxybenzamine, an agent in the patient's medication regimen.[8] Post-excision hypotension is best controlled with epinephrine, norepinephrine, phenylephrine, ephedrine, dopamine, or vasopressin, depending on severity and co-morbidities.[1,2,4,8] Vasoactive infusions

should be, and are, administered through a central line.[5] Individuals requiring post-operative infusion of vasopressors frequently normalize within 12 hours and no longer require hemodynamic support.[7,8]

The anesthetic goals for the pheochromocytoma patient are unvarying, despite the multiple treatment modalities. It is necessary for anesthesia practitioners to be prepared and equipped to monitor and control the extreme hemodynamic changes that accompany this population.

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Reducing the Incidence of Postoperative Nausea and Vomiting

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Keywords: anesthesia, antiemetic, postoperative, nausea, vomiting

Postoperative nausea and vomiting (PONV) has been thought of as a "big little problem" in anesthesia. The incidence of PONV is 30%.[2] However, in high risk patients, PONV is estimated to occur in up to 80% of cases.[1]Several factors including female gender, non-smoking status, history of PONV or motion sickness and genetic predisposition are used to identify high risk patients. The type and length of the surgical procedure also plays a role in the incidence of PONV.[2] In addition to patient discomfort and decreased satisfaction, PONV can increase risk of serious complications

including aspiration, wound disruption, and increased surgical bleeding.[3]

Case Report

A 41-year-old, 57 kg, 170 cm female arrived to the preoperative unit for a robotic left partial nephrectomy. The patient had no known drug allergies and medications consisted of omeprazole 20mg by mouth BID. Medical history consisted of stage II renal cell carcinoma. Surgical history included a left partial nephrectomy three months prior, without subsequent chemotherapy. Postoperatively, the patient described severe PONV lasting 3 weeks, requiring an esophagogastroduodenoscopy (EGD) due to pain and hematemesis. The EGD showed mild esophagitis; the patient denied subsequent symptoms or treatment following the EGD. Preoperative vital signs included a heart rate of 71 beats per minute, blood pressure 104/67 mmHg, respiratory rate 18 breaths per minute. The patient was afebrile. Physical examination, preoperative testing and laboratory values were within normal limits. The patient had fasted for eleven hours.

Midazolam 4 mg was administered intravenously (IV) to reduce PONV associted with excessive anxiety before transport to the operating room.[2] Preoxygenation with 100% O2 via facemask was provided. A bolus of lactated ringer's 500 ml was infused prior to IV induction of anesthesia with propofol 150 mg, ketamine 40 mg, and vecuronium 6 mg. A Macintosh 3 blade was used to visualize the glottic opening. The trachea was intubated with a 7.0 endotracheal tube (ETT). Mechanical volume control ventilation was initiated and ETT placement was confirmed by bilateral auscultation of breath sounds and positive end tidal carbon dioxide detection. Initial ventilation settings were as follows: tidal volume of 400 ml and ventilation rate 10 breaths per minute, with plateau pressures at 20 cm H2O. A flow rate of one liter each of oxygen and air was started. A propofol infusion was titrated for a bispectral index of 40-60.

Two additional large bore IV catheters and a radial arterial line were inserted. Prior to incision the patient received cefazolin 1 gram, acetaminophen 1 gram, magnesium sulfate 2 grams, diphenhydramine 25 mg, droperidol 0.625 mg and dexamethasone 5 mg IV. Ketamine 10 mg IV was administered each remaining hour of surgery. The patient received vecuronium as needed to maintain 0-1 twitches. The patient's vital signs remained stable throughout surgery.

Thirty minutes before extubation, ondansetron 4 mg IV was administered. Neostigmine 3 mg IV and glycopyrolate 0.6 mg IV were administered to obtain train-of-four of 4/4 and greater than five seconds sustained tetany. Mechanical ventilation was discontinued when spontaneous ventilation was adequate. Hydromorphone 1 mg was titrated intravenously prior to case end. The surgeons administered bupivacaine 0.5% subcutaneously to incision sites. Following an uneventful extubation the patient was transported to the post anesthesia recovery unit (PACU). Fluid administration totals included lactated ringer's 3000 ml and albumin 5% 500 ml. The estimated blood loss was 200 ml, and urine output was 1300 ml.

Postoperative assessments by anesthesia professionals were completed at one and two hours in the PACU. The patient denied pain or nausea and vomiting. Four hours post operatively, the patient began to complain of 4/10 pain but declined medication for pain

and denied PONV. On postoperative day one the patient denied PONV. Pain was relieved by hydromorphone IV.

Discussion

Postoperative nausea and vomiting is a multi-factorial problem that needs to be approached with multi-modal therapies to balance the treatment of nausea, vomiting, and pain.[2] When a high-risk patient is identified, the anesthesia practitioner should start therapies to prevent PONV in the preoperative setting. This patient had 3 of 4 risk factors including female gender, non-smoking status, and history of PONV. The fourth risk factor of genetic predisposition was unknown. As anxiety is also a known contributor to PONV, premedication with a benzodiazepine such as versed is advantageous.[2] Preoperative administration of 2ml/kg of crystalloid for every hour of fasting has been shown to be more effective in the prevention of PONV than intraoperative infusion of 3ml/kg of crystalloid.[4]Adequate hydration needs to be continued during the intraoperative period as patients receiving larger volumes of crystalloids have decreased incidence of PONV.[4] According to Haentjens et al.[4] the type of fluid replacement, colloid versus crystalloid, has little effect on the incidence of PONV in patients with minimal blood loss and fluid shifts. It has been shown that balanced administration of colloid and crystalloid in cases with large blood loss and fluid shifts decreased PONV.[4]

The prevention of PONV is the basis for administering intraoperative antiemetics. Numerous studies have shown that the administration of two antiemetics is more efficacious than one, estimating that each antiemetic can decrease PONV by up to 30%.[2] Early administration of dexamethasone, a corticosteroid anti-inflammatory drug, reduces PONV incidence.[5] According to Oliveira Jr. et al.[5] a dose of 4-5 mg IV is equally as effective as an 8-10 mg single dose, or combination therapy. Ondansetron, a $5HT_3$ receptor antagonist, is another effective and commonly used antiemetic. However, it is questionable whether $5HT_3$ receptor antagonists are centrally acting.[1] Aprepitant, a highly selective, brain-penetrating NK_1 receptor antagonist, previously approved for chemotherapy induced nausea and vomiting, has become available for management of PONV.[1] According to Gan et al.[1] a preoperative dose of aprepitant 40mg PO was shown to be superior to ondansetron in the prevention of vomiting for the first 24-48 hours; although there were no significant differences for nausea control, use as a rescue drug, or overall complete response. Aprepitant was not used in this case as it was unavailable at this institution, but considering the long half-life of aprepitant, of 9-12 hours, and its superior drug profile for vomiting, it should be considered in high risk cases.[1] Dopamine antagonists, antihistamines and anticholinergic drugs are also effective antiemetics, although each has side effects that need to be considered.[3] In this case study, the patient received drugs from each of these classes and did not show any negative side effects postoperatively.

It has long been established that total intravenous anesthesia is associated with less PONV than inhalational agents.[3] When propofol is used as both an induction and maintenance agent, PONV is further decreased.[2] The antiemetic effect of propofol can last up to 30 minutes after the operation, aiding in prevention of PONV.[2] The use of short-acting opioids, such as remifentanil infusion or single bolus sufentanil do not increase the incidence of PONV, but neither do these agents provide adequate postoperative analgesia.[2] Since

both inadequate pain control and opioid administration increase incidence in PONV, a multi-modal pain management plan is optimal.[2] Administration of non-opioid medications has the benefit of increasing effectiveness of overall pain management, while decreasing opioid administration.[6] Non-opioid adjuncts effective in pain management include non-steroidal anti-inflammatory drugs such as ketorolac and acetaminophen. Gabapentanoids, local anesthesia, regional anesthesia and NMDA antagonist, such as ketamine are also effective.[6] Using a multi-modal pain management strategy can limit side effects of not only opioids, but of all the agents used in the therapy.[6] However, caution must be taken in choosing which adjuncts are appropriate for each patient. For example, in this case ketorolac was omitted because of the increase risk of postoperative bleeding and the patient's history of renal cell carcinoma.

Neostigmine is an anticholinesterase and has cholinergic effects on the gastrointestinal tract including increased motility and gastric acid secretion.[7] A reversal agent should always be administered when medically necessary following neuromuscular blockade. Side effects from PONV are less severe than those of residual respiratory paralysis.[7]According to Cheng et al.[7] there is no increased incidence of PONV with neostigmine administration.[7] Further studies are needed to assess if dose-dependent administration of neostigmine impacts PONV. In addition, there are limited studies assessing whether administering atropine versus glycopyrolate impacts PONV.

In conclusion, the prevention of PONV starts with risk stratification and continues with tailoring the anesthetic plan based on patient medical history and any surgical concerns. Postoperative nausea and vomiting is not a "big little problem" that ends with patient discomfort, it is an anesthetic risk that can result in serious postoperative complications including increase risk of aspiration, wound disruption, and surgical site bleeding.[3] Postoperative nausea and vomiting is a multi-factorial problem that needs to be approached with multi-modal therapies to balance the treatment of nausea, vomiting, and pain.[2]

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Anesthetic Considerations in a Parturient with Fontan Circulation

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Keywords: Fontan palliation, neuraxial versus general anesthesia

Advances in surgical correction of complex cardiac congenital defects have increased the rate of survival to adulthood to 85%.[1-4] Female survivors reaching child-bearing age often consider pregnancy. Anesthesia is especially challenging in parturients with a single functioning ventricle and Fontan pulmonary circulation.[1-6] Physiologic changes related to pregnancy place these females at a higher risk for cardiac failure due to a limited cardiac reserve.[1-6] Anesthesia practitioners must be familiar with nuances of the Fontan circulation; be aware of the risks and maximally optimize conditions of the parturient preparing for labor.

Case report

A 19-year-old female gravida 1 para 0 presented with intrauterine contractions and 2 cm cervical dilation at 36 weeks of gestation. The patient had a history of hypoplastic left heart syndrome at birth. The patient had previously undergone three palliative surgeries with the final Fontan repair at the age of 3 years. There were no other medical problems or allergies. The parturient stated she had an unremarkable pregnancy until the prior 2 weeks when she developed shortness of breath and fatigue with her regular physical exercise. Medication included aspirin 81 mg once a day. She had no signs of distress and SpO_2 was 89-93 % on room air. A transthoracic echocardiogram showed a dilated single ventricle, ejection fraction (EF) of 45% and patent Fontan conduit. She was assigned class II of the New York Heart Association (NYHA) functional classification by the consulting cardiologist. Laboratory values included: Hb 10.2 g/dL, Hct 29.5 %, platelet count 103 x 1000/mm^3, PT 10.5 s and PTT 31.8 s.

The anesthetic management plan was immediate administration of epidural anesthesia for a vaginal delivery. In the labor suite, 20 and 18 gauge peripheral intravenous (IV) catheters were inserted. The right radial artery was cannulated for continuous blood pressure (BP) monitoring. An epidural catheter was placed in the left lateral position with a midline approach without difficulty. Epidural anesthesia was initiated with a bolus of ropivicaine 0.2% 10 ml and fentanyl 100 mcg followed by a continuous infusion of ropivicaine 0.2% at 6 ml/hr. Heparin infusion was initiated and titrated to a goal PTT between 60-70 seconds. A healthy male infant was delivered vaginally with vacuum assistance and minimal blood loss. Throughout the entire labor period the parturient remained hemodynamically stable. She was medicated with phenylephrine 40-80 mcg for any 20%

drop in baseline BP. Immediately after cord clamping, 20 units of oxytocin were injected into the infusing IV fluids. In the postpartum period, the patient had no signs of cardiac or respiratory distress. Both the epidural and radial artery catheters were removed without hematoma formation after heparin infusion was discontinued for 4 hours. Further anticoagulation therapy was adjusted per cardiology recommendations. The patient and her infant were discharged home uneventfully 2 days later.

Discussion

A Fontan repair is the treatment of choice for congenital lesions with a single ventricle.[1,2,4] The procedure is a multistage process. In the final step of palliation, the entire systemic venous output is rerouted to the pulmonary circuit via a surgically created conduit placed either inside or outside of the heart. The surgery completely separates deoxygenated blood from oxygenated blood.[1-6] The continuum of flow through the conduit and the lungs relies on a pressure gradient between the systemic veins and pulmonary vasculature.[2-5] Any changes in pulmonary vascular resistance (PVR) and preload easily impact the patient's cardiac output (CO).[2,4-6] Average CO after Fontan repair is only 70% of a normal functioning heart.[3,4,8] Within 5 years of repair, 9% of these patients develop class III NYHA heart failure while 70% have symptoms of heart failure within 10 years of repair.[2,6] A single ventricle struggles to match the physiological surplus of blood volume and increased CO demanded in pregnancy and labor.[2,4,6-9]

Limited cardiac capacity jeopardizes maternal and fetal wellbeing. Maternal threats include arrhythmia, thrombo-embolism and heart failure.[5-8] In 26% of these patients supraventricular arrhythmias developed during the third trimester.[2,4,6-8] Fetal risks are prematurity, miscarriage, early membrane rupture and intrauterine growth retardation.[4-9] Invasive monitoring is prudent during anesthesia delivery. Direct arterial pressure monitoring provides beat to beat evaluation of the systemic pressure and immediate access for arterial blood sampling.[6-9] A central venous catheter estimates intravascular volume and responsiveness to fluid treatment.[4,6-9] Echocardiogram is mandated in the peripartum period to evaluate patency of the surgical conduit and ventricular function.[3,4,8] Women whose EF is less than 45% are prone to high maternal morbidity during labor.[1-5, 7,8] The overall goal is to reduce the stress of labor in ways that allow the parturient with a single functioning ventricle to compensate. The typical obstetric plan for these patients is either minimal pushing utilizing forceps or vacuum or complete avoidance of expulsive efforts via an elective cesarean section.[1-9] Despite choices in delivery approach, the optimal anesthesia mode for laboring women post Fontan, is neuraxial anesthesia.[1,4,5,7-9]

Neuraxial anesthesia offers less hemodynamic fluctuation compared with general anesthesia (GA). Risks of GA are a buildup of CO_2 and increased intrathoracic pressure leading to a rise in PVR and decline of maternal CO respectively.[1,4,5,7-9] Such a decline could interrupt fetal oxygen supply.[5-9] Manipulation of the airway during GA causes additional surges of catecholamines followed by unfavorable increases in PVR.[4,5,7-9] General anesthesia is usually avoided and considered only when a regional approach is contraindicated.[5,7-9] Administration of spinal or epidural anesthetics causes a sympathetic blockade.[1-3, 6-9] This block lowers cardiac preload and produces hypotension.[1,4,5,9] However symptoms are less pronounced

with epidural anesthesia.[7-9] Historical reviews of women with repaired cyanotic defects show that early placement of an epidural catheter alleviates workload for the single ventricle and promotes adequate blood flow in the Fontan circulation.[1,4,9] Epidural anesthesia reliably maintains cardiac output and rapidly converts epidural analgesia to a full surgical block in case of obstetric emergency.[5-9] A single shot of spinal anesthetic has proved safe in patients with well-preserved ventricular function only following adequate pre-hydration and exaggerated left uterine displacement.[1-5,7,9] A combined spinal epidural technique attains the same surgical anesthesia as spinal approach and allows a slow titration of epidural anesthetics, minimizing hemodynamic instability.[8,9] Addition of opioids to neuraxial anesthesia improves the control of postpartum pain.[4,5,7-9] An intrathecal or a combined technique should be considered when time to delivery is limited or following an unintended dural puncture. Factors precluding regional anesthesia should be evaluated early.[4,6-8]

Anticoagulation is vital for patency of the Fontan conduit.[5,6] A hypercoagulable state of pregnancy increases the incidence of thrombo-embolism with peak occurrence during the postpartum period.[4,5,8,9] Delivery must be planned well in advance and allow for fine tuning of anticoagulant therapy. A detailed assessment of coagulation status will ascertain timely, effective placement and removal of epidural and other invasive catheters without subjecting the parturient with Fontan circulation to risks of bleeding. In conclusion, women with Fontan palliation have become more commonplace in labor units. These young women must be educated about pregnancy related risks upon entering childbearing age. Early education gives the patient time for decisions and time to establish regular medical surveillance. Delivery under epidural anesthesia in patients after Fontan repair is recommended as a safer alternative to general anesthesia.

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Cardiac Tamponade following Arrhythmia Ablation

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Keywords: atrial fibrillation, pericardial effusion, pericardiocentesis, radiofrequency catheter ablation, cardiac tamponade

Cardiac arrhythmias contribute to the morbidity and mortality of cardiac disease, with atrial fibrillation (AF) the most prevalent, respectively estimated at 2.7 and 6.1 million.[1] Radiofrequency catheter ablation offers an increasingly popular treatment option for the patient suffering from AF.[2] A 2009 study of 32,569 patients undergoing ablation of AF found a mortality rate of 1 in 1,000.[3] Of these mortalities, cardiac tamponade was identified as the most frequent and fatal complication.[3] The importance of the anesthesia provider to promptly identify and manage the patient suffering from tamponade in this setting cannot be overstated.

Case Report

A 74.4 kg, 73-year-old female presented for ablation of left atrial appendage as treatment for paroxysmal AF. Past medical history included: paroxysmal AF and sick sinus syndrome which necessitated placement of an AICD with pacing capabilities, hypertension, gastroesophageal reflux disease, and glomerulosclerosis. Her past surgical history was significant for appendectomy, and cholecystectomy. The patient's medication list included bisoprolol, diltiazem, omeprazole, warfarin, and lovenox, as bridge therapy for warfarin. A preoperative electrocardiogram (ECG) showed atrial pacing with a rate of 84; transesophagealechocardiogram showed moderate aortic regurgitation, mildly dilated left atrium, mild mitral and tricuspid regurgitation, mild atherosclerotic plaque in descending aorta; cardiac computed tomography angiogram outlined codominance of coronary arteries, hiatal hernia, left atrial enlargement, a left atrial appendage, dilated left atrium; AICD interrogation reported DDDR with rate set at 60-130. A complete blood count, comprehensive metabolic panel, and prothrombin time with INR, were available prior to the procedure with all normal values.

Prior to the procedure, the pacemaker was interrogated and the defibrillator was inactivated. An 18 gauge intravenous line was established and the patient was brought to the electrophysiology laboratory (EPL). Standard monitors were applied and the patient was noted to be in AF with a ventricular rate of 80 beats/min, blood pressure 140/80 mmHg, and oxygen saturation was 97% on room air. Intravenous induction was performed with midazolam 2 mg, lidocaine 60 mg, fentanyl 50 mcg, propofol 100 mg, and rocuronium 50 mg. Direct laryngoscopy with a MAC 3 blade provided a grade II view. The trachea was intubated with a cuffed 7.0 mm oral endotracheal tube, placement was confirmed, and respirations controlled via

mechanical ventilation. General anesthesia (GA) was maintained with isoflurane 0.6% for amnesia and remifentanil 0.05 mcg/kg/min for analgesia and akinesia. The patient was positioned supine with arms tucked. Following induction, a right radial arterial line and right internal jugular central venous catheter were placed.

Intraoperatively, the patient received adenosine 18 mg to disrupt the aberrant rhythm of atrial fibrillation; the underlying rhythm was sinus rhythm with a rate of 75 beats/min. Heparin administration for prevention of thromboembolism, totaled 13,000 IU, given in aliquots every hour to maintain an activated clotting time of greater than 300 seconds. Towards the end of the case protamine 40 mg, given over a period of 15 minutes, was administered to reverse the effects of heparinization. Shortly after, the patient developed hypotension with systolic blood pressures ranging from 85-90 mmHg, transiently responding to 240 mcg of phenylephrine, given in 120 mcg aliquots. An additional 240 mcg of phenylephrine, again given in 120 mcg aliquots was administered with concurrent 500 mL fluid bolus. Hypotension persisted and a total of ephedrine 20 mg was administered. While a protamine reaction was being considered, the cardiologist performed a transthoracic echocardiogram and pericardial effusions were identified. Subxyphoid pericardiocentesis under echocardiographic guidance followed and 300 ml of blood was aspirated from the pericardial sac, a pericardial drain was placed, and minimal effusion was visualized via echocardiography post drainage.

The patient remained intubated while her hemodynamics normalized, with pericardial drain in place and was admitted to the coronary intensive care unit. The patient was administered midazolam 2 mg and fentanyl 100 mcg before the transfer. Upon arrival to the unit, the patient was in sinus rhythm with a rate of 78 beats/min, blood pressure 160/90 mmHg, and oxygen saturation 99% on 40% FiO_2. The patient remained intubated until that evening and the drain was removed on postoperative day 2, concluding with patient discharge on postoperative day 4.

Discussion

Radiofrequency catheter ablation of AF under GA shows promising results including higher success rates, decreased procedural and fluoroscopy time, and greater catheter stability and contact for lesion ablation secondary to patient akinesis.[1] Additionally, efficacy depends upon arrhythmia induction to accurately map the point of origin.[4] Therefore, an anesthetic providing akinesis without suppression of arrhythmias or interfering with the cardiac conduction system provides optimal procedural conditions. The most appropriate anesthetic for the procedure is debated and current literature is conflicting. Erb et al. (2002), found isoflurane and propofol to be an equally suitable anesthetic relative to atrioventricular nodal conduction and ventricular repolarization for children and adolescents undergoing radiofrequency catheter ablation.[5] Mandel et al. (2010) point out that while dose-dependent bradycardia and hypotension is associated with remifentanil infusion, it has little effect on arrythmogenicity.[6] The anesthetic delivered to the patient, 0.6% isoflurane and remifentanil 0.05 mcg/kg/min, provided adequate amnesia, analgesia, and akinesia, without suppression of arrythmogenicity. Ablation is not without risk, and may result in chamber wall perforation, leading to rapid accumulation of pericardial effusion.[7] A pericardial effusion causing a rise in intrapericadial pressure leading to

hemodynamic instability, and ultimately cardiogenic shock with circulatory collapse, defines the cardiac tamponade state. The intent of this article is to highlight the identification and management of cardiac tamponade.

The progression of a pericardial effusion exists on a continuum of three phases.[8] Phase one describes the gradual rise of intrapericardial pressure as a result of an increasing accumulation of fluid. As a result, right ventricular (RV) and left ventricular (LV) filling pressures increase to meet physiologic demands. Phase one concludes with the equalization of the intrapericardial and RV filling pressures, at approximately 10 mmHg.[8] Clinically, a slight drop in cardiac output (CO) and arterial pressure during inspiration may be seen. Phase two proceeds with progression of fluid accumulation, further increasing intrapericardial and right atrial (RA) pressures until they match left atrial (LA) at roughly 15 mmHg.[8] A drop in CO will be noticeable in this phase, and an arterial pressure drop nearing 10 mmHg on inspiration will be present. Phase three cardiac tamponade begins when intrapericardial, RA, and LA pressures equalize. CO will continue to decline, arterial pressure will drop > 10mmHg (pulsus paradoxus) after inspiration, and circulatory collapse will occur.[8] Within the EPL, the clinicians have a variety of tools available to identify a patient in distress. The cardiologist has access to fluoroscopy, intracardiac echocardiography, transthoracic echocardiogram, and ECG.[1] The anesthesia provider monitors a 5-lead ECG, pulse oximetry, noninvasive and invasive blood pressure, temperature, end-tidal carbon dioxide, and gas monitoring.[1]Our anesthesia set up included the above monitoring parameters. In addition, we placed a right internal jugular central venous catheter preemptively should a catecholamine stress test, central venous or pulmonary artery pressure measurements, and/or transcutaneous pacing be desired. The arterial line allowed us to assess beat to beat variability. In retrospect, transducing and trending a central venous and pulmonary artery pressures throughout the case would have allowed us to identify the progression of the tamponade state. Early in the patient's hypotensive state, employment of transthoracic echocardiography identified pericardial effusions as the likely source of the patient's hemodynamic instability.

A patient with tamponade may present with dyspnea, chest pain, anxiety, hypotension, and palpitations.[8,9] Classic signs of tamponade include the Beck triad: decreasing arterial blood pressure, increasing jugular vein distention, and muffled heart tones.[9] Additional signs include tachycardia, tachypnea, electrical alternans, and pulsus paradoxus. A patient under anesthesia is unable to convey symptoms, covered by drapes, and body habitus may mask assessment of jugular vein distention and muffled heart tones. Additionally, hypotension may not present until late in the hypertensive patient. Pulsus paradoxus may be masked in the presence of hypotension, aortic regurgitation, right ventricular hypertrophy, pericardial adhesions, and atrial-septal defects.[9] Pulsus paradoxus may also be present without tamponade in a patient with obstructive pulmonary disease, congestive heart failure, obesity, hypovolemic shock, and mitral stenosis.[9] One study found a long procedural time, coronary artery disease, and arterial hypertension to be independent predictors of pericardial effusion during ablation.[7] The patient described above presented with multiple comorbidities which may have predicted and delayed recognition of the tamponade, including obesity, moderate

aortic regurgitation, and hypertension.

An adjunct to clinical exam, echocardiography with doppler allows for determination of size and location of effusion.[8] Pericardial effusion presentation on echocardiogram is viewed as separation of the pericardium from the epicardium during diastole, and graded as small, medium, or large[8]. Pendular motion of the heart, due to movement in the enlarged sac, may be visualized and correlated with electrical alternans on the ECG.[9] Diastolic collapse of the right atrium suggests tamponade. Inferior vena cava plethora, which is dilation of the inferior vena cava and less than 50% collapse during inspiration, predicts RA pressure to be ≥15mmHg, consistent with phase three cardiac tamponade and impending cardiovascular collpase.[8] In one study, inferior vena cava plethora was correlated with a 92% incidence of effusion requiring pericardial drainage.[10]

When tamponade is suspected and echocardiogram has determined the size and location of the effusion, echocardiographic guided percutaneous pericardiocentesis should follow.[10] The subxyphoid approach is most commonly employed in the EPL since it is extrapleural and avoids the coronary, pericardial, and internal mammary arteries.[10] Unsuccessful resolution via pericardiocentesis may necessitate surgical intervention.

As more ablation of AF procedures are performed under GA, anesthesia providers must develop a foundation of knowledge regarding the procedure and complications. The anesthesia provider should be mindful of pre-existing hypertension, obesity, and length of the procedure as independent predictors of effusion. With cardiac tamponade being the most common and fatal complication to ablation, vigilant evaluation of the patient for signs of tamponade by the cardiologist and anesthesia providers are paramount. Early correlation of the clinical signs of tamponade with echocardiographic findings and subsequent echocardiographic guided pericardiocentesis offers favorable outcomes. As in this case, evaluation of the potential causes of hypotension in the anesthetized patient can be difficult, especially when body habitus may mask classic signs of tamponade, such as muffled heart tones or jugular vein distention. Protamine was considered as a potential cause of the hypotension. Protamine can impair hemodynamics through both immune and idiosyncratic nonimmune reactions.[11] Hemodynamic instability following protamine administration may present as hypotension secondary to systemic vasodilation from histamine release by mast cells, myocardial depression, and pulmonary hypertension.[11] It is important to not merely attribute hypotension to the anesthetic, especially late in the case. The patient's history of moderate aortic regurgitation may have masked identification of pulsus paradoxus on the arterial blood pressure waveform. Early in the progression of hypotension, the clinician must communicate with the cardiologist and consider effusion as the cause during ablation procedures, as the patient may quickly deteriorate to the tamponade state.

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Anesthetic Management of Retained Placenta in a Morbidly Obese Parturient

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Key words: postpartum hemorrhage, retained placenta, maternal obesity

The growing prevalence of obesity is a global health problem.[1] Among parturients, obesity is associated with increased morbidity and mortality.[2] As the number of obese parturients continues to rise, anesthesia practitioners must be prepared for the challenges and risks associated with their anesthetic management, in addition to the challenges inherent with labor.[1] Postpartum hemorrhage (PPH) is a potential complication of delivery that occurs in 3% of all births.[3] It is the second leading cause of maternal death and often requires swift intervention and careful anesthetic management.[3]

Case Report

A 22-year-old, 172 cm, 174 kg primigravida with a body mass index (BMI) 49.4 was admitted directly to the labor and delivery unit with the onset of term labor. Her past medical history, surgical history, and physical examination were significant for morbid obesity, a Mallampati III airway classification, and a large neck circumference.

Shortly after admission, a 20 gauge peripheral intravenous catheter was placed and a 1L fluid bolus of 0.9% normal saline was administered. The anesthesia practitioners were then consulted to place a labor epidural. The epidural catheter was inserted without complication and was confirmed with a test dose of 1.5% lidocaine with epinephrine 1:200,000. Neuraxial labor analgesia was obtained via the epidural catheter using 0.25% bupivicaine to achieve a T_{10} sensory blockade.

The anesthesia practitioners were notified within 30 min of spontaneous vaginal delivery that the patient had failed to deliver the placenta and lost over 1L of blood. They were asked to prepare for an emergent dilation and curettage to remove the retained placenta and stop the hemorrhage.

The patient was brought to the operating room where oxygen was administered via nasal cannula at 2 L/min and standard monitors were applied. The patient took sodium citrate 30 mL per OS. Though slightly tachycardic with a heart rate of 102 beats per min (bpm), all other vital signs were stable with an initial blood pressure of 112/64 mmHg. An additional 16 gauge peripheral intravenous catheter was inserted and a rapid infusion of lactated ringers initiated.

The epidural catheter was redosed with an additional 10mL of 2% lidocaine in an attempt to raise the sensory level of the block to T_4. Once the procedure began, however, the patient was in extreme discomfort and the procedure was paused until adequate analgesia could be obtained. Inadequate anesthesia remained after a second administration of 10mL of 2% lidocaine into the epidural catheter. Intravenous fentanyl 50mcg was then administered in addition to intravenous propofol 20 mg. The surgery was then resumed and removal of the placenta was achieved with minimal discomfort. Following the removal of the placenta, 20 units of oxytocin were added to the maintenance infusion.

Over the course of the procedure, an additional 400mL of blood was lost and the patient became hypovolemic and symptomatic with the systolic blood pressure dropping to 78 mmHg and the heart rate reaching 138 bpm. A rapid infusion of 6% Hetastarch 500 mL and incremental boluses of phenylephrine, a total of 350 mcgs, were sufficient to maintain the patient's blood pressure until two units of packed red blood cells (PRBCs) could be administered.

After a total surgical time of one hour, and with the administration of a total of 1500 mL of crystaloid solutions, 500 mL of colloid solutions, and 2 units of PRBCs, the patient was transferred to the post anesthesia care unit. She was alert and oriented with all vital signs within normal limits.

Discussion

Not only is the incidence of obesity growing in the general population, the rates of maternal obesity is also on the rise.[1] The growing number of obese parturients is of concern because obesity is associated with a higher morbidity and mortality.[2] Two areas of specific concern to the anesthetist are the increased risk for aspiration and the increased risk for failed intubation.

Both pregnancy and obesity place the patient at increased risk for aspiration of gastric contents.[1] The American Society of Anesthesiologists (ASA) practice guidelines for obstetric anesthesia support the administration of a preoperative

nonparticulate antacid, such as sodium citrate, in order to decrease the acidity of the stomach. The guidelines also recommend "a timely administration of H2 receptor antagonists, and/or metoclopramide for aspiration prophylaxis." [5]

Complications in managing the airway is the leading cause of anesthesia-related maternal mortality.[2] In a recent review of the literature, Boutonet, Faitot, and Keita[6] indicate that the incidence of difficult intubation in the obstetric population is 1 in 30, and the incidence of failed intubation is 1 in 280. This is attributed to the changes in anatomy associated with pregnancy. The morbidly obese are at even greater risk of difficult intubations. According to one study, difficult intubations occurred in 1 in 3 morbidly obese patients.[2] In the non-parturient population, several studies have identified the combination of a large neck circumference and high Mallampati score as a positive predictor for a difficult intubation.[2]

The literature defines PPH as blood loss greater than 500mL after vaginal delivery or greater than 1000mL after cesarean delivery.[4] The incidence of PPH is 3% of all deliveries and, next to embolism, is the second leading cause of maternal mortality.[3] Among the various causes of hemorrhage, the incidence of retained placenta (RP) is second only to uterine atony, accounting for nearly 10% of the total cases of PPH.[4] The literature supports the use of various anesthetic techniques for RP, but careful extension of the neuraxial blockade in a hemodynamically stable parturient with a functioning epidural catheter is recommended, as it may be the best option to minimize maternal risk.[5,7] General anesthesia with volatile anesthetics may offer some benefits due to their effect on uterine relaxation, but may not be well-tolerated in a hypovolemic patient and pose risks associated with difficult and failed intubations.[7] Another method to achieve uterine relaxation is the administration of intravenous nitroglycerine.[5,7] After removal of the placenta, uterine tone may need to be enhanced with the administration of oxytocin.[3]

Due to the emergent nature of this case, the anesthesia practitioners were only able to administer sodium citrate prior to beginning the dilation and curettage. Considering the patients BMI, airway classification, and the stable vital signs at the onset of the procedure, it was decided that the anesthetic goal for this patient was to avoid the potential risks associated with general anesthesia and endotracheal intubation, and instead to attempt adequate analgesia using the epidural catheter. Once it was determined that the neuraxial analgesia was insufficient to provide optimal surgical conditions and comfort for the patient the anesthesia practitioners judiciously administered opioids and propofol to achieve adequate sedation. This was done with the understanding that systemic opioids have been shown to cause maternal sedation, obstruction, and hypoxia, but may still be favorable to a difficult intubation.[1, 5] Using a combination of fentanyl and ketamine to provide sedation and pain control may have been a beneficial alternative technique, due to ketamine's analgesic properties and more favorable effect on a patient with low blood pressure.[3] Following the removal of the placenta, oxytocin was administered to help minimize any further bleeding. The decision to administer blood was based on continued blood loss and symptomatic, unstable hemodynamics. Though a current type and screen had not been obtained on the parturient on admission to the labor and delivery unit, the anesthesia practitioners administered two units of type O negative

PRBCs, both of which are supported by the current ASA practice guidelines.[5]

As the number of obese parturients continues to rise, it is important to understand how their altered anatomy and physiology can affect the anesthesia plan and place them at increased risk for morbidity and mortality. Having a knowledge of the current literature and practice guidelines for an obese parturient is of paramount importance.

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Iatrogenic Pneumothorax during Electromagnetic Navigational Bronchoscopy

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Keywords: bronchoscopy, Electromagnetic Navigational Bronchoscopy, peripheral lung lesions, pneumothorax, total intravenous anesthesia

Solitary pulmonary nodules frequently occur in patients with multiple comorbidities who are at high risk for lung cancer.[1] Electromagnetic Navigational Bronchoscopy (ENB) is a newer diagnostic tool used for peripheral lung biopsies in patients who would not tolerate biopsy complications such as pneumothorax. Pneumothorax occurs in 15-40% of computed tomography (CT) guided percutaneous biopsy procedures.[1, 2] ENB is considered a safer method to biopsy pulmonary nodules with a much lower pneumothorax complication rate of 1.6-8%.[1,2] Early identification and treatment of iatrogenic pneumothorax during lung biopsy is crucial to minimize potential life threatening complications such as

hemodynamic compromise or tension pneumothorax.

Case Report

A 77-year-old female presented with a 1.5 cm solitary lung nodule in the right lower lobe for biopsy using ENB. Significant past medical history included obstructive sleep apnea, chronic obstructive pulmonary disease (COPD), obesity, gastroesophageal reflux disease, hypertension, and a 100 pack-year smoking history. Past surgical history included: lumbar spine fusion, total knee and hip arthroplasty, and total abdominal hysterectomy. Preoperative vital signs were stable and SpO_2 was 94% on oxygen at 2 L/min. Electrocardiogram (EKG) revealed sinus rhythm with premature ventricular contractions. Abnormal lab values included: white blood cell count 17.2 x 10^3/uL (microliters), blood urea nitrogen 25 mg/dL, and creatinine 1.3 mg/dL.

Induction and intubation were uneventful following administration of propofol, lidocaine, fentanyl, and rocuronium. The patient remained supine and the ENB was started by the pulmonologist. Anesthesia was maintained with a propofol/remifentanil continuous infusion and intermittent boluses of propofol for total intravenous anesthesia. An air leak was noted at the entrance of the bronchoscope through the 8.0 endotracheal tube during positive pressure hand-bag ventilation during the ENB. A diaphragm and petroleum jelly-soaked gauze were used at the connection of the bronchoscope and endotracheal tube to decrease air leakage. End-tidal carbon dioxide was inaccurate during the procedure due to a leak and intermittent suctioning with the bronchoscope. Hemodynamics were stable for the first hour of the case and lung compliance remained normal with manual ventilation. One hour into the surgery, the SpO_2 abruptly declined to 88%, resistance to manual ventilation increased, inspiratory pressures increased, and the EKG showed sinus rhythm with frequent premature atrial contractions. The pulmonologist was notified, the bronchoscope was removed, and the seal at the entrance of the bronchoscope was closed. The SpO_2 remained around 90%. The decision was made to terminate the surgery and the propofol and remifentanil infusion were stopped. Breath sounds were diminished on the right and expiratory wheezes and coughing were noted. Blood pressure and heart rate remained stable. The SpO_2 remained at 90% on 100% FiO_2 with tachypneic respirations in the 30's. Neostigmine and glycopyrrolate were given for antagonization of neuromuscular blockade. The endotracheal tube was removed once awake and 15 LPM O_2 via non-rebreather facemask was applied for transport to the post-anesthesia care unit (PACU).

In the PACU, a chest radiograph revealed a large right-sided pneumothorax. The SpO_2 remained 85-88% despite supplemental oxygen with laborious breathing and audible wheezes. A thoracic vent was placed by a general surgeon in the right second intercostal space midclavicular line in the PACU with a rush of air exiting the catheter upon puncture. Significant improvement in SpO_2 was observed within 45 seconds with decreased respiratory distress. The patient was admitted to the nursing ward on 4 LPM oxygen per nasal cannula with the thoracic vent in place. After subsequent chest tube placements due to persistent right-sided pneumothorax she was discharged home after seven days with no further complications.

Discussion

ENB is performed by obtaining a CT and creating a 3-dimensional (3D) reconstruction using computer software. The 3D view of the airway is then correlated with the CT scan magnetic markers that are placed on the patient and are used to navigate real-time throughout the procedure. Using a bronchoscope, the pulmonologist is able to locate the peripheral lung lesions and obtain biopsies by piercing the bronchial tissue. This technique is used to limit pleural puncture and pneumothoraces.[2] Pneumothorax is an accumulation of air in the pleural cavity and occurred iatrogenically in this case.[3] Iatrogenic pneumothorax is a known complication of ENB from tissue biopsies and must be promptly diagnosed in order to minimize complications.[1-5] Common signs of pneumothorax include increased inspiratory pressures with decreased chest compliance, tachypnea, and dyspnea. These signs may be masked by general anesthesia during ENB.[3] Other signs and symptoms of pneumothorax include diminished or absent lung sounds on the ipsilateral side, decreased arterial saturation, and hypercarbia.[3,6,7] Decreased SpO_2 and decreased lung compliance were initially noted. The bronchoscope was removed, the breathing circuit closed, and high airway pressures, elevated end-tidal CO_2 and diminished breath sounds on the right chest were noted. A pneumothorax was suspected and the procedure was concluded.

An overall consensus does not exist for a superior anesthetic technique for patients undergoing ENB. Any movement by the patient will decrease the accuracy of the navigation and therefore general anesthesia with neuromuscular blockade is appropriate.[2,5] General endotracheal anesthesia with total intravenous medications was chosen due to the poor seal at the bronchoscope entrance to the endotracheal tube. A paralytic was administered per surgeon request to obtain biopsies of smaller nodules in the peripheral and lower lung lobes. Neuromuscular blockade required positive-pressure ventilation and was complicated by a significant air leak. Because of the significant air leak identification and treatment of pneumothorax was difficult due to the inability to monitor peak airway pressures.[6]

Dynamic lung hyperinflation can occur in patients with COPD due to gas-trapping that occurs with positive-pressure ventilation.[6] Positive-pressure ventilation was difficult throughout this case because of the inability to create a seal around the bronchoscope, suctioning, and inconsistent monitoring of ventilatory volumes and pressures. The combination of all of these components, in conjunction with neuromuscular blockade and patient comorbidities, made the identification of a pneumothorax difficult. In the case of a pneumothorax, positive-pressure ventilation increases the air leak into the pleural cavity and creates a one-way valve and may lead to life-threatening tension pneumothorax and hemodynamic instability.[3] The decision was made to extubate this patient following neuromuscular blockade antagonization because it was believed that mechanical ventilation may increase pleural air entrapment and lead to tension pneumothorax.[3,7] The patients' vital signs were stable and allowed for chest radiograph confirmation of pneumothorax prior to decompression.

Minor pneumothoraces may occur during ENB without signs or symptoms. Acute decreases in blood pressure with increased inspiratory pressures and loss of breath

sounds should direct the clinician towards the diagnosis of tension pneumothorax.[3,7] If hemodynamically unstable, a 14 gauge or other available large-bore catheter should be placed in the second intercostal space midclavicular line or in the fourth intercostal space midaxillary line immediately until a chest tube can be placed.[3,7] In this case the first clues to pneumothorax were the desaturation of SpO_2 to 88% with the inability to oxygenate back to 98% and decreased lung compliance via hand-bag ventilation. Auscultated breath sounds were unequal. The endotracheal tube was not malpositioned as the fiberoptic bronchoscope was used to visualize its correct placement. The decision to stop the procedure, antagonize neuromuscular blockade, emerge from anesthesia, extubate, and get a chest radiograph led to the prompt diagnosis and treatment of pneumothorax including thoracic vent placement by the surgeon. The patient was hemodynamically stable allowing for transport to the PACU and a chest radiograph to confirm diagnosis. Had the patient been unstable the chest radiograph could have been performed in the operating room. Also, had a surgeon not been immediately available for placement of a thoracic vent, a needle decompression by the anesthetist with a 14 gauge IV catheter in the second intercostal space midclavicular line or in the fourth intercostal space midaxillary line may have been necessary.[3,7] The prompt placement of a thoracic vent by a surgeon allowed for the pneumothorax to be decompressed and the patient was able to maintain adequate oxygen requirements with 4 LPM oxygen via nasal cannula within 45 seconds of thoracic vent placement.

This case report demonstrates the need for understanding potential complications of ENB, signs of pneumothorax, and prompt decompression to prevent a tension pneumothorax. In the future I would attempt to improve my ventilatory seal with petroleum jelly-soaked gauze. I would also use a properly fitted diaphragm attachment on the endotracheal tube to better monitor peak airway pressures.

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Jet Ventilation for Microlaryngoscopy

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Keywords: jet ventilation, microlaryngoscopy, airway tumor, supraglottic airway

Alternative methods for ventilating patients are an important part of the anesthetic plan. Jet ventilation provides a viable option in ventilating patients when necessary. Jet ventilation was introduced into medical practice in the 1950s.[1] It can be used during procedures such as microlaryngoscopic surgery, rigid bronchoscopy, emergent airway rescue, or for procedures involving the larynx when an unobstructed view is necessary.[2] The surgeon utilizes manual or mechanical high pressure delivery devices attached to the rigid laryngoscope to direct oxygen through the glottic opening using either supraglottic or subglottic approaches.[2]

Case Report

A 48-year-old, 170 cm, 74 kg male was scheduled for microlaryngoscopy with excision of vocal cord growths. The chief complaint noted by the patient included a worsening hoarseness in his speech over several months. The patient's medical history included tobacco use (25 pack year), hypertension, hyperlipidemia and seasonal allergies. His surgical history included an adenotonsillectomy and a laparoscopic appendectomy, both without anesthetic complications. Preoperative laboratory values included: hemoglobin 13.3 g/dL, hematocrit 40.4%, platelet count 189x1000/mm^3. Preoperative vital signs were as follows: blood pressure 132/76 mmHg, pulse 84 beats/minute, SpO_2 96%, and respirations 14/minute.

The patient was classified as an ASA II and an airway assessment revealed a Mallampati II. In the preoperative holding area, the patient was given 2 mg of midazolam intravenously (IV) for anxiolysis after the preoperative assessment. The patient was transferred to the operating room where electrocardiogram, blood pressure, oxygen saturation and temperature monitors were applied. The patient was pre-oxygenated by breathing 100% oxygen through a mask with a tight seal. Once the patient was pre-oxygenated, induction of anesthesia was performed by administration of 150 mcg of fentanyl IV, 200 mg of propofol IV and neuromuscular blockade was achieved with rocuronium 50 mg IV. After induction of anesthesia, the airway was visualized by direct laryngoscopy with a Macintosh 3 blade revealing a grade I view of the vocal cords. Minor tissue erythema and edema was noted laterally to the vocal cords. The patient was intubated with a 6.0 mm endotracheal tube (ETT) on the first attempt. Maintenance of anesthesia consisted of a propofol infusion at 150 mcg/kg/min and a remifentanil infusion at 0.25 mcg/kg/min. The patient was ventilated with a 40% oxygen and 60% air mixture.

Once the patient's airway was secure, the surgeon placed a rigid laryngoscope in an attempt to visualize the surgical area. Due to the location and placement of the ETT, clear and direct visualization of the aberrant tissue was unattainable. In order to perform the surgery, a decision was made to use supraglottic jet ventilation with a respiratory rate between 10 and 20 breaths per minute. The patient was hyperventilated to decrease the end-tidal CO_2 prior to removal of the ETT. After removal of the ETT, a rigid laryngoscope was inserted and the jet ventilator was directed toward the glottic opening. The pressure used for ventilation was gradually increased from 20 psi to 25 psi until chest rise was present and ventilations continued at the set pressure. The patient's SpO_2 remained above 98% during the entire case. At the completion of the procedure, the neuromuscular blockade was antagonized with 0.6 mg of glycopyrrolate IV and 3 mg of neostigmine IV. An oral airway was inserted and the patient was mask ventilated until spontaneous respirations returned. The patient awoke with no complications and was transferred to the recovery unit in stable condition.

Discussion

Surgery of the larynx can be challenging for anesthesia providers as the airway is shared with the surgeon leaving limited airway access. There are several ventilation techniques used to control and secure the airway during surgery of the upper airway. A laryngeal mask airway (LMA), laser-safe ETT, supraglottic jet ventilation, subglottic jet ventilation, and apneic episode ventilation are methods used for ventilation during laryngeal surgery.[2-6]

An LMA is a suitable option for procedures such as a bronchoscopy. Many bronchoscopes will fit down the lumen of an LMA allowing visualization of the vocal cords without impeding ventilations.[4] The LMA is a supraglottic airway device used in many procedures and is also included in the difficult airway algorithm.[7]

Smaller sized laser-safe ETTs may be useful during microlaryngeal surgery.[5] The smaller sized ETT can provide adequate ventilation while allowing a better surgical view of the vocal cords.[5] This type of ETT does provide adequate oxygenation with minimal risks of oxygen desaturation and without elevating the $PaCO_2$ with the inspired oxygen concentration at 21% in patients with normal respiratory function.[5] The lower inspired oxygen decreases risks of airway fires during laser surgery.

Supraglottic jet ventilation is providing ventilation through a cannula that directs high pressure air through the glottic opening. A benefit of this technique is that it allows for an unobstructed view of the larynx and vocal cords.[2] In addition, the technique can be performed with either manual or mechanical jet ventilation.[2] During manual jet ventilation, pressure and flows are controlled by the practitioner. The risks with manual ventilation include: subcutaneous emphysema, pneumothorax, barotrauma, hypoxemia, and hypercarbia.[3,4] The mechanical jet ventilator can allow for inspiratory/expiratory times, driving pressures, respiratory rate, inspired oxygen content, and protection against barotrauma.[4]

Subglottic jet ventilation is performed either by inserting a special tube through the glottic opening or using a transtracheal approach. A specialized tube is inserted into the trachea by direct laryngoscopy and the cuff is inflated. This type of tube has a very small internal diameter of about 3 mm.[8] These smaller tubes can be ventilated with

either a manual or a mechanical jet ventilator. Benefits of this type of ventilation include: decreased risk of aspiration of blood or surgical debris, decreased reliance on the surgeon directing the flow of air, better visualization of the larynx compared to a standard ETT, and the ability to monitor $EtCO_2$.[8] The transtracheal approach utilizes a catheter inserted through the cricothyroid ligament. Advantages of this method are complete visualization of the larynx for surgery, but the complications of this technique include: hypercarbia, bleeding, barotrauma, pneumothorax, and the technique is more difficult to perform.[2]

The last method of ventilation discussed is apneic ventilation or intermittent ventilation. This method involves placement of an ETT, ventilating the patient to optimum oxygenation and then extubating the patient to allow surgery. The patient is monitored to resume ventilation when the SpO_2 falls to a predetermined point.[6] Complications of this technique are the patient's ability to tolerate the apnea for extended periods long enough to allow the surgeon to perform the procedure.[6] This technique does allow full view of the larynx for surgical procedures.

Supraglottic or subglottic jet ventilation is a practical option to provide ventilation for patients undergoing laryngeal surgeries. The anesthesia provider must consider the type of procedure and instruments to be used by the surgeon when developing the anesthetic plan. Safety precautions must be utilized to protect both the patient and members of the surgical team should a laser be used during the procedure.

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Regional versus Topical Anesthesia for Awake Fiberoptic Intubation

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Keywords: awake fiberoptic intubation, anesthesia, systemic lupus erythematosus

Direct laryngoscopy without in-line manual stabilization is often deemed unsafe in the patient with cervical spine trauma or disease. Alternative airway management techniques are often utilized to avoid excessive cervical spine movement and prevent further neurological injury.[1] Although awake fiberoptic intubation (FOI) is the most common method of intubation in known difficult airways and cervical spine surgery, consensus on a specific technique has yet to be determined.[2] Practitioner skill level greatly influences the particular technique utilized. This case study focuses on one technique for topically anesthetizing the airway in an awake patient with systemic lupus erythematosus undergoing cervical spine surgery.

Case Report

A 25-year-old, 49 kg, 160 cm, female presented for a C5-T2 laminectomy for resection of an intradural extramedullary schwannoma causing severe spinal cord compression. The patient's medical history was positive for mild systemic lupus erythematosus (SLE) diagnosed 3 years prior. She had an allergy to pineapple and took no medications or supplements. Physical exam demonstrated a Mallampati II airway classification. Neck mobility and range of motion were not evaluated to avoid any further spinal cord injury. Motor deficits were noted in the bilateral lower extremities; however, the left lower extremity demonstrated diminished proprioception compared to the right. Hyperreflexia, clonus, and Babinski reflex were present in bilateral lower extremities. Bowel and bladder function remained intact. Preoperative laboratory values were unremarkable. Fifteen minutes prior to entering the operating room, glycopyrrolate 0.2 mg was administered intravenously (IV) as an antisialagogue agent.

The patient was transferred to the operating room where standard monitors including noninvasive blood pressure (BP), electrocardiogram (EKG), and pulse oximeter (SpO_2) were applied. The patient's baseline vital signs were: BP = 115/81; heart rate = 90; respirations = 14; $Sp0_2$ = 100%; and normal sinus rhythm on EKG. The patient was preoxygenated with 100% O_2 via facemask and IV sedation consisted of midazolam 2mg and propofol 20mg. The patient gargled and expectorated two times, 5 minutes apart with 4% viscous lidocaine 4 ml to provide oropharyngeal topical anesthesia. An Ovassapian airway (Teleflex Medical, Research Triangle Park, NC) coated with lidocaine 5% ointment 0.5 in was introduced into the oropharynx. After 3 minutes, a LF-GP 4.1 mm flexible fiberoptic bronchoscope (Olympus American Inc., Center Valley, PA), onto which a 7.0 spiral reinforced endotracheal tube was positioned, was introduced through the Ovassapian airway and advanced into the posterior pharynx. The uvula was clearly visualized and the bronchoscope was advanced in the midline to the level of the glottic inlet. Approximately 4 mL lidocaine 4% was injected through the working port of the bronchoscope to provide topical anesthesia to the vocal cords. Two minutes were allowed to pass to permit adequate

abduction of the vocal cords. The bronchoscope was advanced atraumatically through the vocal cords and positioned 2 cm above the carina. The endotracheal tube was advanced over the bronchoscope, through the vocal cords, and into the trachea. Correct placement of the endotracheal tube was confirmed bronchoscopically as well as by the presence of $EtCO_2$ on the anesthesia gas monitor.

After performing a neurological assessment, no new deficits were observed. Induction of general anesthesia was performed with propofol 100 mg, ketamine 100 mg, and vecuronium 10 mg IV. Dexamethasone 50mg IV was administered per the surgeon's request as an anti-inflammatory agent for severe spinal cord compression. The patient was positioned prone while maintaining the cervical spine in the midline position. General anesthesia was maintained by propofol infusion at 150 mcg/kg/min and end-tidal desflurane 2.5%. The procedure was completed without difficulties and the patient was extubated awake after verifying that neurological function was intact and an endotracheal cuff leak was present.

Discussion

Establishing a secure airway in the neurosurgical patient with cervical spine trauma or disease demands thoughtful consideration.[1] Cervical spinal cord damage is a significant risk during intubation for this patient population and must be mitigated with the appropriate anesthetic technique.[3] Many methods of airway management are available for patients with known difficult airways and those requiring cervical spine surgery. However, utilizing an awake fiberoptic intubation technique is the most common.[2] Consensus on a specific technique remains a topic of debate within the anesthesia community and decisions must be made as to sedation, which route to use, and the method of providing topical or regional anesthesia to the airway.[4] In some circumstances, pharmacological adjuvants may eliminate the need for topical or regional anesthetics altogether. Dexmedetomidine and remifentanil have been successfully utilized for this purpose. However, for the purpose of this case study, the efficacy of topical versus regional anesthesia was specifically evaluated. Much of the current literature regarding awake fiberoptic techniques focuses on case studies and observational cohort data. However, there are limited controlled prospective studies that compare different techniques.[2]

Although the patient's anatomical neck landmarks were easily discernible and a regional nerve block was not contraindicated, topical anesthesia was selected primarily due to the anesthesia team's preference and comfort level. Topical anesthesia has been shown to be a safe technique with low risk of systemic lidocaine toxicity while sufficiently suppressing airway reflexes.[2] A review article by Simmons and Schliech found the combination of topical anesthesia and a method to anesthetize the laryngeal/tracheal structures to be the most effective plan for an awake FOI.[4] The topical anesthesia in this case was performed by gargling viscous lidocaine 4%, the application of lidocaine 5% ointment to the oral airway, and direct spray of lidocaine 4% to the vocal cords, which obliterated the patient's cough and gag reflexes during the FOI. The toxicity and total dose of lidocaine administered was also taken into consideration. Although the total dose of 505mg administered exceeds the toxic dose threshold of 245mg for the patient's weight, the patient expectorated the majority of the gargled viscous lidocaine and systemic absorption via the gastric mucosa is limited by first-pass hepatic

metabolism. While the topical technique in this case was successful, a regional nerve block technique may have also been effective.

Unfortunately, there is only one study, published in 1995, comparing a topical anesthetic technique to a regional nerve block technique. Reasoner et al compared twenty-one subjects who received inhaled nebulized lidocaine 4% and a transtracheal injection of lidocaine 4% against nineteen subjects who received glossopharyngeal and superior laryngeal nerve blocks with 4% lidocaine in addition to a transtracheal injection of lidocaine 4%.[5] The quality of the anesthetic was graded on the severity of coughing and gagging during intubation by a blinded observer. No significant difference in quality or safety profile was observed between the two groups.[5] While nerve blocks provide excellent anesthesia, they are technically more difficult and inherently have a higher risk of complication due to the invasive nature of the technique when compared to topical anesthesia.[4] Intravascular injection, nerve damage, and bleeding are all possible complications associated with a nerve block while noninvasive methods carry the risk of local anesthetic toxicity.[4] Maintaining spinal alignment after transtracheal block may also be problematic as coughing is expected and helps spread the local anesthetic.

This patient also had a history of systemic lupus erythematous. SLE has been recognized for over fifty years and has demonstrated laryngeal complications ranging from 0.3% to 30%. Some studies suggest that post intubation subglottic stenosis, laryngeal edema, and acute airway obstruction may be more common in patients with active SLE.[6] Although the patient was diagnosed with mild SLE and was asymptomatic, potential postoperative SLE related sequelae were still contemplated and an endotracheal tube cuff leak test was performed prior to extubation to ensure adequate passage of air.

There are multiple methods available to anesthetize the airway during an awake FOI. However, the anesthetist must assess his or her own skill level as well as the risks and benefits of each technique and develop a plan that provides for safe airway management.[4] Concerns about potential complications with regional nerve blocks may prompt anesthetists to choose an alternative method such as topical anesthesia. The differences in the anesthetist's skill level and a significant lack of research in this area may make topical anesthesia the safest choice for anesthetizing the airway in the patient requiring an awake FOI. Additional consideration must be given when choosing a specific anesthetic technique in patients with active SLE. Further research is needed to evaluate the relative safety profiles, quality of anesthesia, and risks and benefits of each technique.

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Anesthesia for Cesarean Section in a Patient with Hypochondroplasia Dwarfism

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Keywords: Cesarean section, hypochondroplasia dwarfism, neuroaxial anesthesia,
general anesthesia**,** subarachnoid block

Hypochondroplasia dwarfism with a prevalence of approximately one in 15,000 to 40,000 live births is one of the more common subtypes of the greater than 100 different classifications of dwarfism with a prevalence of approximately one in 15,000 to 40,000 live births.[5-7] Physical characteristics of the anomaly present numerous anesthetic challenges for both neuroaxial and general anesthetic techniques without clear evidence-based guidelines. Hypochondroplasia parturients exhibit normal female fertility function, but have an increased incidence of both planned and emergent cesarean sections due to elevated rates of cephalopelvic disporportion.[4,5] This case report describes the anesthetic management for a patient with hypochondroplasia dwarfism undergoing an elective cesarean section.

Case Report

A 21-year-old primigravida presented for scheduled primary cesarean section at 39-2/7 weeks for cephalopelvic disproportion. The female patient was 142 cm in height and 72 kg in weight. Her significant past medical history included seasonal allergies and hypochondroplasia dwarfism. Her only medication was a prenatal vitamin. The patient presented to the anesthesia professionals the morning of the planned cesarean section. The physical exam was significant for macrocephaly, Mallampati grade 2, mild joint laxity, disproportionately short arms and legs, and lumbar lordosis. Hemoglobin, hematocrit and platelets were 9.4 g/dL, 28%, and 175 x 10^9/L, respectively. ASA class 3 was assigned. Subarachnoid block was planned with difficult airway supplies available on standby.

The patient was administered lactated ringers one liter, famotidine 20 mg and metoclopramide 10 mg intravenously. Following transport to the operating room the patient was assisted to the sitting position on the operating table. Standard monitors were applied and oxygen was administered via nasal cannula at two liters per minute. Sterile technique was maintained throughout the subarachnoid block; an introducer needle was placed followed by 25 gauge pencil-point spinal needle between L3 and L4 vertebrae requiring one redirection. The subarachnoid space was identified, clear free flow of

cerebrospinal fluid was observed with no paresthesias reported. Bupivacaine 0.75% in 8.25% dextrose 1.3 mL (9.75 mg) and fentanyl 15 mcg were administered into the subarachnoid space. The patient was repositioned supine with assistance and placed in left lateral tilt position.

The subarachnoid block was assessed to be at the T2 dermatome prior to surgical incision. The support person was brought to the operating room and the cesarean section proceeded uneventfully. Birth of a 3.4 kg viable newborn ensued with 1 and 5 minute APGAR scores 9 and 9, respectively. Oxytocin 10 units was added to continuous intravenous infusion of lactated ringers. Surgical closure was completed with estimated blood loss 450 mL. The patient required two doses of ephedrine 5 mg intravenously following onset of subarachnoid block and was otherwise hemodynamically stable throughout the duration of anesthesia and surgery.

The patient was transferred to the Post Anesthesia Care Unit (PACU) with T8 dermatome sensory block. Bilateral ultrasound guided transverse abdominal plane (TAP) blocks were completed in PACU under sterile technique utilizing a 21 gauge 4-inch stimulating needle. Ropivicaine 0.25% 25 ml was administered bilateral via incremental aspiration.

Discussion

Hypochondroplasia dwarfism is commonly identified as an autosomal dominant mutation in the Fibroblast Growth Factor Receptor 3 (FGFR3) gene resulting in skeletal dysplasia, related to unaffected periosteal bone formation with diminished endochondral ossification.[1,5,6,8] Characteristics of the disease include disproportionately short arms and legs, macrocephaly, short stature less than 148 cm, thoracolumbar kyphosis, narrowing of lumbar interpedicular distance, spinal stenosis, central sleep apnea and obstructive apnea [1,3,5] These clinical manifestations predict difficultly with both neuroaxial anesthesia and airway management. More specific airway characteristics include craniofacial abnormalities, limited neck extension, foramen magnum stenosis, macroglossia, macrognathia, and atlantoaxial instability.[7] In this case study the patient exhibited macrocephaly, mild joint laxity and physical characteristics of FGFR3 mutation.

Neuroaxial techniques utilized as a primary anesthetic for the hypochondroplasia patient present several challenges. Technical difficulties stem primarily from alternations in anatomy, including difficulty in palpation of the lumbar vertebra processes, lumbar lordosis, scoliosis, narrowed lumbar interpedicular distance and spinal stenosis.[4,5] Furthermore it is not uncommon for this population to have undergone prior lumbar laminectomy.[3,5] When a single dose subarachnoid block for neuroaxial anesthesia is utilized local anesthetic spread is unpredictable.[3,7] Factors influencing the dose of intrathecal bupivacaine include height, weight, pregnancy and spinal anatomy. There is limited evidence base for intrathecal dosing for the parturient with dwarfism with recommendation of 30% reduction from standard dosing in the achondroplasia dwarfism.[3] The current evidence supports standard cesarean section intrathecal dosing of 0.06 mg/cm height for 0.5% hyperbaric bupivacaine as the minimum effective dose.[2] Utilizing this calculation for this case study the appropriate dosing would have been hyperbaric bupivacaine 0.5% 1.7 mL (8.5 mg) in comparison to the hyperbaric bupivacaine 0.75% 1.3 mL (9.75 mg)

actually administered. The factors influencing this dosing in clinical practice included patient height, pregnancy, lumbar lordosis, expected length of surgical procedure and practitioner team preference.

There are no clear outlines in the current literature to describe neuroaxial technique dosing in the parturient with dwarfism. In retrospect, utilization of a combined spinal epidural (CSE) technique could have provided the advantage of administering a lower dose of local anesthetic into the subarachnoid space allowing for slow and controlled titration of local anesthetic in the epidural space if T4 dermatome block was not achieved with intrathecal dosing. Thus the risk for high or total subarachnoid block may have been decreased. Yet, as it has been described in the literature, the placement of epidural catheter may be technically challenging and potentially impossible as the epidural space may not even be existant.[3,4,7] Therefore epidural anesthesia is not without risk of subarachnoid catheter placement or migration. Therefore, if a neuroaxial technique is determined to be the technique of choice, the potential for inadequate or high level of anesthesia must be earnestly considered.

General techniques have been described in the literature for use in the event of failed regional and emergency cesarean section in this population.[3,4,7] One of the greatest concerns for the parturient with hypochondroplasia dwarfism undergoing general anesthesia is airway management, as a rapid sequence induction (RSI) is indicated in the presence of a difficult airway.[4] Awake fiberoptic intubation is prudent airway management technique of choice in this patient population. Case studies in the literature describe both successful and unsuccessful RSI airway management. If RSI is planned than considerations surrounding the potential for hypochondroplasia related alteration in pharmacokinetics should be considered as inadequate induction dosing has resulted in a failed airway.[8] The alterations in pharmacokinetics is hypothesized to be due to hypochondroplasia leading to alterations in body organ proportions resulting in an overall higher metabolic requirements from the larger percentage of vessel-rich organ groups when compared to moderate and low energy requiring systems.[8] In addition, the anesthesia professionals need to implement the difficult airway algorithm with judicious consideration for the appropriate utilization of awake fiberoptic intubation. As a result, in the case described the anesthesia professionals were prepared with difficult airway cart, fiberoptic bronchoscope and local anesthetic for topical airway application throughout the course of the anesthetic.

The literature currently does not favor administration of a neuroaxial or general technique in the dwarf population presenting for cesarean section nor does it outline clear neuroaxial dosing recommendations. The research does however demonstrate that this is a high risk obstetrical population. In reflection of this case study the patient demonstrated successful single dose subarachnoid block may have been better managed with a general anesthetic. The plan for airway management would have been awake fiberoptic intubation to allow for safe control of the predicted difficult airway. Overall neuroaxial techniques and general anesthesia are both reported options in the literature for anesthetic care of the hypochondroplasia parturient as long as vigilant anesthetic practice is maintained and potential difficulties are anticipated. Further research is necessary to develop evidence based practice guidelines for

anesthetic care of the hypochondroplasia parturient.

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Anesthetic Adjuncts in a Neurosurgical Patient

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Keywords: neurosurgical patient, opioid abuse, dexmedetomidine, remifentanil, ketamine.

Significant consideration for hemodynamics is required when planning the anesthetic management of the neurosurgical patient. This case report describes the use of anesthesia adjuncts in a 28 year-old female with a history of Arnold Chiari Malformation Type I (ACM-I). This patient underwent suboccipital craniotomy previously, returning four years later with recurrent symptoms of headache and numbness to her hands and feet. Follow up magnetic resonance imaging showed evidence of re-impaction of ACM-I at the level of the foramen magnum. The patient was scheduled for and underwent suboccipital craniotomy for decompression of the recurrence of ACM-I.

Case Report

A 28-year-old female presented to surgery for a suboccipital crainiotomy for decompression of recurrent ACM-I. The patient was 160 cm in height and 77 kg in

weight with no known allergies. Her history included eleven pack years of smoking tobacco, hepatitis C, intravenous substance abuse, anxiety, depression, attention deficit hyperactivity disorder (ADHD), and obsessive compulsive disorder (OCD). In addition to the suboccipital craniotomy four years prior, surgical history consisted of posterior cervical diskectomy, cesarean section, and craniocervical junction surgery. The patient had no previous anesthesia complications. Daily medications included acetaminophen-diphenhyramine, levonorgestern and buprenorphine-naloxone with the last dose of buprenorphine-naloxone twenty-four hours prior. Complete blood cell count, blood chemistry, lung sounds, chest x-ray, and electrocardiogram were normal and liver enzymes elevated.

Preoperative vital signs included: blood pressure 114/63 mmHg; pulse, 62 /min; respiratory rate, 20/min; room air oxygen saturation 96%; temperature 35.9^{o}C. The patient was consented for general anesthesia with an intravenous (IV) induction. A 20-gauge IV catheter and left radial arterial line were inserted.

After receiving IV midazolam 2 mg, the patient was brought to the operating room and positioned supine with the head placed in a neutral position, monitors were placed and measures were: blood pressure 110/55 mmHg; heart rate, 73/min; respirations, 12/min; oxygen saturation, 99%; electrocardiogram showing sinus rhythm. After three minutes of oxygen administered at 10 L/min by mask, a smooth induction was achieved with IV sufentanil 50 mcg, lidocaine 100 mg, etomidate 20 mg, and rocuronium 50 mg. Tachycardia and hypertension during intubation were treated with IV esmolol 80 mg and nitroglycerin 50 mcg to minimize any related increase in intracranial pressure. An atraumatic tracheal intubation was achieved on the first attempt with a number three Macintosh blade. A cuffed endotracheal tube was placed and secured at 20 cm at the lips. The patient was placed on volume controlled ventilation at 13/min, tidal volume 537 ml and peak inspiratory pressure 17 cm H2O with an end tidal carbon dioxide 30-32 mmHg. After induction, the blood pressure was 100/70 mmHg, heart rate 81/min, and vital signs were maintained within 20% of preoperative measurements. After a second IV catheter and foley catheter were inserted, the patient was positioned prone on the operating room table with neck positioned neutral, the table was turned 180 degrees toward the surgeon and a lower body warming blanket was applied.

During the surgery, neuromuscular blockade, urine output, temperature and vital signs were monitored. General anesthesia was maintained with sevoflurane at an expired concentration of 1.5%, remifentanil infusion 0.5 mcg/kg/min, and dexmedetomidine infusion 0.5 mcg/kg/hr. Ketamine 15 mg IV was also administered, along with rocuronium 70 mg, sufentanil 40 mcg, phenylephrine 120 mcg, ephedrine 5 mg, ondansetron 4 mg, and decadron 4 mg during the case. The patient received a total of 1700 mL 0.9% sodium chloride, remifentanil 5.8 mg, dexmedetomidine 86.38 mcg, and ketamine 15 mg.

After the procedure was completed, neuromuscular blockade was antagonized with neostigmine 5 mg and glycopyrrolate 0.8 mg. The endotracheal tube was removed once the patient met extubation criteria of a tidal volume 350 ml and sustained head lift times 5 seconds. She was transported to the postanesthesia care unit without complications.

Discussion

The pathophysiology of the neurosurgical patient with a diagnosis of ACM-I combined with a history of opioid abuse currently taking buprenorphine-naloxone is multifactorial. In this situation, anesthesia management was tailored to decrease the incidence of increased intracranial pressure due to increased stimulation. A current literature review discusses the use of anesthetic adjuncts to control hemodynamics in the neurosurgical patient.

ACM is categorized into four subtypes depending on the degree of herniation of the cerebellar structures through the foramen magnum.[1] ACM-I involves herniation in the cerebellar tonsils and can include syringohydromyelia, hydrocephalus and skull base alterations.[1] ACM-I has been associated with psychiatric symptoms and brainstem nuclei compression in the locus coeruleus, which may be responsible for her anxiety, depression, ADHD, and OCD comorbidities.[1] In addition, psychiatric symptoms and a history of opioid dependence may have further contributed to the patient's hyperresponsiveness to pain and surgical stimulation with the risk of hemodynamic instability. No literature was found on surgical recommendations for continuing or discontinuing buprenorphine-naloxone perioperatively and further studies are suggested. Orman et al. discuss buprenorphine-naloxone use in treatment of opioid dependence.[2] Buprenorphine is a partial mu-opioid receptor agonist well established for an alternative treatment for opioid dependence and with the addition of naloxone, an opioid antagonist.[2] A decrease in the IV abuse liability of the partial opioid agonist is expected by blocking or attenuating the effects of other opioid agonists such as morphine, heroine, in a dose-related manner for at least 25 hours.[2] Buprenorphine-naloxone's elimination half-life from plasma is 37 hours and 1.1 hours respectively.[2] Pharmacological agents like dexmedetomidine, remifentanil, sufentanil, ketamine, esmolol, and nitroglycerin, which were all used in this case contributed to the return of the patient's hemodynamic stability.

The anesthetic goals for this neurosurgical patient were to maintain stable hemodynamics without increasing the incidence of increased intracranial pressure which could have led to intracranial hemorrhage and prolonged hospital stay.[3] Mechanisms responsible for increased intracranial pressure are complicated by hypertensive episodes and dexmedetomidine is now being used to improve hemodynamic stability during stimulating periods of surgery.[3] Dexmedetomidine is an alpha 2 adrenoreceptor agonist and its sympatholytic and antinociceptive properties are effective for blunting the increase in systolic blood pressure.[3] Bekker et al. showed statistical evidence the dexmedetomidine group had fewer hypertensive episodes and were discharged earlier.[3] Panzer et al. described the neuroprotective effects of dexmedetomidine including sympatholysis, preconditioning, and attenuation of ischemia-reperfusion injury as well as decreases in cerebral blood flow.[4] Drown discusses the unique properties of dexmedetomidine including analgesic-sparing and, sympatholytic effects, both reducing opioid requirements and reducing the stress response to surgery, minimizing hemodynamic responses.[5] Grof et al. reported dexmedetomidine as an effective sedative agent but due to the small sample size suggested further research for doses and durations for hemodynamic monitoring.[6] According to Upadhyay et al. dexmedetomidine has been successfully

used as a sedative and analgesic medication.[7]

In this case study, a low analgesia dose of ketamine 15 mg was administered. Ketamine's use in neurosurgery is limited due to its association with increased intracranial pressure. However, Bowles et al. discusses a review of recent clinical literature suggesting ketamine's use as an adjuvant anesthetic agent.[8] Along with mechanical ventilation to maintain normocapnia, Bowles et al. reports with the use of ketamine, ICP remains stable with benign cerebrovascular effects.[8] Anesthesia practitioners may use ketamine as an anesthetic adjuvant with caution in neurosurgical patients and ketamine should not be considered an absolute contraindication due to its properties that may antagonize both neuroprotective and neurodestructive N-methyl-D-aspartate receptor-mediated pathways.[8] The analgesia properties of ketamine may have provided optimal surgical conditions for this procedure by blunting the sympathomimetic response.

Much consideration should be given to maintaining hemodynamic stability for the neurosurgical patient. In this case report, the use of anesthetic adjuncts such as dexmedetomidine and ketamine resulted in stable hemodynamics without signs of increased intracranial pressure. In conclusion, it is vital for the anesthesia practitioner to remain vigilant in assessing hemodynamic changes in a neurosurgical patient. The practitioner must consider numerous factors when formulating a plan of care to provide safe intraoperative management of the neurosurgical patient.

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Mentor: Lisa J. Hogan, CRNA, DNP

Anesthesia Workstation Preparation for a Malignant Hyperthermia Susceptible Patient

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Keywords: Malignant Hyperthermia; Activated Charcoal; Anesthesia; Vapor Free, Preparation

Malignant hyperthermia (MH) is a rare and potentially lethal "pharmacogenetic disorder of skeletal muscle that presents as a hypermetabolic response" to volatile anesthetics and succinylcholine.[1,2] Hallmark signs of MH include muscle rigidity, hypercarbia, tachycardia, tachypnea, acidosis, and hyperthermia. MH susceptible patients should receive anesthesia from a dedicated anesthesia machine free of triggers. This "clean" machine should have all vaporizers removed and all parts saturated with volatile anesthetic replaced.[3] If this cannot be accomplished, the machine should be "flushed with a high-flow fresh gas for 10 to 104 minutes so that an acceptable concentration of <5 parts per million (ppm) is reached."[1,3]

Case Report

The patient was a 37-year-old obese woman who was 165 cm tall, weighed 98 kg, and had a calculated body mass index of 35.26 kg/m^2. She was scheduled to undergo robotic assisted laparoscopic-assisted vaginal hysterectomy for severe cervical dysplasia. The patient's complex medical history included obesity and a familial history of malignant hyperthermia. The patient's father and paternal uncle were both diagnosed with MH using the caffeine-halothane contracture test, the only recognized laboratory test to diagnose MH.[4] The patient had no known drug allergies and did not take any medications. Her physical examination and laboratory values were insignificant. Her surgical history included a loop electrosurgical excision procedure and right knee arthroscopy. She had nothing to eat for 8 hours prior to the surgery.

The patient's history of MH was of utmost concern. To prepare for this anesthetic, the patient's surgery was scheduled to be the first case of the day. Additionally, all operating room (OR) staff members were made aware of the patient's history, precautions, and emergency treatment for MH. The anesthesia machine was prepared using current recommendations from the Malignant Hyperthermia Association of the United States (MHAUS).[5] All vaporizers were removed from the anesthesia machine. The carbon dioxide (CO_2) absorbent and breathing circuit were replaced. O_2 flowed at 15 L/min through the anesthesia workstation for 12 hours, and before the case, the CO_2 absorbent and breathing circuit were replaced again. Dantrolene sodium, the only specific antagonist of MH, was immediately available in the MH cart that was placed outside the operating room suite as well.[1,2]

Preoperatively, midazolam 5 mg intravenous (IV) was administered and the patient was taken to the operating room suite and transferred herself to the OR table. Standard monitors were applied and the patient was preoxygenated for 5 minutes before induction. Induction commenced with the administration of fentanyl 150 mcg, lidocaine 80 mg, and propofol 160 mg. After 2 minutes of easy mask ventilation, rocuronium 40 mg, was administered to facilitate tracheal intubation. Direct laryngoscopy was performed 2 minutes after the administration of rocuronium with a

Macintosh blade size 3. A 7.0 mm oral endotracheal tube was successfully placed, with subsequent verification through end-tidal carbon dioxide tracing on the capnogram and equal bilateral breath sounds upon auscultation. Respirations were controlled using mechanical ventilation and maintenance of anesthesia was achieved via a propofol infusion 150 mcg/kg/min. Two boluses of fentanyl 50 mcg were administered intra-operatively for analgesia. Surgery proceeded and was uneventful. Upon emergence, neostigmine 3 mg, glycopyrrolate 0.4 mg, ondansetron 4 mg, and ketorolac 30 mg were administered. The patient emerged from anesthesia successfully with spontaneous ventilation and adequate tidal volumes. After the patient responded to commands to lift her head for five seconds, she was extubated and continued spontaneous ventilation with O_2 via facemask successfully. The patient was then transferred to the post anesthesia care unit where care continued without any untoward events. No signs or symptoms of MH were noted throughout the case and the patient had a successful surgery.

Discussion

The current recommendations for providing anesthesia to the MH susceptible patient are twofold: one, avoid triggering agents such as succinylcholine and volatile anesthetic agents (VAAs); and two, provide anesthesia using a "clean" anesthesia machine.[1-5] Although the first part of this is easily managed by the anesthesia professional, the later is not as simple.[1,3] Specifically, to provide anesthesia on an anesthesia machine that has not been exposed to VAAs is both unfeasible and cost-prohibitive. However, the general consensus is to reduce the level of exposure to VAAs to under 5 ppm.[1,3-6] In order to accomplish this, precautions must be taken to ensure that the anesthesia machine is adequately cleansed from retained VAAs. Unfortunately many of the internal breathing components in modern anesthesia machines utilize more plastic and rubber parts than older traditional machines. As a result, a significant reservoir exists that retains VAAs and therefore are particularly difficult in "cleaning".[1,3,5] The current recommendation for delivering a "clean" anesthetic that is free from VAAs or meeting the guideline of being under 5 ppm can be accomplished by a variety of methods. The most commonly employed technique is to remove or disable vaporizers, flush the machine with high-flow fresh gas greater than or equal to 10L/min using the ventilator for at least 20 minutes, replace the fresh gas outlet hose, CO_2 absorbent, and breathing circuit.[1] For this case study, the above recommendations were followed in entirety and no adverse events concerning MH took place for the patient.

Unfortunately, a review of the literature has shown that these "current" recommendations for preparing the anesthesia machine for an MH susceptible patient are outdated. The current guidelines were developed for older anesthesia machines that utilized simpler internal breathing circuitry, machines that were free from the highly soluble reservoirs for VAAs that are replete in modern machines.[1,3] Concerning the preparation of modern anesthesia machines for MH susceptible patients, the latest studies show that a much more time and labor intensive process must be utilized to adequately remove all the residual VAAs that constitute the reservoir replete in modern anesthesia workstations.[1] In fact, the studies clearly demonstrate that in order to maintain a safe level of VAAs that will not trigger an MH reaction, specifically under 5 ppm, modern machines should undergo washout with high-flow fresh gas at 10 L/min for a minimum of 122 minutes.[7] It should be

noted that this minimum of 122 minutes flush with high-flow fresh gas is assuming that the ventilator diaphragm and ventilator hose are autoclaved.[7] Without meeting this step, the time increases to a minimum of 151 minutes. Regardless of time spent in preparing the anesthesia machine in washout, the latest studies also point out a common theme, the rebound effect.[1,3,6-7] The rebound effect occurs when modern anesthesia machines that have been undergoing a high-flow flush with fresh gas to meet the low anesthetic concentrations of 5 ppm or lower have the flow reduced to under 10 L/min. When flow is reduced, a surge in anesthetic concentration occurs. All the current studies conclude that in order to keep the anesthetic triggering agents under the concentration of 5 ppm, high-flow fresh gas of at least 10 L/min must be maintained for the entire anesthetic.[1,3,6-7] Unfortunately, this can have deleterious effects in itself to the patient.

Fortunately, an alternative has been proposed. Birgenheier and colleagues discovered a novel approach to dealing with the complex challenge of preparing modern anesthesia machines for the MH susceptible patient that truly ensures safety, namely, a concentration of VAAs under 5 ppm for the duration of an anesthetic.[3] In their landmark study, activated charcoal filters were placed on both the inspiratory and expiratory limbs of multiple anesthesia machines that had been exposed to VAAs. Using infrared spectroscopy, the inspired concentrations of VAAs were measured and recorded every 45 seconds to determine the time until the delivered concentration of a particular VAA was under 5 ppm.[3] Unique to this study was the finding that modern anesthesia machines could be "cleaned" in less than 90 seconds when using activated charcoal filters on both inspiratory and expiratory limbs. Another useful innovation of using activated charcoal filters is that in the event of an MH crisis that is triggered intra-operatively, the anesthesia professional could quickly reduce the level of VAAs below the triggering threshold of 5 ppm while meeting the MHAUS recommendation to not change breathing circuit and CO_2 absorbent in place of treating MH with dantrolene sodium.[3]

In summary, the patient in this case study could have been more effectively and safely managed with the use of activated charcoal filters on the anesthesia machine. Although the patient did not suffer any adverse effects from the anesthetic delivered using the modern anesthesia machine described herein, this outcome is anecdotal evidence to support the use of guidelines that were developed using older anesthesia machines. Going forward, when modern anesthesia machines will be used for the MH susceptible patient, preparation should include the use of activated charcoal filters.

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Mentor: Mark D. Welliver, CRNA, DNP, ARNP

Editorial

Happy Holidays to everyone! I would like to point out that this is the largest issue I have released since taking over for Ron Van Nest (founder of the ISJNA) about 5 years ago. This is clearly evidenced by the extensive list of reviewers for this issue, to whom I am grateful for their hard work. Without our reviewers and editorial board this journal could not exist, so thank you all for your dedication and time.

This issue contains a good variety of reports spanning across many specialty areas, including pediatric, obstetric, cardiothoracic, and neuro-anesthesia. Rare, but potentially life-threatening topics are covered, as well as unusual syndromes and conditions. I hope you will find this interesting reading during your holiday break. As always, but it bears repeating, we appreciate your comments and do accept Letters to the Editor for publication.

Please enjoy a safe, heartwarming holiday season!

Best,

Vicki C. Coopmans, CRNA, PhD
Editor

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

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INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA
GUIDE FOR AUTHORS

MISSION STATEMENT

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

ITEMS ACCEPTED FOR PUBLICATION

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

ITEM PREPARATION & SUBMISSION

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

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

PEER REVIEW

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

General guidelines

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

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

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

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

<u>Case Reports</u>

The student author must have had a significant role in the conduct of the case. The total word count should be between 1200 – 1400 words. References do not count against the word count. Case reports with greater than 1400

words will be returned to the mentor for revision prior to initiation of the review process. The following template demonstrates the required format for case report submission.

Heading (see #9 above in General Guidelines)

[space]

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

[space]

Case Report (bold, 400-500 words)

[space]

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

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

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

[space]

Discussion (bold, 600-800 words)

[space]

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

[space]

References (bold)

[space]

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

[space]

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

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

Research Abstracts

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

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

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

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

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

Heading (see #9 above in General Guidelines)
[space]
Introduction (bold)
[space]
Briefly introduce the reader to the practice issue or controversy, describe the scope or significance or problem, and identify the purpose of your analysis. Describe the theoretical, conceptual, or scientific framework that supports your inquiry.
[space]
Methodology (bold)
[space]
Include the format used for formulating the specific question you seek to answer, search terms and methods used, and levels of evidence.
[space]
Literature Analysis (bold)
[space]

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

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

AMA MANUAL OF STYLE

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

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

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

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

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

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

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

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

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

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

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

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

Author (or if no author, the name of the organization responsible for the site). Title. *Name of journal or website*. Year;vol(issue no.):inclusive pages. doi: or URL. Published [date]. Updated [date]. Accessed [date].

For online journals, the accessed date may be the only date available, and in some cases no page numbers.

Examples:
Kamangar N, McDonnell MS. Pulmonary embolism. *eMedicine*. http://www.emedicine.com/med/topic1958.htm. Updated August 25, 2009. Accessed September 9, 2009.

Gupta A, Aggarwal N, Sharma D. Ultrasound guided ilioinguinal block. *The Internet Journal of Anesthesiology.* 2011;29(1).
http://www.ispub.com/journal/the_internet_journal_of_anesthesiology/volume_29_number_1/article/ultrasound-guided-ilioinguinal-block.html. Accessed August 1, 2011.

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

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

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SUBMISSION CHECK LIST

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

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

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

Abstract
___ The 500 word count maximum is not exceeded.
___ Abstract reports the *outcome* of your study.
___ Includes Introduction, Methods, Results, and Conclusion sections.

EBPA Report
___ The 3000 word count maximum is not exceeded.
___ A critical evaluation of a practice pattern in the form of a precise clinical question about a specific intervention and population is presented.
___ A focused foreground question following either the PICO or SPICE format is used.
___ Includes Introduction, Methodology, Literature Analysis, and Conclusion sections.

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___ AMA Style for referencing is used correctly.
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