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TOPICS IN THIS ISSUE

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Front Cover:
Jennifer Link, MSN, a graduate student enrolled in the Wake Forest Baptist Health Nurse Anesthesia Program, learns how to suture while on a mission trip to the Dominican Republic. Besides nurturing unique clinical skills and a philanthropic spirit, global health experiences typically foster interprofessional education.

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Case Reports

Wolff-Parkinson-White Syndrome: Implications for Anesthesia

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Keywords: Wolff-Parkinson-White syndrome, anesthesia, atrioventricular nodal reentrant tachycardia.

Wolff-Parkinson-White syndrome (WPW) is characterized by the presence of accessory conduction pathways in the heart and occurs in approximately 0.3% of the population.[1] Symptomatic WPW is generally treated with radiofrequency catheter ablation (RFCA) with a 95% success rate.[1] Not only do anesthesia practitioners encounter patients with WPW during electrophysiological (EP) studies and RFCA, they also encounter patients with unsuccessful RFCA treatment and those who receive antiarrhythmic drug therapy. Symptoms of WPW can manifest for the first time intraoperatively.[1-3] An understanding of relevant pathophysiology and anesthesia implications is critical when caring for these patients.

Case Report

A 39-year-old, 163 cm, 49 kg female with WPW presented for breast augmentation and abdominoplasty. The patient had an orthodromic (narrow) QRS presentation of WPW and had undergone unsuccessful RFCA 11 years earlier. As a young adult, she experienced occasional symptomatic tachyarrhythmias that caused palpitations and fainting, for which she took propranolol as needed. She had uneventful vaginal deliveries and no other significant medical history. There were no significant findings on physical examination and lab results were within normal limits. Her preoperative 12-lead electrocardiogram (ECG) tracing showed WPW preexcitation with ectopic ventricular beats in sinus rhythm. Drugs to manage orthodromic tachyarrhythmias associated with WPW, specifically adenosine and β-blockers, were available throughout the perioperative period. A defibrillator was in the room but pads were not placed on the patient.

Prior to induction of general anesthesia, the patient was given midazolam 2 mg intravenously (IV) for anxiolysis and fentanyl 100 mcg IV for analgesia and to blunt the sympathetic response to direct laryngoscopy (DL) and intubation. General anesthesia was induced with lidocaine 50 mg IV, propofol 100 mg IV, and succinylcholine 75 mg IV, and maintained with isoflurane 1.2% inspired concentration in a mixture of oxygen 0.75 L/min and air 0.75 L/min. During the surgery, the patient was given vecuronium for neuromuscular blockade to keep train of four less than two out of four twitches per surgeon request. She was also given fentanyl 150 mcg IV for analgesia, dexamethasone 4 mg, and ondansetron 4 mg IV for post-operative nausea and vomiting prophylaxis. Near the end of the case and after the return of two strong twitches, neuromuscular blockade was antagonized with incremental administration of glycopyrrolate 0.5 mg IV and neostigmine 2.5 mg IV.

Throughout the case, the ECG was monitored for premature atrial contractions (PACs) and premature ventricular contractions (PVCs), both known triggers of tachydysrhythmias associated with WPW.[1-2] No arrhythmias or ECG changes were noted. The anesthetic course of induction, maintenance, and emergence occurred

without incident. The patient was recovered from anesthesia and discharged.

Discussion

Wolff-Parkinson-White syndrome occurs when congenital accessory atrioventricular (AV) conduction pathways bypass normal conduction through the AV node, exciting the ventricles sooner. The congenital accessory pathways (CAPs) manifest on ECG as a short PR interval and an early deflection, a delta wave, before the QRS complex. CAPs are likely remnants of incompletely developed fetal AV tissue and are capable of conducting impulses in one or both directions, thus creating the potential for "reentrant" tachycardias when retrograde conduction occurs.[1-3] Wolff-Parkinson-White syndrome manifests in three types of tachyarrhythmias. Both orthodromic (narrow-QRS) atrioventricular reentrant tachycardia (AVNRT) and antidromic (wide-QRS) AVNRT are types of paroxysmal suprvaentricular tachycardias (PSVT) occuring through the AV node; preexcited atrial fibrillation (AF) is the third tachyarrythmia to be discussed below.

Orthodromic atrioventricular nodal reentrant tachycardia (AVNRT) occurs in 90-95% of patients and is characterized by rapid, regular, narrow-QRS complexes resulting from antegrade conduction through the normal AV nodal pathway that return retrograde via a faster CAP from the ventricle to the atrium. Treatment is targeted at interrupting the reentract circuit at the AV node. Adenosine IV dramatically slows AV node conduction, terminating any tachycardia dependent on this reentry pathway; caution is advised since adenosine also enhances automaticity and can induce AF. Verapamil is effective at terminating PSVT in 80% of adult cases of orthodromic AVNRT by slowing phase IV depolarization in the AV node without effecting the refractory period. Verapamil can exacerbate PSVT by deteriorating into preexcited AF with rapid ventricular response (RVR). β-blockers such as esmolol are less effective than adenosine and verapamil at terminating PSVT. Amiodarone is not generally used for acute conversion given the relatively slow onset.[3]

Antidromic AVNRT is characterized by a wide QRS. These impulses are conducted antegrade via the faster CAP, then return from the ventricles via the normal AV nodal pathway to reexcite the atria. The goal of treatment for antidromic (wide-QRS) AVNRT is to slow conduction via the CAP. The same drugs used to slow AV nodal conduction in orthodromic AVNRT (adenosine, verapamil, β-blockers) are not effective in treating antidromic AVNRT because they may increase forward conduction along the CAP thus increasing ventricular rate. Stable antidromic AVNRT should be treated with class IA antiarrhythmics such as procainamide, which profoundly lengthen the CAP refractory period to slow the ventricular response terminating the arrhyythmia. Electrical cardioversion is performed if the patient is hemodynamically unstable in the face of the tachyarrhythmia or when ventricular response is refractory to drug therapy.[1,3]

In rare cases, both normal and accessory pathways rapidly excite the ventricles, causing preexcited atrial fibrillation (AF), characterized by an irregular, polymorphic, wide QRS tachycardia. Preexcited AF with RVR can quickly deteriorate to ventricular fibrillation and hemodynamic collapse.[1-3] Prompt electrical cardioversion may require the addition of pharmacological management. Procainamide is the drug of choice to lengthen the CAP refractory period

and slow the ventricular rate. β-blockers do not slow ventricular response to CAP conduction.[3] Digoxin and verapamil are also contraindicated as they may decrease the refractory period of CAPs responsible for AF, thus accelerating CAP conduction and increasing ventricular response rate.[1-2]

Preoperative assessment of patients with known history of WPW syndrome involves a thorough history including symptoms (palpitations, AF) and treatments (RFCA, drug therapy). The patient in this case study rarely experienced symptoms requiring treatment with propranolol. Patients requiring regular antiarrhythmics should continue these medications perioperatively.[1-2] Some studies estimate that 20% to 60% of patients with WPW have only intermittent ventricular preexcitation, making preoperative identification by ECG difficult.[4]

In a patient with unresolved WPW, the perioperative anesthetic goal is to minimize sympathetic nervous system (SNS) activation that precipitates tachyarrhythmias. β-blockers, opioids, and intravascular volume should be given to prevent tachycardia.[1-2] In this case, anxiolysis, analgesia, and neuromuscular blockade were carefully selected and administered to prevent sympathetic stimulation. Adequate pain control and minimal SNS stimulation are essential to avoid triggering tachydysrhythmias, through emergence and the post-operative period. Acetaminophen IV may be considered for postoperative pain control.

Propofol data conflicts regarding its antiarrhythmic and proarrhythmic effects, which may be relevant for patients with WPW.[7] One study of 5 adults and 7 children undergoing EP studies and RFCA for AVNRT found propofol had no effect on AV node conduction.[5] Another study of 60 children receiving RFCA showed slowed AV node conduction with propofol.[7] In this case, propofol was used for induction without any observed proarrhythmic effects.

When providing anesthesia to patients with unresolved WPW, anesthesia practitioners should understand the type of arrhythmia they present, and be prepared to administer appropriate antiarrhythmic therapies. It is also critical to minimize SNS stimulation perioperatively and be prepared for electrical cardioversion to address unstable arrhythmias. With successful RFCA, the incidence of unresolved WPW has been dramatically reduced, but anesthesia providers still must be prepared to care for patients with WPW.

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Horner's Syndrome following Lumbar Epidural for Labor

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Keywords: epidural, lumbar epidural anesthesia, labor epidural, Horner's syndrome

Lumbar epidural anesthesia is a common technique for pain management in laboring parturients. Although common, it is not a benign procedure. Horner's syndrome (HS) is a rare complication of epidural anesthesia, often self-limiting with symptom resolution upon regression of the neuraxial blockade. Symptoms of HS include miosis, ptosis, anhydrosis, nasal stuffiness, and fascial flushing and can herald an impending high spinal with associated respiratory compromise. These symptoms produce significant anxiety with patients and labor staff, and anesthetists need to be aware of this possible complication to rapidly exclude other causes of HS and institute appropriate management.

Case Report

A 21- year-old, 177.8cm, 58 kg primigravida presented to the labor and delivery department in active labor at 39 weeks gestation with regular, progressively intensifying uterine contractions. Medical history included asthma and anemia with normal prenatal course and laboratory data. Upon reaching 4 cm cervical dilation, the patient requested epidural pain management. The risks and benefits were discussed and consent was obtained. A bolus of 800 mL of lactated ringer's solution was infused intravenously and the patient placed in the sitting position, prepped and draped in sterile fashion. Appropriate local anesthesia was obtained using 1.5% lidocaine 5 mL between the third and fourth lumbar interspace. An 18 ga Touhy needle was placed using the midline approach and loss of resistance with air. The epidural space was identified and the epidural catheter threaded easily with 5 cm of catheter placed into the epidural space. Aspiration of the epidural catheter was negative for blood or cerebrospinal fluid. A test dose of 1.5% lidocaine with epinephrine 5 mcg/mL 3 mL of was administered with negative indication of intravascular or intrathecal administration. The patient was given 0.25% bupivacaine 8 mL and fentanyl 100 mcg in small aliquots over 5 minutes followed by a continuous infusion of 0.125% bupivacaine and fentanyl 3.6 mcg/mL at 10 mL/hr per hour. Shortly after placement of the labor epidural catheter the patient reported satisfactory pain control with limited ability

to lift legs and sensory level to T6 dermatome level bilaterally.

Approximately 45 minutes after initial bolus administration the patient complained of nasal stuffiness and the patient's family reported left eye drooping. Upon physical examination the patient was awake, alert, and denying respiratory difficulty. Grip strength was equal bilaterally; ptosis and miosis was noted on the left side in comparison to the right. There were no hemodynamic derangements requiring intervention. Fetal monitoring showed no fetal distress. Sensory level to pinprick continued to be T6. Both patient and family expressed anxiety and apprehension pertaining to the undesired symptoms associated with HS requiring significant reassurance. Based on symptom presentation and physical exam, HS was suspected and the epidural infusion was stopped. After 90 minutes, there was partial regression of symptoms with an increase in discomfort associated with labor. The epidural infusion was restarted at a rate of 6 mL per hour without bolus administration. Over the subsequent 90 minutes, labor pain increased and the patient was given a bolus of 0.25% bupivicaine 8 mL and the infusion increased to 10 mL per hour, resulting in pain resolution. Ultimately the patient delivered a healthy baby via cesarean section due to arrest of descent and failure to progress nine hours later.

Discussion

Epidural analgesia is a commonly used technique for the management of pain in the laboring patient. Horner's syndrome results from inhibition of the ascending sympathetic trunk.[1] While HS is usually non-threatening and self-limiting,[2] it can warn of high sympathetic blockade and respiratory compromise[3] as well as induce significant anxiety among patients and staff. Therefore it is prudent for anesthesia practitioners to be aware of the subtle presentation of Horner's syndrome (HS) so that appropriate treatment and reassurance can be provided. Sympathetic pathway blockade allows predominant parasympathetic nervous system control, producing the characteristic symptoms of miosis, ptosis, and anhydrosis, nasal stuffiness and facial flushing.[4] The exact mechanism by which the symptoms occur remains unclear, but the most widely accepted theory suggests disruption of the preganglionic, second order neurons along the oculosympathetic pathway involving the eighth cervical and first thoracic nerve as they exit the spinal cord.[4,5] Originally described in the obstetric setting in 1972 by Kepes et al,[4,6] the most recent literature cites an incidence of 0.4% to 2.5% during labor analgesia, which increases to 4% during epidural anesthesia for cesarean section.[5,6]

Normal anatomical, physiological, and hormonal changes associated with pregnancy may increase the risk for HS among parturients. Greater cephalad spread of local anesthetic may be attributed to the decreased volume and compliance of the epidural space and increased abdominal pressure from uterine contractions.[3-6] Unilateral spread of local anesthetic may be due to the septation within the epidural space[3, 5] or the lateral recumbent position. This may increase the risk for HS on the ipsilateral side due to gravity dependent spread of local anesthetic, but this is not seen in all the reported cases.[4-5] In this case, the patient was positioned from sitting position to the supine position and was not repositioned laterally leaving the potential for other mechanisms to be involved. Thinner, sympathetic nerve fibers already possess an increased affinity and sensitivity to local anesthetics which may result in a sympathetic block several segments above

the sensory level.[4-6] Barbara, Barua, and Tome, et al. proposed that high levels of progesterone during pregnancy may increase nerve fiber sensitivity even more, leading to increased incidence of HS in the parturient.[4] High levels of progesterone may explain some symptoms described in this case study, but not all.

Preganglionic cardioaccelerator fibers of the sympathetic nervous system synapse at the first through fourth or fifth (T1-T4) paravertebral ganglia.[7] Interestingly, the patient did not have any episode of hypotension or bradycardia inferring that the level of blockade had not ascended above the cardioaccelerator fibers at T4. Inadvertent epidural catheter migration outside the epidural space may also lead to symptoms of HS.[4] Subdural placement of the epidural catheter can produce an inconsistent, asymmetrical block that may have sparing of the motor fibers, which seems unlikely in this case due to the patient's even dermatome distribution and limited ability to lift legs off bed to command. While subarachnoid block can lead to motor block and hemodynamic instability, the patient in this case had an initial negative test dose with no reported dyspnea, hypotension, bradycardia and upper extremity grips were equal, indicating that epidural catheter was correctly placed and high spinal blockade was an unlikely event. Onset time of HS and time of resolution of symptoms depends on agent used with longer onset and resolution times expected with bupivacaine and ropivacaine. The patient had onset of symptoms within 45 minutes and partial resolution of nasal stuffiness and ptosis within an hour after initial infusion was stopped. Significant resolution of symptoms occurred within 24 hours, and complete resolution of symptoms occurred by discharge.

This patient was managed according to the most recent literature, which recommends a conservative approach to management once immediate maternal-fetal threats have been ruled out. The patient required increased vigilance due to the potential for high spinal and respiratory compromise. Our patient had no significant history or precipitating events to indicate other causes of HS. The symptoms of HS coupled with the increased attention from both anesthesia and labor staff created significant stress and anxiety to the patient and family, which resolved after reassurance and a thorough explanation on the transient nature of HS. Staff continued to monitor the patient closely, and no further intervention was required.

With the vast number of labor epidurals utilized for the management of pain in the parturient and the unpreventable and unpredictable occurrence of this complication, Horner's syndrome deserves attention from anesthesia providers due to the potential for impending high spinal and the high levels of anxiety it produces among the patient and labor staff.

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Emergence Delirium in the Military Veteran

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Keywords: emergence delirium, post-traumatic stress disorder, anesthesia, military veteran

Emergence Delirium (ED) is a post-anesthetic phenomenon which occurs immediately after emergence from general anesthesia. ED is defined by behavioral symptoms of restlessness, confusion, and possible combative or violent actions after waking from general anesthesia.[1] ED associated behavioral symptoms can create a potentially dangerous environment for patients and health care providers.[2] Due to military involvement in Iraq and Afghanistan, combat veterans continue to serve on active duty. Older generation veterans also present with health related needs. The traumatic physical and psychological effects from the battlefield can carry over into the healthcare environment.[1,3] Interest in ED has developed among military anesthesia professionals because of its increasing incidence within their surgical population.[2]

Case Report

A 90 kg, 42-year-old male Military Veteran presented for a right shoulder arthroscopy with rotator cuff repair. The patient's medical history included allergic rhinitis, hypertension, incomplete left bundle branch block, occasional alcohol use, and gastro esophageal reflux disease (GERD). The patient's mental history revealed panic disorder, insomnia, and post traumatic stress disorder (PTSD). The patient also reported having had a previous computerized tomography (CT) scan of the head due to frequent severe headaches. The results of the exam were negative. The patient's medications included: zolpidem, loratadine, gabapentin, paroxetine, hydrochlorothiazide, lisinopril, omeprazole, and ranitidine. Laboratory values were assessed and found

to be physiologically appropriate. The patient had no known drug allergies.

Noninvasive blood pressure, oxygen saturation, and ECG monitors were applied to the patient and O_2 2 L/min was administered via nasal cannula in preparation for right interscalene brachial plexus block. The block was administered under ultrasound guidance via a 22 gauge 2" nerve stimulator needle. The block was placed easily and without complications. The patient was rolled to the operating room (OR) and general anesthesia with tracheal intubation was induced without complication. The patient was rolled into the left lateral surgical position. The patient remained hemodynamically stable throughout the procedure and the surgery was completed without any complications.

Following closure and dressing of the surgical site, the patient was repositioned supine. The volatile anesthetic was discontinued, and the patient's oropharynx was suctioned above the endotracheal tube. The patient's tidal volume, respiratory rate, and oxygen saturation met criteria for safe extubation. The patient demonstrated movement of all extremities to command along with a sustained head lift of greater than 5 seconds. Prior to extubation, all vital signs remained stable. Immediately following extubation, the patient arched his back and rolled off the left side of the table onto the OR floor. The patient was quickly assessed for injuries by OR personnel and was found to be awake and in stable condition. The patient apologized for jumping off the table stating, "I was trying to escape." The patient reported having a bad dream and felt the urge to "escape." The patient was lifted onto a stretcher by OR personnel and taken to the post anesthesia care unit (PACU). The patient's vital signs were stable in PACU and no injury from the fall was found to be present.

Discussion

Post traumatic stress disorder has four types of symptoms: reliving the event (also called re-experiencing or a flashback), avoiding situations that remind you of the event, feeling numb, and feeling keyed up (also called hyperarousal).[4] The criteria for diagnosis of PTSD includes: 1) exposure to a traumatic event that evoked feelings of fear, hopelessness, or horror; 2) demonstration of at least one significant symptom of re-experiencing, three clinically significant symptoms of avoidance or emotional numbing, and two clinically significant symptoms of persistent arousal; 3) symptoms that persist for at least one month; and 4) symptoms that adversely impact functioning.[5]

A recent qualitative study defined ED within military personnel with PTSD as "any occurrence in which the patient awakens in a violent or thrashing manner with attempts at self-extubation, breath holding, intravenous line displacement, assault on the operating room staff, and/or the want to flee or the risk of falling from the narrow operating room table."[3] The patient in this case would clearly meet the definition of ED with consideration to his mental history and surgical events.

Another recent investigation suggested that there was a correlation between anxiety, depression, PTSD, and ED in a sample of combat veterans.[1] Other studies have demonstrated that members of the military, with deployment/combat history, often emerge from general anesthesia restless and confused. Such events have led to the development of phrases such as "wild wake-up" and "post-anesthetic wind-up" to

describe ED in this patient population.[2] Risk factors for ED in the military population have been identified. A review of the patient's history and psychological disorders may have alerted the OR personnel in this case to a possible ED event.

Potential risk factors for ED within this specific patient population have been categorized into four main areas: environmental (light, noise), social (lack of social support), biological (physical trauma requiring surgery, pain, traumatic brain injury (TBI)), and psychological (PTSD, anxiety, depression).[2] It is unknown exactly which of these factors played the largest role in this particular case. Operating rooms typically have plenty of lights and are often noisy with equipment, instruments, monitors, phones, and continuous conversations. The OR in this case was no exception. Identifiable risk factors for this patient would be attributed to the biological and psychological categories.

The stress response from surgical insult can result in overstimulation of the sympathetic nervous system, which in turn may present in the form of a behavioral response, manifesting as ED.[2] Patients who have not been adequately treated for pain can appear much like a patient experiencing ED. In this case the patient had received an interscalene brachial plexus nerve block for perioperative and postoperative pain management. The patient also received fentanyl 325mcg IV and ketorolac 30mg IV perioperatively in addition to the interscalene nerve block. Untreated pain in the immediate postoperative period may mask the diagnosis of ED, or be the direct cause.[2] TBI is estimated to be between 10-20% of the patient population exposed to combat. The extent of TBI maybe mild in nature and therefore is often undiagnosed. Even though the patient had a previously normal head CT, it is unknown whether this patient had an underlying TBI.

This patient's psychological history included risk factors for ED. He had previously been diagnosed with panic disorder, insomnia, and PTSD. A review of his medication list indicated treatment of insomnia with zolpidem and treatment of depression with paroxetine. Nearly 20% of returning combat veterans develop PTSD; the prevalence is even higher among injured veterans and a recent case study reported ED in a patient with a known diagnosis of PTSD and depression.[2]

Several risk factors may have attributed to the manifestation of ED in this case. The literature review supports that ED is a multifactorial complication.[1] This case report also demonstrates the need for the anesthesia care team to identify potential risk factors for ED in an effort to ensure a safe surgical experience for the military surgical population. Appropriate identification of potential ED patients can allow the anesthesia team to take safety precautions upon emergence from general anesthesia.

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Post Extubation Stridor

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Keywords: inspiratory stridor, laryngeal edema, racemic epinephrine, difficult intubation, respiratory distress, laryngospasm, croup

Inspiratory stridor is an alarming sign that may indicate airway obstruction. Stridor may imply either supraglottic or glottic narrowing.[1] Causes of acute onset inspiratory stridor in the postoperative patient include: laryngospasm, laryngeal edema, vocal cord dysfunction, or foreign body aspiration.[2] Like stridor croup is also associated with multiple tracheal intubation attempts due to post intubation edema around the glottis and subglottic regions .[1] Primary goals for the anesthesia practitioner regarding the patient with inspiratory stridor are to maintain airway patency and abate the laryngeal edema.

Case Report

A 47-year-old male with a history of polycystic kidney disease presented for tenckhoff catheter placement for chronic dialysis venous access. Past medical history was significant for gastroesophageal reflux disease, hypertension, and polycystic kidney disease. The patient was 175 cm tall and weighed 106 kg. Home medications included allopurinol, amitriptyline, amlodipine, aspirin, labetalol, lisinopril, hydrocodone/acetaminophen, esomeprazole, pravastatin, and terazosin. On physical examination the patient had a Mallampati class 1 airway with full neck range of motion and normal dentition. Adequate thyromental distance was noted. The anesthetic plan consisted of a general anesthetic with rapid sequence induction, followed by tracheal intubation.

Preoperatively the patient received sodium citrate 30 ml orally, famotidine 20mg, metoclopramide 10 mg, and midazolam 5 mg. The patient was taken to the operating room and standard monitors were applied. After pre-oxygenation with 100% O_2 for 5 min anesthesia was induced using a rapid sequence induction technique. Direct laryngoscopy was attempted using a Macintosh 3 laryngoscope blade, but only a Cormack-Lehane grade 3 view was obtained and a second attempt revealed a similar view. Following the second attempt the patients' oxygen saturation was 78%. Easy mask ventilation with 100% oxygen was accomplished and the patient's oxygen saturation increased to 99%. A third attempt at tracheal intubation was performed using a C-mac video laryngoscope (Storz, Tuttlingen Germany), but was unsuccessful. A final attempt was made using the video laryngoscope and a flexible intubating fiberoptic bronchoscope - a 7.0 endotracheal

tube was inserted into the trachea. Endotracheal tube placement was confirmed with bilateral breath sounds and positive end-tidal CO_2 capnography. The endotracheal tube was secured and ventilation was maintained with a mechanical ventilator. General anesthesia was maintained with Desflurane 6% inspired concentration in oxygen, and neuromuscular blockade was achieved with cisatracurium 6 mg.

Upon case completion neuromuscular blockade was antagonized with neostigmine 5 mg and glycopyrrolate 1 mg. The patient was extubated after developing a full train of four, performing a sustained head lift and following commands. Soon after oral suctioning and removal of the endotracheal tube the patient exhibited severe stridor. The SpO_2 decreased to 65%. Positive pressure ventilation with a facemask and 100% O_2 was initiated. After 10 minutes of positive pressure ventilation the stridor was unresolved, but the SpO_2 had increased to 95%. The patient's respiratory rate was 30 breaths/min and shallow with continued stridor. The patient was taken to the post anesthesia care unit (PACU) on 100% oxygen via facemask with noted significant increase in stridor during the short transport time. Upon reaching the PACU the patient was awake and alert with SpO_2 below 60%. Breathing was assisted using positive pressure ventilation with 100% oxygen and aerosolized 2.25% racemic epinephrine 0.5 mL was administered ultimately leading to a complete resolution of the stridor and a resultant SpO_2 of 97%.

Discussion

The treatment of stridor is important to all anesthesia practitioners because total laryngeal obstruction can occur at any moment once stridor is present. Stridor occurs when air is rapidly forced through a constricted area.[3] Stridor occurring immediately after extubation is usually caused by laryngospasm.[3] In the case of complete laryngospasm, stridor will be absent due to the complete lack of air movement caused by the closed vocal cords.[3] Stridor following extubation of the trachea does not usually resolve on its own and requires prompt treatment.[3]

The first line treatment of stridor due to laryngospasm is the application of positive-pressure ventilation with 100% oxygen.[1] Pressures greater than 10-20 cm of water may lead to gastric insufflation and distention.[1] While positive pressure ventilation helped with the partial obstruction in this case, it did not completely resolve the stridor. Forward displacement of the mandible and inward pressure toward the base of the skull (also termed the laryngospasm notch) with both middle fingers may alleviate laryngospasm as well.[4,5] This maneuver opens the airway and causes periosteal pain leading to relaxation of the vocal cords.[5] If these methods prove unsuccessful, the anesthesia practitioner should call for help.[4] At any time, if unresolved oxygen desaturation or a complete laryngospasm develops, patients may need to be emergently intubated.

The treatment modality that proved most successful in this case was the use of racemic epinephrine in the recovery unit. There were multiple intubation attempts, which may be associated with airway edema. Stridor associated with multiple intubation attempts usually occurs within three hours of extubation.[1] Racemic epinephrine is a mainstay of treatment for post extubation stridor due to airway swelling.[1] The inhalation dose of racemic epinephrine is administered with cool moist oxygen via facemask. The dose is 0.5ml of a

2.25% solution in 2.5ml of normal saline.[1] An alternate treatment for post extubation stridor includes dexamethasone 0.1-0.5mg/kg IV.[1]

There are many other treatment options in the literature aimed at reducing laryngospasm and croup. Premedication with anticholinergics such as atropine may also reduce the chance of a laryngospasm.[4,5] Other effective treatments used to prevent laryngospasm include administration of IV: a) lidocaine 1mg/kg, b) dexamethasone 0.1-0.5mg/kg, c) propofol 0.25-0.5mg/kg IV, and, d) nitroglycerin 4g/kg.[1,4,5] During extubation, the lungs should be inflated using positive pressure in order to help decrease the abductor response of laryngeal muscles.[5] Extubating when patients are fully awake and opening their eyes spontaneously also reduces the occurrence of laryngospasm during emergence.[5] Treatment for total laryngospasm includes administration of an intubating dose of succinylcholine (1 to 2mg/kg IV or 4mg/kg intramuscularly) and possible re-intubation.[1] For refractory laryngospasm, a superior laryngeal nerve block can relax the cricothyroid muscle and alleviate vocal cord constriction.[5]

Various techniques to prevent and treat stridor and laryngospasm are available to anesthesia practitioners. These options should be individualized to patient needs. Racemic epinephrine should be readily available in both the operating room and post anesthesia recovery unit. Early use of these techniques may prevent morbidity and mortality.

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Bradycardia During Laparoscopic Surgery

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Keywords: Bradycardia, pneumoperitoneum, laparoscopic surgery, decreased venous return, anticholinergic.

Laparoscopic surgery is a growing field that has made particular advances in the last 30 years.[1] Laparoscopy is being used in fields

that include general surgery, urology, and gynecology.[1] The creation of a pneumoperitoneum is the featured element.[2] The most commonly used gas for insufflation is carbon dioxide.[2] As pressure on the diaphragm increases, as a result of the pneumoperitoneum, venous return to the heart decreases.[2] This increase in pressure can lead to excessive parasympathetic activity, which in turn can lead to bradyarrhythmias.[3] Bradyarrhythmias such as this require fast recognition and treatment.[3]

Case Report:

A 27-year-old, 52 kg female presented for a laparoscopic cholecystectomy for treatment of cholelithiasis. The patient was otherwise healthy with no co-existing diseases, no allergies, and was not taking any prescribed or over-the-counter medications. No abnormal laboratory values were noted. No abnormal findings were noted on physical exam.

The patient presented to the operating room with a 20 gauge peripheral IV. A solution of Lactated Ringers was initiated at 450 mL/h. Following a preoperative dose of midazolam 2 mg and pre-oxygenation with 10 L/min of oxygen, intravenous induction of general anesthesia was performed in sequential order with: fentanyl 75 mcg, lidocaine 50 mg, propofol 100 mg, and rocuronium 30 mg. The trachea was intubated with a 7.0 mm cuffed endotracheal tube and respirations were controlled with a mechanical ventilator. Ventilations were controlled at a rate of 10 breaths/min, with a tidal volume of 450 mL, and titrated to maintain an end-tidal CO_2 of 35 mmHg of mercury. Dexamethasone 4 mg IV and ondansetron 4 mg IV were administered for prophylaxis of post-operative nausea/vomiting.

General anesthesia was maintained during the case with sevoflurane 1.8% inspired concentration in a mixture of O_2 1 L/min and air 1L/min, and incremental doses of fentanyl 25 mcg IV to a total of 150 mcg. The patient was placed in reverse Trendelenburg position. The table was tilted approximately 10 degrees to the left. The circulating nurse placed sequential intermittent compression devices to the lower extremities bilaterally following induction.

During the initial maintenance phase, creation of the pneumoperitoneum occurred. Initial insufflation pressures did not exceed 20 mmHg of mercury. Following the initial institution of pneumoperitoneum, the patient's heart rate decreased from a pre-operative 65 to 35 beats/min. The surgeon was immediately notified. The pneumoperitoneum was desufflated. IV fluid rate of lactated ringers solution was increased, and glycopyrrolate 0.2 mg IV was administered. The patient's heart rate returned to the previous rate of 65 beats/min. The pneumoperitoneum was then attempted to be insufflated at pressures less than 15 mm Hg. The patient's heart rate once again decreased from 65 to 35 beats/min. The surgeon was once again informed. The pneumoperitoneum was desufflated and glycopyrrolate 0.2 mg IV was administered. The patient's heart rate once again returned to 65 beats/min. The pneumoperitoneum was insufflated at pressures less than 15 mmHg. Vital signs remained within normal limits. The surgery proceeded uneventfully.

Neuromuscular blockade was antagonized with neostigmine 3 mg IV, and glycopyrrolate 0.4 mg IV. Train of four count was 4/4 with sustained tetany. The patient had a return of spontaneous ventilation at a rate of 10 breaths/min with a tidal volume of 400 mL. Emergence was

uneventful. The patient was placed on O_2 10 L/min and inspired concentrations of sevoflurane were discontinued. The oropharynx was suctioned. The tracha was extubated awake and the patient was transferred to the recovery room with standard monitors and oxygen O2 10 L/min. No adverse events were reported throughout the post-operative recovery phase. The patient was discharged home in good condition after meeting discharge criteria.

Discussion:

Cardiovascular changes in response to the creation of a pneumoperitoneum may be varied. As the pneumoperitoneum is insufflated, intraabdominal pressure is increased.[4] The increased intra-abdominal pressure can lead to compression of the inferior vena cava, which in turn can lead to decreased preload, thus leading to a decreased cardiac output.[4] Typically, insufflation pressures are reduced to 10-15 mmHg in order to minimize cardiovascular complications.[5] Patients particularly at risk of cardiovascular changes are the elderly, due to their decreased ability to compensate, and patients with pre-existing heart disease.[1] Bradycardia is typically associated with decreased preload, as a result of the increase in intraabdominal pressures.[1]

Position may play a prominent role in altering hemodynamic parameters. To facilitate the operative view for the surgeon, Reverse Trendelenburg is the position of choice for the laparoscopic cholecystectomy.[4] This position can result in hypotension as a result of venous pooling in the lower extremities.[4] A reduction in the angle of reverse Trendelenburg may be required to improve venous return.[5] Patients are usually fitted with sequential, intermittent lower extremity compression devices to increase venous return and reduce venous stasis.

Other variables to be considered include preoperative fluid restrictions and the vasodilating properties of anesthetics.[1] Liberal fluid administration is typically employed to offset the decreased venous return caused by the increase in intra-abdominal pressure.[5] Although not done in this case, it is also recommended to hydrate patients preoperatively with the administration of intravenous crystalloid to minimize hypovolemia and decreased preload in response the creation of the pneumoperitoneum.[2]

Anticholinergic drugs can be beneficial in the treatment of bradycardia during laparoscopic surgery.[3] Anticholinergic drugs block the parasympathetic muscarinic receptors.[3] As a result of this, parasympathetic activity is decreased, and sympathetic activity is increased.[3] The end result is an increase in heart rate. The study performed by Aghamohammadi, Mehrabi, and Ali Beigi in 2009 recommended prophylactic treatment with anticholinergic agents, such as atropine, to counter parasympathetic activity such as bradycardia associated with laparoscopic surgeries.[3] In their study, bradycardia was much more commonly seen in a control group that did not receive an anticholinergic agent preoperatively.[3] Although, the control group's symptoms did resolve if an anticholinergic agent was administered in response to the bradyarrhythmia.[3]
In the case being discussed, the patient's heart rate decreased from 65 to 35 beats per minute in response to the creation of the pneumoperitoneum. Suggested causes of this decrease in heart rate are: hypovolemia from preoperative fluid restriction, decreased venous return caused by creation of the pneumoperitoneum, decreased venous

return from the reverse Trendelenburg position, and decreased preload from the vasodilating effects of anesthetic agents. Treatment modalities employed in this case were the use of sequential lower extremity compression devices to aid venous return. Intravenous fluids were administered at a liberal rate of 450 mL/h. The surgeon was notified immediately, and desufflation of the pneumoperitoneum occurred. Glycopyrrolate, an anticholinergic agent, was administered at a dose of 0.2 mg. When the event repeated in response to recreation of the pneumoperitoneum, the interventions were repeated, and ultimately were successful.

Additional interventions could have been employed and possibly prevented the bradyarrhythmia from occurring. Replacing the patient's fluid volume deficit preoperatively may have prevented hypovolemia and increased the patient's preload. Decreasing the angle of the reverse Trendelenburg position may have helped improve venous return and could have prevented the second episode from occurring. This is not always feasible due to surgical exposure needs. The study by Preiss and Berguson (1983) demonstrated a linear relationship regarding increased heart rate and increased dose of glycopyrrolate.[6] A higher initial dose of glycopyrrolate, such as 0.4 mg, could have been administered initially, although the initial symptoms did resolve with only the 0.2 mg. Due to atropine's increased affect on heart rate, the use of atropine instead of glycopyrrolate is also an option that could be debated.

I anticipate that this scenario will repeat itself sometime again during my career. I would advocate for the replacement of fluid volume deficits with intravenous crystalloid solutions preoperatively. The time constraints placed on anesthesia practitioners to minimize turnover mandates good coordination between anesthetists and preoperative nurses for preoperative fluid replacement. It is difficult to recommend any change in position for this procedure. The reverse Trendelenburg position is used to improve the surgeon's view for this procedure.[2] Using the minimum angle possible would be recommended to minimize the decrease in venous return. Intermittent breaks from the position may be needed should bradycardia occur. Anticholinergic agents such as glycopyrrolate will continue to be recommended to improve cardiac function in response to bradycardia.[3] Ultimately, I feel the most important interventions are close coordination and discussion with the surgeon, and vigilance by the anesthesia practitioner.

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Cricoid Pressure Effective in Aspiration Prevention with Emergent Appendectomy

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Keywords: cricoid pressure, rapid sequence induction, Sellick's maneuver, full stomach precautions, aspiration prevention

Cricoid pressure (CP) is considered a standard of care when full stomach precautions must be exercised during the induction of general anesthesia.[1,2] CP is intended to prevent aspiration of gastric contents by mechanically occluding the esophageal passageway. Techniques that may be employed to achieve this include: (1) pressure applied at the cricoid cartilage with force directed towards the posterior aspect of the patient's neck, (2) apply CP with force directed posteriorly and cephalad, or (3) utilize the "BURP" (backward, upward, rightward pressure) maneuver.[1,3] Force requirements for effective CP ranges from 20 Newton (N) for the awake patient, to 44 N when loss of consciousness is achieved.[1]

Case Report

A 39-year-old, 95 kg, 152 cm male presented via the emergency department with a diagnosis of acute appendicitis and was scheduled for an emergent laparoscopic appendectomy. History and physical revealed a history of gastroesophageal reflux, symptoms of abdominal pain in the right lower quadrant, and nausea with one episode of vomiting over the prior twelve hours. Surgical history included an inguinal hernia repair five years prior with no surgical or anesthesia complications. Current medications included calcium carbonate as needed for reflux. The patient had no reported allergies. Assessment showed a class two Mallampati score and thyromental distance of approximately six cm.

Once the patient was consented for anesthesia, metoclopramide 10 mg IV and ranitidine 50 mg IV were administered in the preoperative holding area. Midazolam 2 mg IV was administered immediately prior to transport to the operating room (OR). Once in the OR, standard monitors were applied, preoxygenation was provided via facemask, and approximately 15 degrees of reverse Trendelenburg position was utilized. The decision was made to employ a rapid sequence induction due to full stomach precautions. The patient was informed that he would feel slight pressure being applied to his neck as he "was going to sleep". The circulating nurse applied cricoid pressure, and induction was initiated with fentanyl 100 mcg IV, lidocaine 100 mg IV, vecuronium 1 mg IV as a defasciculating dose, propofol 200 mg IV, and succinylcholine 140 mg IV.

It was observed that the circulating nurse's hand appeared to be abnormally close to the patient's chin, at which point the clinician asked if cricoid pressure was being applied. The supervising anesthesia professional inspected and corrected the position of the cricoid pressure was being applied to a more caudad position where pressure was applied directly to the cricoid cartilage, as opposed to the thyroid cartilage. Instruction on proper pressure requirements for CP was given, as well as instruction to maintain CP until the endotracheal tube (ETT) was properly secured and placement was confirmed in the trachea.

Direct laryngoscopy was performed using a Miller 2 laryngoscope blade, yielding a Cormack laryngoscopic grade 1 view.[4] A 7.5 cuffed ETT was placed into the trachea under direct visualization. Minimal occlusive pressure was applied to the ETT cuff, end-tidal CO2 tracing was observed on the monitor; bilateral breath sounds were auscultated, and CP was released. Ventilation was controlled via a mechanical ventilator, and general anesthesia was maintained with desflurane 6.6% end-tidal concentration in a mixture of oxygen 1 L/min and air 1 L/min. After return of twitches utilizing a train-of-four monitor, vecuronium 7 mg IV was administered. An orogastric tube was temporarily placed after induction, which yielded an initial volume of 150 ml of gastric content when placed to low suction. Dexamethasone 4 mg IV and ondansetron 4 mg IV was administered approximately 35 minutes prior to emergence from anesthesia. Neuromuscular blockade was antagonized, and the endotracheal tube was removed without complications.

Discussion

The use of CP is a point of debate among anesthesia professionals, primarily due to lack of strong evidence to support or refute its use, as well as conflicting test data results. As was the case for this patient, CP is indicated for cases where full stomach precautions should be exercised, as well as in emergency situations where CP is deemed appropriate, such as in patients with a high risk of aspiration or a history of gastroesophageal reflux.[1,2,5,6]

Neilipovitz and Crosby cite that emergency surgery carries a greater than six-fold increased risk of aspiration versus elective surgery.[6] Preoxygenation was provided via facemask for approximately four minutes, during the time interval from when the patient was positioned onto the OR table, all monitors were placed, the OR table was positioned in reverse Trendelenburg, and baseline vital signs were obtained. Preferred methods of preoxygenating the patient include three to five minutes of tidal volume breathing, or at least eight vital capacity breaths in a 60 second interval.[6] Furthermore, Neilipovitz and Crosby state that incorporating the head-up position during preoxygenation "…extends the safe apnea time in obese patients when compared with the supine position". [6]

In this case, CP was initially applied as the patient was awake, and the force was increased with loss of consciousness. The appropriate pressure to apply in the awake patient is approximately 20 N and increases to approximately 44 N with loss of consciousness.[1,3,5] Proper finger placement for CP includes single-handed downward pressure with the index finger over cricoid cartilage, and thumb and middle finger each side.

Although rapid administration of induction medications may not be appropriate for all patients, this patient was considered a high risk for aspiration and was deemed appropriate. This coincides with current literature; although this practice may carry the risk of hemodynamic instability, it did not occur in this case.[6]

Although there are several variations of exactly how to apply CP, in this case pressure was applied directly in a posterior manner, resulting in an acceptable view to allow the airway to be secured. Snider et al.[7] studied the effects of utilizing the BURP maneuver and its resultant effect on glottis view, and although subjective in nature, this study found that the BURP maneuver actually worsened the view during laryngoscopy. Much of the debate around the effectiveness of CP seems to revolve around the position of the esophagus relative to the position of pressure application at the cricoid. Rice et al.[8] looked at the anatomy involved in CP application, as well as the effectiveness of occluding the passageway to the esophagus, and concluded that the cricoid cartilage and postcricoid hypopharynx maintain their anatomical relationship, thus acting as a functional "unit". This study utilized sniffing (as was utilized in this case), neutral, and extended head positions to evaluate the effectiveness of CP, and found that the relationship of the hypopharynx and cricoid ring is maintained due to anatomical connections, which is of primary importance when attempting to prevent flow of gastric contents in regurgitation and aspiration.[8] In this study, Rice et al. assert that the true work of CP actually occurs above the origin of the esophagus, and is thus "…independent of the positioning of the esophagus, given that virtually all of the "action" of CP takes place above the esophageal origin", concluding that CP is an effective means of occluding the passageway between stomach contents and the pharynx.[8]

Use of cricoid pressure in anesthesia practice was introduced over 50 years by Dr. Sellick.[9] However, there is still much debate over the effectiveness of CP. One consistent theme demonstrated in literature is the need for training and communication if CP is to be appropriately and effectively applied. Unfortunately, as Lerman[3] suggests, anesthesia professionals may not be taking an active role in educating others on the proper techniques for effective CP, even though improper CP application is one potential cause of failure to prevent regurgitation and aspiration. Further highlighting the need for educating proper CP application, Ellis, Harris and Zideman state that if CP is not being applied as recommended, then its usefulness "…becomes a secondary argument", as its efficacy cannot truly be evaluated. [5]

It seems evident that open communication and education of proper CP is essential, and there are several techniques in the literature that may prove useful given the current training tools at the anesthesia professional's disposal. First, Snider et al. utilized a marking pencil to identify the cricoid cartilage, which would intuitively lend itself to lessen the risk of pressure being applied at an incorrect position.[7] Secondly, utilizing a tool to "calibrate" ones hands to the proper amount of force would seem to increase the chances that proper force is being applied. Although tools as simple as a weighted brick have been utilized, weighted scales and makeshift simulators have also been employed and may play a significant role in the outcomes of each respective study.[2,7,8] Finally, consistently revisiting training to ensure that skills are retained appears key to the effectiveness of proper CP technique and success.[2,5]

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Converting Monitored Anesthesia Care to General Anesthesia

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Keywords: Monitored anesthesia care, MAC, sedation, rapid sequence induction, aspiration

Various levels of anesthesia are required for today's procedures**.** The depth of anesthesia required is dependent on the patient's condition and procedure being performed. The American Society of Anesthesiologists (ASA) states that monitored anesthesia care (MAC) may include varying levels of sedation, analgesia, and anxiolysis. The MAC provider "must be prepared and qualified to convert to general anesthesia when necessary."[1] Additionally, a provider's ability to intervene to rescue a patient's airway from sedation-induced compromise is a prerequisite to the qualifications to provide MAC.[2] The anesthesia professional must be vigilant and always prepared to convert to general anesthesia.

Case Report

A 69-year-old female presented to the surgical holding area for emergent cystoscopy with possible bladder clot evacuation. The patient weighed 60kg, was 157cm and had a documented allergy to penicillin. Medical history was significant

for hypertension, mitral valve prolapse secondary to rheumatic fever, renal insufficiency, cervical cancer with attributable radiation colitis, radiation proctitis, and radiation cystitis. Surgical history included hysterectomy with postoperative radiation and colon resection. Current medication regimen included atenolol, calcium, lisinopril, and tramadol. Informed consent was obtained and the patient was evaluated by the anesthesiologist and nurse anesthetist. The patient was given an ASA classification of 3E. Moderate sedation was requested by the surgeon due to expected short length of case and patient's acuity. The patient's NPO status was greater than 8 hours.

Upon arrival to genitourinary operating room the patient was transferred to the cystoscopy table via the operating room team. Oxygen was administered via face mask at 6L/min and standard ASA monitors were applied. Intravenous induction of anesthesia began with midazolam 1 mg, fentanyl 50 mcg, and propofol 50 mg. Spontaneous respirations continued with supplemental oxygen at 6 L/min as the desired level of sedation was achieved via a propofol infusion. The patient was then placed in lithotomy position.

Prior to start of procedure, a small amount of light green fluid was noted in the patient's mouth. The patient's mouth was immediately suctioned. Vital signs at this time included HR: 75, BP: 119/65, O2Sats: 98%. The surgeon was notified of the patient's change in status and procedure preparations were terminated. Suctioning of the oropharynx was continued due to increased emesis. A rapid sequence induction was initiated. Cricoid pressure was applied and succinylcholine 100mg was administered. Blow by oxygen via mask was maintained until muscle fasciculation had ceased. Direct laryngoscopy resulted in a Grade I view with no stomach content noted. The patient was successfully intubation after one attempt. Bradycardia (50s) and hypotension (70s/40s) were noted and epinephrine 0.6mg IV was administered. Bronchoscopy was utilized by the anesthesiologist to assess for possible aspiration. No gastric contents were noted distal to the vocal cords. The patient's vital signs stabilized, HR: 87, BP 138/89, O2Sats 100%, and the cystoscopy proceeded.

Cystoscopy was completed and the patient was found to have a perforated bladder which was suspected due to previous radiation treatments. This finding required emergent laparotomy. The patient was then transported to a general operating room. Upon arrival to a general operating room, a left radial arterial line and right internal jugular central venous line were placed. The patient's vital signs continued to be stable and the laparotomy was completed without incident. The patient remained on mechanical ventilation and was transported to the intensive care unit in stable condition.

Discussion

Today, MAC is the first option of anesthetic techniques in approximately 10-30% of all the surgical procedures. Among anesthesiologists and Certified Registered Nurse Anesthetists (CRNAs) surveyed in the United States, use of MAC averaged 27% and 29% of anesthetic techniques and 16% and 24% of anesthesia time respectively.[3] In the case presented, MAC was the preferred choice of anesthetic technique due to patient frailty and case duration.

MAC cases are multi-faceted and may include multiple levels of anxiolysis, analgesia and sedation. Sedation, as a continuum, is not always predictable and

each individual patient has the potential to respond differently as evidenced by the patient's rapid decline in the case presented. Anesthesia professionals intending to induce and maintain a given level of sedation should be prepared to rescue a patient whose level of sedation becomes deeper than initially intended. Individuals administering moderate sedation should be able to rescue patients who enter a state of deep sedation, while those administering deep sedation should be able to rescue patients who enter a state of general anesthesia.[4] In contrast to general anesthesia, moderate sedation is not expected to induce depths of sedation that would impair the patient's own ability to maintain airway integrity. In the case presented, the patient's acuity, the type of case, and the surgeon's request were all components of the decision to use MAC. Ekstein, et al state that because of cognitive decline in elderly patients after general anesthesia, MAC is a better choice for the elderly.[5]

Upon arriving to the operating room, the patient was able to maintain her own airway and was breathing sufficiently enough to maintain oxygen saturations >95%. The patient's protective pharyngeal and laryngeal reflexes were depressed due to the administration of sedation as well as her debilitated state, and advanced age. The patient was carefully and continuously monitored with special attention to preservation of the airway. During normal physiologic respiration, inspiration is subatmospheric creating a tendency for the airway to collapse. This tendency is opposed by upper airway muscles that contract just prior to diaphragmatic contraction. This upper airway motor control appears extremely sensitive to sedative-hypnotic drug administration such as propofol and midazolam. Sedative doses of midazolam have been reported to increase upper airway resistance three to fourfold, an effect exaggerated in the elderly.[6] Additionally, progressive levels of sedation result in decreased muscle tone, allowing for gastric contents to enter the oropharynx and potentially the lungs. While continual assessment of the patient's physiologic state is always required during anesthesia, MAC especially requires heightened vigilance.[7] At times, the patient's physiological status may require conversion of a MAC to a general endotracheal technique. As in this case, as the patient's status changed, so did the anesthetic management. This rapid change was facilitated by the preparation of the anesthesia practitioners for the delivery of general anesthesia.

When developing any anesthesia plan, it is imperative that a thorough patient assessment be completed and pertinent anesthetic plan created. It is possible for anesthetic plans to change to a general anesthetic in response to a change in patient's status. Due to appropriate response and timely intervention, there was no evidence of aspiration noted on bronchoscopy immediately after the incident or at 24 hours postoperatively. Patient safety is dependent on effective communication and preparation for expected as well as unexpected complications.

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Management of Obese Patients Undergoing Robotic Surgery

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Keywords: morbid obesity, robotic-assisted, pneumoperitoneum, body mass index, steep Trendelenburg.

A growing concern for anesthesia professionals in the United States is the rising rate of obesity. Obesity not only contributes to additive comorbid conditions but complicates anesthetic technique. A recent meta-analysis concluded that approximately 30% of the population across age and gender classifications are obese, and that 1 in every 20 Americans has a body mass index (BMI) of greater than 40.[1] Obesity has also been identified as a risk factor for endometrial cancer. Robotic-assisted hysterectomy requires steep Trendelenburg positioning and institution of pneumoperitoneum. In the obese patient, the combination of these two conditions may pose difficulties in both the anesthetic and surgical management. Adequate planning,

positioning, pre-oxygenation, and airway management skills are essential for a safe and effective anesthetic.

Case Report

A 53-year-old, 165 cm, 174 kg female presented for robotic hysterectomy secondary to the presence of endometrial carcinoma. Her current medical history included super morbid obesity with a BMI of 64 kg/m^2, well-controlled hypertension with baseline a blood pressure of 136/83 mmHg and SpO_2 of 97% on room air, well-controlled epilepsy with absence of seizures in the last two years, and Type II diabetes mellitus with preoperative blood glucose of 128 mg/dl. No hemoglobin A1C was available in the patient's chart. Her medication profile included atenolol, furosemide, levetiracetam, exenatide and metformin. Prominent findings on airway assessment included an increased neck circumference and poor dentition. Respiratory assessment revealed a one pack per day smoking history for the last 10 years. Bilateral breath sounds were clear to auscultation preoperatively. The anesthetic plan and concern for potential postoperative endotracheal intubation and mechanical ventilation were discussed with the patient. The patient denied any further medical history. Physical exam findings and laboratory data were otherwise within normal limits.

In the operating room, the patient was assisted to a sniffing position with two pillows under the shoulders and reverse trendelenburg positioning at 15º. She was pre-oxygenated for 5 min to an end tidal O_2 concentration > 90%. General anesthesia was induced with lidocaine 100 mg, fentanyl 100 mcg, and propofol 200 mg. After placement of an oropharyngeal airway, the ability to bag mask ventilate the patient was confirmed. Direct laryngoscopy, facilitated with rocuronium 100 mg, yielded a grade II Cormack-Lehane view. The trachea was intubated, and endotracheal tube (ETT) placement was verified with a positive $ETCO_2$ and auscultation of bilateral breath sounds. The patient was placed on volume-controlled mechanical ventilation. Isoflurane 1% in oxygen 1L/min and air 1L/min was delivered via inhalation.

The patient was positioned in dorsal lithotomy position, and the table was placed in steep Trendelenburg position at 30º. Within minutes, an increase in peak inspiratory pressures to 60 cm H_20 and a decrease in SpO_2 to 94% were noted. Auscultation revealed wheezing bilaterally with diminished breath sounds over the left lung fields, suggestive of a right mainstem intubation.

The patient was returned to the supine position and albuterol was administered via the ETT. The ETT was withdrawn 1 cm and secured at this location. Proper position was confirmed with decreased airway pressures and equal bilateral breath sounds. Maintenance of equal breath sounds were confirmed after the bed was returned to Trendelenburg at 25º. Abdominal insufflation pressures were limited to 13 mmHg by the surgical team. Pressure-controlled ventilation was utilized with peak airway pressures of 38-44 cm H_20 and tidal volumes of 407-682 ml. The I:E ratio was increased to 1:2.5 and positive-end expiratory pressure (PEEP) of 5 mmHg was utilized to improve compliance and maintain oxygenation. Oxygen saturations above 92% were considered acceptable. Pulse oximetry was maintained between 94-100%, and $ETCO_2$ was maintained at 36 mmHg. Upon completion of the case, isoflurane was titrated off and neuromuscular blockade was antagonized with neostigmine 5 mg and

glycopyrrolate 0.8 mg. A 36 Fr nasal airway was placed in the right naris after administration of two puffs of oxymetazoline nasal spray and appropriate lubrication with 2% lidocaine jelly.

After 10 min of assisted spontaneous ventilations, the patient was taking regular, 200 ml tidal volume breaths (3.8 ml/kg of her ideal body weight). She had an equal TOF ratio, equal double burst stimulation response, and was able to sustain a head lift on command. The ETT was removed without complication and she maintained an SpO_2 98% and $ETCO_2 < 55$ mmHg. The patient was transported to the post anesthesia care unit with the nasal airway in place. Her postoperative course was uneventful, and she was discharged to home on the second postoperative day.

Discussion

Robotic hysterectomy presents many challenges for the morbidly obese patient. General anesthesia alone contributes to decreased lung volumes and atelectasis. Obesity further reduces functional residual capacity, contributes to restrictive lung disease, and increases oxygen requirements. Co-morbidities such as heart disease, diabetes mellitus, and hypertension are often present in the obese patient. Delayed gastric emptying secondary to diabetes mellitus may have contributed to an increase in gastric contents. Given this possibility, a rapid sequence induction could have been warranted.

Anesthetic risks associated with the steep Trendelenburg positioning and pneumoperitoneum required for Robotic-assisted procedures include subcutaneous emphysema, optic neuropathy, laryngeal edema, pneumothorax/pneumomediastinum, increased intracranial pressure, increased intra-abdominal pressure, decreased pulmonary compliance, and hypercapnia.[2] Further physiologic effects include increased preload, increased airway pressures, and decreased arterial oxygen saturation.[3]

Airway protection was achieved with the utilization of general endotracheal anesthesia and gastric decompression via an orogastric tube. As the patient had no significant reflux and had been compliant with NPO guidelines, rapid sequence induction was not performed. The addition of PEEP), albuterol administration, reduced Trendelenburg positioning, and reduced insufflation pressures may have contributed to the avoidance of postoperative complications and the need to transition to an open procedure. However, further optimization of this case via recruitment maneuvers, varying anesthetic selection and efficient management of neuromuscular blockade could have ameliorated ventilatory difficulties in this patient.

Recruitment maneuvers such as the application of 40 seconds of continuous positive airway pressure at 40 cm H_20 after institution of pneumothorax could have proven advantageous to attenuate atelectasis.[4] Utilization of this technique, preoperative administration of albuterol, and early use of PEEP may have assuaged the spike in airway pressures during initial Trendelenburg positioning. Also, a more thoughtful choice of anesthetic may have been beneficial. Isoflurane has a higher blood:gas partition coefficient than other volatile agents and may not have been the best choice for this procedure. Current literature maintains that volatile anesthetic selection for the morbidly obese patient is controversial; however, there is evidence supporting the efficacy and superiority of desflurane for this patient population. Both sevoflurane and desflurane have been

marketed based on their reduced absorption in the tissues, with sevoflurane possessing the added advantage of reduced airway irritation.

McKay and colleagues studied the use of desflurane as compared to sevoflurane and noted more expedient and consistent return of protective airway reflexes and response to verbal commands.[5] Jakobsson discussed the hemodynamic stability achieved with desflurane which would combat alterations in preload and cardiac output resulting from robotic techniques.[6] While desflurane does allow for faster recovery from anesthesia, it does exhibit increased pungency in comparison to isoflurane and sevoflurane and also can increase heart rate and blood pressure due to sympathomimetic properties. Given desflurane's decreased potency coupled with a prolonged case duration, utilization would be higher, representing another drawback to the use of desflurane for this case. Bispectral index monitoring was utilized and ranged between 40-60 during the case. Reduced concentrations of isoflurane between 0.4-0.6% combined with efforts to maintain bispectral index between 50-60 at the end of the case may have contributed to a more rapid emergence.

Aside from alterations in selection and utilization of volatile anesthetics, further pharmacologic changes could have been made to improve patient outcomes. Opioid management, however, was appropriately minimized for this patient and acetaminophen 1,000 mg IV, was used as a non-opioid adjuvant with excellent pain control noted postoperatively. Optimization of neuromuscular blockade could have been achieved with continuous infusion of a non-depolarizing muscle relaxant. Additionally, induction doses of relaxant based on ideal body weight rather than actual body weight may have prevented over-administration initially.[7] This technique would promote more effective relaxation and would provide more prompt return of twitches in preparation to antagonize the neuromuscular blockade.

In conclusion, the morbidly obese patient presents many challenges to anesthesia professionals especially when cases are technically complex as with robotic-assisted hysterectomy. While this case utilized an appropriate anesthetic, enhanced planning and heightened vigilance intraoperatively may have allowed for smoother conduct of the case and more rapid return of adequate spontaneous ventilation. Selection of volatile and intravenous agents should take into consideration the volume of distribution of obese patients as well as their metabolic state. Pulmonary recruitment maneuvers have been studied to improve ventilation for such patients undergoing robotic surgery, but with the ever-rising prevalence of obesity, further research is required to optimize patient outcomes in the future.

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Sacrococcygeal Teratoma Resection in a Pediatric Patient

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Keywords: sacrococcygeal teratoma, pediatric, thermoregulation, blood loss, pain control

A sacrococcygeal teratoma (SCT) is a common congenital tumor of the neonate occurring in 1:40,000 births.[1] Most SCTs are benign, consisting of a combination of solid and cystic components.[1] While benign in nature, significant problems associated with the tumor's mass and extensive blood supply can arise. Intraoperative management of SCT resection can be complicated by several factors. These may include difficulty positioning the patient supine for endotracheal intubation, difficulty ventilating the lungs in the prone position, problems with thermoregulation, cardiovascular instability, massive blood loss requiring transfusion, and tumor lysis.[1] These confounding factors can present unique challenges for the anesthetist.

Case Report

A 2-day-old, 3.4 kg, female infant presented for surgical resection of a large, type II externalized SCT with pelvic extension. The prenatal history was unremarkable until a SCT was diagnosed by prenatal ultrasound. Delivery occurred at 39 weeks gestation via cesarean section. The infant had no medical or surgical history, no current medications, and no known allergies. Preoperative labwork included a blood type and crossmatch, a complete blood count, and a basic metabolic panel. All labwork was within normal limits with an optimal starting hemoglobin of 15.3 g/dl and hematocrit of 47.3%. Physical examination revealed clear breath sounds bilaterally and a regular cardiac rhythm and rate. The last oral intake was greater than 8 hours prior to surgery. An intravenous (IV) infusion of D10W was infusing via a 24 gauge peripheral IV catheter at 12 ml/hr. A radiant heat warmer was in use to maintain thermoregulation.

The infant was transported to a pre-warmed operating room and placed on the operating table in a prone position with a pediatric underbody forced air warmer. Standard noninvasive monitors, including continuous temperature monitoring, were utilized. An IV fluid warmer was used. Preoxygenation commenced, and an IV induction was achieved using propofol 10 mg and rocuronium 4 mg. The infant was then carefully positioned supine for tracheal intubation using folded sheets to elevate the infant's head and torso to a position level with the SCT. Laryngoscopy was performed utilizing a Miller 1 blade and the infant's trachea was intubated with a 3.5 uncuffed endotracheal tube without complication. General anesthesia was maintained with sevoflurane. An additional 22 gauge peripheral IV was started for anticipated blood transfusions. The infant was then repositioned prone and surgery proceeded. Estimated blood loss during surgery was 50 ml. The infant received 4 transfusions of 10 ml of packed red blood cells (PRBCs) intraoperatively. The surgery was otherwise uneventful. The neuromuscular blockade was antagonized with neostigmine 0.2 mg IV and glycopyrrolate 0.04 mg IV after spontaneous return of breathing. The infant was repositioned supine and extubated without complications after respiratory rate and tidal volume were deemed adequate. SpO_2 was 100% on room air. The infant was wrapped in warm blankets and transported to the post anesthesia care unit (PACU). Upon arrival to the PACU, the infant was reassessed and noted to have stable vital signs with no acute distress noted. Care was transferred to the PACU nurse.

Discussion

Surgical resection of large SCTs in the pediatric patient has the potential to be fatal and is associated with a high rate of perinatal mortality.[2] SCTs are embryonal neoplasms that arise when totipotent germ cells located near Hensen's node in the embryo migrate to tissues other than the anatomical site of origin. [2] They consist of germ cell tissues that may include the ectoderm, mesoderm, and endoderm layers.[3] Depending on where these cells migrate to, teratomas can be found in a variety of sites, with the sacrococcygeal area being one of the most common.[3] The definitive treatment for SCTs is surgical resection. Caution was warranted when conducting anesthesia for this case given the surgical risk of resection in a very young pediatric patient.

Hypothermia is a well documented, preventable complication that can cause adverse effects following surgical interventions.[4] It has been associated with many detrimental effects which include increased oxygen consumption, decreased metabolic rate, decreased cardiac output, metabolic acidosis, prolongation of muscle relaxants, altered clotting functions, and an increased incidence of postoperative infection.[4] In this case, the infant's age and weight made her particularly susceptible to hypothermia. Newborns have limited capability for thermoregulation, especially during the first weeks of life.[5] Specific interventions by the anesthetist should be undertaken to prevent hypothermia from occurring. These interventions include warming the operating room, using a forced air warming device, using an IV fluid warmer, swaddling the infant in warm blankets, placing a warm hat on the infant, using an overhead radiant warmer as needed, and continuously monitoring the infant's temperature. All available measures were utilized during this case to prevent hypothermia, reducing the significant complications associated with it. Interventions to maintain thermoregulation

resulted in a normal temperature throughout the case and upon arrival to the PACU.

When a SCT has extensive vascularization and a size exceeding 10 cm, there is a high risk of rupture and profuse bleeding during surgical resection.[6] The increased baseline concentrations of hemoglobin, increased oxygen consumption, increased affinity of residual fetal hemoglobin for oxygen, and the absolute blood volume of the infant must be taken into account when deciding to transfuse blood.[7] These factors, as well as other physiological and surgical factors, demand extreme vigilance in monitoring and replacing blood loss. In this case the SCT was 12 cm, however, vascularization was only moderate, and the surgeons were able to ligate and cauterize bleeding arteries and veins during resection. Fortunately, the starting hematocrit for this case was 47.3%, and blood loss was limited to 50 ml. A total of 40 ml PRBCs was transfused during surgery to prevent complications associated with blood loss. It is important to note that prior preparation for massive blood loss was taken, including having type specific blood available, an IV blood set with blood warmer, and additional IV access. Careful monitoring of blood loss and timely transfusion of PRBCs to replace losses resulted in a hemodynamically stable patient. The postoperative hematocrit of 41.5% reflects this attentiveness.

Physiologic stressors associated with surgery can adversely affect the infant. In the first few days of life, infants subjected to stressors such as hypoxia, hypothermia, pain, and acidosis can manifest with severe increases in pulmonary vascular resistance.[8] The increased workload imposed on the right ventricle can promote right to left shunting via the foramen ovale, and if pulmonary vascular resistance exceeds systemic vascular resistance, a right to left shunt via the ductus arteriosus can occur.[8] This phenomenon is known as a return to fetal circulation. The resultant admixture of oxygenated and deoxygenated blood further contributes to the increase in pulmonary vascular resistance, establishing a vicious cycle. The infant in this case was only 2 days old, making her particularly susceptible to a return of fetal circulation. Anesthetic management of these cases should focus on reducing stressors that increase pulmonary vascular resistance, thereby controlling the development of right to left shunts and a return to fetal circulation.[8] Measures to reduce stressors during this case included oxygenation, prevention of acidosis, and maintenance of normothermia. Ultimately, these measures proved successful in maintaining normal neonatal circulation, and a return to fetal circulation was avoided.

This case highlights the importance of understanding the anesthetic implications and management during surgical resection of a SCT in a pediatric patient. The physiology of the pediatric patient must be considered to anticipate and manage potential complications that may occur. While surgical resection in the pediatric patient is risky, the survival rate is high and the literature supports an overall good prognosis.[3] A knowledgeable, prepared anesthetist that can easily recognize and treat complications is the patient's best defense against poor outcomes.

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Occupational Exposure to Ethylene Oxide and Pseudocholinesterase Deficiency

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Keywords: ethylene oxide, pseudocholinesterase deficiency, succinylcholine, neuromuscular blockade, and occupational exposure

Pseudocholinesterase deficiency is a genetic or acquired alteration in the metabolism of choline esters commonly found in anesthesia medications. While there are no direct negative health effects resulting from pseudocholinesterase deficiency, the most common consequence is prolonged paralysis and apnea following the administration of succinylcholine or mivacurium. Pseudocholinesterase is produced in the liver and numerous factors can lead to a deficiency including liver and renal disease, malnutrition, and exposure to organophosphate insecticides.[1] While some causes of pseudocholinesterase deficiency are evident in histories and physical exams, exposure to hazardous materials may be more difficult to assess, causing unanticipated prolonged paralysis.

Case Report

A 48-year-old, 177.5 cm, 102.9 kg male presented for cervical spine level 4-5/5-6/6-7 anterior cervical discectomy and fusion for management of herniated cervical discs with radiculopathy. His medical history was significant for hypertension and gastroesophageal reflux. His current medication regimen included enalapril, hydrochlorothiazide, pantoprazole, and acetaminophen. The patient's surgical history included left scapula lipoma excision, nissen fundoplication, and left inguinal hernia repair, none of which were associated with any anesthetic or surgical complications. The patient has worked for a medical equipment manufacturing company for over 25 years.

In the operating room, the patient was given intravenous (IV) midazolam 2 mg, fentanyl 50 mcg, hydromorphone 0.8 mg, and ondansetron 4 mg while being placed on noninvasive monitors. Pre-oxygenation was initiated via facemask at 10 L/min flow while a propofol infusion was started at 75 mcg/kg/min. After reaching a deep level of sedation, baseline motor and somatosensory evoked potentials were obtained, after which general anesthesia was induced with IV fentanyl 50 mcg, propofol 200 mg, and succinylcholine 140 mg. A direct laryngoscopy was then performed with a GlideScope (Verathon Inc., Bothell, WA) and an 8.0-cuffed endotracheal tube was advanced through the glottis. Positive bilateral breath sounds were auscultated and positive end tidal carbon dioxide was noted. The patient was then placed on volume control ventilation. General anesthesia was maintained with a total IV anesthetic technique. The propofol infusion was increased to 150 mcg/kg/min and supplemented with intermittent doses of hydromorphone totaling 2 mg and fentanyl totaling 350 mcg. During the procedure, motor evoked potentials were frequently assessed. The neurophysiologist stated that motor evoked potentials did not return until approximately 45 minutes after the administration of succinylcholine, indicating a prolonged neuromuscular blockade.

At the conclusion of the procedure, IV dexamethasone 6 mg was administered and the propofol infusion was discontinued. The patient was then weaned from the ventilator utilizing pressure support ventilation. Train-of-four monitoring and the ability to purposefully follow commands, including sustained head lift, indicated wakefulness and return from neuromuscular blockade. Once the patient was able to maintain adequate respiratory function based on tidal volume and respiratory rate, he was extubated and transferred to the post anesthesia care unit without incident.

During the post anesthesia evaluation, it was discovered that the patient had spent over 25 years working in the ethylene oxide sterilization department. Upon further questioning, he stated he was not required to use personal protective equipment (such as respiratory protection) unless he was going inside the sterilization chamber while it was in operation.

Discussion

Pseudocholinesterase, an enzyme formed in the liver and found in plasma, is responsible for metabolizing succinylcholine through a hydrolysis reaction.[2] Low levels or atypical variants of pseudocholinesterase can lead to prolonged neuromuscular blockade following administration of succinylcholine. Causes of decreased pseudocholinesterase include liver and renal disease, malnutrition, pregnancy, malignancy, burns, cardiopulmonary bypass, leprosy, and exposure to cholinesterase inhibiting drugs or toxins.[1,2]

In order to recognize and respond to a prolonged neuromuscular blockade associated with a pseudocholinesterase deficiency, anesthesia professionals should utilize peripheral nerve stimulators with administration of all neuromuscular blocking agents, including succinylcholine. If the patient does not exhibit a response to train-of-four stimulation 15 minutes after administration of succinylcholine, then a pseudocholinesterase deficiency should be suspected.[1]

In this case, the patient exhibited a prolonged neuromuscular blockade following the administration of succinylcholine. Many factors could result

in prolonged effects of succinylcholine related to deficient or defective pseudocholinesterase. The patient denied any episodes of prolonged neuromuscular blockade in either his or his family's anesthetic history. One unique aspect to this patient's history was his long-term occupational exposure to ethylene oxide. Ethylene oxide is a widely utilized industrial chemical often employed as a sterilizing agent, disinfectant, pesticide, and an intermediary in the synthesis of ethylene glycol and other products.[3, 4]

Ethylene oxide is considered a probable carcinogen and has been associated with the formation of non-Hodgkin's lymphoma, multiple myeloma, and chronic lymphoid leukemia, among numerous other illnesses.[3] The mechanism of carcinogenicity of ethylene oxide is not well understood but is thought to involve the creation of deoxyribonucleic acid (DNA) adducts and has been shown to be mutagenic in a variety of in vitro and in vivo systems.[4] DNA adducts are useful markers of exposure to environmental and endogenous genotoxicants. Identification of DNA adducts can be used to determine the risk of a genotoxic chemical, such as a pharmaceutical, in leading to development of cancer.[5]

The current standard treatment for prolonged neuromuscular blockade following administration of succinylcholine resulting from pseudocholinesterase deficiency involves waiting for spontaneous recovery. The time to spontaneous recovery depends on genetic and acquired factors that may be difficult to predict.[1] Other treatments that have been reported in the literature to reduce the time required to recover from succinylcholine include transfusion of whole blood, transfusion of fresh frozen plasma, and administration of human serum cholinesterase. While transfusion of whole blood has been shown to consistently reduce the time needed to recover from succinylcholine, fresh frozen plasma administration has shown less consistent results.[1] Human serum cholinesterase administration has been shown to shorten recovery time and has a safety profile similar to that of administration of human albumin, however its use is cost prohibitive and it is currently unavailable in the United States.[1]

Once an individual is suspected to possess a pseudocholinesterase deficiency, appropriate laboratory tests should be obtained, including serum pseudocholinesterase activity and dibucaine and fluoride inhibition tests. Measurement of serum pseudocholinesterase is a sensitive tool to measure the synthesizing capacity of the liver. A decrease in activity will reflect decreased liver production. The reference range is 2900 to 7100 units/L, but can vary widely by different laboratory standards and is subject to variability between patients. Dibucaine and fluoride are the most common inhibition tests and are used to identify the functional ability of the individual's enzymes.[1,2] It is important to utilize both the serum plasma cholinesterase level and inhibition tests such as dibucaine and fluoride to accurately determine if the prolonged apnea and neuromuscular blockade following succinylcholine is due to a genetic variant in plasma cholinesterase or the result of low levels of normal plasma cholinesterase.[1,2]

In this particular case, the surgical procedure was of sufficient length to allow for spontaneous recovery, which was frequently assessed using motor evoked potential monitoring. If insufficient time had elapsed, the patient would have been appropriately sedated until extubation criteria were met

and later informed of the situation. Family members should also be notified and tested. Those with pseudocholinesterase deficiencies should be identified and issued medical ID cards or bracelets notifying healthcare providers.[1]

Given the carcinogenicity of ethylene oxide, one could speculate that prolonged exposure could be linked to the development of a pseudocholinesterase deficiency due to its wide spread effects on cellular and molecular functioning. In order to characterize the cause of the prolonged neuromuscular blockade following administration of succinylcholine, serum pseudocholinesterase activity as well as dibucaine and fluoride inhibition tests should have been collected. Unfortunately, it is unknown if this patient received this testing to evaluate the source of his prolonged neuromuscular blockade.

In conclusion, this case serves as an example of the potential association between occupational exposure to chemicals such as ethylene oxide and the occurrence of unexpected reactions to medications. Further research is warranted to investigate the relationship between occupational exposure to hazardous materials and the development of pseudocholinesterase deficiencies.

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Mentor: Kelly L. Wiltse Nicely, PhD, CRNA

Hypotension in a Patient on Corticosteroids

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Keywords: adrenal insufficiency, corticosteroids, anesthesia, perioperative steroids, glucocorticoids

In surgery, it is common to encounter patients who are being treated with corticosteroids for various comorbidities. Patients who take chronic steroids may

exhibit symptoms of adrenal insufficiency during the perioperative period when stressed. Exogenous corticosteroids are often given prophylactically to patients who are at risk for adrenal insufficiency. Rather than administering a routine dose to every patient taking corticosteroids, a tiered regimen for dosing has been recommended.

Case Report

An 81-year-old male presented for elective endovascular repair of a 6.8 cm infrarenal aortic aneurysm. The patient's past medical history was significant for hypertension (baseline blood pressure 148/52), atrial fibrillation, permanent pacemaker implantation due to symptomatic bradycardia, aortic stenosis, type II diabetes mellitus, asthma, gastroesophageal reflux, obstructive sleep apnea, and benign prostatic hypertrophy. The patient's medical history also included chronic lymphocytic leukemia (CLL) for which he was treated with chemotherapy and corticosteroids. The patient's medications regimen included metformin, budesonide/formoterol, alprazolam, omeprazole, finasteride, atenolol 50 mg, losartan, and prednisone 10 mg daily. On the morning of surgery, the patient took oral atenolol 50 mg and prednisone 10 mg.

After the preoperative assessment was conducted, the patient was brought into the operating room and pulse oximetry, electrocardiogram, and non-invasive blood pressure monitors were applied. The patient was noted to be ventricularly paced at a rate of 60 bpm with a baseline blood pressure of 140/80 mmHg. Preoxygenation and denitrogenation was achieved with 100% oxygen applied by face mask. Induction of anesthesia and tracheal intubation was performed after the administration of fentanyl 50 mcg IV, lidocaine 100 mg IV, propofol 120 mg IV, and rocuronium 50 mg IV. Direct laryngoscopy was performed with a Miller 2 laryngoscope blade and the trachea was intubated with a 7.5 cuffed endotracheal tube. Correct placement of the endotracheal tube was confirmed with the presence of end-tidal CO_2 and auscultation of bilateral breath sounds. General anesthesia was maintained with sevoflurane 1.5-2.5% inspired concentration in a mixture of oxygen 1 L/min and air 1 L/min. A second 18 gauge peripheral intravenous (IV) catheter and left 20 gauge radial arterial line were placed after induction of anesthesia.

Bilateral femoral cutdown was utilized by the surgeon for surgical access during which the patient experienced a 1500 ml blood loss. The patient became hypotensive with systolic blood pressure (SBP) readings between 70-80 mmHg and mean arterial pressures (MAP) between 50-60 mmHg. Plasmalyte fluid administration and phenylephrine boluses IV (100 mcg increments) were used to treat the hypotension. As surgery progressed, the patient's blood pressure remained labile and a phenylephrine infusion was started at 50 mcg/min and titrated to maintain the blood pressure within 20% of the patient's baseline SBP of 140-150 mmHg. A complete blood count (CBC) was obtained and the results showed a stable hemoglobin of 10 g/dL and a hematocrit of 29.2%. The surgical procedure continued without any further significant blood loss. Total fluid administration for the procedure included 2500 ml plasmalyte and 500 ml of 5% albumin. Urinary output was measured at 50-100ml/ hr and total estimated blood loss was 1700ml. The phenylephrine infusion was weaned for a SBP greater than 110 mmHg and discontinued by the completion of the procedure. The patient was emerged and extubated in stable condition prior to

transport to the post anesthesia care unit (PACU).

Upon arrival to the PACU, the patient experienced a hypotensive event with a SBP of 80-90mmHg. A 500 ml plasmalyte fluid bolus was given and the phenylephrine infusion was restarted at 50 mcg/min. The patient remained hypotensive and an infusion of norepinephrine was initiated at 5 mcg/min and titrated to effect. Moderate infusion rates of both vasopressors were required to stabilize the patient's SBP's to range between 90-100 mmHg and a MAP of 60. Laboratory values remained unchanged and the patient remained awake and alert. Adrenal insufficiency was considered as a potential cause of hypotension and hydrocortisone 100 mg IV was given to the patient. The patient was then transported to the intensive care unit (ICU). The vasopressor infusions were weaned according to ICU hospital protocol and the patient maintained stable vital signs until the time of discharge.

Discussion

The body's response to stress is dependent upon the normal function of the adrenal glands. Acute adrenal crisis is a life threatening emergency that can be induced by surgical stress in patients who are taking chronic corticosteroid therapy. Cortisol facilitates catecholamine synthesis and contributes to vascular tone and cardiac contractility, and its production is controlled by the hypothalamic pituitary axis (HPA).[1]

Surgery is one of the most potent activators of the HPA.[2] "The degree of activation of the axis depends on the magnitude and duration of surgery and the type and depth of anesthesia".[1] When the HPA is stimulated by surgical stress, the suppressed patient is unable to respond normally by increasing cortisol production. This can result in hypotension, lethargy, hypovolemia, and ultimately cardiovascular collapse.[1,2] The prophylactic use of corticosteroids during the perioperative period may prevent these effects. The hypotension seen in this patient may have been an indicator of a suppressed HPA. The question of whether or not to administer steroids to this patient population remains controversial.

A systematic review of randomized controlled trials was conducted by Yong et al to determine the necessity of perioperative steroid supplementation in adult patients taking long term corticosteroids.[3] The review found that some studies have reported supplemental perioperative steroids were not required for patients with adrenal insufficiency.[3] However, these trials only included a total of 37 patients and owing to this small sample size the results may not be representative and applicable to a larger patient population.[3] There has been a shift in clinical practice in favor of administering lower doses of corticosteroids according to the duration and type of surgery.[2,4] This is consistent with the body's natural response to surgical stress; major surgery of long duration produces greater adrenal output than minor surgery of short duration.[5]

The trigger for hypotension seen in this case is unknown and may be multifactorial. Differential diagnoses include; hypotension secondary to acute blood loss, retroperitoneal bleeding, sepsis secondary to intestinal ischemia, a decrease in cardiac output secondary to atrial fibrillation and aortic stenosis, and anemia due to CLL. Adrenal insufficiency can certainly be considered as a contributing factor to the hypotension seen in this case. This patient experienced a significant amount of blood loss in a short period of time. Normally, patients are able to compensate for this type

of insult when treated appropriately with volume and vasopressors as needed. However, this patient required the prolonged administration of vasopressors and was not able to compensate. The patient had been on chronic steroid therapy since 2011, which had been tapered down to his recent regimen of prednisone 10 mg daily. Patients receiving 10 mg/ day may have HPA suppression and the literature suggests supplementation.[1,6] In this case, the benefits of steroid supplementation outweighed the side effects such as delayed wound healing, immunosuppression and increased blood glucose levels. Additionally, it was found that short courses of increased steroid therapy rarely cause significant complications.[2]

A tiered approach to perioperative steroid supplementation with hydrocortisone has been proposed by Jung et al.[2] Lower doses (25mg) are suggested for minor surgery (laparoscopic cholecystectomy) with higher doses (50-75 mg) for moderate surgery (colon resection) and 100-150mg for major surgery (cardiothoracic, liver).[5] These doses are to be given by IV injection prior to the induction of anesthesia. By following this guideline, steroid dosing is tailored to each individual patient. The length of surgery and severity of surgical stress are considered, and the minimal therapeutic dose is given.[2,5]

Since the patient took his usual dose of prednisone the morning of surgery, the anesthesia care team concluded that additional supplementation was not necessary. Additional steroids were not administered until the patient arrived in the PACU with ongoing hypotension. The effect of hemodynamic stabilization secondary to the administration of corticosteroids in this case is inconclusive. The literature review supports the use of supplemental steroids in this patient population, especially if intraoperative events occur that increase physiologic stress and hemodynamic instability. In combination with good clinical judgment these proposed guidelines can assist the anesthesia practitioner to determine an appropriate dose for steroid supplementation.

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Mentor: Brenda A Wands, CRNA, MBA, PhD

Editorial

I would like take this time to extend my sincere gratitude to all of my editors and reviewers for their hard work this summer in helping me work through our abundance of submissions. It is wonderful that we have so many programs participating, using this journal as a means to introduce their students to the process of publication. It has been a very busy season trying to catch up and I am almost there! I do want this to be a positive experience for everyone, and my goal is to keep submission to publication turnaround at about three months – sometimes I succeed and sometimes I don't, so I appreciate everyone's patience and hard work in this purely volunteer endeavor. I was very fortunate to recruit and welcome a number of new reviewers, and I would also like to welcome the following individuals who have joined our Editorial Board during the past year:

Laura S. Bonanno, CRNA, DNP - Louisiana State University Health Sciences Center
CDR Johnnie Holmes, CRNA, PhD, NC, USN - Uniformed Services University
CDR Justice Parrott, CRNA, DNAP, NC, USN - Uniformed Services University
Jo Ann Platko, CRNA, BC, PhD - Somnia Anesthesia, Inc.

These additions have helped tremendously, but we can always use more reviewers. Or, if having served as a reviewer you are interested in joining the editorial board please contact me!

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.**"

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

www.aana.com/studentjournal

INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA
GUIDE FOR AUTHORS

MISSION STATEMENT

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

ITEMS ACCEPTED FOR PUBLICATION

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

ITEM PREPARATION & SUBMISSION

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

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

PEER REVIEW

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

General guidelines

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

2. Report appropriate infusion rates and gas flow rates:
    a. When reporting infusion rates report them as mcg/kg/min or mg/kg/min. In some cases it may be appropriate to report dose or quantity/hr (i.e. insulin, hyperalimentation). If a mixture of drugs is being infused give the concentration of each drug and *report the infusion rate in ml/min*.
    b. Keep the gas laws in mind when reporting flow rates. Report the liter flows of oxygen and nitrous oxide and the percent of the volatile agent added to the gas mixture. Statements such as "40% oxygen, 60% nitrous oxide and 3% sevoflurane" do not = 100% and are thus incorrect. For example, "General anesthesia was maintained with sevoflurane 3% inspired concentration in a mixture of oxygen 1 L/min and air 1 L/min".
3. Only Microsoft Word file formats will be accepted with the following criteria:
    a. Font - 12 point, Times New Roman
    b. Single-spacing (except where indicated), paragraphs separated with a double space (do not indent)
    c. One-inch margins
    d. Place <u>one</u> space after the last punctuation of sentences. End the sentence with the period before placing the superscript number for the reference.
    e. Do not use columns, bolds (except where indicated), or unconventional lettering styles or fonts.
    f. Do not use endnote/footnote formats.
4. Do not use Endnotes or similar referencing software. Please remove all hyperlinks within the text.
5. Avoid jargon.
    a. *'The <u>patient</u> was reversed'* - Did you physically turn the patient around and point him in the opposite direction? "Neuromuscular blockade was antagonized."
    b. *The patient was put on oxygen.* "Oxygen was administered by face mask."
    c. *The <u>patient</u> was intubated and put on a ventilator*. "The trachea was intubated and respiration was controlled by a mechanical ventilator.
    d. *The patient had been on Motrin for three days.* "The patient had taken ibuprofen for three days."
    e. Avoid the term "MAC" when referring to a sedation technique - the term sedation (light, moderate, heavy, unconscious) sedation may be used. Since all anesthesia administration is monitored, the editors prefer to use specific pharmacology terminology rather than reimbursement terminology.
6. Use the words "anesthesia professionals" or "anesthesia practitioners" when discussing all persons who administer anesthesia (avoid the reimbursement term "anesthesia practitioners")
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. Diag must have permission from original author. This is the most important part of the article. In terms of space and word count this should be longer than the case presentation. End the discussion with a summary lesson you learned from the case, perhaps what you would do differently if you had it to do over again.

[space]

References (bold)

[space]

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

[space]

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

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

Research Abstracts

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

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

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

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

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

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

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

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

AMA MANUAL OF STYLE

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

ACADEMIC INTEGRITY
Issues of academic integrity are the primary responsibility of the author and mentor. Accurate and appropriate acknowledgement of sources is expected. **Any violation will be cause for rejection of the article.**

"Plagiarism is defined as the act of passing off as one's own the ideas, writings, or statements of another. Any act of plagiarism is a serious breach of academic standards, and is considered an offense against the University subject to disciplinary action. Any quotation from another source, whether written, spoken, or electronic, must be bound by quotation marks and properly cited. Any paraphrase (a recapitulation of another source's statement or idea in one's own words) or summary (a more concise restatement of another's ideas) must be properly cited."
http://grad.georgetown.edu/pages/reg_7.cfm

HOW TO SUBMIT AN ITEM
Manuscripts must be submitted by the mentor of the student author via e-mail to **INTSJNA@aol.com** as an attachment. The subject line of the e-mail should be "Submission to Student Journal". The item should be saved in the following format – two-three word descriptor of the article_author's last name_school abbreviation_mentor's last name_date (e.g. PedsPain_Smyth_GU_Pearson_5.19.09)

REVIEW AND PUBLICATION

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

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

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

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

PHOTOS

Photos of students for the front cover of the Journal are welcome. Include a legend describing the activity and who is in the photo and identify the photographer. Only digital photos of high quality will be accepted via email to INTSJNA@aol.com. There must be a follow up hard copy signed by all present in the photo, as well as the photographer/ owner of the original photo, giving consent to publish the photo. Mail that consent to:

Vicki C. Coopmans, CRNA, PhD
Goldfarb School of Nursing at Barnes-Jewish College
4483 Duncan Ave., Mailstop 90-36-697
St. Louis, MO 63110

SUBMISSION CHECK LIST

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

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

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

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

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

References
___ AMA Style for referencing is used correctly.
___ Reference numbers are sequenced beginning with one and superscripted.
___ References are from anesthesia and other current primary source literature.
___ All inclusive pages are cited, texts as well as journals.
___ Journal titles are abbreviated as they appear in the PubMed Journals Database.
___ Number of references adheres to specific item guidelines.
___ Internet sources are currently accessible, reputable, and peer reviewed.

Transmission
___ The article is sent as a attachment to **INTSJNA@AOL.COM**
___ The file name is correctly formatted (e.g. PedsPain_Smyth_GU_Pearson_5.19.09)
___ It is submitted by the mentor with cc to the student author
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