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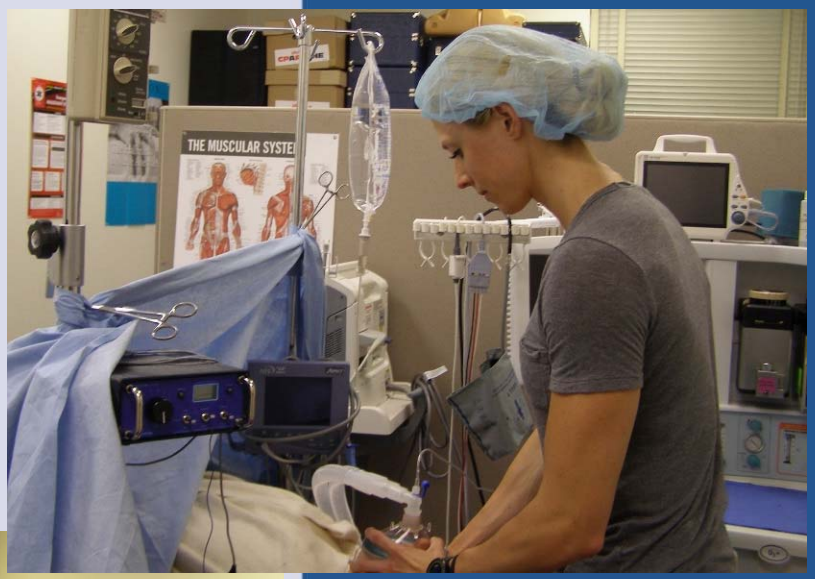

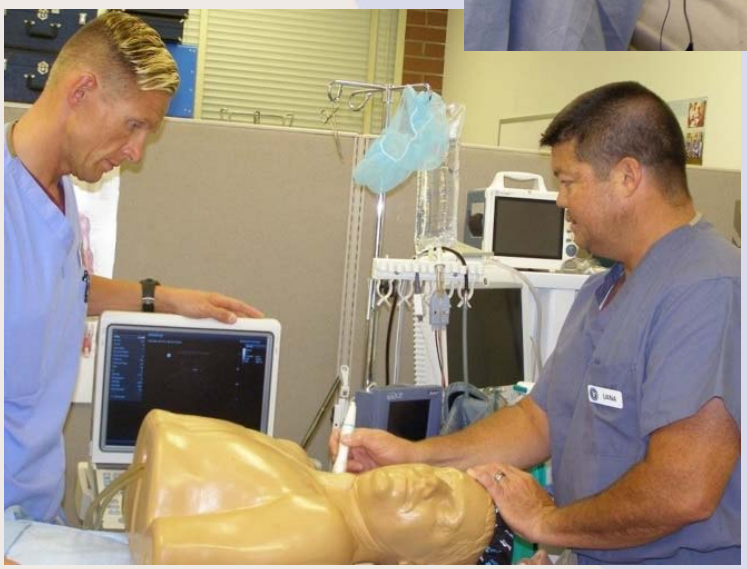

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Front Cover:

Pictured above, Felicia Anderson, RN, BSN, a graduate student enrolled in the Westminster College nurse anesthesia program (NAP) provides anesthesia in the program's high-fidelity simulated operating room. Pictured below, Art Shimata, CRNA, MAE, professor for the Westminster College NAP works with Dan Bunker, CRNA, MSNA, new assistant professor and alumnae of the Westminster NAP, in the program's simulation laboratory to plan instruction on ultrasound-guided nerve blocks.

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Evidence-based Practice Analysis Reports

Amrutha Panakkal, MSN
University of Pennsylvania

Keywords: supraorbital craniotomy, perioperative hematoma, nasopharyngeal osteosarcoma

The endoscopic supraorbital approach to craniotomy has changed the amount of exposure needed to operate on tumors located at the cranial base. Complications such as transient supraorbital nerve injury, cerebrospinal fluid leak, rhinorrhea and pseudomeningocele formation are known risks.[1] However, less well documented is the formation of a hematoma during the perioperative or immediate postoperative period, which may arise from prolonged surgery via the supraorbital route. Hematoma formation around the orbit must be rapidly identified and treated in order to prevent permanent damage to the globe and optic nerve.

Case Report

A 31-year-old, 165 cm, 66 kg male presented for resection of a right skull base nasopharyngeal osteosarcoma via supraorbital craniotomy. His medical history included essential hypertension, nasopharyngeal cancer, right cranial nerve VI compressive neuropathy and cerebrovascular accident (CVA) three years prior with no residual extremity weakness. His past surgical history included a percutaneous gastrostomy insertion post CVA that was not associated with any anesthetic complications. His current medication regimen included dexamethasone and hydrochlorothiazide.

Pre-operative airway evaluation identified a Mallampati classification III, a thyromental distance less than 6 cm, an inter-incisor distance less than 4 cm, intact dentition, and narrow neck with full range of motion predicting a difficult airway. The anesthesia team planned an awake fiberoptic intubation to secure the airway for the procedure.

In the pre-operative area, intravenous (IV) access was established and the patient's oral and hypopharynx was topicalized with 4% lidocaine jelly 5 mL applied to a gauze-wrapped tongue depressor. The patient was brought into the operating room (OR) and standard American Society of Anesthesiologists monitors were applied with oxygen supplementation at 5 L/min via a nasal cannula. Midazolam 2 mg IV was administered for anxiolysis and a 7.0 mm cuffed endotracheal tube (ETT) was passed through the larynx using a pediatric fiberoptic scope following an unsuccessful attempt with an 8.0 mm cuffed ETT. Once end-tidal carbon dioxide ($ETCO_2$) was confirmed and bilateral breath sounds were noted, anesthesia was induced with propofol 200 mg, rocuronium 30 mg and dexamethasone 10 mg were administered IV. General anesthesia was maintained with Sevoflurane 1.8 – 2.2% inspired concentration in a mixture of oxygen 1 L/min and air 1 L/min.

The surgical procedure was uneventful. Upon conclusion of the five hour case, neuromuscular blockade was antagonized with neostigmine 3 mg and glycopyrolate 0.5 mg IV. When the patient was able to maintain a head lift for >5 seconds, had a + 5 hand grip with bilateral upper extremities and consistently inspired tidal volumes greater than 400 ml, the ETT was removed.

Following extubation, blood began to ooze from his right eye socket and a hematoma was noted which

continued to develop over time. The anesthetic team titrated midazolam 1 mg IV and fentanyl 75 mcg IV to the patient's comfort while maintaining a patent airway. Prior to extubation, a total of hydromorphone 1 mg and fentanyl 250 mcg was administered throughout the case. The surgeon immediately identified, assessed, and evacuated the hematoma. An ophthalmologist was consulted to assess the patient's eyesight in the OR. The patient's vision was determined intact and he was transported to post-anesthesia care unit (PACU). In the PACU and throughout his hospital stay, the patient did not have any further deterioration of sight in his right eye.

Discussion

The supraorbital endoscopic approach to craniotomy is characterized as the keyhole principle, a small incision that allows wide access to deeper surgical lesions without the need for fixed brain retraction.[1] Prior to the supraorbital endoscopic approach, the "cranio-orbito-zygomatic" or "bifrontal approach" was used for tumors at the cranial base.[2,3] These craniotomies required prolonged retraction of the frontal lobes and an incision that involved the forehead and scalp.[2,3,4] The supraorbital endoscopic approach is being increasingly favored by neurosurgeons because it decreases the amount of trauma to the patient and provides increased exposure for surgery.[3,5] This approach has also become cosmetically appealing to patients.[4] Although the incision is small, there remain complications associated with this minimally invasive approach.

In order to avoid injury to the neural and vascular structures around the supraorbital incision site, an in-depth knowledge of anatomic landmarks is required.[2] Complications are very rare in this type of craniotomy, but some of the potential complications discussed in the literature are transient forehead numbness, transient frontalis weakness, pseudomeningocele and cerebrospinal fluid (CSF) rhinorrhea.[3] Damage to the supraorbital nerve is the cause of transient forehead numbness and it is one of the common complications in the early postoperative period.[3] Transient frontalis weakness from injury, which is caused by stretching of the frontalis branch of the facial nerve, can also be seen immediately after surgery.[3] Both transient forehead numbness and transient frontalis weakness are related to the soft tissue dissection and retraction of surgery.[3]

Pseudomeningocele can occur when CSF collects in the pocket of space created over the frontal bone and CSF rhinorrhea may occur if the frontal sinus is interrupted or inadequately repaired.[3] If the patient develops CSF rhinorrhea postoperatively and requires immediate repair of the leak, anesthesia providers need to be cautious in providing positive pressure mask ventilation during induction, as this might cause pneumocephalus.[1] Additionally, a pneumocephalus may also occur when positive pressure is present in the upper airway, as with an airway obstruction, because air can be forced "into the skull through any openings of the facial bone."[1]

Smooth emergence is critical in endoscopic supraorbital craniotomies due to the increased risk of pneumocephalus and also pseudomeningocele.[1] Applying gentle pressure over the surgical site during extubation can decrease the incidence of these potential complications.[3] During supraorbital approach craniotomy the anesthesia provider has limited access to the airway as the operating table is turned 180 degrees.[6] Therefore, it is important to extubate the patient after the anesthesia provider has complete access to the airway and necessary anesthesia equipment. During emergence, coughing and bucking on the endotracheal tube should be avoided as this may cause bleeding in the surgical site, resulting in hematoma formation.[6] Extubating the patient under deep anesthesia may ameliorate this complication

decreasing the potential for coughing. However, the decision to extubate the patient deep versus awake will depend on if securing the airway was easy or difficult and risk of aspiration.[6,7]

During this case, recognizing signs and symptoms of a hematoma was critical. Supraorbital hematoma can quickly develop into a periorbital hematoma and a periorbital hematoma can progress into a retrobulbar hemorrhage.[8] Retrobulbar hemorrhage in turn may lead to blindness due to the untreated pressure maintained on the optic nerve.[8] Immediate assessment of the patient's visual acuity is a priority because changes in visual acuity can be an early indicator of retrobulbar hemorrhage.[8] Additional signs and symptoms include an afferent pupillary defect, resistance to retropulsion, proptosis or increased intraocular pressure.[8] While the consequences of a periorbital hematoma progressing to a retrobulbar hemorrhage are detrimental, the incidence of developing a hematoma post supraorbital craniotomy is low. A study conducted by Kabil and Shahinian (2006), looked at complications post supraorbital endoscopic approach and indicated that there was no evidence of hematoma formation postoperatively in their study group. Incidentally one patient in the study group developed transient swelling and reddening of the periorbital area.

Endoscopic supraorbital craniotomy is a safe and effective method for surgically removing tumors located at the base of the skull. Similar to other endoscopic craniotomies, this approach carries some complications. These complications can be prevented by a meticulous surgical approach and a smooth emergence from anesthesia. Minimal retraction and manipulation of the orbicular and frontal muscular layer is essential to avoid a postoperative periorbital hematoma. A supraorbital hematoma can form with greater manipulation.[4] Early opioid administration, especially hydromorphone, aids in the patient's comfort post-extubation. This can facilitate hematoma evacuation if warranted, such as was the case with the patient presented. Applying pressure at the surgical site during extubation may also prove effective in prevention of hematoma formation. Supraorbital hematoma is a very rare complication that can develop after endoscopic supraorbital craniotomy. However, as the number of patients that opt for minimally invasive craniotomies increases, the incidence of this complication may rise.

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Acute Intermittent Porphyria

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Keywords: acute intermittent porphyria, ketamine, propofol, sevoflurane, sufentanil

Acute Intermittent Porphyria (AIP) is a rare, inherited disorder affecting the liver heme synthesis pathway which is regulated by aminolevulinic acid (ALA) synthetase, the rate limiting step in the production of heme. Many anesthetic drugs require the cytochrome P450 for metabolism, which increases the production of ALA synthetase and thus heme. When the normal pathway is blocked, enzyme substrates can accumulate causing an acute porphyria crisis. For patients at risk for AIP the anesthesia provider should choose anesthetic agents that will minimize the chance of triggering an acute porphyria attack during the peri-operative period.[1-4]

Case Report

A 37-year-old, 97 kg, 175 cm male, presented for a C4-C7 anterior cervical discectomy and fusion with a diagnosis of spondylosis with myelopathy. The patient's past medical history included AIP with two episodes of pancreatitis requiring hospitalization within the prior six months and report of alcohol use of undetermined frequency. The patient suffered from chronic neck pain and was taking klonopin, gabapentin, oxycontin, and oxycodone. With the use of these medications he reported that his level of pain was never below a 6/10. The patient had limited neck range of motion and his airway exam indicated he was Mallampati class II.

An 18 gauge peripheral intravenous catheter (PIV) was started in the preoperative area and midazolam 5 mg intravenous (IV) was administered before transporting him to the operating room (OR). Upon arrival to the OR, standard monitors were placed and the patient was preoxygenated with O_2 10 L/min. The patient received lidocaine 100 mg, propofol 200 mg, fentanyl 250 mcg, and rocuronium 50 mg IV for induction of anesthesia. The trachea was easily intubated while using the Glidescope (Verathon Inc., Bothell, WA) with cervical spine stabilization by a second anesthesia professional. Respirations were controlled with a mechanical ventilator with a mixture of oxygen 1 L/min and air 1 L/min.

A second 18 G PIV, right radial arterial line, and foley catheter were placed after induction of anesthesia. Needle electrodes were placed to monitor somatosensory evoked potentials (SSEP) and motor evoked potentials (MEP). Anesthesia was maintained throughout the case with sevoflurane at 0.5 minimum alveolar concentration (MAC), a propofol infusion at 100-150 mcg/kg/min for a total of 3250 mg, a sufentanil infusion of 0.3-0.5 mcg/kg/hr for a total of 230

mcg, which were all discontinued 30 min prior to the end of the case. He also received a 2-hour ketamine infusion, started at the beginning of the case at 0.2 mg/kg/hr for a total of 38 mg. The patient received dexamethasone 10 mg, cefazolin 2 g, and hydromorphone 2 mg IV prior to incision.

Near the end of the 8-hour case, the patient received acetaminophen 1 g and ondansetron 4 mg IV. The total IV crystalloid fluid intake was 2550 mL, blood loss was 100 mL, and 1000 mL of clear yellow urine was recorded. At the conclusion of the case, the patient was not spontaneously breathing or responding to noxious stimuli despite discontinuation of the sevoflurane and all infusions. Neostigmine 5 mg with glycopyrrolate 0.6 mg was administered despite receiving only an initial dose of rocuronium and full neuromuscular recovery as indicated by four out of four twitches followed by sustained tetany on the neuromuscular monitor. The patient was also dosed with naloxone 0.04 mg IV. After an hour of an unsuccessful emergence in the OR, the patient remained intubated and was transported to the post anesthesia care unit where respirations were controlled with a mechanical ventilator. He met criteria for extubation 1.5 hours later and was discharged home the following day with no evidence of an acute AIP crisis.

Discussion

AIP is a rare (1:20,000), autosomal dominant inherited metabolic disease affecting the heme synthesis pathway causing an overproduction of porphyrins.[2,4,5] The synthesis of porphyrins occurs as a result of a series of pathway enzymes. A defect, specifically in the enzyme porphobilinogen deaminase, part of the heme synthesis pathway, is found in patients with AIP thereby causing a buildup of pathway intermediaries, interfering with the negative feedback loop. An increase in heme requirements, such as the induction of cytochrome P450 can result in an accumulation of pathway intermediates. Many drugs commonly administered in anesthesia induce ALA synthetase, which is dependent on the formation of heme. The production of ALA synthetase is increased in order to increase production of heme in response to drugs that utilize cytochrome P450 for drug metabolism. The increase in ALA synthetase stimulates the heme production pathway causing an accumulation of enzymes, because of the defective porphobilinogen deaminase and thus the production of excessive amounts of porphyrin. An acute crisis can be caused by either a porphyrin accumulation or reduction in the production of heme. An acute crisis is manifested as severe abdominal pain, nausea, vomiting, neuropathies, tachycardia, or hypertension.[1-4]

The literature indicates that barbiturates, anti-epileptics, sulfonamide antibiotics, and ketorolac should be avoided in patients with known AIP, whereas other medications such as opioids, propofol, nitrous oxide, many local anesthetic agents, and neuromuscular blockers are considered safe.[1-5] Controversial medications include sevoflurane, etomidate, and ketamine which may contribute to an acute crisis.[1,3,4] It is important in the perioperative setting to be aware that other conditions such as dehydration, alcohol use, fasting, pain, psychological stress, infection, and nausea, and vomiting can trigger an acute attack.[2,3]

Propofol, opioids, rocuronium, and acetaminophen have been proven to be safe drugs and can be administered to a patient with AIP without precipitating an acute crisis.[2-5] Propofol is considered the drug of choice for induction of anesthesia in AIP patients.[2,3] Researchers and experts in

porphyria recommend desflurane as the preferred volatile anesthetic agent. Nitrous oxide has also been used safely in patients with AIP.[2-4,6] Enflurane is absolutely contraindicated and evidence is inconclusive regarding the other volatile anesthetics. Although there is questionable research pertaining to the use of sevoflurane, there have been several case reports of it safely being used for general anesthesia,[1,2,6] Ketamine is another controversial agent as the evidence is limited and conflicting. Some experts recommend avoiding the ketamine in patients with known AIP because they believe it can contribute to delirium that may mask an acute crisis while some endorse its use.[1,3,4] Researchers have also found that prolonged administration of any anesthetic agent including propofol may contribute to an acute crisis; therefore, using short-acting agents for the minimal amount of time possible is recommended.[2-4]

The length of the surgery, the patient's chronic pain, and the neurosurgical monitoring required for the surgery dictated the choice of drugs and infusions to maintain adequate anesthesia. Additionally, his chronic pain history further complicated the choice of anesthetic agents. It was important to maintain a satisfactory level of analgesia with a variety of drugs that act on the various pain receptors to avoid an acute crisis.[4,7]

While the patient in this case did not appear to suffer an acute porphyria attack within the first 24-hours postoperatively, he had delayed emergence from anesthesia necessitating mechanical ventilation into the recovery period. The exact cause for delayed emergence from anesthesia could be due to a variety of factors related or unrelated to the medications given. Anecdotal case studies and dated research guide the anesthetist's knowledge of safe, unsafe and undetermined safety of anesthetic drugs. In addition, the stress of surgery, infection or other factors such as alcohol use, drugs taken by the patient prior to surgery or electrolyte disturbances may have contributed to the delayed awakening.

Although it can never be conclusively determined the cause for the delayed awakening after anesthesia in this case, there are factors that may have improved the outcome. Using Bispectral Index monitoring (Aspect Medical Systems, Inc.) during this case could have better guided the anesthetic plan. Using desflurane rather than sevoflurane may have been a better volatile anesthetic of choice. The prolonged infusion of propofol or the combination of the several anesthetic modalities administered may have contributed to the delayed awakening.

There is much to be discovered about AIP and the associated crises that can develop after receiving anesthetic drugs. No current clinical research has been conducted on this rare disease and it is unlikely that AIP will receive funding for studies. Therefore, much of the information that is derived from anesthetic management is related to dated studies, clinical case reports, and expert opinions available in the research. It is recommended that anesthesia professionals continue to publish AIP case reports that discuss the specific drugs utilized in anesthesia with their associated outcomes.

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Total Intravenous Anesthesia for Malignant Hyperthermia

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Keywords: malignant hyperthermia, total intravenous anesthesia, propofol infusion, laryngeal mask airway, anesthetic triggering agents

Malignant hyperthermia (MH) is a hypermetabolic condition that occurs in genetically susceptible patients after being exposed to an anesthesia triggering agent.[1] Malignant hyperthermia cases are rare, but can be fatal, with an estimated incidence around 1:50,000.[2] The most common triggering anesthetics include succinylcholine and all volatile anesthetic agents.[1] Delivering anesthesia to a patient with a history of malignant hyperthermia presents a distinctive anesthetic challenge. The following case explains the importance of adequate preparation and the use of total intravenous anesthesia (TIVA) in the anesthetic management in a patient at risk for malignant hyperthermia.

Case Report

A 29-year-old, 150 cm, 79 kg female with a left ankle fracture presented for removal of hardware from the left ankle. Her medical history was significant for hypertension, asthma, Type 1 Diabetes, migraines, penicillin allergy, bipolar and anxiety disorders, anemia, and a personal history of malignant hyperthermia with prior surgery. The operating room (OR) was prepared by the anesthesia team by placing a new breathing circuit and new carbon dioxide absorbent on the anesthesia machine. The vaporizers were removed, and the anesthesia machine was flushed with oxygen at a rate of 10 L/min for 30 min. Different anesthesia machines and hospitals have varying MH protocols, but all of them stress the importance of flushing the machine with oxygen. The hospital's standard malignant hyperthermia cart was placed inside the OR suite for immediate access if necessary, and the contents of the cart were verified.

A peripheral intravenous (IV) line was started, and midazolam 2mg IV along with fentanyl 100 mcg IV was administered as premedication. A popliteal and a saphenous nerve block were performed using 0.5% bupivacaine, and catheters were left in place for each block. Once the patient was transported into the OR, the patient was positioned supine on the OR table, and monitors including a pulse oximeter, electrocardiograph, noninvasive blood pressure cuff, and cutaneous temperature probe were applied. Oxygen via face mask was administered at 12 L/min for pre-oxygenation. The left lower extremity was assessed to be numb to touch by the surgeon. Induction medications included lidocaine 20 mg and propofol 200 mg IV. A laryngeal mask airway (LMA), size #4, was inserted. Bilateral breath sounds and end tidal carbon dioxide ($EtCO_2$) were confirmed. Oxygen via the LMA was administered at a rate of 4 L/min. A continuous infusion of propofol was started at a rate of 100 mcg/kg/min. Vancomycin 1.5 g IV was administered as antibiotic therapy.

The patient maintained spontaneous ventilation through the LMA, with $EtCO_2$ between 35 and 40 mm Hg. The patient remained hemodynamically stable, with a heart rate between 60 and 75/min and systolic blood pressure ranging from 110 to 140 mm Hg. Titration of the rate of the propofol infusion and fentanyl boluses was based on patient movement and vital signs, such as tachycardia or hypertension.

In total the patient received propofol 800 mg and fentanyl 450 mcg throughout the entire 120-min case. The patient received 1200 mL of Lactated Ringers and estimated blood loss was 15 mL. Ondansetron 4 mg IV was administered as an antiemetic. The patient was transferred to the Post Anesthesia Care Unit (PACU) where she remained for an additional 120 min. She required fentanyl 50 mcg IV bolus three times for pain scores of 6 out of 10, for a total of fentanyl 150 mcg. In addition, the block team re-dosed her popliteal and saphenous nerve blocks with diluted bupivacaine for postoperative pain relief via block catheters. Vital signs remained at baseline. No tachycardia, sweating, hyperthermia, arrhythmias, muscle rigidity, or change in urine color noted throughout the procedure.

Discussion

Malignant hyperthermia is a hypermatabolic condition generally occurring in genetically susceptible patients, typically with an autosomal dominant mutation, after being exposed to an anesthetic triggering agent.[1] The triggering anesthetics include all inhalational agents and succinylcholine, a depolarizing neuromuscular blocker.[2] Malignant hyperthermia is most likely caused by a reduction in the reuptake of calcium by the sarcoplasmic reticulum, which is needed to stop muscle contraction. When this occurs, muscle contraction is sustained, causing signs of hypermetabolism and hyperthermia.[1,3]

A personal or family history of anesthetic problems, such as unexplained fevers or death during anesthesia, or personal history of malignant hyperthermia suggests susceptibility for malignant hyperthermia, and the anesthesia plan should take special attention to avoid a crisis.[1] The MH cart, with 36 vials of dantrolene, should be available in the OR should a crisis occur. Dantrolene, a muscle relaxant, is the treatment for MH, inhibiting calcium release from the sarcoplasmic reticulum.[3] Due to the possibility of residual inhalational agent, the anesthesia machine needs to be prepared by changing the carbon dioxide absorbent and fresh gas tubing, disconnecting the

vaporizers, using a disposable breathing circuit, and flushing the machine with oxygen at a rate of 10 L/min for at least 10 minutes.[1,2] My hospital policy suggests flushing the machine for 30 minutes, so that is what we did. Local or regional anesthesia should be considered, but general anesthesia using non-triggering agents is acceptable.[1] In this case, peripheral nerve blocks were administered preoperatively, however it was determined that the blocks were not going to provide adequate pain relief alone, and TIVA was needed for general anesthesia.

Larach et al studied 291 cases involving patients that experienced a malignant hyperthermia event, and reported that 8 (2.7%) resulted in cardiac arrests and 4 (1.4%) resulted in death.[4] Early signs of malignant hyperthermia should be closely monitored, so treatment can be initiated immediately. Early clinical signs include elevated CO_2, sweating, tachycardia, arrhythmias, unstable arterial pressure, masseter spasm, and muscle rigidity. Later signs include hyperkalemia, hyperthermia, elevated blood creatine phosphokinase and blood myoglobin levels, dark colored urine associated with myoglobinuria, and cardiac arrest.[2] If MH is suspected, discontinue all triggering agents, hyperventilate with 100% oxygen at high flow, convert to TIVA if not already using this method, stop surgery, disconnect the vaporizer, change the circuit and machine, obtain labs, and treat symptoms appropriately.[1,2] Dantrolene 2.5 mg/kg IV (to maximum dose of 10 mg/kg) should be administered. Larach et al also reported that the mortality rate for MH was 70% without Dantrolene administration, but decreased to 1.4% when promptly treated with Dantrolene.[4]

Inhalational agents had to be avoided in this patient because of her personal history of MH, so general anesthesia was maintained with a propofol infusion and fentanyl boluses. Propofol is considered the most suitable anesthetic agent for TIVA.[5] Propofol allows for quick changes in anesthetic depth, and a speedy recovery, due to its ability to be rapidly metabolized. The low context–sensitive halftime makes propofol the best available agent for TIVA procedures.[3,5] Lastly, propofol attenuates airway reflexes so the LMA may be positioned easily without neuromuscular blockade, which was useful in this case.[5]

Since the patient was a low risk for aspiration, surgical time was under 2 hours, and succinylcholine had to be avoided, an LMA was used to maintain the patient's airway during the case as opposed to an endotracheal tube (ETT). Researchers at the University of Cape Town found that the LMA, compared to the ETT, was a safe alternative airway during TIVA. This study compared four groups of patients receiving TIVA with either an LMA or an ETT, and either neuromuscular blockade or no blockade. The study found that the patients in the group with an LMA and no neuromuscular blockade were more hemodynamically stable than those with an ETT. The LMA was found to be an effective device during TIVA, and easier to manage than the ETT in the absence of neuromuscular blockade.[6] Since neuromuscular blockade was not utilized in this case, the LMA was a safe alternative airway.

As an anesthesia practitioner, avoiding MH in a susceptible patient, while maintaining general anesthesia and a hemodynamically stable patient, was my goal. Recognizing this patient was at high risk for MH because of her personal history of MH, an anesthetic without any triggering agents was necessary. TIVA with a propofol infusion and fentanyl boluses, along with both a popliteal and saphenous nerve block, was my choice to diminish the risk of anesthetic complication, since it avoided inhalational anesthetics and succinylcholine. The patient's post

anesthesia course, both without excruciating pain or any signs of MH, confirmed the anesthetic was a success. In future cases, TIVA could be established with dexmedetomidine instead of propofol, since new studies are showing how this alpha-2 agonist provides safe and effective sedation, amnesia, and analgesia without respiratory depressant effects, and without triggering MH.

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High Spinal in a Morbidly Obese Patient

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Keywords: high-spinal, total spinal, morbid obesity, spinal anesthesia, neuraxial anesthesia

High and total spinals are acute, potentially life threatening risks of spinal anesthesia, occurring when the cephalad spread of the local anesthetic (LA) results in an unexpected high neuroblockade.[1] The morbidly obese are a rapidly growing segment of the United States population.[2-4] There is evidence that increased BMI correlates to increased block height with neuraxial anesthesia.[2] This suggests that the morbidly obese may be at increased risk of high or total spinals when dosing is not adjusted in this population. Symptoms of high and total spinals must be identified and treated early, to prevent devastating consequences, even death.[1]

Case Report

A 170 cm, 141 kg, 64-year-old male with a BMI of 48.7 kg/m^2 was scheduled for a total knee arthroplasty due to osteoarthritis. His medical history included: morbid obesity, osteoarthritis, hypertension, atrial fibrillation, obstructive sleep apnea (OSA), and diabetes mellitus. The patient's current medications included: warfarin, potassium chloride, furosemide, lisinopril, testosterone, bupropion, diltiazem, metformin and simvastatin. He had no known drug allergies. The last dose of warfarin was five days preceding surgery and the international normalized ratio was 1.2.

In the preoperative area intravenous access was secured, oxygen was administered by nasal cannula at 4 L/min, noninvasive monitors were applied, and midazolam 4 mg was given before peripheral nerve blocks were placed. Femoral and sciatic blocks were achieved each using 20 mL ropivacaine 0.5%. A catheter was threaded into the femoral nerve block space and secured. Replacement of the patient's calculated fluid deficit was started preoperatively as spinal anesthesia was planned for the surgery. One liter of Lactated Ringer's (LR) solution had been infused before the patient was taken to the operating room.

In the operating room, oxygen administration was continued by nasal cannula and noninvasive monitors were reapplied. The patient was placed in the sitting position to perform the subarachnoid block (SAB). Due to the patient's body habitus we were unable to locate bony landmarks indicative of the lower lumbar interspaces nor determine exactly which interspace was injected. The injection site was the lowest level where spinous processes were palpable. Visually, the space injected was estimated to be approximately L1. Free flow of cerebrospinal fluid was confirmed via aspiration. No blood was aspirated and the patient experienced no paresthesias during placement of the SAB. Isobaric 0.5%bupivacaine 3 mL was injected at a moderate speed through a 22 gauge Whitacre needle.

Immediately after SAB placement the patient was assisted to the supine position. Shortly after lying supine, he became nauseated and pale and complained of difficulty breathing. His blood pressure decreased from 166/83 mm Hg to 96/38 mm Hg and his heart rate decreased from 78 to 53/min. The patient's voice deteriorated into a whisper and he was minimally able to lift his upper extremities off the bed. A high spinal was recognized. Rapid infusion of one liter of LR solution and two phenylephrine doses of 100 mcg each were administered. His heart rate remained in the mid-50s with administration of phenylephrine.

Preoxygenation was achieved by face mask through the anesthesia circuit at 10 L/min; mask ventilation was easy with placement of an oral airway. Rapid sequence induction with propofol 140 mg and succinylcholine 200 mg was used to facilitate tracheal intubation for mechanical ventilation. Anesthesia for tolerance of the endotracheal tube was maintained with sevoflurane 1.0% expired concentration and oxygen at 2 L/min. A phenylephrine infusion was required intermittently throughout the surgery to maintain a mean arterial blood pressure greater than 65 mm Hg.

At the end the surgical case (3 hours), the patient was spontaneously ventilating. Emergence from general anesthesia and extubation of the trachea were uneventful. The patient was

transferred to the recovery area with oxygen by non-rebreather facemask. Recovery and postoperative period were uneventful and patient was discharged without sequelae.

Discussion

Morbid obesity, a BMI of 40 or more,[2-5] affected 15.5 million Americans by 2010.[4] Trend estimates suggest the rate of morbid obesity continues to rise rapidly.[4] Numerous health problems associated with morbid obesity cause this population to require or desire surgery frequently.[2,3]

Regional anesthesia presents several advantages and challenges for the morbidly obese.[2,3] Potential advantages include: minimal airway manipulation, less cardiopulmonary depression, better postoperative pain control with decreased use of opioids, reduced postoperative nausea and vomiting, shorter lengths of stay in both post-anesthesia care units and hospitals, fewer complications, and fewer unplanned hospital admissions.[2] Challenges include: positioning, difficulty identifying landmarks, fat pockets resulting in false-positive loss of resistance during needle placement, needing longer needles, increased incidence of accidental dural puncture or epidural venous puncture, and ineffective blocks more often than in the nonobese population.[2,3,5]

Despite technical challenges of placing a SAB and conflicting data regarding local anesthetic dosing, the actual incidence of complications seems to be low in obese patients.[2] This patient's history of morbid obesity and OSA made SAB an appropriate choice for his anesthesia.

Two significant, potential complications of SAB are high and total spinals.[6] A high spinal affects vasomotor tone, and therefore blood pressure, when LA spread blocks the sympathetic fibers arising from T5-L1.[6] When LA reaches the T1-T4 (cardiac accelerator fibers), heart rate is affected (bradycardia).[6] A total spinal occurs when the local anesthetic affects the entire spinal cord and possibly the brainstem.[1] Respiratory depression and unconsciousness accompany hypotension and bradycardia in a total spinal.[5]

If a high or total spinal occurs, prompt recognition and intervention are key to preventing permanent damage or death.[1] The first symptoms that LA has reached a higher level than intended are often dyspnea, and numbness, tingling, or weakness in the upper extremities.[6] Nausea commonly precedes hypotension. As cervical spinal nerves are affected, patients exhibit severe hypotension, bradycardia, difficulty speaking and respiratory insufficiency. A total spinal results in loss of consciousness and apnea. If unrecognized and untreated, hypotension, bradycardia and hypoxemia lead to cardiac arrest. However, outcomes are improved when respiratory and cardiovascular system depression are quickly identified and supported.[1,6] In this case, the patient's BP was supported with vasopressors and fluid and his respiratory system supported by intubation of his trachea and mechanical ventilation. Interventions were swift and the patient suffered no long term effects.

Many factors may contribute to increased cephalad spread of LAs in the intrathecal space.[1,6] Such factors include: dose-related factors of volume, dosage, and baricity; administration technique including needle type, site of injection, needle direction, injection velocity and use of

barbotage; and patient characteristics such as height, age, gender, intra-abdominal pressure and anatomy of the spinal cord.[1,6]

Morbid obesity alters drug distribution,[2,5] but there is conflicting data as to whether obesity affects the spread of local anesthetics in neuraxial anesthesia.[5] Some studies have found lower anesthetic requirements for neuraxial anesthesia in obese patients when compared to non-obese patients[3,5] or a strong correlation between increasing BMI and sensory block height.[2] Yet other studies demonstrated no correlation between BMI and the spread of local anesthetic in neuraxial anesthesia.[2,5] There is a paucity of information regarding how morbid obesity affects regional anesthesia, thus controversy remains as to whether smaller local anesthetic doses should be used for neuraxial anesthesia in obese patients.[2]

When landmarks are impalpable, lumbar ultrasonography has been used to aid neuraxial placement in obese patients.[2,5,7] Imaging can determine the correct interspinous space, accurately identify midline and estimate depth of the ligamentum flavum and dura in most patients.[7] In cases of extreme obesity, ultrasound guidance may only aid in identifying spinous processes and midline.[5] Many of our patient's bony landmarks could not be palpated, making him an appropriate candidate for ultrasound guidance. Experience in US use and required equipment is paramount in applying this technology.

Anesthetic management of the morbidly obese is challenging. Because the prevalence is increasing rapidly, we must identify ways to safely and effectively provide anesthesia for this population. Though regional anesthesia is commonly a safe mode in this population, placement of blocks can often be technically difficult. Ultrasound guidance for placement of neuraxial blocks is one way to mitigate the challenge. Furthermore, continued research is needed to determine dosing guidelines and techniques for this population.

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Anesthetic Management of a Patient with Myasthenia Gravis

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Keywords: Myasthenia Gravis, Autoimmune Neuromuscular Disorder, Neuromuscular Junction, Acetylcholine Receptors

Myasthenia Gravis (MG) is an autoimmune neuromuscular disorder affecting predominantly young women and older men, the prevalence in the United States is estimated at 0.5-14.2 per 100,000.[1] The hallmark of this disease is the destruction of nicotinic receptors resulting in reduced synaptic transmission at the neuromuscular junction leading to incomplete or absent muscle depolarization. Symptoms include skeletal muscle weakness exacerbated by activity and relieved by rest. Individuals affected by MG have an unpredictable response to illness, stress and pregnancy and can exhibit a range of symptoms involving weakness of the ocular muscles to respiratory failure described as *myasthenic crisis.*[2]

Case Report

A 63 year-old, 62 kg, 160 cm tall female patient with a past medical history significant for MG but otherwise healthy experiencing pain and numbness in her lower back and bilateral upper legs presented for a minimally invasive lumbar discectomy of L3-L4. The patient takes pyridostigmine 60 mg daily and noted an increased prednisone dose to 7 mg daily to control symptoms over the previous 12 months. An 18 gauge intravenous catheter was obtained in the left hand and a lactated ringers infusion was initiated. The patient was premedicated with fentanyl 50 mcg prior to transport to the operating room.

Upon arrival in the operating room, noninvasive monitoring was initiated and preoxygenation was provided with oxygen 10 L/min. General anesthesia was induced with lidocaine 50 mg, propofol 140 mg and alfentanil 1000 mcg. Laryngoscopy was performed with a Mac 3 blade and the trachea was intubated with a 6.5 mm cuffed oral endotracheal tube. Placement was confirmed with bilateral breath sounds and end tidal CO_2. Intraoperatively respirations were controlled with volume controlled mechanical ventilation and a fresh gas flow totaling 2 L/min of an equal mixture of oxygen and air. General anesthesia was maintained with a continuous infusion of remifentanil titrated between 0.15-0.2 mcg/kg/min and isoflurane 1%. The patient's eyes were lubricated and taped prior to prone positioning on the operating table for optimal surgical exposure. Pressure points were padded, arms were abducted less than 90 degrees, eyes and ears were free from pressure. Prophylactic antibiotics were administered as well as supplemental corticosteroids, cefazolin 1 g and dexamethasone 10 mg respectively.

Approximately 30 minutes prior to the end of the case, ondansetron 4 mg was administered and the isoflurane was discontinued and general anesthesia was maintained with fresh gas mixture of N_2O 2 L/min, O_2 1 L/min and remifentanil 0.2 mcg/kg/min. After the closing sutures were completed by the surgeon, the patient was turned supine onto the stretcher, all agents were discontinued and oxygen flow was increased to 10 L/min. As the patient began to initiate spontaneous respirations, mechanical ventilation was discontinued and manual positive pressure ventilation was initiated. Once adequate tidal volumes were reached at a rate within normal limits and the patient opened her eyes to command she was extubated and transported with supplemental oxygen 10 L/min via face mask to the post anesthesia care unit. Muscle weakness was assessed according to bilateral strength of hand squeeze and dorsiflexion of the feet. The patient exhibited no signs of weakness with these actions. The patient's postoperative recovery was uneventful without respiratory complications and the patient was discharged home the same day.

Discussion

Myasthenia Gravis is a chronic autoimmune neuromuscular disorder in which the nicotinic acetylcholine receptors at the motor end-plate of skeletal muscles are inactivated by circulating autoantibodies and complement mediated destruction. The weakness associated with the disease manifests in different patterns ranging from involvement of only specific muscle groups such as ocular muscles to generalized fatigability affecting the bulbar and limb muscles. Patients with MG may present with symptoms of laryngeal and pharyngeal muscle weakness resulting in dysphagia, dysphonia and difficulty clearing secretions leading to an increased risk for aspiration. A more severe manifestation of the disease includes respiratory muscle weakness resulting from illness, infection, emotional stress, or surgery which can lead to respiratory failure described in the literature as *Myasthenic crisis*.[3]

The hallmark treatment of MG patients are cholinesterase inhibitors such as pyridostigmine which flood the neuromuscular junction with acetylcholine increasing the availability of acetylcholine for activation of intact nicotinic receptors. Pyridostigmine should be continued in the perioperative period.[5] However, when anticholinesterase agents are used perioperatively patients taking pyridostigmine are at risk for cholinergic crisis resulting from overstimulation of muscarinic receptors leading to symptoms of salivation, lacrimation, urination, defecation, GI distress and emesis (SLUDGE).[1]

As the disease increases in severity, corticosteroids and immunomodulators are often included in the pharmacological management of the disease. This patient's history of chronic corticosteroid therapy as part of her treatment regimen and noted increase in prednisone consumption over the previous twelve months prior to her surgery indicated that a stress dose of supplemental corticosteroids be administered intraoperatively.[2]

Patients with MG are often sensitive to the respiratory depressant effects of narcotics and benzodiazepines and should be used with caution preoperatively.[3] For this reason midazolam was not included in the anesthetic plan, however, a low dose of fentanyl was safely administered prior to transport to the operating room in order to relieve pain and anxiety verbalized by the patient.

Myasthenia Gravis patients often exhibit a profoundly increased sensitivity to non-depolarizing neuromuscular relaxants (NDMRs) due to the decreased number of functional acetylcholine receptors and may lead to prolonged intubation and/or residual muscle weakness postoperatively.[2] As a result, NDMRs should only be included in the anesthetic plan for these patients when absolutely necessary, given at decreased doses and with close attention to neuromuscular monitoring.[3] The response to depolarizing agents is often unpredictable. Patients can exhibit a resistance to succinylcholine or to the anticholinesterase medications with which these patients are medically managed, and may lead to a prolonged duration of action.[2] These medications were excluded from the anesthetic plan in order to avoid potential complications of increased sensitivity and prolonged time to extubation. Alternatively, short acting opioids were chosen for induction and maintenance of anesthesia for this case based on the observation that the muscle weakness associated with MG often allows for tracheal intubation. Additionally, volatile anesthetics have neuromuscular blocking properties which are often magnified in MG patients with the added benefit of no residual block after these agents are discontinued.[3]

For this patient, tracheal intubation was accomplished using propofol, isoflurane and alfentanil. Alfentanil has been shown to provide optimal intubating conditions when NDMRs are contraindicated.[4] Respiratory depression results from alfentanil rapidly crossing the blood brain barrier with an equilibration time significantly lower than that of fentanyl and sufentanil and was chosen based on the rapid pharmacokinetics of onset and elimination of the drug. Maintenance of anesthesia relied on isoflurane and remifentanil to provide adequate amnesia, analgesia and akinesia. Remifentanil was used in consideration of the respiratory effects of longer acting opioids. These agents can persist into the postoperative period and the desire was for this patient to exhibit a strong respiratory drive at extubation and in recovery. The drug was chosen owing to metabolism by non specific blood and tissue esterases which allows for rapid clearance.[4] Additionally, remifentanil has also been shown to enhance the volatile anesthetic properties that produce akinesia and therefore provided an excellent adjuvant in this case.

Respiratory muscle weakness and respiratory depression are key concerns in caring for a patient with MG and should be closely monitored for ensuring that the patient exhibits adequate strength to maintain spontaneous respirations postoperatively. Though all muscle relaxants were avoided in this case, it remains important to establish that the opioid infusion has adequately metabolized to the point that respirations are no longer inhibited. For this patient, after the remifentanil infusion was discontinued adequate respiratory drive and rate with sufficient tidal volumes was ensured to confirm the safety of extubation. The patient's strength was also evaluated with hand squeezes and plantar/dorsiflexion which was strong and equal bilaterally. The patient was able to follow commands and showed no signs of weakness during emergence and throughout the recovery period.

Planning an anesthetic for a patient with MG can be challenging due to the increased sensitivity of these patients to anesthetic medications as well as the risk for disease exacerbation that can result from surgical stress. It is important to consider the potential complications that can accompany the inclusion of muscle relaxants in the anesthetic plan for patients with Myasthenia Gravis.

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Bradycardia during Robotic-Assisted Cystoprostatectomy

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Keywords: hemodynamic changes robotic, cystoprostatectomy, robotic-assisted prostatectomy, bradycardia with insufflation, abdominal insufflation risks

A robotic-assisted cystoprostatectomy is a minimally invasive surgery which allows the surgeon to perform the procedure by remotely controlling surgical instruments attached to robotic arms.[1] Major advantages to robotic-assisted procedures include surgical precision, decreased blood loss, decreased post-surgical pain, faster recovery, smaller incisions, decreased risk of infections, minimal scarring, and shorter hospital stays.[2] However, they require lengthy positioning of the patient in a steep Trendelenburg position (30°-45°), and also involve insufflation of carbon dioxide (CO_2) into the peritoneal cavity, producing pneumoperitoneum. The pneumoperitoneum and alteration of position elicit changes in the patient's cardiovascular system, altering the patient's hemodynamics.[2]

Case Report

A 69-year-old, 188 cm, 90 kg male with a history of bladder cancer presented for a robot-assisted cystoprostatectomy with conduit diversion, a pelvic lymph node dissection, and an open bilateral ureteral stent placement. He also had a history of poorly controlled insulin dependent diabetes mellitus, chronic obstructive pulmonary disease, carotid artery disease, and cystoscopy with transurethral resection of a bladder tumor one month prior to the current surgery. His medication regime included gabapentin, glyburide-metformin, lorvastatin, albuterol, and insulin detemir. The pre-anesthetic evaluation was completed in the pre-anesthesia clinic, where the patient's pre-existing conditions were optimized for surgery. The risks related to anesthesia were discussed with the patient before the anesthesia consent was signed.

Midazolam 2 mg and fentanyl 100 mcg were given intravenously (IV) in the holding room. In the operating room, standard non-invasive monitors were applied. The patient was hemodynamically stable with a heart rate of 62/min. The patient was pre-oxygenated and was given fentanyl 50 mcg, lidocaine 100 mg, propofol 150 mg, and rocuronium 50 mg for induction of general anesthesia. Successful tracheal intubation with a 7.5 mm endotracheal tube was verified and the endotracheal tube was secured.

Anesthesia was maintained with an inspired isoflurane concentration of 1%. The patient was secured on the bed with his arms padded and tucked. The bed was moved into a steep Trendelenburg position as a trial for hemodynamic and ventilatory stability during surgery. The patient's vital signs, tidal volumes, and peak inspiratory pressures remained stable during this trial. The patient was then positioned in low lithotomy position, and the surgical team began preparation of the abdomen for surgery. The abdomen was insufflated with CO_2 by the surgeon. During insufflation the patient's heart rate decreased from 62 to 30 beats per minute. Ephedrine, 20 mg was given IV, with no immediate response. Glycopyrrolate 0.2 mg IV was then given. The patient's heart rate increased to 60/min and remained stable at that level for the remainder of the case. There was no interruption of abdominal insufflation. The rest of the anesthetic course was uncomplicated.

Once the robotic portion of the procedure was completed, the patient was placed into the supine position for the remainder of the surgery. At the conclusion of surgery, neuromuscular blockade was antagonized with neostigmine 3 mg and glycopyrrolate 0.4 mg. When the patient resumed spontaneous respirations and appropriately followed commands, the trachea was extubated. Oxygen at 6 L/m was administered via a Mapleson C circuit. The patient was transferred to the post-operative care unit in stable condition.

Discussion

In the past decade, robotic-assisted cystectomy and prostatectomy surgeries have become more common due to reports of shorter recovery periods, shorter hospital stays, and earlier return to daily activities compared with open radical cystectomy and prostatectomy surgeries.[3] However, some studies have shown significant hemodynamic changes during robot-assisted laparoscopic prostatectomies and cystectomies when patients were in the steep Trendelenburg position (30°-45°) with high-pressure CO_2 pneumoperitoneum.[3,4] Several of these patients had no significant cardiac history.[4-6] Of these reported cases, the patients' heart rate returned to baseline after removal of the CO_2.[4]

It is well known that using CO_2 for pneumoperitoneum has risks of cardiovascular changes, such as bradycardia and cardiac arrest. During CO_2 insufflation, traction and pressure is exerted onto the mesentery. Rapid peritoneal distention with abdominal insufflation causes a vagal-mediated cardiovascular reflex. This reflex occurs from both decreased venous return to the heart and stimulation of the celiac plexus in the abdominal cavity.[5,7] The pressure on the mesentery from the pneumoperitoneum stimulates the vagus nerve which decreases the patient's heart rate.[7] The bradycardia could also be a paradoxical reflex-induced response resulting from decreased venous return. This response activates the vagal response through baroreceptors and stretch receptors.[7]

There are several treatment options when this reflex is stimulated. Pharmacological interventions, like in the above case, are simple, effective methods to increase the patient's heart rate. Treating bradycardia with medication prevents interruption in the surgery and causes minimal to no harm to the patient. The risks associated with pharmacologic treatment include a failed desired response and a prolonged episode of bradycardia. This delay could decrease the patient's cardiac output, decrease perfusion to primary organs, and possibly result in asystole. However, the anesthesia provider and the surgeon could minimize the pathophysiologic changes from the pneumoperitoneum by limiting the intra-abdominal pressure below 12-15 mm Hg. This can also be accomplished by keeping the rate of insufflation slow instead rapid. By decreasing stimulation to the celiac plexus, the patient's heart rate should return to baseline.

Several studies have demonstrated the effectiveness of these techniques. In one study, a patient became asystolic and was treated immediately by decreasing the insufflation pressure on the abdomen. The patient was also given chest compressions and pharmacological intervention, which resulted in a return of spontaneous circulation.[5] Other patients studied were only treated with pharmacological intervention and had positive results, such as the patient in the case report.[5] However, it is unclear in this study what pharmacological drugs were given.

When the reported patient started to show signs of bradycardia during insufflation, ephedrine was given to stimulate beta-1 receptors and increase the heart rate. When a response to the ephedrine was not seen, glycopyrrolate was given to block muscarinic receptors, which should also result in an increased heart rate. Within 1 to 2 minutes of giving the glycopyrrolate and 4 minutes of giving the ephedrine, the patient's heart rate returned to baseline. Additionally, the surgeon could cease abdominal insufflation or decrease the intra-abdominal pressure. If the previous interventions were unsuccessful in stabilizing the patient's heart rate, the patient's position could be changed from steep Trendelenburg to flat supine. By deflating the insufflation pressure and positioning the patient supine, venous return would increase and the vagal reflex would be inhibited.[7]

In the present case, the patient's bradycardia was treated with pharmacological interventions prior to changes in abdominal insufflation pressure or positional changes. The patient's positive response from the pharmacological intervention prevented any delay in the surgery. After treatment, the patient's baseline heart rate was maintained. The perioperative management of robotic-assisted urologic surgeries creates both advantages and challenges, but the literature on hemodynamic changes during these surgeries is limited. Several studies observed physiological effects of pneumoperitoneum in the Trendelenburg position; however, only one study discusses successful treatment for these adverse events.[5,6,8]

In conclusion, the perioperative management of a patient during robotic-assisted prostatectomy or cystectomy can bring challenges that both the surgeon and the anesthesia provider must anticipate. Close coordination between the anesthesia and surgical teams is required for a successful surgery.[8] This case presents the anesthetic management of a patient with complications consistent with existing current literature and best available evidence. However, further research is needed to examine management techniques that benefit patients who have unexpected hemodynamic changes during robotic-assisted urologic surgeries.

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Preoperative ACE inhibitor use and Refractory Hypotension

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Keywords: angiotensin-converting enzyme inhibitor (ACEI), perioperative hypotension, refractory hypotension, vasopressin, anesthesia

Over 60 million Americans are diagnosed with hypertension and currently receiving treatment. Several classes of drugs are used to effectively control hypertension.[1] Angiotensin-converting enzyme inhibitors (ACEI) are frequently used for their additional therapeutic effects including; stabilization of atherosclerotic plaques, inhibition of left ventricular hypertrophy, prevention of myocardial remodeling, decreased systemic vascular resistance, decreased platelet aggregation, decreased intraglomerular pressure, decreased fluid retention, increased insulin sensitivity and anti-inflammatory properties.[1] While these effects are important when used preoperatively, a review including 434 patients revealed 100% use of vasopressors for intraoperative hypotension associated with ACEI use prior to surgery.[2] This hypotension proves an intraoperative challenge when refractory to standard treatment by anesthesia professionals.

Case Report

A 66-year-old African American female (83 kg, 160 cm) presented for a deep inferior epigastric perforator (DIEP) flap, related to history of breast cancer and mastectomy. The patient had no

known drug allergies, no history of smoking or alcohol use. Additional medical history included hypertension, hyperlipidemia, fatty liver, lymphedema of her right arm and anemia. Her medications included metoprolol, lovastatin, acetylsalicylic acid, letrozole, chlorthalidone, multi-vitamin, and venlafaxine. Surgical history included: hysterectomy, nasal endoscopy, dacryocystorhinoplasty and mastectomy. She reported no anesthetic complications following these procedures.

She presented to the pre-operative clinic 13 days before scheduled surgery with a blood pressure (BP) of 188/98 mm Hg and a mean arterial pressure (MAP) of 128 mm Hg, and was referred to her primary care provider for improved control of hypertension prior to surgery. She was prescribed lisinopril, she self-administered metoprolol and lisinopril the morning of surgery. The morning of surgery her BPe was 178/89 mm Hg (MAP 118 mm Hg). A complete pre-anesthetic evaluation revealed no other significant findings. Lab values were within normal limits. Physical examination revealed a Mallampati Class III, thyromental distance of three fingerbreadths, 3 cm oral aperture, and normal Atlanto-occipital joint extension. A 20 g intravenous catheter was inserted and midazolam 2 mg was administered.

In the operating room, standard monitors were applied; the patient was pre-oxygenated with O_2 10 L/min for approximately 5 minutes. General anesthesia was induced intravenously with lidocaine 100 mg, propofol 150 mg, and rocuronium 70 mg. The patient was easily mask ventilated upon introduction of a 9 mm oral airway. A size 3 Macintosh blade was used to place a 7.0 mm endotracheal tube (ETT). Placement was confirmed with positive end-tidal CO_2 tracing, bilateral breath sounds and equal chest rise it was secured at 22 cm at the lip. An additional 18g intravenous catheter was inserted. Anesthesia was maintained with 0.5-1 mean alveolar concentration (MAC) of isoflurane, 50% nitrous oxide and vecuronium. A Brain Function Monitoring System (BIS by Covidien, Mansfield, MA) was placed to monitor depth of anesthesia so that volatile gas administration could be minimized.

The patient's MAP fluctuated from 60-100 mm Hg during the procedure, with a target MAP of 70 mm Hg as requested by the surgeon. The patient received 5% albumin 1 L, normal saline 1 L, and plasma-lyte 4500 mL administered over 11 hours. Urinary output was 60-300 mL/hr for a total of 1330 mL. Blood pressure was maintained the first five hours of the case with total bolus doses of phenylephrine 1080 mcg and ephedrine 165 mg. A phenylephrine infusion was then initiated for the final 2 hours, totaling 2735 mcg. Seven hours in to the case vasopressin 2 units IV was administered and no additional vasopressors were required.

At the conclusion of the procedure, neuromuscular blockade was antagonized with neostigmine 3 mg and atropine 0.4 mg IV. Estimated blood loss was 225 mL. The patient demonstrated return of spontaneous ventilation at 15/min, tidal volumes of 350 mL, and ability to follow commands. The ETT was removed with positive pressure. The patient was transferred to the post-anesthesia care unit with O_2 10 L/min via facemask, stable vital signs (BP 123/62 mm Hg, MAP 84 mm Hg, heart rate 88/min, respiratory rate 15/min, SpO_2 100%), and a patent airway.

Discussion

Anesthetic priorities for a DIEP flap include muscle relaxation, keeping the patient warm and well hydrated. These interventions minimize peripheral vasoconstriction which could impair graft perfusion.[3] The surgeon also requested a MAP of 70mmHg because hypotension and vasopressor use causes the vessels to spasm and making anastomosis under a microscope very difficult. This hemodynamic parameter was antagonized by the patient's continued ACEI therapy. Discontinuation of ACEI therapy on the day of surgery is common practice, although steadfast guidelines are not available.[4,5] The American College of Physicians initially recommended to stop ACEI therapy on the day of surgery and then later modified its recommendation to continue ACEI with caution in patients with cardiac failure.[4] The American Society of Anesthesiologists (ASA) does not have any guidelines or recommendations regarding ACEI. In 2012, the ASA's chair of the committee on regional anesthesia, who is also the clinical practice chair of the Mayo Clinic's department of anesthesiology, reported that all Mayo Clinic surgical patients are requested to hold their ACEI on the day of surgery.[5]

Blood pressure is regulated by three systems in the body; the sympathetic nervous system, the renin-angiotensin aldosterone system (RAAS) and the vasopressin system. ACEI's inhibit the RAAS. General anesthesia inhibits the sympathetic nervous system so the body is left to compensate with the vasopressin system.[1] Hypotension was evident in our patient even after induction medication effects had subsided. We started replacing volume using crystalloids with minimal results and supplemented with IV bolus doses of phenylephrine and ephedrine as necessary to support her MAP. Colloid administration was our next action in replacing the volume and avoiding vasopressors. Volume resuscitation was evaluated by urinary output and anesthetic fluid replacement calculations. Maintenance of 120 mL/hr, deficit of 960 mL and third space loss of 4 mL/kg/hr was replaced. The patient's MAP remained in the low 60's after adequate volume resuscitation despite titrating the volatile anesthetic to an appropriate BIS reading and administering multiple ephedrine and phenylephrine boluses. A phenylephrine infusion was then initiated; 25 mcg/min was required to keep the patient's MAP near 70 mm Hg. Phenylephrine and ephedrine are considered first-line agents for treating hypotension during anesthesia but several studies have shown this therapy is not adequate to treat refractory hypotension in patients receiving ACEIs.[4] At this point, the surgeon was under the microscope working on anastomosis of the transplanted vasculature. We frequently consulted regarding the blood pressure and spasm of the vessel. We gave 2 units of Vasopressin IV bolus and for the remaining three hours of surgery no other vasopressor therapy was needed.

Review of a similar case report identified several risk factors contributing to the patient's hypotension: age, use of ACEI on day of surgery, other antihypertensive medications (metoprolol and chlorthalidone), and administration of propofol at induction.[6] Analgesia was achieved with totals throughout the case of fentanyl 250mcg, acetaminophen 1000mg and ketamine 40mg. This choice was supported in the literature because ketamine increases heart rate and blood pressure without causing significant change in autonomic nervous system.[4]
If a case similar to this presented, alternative anesthetic choices should be considered. Induction was achieved with 1.8 mg/kg of propofol, the patient had received approximately 200mL of crystalloid prior. Studies have shown that etomidate or methohexital would be a more cardiostable agent.[4] Also, a dose of 1.3mg/kg of propofol is associated with the least need for

pharmacologic intervention to treat bradycardia and hypotension in patients on ACEIs. [7] An intravenous bolus of 10 mL/kg of crystalloid administered prior to induction has also been shown to minimize resultant hypotension.[4]

Comparing maintenance of anesthesia at 0.5 MAC between isoflurane, sevoflurane and desflurane, little difference is noted in respect to their effects on sympathetic nervous system and catecholamine levels. Above 0.5 MAC desflurane is associated with higher sympathetic nerve conduction and higher catecholamine concentrations.[4] After initial efforts with ephedrine and phenylephrine were unsuccessful, norepinephrine could have been considered for its alpha and beta 1 effects; addressing that a patient on ACEI's has a lower amount of circulating catecholamines.[4] A reported disadvantage of administration of norepinephrine when compared to vasopressin is more episodes of hypertension.[4] Vasopressin-mediated vasoconstriction is not attenuated by ACEI therapy as it works independently of the RAAS, and hence why it was chosen to treat the hypotension refractory to ephedrine and phenylephrine. Alterations in the induction dose, volatile choice, and vasopressor use could have better optimized the patient's blood pressure. The phenylephrine infusion may have been avoided, and perfusion better supported ultimately avoiding vasospasm of the vasculature and decreasing surgical time.

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Anesthetic Management of Mitral Valve Prolapse

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Keywords: mitral valve, prolapse, regurgitation, leaflets, valvular disease

Anesthetic considerations related to mitral valve prolapse depends on the severity of the disease and the patient's self-report of symptoms. Mitral valve prolapse (MVP) is defined as the upward movement of one or more of the leaflets during systole, regurgitant flow may or may not be present.[1] This cardiac abnormality receives less attention compared to other valvular defects, but it is the most common valve disease, affecting up 2.5% of the healthy population.[2] To safely treat patients diagnosed with mitral valve prolapse (MVP), the underlying symptoms and disease severity should be known prior to implementing an anesthetic plan.

Case Report

A 68-year-old, 80 kg female with five year history of lower back pain and leg numbness, was scheduled for a posterior lumbar interbody fusion of L3-5. Additional medical history included: diabetes mellitus type II, hypertension, hypothyroidism, splenic aneurysm, and mitral valve prolapse with mild regurgitation. At the time surgery, there were no echocardiogram results and a recent EKG showed sinus rhythm. Of note the patient was prescribed a daily fentanyl patch for control of chronic pain related lumbar disc disease.

A pre-anesthesia assessment and physical examination was performed, and the patient was noted to have a Mallampati class 3, thyromental distance and oral aperture of 3 finger breadths. Laboratory work was remarkable for anemia with a hemoglobin of 9 g/dL and hematocrit of 30 g/dL. The anesthetic plan was discussed with the patient and informed consent was obtained. A general anesthetic was planned for the procedure with a kneeling prone surgical position. An 18 gauge peripheral IV was placed in the holding room with a slow infusion of normal saline. Midazolam 1 mg was administered, and the patient was transported to the operating room. The patient remained on the stretcher for induction, and all standard monitors were placed. Oxygen was administered via face mask at 10 L/min until expired O_2 concentration was greater than 80%.

Once adequate pre-oxygenation was achieved, the patient was induced with intravenous: lidocaine 80 mg, propofol 160 mg, fentanyl 100 mcg, and rocuronium 50 mg. Phenylephrine in the amount of 720 mcg was given post induction to maintain blood pressure within 20% of baseline. Elective use of an endotracheal light wand was used to place a 7.0 ETT due to patient's body habitus and airway exam. Final surgical position was prone with use of chest rolls and ProneView (Dupaco, Inc., Oceanside, CA). Adequate padding was used on all pressure points.

Anesthesia was maintained with a fentanyl infusion which was discontinued 1 hour prior to closure, isoflurane at 0.8% inspired concentration with 1 L/min flow of nitrous oxide and O_2, and rocuronium boluses. At the start of closure, 5 mg of neostigmine along with 1 mg of glycopyrrolate was given to adequately antagonize the neuromuscular blockade. The patient was

returned to supine position on the stretcher. The patient achieved adequate tidal volumes, responded to verbal command, and the endotracheal tube was removed. The patient maintained a patent airway with stable vital signs, and was transported PACU on supplemental oxygen. A total of 1 L of normal saline and 1.2 L of plasmalyte was given during the procedure. Blood loss was estimated at 200 mL. Report was given in PACU, and the patient remained in the hospital overnight for observation.

Discussion

Mitral valve prolapse (MVP) can occur in patients with no prior, related medical history. The presence is often benign, showing no symptoms.[3,4] Females have a higher incidence of the disease, as do patients with a history of rheumatic fever, myocarditis, lupus, and Marfan syndrome.[1] Patients may be asymptomatic or suffer from cardiac dysrhythmias, dyspnea, or fatigue.[3] These symptoms typically will vary with the severity of the prolapse and whether or not regurgitant flow is present.[4] A person with severe mitral valve prolapse may have similar symptoms as someone with mitral valve insufficiency such as cough, fatigue, shortness of breath, and chest palpitations.[3] Surgical correction is often considered once dyspnea is present with moderate to severe regurgitant flow through the valve.[1,2] More severe cases pose potential serious complications such as embolic stroke, congestive heart failure, lethal dysrhythmias, and cardiac sudden death.[1]

Patients may be diagnosed with mitral valve prolapse by expressed symptoms or a mid-systolic murmur heard by auscultation.[1] A definitive diagnosis is made by transesophageal echocardiography, which observes mitral valve movement during the cardiac cycle.[4,5] An echocardiogram can also help visualize flow dynamics, determine if any regurgitant flow is present, and provide information to accurately diagnose the severity of the condition and proceed with the appropriate treatment.[5,6]

The anesthesia practitioner should have a general overview of optimal physiologic conditions for patients with mitral valve prolapse.[1,6] A baseline or recent EKG will help define the presence of frequent premature ventricular complexes and/or QT interval changes possibly related to MVP.[2] Previous studies indicate that reducing left ventricular (LV) preload will increase the amount mitral valve prolapse, thus increasing the regurgitant flow.[4] Anesthetic considerations should include; maintaining systemic vascular resistance (SVR) at or near the patient's baseline level, avoiding increasing cardiac contractility, attenuating changes in heart rate and ensuring a euvolemic volume status.[1,7] Volume overload or fluid depletion could increase symptoms in a patient with MVP by altering LV preload.[4,7] The sitting or Trendelenburg surgical positions can also decrease LV filling and should be avoided if possible.[4]

Additional equipment, such as an arterial line, a foley catheter, and a pulmonary artery catheter may serve as useful tools to monitor SVR and volume status during surgery.[1,4] Volatile and intravenous anesthetics are safe to use with MVP as long as the dose is titrated to avoid prolonged hypotension.[4] Systemic hypotension may be treated with phenylephrine to increase SVR. Previous research suggests to avoid drugs, such as ketamine and desflurane that can increase a sympathetic cardiac response.[4] Increasing heart rate and contractility can exacerbate symptoms of MVP; therefore, drugs that mimic this response should be avoided.[1,4] Opioids are

also an appropriate choice for these patients due to the blunted sympathetic response, lack of cardiac depression, and decreased reaction to surgical stimulus they provide.[1,7]

In the case presented, adequate volume resuscitation was used to maintain an appropriate volume status and counteract intraoperative blood loss as well as initial fluid deficit. In addition, the patient was appropriately treated with opioid analgesia to decrease the sympathetic response caused by direct laryngoscopy and surgical incision. Prior to surgery, the patient was on an opioid regimen at home; therefore, opioids were tolerated well and liberally administered to maintain appropriate analgesia. Phenylephrine was given post induction to counteract hypotension associated with the anesthetic initiation. Conscientious attention was given to blood pressure and fluid management to avoid drastic changes from baseline status. Maintaining hemodynamics and fluid volume was aimed at preventing any additional MVP or altering cardiac output status associated with the anesthetic. The patient's hemodynamics were stable during the remainder of the case with no signs of cardiac instability. The patient was required to be in the kneeling, prone position for surgery, which did not appear to cause any hemodynamic alterations or present issues with the anesthetic plan. The kneeling prone position is known to sequester blood in the lower extremities and increase intra-abdominal pressure which leads to a decrease in cardiac preload. Overall, the procedure was tolerated well by the patient without any intra- or post-operative sequelae.

Patients with MVP can safely undergo surgical procedures with general anesthesia; however, caution and appropriate technique must be employed by the anesthesia practitioner to protect patients from possible complications related to the anesthetic. Prior to surgery, planning and discussion by the anesthesia team will reduce risks and provide optimal operative conditions for the patient. Anesthesia professionals should follow current practice guidelines and apply the appropriate monitors during surgery to reduce risks, while also educating the patient how their condition relates to the anesthetic plan.

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Converting to General Anesthesia in a Complex Parturient

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Keywords: Obstetric anesthesia, granulosa cell tumor, cesarean section, general anesthesia, American Society of Anesthesiologists (ASA) Practice Guidelines for Obstetrical Anesthesia

Neuraxial anesthesia is frequently the anesthetic method of choice for cesarean sections due to the airway and respiratory changes associated with pregnancy. However, a general anesthetic may be required due to a neurologic or coagulation disorder, inadequate dermatome coverage, or procedure urgency. Fortunately, epidural anesthesia may be re-dosed when utilized, however, the incidence of failed neuraxial anesthesia during cesarean sections thus necessitating conversion to general anesthesia is reported at 1.7%[1] General anesthesia offers benefits during obstetrical emergencies, yet can pose risks, including the significant incidence of 1/533 failed intubations.[1] With updated algorithms, expert anesthesia professionals, and availability of advanced airway devices, general anesthesia can be safe for both mother and fetus.[1]

Case Report

A 30-year-old gravida 1 at 41 weeks' gestation underwent induction with an epidural for labor analgesia. The patient was 170 cm tall and weighed 105.5 kg. The patient's medical history included a documented vicodin allergy, left ovarian cyst, pancreatitis, and a history of chemotherapy for stage II granulose cell tumor of the right ovary from one year prior. During her pregnancy, a questionable pelvic cyst was noted on ultrasonic imaging demonstrating characteristics consistent with ovarian granulosa cell tumor (OGCT). The patient's current medication regime consisted solely of prenatal vitamins. Laboratory values upon admission included: hemoglobin 10.7 g/dL, hematocrit 31.2 %, platelets 154,000/μL and chemistries within normal limits.

When she progressed to 9 cm dilation, the baby began to demonstrate reduced variability and late decelerations on fetal monitors. The patient was immediately brought to the operating room for emergency cesarean section. An18-gauge peripheral intravenous catheter was in place. The patient's airway was classified as Mallampati II. The initial anesthesia plan consisted of utilizing the previously placed epidural with a lidocaine 2% bolus of 20 ml. A T4 dermatome level was obtained with the epidural bolus and augmented with fentanyl 100 mcg, which provided sufficient coverage for incision. Upon arrival to the operating room, oxygen was administered via nasal cannula at 4 L/min, standard monitoring initiated. The patient was placed in left uterine displacement. The patients' vital signs at this time were HR: 86, BP: 123/80, SpO_2: 98%. Upon delivery, oxytocin 20 U/L was initiated with an estimated blood loss of 1000 ml. The obstetrician explained to the patient and the anesthesia providers the need for oncology to assist with debulking a pelvic tumor. As the surgical procedure progressed, the patient complained of increasing discomfort. Instead of rebolusing the epidural, intravenous anesthesia was utilized. However, the patient's discomfort was refractory to multiple administrations of ketamine and fentanyl, totaling 50 mg and 100 mcg, respectively. As the laparotomy progressed, a

hemorrhagic mass versus hematoma in the posterior cul-de-sac was found. The patient, anesthesia providers and the surgical team decided to convert to a general anesthetic due to the increased possibility of hemorrhage and insufficient dermatome coverage provided by the epidural. A rapid sequence induction (RSI) with cricoid pressure was performed using fentanyl 100 mcg, propofol 150mg and succinylcholine 100 mg. Direct laryngoscopy with a Miller #2 blade was performed and resulted in a grade I view of the glottis. The airway was secured without difficulty with a #7 endotracheal tube. General anesthesia was maintained during the case with sevoflurane 1.8% inspired concentration in a mixture of oxygen 2 L/min and nitrous oxide 2 L/min. An additional large-bore intravenous and arterial line were established and the remaining two-and-a-half-hour surgical time was focused on tumor debulking, hemostasis maintenance, and abdominal closure. An additional administration of oxytocin 20 U/L was hung and five liters of normal saline were infused to maintain the patient's blood pressure throughout the case. Despite the increased blood loss, totaling 1800 ml, the bedside hemoglobin levels were 8.0 g/dl. The remainder of the surgery progressed uneventfully. The patient was extubated and transferred to the post anesthesia care unit, alert and hemodynamically stable.

Discussion

Currently, the ASA Task Force on Obstetrical Anesthesia recommends epidural or spinal anesthesia over general anesthesia for cesarean sections. While time from anesthesia start to delivery is shortest with general anesthesia, maternal and fetal complications increase.[2] Neuraxial anesthesia decreases maternal stress, improves patient satisfaction, and provides sufficient pain relief during labor.[3] The benefits of neuroaxial anesthesia over GA for cesarean section are due to limited drug transfer to the fetus, and the respiratory/airway changes associated with pregnancy.[1]

As with all anesthetic plans, cesarean deliveries should include possible conversion to general anesthesia. It requires advanced planning, even for experienced obstetric anesthesia practitioners, to safely manage an emergency conversion.[4] The complex physical alterations in pregnant women can create additional challenges when providing general anesthesia to this patient population. The case study presented above reflects the suggested ASA guidelines when requiring conversion to general anesthesia.[2]

This patient's medical history supplied an additional obstacle, making a conversion to general anesthesia much more likely. OGCT are rare, accounting for only 2-5% of all ovarian neoplasms.[5] These tumors are described as slow growing, with a later incidence of reoccurrence. Reoccurrence ranges have been documented from five to thirty years, with an overall unfavorable prognosis.[5] There are currently no standard guidelines for OGCT, however, improved outcomes have been achieved with surgical debulking combined with chemotherapy. In this case, an initial plan was established for standard vaginal delivery, yet, if a cesarean section proved necessary, the surgical team had obtained consent for an exploratory laparotomy.

With the expanded scope of this surgery and inadequate dermatome coverage, general anesthesia with an RSI was our choice. Since parturients are considered to have full stomachs, and are at an increased risk of aspiration, a rapid sequence induction is the preferred method to secure the airway.[2] There are reports of an increase in difficult intubations in term parturients, upwards of

16%; the risk of failed intubations is at least 10 times greater in parturients as compared to the general surgical population.[6] The rapid change in anesthetic plan was made in accordance with current guidelines and standards.[2] Conversion to general anesthesia was made with the foresight that the patient could be a difficult intubation. Advanced airway devices, including a bougie and Glidescope (Verathon Inc., Bothell, WA) were readily available, but were not utilized for the case. A recent observational study of 5,000 general anesthetics on pregnant women did not note any incidence of aspirations.[1] The results of this study, however, do not reject the possibility of an aspiration occurring. Given the myriad of complications, anesthesia providers should weigh the risks associated with general anesthesia in order to best maintain a secure airway.

With the increased possibility of blood loss necessitating rapid fluid administration, a second large-bore intravenous was established. Current literature has yet to address central venous access use alone for obstetrical patients. Yet, recommendations provided by the ASA suggest large-bore access when hemorrhagic emergencies are encountered.[2] Additionally, current recommendations provided by the ASA suggest invasive hemodynamic monitoring should be initiated individually based on clinical presentation.[2] Given the increased surgery scope, an arterial line was inserted to monitor the patient's hemodynamics, as well as to obtain blood specimens.

Parturients experience a hemodilutional anemia due to increased plasma levels, along with slightly increased hemoglobin. This relative anemia rarely requires a blood transfusion given the increased circulating volume. Particularly with this patient's hemoglobin of 8.0 G/dl, blood transfusion was not required to improve the patient's oxygen carrying capacity. The patient likely experienced some extent of hemodilution, considering the amount crystalloid administered. Significant post-partum hemorrhage occurs in <1% of all deliveries.[7] Research also demonstrates a hemoglobin level of 7.0 G/dl is the cut off to provide sufficient oxygen carrying capacity in healthy women.[7] Expected estimated blood loss for cesarean section can reach upwards of 1000 ml. As the case progressed, there was a reduction in blood loss rate and hemostasis was adequate, thus a coagulation panel was not deemed necessary.

Neuraxial anesthesia can sufficiently blunt the noxious stimuli of a cesarean section. However, this case demonstrates the need for constant vigilance, communication with the patient and surgical team, and preparation for immediate conversion to general anesthesia, when neuraxial anesthesia is not sufficient. With the infrequent need for general anesthesia in this unique patient population, the risks and complexities may not be familiar to anesthesia providers. It is necessary for anesthesia providers to be aware of complications associated with obstetrical anesthesia and to plan ahead to avoid them, as well as maintain flexibly to safely adapt as patients' needs vary. With the rare complexities of this case the conversion from neuraxial to general anesthesia was the appropriate decision and was managed according to current recommendations based upon best evidence.

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Negative Pressure Pulmonary Edema in a Pediatric Patient

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Keywords: Negative pressure pulmonary edema, post-operative pulmonary edema, post-obstructive pulmonary edema, obstructive sleep apnea, tonsillectomy

Negative pressure pulmonary edema (NPPE) or post-operative pulmonary edema (POPE) can rapidly become a life-threatening anesthetic emergency. The presentation of NPPE can be immediate or delayed and can be confused with other causes of postoperative respiratory distress. The disorder has been classified into 2 types, each with similar clinical pictures, but with differing etiology. Type I follows a sudden severe episode of upper airway obstruction (UAO) such as laryngospasm, while Type II develops after relief of a chronic obstruction such as enlarged tonsils or upper airway tumor.[1] This report details a case of respiratory distress in a pediatric patient following tonsillectomy

Case Report

A 5-year-old male, measuring 39 kg in weight and 110 cm in height, presented for tonsillectomy and adenoidectomy. His past medical history included chronic otitis media, obstructive sleep apnea (OSA), enlarged tonsils, and chronic upper respiratory infections. A sleep study evaluation revealed an apnea-hypopnea index (AHI) of 6. Past surgical history included bilateral

myringotomy and tube insertion. The patient's medications included albuterol metered dose inhaler, 2 puffs every 6 hours, as needed. Three weeks prior to admission, he completed a 7 day course of amoxicillin/clavulanic acid for reoccurring sinusitis and otitis media. On physical examination, breath sounds were clear to auscultation and clear nasal drainage was noted. The patient's mother reported that he was free of any recent cough or fever. Assessment of the airway revealed a Mallampati score of I, normal dentition, and 2+ tonsillar hypertrophy. The patient had nothing by mouth for 8 hours prior to surgery. He received acetaminophen 10mg/kg orally in the pre-operative unit.

Accompanied by his mother, the patient was taken to a pre-warmed operating room and placed supine on the operating table. A pulse oximetry monitor was applied and a mask induction was initiated with a blend of 3 L of oxygen, 6 L of nitrous oxide, and 8 % sevoflurane. Once induction of anesthesia was accomplished, the mother was escorted from the room. Standard monitors were applied and the eyes were taped. A 22-gauge peripheral intravenous (IV) catheter was inserted in the left hand. Propofol 50mg and fentanyl 30 mcg were administered through the IV. The nitrous oxide was discontinued and the patient's trachea was intubated under direct laryngoscopy with a Miller 2 blade and a 5.0 mm cuffed oral endotracheal tube (ETT). Clear, thick post-nasal secretions were noted during laryngoscopy. Placement of the ETT was confirmed by the presence of end tidal carbon dioxide ($etCO_2$) and bilateral breath sounds. General anesthesia was maintained with 2.5% end-tidal sevoflurane with 1L/min air and 1L/min of oxygen. The patient was mechanically ventilated on volume control with a respiratory rate of 20 breaths per minute and a tidal volume of 250mL. The temperature was monitored through an axillary probe. A forced air lower body warming unit was applied.

The surgery was uneventful with the patient's SpO_2 in the range of 99-100% throughout and blood loss was estimated at 100 ml. Upon return of spontaneous breathing, 1.6 mg of morphine sulfate was titrated in 0.2mg IV incremental boluses according to respiratory rate. At the conclusion of the procedure, the surgeon inserted a 16 French orogastric tube (OGT) and suctioned a moderate amount of bloody secretions. Following this, the oropharynx was suctioned by the anesthesia professional using a yankauer tonsil tip catheter and a 70 mm oral pharyngeal airway was inserted. The patient was suctioned a third time with a Y-suction catheter through the oral airway to remove thick yellow secretions. The ETT was removed from the trachea with the patient breathing spontaneously at a rate of 15 breaths per minute and in a deep plane of anesthesia expiring 3.4 % end tidal concentration of sevoflurane in 3 L/min of oxygen. Following extubation, the patient continued to spontaneously breath with no signs of obstruction, and was transported to the post anesthesia care area (PACU) with a face-mask of oxygen at 8 L/min.

Upon arrival in the PACU, the SpO_2 was 96%. Auscultation of the lungs revealed coarse lung sounds. He was suctioned through the oral airway and yellow secretions were again removed. Upon awakening and approximately 15 minutes after arriving in the PACU, the patient was restless, and SpO_2 was 92%. Breath sounds continued to sound coarse and a chest radiograph was ordered. A face-mask was applied and humidified oxygen was administered at 6 L/min. The SpO_2 continued to stay at 92% for about 15-20 minutes and began to slowly trend up to 94%. After approximately 30-45 minutes, the PACU anesthesia physician and surgeon decided to

admit the patient for observation based upon symptoms. He was discharged 24 hours later with no complications and an unremarkable chest radiograph.

Discussion

Obstructive sleep apnea (OSA) occurs when the upper airway is obstructed in some way during sleep, which causes "repetitive arousal from sleep to restore airway patency, which may result in daytime hypersomnolence or other daytime manifestations of disrupted sleep…".[2] It can result in episodes of hypercarbia and oxygen desaturationswhich can compromise cardiac function. Common signs of OSA include a history of snoring, frequent somnolence despite adequate sleep time, and pauses in breathing or a choking sensation during sleep.[3] Conditions associated with OSA include obesity, hyperplasia of the tonsils and adenoids, muscular dystrophy, Down syndrome, and craniofacial abnormalities.[2]

This case report describes an example of respiratory distress following tonsillectomy and adenoidectomy in a pediatric patient with a history of OSA. Patients with a history of enlarged tonsils and OSA are at risk for developing NPPE, Type II following surgical relief of the upper airway obstruction. A review of the pathophysiology, clinical manifestations, differential diagnosis and management of patients manifesting signs of Type I and Type II NPPE will be presented.

The pathogenesis of NPPE is multifactorial and related to disturbances of one of four mechanisms: increased hydrostatic pressure in the pulmonary capillary bed, decreased osmotic pressure of plasma, increased permeability of the membrane, and decreased return of fluid to the circulation by way of the lymphatics.[1] The predominant mechanism is forced inspiration against a closed glottis inducing a negative gradient of intrapleural and transpulmonary pressure.

In children, normal intrathoracic pressure is -2.5 to -10 cmH_2O; however, breathing against an obstructed airway can create a large negative intrathoracic pressure (>-30cmH_2O).[3] The high pressure gradient causes the transudation of fluid from the pulmonary vessels into the interstitial spaces and alveoli of the lungs, resulting in pulmonary edema.[4] When a large amount of fluid enters the interstitial compartment, alveoli flooding occurs. The marked increase in intrapleural negative pressure leads to increased venous return to the right ventricle. The result is decreased left ventricular output leading to an elevation in pulmonary blood volume and microvascular pressure. The increased volume and pressure in the right ventricle causes distension of the septum into the left ventricle. This results in a decrease in compliance, increase in workload and increase in end diastolic and end systolic ventricular volumes.[1] In addition, lack of alveolar oxygenation during the obstruction results in hypoxemia. The result is a hyperadrenergic state. Peripheral vasoconstriction shunts blood centrally. Damaged pulmonary capillaries exhibit increased permeability and hypoxia-mediated pulmonary vasoconstriction all of which favor the formation of pulmonary edema.[1]

NPPE is categorized as Type I or Type II. Type I occurs directly after an acute upper airway obstruction, commonly from laryngospasm. The etiologies for Type I are strangulation, upper airway tumors, epiglottitis, croup, ETT obstruction, and mononucleosis.[1] The cause of Type II is less clear. It develops in patients when a chronic upper airway obstruction (UAO) such as

hypertrophic tonsils and adenoids or upper airway tumor is removed.[1] In Type II NPPE, it is postulated that UAO leads to a ball valve effect, whereby large positive intrathoracic pressures are generated. It is thought that the obstruction may actually limit the fall in oxygen saturation and transpulmonary capillary pressure as the result of positive end-expiratory pressure (PEEP) it creates. Once the obstructive tonsils or mass are removed, PEEP disappears. Venous return then increases, along with pulmonary blood volume and pulmonary hydrostatic pressure, creating a negative intrapulmonary pressure and transudation of fluids into interstitial space and alveoli of lungs.[1] The incidence of Type I NPPE developing with acute postoperative upper airway obstruction is 9.6–12%; for Type II NPPE the rate of manifestation is 44%. [1] In adults, 50% of the NPPE cases are a result of post-operative laryngospasm.[1]

Symptoms of NPPE can develop rapidly after extubation or can be delayed a few hours due to the positive pressure created by the patient expiring against a closed glottis. Therefore, a patient at risk must be observed in the PACU for an extended period of time. The initial clinical presentation usually includes decreased oxygen saturation, pink frothy sputum, wheezing, and chest radiograph with diffuse infiltrates.[3] Signs of acute airway obstruction include stridor, use of accessory muscles for inspiration and retractions, tachypnea, tachycardia, secretions, and a an expression of panic.[1] On auscultation, crackles and wheezes can be heard. Hypoxemia is the result of impaired diffusion of oxygen and ventilation/perfusion mismatching.[1]

This patient presented in this case report was admitted for observation because he was showing early signs and symptoms consistent with NPPE, Type II. This patient had a history of OSA with an AHI of 6, which is considered mild to moderate OSA, and suggests need for surgical treatment. This put him at risk for development of NPPE.[2]

The initial management for patients exhibiting NPPE is oxygen therapy, correction of the hypoxemia, and removal of the airway obstruction.[1] Oxygen administration can be supplied with 5-10 cmH_2O of PEEP or continuous positive airway pressure to resolve the pulmonary edema.[5] In some severe cases of NPPE, intubation with an ETT and 100% oxygen mechanical ventilation is needed. Diuretics to decrease the intravascular volume are controversial because it can cause hypovolemia and hypotension in post-surgical patients.[5] According to Bhaskar, "NPPE resolves generally within 24 h. However, when recognition is delayed, patients with NPPE have mortality rates ranging from 11% to 40%."[1]

Currently, there are no measures to accurately predict a child's risk for developing NPPE after the obstruction is corrected.[3] However, to decrease the risk, the anesthesia professional should take measures to avoid laryngospasm by assuring adequate anesthesia during mask ventilation or deep extubation, or ensure a patient is fully awake with adequate airway function prior to extubation.[5] This patient was adequately anesthetized for a deep extubation with an end-tidal sevoflurane concentration of 3.4%, along with no changes in heart rate or cough with oropharynx suctioning. His airway was clear after extubation and he did not exhibit laryngospasm. The patient was admitted for observation due to an awake SpO_2 of 92% on oxygen, coarse lung sounds and a history of OSA. Symptoms resolved in less than 24 hours.

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Anesthetic Implications for a Patient with Gitelman's Syndrome

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Keywords: Gitelman's syndrome, metabolic acidosis, Bartter's syndrome, renal tubular disorder

Gitelman's syndrome (GS), first described in 1966 and also referred to as Gitelman's variant of Bartter's syndrome, affects an estimated 1 in 40,000 people worldwide.[1] GS is a congenital, autosomal recessive, renal tubular disorder, characterized by chronic hypokalemia, hypomagnesemia, and hypocalciuria associated with metabolic alkalosis and normal blood pressure. During routine anesthesia care, the metabolic acidosis associated with GS can be further exacerbated by preoperative fasting, anesthetic medications, endotracheal intubation and controlled respirations, and intraoperative fluid and electrolyte management.[1] Without proper anesthetic planning and strict patient monitoring, GS can precipitate perioperative respiratory, cardiovascular, musculoskeletal, and neurologic complications.

Case Report

A 37-year-old, 160 cm, 62.7 kg, female presented for urgent laparoscopic cholecystectomy following acute cholecystitis with cholelithiasis. The patient's medical history was significant for GS and chronic hypokalemia. Her surgical history included functional endoscopic sinus surgery under general anesthesia. Her medication regimen included amiloride hydrochloride and potassium chloride. A preoperative electrocardiogram (ECG) showed normal sinus rhythm with a heart rate of 78 beats/minute, a PR interval of 0.17 seconds, a QRS interval of 0.09 seconds, a QT interval of 0.48 seconds, and regular ST segments. Preoperative blood pressure was 97/60 mm Hg, heart rate was 72 beats/min and potassium level was 2.9 mEq/l.

On arrival to the operating room, the patient was given intravenous (IV) midazolam 2 mg and noninvasive monitors were applied. Pre-oxygenation was initiated with O2 flows at 6 L/min via face mask. IV induction was achieved with fentanyl 100 mcg, lidocaine 50 mg, propofol 150 mg, and rocuronium 40 mg. The trachea was intubated and placement was confirmed with positive end-tidal CO2 and auscultation of bilateral breath sounds. The patient was then placed on synchronized intermittent mandatory ventilation.

Anesthetic maintenance included an expired sevoflurane concentration of 2% with 1.5 L/min of O2 and 1.5 L/min of air. The patient was given acetaminophen 1000 mg and three fentanyl 50 mcg boluses for procedural pain control. Dexamethasone 10 mg and ondansetron 4 mg were given prior to incision. Abdominal insufflation was initiated without complication, and trendelenburg positioning was utilized for increased laparoscopic visualization. ECG was continually monitored and consistently correlated to preoperative baseline measurements. An additional 10 mg of rocuronium was used for muscle relaxation during the procedure. A total of 1500 ml of Lactated Ringer's (LR) was given.

Following completion of the procedure, neuromuscular blockade was antagonized with neostigmine 3 mg and glycopyrrolate 0.4 mg. When extubation criteria were met the oropharynx was suctioned and the endotracheal tube was removed without complications. 6 L O2 via simple mask was utilized with SpO2 98%. Transfer to the recovery room was uneventful and vital signs remained stable.

Discussion

GS is typically diagnosed in adolescence and early adulthood when symptoms such as muscle weakness, cramping, spasms, tetany, and/or fatigue present along with laboratory abnormalities such as metabolic alkalosis and hypokalemia.[1] GS is associated with distal convoluted tubule defects, and has been linked to loss of function in the thiazide sensitive Na-Cl cotransporter (TSC or NCCT).[2] The NCCT is responsible for reabsorption of Na-Cl across the basolateral membrane of renal intraluminal cells. Patients with defective NCCTs experience salt wasting, which eventually leads to hypovolemia and metabolic alkalosis.[2] Etiology of the NCCT dysfunction is controversial, but in 1996 Simon et al[3] first displayed a variety of nonconservative amino acid substitutions and splice site mutations on chromosome 16q13 of the NCCT gene. Since then, more than 140 mutations of the NCCT gene (SLC12A3) have been described.[3]

The management of GS patients undergoing general anesthesia is complex due to the many pathophysiologic changes. It is imperative for the Certified Registered Nurse Anesthetist (CRNA) to consider these changes as they relate to cardiac monitoring, impaired vascular tone, fluid volume management, electrolyte monitoring and replacement, and medication management.

Potassium and/or magnesium depletion occurring in GS can lead to prolonged action potential duration of a cardiac myocyte, thus increasing the QT interval on ECG. A prolonged QT interval will increase the risk for developing ventricular arrhythmias, which could advance to syncope and sudden cardiac death.[4] The case study patient had a decreased potassium level upon admission, and had a slightly increased QT interval on initial ECG. Her acute cholecystitis could have further exaggerated the decreased potassium, sodium, chloride, and magnesium levels.

Subsequently, there was the potential for further metabolic alkalosis and QT interval prolongation. The preoperative ECG obtained was compared with intraoperative and postoperative ECG tracings to monitor for acute changes. Also, preoperative and postoperative blood draws were completed to strictly monitor electrolyte status.

Patients with GS typically present with a decreased intravascular fluid volume, reduced peripheral vascular resistance, and normo to hypotension.[1] This normotension to hypotension is consistent despite activation of the Renin-Angiotensin-Aldosterone System (RAAS) by decreased sodium levels. Activation of the RAAS typically leads to hypertension through angiotensin II signaling throughout the body, yet this system is overcome by the salt wasting associated with GS.[5] The decrease in vascular tone has been attributed to defective sympathetic and parasympathetic nervous systems[6]; and/or decreased responsiveness to vasoconstrictors through altered cellular signaling transduction systems coupled with the upregulation of the nitric oxide synthetase system.[5]

For elective surgical procedures, Bonfante et al[7] describe managing a GS patient with pre-procedure saline infusions (1.5 Liters/3 times per week) in order to overcome the chronic hypovolemia, hypokalemia, and hypotension. The case study patient was slightly hypotensive in the preoperative holding room: BP 97/60 mmHg. Throughout the procedure the IV fluids and two boluses of phenylephrine 100 mcg were utilized successfully for blood pressure control. In future GS cases, an operating room with blood tubing and fluid warmer prepared, large bore IV access, and a colloid readily available, would be more prudent in the event of difficult blood pressure control.

A higher prevalence of polyuria and nocturia, accentuating their decreased circulating fluid volume[8] places the GS patient at risk for developing chronic nephropathy due to acute prerenal failure, secondary to chronic hypovolemia.[7] Preservation of kidney function is further complicated by the fasting status of surgical patients. Anesthesia practitioners need to recognize the chronic and acute fluid volume deficits associated with GS patients. The case study patient was managed with LR infusion according to fluid volume requirements calculated from patient ideal body weight. A total of 1500 mL of LR was administered. Based on the evidence, it may have been beneficial to give an additional 1500 mL of LR to compensate for the chronic hypovolemia and NPO status of the patient. Additionally, the placement of a urinary catheter may have been beneficial to improve intraoperative fluid volume assessment and guide fluid management.

Hypokalemia leads to fatigue, weakness, muscle cramps, and pain. Severe manifestations of hypokalemia can include hand and feet spasms, tetany, rhabdomyolysis, and paralysis.[5] In the 2001 descriptive study by Cruz et al[8] 84% of the GS patients reported having some degree of cramping, with 42% reporting frequent cramping. Patients who sought medical attention for the musculoskeletal manifestations of GS were typically treated with intravenous potassium and/or magnesium and started oral supplementation for prophylaxis.[8]

The patient in the case report was asymptomatic despite her hypokalemia: potassium level 2.9 mmol/L. After anesthetic planning and discussion the CRNA and anesthesiologist realized her potassium level was in her normal range and did not want to increase the level because her body

had adapted to this hypokalemia. As discussed previously, a baseline ECG and continuous ECG monitoring in the operating room were compared to assess for symptomatic electrolyte disturbances needing attention during the perioperative period.

GS is one of the most common inherited renal disorders affecting patients today. An awareness of the potential complications common in patients with GS will allow for more consistent and precise monitoring, patient management, and delivery of anesthesia for these patients. Anticipating and thoroughly preparing an evidence-based and patient specific anesthetic plan will help minimize perioperative management difficulties and enhance patient safety.

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Anesthetic Considerations a for Patient with Cri du Chat Syndrome

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Keywords: Cri du Chat, cat cry syndrome, hypotonia, microretrognathia, airway anatomy

Also known as cat cry syndrome or 5p-syndrome, Cri du Chat is a rare genetic condition that is caused by deletion of genetic material on the small arm (the p arm) of chromosome 5.[1] The most common manifestations usually include a high-pitched cat-like cry, mental retardation, delayed development, distinctive facial features, small head size (microcephaly), widely-spaced eyes (hypertelorism), low birth weight, and hypotonia.[2] Of highest concern are the adverse effects on the respiratory, cardiac systems, and neurologic systems, but the gastrointestinal, musculoskeletal, and endocrine systems must also be evaluated judiciously prior to development of an anesthetic plan. A detailed summary of these anesthetic implications will be discussed.

Case Study

A 14-year-old female with known Cri du Chat syndrome was scheduled for tooth extraction. The patient weighed 35.5 kg; an accurate height was unable to be obtained due to contractures. The patient's medical history included Cri du Chat, severe muscle hypotonia, nonverbal, dysphagia, percutaneous endoscopic gastrostomy tube, and gastroesophageal reflux disease. Patient's parents denied cardiac, pulmonary, or additional neurologic conditions. The patient had undergone knee surgery in the past with a reported history of difficult intubation and respiratory arrest post narcotic administration. Additionally, she had undergone bilateral myringotomy tubes, hip joint surgery, eye surgery, and gastrostomy without complications.

Preoperative vitals were as follows; blood pressure 76/47 mm Hg, heart rate 100/min, respiratory rate 16/min, oxygen saturation on room air 100%. No laboratory values were obtained. Lung sounds were clear to auscultation bilaterally, and heart rate and rhythm were regular. The patient had a very small mouth opening with a decreased thyromental distance and a Mallampati 3 classification. Hypotonia was present in the patient's neck muscles, and she was unable to support her head independently. After administration of midazolam 15 mg via gastric tube, a 22G IV was placed in the left antecubital region. The patient then received IV ondansetron 4 mg.

Following transport to the operating room, pulse oximetry, blood pressure cuff, and electrocardiogram leads were placed, and the patient was preoxygenated with 10 L/min O_2 for 5 minutes. Due to patient's history of difficult intubation, video laryngoscopy and difficult airway cart were immediately available in the operating room. General anesthesia was induced with lidocaine 40 mg and propofol 100 mg. Mask ventilation was established without difficulty and rocuronium 10 mg was administered. Additionally, sevoflurane was administered via face mask. Direct laryngoscopy with a Miller 2 blade provided a grade 1 view of the patient's vocal cords. Placement of a 6.0 cuffed endotracheal tube was attempted unsuccessfully. Placement of a 5.5 cuffed endotracheal tube was successful with inflation of cuff provided with 0.5 ml air. The endotracheal tube was taped at 18 cm at the lip, and there was no change in dentition or mucosa from preoperative state. Patient was placed on pressure support ventilation to maintain tidal

volumes 215-240 ml until return of spontaneous ventilation.

General anesthesia was maintained with sevoflurane 1.7-2.0% end tidal concentration in O_2 2 L/min. Vital signs remained stable throughout induction and no vasopressors were required throughout the case. The oral surgeon localized the extraction sites with bupivacaine 0.25% 6 mL, and no opioid was administered throughout the case. With return of spontaneous ventilation, patient maintained adequate tidal volumes and neuromuscular blockade was antagonized with neostigmine 1 mg and glycopyrrolate 0.2 mg at the end of the case. The endotracheal tube was removed after pupils were midline and patient eye opening to voice. She was monitored for apnea in the post anesthesia care unit for 45 minutes and discharged home the same day without need for opioid administration in recovery.

Discussion

Most cases of Cri du Chat are not inherited. Rather, the chromosomal deletion occurs as a random event during the formation of reproductive cells or in early fetal development, and is diagnosed at birth. The cause of this deletion is unknown and occurs in 1:15,000 to 1:50,000 births.[2] People with Cri du Chat typically have no history of the condition in their family.[1] In 10-15% of cases, one of the parents is a carrier of a chromosomal abnormality, known as a translocation. The parent, not showing any signs of having the syndrome itself, unknowingly transfers the abnormality to his/her offspring. Because of this, it is recommended that parents receive genetic testing following diagnosis of their child.

Patients with Cri du Chat have various presentations. The presence of such variations may be related to the size of the chromosomal deletion. These patients may present with characteristic high-pitched cry, facial abnormalities, mental and physical retardation, hypotonism, microcephaly, microretrognathia, possible high palate, and varied neural, renal and cardiac abnormalities.[3] Most individuals with Cri du Chat syndrome have difficulty with speech and language development, but half of these children learn sufficient verbal skills to communicate.[1] Additional characteristics may include feeding difficulties, delays in walking, hyperactivity and scoliosis. Although a small number of children are born with serious organ defects and other life-threatening medical conditions, most individuals with Cri du Chat syndrome have a normal life expectancy. Not all patients with Cri du Chat present with all of these symptoms, but potential abnormalities involving airway anatomy as well as neurological, cardiac and respiratory systems require particular assessment.

As this patient did not exhibit any cardiac or neurologic concerns, other than muscular hypotonia, the main concern was the patient's known difficult airway and history of respiratory compromise following opioid administration. A thorough airway assessment was difficult to obtain due to the patient's inability to cooperate. Obvious visible concerns were present, including a small recessed chin and small mouth opening. Difficult airway supplies were immediately available in the operating room, including Glidescope, laryngeal mask airways, bougies, multiple endotracheal tubes and laryngoscope blades, as well as emergency airway supplies. After consultation with the surgeon, it was determined that a regular oral endotracheal tube would be acceptable for these two extractions, avoiding nasal intubation and potential trauma to the airway. Trauma to the airway could result in swelling and a bloody field obscuring

visualization and leading to laryngospasm. Elective fiberoptic intubation is another option that could have been utilized in this situation. This could be accomplished safely with a ketamine induction to maintain spontaneous respiration, intranasal oxymetazoline to prevent epistaxis, nasal airway, laryngotracheal topical anesthetic for supraglottic and glottic anesthesia, and a pediatric fiberoptic bronchoscope for visualization of the airway.[4] This patient was successfully and atraumatically intubated with direct laryngoscopy.

Gastrointestinal reflux is common in Cri du Chat patients, and general anesthesia exacerbates weak sphincter tone at the gastroesophageal junction. Particular attention should be paid to NPO status and home medications, with consideration then given to preoperative treatment. Rapid sequence intubation should be utilized if indicated.

The patient's hypotonia was taken into careful consideration. An anesthetic technique that minimizes respiratory depression should be utilized with drugs that are short-acting and rapidly metabolized.[5] Children with neuromuscular disease will typically have an increased sensitivity to non-depolarizing neuromuscular blocking agents. The use of a nerve stimulator and short-acting neuromuscular blocking agents is recommended only if surgically necessary. In patients with neuromuscular disease, succinylcholine should generally be avoided as there can be dramatic, life-threatening increases in serum potassium, as well as an increased risk of malignant hyperthermia and rhabdomyolysis.[5] However, there is no known association with these risks in hypotonia produced by Cri du Chat syndrome.[6] Furthermore, administration of opioid decreases patient response to hypercarbia, decreasing the respiratory drive and compromising the respiratory system in a patient with concurrent baseline muscle weakness. Hypotonia can include pharyngeal muscles, and patients should be observed closely for postoperative airway obstruction.[6] In this case, propofol and a small dose of rocuronium were administered to facilitate tracheal intubation. No opioid was administered and the patient's respirations were supported transiently until timely return of spontaneous ventilation. Neuromuscular blockade was antagonized at the end of the case to ensure adequate return to baseline respiratory function. Patients with Cri du Chat can present with multisystem involvement, which almost always include airway abnormalities. When caring for a patient with Cri du Chat syndrome, the anesthesia practitioner must tailor the anesthetic plan to the unique needs of this population, paying particular attention to airway concerns and muscular hypotonia.

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Managing Accidental Dural Punctures with Spinal Catheters

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Introduction

Regional anesthesia is the most commonly used non-emergent technique in the parturient patient population.[1] It allows a safer delivery of anesthesia for both mother and baby when compared to general anesthesia in non-emergent situations.[1] However, there are still risks with this technique. One of these risks is an accidental dural puncture (ADP). An ADP is typically encountered during attempts to locate the epidural space when the Tuohy needle is unintentionally inserted past the epidural space, through the dura, and into the subarachnoid space. The passage of the Tuohy needle into the subarachnoid space produces a tear in the dura, creating a route for cerebral spinal fluid to leak out of the spinal canal, even after the needle has been removed. The leakage of CSF lowers intracranial pressure and is thought to be the main causative factor in the development of postdural puncture headache (PDPH).[2]

While there is a multitude of available research on the treatment of PDPH, less information is available on the prevention on PDPH following ADP. Prior to the 1990s, when an ADP occurred, it was common practice to remove the needle and re-site the epidural.[3] An alternative method of managing an ADP by passing an epidural catheter through the needle into the subarachnoid space was first described by Cohen et al. in 1989.[3] Since the inception of this spinal catheter technique many benefits have been noted in the literature regarding this approach, which include; reduced rates of PDPH, a more rapid establishment of analgesia, and a lower rate of repeat dural puncture and/or epidural catheter failure.[4-7] In contrast, the literature also implies mixed results and will require further review in order to determine firm evidence-based practice recommendations. The purpose of this analysis is to determine if the evidence supports utilization of a spinal catheter following ADP as best practice. Current literature was reviewed to better understand the incidence of ADP, complications resulting from ADP, and an investigation of the two management techniques.

Methodology

Evidence-based Analysis Model

The PICO format assisted in framing a clinical question into a relevant search criterion. The PICO parameters include: P (patient population) = Parturient patients with an ADP, I (current intervention) = placement of an epidural catheter into the subarachnoid space, C (contrasting intervention) = removal of epidural needle and reattempting placement of an epidural catheter into an adjacent epidural space, O (outcome of interest) = the rate of PDPH and the rate of complications secondary to ADP management technique.

Purpose

The purpose of this evidence-based practice analysis is to examine the incidence of ADP complications following either placement of an epidural catheter in the subarachnoid space or reattempting epidural catheter placement into an adjacent epidural space in the laboring parturient population.
The clinical questions that guided the literature search were: (1) In the parturient population does the ADP management technique impact the rate of PDPH and (2) Is there a significant difference in the rate of complications secondary to the chosen ADP management technique?

Search Models
Research articles were obtained through electronic databases such as: PubMed, EBSCO, and OVID. The search for literature was confined between the years 2009-2014 in order to ensure the most current information was included in this literary analysis.

Search Terms
Accidental dural puncture, inadvertent dural puncture, spinal catheter, intrathecal catheter, epidural catheter, complications, and postdural puncture headache.

Levels of Evidence
Eleven studies were included in the evidence-based practice analysis. Of the eleven studies there were one meta-analysis and one quantitative systematic review, both provided Level I evidence. Three studies which produced Level II evidence, a non-randomized prospective controlled cohort study and two retrospective cohort study. Lastly, six articles are deemed expert opinions and are classified Level V evidence.

Literature Analysis

The incidence of ADP during epidural placement has a range of 0.19% to 3.6% in the obstetric population, but is found to be as high as 6.6% in one study.[5,8] The incidence of PDPH following ADP with an epidural needle has been found to range from 52% to 88%.[2] PDPH is a serious complication of ADP because symptoms can be severe enough to prevent the mother from caring for her newborn, increase length of hospital stay, and relate to a possible increase in morbidity and mortality in the obstetric population.[8] Serious complications resulting from an ADP include: postural headache, orthostatic hypotension, diplopia, tinnitus, dizziness, neck pain, myalgia, nausea, vomiting, and subdural hematoma.[2,4] Typically, PDPH "symptoms develop within 48 hours and may be delayed up to five days".[4 (p3)] "Generally, there is gradual improvement in the symptoms with time. Seventy percent tend to resolve within one week, 95% within six weeks, and 96% within six months".[4 (p3)]

Common practice for the management of an ADP is to remove the epidural needle and to reattempt insertion of the epidural needle at an adjacent vertebral interspace. Reattempting epidural placement in a patient that has already experienced an ADP carries a 9% risk of another ADP.[7] Laboring anesthesia can be administered through the spinal catheter allowing for a more rapid progression of regional anesthesia, which negates the need for a second epidural attempt.[5,7,9] Both ADP management techniques carry a risk for complications; however, the literature demonstrates the rate of complications to be three times greater in the epidural re-site group compared to the spinal catheter group.[9] The literature also demonstrates that a spinal catheter results in a reduction in the rate of PDPH, although conflicting data on this topic is present in the current literature. In an effort to provide the most current evidenced-based practice recommendations, six articles with research data were reviewed. The results are summarized in Table 1.

Table 1. Recent literature regarding PDPH rates and neuraxial complications

Articles	Description	Results
Apfel, Saxena, Cakmakkay, Gaiser, George, and Radke, 2010[8]	Quantitative systematic review Prevention of postdural puncture headache after accidental dural puncture	<u>Short-term spinal catheter versus re-siting epidural:</u> Spinal catheter: 145 total ADP, 90 PDPH (62% PDPH rate) Epidural re-site: 161 total ADP, 124 PDPH (77% PDPH rate) Test for overall effect: *Z*=0.99 (P = 0.32) <u>Long-term spinal catheter versus re-siting epidural:</u> Spinal catheter: 104 total ADP, 32 PDPH (30% PDPH rate) Epidural re-site: 214 total ADP, 123 PDPH (57% PDPH rate) Test for overall effect: *Z*=1.20 (*P*=0.23) Reduction of PDPH was not significant in the short-term or long-term spinal catheter group
Heesen, Klöhr, Rossaint, Walters, Straube, Van de Velde, 2013[5]	Meta-analysis Insertion of an intrathecal catheter following accidental dural puncture	Spinal catheter: 469 total ADP, 258 PDPH (55% PDPH rate) Epidural re-site: 470 total ADP, 310 PDPH (65% PDPH rate) Test for overall effect: *Z* = 1.85 (*P* = 0.06) The incidence of PDPH headache was reduced but not to a rate that concluded significant results.
Russell, 2012[6]	Prospective controlled study Continuous spinal analgesia versus repeat epidural analgesia after accidental dural puncture in labor	Spinal catheter: 50 total ADP, 36 PDPH (72% PDPH rate) Epidural re-site: 47 total ADP, 29 PDPH (62% PDPH rate) (*P* = 0.2) PDPH were demonstrated to be higher in the spinal catheter group compared to the re-site epidural group. The difference in the PDPH could not conclude significant results Risk of another ADP with reattempting epidural placement was demonstrated to be 9 % Participants in the repeat epidural group suffered complications at a rate three times greater than those in the spinal catheter

		group
Van de Velde, Schepers, Berends, Vandermeersch, De Buck, 2009[7]	Retrospective cohort study Ten years of experience with accidental dural puncture and post-dural puncture headache in a tertiary obstetric anesthesia department	Spinal catheter: 27 total ADP, 14 PDPH (52% PDPH rate) Epidural re-site: 28 total ADP, 17 PDPH (61% PDPH rate) ($P > 0.05$) Review of literature: Spinal catheter: 317 total ADP, 161 PDPH (51% PDPH rate) Epidural re-site: 282 total ADP, 187 PDPH (66% PDPH rate) $Chi^2 = 14.8$; $P < 0.001$ Significant results concluded that spinal catheters reduce the rate of PDPH. However, "most trials or case series were non-randomised, unblinded or of insufficient power to detect a difference"[9 (p334)]
Rahim, Ranasinghe, Moaveni, Cohn-Hochman, 2013[11]	Retrospective cohort study Complications of Continuous Intrathecal Catheters in Obstetric Patients	Spinal catheter: 761 total ADP, 312 PDPH (40.9% PDPH rate) Overall failure rate of spinal catheters was 5.9%, 45 of the 761 inserted spinal catheters Four patients in the spinal catheter group requiring intubation and ventilation resulting from a high block There were no reported cases of meningitis, arachnoiditis, cauda equina syndrome or epidural hematoma
Silva and Halpern, 2010[10]	Expert Opinion review of literature Complications of neuraxial analgesia	Complication — Epidural — CSE Failure rate — 14% — 10% Dural puncture — 0.21%-1.6% — 0.20%-1.7% Nerve damage cause by needle trauma — 0.6:100,000 — 3.9:100,000 Epidural abscess — 3 per 100,000 — ? Meningitis — 0–3.5:100.000 — 0–3.5:100,000 Epidural hematoma — 1:168,000 — ?

PDPH Prevention

The two main neuraxial regional techniques utilized in the parturient population involve the delivery of anesthesia either into the epidural space or into the spinal canal. Both techniques carry an inherent risk for PDPH. Since the 1960s, there has been a bounty of literature on the prevention of PDPH with the administration of spinal anesthesia, termed a subarachnoid block**.** However, until recently, literature has been limited on interventions that can be utilized to reduce the rate of PDPH following ADP with an epidural needle. In 1989, a technique was published involving passing an epidural catheter into the subarachnoid space and dosing this catheter in increments appropriate for a spinal dose in order to provide laboring analgesia.[3] In this case study all 10 patients obtained appropriate relief from labor pain, but what Cohen et al. also noted was that only two of his 10 patients developed a PDPH.[3] With the spinal catheter technique, a 20% rate of PDPH following ADP was demonstrated, which was found to be much lower than the national average of 52 to 88% with re-siting the epidural.[2,3] While the evidence from this study appears convincing, a control group and power analysis are not included and therefore the impact on current practice is not clear.[3]

Cohen et al's study was the first to examine the spinal catheter as an intervention following ADP, while it lacked the ability to independently change current practice at that time, it did create a foundation for research. Apfel et al. and Hessen et al. each performed a meta-analyses to determine if a spinal catheter demonstrated a reduced rate of PDPH when compared to re-siting the epidural.[5,8] Use of the spinal catheter technique did not demonstrate a statistical difference in the prevention of PDPH, with a RR of 0.88 (95% CI 0.68–1.14, $P < 0.32$) for PDPH for short-term spinal catheterization and a RR of 0.21 (95% CI 0.02–2.65,$P < 0.23$) for long-term spinal catheterization, defined as a period longer than 24 hr.[5,8] Van de Velde et al. performed a prospective non-randomized control trial and concluded similar results as the two previous studies, with spinal catheters showing no clear benefit on PDPH rates.[7] "When data from obstetric reports are combined, prolonged intrathecal catheter placement significantly reduces the risk of PDPH after ADP to 51% compared with 66% in those who have the catheter re-sited epidurally ($Chi^2 = 14.8$: $P < 0.001$)". [7 (p334)] It was noted in this study that most trials in the review of literature were non-randomized, the researcher was not blinded to data collection, or the trial was of inadequate power to detect a difference.[9] One conclusion that was universally agreed upon is that spinal catheter placement following ADP is a promising intervention for preventing PDPH and that a large, multi-center, prospective, randomized, blinded trial is needed to clarify this issue.[5-8]

Decreased rate of complications with spinal catheters

Russell performed a prospective controlled study of continuous spinal analgesia versus repeat epidural analgesia after accidental dural puncture in labor, and what was demonstrated in this study was an increased rate of complications when the epidural was reattempted following an ADP.[6] The risk of another ADP with reattempting epidural placement was demonstrated to be 9 %; this was in line with other previous studies.[6,9] In fact, it was noted in this study that participants in the repeat epidural group suffered complications at a rate three times greater than those in the spinal catheter group.[9] Common complications encountered in each study include: multiple repeat ADP, ineffective analgesia when the epidural is placed on subsequent attempts, epidural catheter failure, total spinal block, and epidural vein puncture with a subsequent high block.[6,9] In addition, the use of a spinal catheter was not without its complications in this study, including spinal catheter failure, failure due to paresthesia, failure to pass spinal catheter, and a total spinal.[6]

In a retrospective quality assurance study, 12,590 regional anesthetic procedures for labor analgesia were reviewed, in this study it was noted that 14% ($P < 0.001$) of inserted epidurals failed.[10] In this trial, 5.6% of the epidural catheters placed had to be removed and replaced, despite functioning initially upon insertion.[10] While this study does not offer a comparative data analysis due to the fact that spinal catheters were not specifically addressed, it should be noted inadequate analgesia with the epidural catheter alone was reported at 8.4% while the combined single shot spinal-epidural (CSE) group reported 4.2%.[10] One can deduce that incorporation of spinal anesthesia improved patient analgesia compared to the use of the epidural as the sole route of delivery.[9]

Rahim et al performed a retrospective cohort study examining the rate of spinal catheter complications following ADP.[11] A total of 761 patients had spinal catheters placed during the

study period, and of the 761 catheters placed, 45 failed (5.9%).[11] The other complication noted was that four patients had respiratory problems resulting from a high block and required intubation.[11] These findings indicate spinal catheters have a relatively low rate of failure when compared to epidural catheters, 5.9% compared to 14%. When anesthesia personnel cause an ADP after having difficulty finding the epidural space, it appears more beneficial to place a spinal catheter and provide a route for rapid anesthesia rather than to place the patient at risk for further complications with re-siting the epidural.[6,9,11]

Despite the literature available to support spinal catheter use, recent surveys have found that only 25 to 36% of ADPs are managed with spinal catheter insertion.[2] Accidental misuse and infections were the most common listed reason for not placing a spinal catheter and opting to re-site the epidural. The fear of misuse is that anesthesia personnel would dose the spinal catheter as though it were in the epidural space, which could be fatal. There is potential for this problem in that the catheters look identical in appearance; however, this can be easily managed with firm adherence to clear policies and protocols.[2] Infection is the other concern anesthesia personnel list as a reason for abstaining from the insertion of a spinal catheter following ADP. The main issue is that spinal catheters present a direct connection to the central nervous system, which could potentially increase the risk of developing meningitis. In review of the literature, only one retrospective cohort study was present, yet this study involving 761 parturient patients with spinal catheters did not observe any incidences of meningitis or arachnoiditis .[11] However, there is a larger pool of data available on epidural, single shot spinal, and CSE infection rates that give potential insight into the rate of infection with neuraxial techniques in the parturient population.[10] Silva and Halpern reviewed the findings of 10 surveys and found the incidence of epidural abscess to be 0.2-3.7 per 100,000 obstetric epidurals. Similar results were obtained concerning bacterial meningitis following spinal anesthesia, 0-3.5 per 100,000 (95% CI).[10] One would conclude that this risk for infection is very low regardless of the neuraxial technique. However, further research is warranted to confirm a similar rate of infection in relation to spinal catheters and the other neuraxial techniques in practice.

Conclusion

There is conflicting evidence in the literature regarding the effectiveness of spinal catheters providing a decreased risk of PDPH. Considering that ADP has a demonstrated PDPH incidence of 52 to 88% in the obstetric population, prophylactic interventions need to be considered.[2,8] PDPH symptoms can be severe enough to prevent the mother from caring for her newborn, increase length of hospital stay, and relate to a possible increase in morbidity and mortality in the obstetric population.[8] It would therefore appear to be more sensible to enact preemptive interventions aimed at preventing the onset of PDPH rather than starting treatment after the clinical presentation.[8] Anesthesia professionals must be sure that these preemptive interventions are not responsible for further increasing morbidity and mortality.[5] More research in the form of a large, well-designed, double-blind, randomized controlled multicenter trial is necessary in order to draw more definitive conclusions in regards to spinal catheters impact on the incidence of PDPH following ADP.[5-8]

Present literature on the incidence of complications following an ADP illustrates a pattern of decreased risk with the utilization of the spinal catheter technique.[6-8] Studies by Hessen et al.,

Russell, Gaiser, and Van de Velde et al. suggest that when the anesthesia personnel has had difficulty identifying the epidural space prior to an ADP, introduction of a spinal catheter greatly reduces the risk of complications stemming from re-siting the epidural, mainly a repeat ADP and catheter failure.[5,6,9,11] The spinal catheter technique is optimal because it relieves the patient of potential suffering through another epidural or a repeat ADP.[6,9] Lastly, the spinal catheter technique allows for the anesthesia personnel to provide a more rapid and highly successful anesthetic in comparison to re-siting the epidural.[2,4-7] Based on these benefits, change in current practice should be directed toward introduction of a spinal catheter following an ADP, particularly if prior identification of the epidural space was difficult.

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Mentor: Laura S. Bonanno, CRNA, DNP

Editorial

I am always excited when I receive a submission from a nurse anesthesia program that has never submitted before. Newman University has their first (of many, I hope) report published in this issue. Since I assumed the role of Editor the number of programs represented on the board has almost doubled, and the number of individuals serving on the board has more than doubled!

On that note, I am also very pleased to welcome two new members of the Editorial Board:

Johanna Newman, CRNA , DNAP - Florida International University
Bryan Tune, CRNA, DNP - National University

As always, I am grateful for all of the hard work and time contributed by the volunteers for this journal!

Sincerely,

Vicki C. Coopmans, CRNA, PhD
Editor

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

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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.)

Case Reports

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

Heading (see #9 above in General Guidelines)

[space]

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

[space]

Case Report (bold, 400-500 words)

[space]

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

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

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

[space]

Discussion (bold, 600-800 words)

[space]

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

[space]

References (bold)

[space]

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

[space]

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

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

Research Abstracts

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

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

EBP Analysis Reports

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

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

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

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

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

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

AMA MANUAL OF STYLE

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

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

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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.

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

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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.**

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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.

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

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

SUBMISSION CHECK LIST

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

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

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

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.
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___ References are from anesthesia and other current primary source literature.
___ All inclusive pages are cited, texts as well as journals.
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