

Volume 16 Issue 1 Spring 2017

The International Student Journal of Nurse Anesthesia

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INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA
Vol. 16 No. 1 SPRING 2017

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Front Cover:

Scott Brown, RN, BSN, a graduate student enrolled in the Bryan College of Health Sciences Nurse Anesthesia Program, demonstrates his airway management skills while caring for a patient as part of his clinical practicum.

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Managing Blood Loss and Blood Transfusion for the Pediatric Patient

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Key words: Apert's syndrome, pediatrics, tranexamic acid, blood transfusion, craniofacial surgery

Apert's Syndrome is a genetically inherited disease characterized by craniosynostosis, craniofacial anomalies, and symmetrical cutaneous and bony fusion of the hands and feet.¹ Children diagnosed with this syndrome undergo numerous surgical procedures to correct the associated anomalies, many of which result in significant blood loss, requiring administration of blood products. This case study examines blood loss and transfusion requirements in a pediatric patient with Apert's Syndrome presenting for craniofacial surgery.

Case Report

A 5-year-old, 15.28 kg, 109.2 cm male presented for a LeFort II midface osteotomy, advancement with distraction osteogenesis, major cranioplasty, periorbital flap, and lateral canthal reconstruction. Pertinent medical history was significant for Apert's Syndrome, obstructive sleep apnea, and syndactyly of fingers and toes. Previous surgical history included tracheostomy, gastrostomy with feeding tube insertion, repair of cleft hand deformity, and repair of craniofacial anomalies. Due to the complexity of the planned procedure and anticipation of significant blood loss, blood typing with crossmatch was completed preoperatively. The beginning hemoglobin was 11.3 g/dL and estimated total blood volume was 1125 mL. Blood products (packed red cells and fresh frozen plasma) were available in the operating room at the start of the surgical procedure.

Anesthesia was induced by mask inhalation induction with O₂ 8 L/min and sevoflurane. After loss of lid reflex, a 22 gauge peripheral intravenous (IV) catheter was placed in the left foot and rocuronium 20 mg was administered. A Glidescope (GVL; Verathon Inc., Bothell, WA) was used to facilitate tracheal intubation and a 5.0 mm endotracheal tube inserted. Following the securing of the airway, a second 22 gauge peripheral IV was placed in the right foot, and an arterial line was inserted with sterile technique in the right radial artery. Anesthesia was maintained with isoflurane 0.8-1.5% expired concentration in a mixture of O₂ 0.5 L/min and air 1.5 L/min. Intermittent bolus doses of fentanyl 50 mcg and hydromorphone 0.1 mg were administered for analgesia throughout the case and neuromuscular blockade was maintained with vecuronium. The intra-operative temperature was measured using an axillary temperature probe; forced air warming devices and fluid warmers were utilized.

Arterial blood gases were obtained hourly and transfusions were guided by hemoglobin values, estimated blood loss, and physiologic parameters of heart rate, blood pressure and urine output. A single dose of tranexamic acid 154 mg was administered IV over 15 minutes, followed by a controlled infusion of 1 mg/kg/hour. PRBC and FFP were administered at a 1:1 PRBC (mL) to FFP (mL) ratio. Normal saline was used for maintenance IV fluid at 25 mL/hour.

Surgery and anesthesia time was 6 and 8 hours respectively. Vital signs were stable throughout without need for pharmacologic support. Intraoperative hemoglobin values ranged from 10.6 g/dL to 13.9 g/dL; 820 mL of PRBC and 821 mL FFP were transfused. The estimated blood loss was 850 mL, approximating 75% of the estimated total blood volume. While vital signs were stable at surgery culmination, due to the length of the procedure, transfusion requirements, and noted facial edema, the trachea was not extubated, the patient was directly admitted to the pediatric ICU, and placed on the ventilator per the respiratory therapist.

Discussion

Craniofacial surgery is associated with substantial bleeding, lengthy surgical times, and the need for blood transfusion.² The hemostatic changes during these procedures may be related to hyperfibrinolysis, surgical complexity, and loss of coagulation factors.² Maintenance of normovolemia is important during pediatric surgery because hypovolemia is the most common cause of anesthesia-related cardiac arrest in this population.² Predictors of blood loss during craniofacial surgery include surgical time greater than 5 hours, age less than 18 months, the presence of multiple-suture craniosynostosis, and syndromic craniosynostosis, including Apert's Syndrome.¹ This child had two risk factors for significant blood loss: surgical time greater than 5 hours and syndromic craniosynostosis.

Assuring 'normal-for-age' starting hemoglobin is imperative and blood availability prior to incision is necessary to facilitate timely administration. Appropriate measures to maintain normothermia are also critical since hypothermia leads to impaired coagulation and bleeding.² Preparation for maintenance of adequate hemoglobin began preoperatively by blood typing with crossmatch. Establishing appropriate venous access also allowed for fluid replacement and blood product administration.^{1,2} An arterial catheter facilitated beat-to-beat analysis of blood pressure, which is one variable to determine volume status, as well as aid with blood draws.^{1,2}

Although it is often necessary to transfuse during craniofacial surgery, transfusions are not benign and carry substantial risks. Potential (noninfectious) risks of transfusions include transfusion-related acute lung injury, hemolytic transfusion reactions, allergic reactions, transfusion-related circulatory overload, alloimmunization, and immunomodulation.³ The noninfectious risks account for 87-100% of fatal complications of blood transfusions.³ There are, however, numerous techniques available to minimize blood loss and transfusion; these include the preoperative administration of erythropoietin, deliberate hypotension, cell saver, antifibrinolytic administration, and acute normovolemic hemodilution.³ Although these practices are commonly employed in the adult population, limited data exists regarding these techniques in the pediatric population.

In regards to techniques available to minimize blood loss, erythropoietin stimulating agents may increase red blood cell mass in the pre-surgery time frame, but have been associated with seizures and the development of venous thrombosis in some studies.² Acute normovolemic hemodilution is a process involving the withdrawal of one's own blood and re-infusing it once intraoperative hemorrhage has subsided.³ This technique has questionable utility in children because the patient may be unable to adjust to the normal adult compensatory mechanism for anemia, which is an increase in stroke volume.³ Autologous blood transfusion, also known as

'cell salvage', is not routinely used during pediatric surgery due to the difficulty encountered with collection and processing variances compared to the adult.² Tranexamic acid has been used successfully during cardiac and orthopedic surgery in the pediatric population.⁴ Tranexamic acid is an antifibrinolytic agent administered intravenously that competitively acts to decrease the activation of plasminogen to plasmin.^{3,4} One study reported an 85% total volume reduction in red blood cell transfusion during surgery with tranexamic acid infusions.⁴ Techniques utilized to minimize blood loss and subsequent transfusion in this case included the administration of tranexamic acid.

Transfusion guidelines for infants greater than 4 months of age rarely recommend blood administration when the hemoglobin is greater than 10 g/dL.¹ These guidelines almost always recommend for hemoglobin concentrations less than 6 g/dL.¹ The determination of whether intermediate hemoglobin concentrations (6.0 - 10 g/dL) warrant transfusion should be based on the overall risk of complications and the use of a single hemoglobin trigger is not recommended.¹ Tachycardia, tachypnea, and decreased urine output can also indicate the need for an increased oxygen-carrying capacity. Other clinical indices to determine volume status, such as stroke volume variation (SVV), have been studied in a subset of the pediatric population during acute normovolemic hemodilution protocols.⁶ Significant changes in SVV were identified during acute normovolemic hemodilution and a strong correlation was identified between SVV and estimated blood volume throughout the protocol.⁶ More clinical trials are needed to determine the feasibility of using SVV determinates as guides to transfusion in the pediatric population. Although SVV measurements were not utilized in this case, it may have been beneficial for guiding fluid needs and transfusion decisions.

In this case, the decision to transfuse PRBCs and FFP was based more on clinical indices than isolated hemoglobin values. The use of albumin was not considered because it increases the incidence of postoperative administration of blood products and postoperative coagulation derangements after craniofacial procedures.⁵ It was identified, however, that intraoperative fresh frozen plasma administration has the opposite effect.⁵ During trauma surgery, early transfusion of FFP in a 1:1 ratio with PRBC may improve survival.² FFP and PRBC were administered in a 1:1 ratio for this case.

In conclusion, pediatric craniofacial surgeries are associated with significant blood loss. This case validates the need for a comprehensive pre-anesthesia evaluation and preparedness for transfusion. Additionally, knowledge of current evidence based transfusion guidelines, assuring availability of blood products, preventing hypothermia, employing a variety of monitoring devices, and utilizing pharmacologic and non-pharmacologic techniques to maintain adequate oxygen carrying capacity should be considered.

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Monitoring of Cerebral Hypoperfusion in the Beach Chair Position

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Keywords: beach chair position, cerebral hypoperfusion, shoulder arthroscopy, cerebral perfusion pressure, cerebral desaturation, cerebral autoregulation

The human brain consumes 20% of the body's oxygen supply making it highly susceptible to cerebral ischemia during episodes of prolonged cerebral hypotension.¹ Surgical procedures in the beach chair position (BCP) predispose patients to significant decreases in mean arterial pressure (MAP) and cerebral perfusion pressure (CPP) leading to cerebral oxygen desaturation and neurological insult.^{2,3} Therefore, identification and utilization of multiple monitoring techniques to minimize the risk of cerebral hypoxia should be employed during anesthesia management for BCP procedures.¹⁻⁴ The purpose of this case report is to analyze multiple monitoring techniques for preventing cerebral ischemia in the BCP.

Case Report

An 83-year-old, 103 kg, 168 cm female presented for a left total shoulder arthroplasty due to degenerative joint disease. The patient's past medical history included diverticulitis, hypertension, dyslipidemia, hypothyroidism, venous insufficiency, deep venous thrombosis, abdominal hernia, zygomatic fracture, gastroenteritis, bronchitis with bronchospasm, otitis media, pharyngitis, ileus, osteoarthritis, and high cholesterol. The patient's surgical history included cholecystectomy, appendectomy, bilateral knee arthroplasty, colectomy, open reduction internal fixation of the right zygomatic bone, wound debridement, and hernia repair. No previous anesthesia complications were noted. The patient's current medication regimen included alprazolam, amlodipine, aspirin, clonidine, doxepin, furosemide, levothyroxine, and losartan. The patient had an allergy to sulfamethoxazole-trimethoprim and sulfonamide antibiotics. Laboratory results were unremarkable. No additional diagnostic testing was ordered. A pre-

anesthetic evaluation revealed that the patient had taken amlodipine, clonidine, furosemide, and losartan prior to the procedure. Following the pre-procedural time out, midazolam 2 mg was administered intravenously (IV).

The patient was taken to the operating room, transferred to a beach chair-operating table and placed in the supine position. Intraoperative monitors, including pulse oximetry, electrocardiograph, and noninvasive blood pressure cuff, were applied. Initial vital signs were heart rate (HR) 92/min, blood pressure (BP) 159/80 mm Hg, SpO₂ 97%, respiratory rate (RR) 18/min, and temperature 36.2°C. Oxygen was administered via facemask at 12 L/min until expired O₂ concentration was greater than 85%. General anesthesia was induced with lidocaine 50 mg, fentanyl 150 mcg, propofol 200 mg, and vecuronium 8 mg intravenously. Direct laryngoscopy with a Macintosh #3 blade was performed and airway secured with a 7.0 mm endotracheal tube. Airway placement was verified via capnography, visible chest rise, and bilateral breath sounds.

The patient was placed in the BCP and a continuous noninvasive arterial blood pressure device (CNAP) was applied. Following CNAP calibration, the patient's BP was 71/39 mm Hg and MAP 50 mm Hg. Administration of phenylephrine 100 mcg, ephedrine 5 mg, and 5% albumin 500 mL intravenously resulted in a rise and stabilization of MAP. The patient's MAP ranged from 85 to 110 mm Hg for the duration of the procedure meeting the predetermined target MAP of 90 mm Hg. Anesthesia was maintained with sevoflurane concentrations of 1.8% in a mixture of medical air 1 L/min and O₂ 1 L/min. Fentanyl 50 mcg doses were administered intermittently IV.

Once incisional closure began, administration of lidocaine 50 mg, neostigmine 5 mg, and glycopyrrolate 0.8 mg was given intravenously. Spontaneous respirations returned with adequate tidal volumes. Fentanyl 25 mcg and morphine 3 mg were titrated intravenously according to respiratory rate. The endotracheal tube was removed under positive pressure and airway remained patent. The patient was transferred to the post-anesthesia care unit with O₂ 10 L/min via facemask. Vital signs following the procedure were HR 75/min, blood pressure 139/60 mm Hg, RR 14/min, SpO₂ 100%, and temperature 36.6°C. The patient was admitted for overnight evaluation. Follow-up care revealed no neurological insults or adverse events.

Discussion

General anesthesia attenuates hemodynamic compensatory mechanisms of the sympathetic nervous system and increases the risk of neurological complications due to significant reductions in cerebral oxygen supply for patients in the BCP.² The attenuation of the compensatory mechanisms decreases cerebral MAP and CPP leading to cerebrovascular ischemia due to sustained periods of cerebral hypoperfusion.⁵ While the incidence of cerebrovascular decline in the BCP is rare, a multitude of neurological insults such as brain and spinal cord ischemia, hemiplegia, transient visual loss, and ophthalmoplegia can occur.^{2,4} Anesthesia management during the BCP should include a multitude of monitoring techniques that focus on ensuring adequate cerebrovascular oxygenation, and not merely rely on traditional blood pressure monitoring.¹⁻⁵

Prolonged cerebral hypoperfusion is a predictive factor that can lead to neurological decline, but inconsistencies regarding the safe lower limits of cerebral autoregulation persist among health care professionals.⁴ Historically, the safe lower limits of cerebral autoregulation were thought to be 50 mm Hg, but recent research disputes this limit, which may predispose patients to cerebral ischemia.^{2,4} Safe lower limits of cerebral autoregulation have now risen to a MAP of 70 mm Hg, but may not be adequate for individuals with certain co-morbidities and positional concerns.^{1,2,4} The BCP may require increased lower limits of cerebral autoregulation to account for differences in height between the site of blood pressure measurement and brain perfusion as every 10 cm of elevation in vertical height from the site of measurement to the brain will result in a decrease of CPP of 8 mm Hg.⁴ Despite these claims, blood pressure management with non-invasive blood pressure cuffs and CNAP in the BCP have no definitive standards of MAP regulation.

Currently, there are two ideologies surrounding blood pressure management for BCP procedures.⁴ In the siphon concept, gravitational pull acts simultaneously on both the arterial and venous systems resulting in equal decreases in MAP without significant decreases in CPP.⁴ In contrast, the waterfall concept proposes that veins collapse and prevent the siphon effect from occurring, as gravitational pull causes blood to fall on the venous side making the heart compensate by pumping blood back towards the brain.⁴ Despite either ideology on CPP, cerebral desaturation appears to be a frequent occurrence even when blood pressures are maintained within 20% of baseline levels.⁴ Therefore, blood pressures in the BCP should be adjusted to account for hydrostatic pressure gradients and deliberate hypotensive techniques avoided.^{4,5} Although blood pressure monitoring does not entirely protect patients from neurological insults, it should help guide anesthesia management; but, alternative cerebral oxygenation techniques should be implemented to detect periods of cerebral desaturation that are clinically imperceptible.

While blood pressure management provides guidance for maintaining adequate CPP, it is inadequate to definitively assess periods of cerebral hypoperfusion and cerebral desaturation.¹ When blood pressure monitoring techniques are used in conjunction with a multi-system monitoring approach optimal monitoring of cerebral oxygenation maybe achieved.¹ Cerebral oximetry is a non-invasive near infrared spectroscopy monitoring technique that can be utilized to measure cerebral oxygenation and hypoxia through quantification of total hemoglobin concentration levels in bodily tissues.^{1,2} A decrease in CPP can result in decreases in total hemoglobin levels in the tissue vasculature and indicate episodes of cerebral oxygen desaturation.^{1,2,3} Although the cerebral oximetry can be a pivotal monitoring technique in determining the significance of cerebral hypoxia, it is not without limitations.² Cerebral oximetry can inaccurately interpret cerebral oxygenation due to the inability to differentiate between arterial and venous hemoglobin saturations and regional versus global cerebral hypoxia.² Due to these limitations, cerebral oximetry cannot stand alone as a singular monitor to prevent against neurological insults. Another monitor that may help determine if CPP are adequate is the bispectral index (BIS).

Although the BIS is designed to measure the degree of consciousness during general anesthesia, it may prove viable for detecting periods of cerebral ischemia.³ The BIS can be an effective monitoring technique for detecting neurological decline, as acute slowing of the electroencephalogram (EEG) is an early indication of cerebral hypoperfusion.³ As CPP declines,

EEG activity will decrease and result in a lowering of the BIS; therefore, unintentional decreases in the BIS maybe indicative of cerebral hypoxia.³ However, BIS monitoring maybe limited in anesthetic cases using nitrous oxide, ketamine, neuromuscular blockers, and volatile anesthetics due pharmaceutical manipulation of EEG activity revealing inaccurate results.⁶

The BCP presents unique challenges for anesthesia professionals that can cause detrimental neurological impairments due to decreases in MAP and CPP.^{2,3} Anesthesia professionals must be vigilant in their anesthesia management of the BCP as complications can arise due to inability to accurately monitor cerebral oxygenation. It is imperative that the sole reliance on one singular monitoring technique does not dictate anesthesia practice as the consequences may prove disastrous.¹⁻⁴ Anesthesia management for the BCP should encompass a multitude of cerebral perfusion monitoring techniques to ensure adequate cerebral oxygenation.¹⁻⁴

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Sickle Cell Disease and Ketamine Use in Endoscopy

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Keywords: ketamine, propofol, endoscopy, sickle cell disease, sickle cell crisis, opioid tolerance

Sickle cell disease (SCD) is characterized as a group of disorders that cause red blood cells to become misshapen and break down, leading to anemia and other complications such as painful

vaso-occlusive crisis, also known as sickle cell crisis. The most common treatment for vaso-occlusive crisis for SCD includes opioids combined with nonsteroidal anti-inflammatory agents (NSAIDs).¹ Tolerance and opioid-induced hyperalgesia can occur despite escalating doses of opioids. Patients tolerant to opioids require more propofol during endoscopy procedures which can cause cardiorespiratory instability and depression.²⁻⁴ This case report is an example of multimodal therapy used in an endoscopy procedure.

Case Report

A 48-year-old, 73.3 kg, 181 cm male with a body mass index (BMI) of 22.4 kg/m² presented for an esophagogastroduodenoscopy (EGD) due to dysphagia. His past medical history included coronary artery disease (CAD), sickle cell anemia with admission to the hospital due to sickle cell crisis, transfusion hemosiderosis, liver cirrhosis, sleep apnea, and chronic pain syndrome. The surgical history included a ventral hernia repair and surgical drainage of a dental abscess. The patient had no known drug allergies. The patient's home medications consisted of amlodipine, deferoxamine, flunisolide, folic acid, gabapentin, hydrocodone-acetaminophen, hydroxyurea, lactulose, polyethylene glycol, naproxen, oxycodone, and promethazine.

An electrocardiogram showed sinus rhythm with a first degree atrioventricular block with premature atrial contractions (PAC's) with a rate of 93/min, T-wave abnormality with anterior ischemia, and a prolonged QT interval. The echocardiogram identified an ejection fraction of 60%, grade I diastolic dysfunction, and mild/moderate increased right ventricular systolic pressure. The chest x-ray showed cardiac enlargement with vascular congestion. All laboratory values were within normal limits with the exception of the following: hemoglobin 8.6 g/dL, hematocrit 26.2%, alkaline phosphatase (ALP) 163 units/L, aspartate aminotransferase (AST) of 89 units/L, alanine aminotransferase (ALT) 60 units/L, and lactate dehydrogenase (LDH) 567 units/L.

The anesthetic plan consisted of a general anesthetic with a propofol infusion as well as lidocaine, ketamine, and midazolam preoperatively. Preoperative vital signs included heart rate of 93/min, blood pressure 155/88 mm Hg, respiratory rate 20/min, SpO₂% 100% on 4 L/min via nasal cannula, and temperature of 36.5 °C. The physical examination revealed a Mallampati class III airway with several missing teeth and nearly full neck range of motion.

Noninvasive monitors were placed on the patient and O₂ 4 L/min was continued via nasal cannula. End-tidal CO₂ was measured through the nasal cannula. Midazolam 2 mg, ketamine 30 mg, lidocaine 50 mg, and propofol 30 mg was administered, and the procedure began once the patient appeared relaxed and no longer responded to verbal command. A propofol infusion of 100 to 200 mcg/kg/min was titrated to maintain patient comfort. Vital signs remained within 20% of baseline.

The total surgical procedure time was 17 minutes with a total of 100 mg of propofol given. Upon transport to the recovery area, the patient was alert, oriented, and stable. Fluid administration included normal saline 200 mL with no blood loss. Vital signs remained within normal limits. The patient reported no nausea, vomiting, or postoperative pain prior to being transported back to his inpatient room.

Discussion

Inherited at birth, sickle cell disease can lead to painful vaso-occlusive crisis. Sickle cell disease can be described as an acute episode of pain resulting from tissue ischemia due to occlusion of the microcirculation by sickled erythrocytes.¹ These episodes of pain, which can start at a very young age, are commonly treated with opioids. Tolerance to opioids occurs due to chronic exposure and pain can only be overcome with increasing doses of opioids.¹ Propofol is commonly used as a unimodal medication in endoscopy due to its early-onset effect and short half-life. Although propofol has many benefits, when given in larger doses it can lead to hypotension, bradycardia, and respiratory depression.⁵ Patients that have opioid tolerance require increased doses of propofol before becoming unconscious in preparation for an endoscopy procedure. Ketamine, used in combination with propofol in this patient population, hastens necessary sedation levels while decreasing cardiopulmonary side effects.^{3,5}

Ketamine has many uses within anesthesia and is well known as an adjunct to opioid-induced analgesia.⁶ Ketamine inhibits N-methyl-D-aspartate (NMDA) receptors which produce antinociceptive action and activate the descending inhibitory monoaminergic pain pathway. Ketamine also stimulates the sympathetic nervous system and increases blood pressure as well as heart rate which contribute to its cardio stimulant properties.^{2,3,6} The use of ketamine and propofol in combination, especially in the endoscopy setting, attenuates the cardiovascular response while providing analgesic properties that remove the need for respiratory depressing opioids.^{3,7} Patients who suffer from chronic pain, especially those with high levels of opioid use and tolerance such as patients with sickle cell disease, benefit from the use of low dose ketamine.⁸

The benefits of using low dose ketamine in the endoscopy setting extend beyond its cardio stimulant properties. The medication combinations have an additive hypnotic and anesthetic effect leading to reduced total propofol dosing requirements.⁷ Low dose ketamine, 0.15 mg/kg, along with propofol for an EGD has also been shown to decrease the incidence of the gag reflex.⁷ Ketamine, in subanesthetic doses, has been shown in several studies to impair memory and recall, while propofol alone has a relatively high incidence of recall. The use of ketamine with propofol compared to propofol alone leads to a reduced anesthetic recall by inhibiting the long-term potentiation of synaptic transmission via the NMDA receptor block.⁶

The patient in this case report received a combination of ketamine and propofol for intraoperative sedation. Although the patient had sickle cell disease, which led to his requirement for large doses of opioids, he required minimal amounts of propofol. In theory, had the ketamine not been used, a much larger dose of propofol would have been needed in order to reach the level of sedation necessary for the EGD. Increasing doses of propofol may lead to risks of hypotension, respiratory depression and possible airway intervention. The literature review showed that combining ketamine with propofol in the endoscopy setting, especially for an opioid tolerant patient, allowed for decreased complications in comparison to propofol alone. The EGD was uneventful, the patient's vital signs remained stable, and the patient protected his own airway during the perioperative period. The patient was alert and oriented upon arrival to the recovery room and reported no pain. With the findings of this case report and the evidence

found in the literature review, the anesthetic management for this patient was appropriate and required no change.

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Anesthetic Considerations for Craniotomy for Chiari Malformation

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Keywords: Craniotomy, cervical decompression, cerebellum, cerebral spinal fluid, Chiari Malformation

Chiari malformation (CM) is a structural defect in the posterior part of the brain, which leads to overcrowding of the cerebellum and brainstem. The posterior fossa of the brain is responsible for respiratory control as well as cardiovascular systems, thus, promoting possible challenges for the anesthesia practitioner.¹ Some types of CM require a suboccipital craniotomy, cervical

laminectomy, and possibly a shunt for patients with severe hydrocephalus. The incidence of CM is approximately 1 in every 1,000 births, with a higher prevalence in women.² Anesthetists need to be aware of the physiologic alterations which accompany CM, in order to anticipate perioperative needs.

Case Report

A 19-year-old, 162 cm, 74.2 kg Caucasian female presented for a suboccipital craniotomy with C₁-C₂ laminectomy and decompression for Chiari Malformation. This was found due to complaints of chronic migraine headaches over the last couple years ultimately leading to a diagnosis via Magnetic Resonance Imaging. The patient's past medical history included chronic migraines and history of tobacco abuse of one half a pack to a pack of cigarettes per day for approximately one year. The patient's past surgical history included a Cholecystectomy and wisdom teeth extraction. After a preoperative neurological exam, cranial nerves were all found to be intact.

A preoperative airway exam revealed a Mallampati 2 classification, thyromental distance of more than three fingerbreadths, full range of motion of the neck, and adequate mouth opening with incisor distance of greater than 3 cm. 18-gauge peripheral intravenous lines were placed in bilateral hands. The patient was premedicated with dexamethasone 8 mg, famotidine 20 mg, fentanyl 50 mcg, midazolam 2 mg, ondansetron 4 mg, and a scopolamine patch due to a history of postoperative nausea and vomiting. The patient was transferred to the operating room, placed on standard monitors, and initial vital signs were obtained prior to induction of anesthesia. The patient was pre-oxygenated with O₂ 10 L/min via mask for approximately 3 minutes. Intravenous induction of general anesthetic included lidocaine 80 mg, fentanyl 100 mcg, propofol 150 mg, and rocuronium 40 mg. To minimize manipulation of the neck, a Glidescope (Verathon Inc, Bothell, WA) was utilized, and a grade 1 view of the glottis was achieved. Successful intubation of the trachea with a size 7.0 cm endotracheal tube (ETT) was performed. An esophageal temperature probe was placed after the ETT position was confirmed and secured.

After induction, the depth of anesthesia was increased using 50 mg of propofol prior to placement of the Mayfield frame and tongs by the neurosurgeon for stabilization of the head. This was necessary in order to prevent the patient from coughing/bucking, with the potential to cause increased intracranial pressure and ischemia in the brain. The patient was placed in the prone position on the operating table. Maintenance of anesthesia was obtained with an expired concentration of 2.1% of sevoflurane in a mixture of O₂ 1 L/min and air 1 L/min, as well as periodic doses of 50 mcg of fentanyl. The patient received a total of 800 mL of normal saline.

After the patient was repositioned to the supine position on the transfer cart, the surgeon removed the Mayfield frame and pins, and a dressing was applied. Neuromuscular blockade was antagonized with neostigmine 3 mg and glycopyrrolate 0.4 mg intravenously after a return of 4 out of 4 twitches. The patient maintained adequate tidal volumes of 350-400 mL and 14 breaths/min. The anesthetic gas was turned off and oxygen flows were increased to 10 L/min. After the patient appropriately followed commands, the trachea was extubated and the patient was transferred to the recovery room on a simple face mask with O₂ 8 L/min, and maintained stable vital signs.

Discussion

The four types of Chiari Malformations are classified as type I, II, III, and IV. Type I (what this particular patient had) involves herniation of the cerebellar tonsils through the foramen magnum.³ Type II is the herniation of not only the cerebellar tonsils, but also the vermis, fourth ventricle, pons and medulla move downward.³ Type III is a defect in the back of the head or neck in which the brainstem and/or cerebellum is herniated.³ The final classification, Type IV, is very rare because these patients usually do not survive past infancy.³ As a result of the brainstem and cerebellum being pushed downward into the foramen magnum, the pressure exerted on them causes these structures to malfunction and potentially block the flow of cerebrospinal fluid (CSF) in and out of the brain. People with CMs typically require surgery to make room in the posterior part of the skull for the brainstem and cerebellum and to allow for better flow of CSF in and out of the brain. A craniectomy involves removing part of the base of the skull to enlarge the foramen magnum to allow for better flow of CSF. Laminectomy and decompression involves the removal of the arch of one or more vertebrae (in this particular case it was C₁–C₂) and to open up the posterior fossa space to decrease crowding.

Patients with CM may remain asymptomatic for years. Anesthesia practitioners must be educated on some of the characteristics to look for during the preoperative evaluation. Signs of brainstem dysfunction can include altered respirations and possible sleep apnea, whereas signs of cranial nerve dysfunction include dysphagia, absent gag reflex, and changes in phonation.¹ Signs and symptoms of neurological dysfunction are very important to note prior to surgery so as to adequately assess for any changes in neurological function after surgery. This patient denied having sleep apnea, and regular, nonlabored respirations were observed during the preoperative evaluation.

Anesthetic goals for patients undergoing craniotomy and/or laminectomy and decompression of the cervical spine include maintenance of cerebral perfusion pressure, avoidance of coughing or straining, and hemodynamic stability.¹ Brainstem manipulation can cause blood pressure and heart rate instability. The goal was to maintain the blood pressure within 20% of the patient's baseline reading. It is highly important to avoid any hypertensive spikes during the more stimulating parts of the case (tracheal intubation, Mayfield pin placement, and opening of the Dura), as elevated blood pressure could lead to increased intracranial pressure and ischemia. The use of an arterial line for continuous blood pressure monitoring is dependent upon practitioner preference. Exact mechanisms utilized throughout this specific case were discussed earlier in the case report.

Cardiac arrhythmias are also common during the manipulation of the brainstem, with bradycardia being the most common.¹ The arrhythmia typically resolves on its own once the surgeon is done with manipulation, however, the patient can be treated with medications such as glycopyrrolate or atropine. The patient did not have any arrhythmias throughout this case. Other possible complications that the practitioner should be ready for include the presence of a venous air embolism (VAE). A sudden, abrupt decrease in end-tidal CO₂ along with a decrease in blood pressure are signs of possible VAE, and a precordial doppler and trans-esophageal echocardiogram (TEE) are sensitive monitors for the detection of a VAE.⁵ Standard monitors (End-Tidal CO₂ and blood pressure) were utilized, and a TEE was readily available if needed.

Venous air embolism can occur at any time when the head is five degrees above heart level. The anesthesia practitioner must be cognizant of these signs and ready to treat by notifying the surgeon immediately, turning on 100% oxygen (removing nitrous oxide), fluid resuscitation, lowering the head to heart level when possible, compressing bilateral jugulars, and possible aspiration of air embolism (requires a central venous catheter).³ Other risks for this procedure include wound infection, cerebrospinal fluid leak, and risk of injury to cranial nerves, brainstem somatosensory pathways, and spinal cord.⁴

Overall, this case went as expected with minimal blood loss and continued hemodynamic stability, and the patient did not show any signs of neurologic deficit after the surgery. Despite the patient's stability however, placement of an arterial line would be beneficial in future cases for constant assessment of arterial pressure in order to manage any acute fluctuations in pressure. Although this case was uneventful, anesthesia practitioners must remain attentive to the physiological characteristics found in patients with CM in order to prepare for specific perioperative needs.

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Hemodynamic Management during Carotid Endarterectomy

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Keywords: carotid endarterectomy, anesthetic management, hemodynamic management, cerebral perfusion, cerebral vascular disease, hyperperfusion syndrome

In the United States, cerebral vascular disease is among the leading causes of morbidity and mortality. Carotid artery atherosclerotic disease accounts for nearly one third of ischemic

strokes.¹ Carotid endarterectomy (CEA) is commonly employed to attenuate injury from progression of carotid atherosclerosis. Given the nature of surgical intervention and comorbidities characteristic of this patient population, perioperative stroke and myocardial infarction are serious risks.²⁻⁴ Thus, achievement of hemodynamic stability during CEA is a challenging and necessary goal for anesthesia practitioners. A brief case report and review of literature demonstrating hemodynamic challenges anesthesia practitioners may confront while conducting anesthesia for CEA follows.

Case Report

A 76-year-old, 77 kg, 178 cm Caucasian male presented for elective right CEA. History of present illness included right carotid artery stenosis and cerebral vascular accident (CVA) two weeks prior with residual left hemiparesis and mild expressive aphasia. Past medical history included hypertension, coronary artery disease (CAD), myocardial infarction (MI), atrial fibrillation, and polymyalgia rheumatica. Past surgical history consisted of percutaneous coronary intervention with stenting and placement of pacemaker/implantable cardioverter-defibrillator (ICD). Current medications included aspirin, warfarin, lisinopril, spironolactone, prednisone, and nitroglycerine. An electrocardiogram (ECG) demonstrated underlying atrial fibrillation with ventricular pacing at 86/min. Echocardiography revealed reduced left ventricular ejection fraction of 40-45% and moderate concentric left ventricular hypertrophy. Bilateral carotid duplex ultrasonography demonstrated greater than 90% stenosis of right internal carotid artery.

Upon arrival to operating room, the patient was positioned supine, standard monitors were applied, and a magnet was placed over pacemaker/ICD. Pre-induction vital signs included heart rate (HR) 82/min ventricular paced, blood pressure (BP) 158/66 mm Hg, and mean arterial pressure (MAP) 97 mm Hg. The patient was pre-oxygenated with oxygen 8 L/min and induced with fentanyl 100 mcg, lidocaine 30 mg, propofol 150 mg in divided doses, rocuronium 40 mg, and phenylephrine 100 mcg. The trachea was intubated with 7.5 mm endotracheal tube (ETT) devoid of complications. Hydrocortisone 100 mg was administered and general anesthesia was maintained with Sevoflurane 1.5-2% inspired concentration in O₂ 1.25 L/min and air 0.75 L/min. A phenylephrine infusion of 60-100 mcg/min was titrated to maintain MAP 90-110 mm Hg.

Following incision, HR was 108/min, BP was 100/60 mm Hg, and the ECG demonstrated bigeminy. The pacemaker initiated the normal ventricular paced contraction, which alternated with a premature ventricular contraction (PVC). During the PVC, the pacemaker fired at varying times throughout the cardiac cycle. Lidocaine 100 mg was administered and normal ventricular pacing resumed at 70-80/min throughout surgery. Prior to emergence, neostigmine 4 mg, glycopyrrolate 0.6 mg, ondansetron 4 mg, and oxygen 10 L/min were administered. Spontaneous respirations were achieved and phenylephrine infusion and sevoflurane were titrated off. During emergence, vital signs were HR 78/min, BP 190/90 mm Hg, and MAP 123 mm Hg. Nicardipine 300 mcg was administered, followed by infusion at 5 mg/hr. Patient was extubated and neurologic assessment demonstrated no change from baseline. Nicardipine infusion was discontinued, and patient was transferred to the recovery room after obtaining a BP of 120/62 mm Hg and MAP 81 mm Hg.

Upon arrival to post anesthesia care unit (PACU), vital signs were HR 71/min, BP 125/51 mm Hg, and MAP 76 mm Hg. The patient remained hemodynamically and neurologically stable throughout stay in PACU.

Discussion

Research suggests most neurologic deficits during CEA have etiology in thromboembolic events from carotid cross-clamp and shunt placement.¹⁻⁵ Thus, the goal of maintaining cerebral perfusion and oxygenation is essential during carotid cross-clamp when cerebral perfusion becomes dependent on collateral blood flow.¹⁻³ This is facilitated by maintaining MAP at or in excess of 20% preoperative level.¹⁻⁴ Although hypothetically beneficial, a MAP in excess of 20% preoperative level may cause myocardial strain and possible ischemia/infarction.^{1,3} Similarly, hypertension following CEA may precipitate poor outcomes such as cerebral hyperperfusion syndrome (CHS) and intracerebral hemorrhage (ICH).²⁻⁴ Therefore, the ultimate anesthetic goal for the patient undergoing CEA is perioperative and postoperative hemodynamic control.

Maintaining such perioperative hemodynamic control begins with preoperative optimization, assessment, and planning. The patient was identified as high-risk for intraoperative hemodynamic instability given history of poorly controlled HTN and two-week interval between CVA and CEA.³⁻⁴ Uncontrolled HTN increased the patient's perioperative risk for CVA, while history of CAD and MI increased his risk for perioperative MI.² Such risk necessitated testing and optimization, which were conducted prior to surgery. A preoperative neurologic assessment was conducted to facilitate early postoperative comparison to identify adverse outcome. Based upon surgeon preference, patient considerations, and recent evidence demonstrating no difference in outcomes (CVA, MI, and death) when comparing CEA under general anesthesia to CEA under local anesthesia,⁵ a general anesthetic was selected. Preoperative assessment, optimization, and selection of anesthetic facilitated planning for interventions expected to facilitate hemodynamic control and thus, cerebral/myocardial perfusion and oxygenation.

Intraoperative cerebral and myocardial perfusion and oxygenation were ensured through employing numerous interventions aimed at preventing/treating hypotension. One such intervention was coupling propofol with phenylephrine for induction of anesthesia. Current literature recommends a smooth intravenous induction with attention to maintenance of preoperative MAP.² Induction with etomidate may have been more ideal given the patient's diminished cardiac reserve. However, the patient was suspected of having iatrogenic adrenal insufficiency (AI) given their long-term glucocorticoid therapy for polymyalgia rheumatica. Research has demonstrated impairment in adrenal gland functioning following a single induction dose of etomidate, yet such impairment has not been linked to increased mortality when compared with other induction agents.⁶ In concern for further AI, etomidate was not utilized, although it may have been more efficacious for maintaining cerebral and myocardial perfusion.

Another intraoperative intervention employed to attenuate the hypoperfusion associated with bigeminy was the administration of lidocaine 100 mg. During bigeminy, the pacemaker was firing randomly throughout the cardiac cycle during the PVCs. This was due to inactivation of the ICD and pacemaker being converted to asynchronous mode following magnet application. Given concern for the pacemaker firing on a T-wave and potential for ventricular fibrillation,

immediate treatment was implemented. Although, lidocaine administration was successful in terminating bigeminy, current treatment guidelines recommend beta-adrenergic antagonism (esmolol) as first-line therapy and reserve lidocaine for an alternative if beta blockade is unsuccessful.⁷ Although unique to this case study, any patient specific intervention required to maintain cerebral and/or myocardial perfusion and oxygenation must be employed during CEA to optimize patient outcomes.

With the goal of preserving cerebral perfusion and oxygenation, vasopressors are frequently administered to achieve a MAP within excess 20% baseline value.¹⁻⁴ Phenylephrine infusion 60-100 mcg/min was administered throughout the case to maintain cerebral and coronary perfusion. Although there is a lack of evidence supporting choice of vasopressor and outcome during CEA, it seems logical to select a vasopressor based upon its pharmacologic profile. Phenylephrine was selected given its quick onset, short duration of action,⁷ and patient's cardiac history. Reflex bradycardia was deemed beneficial, given that bradycardia allows more time for ventricular filling and coronary perfusion. Significant bradycardia with hemodynamic compromise was not a concern because it would be attenuated with ventricular pacing. Phenylephrine administration to achieve mild HTN during CEA was essential for maintenance of cerebral and myocardial perfusion and oxygenation.

Although mild HTN is desirable during CEA, research suggests that normotension is desirable during emergence following correction of carotid atherosclerosis to prevent CHS and ICH.¹⁻⁴ During emergence, the patient became hypertensive. Hypertension was quickly attenuated with bolus administration of nicardipine followed by infusion. Again, there is limited data comparing efficacy of agents in the prevention and treatment of HTN in patients enduring CEA.³ Therefore, selection should be based upon pharmacologic knowledge of such agents. Nicardipine was chosen given its quick onset, short duration of action, and limited adverse effect profile.⁷ There is concern that vasodilators may cause detrimental cerebral vasodilation leading to a steal phenomenon following correction of carotid atherosclerosis, particularly following high grade stenosis.³ However, it is believed that such concern is overshadowed by the benefit of obtaining a normotensive environment.³ Fortunately, the patient sustained a normotensive state following emergence and did not require vasodilators during the postoperative period. The aggregate of interventions aimed at achieving hemodynamic stability previously discussed may have contributed to this patient's positive outcome.

Numerous interventions, both patient specific and common to CEA, must be employed in an attempt to obtain hemodynamic control of the patient undergoing CEA. Such interventions have common goals of achieving cerebral and myocardial perfusion and oxygenation, thus preventing CVA and MI. More research is needed to provide stronger evidence to guide practice and improve the management of patients undergoing CEA.

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The Challenges of Pericardial Disease and Anesthesia

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Keywords: anesthesia, pericardiectomy, patent foramen ovale, pericardial disease, hemodynamics

Constrictive pericarditis (CP) is a result of chronic inflammation of the pericardium, a membrane that encases the heart.¹ This inflammation leads to a lack of elasticity and ultimately ventricular dysfunction.¹ Pericardiectomy is the treatment of choice for patients with CP, with 80% relief of heart failure symptoms.¹ However, administering anesthesia to a patient with pericardial disease pose great challenges for the anesthetist. CP is marked by a fixed cardiac output state which leads to overall physiological impairment.² This case report follows the management and evidence-based anesthesia practice of a patient with CP undergoing an off-pump pericardiectomy.

Case Report

A 74-year-old, 96 kg, 177 cm male with a diagnosis of CP was to undergo a pericardiectomy. The patient's medical history included: coronary artery disease, hypertension, chronic obstructive pulmonary disease, asthma, pericardial effusion, and a patent foramen ovale (PFO) with right to left shunt diagnosed through a transthoracic echocardiogram (TEE) one year prior. Blood pressure was 118/79 mm Hg, heart rate 87/min, respiratory rate 22/min, temperature 35.9°C, and SpO₂ 92% on non-rebreather mask at 15 L/min. A 2-dimensional echocardiogram revealed an ejection fraction of 55%. Laboratory studies were within normal range. Blood products were typed and cross matched for possible transfusion. An arterial line was placed in the left radial artery while in the preoperative holding area.

Prior to induction, the patient was transferred to the operating room table and quickly desaturated with movement to a SpO₂ of 76%. The patient was then preoxygenated with a facemask in the sitting position with O₂, 10 L/min. The patient's SpO₂ improved to 79%. Anesthesia was then induced with midazolam 3 mg, lidocaine 60 mg, etomidate 15 mg, and rocuronium 70 mg. The patient was ventilated by mask prior to intubation of the trachea. There was little improvement in the oxygen saturation when compared to preinduction SpO₂. A Glidescope (Verathon Inc., Bothell, WA) was used to intubate the trachea after 2 minutes of mask ventilation. The patient was then ventilated with O₂ 10 L/min; SpO₂ increased to 85%.

General anesthesia was maintained with isoflurane 0.5% inspired concentration with O₂ 2 L/min and the bispectral index was maintained between 40 and 50 to monitor for intraoperative awareness. Following induction of anesthesia, the blood pressure decreased to 81/50 mm Hg. Phenylephrine 80 mcg IV was given with minimal improvement to blood pressure. Following intubation, a central venous catheter was placed into the right internal jugular vein. Immediately after, while placing the pulmonary artery (PA) catheter, SpO₂ decreased to 74% and blood pressure decreased to 77/43 mm Hg. At this time, epinephrine 50 mcg was given with slight improvement in blood pressure. The surgeon was notified and the case was started immediately. The patient received sufentanil 50 mcg and the chest was opened through a median sternotomy and the pericardium was freed from mediastinal tissue.

Throughout the case the mean arterial pressure (MAP) ranged from 50 to 55 mm Hg although central venous pressure (CVP) ranged from 14 to 18 mm Hg. The PA pressure was 46/39 mm Hg at the start of the case. Volume expansion was used to treat hypotension with little response. The patient received 5% albumin 500 mL, and normal saline 1 L. Epinephrine boluses of 50 mcg were given twice throughout the case. An epinephrine infusion at 1 mcg/min and a norepinephrine infusion of 4 mcg/min were started and titrated in order to maintain a MAP >60 mm Hg. A vasopressin infusion of 4 units/hr was also initiated. Midway through the case milrinone 0.25 mcg/kg/min to maintain a cardiac output (CO) of 2 L/min. In addition, calcium chloride 200mg was administered and CO increased to 4 L/min. At the end of the case, CVP was 17 mm Hg and PA pressures were 32/17 mm Hg. Total surgical time was 2 hours and 3 minutes. The patient's blood pressure and SpO₂ remained unstable throughout the case and the patient was transferred to the intensive care unit intubated, with all vasopressors infusing.

Discussion

Constrictive pericarditis causes constricting of the pericardium during diastole.³ This dysfunction results in a fixed stroke volume which makes cardiac output dependent on increases in heart rate.⁴ Pericardiectomy, the surgical stripping and removal of the adherent pericardium, with sternotomy is the recommended procedure for CP.⁵ Cardiopulmonary bypass may be used to facilitate pericardial stripping if hemorrhage becomes difficult to control.⁵ Patients with pericardial disease may experience hemodynamic instability under anesthesia due to the positive pressure ventilation precipitating cardiac tamponade.⁵ Anesthetic drugs and techniques which minimize changes to heart rate, venous return, myocardial contractility, and systemic vascular resistance should be selected.⁵

In order to form an anesthetic plan for a patient with CP, a number of clinical parameters must be integrated into the plan of care.⁴ During the preoperative period, a complete evaluation should be performed by the anesthesiologist, and prior to transport to the operating room, the evaluation of possible cardiac tamponade should be explored through echocardiogram. In this case, the patient was dyspneic in the preoperative holding room despite the administration of 100% FiO₂ via a non-rebreather facemask. There were no pulmonary function tests recorded, however a computed tomography of the chest was performed to rule out possible pulmonary embolism. All other signs and symptoms to suggest tamponade were ruled out. An invasive arterial blood pressure monitor should be placed to monitor hemodynamic changes.⁵ Central venous pressure monitoring is essential during a pericardiectomy to monitor right ventricular filling pressures and also to observe the decrease in venous pressure following the removal of both parietal pericardium and visceral peel.²

During induction, cross-matched blood and adequate fluids for resuscitation should be available. Vasopressors such as phenylephrine, epinephrine, and norepinephrine should be prepared and ready in the room.⁴ The hemodynamic goals for patients under general anesthesia should be: maintain preload, afterload, and contractility.⁴ Heart rate should also be maintained in a sinus rhythm in order to better facilitate ventricular filling in patients with diastolic dysfunction.⁴ The use of a sevoflurane inhalation induction is recommended because it minimizes coughing while maintaining spontaneous ventilation.⁴ If a patient is not a candidate for inhalation induction, an intravenous induction can be performed.⁴ An anesthetic agent that does not decrease venous return or depress the myocardium should be considered.² Hypotension from vasodilation should be treated with a continuous vasopressor infusion, and if hemodynamics deteriorate during induction, the surgical procedure should be facilitated immediately following intubation of the trachea.⁴ Nitrous oxide should be avoided during the case due to potential breach of the pleural space and hypoxemia.⁴ Muscle relaxation can be used during the case if the patient is tolerating positive pressure ventilation and short or intermediate-acting muscle relaxants are recommended.⁴

Perioperative arrhythmias are common in patients with pericardial disease due to the alterations in the autonomic nervous system during anesthesia and in surgery.² In order to minimize arrhythmias intraoperatively, it is imperative to provide a balanced anesthetic. One example would be the use of lidocaine prior to induction to attenuate the hemodynamic response to laryngoscopy and intubation.² In this case, lidocaine 60 mg was given prior to tracheal intubation. Tachycardia during induction can lead to increased oxygen demand, along with a reduction in blood supply that decreases coronary blood flow.² The continuous use of vasopressors and inotropes may be required to maintain hemodynamic stability during the case.⁴ However, the use of vasopressors should be weaned as tolerated, as excessive vasoconstriction can restrict CO.⁴

Positive pressure ventilation should be delivered with a minimal inspiration pressure required to provide adequate minute ventilation.⁶ Ventilation settings for patients with PFO are critical in the management of care. Due to the presence of a PFO, no positive end expiratory pressure (PEEP) was used on the ventilator and smaller tidal volumes were delivered. Shunt fraction was monitored throughout the case by a TEE. PEEP application in patients with PFO will increase the right-to-left shunt due to the rise in right ventricular afterload and right atrial pressure.⁶ This

increase in shunting is known to cause systemic hypoxemia. Along with hypoxemia, positive pressure delivered from the ventilator decreases venous return.⁴ This, in combination with vasodilatory effects of anesthetic agents, can lead to hemodynamic deterioration.⁴

Postoperative ventilatory insufficiency will require continued mechanical ventilation.⁵ Although hemodynamic parameters are expected to improve after surgery, the result of decreased SpO₂ would not improve in this case until closure of the PFO. Due to the acute state of the constrictive pericarditis and hemodynamic instability during the case, the surgeons were unable to close the PFO during the procedure. Once weaned from vasopressors, the PFO was closed with an amplatzer septal occluder in the cardiac catheterization lab three days after the initial surgery. Combined pharmacologic management of a patient with CP is imperative to providing an adequate and safe anesthetic. The anesthesia professional should be vigilant to hemodynamic changes that occur during the case and current evidence-based anesthesia practice.

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Anesthesia for Abdominal Aortic Aneurysm Repair

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Key words: abdominal aortic aneurysm, anesthesia management, hemodynamics, risk factors

An abdominal aortic aneurysm (AAA) is the abnormal dilation of the aorta, usually below the level of the renal arteries.¹⁻³ The largest concern with an abdominal aortic aneurysm is the risk of rupture; there is a linear relationship between the risk of rupture and diameter of the aneurysm.² For known aneurysms, patients are usually carefully monitored and followed, and once the

diameter exceeds 5 cm surgical intervention is required.² Anesthesia for abdominal aortic aneurysm repair can be complex and requires the anesthetist to have a strong understanding of the disease pathology, surgical interventions, and post-operative complications.

Case Report

A 69-year-old, 86 kg, 193 cm male presented for repair of left external iliac artery aneurysm measuring 5cm. The patient also presented with an abdominal aortic aneurysm measuring 4.7 cm x 4.3 cm x 4.7 cm with a 60 % narrowing of the lumen. The patient's medical history consisted of hypertension, mild tricuspid regurgitation, peripheral vascular disease, pulmonary embolism, and deep vein thrombosis. The patient's surgical history included a femoral bypass, repair of a hip fracture and an incision and drainage of an abscess resulting from the femoral bypass. Patient medications consisted of aspirin 81 mg, atorvastatin 20 mg, nifedipine 60 mg, and rivaroxaban 20 mg. Preoperative blood pressure (BP), mean arterial pressure (MAP), and heart rate (HR) were 155/102 mm Hg, 129 mm Hg, and 77/min respectively.

A 12-lead electrocardiogram displayed normal sinus rhythm. A 2-dimensional echocardiogram revealed trace of mitral regurgitation with abnormal left ventricular diastolic filling, mild tricuspid regurgitation, and an estimated left ventricular ejection fraction of 55%. Preoperative blood work consisted of complete blood count, comprehensive metabolic panel, prothrombin and international normalized ratio; all results unremarkable. In preoperative holding, midazolam 2 mg was administered; another 2 mg was administered on the way to the operating room.

Mask ventilation with O₂ 10 L/min was performed for 5 minutes prior to laryngoscopy. Anesthesia was induced with propofol 130 mg, lidocaine 50 mg, rocuronium 60 mg, and fentanyl 200 mcg. After tracheal intubation, a right radial arterial line and a central venous line was inserted. Anesthesia was maintained with isoflurane 1.0-1.7% expired concentration in O₂ 1 L/min and air 1L/min. An additional 200 mcg of fentanyl was administered prior to incision.

After appropriate access was established, surgery was able to commence, starting with exposure of both right and left groins, proceeding to the abdominal incision. Initially 10,000 units of heparin was administered prior to aortic cross-clamping, followed by 4,000 units 20 minutes after clamps were on. The right common iliac artery and the aorta were clamped infrarenally. At this time mannitol 20% 12 grams was given.

Forty-five minutes after initial clamp placement, the right leg and aortic clamps were released. Hypotension following reperfusion ranged from 90/47 mm Hg to 100/54 mm Hg and was treated with 500 mg calcium chloride, 80 mcg boluses of phenylephrine, 500 mL of albumin 5%, and 25 mEq of 8% sodium bicarbonate. Blood pressure normalized and was maintained at 130/87 mm Hg to 135/91 mm Hg following administration of these medications.

Total surgical time was 4 hours with repair of abdominal aortic aneurysm achieved by placement of a 16-18 mm Gore-Tex vascular bifurcated graft. Intravenous fluid for the case totaled 3 L; blood loss was 2 L. Cell saver was utilized and the patient received 600 mL of blood from the cell saver. Total urine output was 270 mL and central venous pressure fluctuated from 6 to 10 mm Hg. Neuromuscular blockade was antagonized, anesthetic agents were titrated off, and

patient was spontaneously breathing as morphine 5 mg was titrated to respirations of 10-12 breaths per minute. The patient was extubated after an uneventful emergence and an additional 5 mg of morphine was administered once the patient was awake and in recovery.

Discussion

The goal of open AAA repair is to maintain hemodynamic stability and organ perfusion in the face of aortic clamping and unclamping. However, goals to aid in repair and stabilization of the aneurysm start well before surgical intervention takes place. First, assessment of AAA must occur. This includes determining size, location and stability of the aneurysm. Ultrasound, which is almost 100% reliable for detecting AAA; followed by a computed tomography angiography (CTA) to examine the size and involvement of surrounding structures is the current gold standard of AAA identification.⁷

After assessment of the aneurysm, lifestyle changes need to be made. The most importantly modifiable risk factor is smoking cessation. Smoking carries a large risk for development of AAA and thus prior to surgical repair, smoking should cease.⁵ In addition to modifiable risk factors current medication recommendations also exist. These include, statin therapy, beta-blocker therapy, and an aspirin regimen, which should all be continued until the day of surgery.^{4,5}

The patient in this case study followed these suggested guidelines preoperatively taking aspirin and atorvastatin the day of surgery. The goal of this specific treatment is to maintain myocardial function and decrease the risk of myocardial ischemia, as postoperative myocardial infarction risk is as high as 40%.⁶ Although beta-blocker therapy is recommended, this patient was prescribed a calcium-channel blocker despite current research which has demonstrated an increased incidence of perioperative mortality in patients taking calcium-channel blockers who present for AAA repair.⁶

Cross-clamp placement and reperfusion is the major concern during the intraoperative phase. It is critical that the anesthetist is knowledgeable of hemodynamic changes that exist during these time periods and understands how to treat these hemodynamic changes. When the aortic clamp is placed, the body compensates with an increase in mean arterial pressure and systemic vascular resistance.^{2,4} Suggestions for combating the hypertension that occurs include use of vasodilators, opioids, or increased depth of anesthetic.⁴ One suggestion for reducing blood pressure is adding positive end-expiratory pressure (PEEP) right before clamping and keeping that pressure throughout the case as it decreases venous return and can provide hypotension. PEEP should be taken off just prior to unclamping.² Unclamping of the aorta causes a release of lactic acid mediators that results in significant hypotension.² Understanding that hypotension will occur with unclamping is important, and one way the anesthetist can start to tackle this problem is administering crystalloid once the aorta is clamped. Clamp time provides an adequate timeframe in which crystalloid can help increase preload. Thus, when unclamping occurs, overall increase in fluid volume aids in combating hypotension.² As the clamp is coming off, it is important to be ready with vasoconstrictors and inotropic drugs which may need to be administered at this transient period as metabolites are released into circulation.⁴

Thermoregulation is impaired while under anesthesia, and hypothermia measuring less than 36°C results in lower cardiac output, increased risks of tachycardia and arrhythmias postoperatively.⁷ Therefore, all warming techniques must be utilized during AAA repair to maintain adequate perfusion.^{4,6} The use of humidified anesthetic gas, warm blankets, fluid warming devices, and forced air warmers have proven to be sufficient to maintain adequate body temperature.⁷ Hypothermia was avoided during this case study with the use of fluid warmers on two intravascular lines, humidification of volatile agent, as well as an upper body forced air warmer.

Significant volume loss occurs during AAA repair. Despite lengthy research, no studies have been able to determine a specific fluid replacement strategy or transfusion threshold.⁷ Suggestions however include preloading fluid replacement with crystalloids and maintaining a central venous pressure greater than 12 mm Hg.⁷ Multiple studies suggest maintaining a hematocrit of 30% and a hemoglobin of greater than 9 g/dL; maintaining these values may require the administration of packed blood cells along with continuous assessment and lab testing to determine the need for addition blood products (fresh frozen plasma, platelets, and cryoprecipitate).^{4,5} Although it has not been proven to decrease the risk of intraoperative transfusion, it is still suggested to incorporate cell-saver technology if available.

Pain control after repair of AAA is often difficult; patients report significant pain from the large incisions required for repair. Uncontrolled pain can result in poor respiratory effort leading to pneumonia, tachycardia and hypertension, which can be detrimental for the patient recovering from AAA repair. However, the use of an epidural catheter for analgesia during AAA is controversial. These patients are often on anticoagulants, which is often a contraindication for neuraxial anesthesia. Thus, some anesthesiologists are uncomfortable placing epidural catheters in this particular patient population.⁷ The patient within this case study decided against epidural placement and received 10 mg of morphine incrementally administered upon emergence and into the recovery room. However, follow-up on this patient the next day demonstrated significant difficulty in finding a pain regime that offered relief.

The typical patient presenting for AAA repair suffers from significant comorbidities, which make negating the hemodynamic changes that can occur during AAA repair important. Understanding the physiological changes that can occur with clamping and unclamping of the aorta is the most important factor in providing care throughout AAA repair. From this patient's preoperative laboratory testing, aneurysm assessment, and medication regime, to the anesthesiologist's intraoperative interventions, the care provided throughout this case was based on current evidence-based research. This case solidifies that existing guidelines regarding AAA repair aid in positive patient outcomes.

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Pneumothorax in Robotic Total Laparoscopic Hysterectomy

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Keywords: laparoscopic, hysterectomy, robotic, insufflation, pneumothorax

Intra-operative pneumothorax is a risk associated with laparoscopic procedures. Although extremely rare, this can be life threatening if not early identified and treated promptly. Signs and symptoms that prompt the anesthesia practitioner to suspect pneumothorax include sudden decline in SpO₂, increase in end tidal CO₂, increased airway pressures, and hemodynamic changes.^{1,2} Additionally, more than 50% of complications occur during entry into the abdomen and trocar placement, so anesthesia practitioners should remain extremely diligent during this period.⁵

Case Report

A 58-year-old, 77 kg, 166 cm Caucasian female presented for an elective robotic laparoscopic total hysterectomy and bilateral salpingo-oophorectomy secondary to abnormal post-menopausal bleeding. Her past medical history was significant for obstructive sleep apnea (OSA) and endometrial cancer. A preoperative assessment was completed with no significant findings including her OSA which she required no assistive devices. Midazolam 2 mg was administered just prior to departure to the operating room. In the operating room noninvasive monitors were applied and O₂ 6L/min was administered via face-mask. Intravenous (IV) induction was initiated with fentanyl 200 mcg, lidocaine 80 mg, and propofol 150 mg. Rocuronium 40 mg was administered after mask ventilation was confirmed successful. After three minutes of pre-oxygenation, the trachea was intubated. Endotracheal tube (ETT) placement was confirmed with sustained end tidal CO₂ and auscultation of bilateral breath sounds. General anesthesia was maintained with desflurane 7-8% end tidal concentration in a mixture of air 0.6 L/min and O₂ 0.6 L/min on pressure control ventilation. After successful trocar placement, the abdomen was

insufflated with CO₂ and pressures were maintained at approximately 15 cm H₂O. The patient was then placed into steep trendelenburg position.

Quickly following this position change, declining SpO₂ and increased end tidal CO₂ was observed on capnography. O₂ 10 L/min was initiated to provide 100% FiO₂ delivery to the patient. The patient's SpO₂ continued to decline. Manual ventilation was initiated to assess airway resistance, with minimal resistance was noted. Due to high suspicion of right main stem migration associated with position change, ETT depth was reassessed and confirmed by auscultation. At that time, breath sounds were noted to be diminished on the right side. Albuterol 8 puffs were administered through the ETT without improvement in SpO₂. Hemodynamic compromise was observed with a blood pressure of 73/49 mm Hg. Phenylephrine 200 mcg IV bolus was administered. Due to minimal hemodynamic improvement, an additional bolus was administered and IV infusion initiated at 0.5 mcg/kg/min.

Re-auscultation of breath sounds revealed coarse lungs sounds on the left and severely diminished breath sounds on the right. Due to severely declining hemodynamics, the patient was taken out of steep trendelenburg position and the supine position was achieved. Hemodynamic compromise continued, therefore surgery was ceased and the abdomen was no longer insufflated. Epinephrine 10 mcg IV bolus was administered and the phenylephrine infusion was increased to 0.8 mcg/kg/min. Additional anesthesia practitioners arrived to assist with the patient management. A fiberoptic bronchoscope was used to assess the position of the ETT and correct placement above the carina was confirmed. A chest x-ray was obtained to confirm the differential diagnosis of spontaneous pneumothorax. Once this diagnosis was confirmed, the in house trauma service was contacted for insertion of a chest tube to relieve the pneumothorax.

After chest tube placement, vital sign improvement was noted with immediate rise of SpO₂ to 100%, return of blood pressure to baseline and the phenylephrine drip was titrated off. The surgical team, trauma team, and anesthesia practitioners determined it safe to proceed with surgery based on return of hemodynamics to baseline and the need for surgery to remove cancerous tissue. The abdomen was re-insufflated and a modified steep trendelenburg position was achieved while maintaining patient safety and hemodynamics. Surgery was successfully completed without any additional unstable hemodynamics. Neuromuscular blockade was antagonized at the end of case with glycopyrrolate and neostigmine. Bilateral grip strength and adequate tidal volumes of 450-500 mL were observed prior to extubation. Oxygen 6 L/min was administered via simple face-mask and the patient was transported with SpO₂ monitoring to post anesthesia care unit.

Discussion

Laparoscopic approaches have become more common in gynecological surgeries. Literature has reported that this approach has benefits including less post-operative pain, faster recovery, quicker return of bowel function and a lower infection rate.³ Although laparoscopic techniques have many benefits to patients they are not without risk. General anesthesia requiring an ETT is the preferred technique for laparoscopic procedures to help decrease the risk of complications including aspiration, atelectasis, and CO₂ absorption. However, anesthesia practitioners face a variety of challenges including difficulty with ventilation and maintaining adequate blood

pressure due to the effects steep trendelenburg positioning may have on hemodynamic stability.⁴ Steep trendelenburg position is defined as greater than thirty to forty-five degrees of feet elevation above the head. One retrospective review of 968 laparoscopic cases revealed a pneumothorax or pneumomediastinum in 1.9 % of patients.⁵ Although rare, intra-operative pneumothorax can present in many different ways including hypotension, increased or decreased end tidal CO₂, hypoxia, increased peak airway pressures. Treatment interventions such as cessation of surgery and/or chest tube decompression may or may not be needed to resolve all laparoscopic induced pneumothoraxes. Invasive interventions, such as chest tube decompression, should take place when the patient becomes hemodynamically unstable.

Steep trendelenburg position can result in dramatic hemodynamic and respiratory instability. In this position increased venous return, decreased systemic vascular resistance (SVR) and increased cardiac output (CO) can be observed initially. Additionally, a decreased pulmonary compliance, vital capacity and increased peak airway pressures can ensue due to the cephalad movement of abdominal organs onto the diaphragm.⁴ Pressure control ventilation, compared to volume control ventilation, is a more effective mode of ventilation in laparoscopic procedures and was utilized in this case. One reason that pressure control ventilation is preferred is because it delivers a faster tidal volume earlier during inspiration. Also, pressure control ventilation has a high and decelerating inspiratory flow which helps compensate for the potential reduction in tidal volume caused by pressure limitation.⁶ Lastly, pressure control ventilation utilizes lower plateau pressures than volume control which helps reduce the risk of barotrauma while still allowing adequate gas exchange.⁶ After steep trendelenburg position is attained, the ETT placement should be reassessed to confirm a right main stem migration of the tube has not occurred. This is a common cause of declining SpO₂. In a right main stem ETT placement scenario, the anesthesia practitioner would auscultate decreased breath sounds on the left and increased lung sounds on the right, the exact opposite of our findings in our case. These paradoxical findings increased our suspicion of a pneumothorax. Additional techniques that can be instituted to help counter the hemodynamic effects of this position include keeping intra-abdominal pressure less than 15 cm H₂O and positive airway pressures less than 40 cm H₂O.⁴

Insufflation of the abdomen with CO₂ (pneumoperitoneum) drastically increases intra-abdominal pressures which would lead the gas to pass through anatomical, congenital or acquired diaphragmatic defects into the pericardial or pleural spaces. This would cause detrimental hemodynamic compromise to patients including pneumomediastinum, pneumopericardium and pneumothorax.⁴ Although rare, congenital defects of the diaphragm (ex: congenital patent diaphragmatic foramen) need to be considered as a possible cause of pneumothorax during laparoscopic procedures. Due to high intra-abdominal pressures these potential junction sites now become open and CO₂ is able to enter intrapleural spaces.^{1,2} During pneumoperitoneum, CO₂ can become trapped in the intrapleural space and cause rupture of the parietal pleura of the lung causing a pneumothorax.¹ Barotrauma is a cause secondary to increased airway pressures and decreased pulmonary compliance as a result of abdominal insufflation or patient positioning. Ultimately this can also result in a pneumothorax.⁵

Subtle changes in vital signs, such as SpO₂, end tidal CO₂, and blood pressure offer significant information for anesthesia practitioner to investigate his/her patient's risk of having developed a pneumothorax. Increased end tidal CO₂ and declining SpO₂ did prompt us to investigate our

patient for the cause of this sudden change in status. A greater than 20% decrease in blood pressure is significant and requires not only intervention, but determination of the source. Breath sounds auscultated on the left side immediately ruled out the differential diagnosis of right main stem ETT migration and was a key indicator for pneumothorax in this case.

Pneumothorax may resolve spontaneously if due to CO₂ insufflation. If barotrauma is identified as the cause then surgical decompression and chest tube placement is often needed.⁶ The cause of the pneumothorax in this case is unknown and required chest tube placement due to hemodynamic compromise, size of the pneumothorax, and for surgery to be safely continued.

Although rare, pneumothorax is a risk associated with laparoscopic procedures. A rise in end tidal CO₂, abnormal breath sounds, and declining SpO₂ and blood pressure can all be indicative of a spontaneous pneumothorax. The anesthesia practitioner must rule out other possible causes of declining SpO₂ such as right main stem tube ETT migration, which is common when positioning patients in steep trendelenburg. We recommend a thorough investigation be performed by the anesthesia practitioner of all possible causes for vital sign changes in any laparoscopic case. Ruling out other causes may lead to the rare, but potential diagnosis of pneumothorax.

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Anesthetic Approach for an Adult with Mastocytosis

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Keywords: orthopedic, mastocytosis, peripheral nerve block

Mastocytosis is characterized by abnormal mast cell proliferation.¹ The incidence of mastocytosis is rare with 1:150,000 individuals affected by the condition. However, while rare, immediate hypersensitivity reactions due to mast cell degranulation can be life threatening in these individuals,¹ various stimuli in the intraoperative setting can induce mast cell degranulation causing mild cutaneous symptoms such as pruritus, urticaria, and flushing; to severe hypotension, tachycardia, and cardiovascular collapse.² There are currently no formal guidelines regarding perioperative management of mastocytosis. Anesthetic techniques should be directed towards preventing the release of histamine and other mast cell mediators.¹

Case Report

A 57-year-old, 173 cm, 73 kg male patient presented for a removal of hardware and basal joint arthroplasty of the right thumb with tendon transfer. The patient's medical history included nicotine use, recreation marijuana use, anxiety with panic attacks, hypertension (HTN), occasional palpitations, mitral valve prolapse (MVP), chronic obstructive pulmonary disease (COPD), degenerative disc disease, lumbago, mastocytosis, osteoporosis, osteoarthritis. His surgical history included a lumbar discectomy, an inguinal hernia repair, and a carpometacarpal fusion of the right thumb. The patient's current medication regimen included gabapentin, amitriptyline, fluoxetine, baclofen, oxycodone/acetaminophen, and aripiprazole. The patient states that his HTN is well controlled without any medications. The patient also reported rare episodes of dyspnea and wheezing which did not require intervention or further evaluation. Physical examination of the patient and laboratory values were noted as unremarkable.

Consent was obtained for a right brachial plexus block to help manage postoperative pain. A 20-gauge peripheral intravenous catheter (PIV) was placed and an infusion of lactated ringers (LR) was initiated per gravity. Midazolam 2 mg and fentanyl 100 mcg were administered intravenously (IV) for procedural sedation. Utilizing an ultrasound guided supraclavicular approach, the trunks of the right brachial plexus, first rib, and axillary artery were identified. Bupivacaine 0.5% was administered via a single injection technique for a total of 20 mL.

Upon reassessment of the peripheral nerve block, the patient had minimal movement of his right fingers and wrist. An additional 1 mg of midazolam and 50 mcg of fentanyl were administered IV upon leaving the preoperative holding area. Once in the operating room, the patient moved himself to the operating table where electrocardiogram, non-invasive blood pressure monitoring, and pulse oximetry monitors were applied. The patient was placed in the sniffing position and preoxygenated with oxygen (O₂) at 10 L/min via facemask for 3 minutes. General anesthesia was induced via inhalation with sevoflurane 3% inspired and a combination of lidocaine 60 mg and propofol 80 mg administered IV. After anesthesia induction and the ability to mask ventilate, a size-5 iGel supraglottic airway (Intersurgical Inc., Liverpool, NY) was inserted and subglottic

movement of air was auscultated and verified by capnography. Fresh gas flows were adjusted to O₂ 1L/min, sevoflurane inspired concentration of 1.5%, and nitrous oxide (N₂O) at 2L/min. Metoclopramide 10 mg and famotidine 20 mg were administered IV to enhance gastric motility and decrease stomach acidity. Antibiotic prophylaxis was achieved with the administration of cefazolin 2 g IV.

The patient was maintained on sevoflurane 0.8% inspired concentration, N₂O 1.25 L/min and O₂ 0.75 L/min. Pressure support ventilation was utilized to maintain adequate tidal volumes and help prevent atelectasis. An additional fentanyl 50 mcg IV was given for suspected tourniquet pain. Total tourniquet and surgical time was 96 and 116 minutes respectively. Upon surgical closure, N₂O was discontinued, O₂ was increased to 4 L/min and the inspired concentration of sevoflurane was increased to 1.5%. Ondansetron 4 mg IV was administered at this time to reduce the incidence of post-operative nausea and vomiting (PONV). Emergence was initiated by turning off the volatile anesthetic and increasing the O₂ to 10 L/min. Within 2 minutes, the patient aroused to name and the airway was removed without incident. The patient was then transferred to the post anesthesia care unit (PACU) where he denied any PONV. Once in the PACU, the patient complained of chronic back pain and was treated with hydrocodone/acetaminophen 5/325 mg 2 tablets taken by mouth. He was subsequently discharged home approximately 90 minutes later.

Discussion

Mastocytosis contains a spectrum of disorders with varying clinical presentations characterized by mast cell hyperplasia in the skin and in other organs.² It is associated with somatic point mutations at codon D816V³ in the transmembrane receptor c-kit tyrosine kinase, which is implicated in the survival and activation of mast cells.^{1,4} Approximately 10% of individuals diagnosed with the initial cutaneous form, urticaria pigmentosa, will have mast cell infiltration beyond the skin into various organ systems.⁴ Systemic mastocytosis has various forms depending on clinical characteristics and severity of organ infiltration.¹ The most common type of mastocytosis seen in adults is indolent systemic mastocytosis which carries a good prognosis.¹

Perioperative management of mastocytosis is based on the potential for intraoperative mast cell degranulation and a potentially life threatening anaphylactoid reaction.² Prevention of mediator release is a major anesthetic goal in patients with mastocytosis¹. Various intraoperative stimuli can cause mast cell degranulation resulting in release of vasoactive and immunoregulatory mediators (histamine, leukotrienes, prostaglandins, and proteases).³ This anaphylactoid response appears similar to an allergic reaction, yet it differs as bronchospasm and angioedema do not typically occur.¹

The incidence of immediate hypersensitivity reactions under anesthesia in patients with mastocytosis is unknown.¹ Mastocytosis has not been studied to any extent with regards to perioperative management, and it is likely that the majority of uneventful procedures go unreported.¹ The majority of case studies reflect an uneventful intraoperative course¹, however life-threatening reactions have been reported even in minor procedures.^{1,2}

During the preoperative assessment, the patient could not provide specific triggers related to his mastocytosis. He stated that he follows a hematologist annually. A history of anxiety was reported with symptoms of palpitations and chest tightness, symptoms sometimes reported with immediate hypersensitivity reactions.³ Prior allergies included Darvocet which caused pruritus and a rash, symptoms that have been reported with medication induced reactions in mastocytosis.³ Cutaneous assessment of the patient was unremarkable, which is not uncommon even in the cutaneous forms of mastocytosis.³ Osteoporosis, which the patient reported, is a common occurrence due to excessive histamine release⁴ and care should be taken when positioning to avoid causing a fracture.¹ Preoperative skin testing is only recommended if the patient has had a perioperative reaction in the past and the agent remains unknown.¹

Anxiolysis is recommended in patients with mastocytosis as psychological stress may cause a reaction.¹ Premedication with histamine (H₁ and H₂) receptor antagonists and/or corticosteroids is typically recommended, yet this has not been studied in placebo-controlled trials.¹ The patient received midazolam and famotidine during the perioperative course. Hydrocortisone and diphenhydramine have also been used in addition to famotidine in other case studies.³

Temperature should be monitored and maintained during the case as extreme temperatures have been shown to initiate degranulation.¹ Mechanical irritation or tourniquet use on skin lesions may induce a reaction.¹ The patient did not have any skin lesions, yet surgical trauma and pain alone may cause degranulation.¹

There are no formal recommendations or cohort studies concerning management of pain and intraoperative stressors for patients with mastocytosis. The use of opioids intraoperatively is a widely suggested accepted safe practice.¹ The patient received a supraclavicular brachial plexus block. This approach provides effective and consistent coverage of the upper extremity for both anesthetic purposes and postoperative pain management.⁵ The medial arm is not anesthetized in a supraclavicular approach; therefore, tourniquet discomfort may initiate a reaction. Fentanyl was utilized in response to increases in blood pressure and respiratory rate. The use of high-dose remifentanyl (1.0 mcg/kg/min) has been shown to attenuate the stress response due to tourniquet pain⁶ and with its rapid elimination half-life, it may be useful to control intraoperative pain in patients with mastocytosis. Inhibiting surgical stress response in patients with mastocytosis with the use of remifentanyl is a clinical area that has yet to be studied.

An iGel supraglottic airway (Intersurgical Inc., Liverpool, NY) was selected as a less invasive approach to a standard endotracheal tube (ETT). Use of a supraglottic device, such as a laryngeal mask airway (LMA), versus an ETT has been associated with less hemodynamic instability and a lower incidence of laryngospasm and cough during emergence.⁷ The iGel has been shown to cause less pharyngeal trauma and less postoperative pain over the traditional LMA.⁸ Limiting stimulation and hemodynamic fluctuations results in less medication administration which decreases the risk for a hypersensitivity reaction due to pharmaceutical triggers.

Histamine releasing drugs and known drugs that trigger a reaction should be avoided.³ It is recommended to avoid atracurium, mivacurium, and nefopam due to their histamine releasing action.¹ Antibiotics or other infusions of medications that may cause histamine release should be avoided if possible.¹ Clinical case studies of patients with mastocytosis typically recount an

uneventful postoperative course with only mild adverse effects reported possibly from intraoperative mechanical stressors.¹

Perioperative management of mastocytosis is largely unstudied and has no standardized recommendations. Provided that triggers for mast cell degranulation are avoided patients can typically have a safe and uneventful perioperative course.

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Laryngeal Mask Airway use for Pediatric Tonsillectomy and Adenoidectomy

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Keywords: laryngeal mask airway, tonsillectomy, adenoidectomy, adenotonsillectomy, pediatrics

Tonsillectomy and adenoidectomy is the second most common operation performed on children younger than 15 years of age in the United States.¹ Historically, recurrent tonsillitis and its sequelae has been the most prevalent indication for the procedure. However, recently obstructive sleep apnea in children has become the primary indication for removal of the tonsils and

adenoids, partially attributed to the pediatric obesity epidemic.¹ General anesthesia with an endotracheal tube (ETT) has long been considered standard care for the pediatric patient undergoing an adenotonsillectomy, however in recent years the use of laryngeal mask airways (LMA) has become more accepted.^{2,3}

Case Report

A 6-year-old, 114 cm, 19 kg female presented for adenotonsillectomy. The patient's past medical history consisted of hypertrophic obstructive tonsils and adenoids and recurrent tonsillitis. The patient tested positive for strep throat 3-days prior to surgery, however symptoms cleared with antibiotic therapy. Her current medication regimen included amoxicillin, atomoxetine, and nasal fluticasone. Physical examination and pre-anesthetic evaluation of the patient revealed no abnormalities except hypertrophic tonsils.

Preoperatively the patient was given 10 mg of oral midazolam for anxiolysis 20 minutes prior to induction of anesthesia. The patient was transferred to the operating room where standard monitors were placed. Induction of anesthesia was initiated with a mixture of O₂ 3 L/min and nitrous oxide 7 L/min by mask. Sevoflurane was provided in increasing increments up to an inspired concentration of 8% in O₂ 8 L/min. General anesthesia was achieved and spontaneous ventilations were maintained. A 22-gauge peripheral intravenous line was placed in the patient's right hand and an infusion of 0.9% saline was initiated. The airway was maintained using a 2.5 sized flexible reinforced LMA with one attempt. Placement was confirmed with positive end-tidal carbon dioxide and bilateral breath sounds. The LMA was attached to the pediatric anesthesia circuit and spontaneous ventilations were maintained. Intravenous hydromorphone 0.2 mg and dexamethasone 3 mg were administered. General anesthesia was maintained with sevoflurane 2.8-3.2% inspired concentration in a mixture of O₂ 1 L/min and air 1 L/min. The operating table was then turned 90° and the surgical procedure was started.

Once the desired stage of general anesthesia was achieved, a Crowe-Davis mouth-gag was inserted in the mouth by the surgeon and suspended from a mayo stand. A red rubber catheter was passed through the nose and the soft palate was suspended. Bupivacaine 0.25% was injected bilaterally into the tonsillar pillars. The left tonsil was grasped and retracted medially and then removed with a coblation wand from superior to inferior pole. The process was repeated on the right tonsil. The adenoids were then removed with the coblation wand.

At the end of the procedure the table was rotated back to its initial position. Sevoflurane inspired concentration was reduced to 2% in O₂ 8 L/min. Intravenous ondansetron 2 mg and hydromorphone 0.1 mg were administered. The oropharynx was suctioned above the LMA and it was removed while the patient continued spontaneously breathing. An oropharyngeal airway was inserted and the patient was placed on O₂ 8L/min via simple face mask. The patient was moved to the transport bed and placed in a lateral position. She was taken to the post-anesthesia care unit where her wake-up was uneventful.

Discussion

In the United States over 737 000 ambulatory tonsillectomies were performed in 2006 according to estimates by the Centers for Disease Control and Prevention.⁴ Historically, general anesthesia with an endotracheal tube has been the most common anesthetic management for adenotonsillectomy. Reasons for this may include the anesthesia professionals need for airway protection and control. During the procedure the airway will be 'shared' with the surgeon, often with the head of the bed rotated away from the anesthesia practitioner.³ In recent years the use of the flexible reinforced LMA has become increasingly accepted for use during adenotonsillectomy in the pediatric population.^{2,3}

The use of the LMA provides multiple advantages to the anesthesia practitioner. Insertion of the device avoids the need for laryngoscopy and endotracheal tube stimulation of the vocal cords and larynx, thus attenuating the sympathetic response associated with endotracheal intubation.⁵ The use of an LMA is also shown to demonstrate a statistically significant reduction in coughing and laryngospasm during emergence from anesthesia, postoperative sore throat, and hoarseness when compared to the use of an endotracheal tube.⁶ Other reported advantages of LMA use include elimination of muscle relaxant use and possible adverse effects, decreased opiate use, decreased anesthetic requirement, decreased operating room times, and lower overall costs.⁵ The flex reinforced LMA was designed for ears, nose, and throat (ENT) procedures. The flexible shaft of this device allows for rotation, flexion, and extension of the head and neck during surgery.⁷ Because the laryngeal mask is located below the surgical site, a majority of blood and secretions are prevented from entering the lower airway and stomach, thus decreasing the likelihood of aspiration and laryngospasm.⁸

Disadvantages of LMA use during adenotonsillectomy have also been reported. Visualization of the surgical field may be hindered by the LMA, leading to decreased surgeon satisfaction with their use.⁵ Surgeons may also find it easier to work with an endotracheal tube as they may find the LMA more burdensome and bulky and more likely to become dislodged.³ Laryngeal mask airway failure during adenotonsillectomy has been reported as high as 6.8%, with a lower incidence occurring with adenoidectomy alone.² Most complications were associated with LMA insertion and mouth gag placement requiring a conversion to tracheal intubation and associated additional airway manipulation.² Problems may also arise with oxygenation, difficulty with ventilation, and kinking or leaking of the LMA.⁵

The surgical method for performing a tonsillectomy with adenoidectomy may also influence whether an LMA is a suitable option when providing general anesthesia as opposed to an endotracheal tube. It could be assumed that a cuffed endotracheal tube is better suited for airway protection in the instance of profuse intraoperative bleeding. The otorhinolaryngology surgeons at this institution utilized coblation when performing adenotonsillectomy. Coblation uses bipolar radiofrequency rather than heat to remove tissue and has been associated with less postoperative pain when compared to electrocautery.¹ The incidence of intraoperative bleeding with coblation is low. One prospective study found that intraoperative blood loss was less than 5 mL in more than 90% of patients with no patients experiencing greater than 20 mL of blood loss.⁹ Low blood loss with this method of surgical intervention may make it ideal for use with an LMA. It is also

important to consider that a majority of blood accumulation will be located above the laryngeal mask and can be easily suctioned away by the surgeon.

As discussed, many considerations are to be made when planning an anesthetic that utilizes an LMA for maintaining an airway during an adenotonsillectomy in the pediatric patient. Most of all there must be a level of comfort and constant line of communication between the anesthesia professional and the surgeon. If either feel uncomfortable with the decision to use an LMA the operation should be performed using an endotracheal tube. Patient safety is the anesthesia professional's number one priority. The use of the flexible LMA during ENT procedures including adenotonsillectomy has been demonstrated to be safe. This institution does a majority of its pediatric ENT and dental procedures with LMAs when it suits the patient's anesthetic needs. As with all forms of anesthesia, the anesthesia practitioner must be skilled in the use of LMAs for these types of procedures and have the ability to troubleshoot any problems if they occur.

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Charcot Marie Tooth Disease and the use of Neuromuscular Blockers

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Keywords: Charcot Marie Tooth disease, Charcot Marie Tooth disease type 1A, neuromuscular blocker, anesthesia implications, and general anesthesia

Charcot Marie Tooth disease (CMT) is one of the most prevalent neurogenetic conditions with an estimated prevalence of 4 in every 10,000 people.¹ Classic symptoms include peripheral muscle weakness and sensory loss in extremities due to demyelination of the peripheral nerve fibers.¹ Over 25 genes are associated with this disorder, resulting in variability of symptom onset, presentation and rate of progression.² Chief concerns regarding general anesthesia in a patient with CMT relate to the decision of whether to use neuromuscular blockers and the ability to fully reverse their paralytic effects within an expected timeframe.

Case Report

A 38-year-old, 58 kg, 165 cm male presented for left extracorporeal shock wave lithotripsy. Medical history included hypertension, melanoma, decreased lung capacity, osteopenia, leukocytopenia, poorly-controlled gastroesophageal reflux disease (GERD), CMT type 1A, migraine headaches, nonischemic cardiomyopathy, sleep-related hypoventilation due to neuromuscular disorder, sleep apnea, kidney disease, and nephrolithiasis. Current medications included oxycodone-acetaminophen, ciprofloxacin, butalbital-acetaminophen-caffeine, metoprolol succinate and cholecalciferol.

The patient received midazolam 1 mg intravenously (IV) and fentanyl 50 mcg IV in the pre-operative holding room due to anxiety and pain. An hour later, the patient was transported to the operating room and was carefully positioned onto the lithotripsy table. The electrocardiogram, non-invasive blood pressure cuff and finger pulse oximetry monitors were applied.

The patient was pre-oxygenated via face mask at 10 L/min. Induction medications for an induction with applied cricoid pressure were administered IV and included: midazolam 2 mg, fentanyl 100 mcg, lidocaine 40 mg and propofol 200 mg. With manual in-line stabilization and cricoid pressure applied, a GlideScope® (Verathon Inc., Bothell, WA) size 3 blade was used to visualize the patient's vocal cords and successfully intubate with a 8.0 endotracheal tube (ETT) following an unsuccessful attempt utilizing a size 4 blade. Bilateral breath sounds were auscultated and respiration was controlled by a mechanical ventilator. General anesthesia was maintained with sevoflurane 2% inspired concentration in a mixture of O₂ 1 L/min and air 1 L/min.

Near the conclusion of surgery, with appropriate ventilation adjustments, the patient began to breathe spontaneously. Sevoflurane was discontinued and the patient was placed on 100% FiO₂. The total procedure time was 167 minutes. The patient quickly met extubation criteria once he moved all extremities to command, and was breathing spontaneously with a regular respiratory rate and tidal volumes greater than 400 mL. The patient's airway was suctioned and extubated without complication, and oxygen 4 L/min was applied via nasal cannula. The patient was carefully moved to his bed and transferred to the post anesthesia care unit (PACU). Upon arrival

to the PACU, the patient was able to answer all questions appropriately and denied pain or nausea. The patient's PACU stay was uneventful and he was discharged later that day without any complications.

Discussion

The present discussion is concerned with the appropriate use of neuromuscular blocking agents in a patient with CMT, which classically causes peripheral muscle weakness and sensory loss due to demyelination of peripheral nerve fibers.¹ Denervation of nerve fibers predisposes affected muscle tissue to potassium release upon exposure to succinylcholine and therefore, the use of succinylcholine in CMT patients is typically avoided.³ Similar to other neuromuscular diseases, succinylcholine use for induction may result in severe hyperkalemia, as these patients may have an "up-regulation of extrajunctional acetylcholine receptors, which renders them more likely to have an exaggerated release of potassium upon exposure to succinylcholine."^{4(p2)} For critical situations in which the use of succinylcholine cannot be avoided, use of "a small defasciculating dose of a non-depolarizing neuromuscular blocker may lessen the potassium release from diseased muscle."^{3(p.399)} If hyperkalemia does occur, reactions may range from subtle EKG changes or peaked T waves to malignant arrhythmias or even cardiovascular collapse.³ Other potential risks for neuromuscular blocker use in a CMT patient include the potential for weakness upon emergence, inability to obtain/maintain adequate tidal volumes, and risk of prolonged intubation.⁵ Classically affected CMT patients with existing generalized muscle weakness may further experience prolonged respiratory dysfunction if administered non-depolarizing neuromuscular blocking agents.⁴ In addition, the use of volatile anesthetics can potentiate the effects of non-depolarizing neuromuscular blocking agents.¹

Multiple factors were considered when making decisions for this case, including the patient's medical history, evidence of advanced CMT, and available resources of the surgical center. The patient's history of poorly controlled GERD resulted in the need for an induction with cricoid pressure and ETT. The history of surgical spinal fusion led to the decision to apply manual in-line stabilization. The use of succinylcholine was ruled out due to the possible up-regulation of acetylcholine receptors and potential response of hyperkalemia.⁴ Two options remained: to use a nondepolarizing neuromuscular blocker or use increased doses of induction agents and avoid the use of a neuromuscular blocking agent. The patient's CMT disease was considered advanced as evidenced by being wheel-chair dependency for several years along with numbness, weakness and extensive limb deformities in both his upper and lower extremities. Therefore, due to the patient's extensive disease and concern for a prolonged neuromuscular block, the decision to avoid the use of a neuromuscular blocker was made. By giving higher doses of induction agents the patient's airway was safely secured without using a neuromuscular blocker.

One major difference between this case and what was found in the literature review, was the severity of this patient's disease, as only one other case was found which involved a wheel-chair bound patient. Interestingly, of the two most recent case studies found in the literature, one avoided the use of a neuromuscular blocker while the other used a reduced intubating dose of rocuronium.^{4,5}

Although research regarding the anesthetic management of patients with CMT is limited—and occasionally conflicting—research has shown that children and adults with CMT who undergo general anesthesia for surgery do recover within the normal, expected time frame when given a standard intubating dose of a neuromuscular blocker (either depolarizing or non-depolarizing), as compared to normal, healthy patients without CMT disease.^{1,3,6} One potential reason for conflicting results among this population is the variation in symptom presentation, disease progression and severity, coupled with the fact that patients present for surgery all along the continuum.⁷ This variability also makes it extremely difficult to make generalizations for patients with CMT.

The use of neuromuscular blockers in CMT patients must be evaluated on a case-by-case basis, taking into consideration the extent of the patient's disease and type of surgery performed, as well as available resources. Further research is needed to make appropriate generalizations regarding anesthetic management in CMT patients.⁷

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Subcutaneous Emphysema during Laparoscopic Cholecystectomy

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Keywords: hypercarbia, subcutaneous emphysema, crepitus, pneumoperitoneum, laparoscopy

Since the first successful video laparoscopic cholecystectomy was performed in 1988, the laparoscopic technique has revolutionized surgical procedures.¹ Laparoscopic surgery is now a common alternative to open surgical techniques and offers many benefits to patients. Some of these benefits include a shorter hospital stay, more favorable cosmetic results, less risk of hemorrhage, and less postoperative pain. However, the creation of a pneumoperitoneum using CO₂ during laparoscopic procedures brings physiological changes and potential complications as well. One of the less common but potentially severe complications is the creation of subcutaneous emphysema.²

Case Report

A 77-year-old, 163 cm, 61 kg female with a history of pancreatitis, gastroesophageal reflux disease, thyroid cancer, anemia and rheumatoid arthritis presented for a laparoscopic cholecystectomy. Her past surgical history included a thyroidectomy, abdominal hernia repair, breast biopsy, hysterectomy and an endoscopic retrograde cholangiopancreatography with no history of anesthetic complications. Prescribed medications included ceftriaxone, enoxaparin, levothyroxine, metronidazole and pantoprazole.

Upon arrival to the operating room, standard monitoring was applied while preoxygenating and denitrogenating with O₂ 8 L/min for 5 minutes. Anesthesia was induced with fentanyl 100 mcg, lidocaine 80 mg and propofol 140 mg intravenously (IV). Rocuronium 30 mg IV was administered after confirmation of adequate mask ventilation. The trachea was intubated with a 7.0 mm endotracheal tube (ETT), the cuff inflated, correct placement confirmed with end-tidal CO₂ (ETCO₂) tracing, bilateral chest rise and equal, bilateral breath sounds and secured at 21 cm at the lips. The ventilator was set to volume auto flow mode with a rate of with a rate of 12/min, tidal volume (TV) of 420 mL/breath and positive end-expiratory pressure of 5 cm H₂O. Anesthesia was maintained with isoflurane 1.1% end-expired concentration in a mixture of air 0.5 L/min and O₂ 0.5 L/min.

At the time of incision, the patient had a heart rate (HR) of 86/min, blood pressure (BP) of 98/56 mm Hg, SpO₂ 99%, esophageal temperature of 37°C, ETCO₂ of 30 mm Hg and a peak inspiratory pressure (PIP) of 18 cm H₂O. Insufflation was maintained with CO₂ at a pressure of 15 mm Hg or less throughout the procedure. Approximately 10 minutes after insufflation, the ETCO₂ rose to 48 mm Hg and ventilator settings were increased to a rate of 15 breaths/min and TV 470 mL/breath. Ten minutes later, the ETCO₂ had risen to 54 mm Hg and ventilator settings were increased to a rate of 16 breaths/min and TV 550 mL/breath. Despite increased ventilation, the ETCO₂ continued to climb above 60 mm Hg. The surgeon was notified and insufflation pressures were verified to be less than 15 mm Hg.

Approximately 30 minutes post-insufflation, ET_{CO}₂ had risen to 63 mm Hg and crepitus of the neck, face, and eyelids was discovered when verifying that the ETT had not migrated. The surgeon was notified and upon inspection of the surgical site, it was discovered that a trocar had migrated cephalad and into subcutaneous tissue. The trocar was removed and the procedure was completed within 15 minutes. ET_{CO}₂ peaked at this point at 76 mm Hg. The patient's HR was 98/min, BP was 160/92 mm Hg and SpO₂ 99%. Neuromuscular blockade was antagonized with glycopyrrolate 0.6 mg IV and neostigmine 4 mg IV.

The intubated but spontaneously breathing patient was transported to the postanesthesia care unit and sedated with dexmedetomidine 0.5 mcg/kg/hr. Approximately 90 minutes later and after confirmation of normal arterial blood gas results and an adequate cuff-leak, the dexmedetomidine was discontinued and the patient was extubated to an open face mask with O₂ 6 L/min. Over the next 24 hours, the subcutaneous emphysema resolved and the patient had no further apparent complications.

Discussion

Insufflation with CO₂ during laparoscopy allows the surgeon to visualize the surgical field using a magnified view. Although this technique provides benefits to the surgeon, it comes with potential complications. Once the pneumoperitoneum is created, physiological changes can occur including increased HR, increased mean arterial pressure, increased systemic vascular resistance (SVR), increased peak airway pressure, reduced venous return, reduced functional residual capacity and reduced pulmonary compliance.^{1,2} In addition, the laparoscopic patient is at risk of hypercarbia, arrhythmias, CO₂ embolism, vascular injuries, atelectasis, pneumothorax, pneumomediastinum and subcutaneous emphysema.² More than half of all laparoscopic complications are attributed to insertion of the trocars and entry into the abdomen.¹

The incidence of subcutaneous emphysema detectable during laparoscopic surgery ranges from 0.43% to 2.34%.³ Because significant subcutaneous emphysema during laparoscopy is rare, detection can be delayed as other more common causes for increasing ET_{CO}₂ are typically ruled out first. During the case described here, hypercarbia was initially thought to be due to inadequate ventilation or absorption of insufflated CO₂. However, ET_{CO}₂, HR and BP continued to rise in the presence of increased ventilation. Other causative factors considered were bronchospasm, ETT malfunction, exhausted CO₂ absorbent, inspiratory/expiratory valve failure, right main stem intubation and malignant hyperthermia. The crepitus was discovered when assessing for masseter muscle rigidity and ETT depth.

Considering the difficulty in early recognition of crepitus, massive subcutaneous emphysema can develop. There are published reports describing rising ET_{CO}₂ despite increasing minute ventilation while subcutaneous emphysema remained undetected beneath the surgical drapes.⁴ Researchers have reported that certain factors increase the likelihood of subcutaneous emphysema during laparoscopic surgery. These include intra-abdominal pressure greater than 15 mm Hg, multiple abdominal entry attempts, veress needle placement outside of the peritoneal cavity, loose skin around the cannula site, use of more than 5 cannulas, use of the cannula as a fulcrum and procedures lasting more than 3.5 hours. If one of these factors is present, changes observed with subcutaneous emphysema include crepitus, insufflation variability with flow and

pressure, ETCO₂ greater than 50 mm Hg, hypertension, sinus tachycardia, arrhythmias, acidosis and changes in lung compliance.⁵

During laparoscopy, CO₂ is absorbed through the peritoneum and blood levels of CO₂ will rise. However, the resulting acidosis can usually be compensated by increasing the minute ventilation up to 30%.³ If minute ventilation is not increased, hypercapnia will stimulate the sympathetic nervous system leading to an increase in BP and HR, and the myocardium will become sensitized to catecholamines.³ Thus, the patient is at an increased risk for cardiac dysrhythmias. Furthermore, many laparoscopic procedures require trendelenburg positioning which results in physiological effects that can contribute to significant cardiopulmonary changes including arrest.⁶ The oxygen demand with increased venous return due to trendelenburg positioning combined with the increase in SVR and catecholamines due to insufflation can lead to a significant increase in myocardial workload that can be dangerous for patients with pre-existing cardiac disease.⁶

The increase in CO₂ due to absorption from insufflation is normally 26%. However when subcutaneous emphysema is present, CO₂ uptake has been known to increase 113%.² Researchers have found that up to 77% of laparoscopic patients have undetected subcutaneous emphysema and 20% have postoperative chest radiographs confirming a pneumomediastinum.² Furthermore, 56% of patients who have computed tomography scans within 24 hours of a laparoscopic cholecystectomy show otherwise undetectable subcutaneous emphysema.² The volume of subcutaneous emphysema is the concern as CO₂ gas insufflation is a required step of the laparoscopic process. Excessive subcutaneous emphysema can lead to airway compromise along with physiological changes due to increased CO₂ absorption and pressure on the lungs and mediastinal vasculature.¹

Subcutaneous emphysema should be considered early with increasing ETCO₂ during laparoscopic procedures. During the case described here, the primary causative factor was the displaced trocar. Although rare, this surgical complication should be considered along with the other more common factors in the presence of rising ETCO₂ despite increased ventilation. Recommendations for the anesthesia practitioner include communicating with the surgeon, evaluating the patient for a pneumothorax, ensuring that the CO₂ absorbent is not exhausted or desiccated, increasing minute ventilation and assessing the airway with a cuff-leak test before extubation to ensure that complete airway compression is not present.²

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Shoulder Surgery in Beach Chair Position under General Anesthesia

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Keywords: shoulder surgery, shoulder arthroscopy, interscalene block, beach chair position, sitting position

Outpatient surgery centers have seen a four-fold increase in shoulder procedures in the past two decades with an estimated one-third of those surgeries performed in the beach chair or sitting position.^{1,2} The beach chair position aids the surgeon in completing these procedures efficiently and effectively. Its associated risk of hypotension leading to decreased cerebral perfusion has sparked interest in understanding the relationship between systemic blood pressure and cerebral blood flow.^{3,4} This case report details the anesthesia care of a patient for outpatient shoulder surgery performed in beach chair position, and elucidates the current literature surrounding its appeal, risks, and management.

Case Report

A 40-year-old, 86 kg, 175 cm male presented for left shoulder arthroscopy and possible arthrotomy, due to instability of the left shoulder joint. The patient was taking hydrocodone 10 mg/acetaminophen 325 mg for shoulder pain, but denied any chronic medical conditions or allergic reactions. His past surgical history included a nasal septoplasty. The airway assessment revealed a Mallampati classification II airway, thyromental distance greater than 6 cm, and full cervical range of motion. All preoperative laboratory tests, electrocardiogram, and chest x-ray were normal. The patient was consented for both general anesthesia and interscalene brachial plexus nerve blockade for postoperative pain relief.

The preoperative vital signs were: blood pressure 138/92 mm Hg, heart rate 78/min, respirations 12/min, and room air SpO₂ 98%. A 20 gauge intravenous catheter was inserted in the right hand. After midazolam 2 mg was administered intravenously, the patient was brought to the operating room and transferred to the operating table. Preoxygenation commenced using the facemask with oxygen flow at 10 L/min. Standard monitors were applied, including a blood pressure cuff on the right arm. Induction was then initiated with fentanyl 50 mcg and propofol 300 mg. A number 5 laryngeal mask airway was placed and inflated with 40 mL of air to achieve occlusive pressure. An audible leak was confirmed at 20 cm H₂O pressure with the use of the adjustable pressure-

limiting valve; bilateral breath sounds were auscultated and capnography was verified. The airway device was secured in place as spontaneous, regular ventilation returned.

Sevoflurane 3% inspired concentration with oxygen at 4 L/min flow maintained anesthetic depth as the patient was then positioned sitting upright. The head was secured with tape across the forehead and jaw. The right arm was propped on an armrest in correct alignment with pulses palpable. At the time of incision, the blood pressure registered 108/82 mm Hg, heart rate 80/min, and SpO₂ 100%. The blood pressure steadily increased to 125/85 mm Hg before the surgeon converted to open technique. Adjustments in sevoflurane levels maintained the systolic blood pressure between 110-115 mm Hg and the average mean arterial pressure (MAP) at 60 mm Hg. Towards the end of the procedure, the sevoflurane was discontinued as O₂ was increased to 8 L/min flow. Spontaneous respirations were 14 breaths per minute and SpO₂ 100%. The laryngeal mask airway was removed and an oral airway inserted. The patient was positioned supine and jaw thrust was provided until the patient emerged fully from anesthesia.

During recovery, the patient complained of severe left shoulder pain and was amenable to regional anesthesia. An ultrasound and nerve stimulator-guided interscalene block was performed using sterile technique. Left biceps stimulation presented at 0.8 mV and disappeared at 0.3 mV, indicating the appropriate injection site. Preservative-free 2% lidocaine 10 mL and 0.5% bupivacaine 10 mL were administered slowly. The patient reported pain relief, and after 20 minutes of observation, was discharged home without complications.

Discussion

The beach chair position is conducive for shoulder arthroscopy due to its ease in converting to open procedure, increased mobility of the operative arm, and improved visualization of the shoulder joint.^{1,5} However, injury to the hypoglossal nerves at the neck or brachial plexus from improper positioning may be sustained.⁵ Cervical neutrality with alignment of the carotid and jugular vessels should be paramount when using tape to fasten the head to the upright operating table since compression will impede blood flow to and from the head.¹ For this case, securing the forehead and jaw to the OR headrest with tape effectively stabilized cervical alignment.

Shoulder surgery in the sitting position performed under general anesthesia presents a dilemma of balancing hypotension with adequate cerebral perfusion. General anesthesia induces systemic vasodilation and myocardial depression, exacerbating decreased preload from the preoperative fasting period. When the patient is then positioned upright, gravity pools venous return in the lower extremities and the patient may not hemodynamically compensate for position changes due to the blunted sympathetic response from general anesthesia.¹ Moreover, surgeons may request a hypotensive technique for shoulder surgeries to control for bleeding in the surgical field. This surgeon did expect the lowest allowable blood pressures for the patient during this procedure.

Reports of rare but significant incidences of vision changes, stroke, and brain death in otherwise healthy individuals have prompted investigations into how blood pressure should be monitored while the patient is under general anesthesia in the beach chair position since low blood pressure reflects low cerebral perfusion.¹⁻⁴ Ankle blood pressure cuff measurements compared to brachial

blood pressure cuff measurements are significantly higher and may mislead anesthesia practitioners if they do not account for height differences.⁴ Brachial measurements in the non-operative arm are recommended, and if ankle measurements must be performed, it is imperative to calculate and correct for distance between the head and extremity: a 2 mm Hg blood pressure decrease for every 2.5 cm height difference between the head and measurement site.^{1,4}

Despite routine methods to detect hypotension using noninvasive blood pressure cuffs, continued interest remains in identifying cerebral hypoperfusion under general anesthesia in the beach chair position.^{1,2} Autoregulation of cerebral blood flow exists when MAP is between 50 to 150 mm Hg; its compromise is associated with a drop of the MAP lower limit.⁴ Researchers have noted a relationship between the beach chair position and decreased cerebral autoregulation compared to supine positioning, though no cognitive impairments were observed postoperatively.² A standard of lower limit for MAP or preference for invasive over noninvasive blood pressure monitoring specifically in the outpatient setting has not been established. Cerebral oximetry by near-infrared spectroscopy may provide real-time readings of cerebral hypoperfusion despite MAP measurements above 60 mm Hg; however, MAP remains the gold standard in detecting cerebral desaturation.³ Recent literature cautions lowering blood pressure further than 20% of the patient's baseline, especially when accounting for patient comorbidities such as cardiovascular disease and hypertension.¹ Therefore, it is prudent to establish both lower MAP and blood pressure limits on an individual basis. For this case, the patient's lower limits were calculated as 110 mm Hg systolic blood pressure and MAP of 60 mm Hg, 20% of his baseline measurements. These parameters yielded favorable surgical conditions without compromising cerebral perfusion.

Anesthesia technique has also been studied in an effort to avoid general anesthesia altogether with surgeries performed in the sitting position. An interscalene brachial plexus block provides anesthesia solely to the operative arm while preserving spontaneous ventilation and systemic vascular resistance.^{6,7} Current literature recommends this block for shoulder surgery should be the primary technique to avoid cerebral desaturation associated with the sitting position.^{1,7} In our case, surgeon preference for general anesthesia was respected, and the interscalene block was performed postoperatively. This regional technique combined with general anesthesia has not shown to fast-track shoulder surgery patients out of the recovery room when compared with general anesthesia alone.⁶ Postoperative pain scores however do improve, decreasing the need for opioids during recovery.⁶ Achieving adequate pain relief expedites turnover, which was observed in our patient's postoperative management.

Though a regional technique could not be the primary anesthetic for this case, spontaneous ventilation with a laryngeal mask airway under general anesthesia was maintained. Current literature suggests the ventilation mode may influence MAP as positive pressure ventilation delivered under general anesthesia worsens hypotension by further decreasing venous return.¹ In contrast, spontaneous ventilation allows for adequate MAP and cerebral blood flow. In our case, the patient returned to spontaneous ventilation after the LMA was placed and continued to do so throughout the procedure.

Hypotension is a recognized and potentially dangerous complication of the beach chair position for shoulder surgery particularly when performed under general anesthesia. The location and

measurement correction of non-invasive blood pressure monitoring, maintenance of spontaneous ventilation, and utilization of regional blockade can help determine the best management in the outpatient setting.

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Postoperative Nausea and Vomiting Prevention

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Keywords: postoperative nausea and vomiting prevention, serotonin antagonist, steroids, droperidol, haloperidol, dexmedetomidine, acetaminophen, mirtazapine

Approximately 20-30% of all patients undergoing general anesthesia will experience postoperative nausea vomiting (PONV).¹ Patient's with a history of PONV have a 50-80% chance of experiencing repeated PONV.¹ PONV is triggered by stimulation of multiple pathways

of the vomiting center within the brain. PONV not only leads to patient dissatisfaction but also increased complications and prolonged hospital stay. A survey conducted on what one would pay to avoid PONV found that 50-100 dollars more would be paid by the patient to prevent PONV.²

Case Report

A 22-year-old, 63 kg, 134 cm female patient presented for a thyroidectomy with central lymph node dissection secondary to a history of thyroid cancer. The patient's medical history was significant for anxiety, depression, thyroid cancer, and PONV following her previous partial thyroidectomy. Her past surgical history was partial thyroidectomy approximately four weeks prior to the scheduled thyroidectomy completion surgery. Medications taken at home included: docusate sodium, oxycodone/acetaminophen, acetaminophen/aspirin/caffeine, levonorgestrel, and ondansetron.

After the patient arrived in the preoperative holding room, a scopolamine patch 1.5 mg, was applied by the preoperative nursing staff. An IV of Lactated Ringers (LR) was initiated and midazolam 2 mg was given intravenously for anxiolysis prior to being brought to the operating room. Once the patient was in the operating room, EKG leads, a non-invasive blood pressure cuff, and an O₂ saturation monitor were applied. The patient was given fentanyl 100 mcg IV and pre-oxygenation was initiated via mask at 10 L/min. General anesthesia was induced with propofol 150 mg, lidocaine 20 mg, and rocuronium 35 mg IV to facilitate tracheal intubation.

Laryngoscopy was performed with a Miller 2 blade, yielding a grade 1 view. The trachea was intubated with a 7.0 endotracheal tube (ETT). Confirmation of the tracheal ETT placement was confirmed by direct visual laryngoscopy, positive fogging of the ETT with ventilations, positive end-tidal CO₂ wave form, equal rise and fall of chest with ventilation, and auscultation of clear and equal bilateral breath sounds with ventilation. The patient was placed on a mechanical ventilator with a mixture of O₂ 1 L/min and air 1 L/min. General anesthesia was maintained with a propofol infusion at 150 mcg/kg/min and titrated to keep the BIS value between 40-60.

The following medications were then given intravenously: acetaminophen 1000 mg, dexamethasone 10 mg IV, and cefazolin 2 g. The surgery proceeded without incident and the patient remained hemodynamically stable through the case without any episodes of hypotension. Ondansetron 4 mg IV and ketorolac 30 mg IV were given at the end of the case. The surgical procedure lasted approximately 60 minutes. The patient's neuromuscular blockade was antagonized with neostigmine 3 mg IV and glycopyrrolate 0.4 mg IV. After the administration of neostigmine and glycopyrrolate, four out of four twitches without any fade and sustained tetany were noted. The patient was extubated without incident, and a nasal cannula was applied with 4 L/min of oxygen.

The patient was taken to PACU and received fentanyl IV only twice for pain. The patient did not experience PONV in the PACU. The patient was discharged home later in the day without any complications. The patient did not have any episodes of PONV that were noted after her stay in the PACU.

Discussion

Nausea and vomiting are stimulated from receptors located in the chemoreceptor trigger zone (CRTZ) and nucleus tractus solitaries (NTS) which are located in the medulla oblongata and the brainstem. The blood brain barrier is not as developed around the CRTZ and the NTS as in other parts of the central nervous system. This can lead to direct stimulation from metabolites and medications.²

The receptors in the CRTZ and NTS are stimulated from various neurotransmitters secreted from special cells throughout the body. Enterochromaffin cells are located within the GI tract, and with direct stimulation of the vagus nerve, these cells release serotonin when stimulated. The serotonin that is released binds to 5-hydroxytryptamine-3 (5HT₃) receptors.² The CRTZ communicates with the NTS directly through the dopamine 2 receptors (D₂).² Neurokinin 1 (NK₁) receptors are stimulated by the afferent impulse from the vagal tract and substance P.³

A simplified risk factor scoring system was developed for PONV prediction, which aimed to help guide anesthesia practitioners in treatment for patients that are high-risk for developing PONV. The criteria which help to determine if a patient is at a high risk for PONV are female gender, nonsmoker, history of PONV, and postoperative opioid usage. The scoring system has a range of 10 to 70% likelihood based on the number of risk factors. However, it was determined that nothing could predict with absolute certainty whether or not a patient will develop PONV.¹

Serotonin antagonists are a commonly used antiemetic either for prophylaxis or treatment of PONV. There is strong evidence supporting the use of serotonin antagonist medications. Newer serotonin antagonists show a greater duration of action in the prevention of PONV than earlier generation serotonin antagonists.³ Ondansetron has been shown to be most efficacious when given towards the end of surgical cases lasting greater than two hours.⁴

This patient was given dexamethasone 5 mg during her first thyroid surgery. She was given dexamethasone 10 mg during this surgery with the intent of preventing PONV. Dexamethasone, has been extensively studied and has been proven to be an, effective, safe medication with no effect on wound healing or increasing the risk of infection. Dexamethasone is thought to help reduce PONV from a decrease in the inflammatory response that could lead stimulation of the PONV. Steroids, given within a multimodal approach, are effective in preventing PONV.⁵

The patient in the current case report had a transdermal scopolamine patch (TDS) applied upon arrival in the preoperative holding room. Two hours lapsed from arrival into the facility until the start of general anesthesia. TDS has been proven to be a safe and effective adjunct in helping to reduce PONV by binding the histamine 1 (H₁) receptors in the CRTZ and NTS.⁶ Adverse side effects have been reported and, are usually seen in patients where the patch was applied 24 hours prior to surgery.⁶ TDS has been shown to be most efficacious when applied at least two hours before the start of general anesthesia in patients at a high-risk for PONV.⁶

Given the patient's history of PONV during her previous hemithyroidectomy, an anesthetic plan that included interventions to decrease the modifiable risk factors was developed. Total intravenous anesthesia (TIVA) utilizing propofol was utilized. PONV is reduced when propofol

is utilized as a TIVA method when compared to inhalation agents.⁷ Propofol in sub-hypnotic doses has been shown to decrease PONV as it reduces serotonin 5HT₃ receptor stimulation.

Decreasing the amount of opioids used in the perioperative setting helped to reduce the incidence of PONV. The best way to maintain adequate pain relief while reducing the amount of opioids is through a multimodal approach for pain management. Ketorolac and IV acetaminophen and were given in an attempt to decrease the amount of opioids needed in this case. Intravenous acetaminophen has shown some benefit in providing adequate pain relief and reducing the amount of opioids needed, and has been shown to achieve steady state and enter into the CSF more rapidly than oral acetaminophen.⁸ Binding to pain receptors occurs earlier, which decreased the amount of opioids that were required to adequately control pain 24 hours postoperatively when compared to than the oral or rectal acetaminophen groups.⁸

Research supports that when dexamethasone and ondansetron are given together they are more efficacious than when either is given alone for PONV prevention.⁵ Longer acting antiemetics may be warranted in patient specific situations to help prevent PONV for outpatients that develop nausea and vomiting after being discharged home which is known as post discharge nausea and vomiting (PDNV).

Future research should focus on developing methods to identify the most efficacious dosages for antiemetics based on the risk factor scoring system develop by Apfel et al.¹ Additional research could also focus on identifying adjunctive medications that may be utilized to decrease the amount of opioids required perioperatively. Lastly, as longer acting antiemetics are developed, more research is needed to focus on appropriate dosages, efficacy of timing, and efficacy in multimodal treatment.

PONV can increase complications, decrease patient satisfaction, and may lead to preventable admission to the hospital for treatment. Prevention and treatment of PONV can be achieved with simple steps for low risk patients. A multimodal approach can help decrease the incidence of PONV for both low and high risk patients. While there is no absolute way to prevent PONV in all patients, the use of current evidence based guidelines likely will prevent PONV in the majority of patients who are at risk.

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Pulmonary Recruitment Maneuver Affects on Laparoscopic Complications

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Keywords: laparoscopy, pain, shoulder pain, pneumoperitoneum, PONV, analgesia, surgery, pulmonary maneuver, anesthesia

Introduction

Postoperative shoulder pain is a common complication following laparoscopic surgery with an incidence as high as 80%.¹ The etiology of the shoulder pain is thought to be due to residual carbon dioxide in the abdominal cavity, however the exact mechanism has not been fully elucidated.² Anesthesia practitioners are challenged with providing effective postoperative pain relief while minimizing complications. Evidence in the literature suggests laparoscopic related complications such as pain and postoperative nausea and vomiting (PONV) can be reduced by implementing a pulmonary recruitment maneuver in Trendelenburg position (30°) at the end of surgery.³ The maneuver may also have an affect on postoperative analgesic requirements.^{4,5} The purpose of this evidence-based-analysis is to investigate the effects of a pulmonary recruitment maneuver at the end of laparoscopic surgery on postoperative pain, analgesic requirements, and PONV.

Methodology

A population, intervention, comparison, and outcome (PICO) question was used to provide the clinical framework, “What are the effects of a pulmonary recruitment maneuver (I) in adult laparoscopic surgical patients (P) on postoperative pain, analgesic requirements, and PONV (O) compared to patients who do not receive a pulmonary recruitment maneuver (C)?”

A literature review was performed by searching the electronic databases *CINAHL* and *PubMed*. Keywords used for the search included: *shoulder pain, surgery, pulmonary maneuver, laparoscopy, pain, pneumoperitoneum, PONV, analgesia*. The author found one meta-analysis that examined the effects of a pulmonary recruitment maneuver on postoperative pain, which was determined to be level I evidence according to the Joanna Briggs Institute Levels of Evidence. The author also found six RCTs (Table 1), level II evidence, with similar methodologies that were used for this review. In each RCT, independent variables for removal of CO₂ from the abdomen included either a pulmonary recruitment maneuver or passive exsufflation.

Literature Review

In this literature review the “treatment group” refers to a group of subjects receiving a pulmonary recruitment maneuver. “Control group” refers to a group of subjects that did not receive a pulmonary recruitment maneuver.

Postoperative Pain. The pathophysiology of shoulder pain following pneumoperitoneum is poorly understood. It is thought to be caused by retained CO₂ in the abdomen, which leads to diaphragmatic irritation and peritoneal stretching.^{1,4} However similar pain can be present with gasless laparoscopy.⁶ The incidence of shoulder pain usually peaks 24-48 hours postoperatively⁴ and affects the right shoulder more than the left.⁷

A pulmonary recruitment maneuver at the end of surgery can reduce the incidence of shoulder pain.^{1,3-5} The extent to which incidence was decreased varied among studies. Incidence of shoulder pain was significantly less in the treatment group compared to control at 12, 24, and 36 hours postoperatively;³ 4, 12, 24, and 48 hours postoperatively;⁴ 12, 24, and 48 hours postoperatively;¹ and a decreased overall incidence.⁵ One consistent finding across studies was that the pulmonary recruitment maneuver intervention significantly reduced the incidence of shoulder pain between 12 and 48 hours postoperatively compared to passive exsufflation.

Results from four studies showed a pulmonary recruitment maneuver at the end of surgery reduced the severity of shoulder pain.^{1,3,4,8} The severity of shoulder pain was significantly less in the treatment group compared to control at 12, 24, and 36 hours postoperatively.³ However, there was no significant difference in shoulder pain between groups at discharge.³ A weakness in this study is that pain scores were self-reported and subjects were instructed to only report shoulder pain, not surgical pain. Subjects in this study may have reported surgical pain instead of shoulder pain, which may cause the results to be misrepresented.

Sharami et al. found the severity of shoulder pain was significantly lower in the treatment group compared to control at 4, 12, 24, and 48 hours postoperatively. Additionally, the mean duration of surgery was significantly lower in the control group.⁴ Khanna et al. found pain severity was less in the treatment group compared to control at 12 and 24 hours postoperatively.⁸ However, a weakness was that their working definition of pain was broad and included “both abdominal and shoulder tip pain”⁸ (1291). Tsai et al. found shoulder pain severity was less in the treatment group compared to control at 12, 24, and 48 hours postoperatively.¹ It is important to note the

treatment group received both an intraperitoneal infusion of normal saline and a pulmonary recruitment maneuver to aid in exsufflation of the abdomen in the aforementioned study¹. In two studies, there was no significant difference between groups with respect to shoulder pain severity.^{5,9}

A pulmonary recruitment maneuver can significantly reduce the severity of postoperative abdominal pain.^{1,5,8,9} Definitions of abdominal pain varied somewhat among studies. Liu et al.⁵ examined pain during movement and at rest. Dynamic pain, defined as pain while standing or coughing, was significantly lower in the treatment group compared to control at 0, 4, and 24 hours postoperatively.⁵ Static pain, defined as pain at rest, was significantly lower in the treatment group compared to control at 0, 2, and 24 hours postoperatively.⁵ Tsai et al. collected data on both upper abdominal pain and surgical pain.¹ Upper abdominal pain was significantly lower in the treatment group compared to control at 12 and 24 hours postoperatively. Tsai et al. found no significant difference in the incidence or severity of surgical wound pain between groups.¹ Tsai et al. collected data on upper abdominal pain and lower abdominal pain.⁹ They found no significant difference in lower abdominal pain severity or incidence between treatment and control groups. Upper abdominal pain severity was significantly less in the treatment group compared to control at 12 and 24 hours postoperatively.⁹ A weakness in the study design was that the investigators collecting postoperative data were not blinded to subject allocation.⁹ Two of the studies did not collect data on abdominal pain.^{3,4}

Postoperative Nausea and Vomiting. Laparoscopic surgery may be implicated in PONV. The pneumoperitoneum decreases intestinal blood flow, which can lead to gastrointestinal ischemia. Serotonin, a highly emetogenic substance, is released in response to ischemia which may lead to PONV.¹⁰ Complications associated with PONV can lead to prolonged hospital stay and increased costs.¹¹ A pulmonary recruitment maneuver following laparoscopic surgery has been shown to decrease PONV.³

Four of the six studies collected data regarding the incidence of PONV. Three studies found no significant difference in the incidence of PONV between groups.^{1,5,9} However neither study by Tsai et al.^{1,9} collected data on intraoperative opioid consumption. Intraoperative opioids increase the risk for PONV. The amount of intraoperative opioids subjects received in these two studies may have affected the incidence of PONV. In contrast, Cakmakkaya et al. found the incidence of PONV was significantly lower in the treatment group compared to control.³ There was no difference in the amount of postoperative opioids administered between the groups. Sharami et al. noted that NSAIDs were routinely used at their facility and the incidence of PONV was low⁴.

Analgesic Requirements. The American Pain Society recommends multimodal analgesia for postoperative pain management utilizing a variety of both pharmacological and nonpharmacological interventions.¹² A pulmonary recruitment maneuver could be a potential nonpharmacological intervention to help decrease postoperative pain and reduce analgesic requirements.

Postoperative analgesia regimens varied among studies and included: IV meperidine, diclofenac suppository, and IV tramadol. Postoperative analgesic consumption data was reported in five of the studies. There was no significant difference in analgesic requirements between groups in

three of the studies.^{3,5,9} However, the authors in two of the aforementioned studies did not discuss the use of analgesics intraoperatively, which may have affected postoperative analgesic requirements.

Analgesic requirements were significantly lower in the treatment group compared to control in two of the studies.^{4,5} Sharami et al. utilized a diclofenac suppository for postoperative analgesia followed by IV meperidine if the diclofenac was ineffective⁴. A weakness in the study is they collected data regarding diclofenac consumption, but not meperidine consumption. Furthermore, the external validity may be somewhat limited because NSAIDs were used as first line treatment for postoperative pain, which may vary across practice settings. Liu et al. found IV tramadol use was lower in the treatment group compared to control⁵. Again, external validity may be limited because IV tramadol for postoperative analgesia may not reflect common practice. Regardless of the type of postoperative analgesic regimen, postoperative analgesic consumption was decreased when a pulmonary recruitment maneuver was implemented at the end of surgery in two studies^{4,5}.

Strengths/Limitations. There are strengths and limitations of the studies reviewed that impact the quality of this evidence based practice analysis. General endotracheal anesthesia was the anesthesia technique utilized in each study. Regarding the number of manual pulmonary inflations, five of the six studies implemented five manual pulmonary inflations, while one study implemented two manual pulmonary inflations each held for 5 seconds. This is a strength as the intervention was similar across studies. Pressure of pulmonary inflation varied slightly among studies—four studies used a pressure of 60 cmH₂O while two studies used 40 cmH₂O. Five studies held the last pulmonary inflation for 5 seconds. In both control and treatment groups, the trocar sleeves were fully open to allow CO₂ to escape. Timing of the intervention was consistent and performed at the end of surgery. The degree of Trendelenburg (30°) was the same in each study. Intraabdominal pressure varied only slightly from 12-15 mm Hg among studies and intraabdominal gas flow rates were 1-2 L/min. Five of the six studies utilized a visual analogue scale to measure pain. One study used a numeric rating scale to measure pain. Pain scores were self-reported in one of the six studies.

Induction, maintenance, and emergence varied among studies. Three of the studies did not define the specific anesthetics administered, only that the anesthesia technique was standard and did not vary.^{1,8,9} Furthermore, these studies did not report the amount of intraoperative analgesics subjects received. This is a significant weakness because intraoperative analgesics have an affect on postoperative pain. Three studies described the anesthetic technique and included induction with a hypnotic, fentanyl, and a nondepolarizing neuromuscular blocking agent.³⁻⁵ In one study, fentanyl boluses were repeated “according to clinical needs”³ (1156) but the authors did not report the total amount of intraoperative fentanyl administered. In the remaining two studies, anesthesia practitioners gave a single dose of fentanyl during induction to all subjects.^{4,5} TIVA with propofol and remifentanyl was utilized in one study and there was no significant difference in cumulative remifentanyl doses between groups.⁵

A significant selection bias was present in the articles reviewed, both in demographics and surgical specialty. A total of 571 subjects were included in the studies, of which 534 were female. Furthermore, the majority of subjects (n=495) underwent laparoscopic gynecological

procedures. The remaining procedures were laparoscopic cholecystectomy (n=47) and laparoscopic inguinal hernia repair (n=29). All subjects were relatively healthy according to their ASA physical status of I or II with the exception of one study that included 5 subjects with an ASA physical status of III. Thus, external validity is somewhat limited to a specific population because of the relatively homogenous sample, healthy females undergoing laparoscopic gynecological surgery. This could be perceived as a potential strength or weakness.

Tsai et al.¹ and Tsai et al.⁹ examined the effects of an intraperitoneal normal saline infusion combined with a pulmonary recruitment maneuver, which limits comparison of literature. Tsai et al. compared a group receiving an infusion of normal saline (15-20 mL/kg) in the upper abdominal cavity combined with a pulmonary maneuver to a control group.¹ Tsai et al. also compared a group receiving intraperitoneal normal saline combined with a pulmonary maneuver, however, they included a group that received only a pulmonary recruitment maneuver.⁹ This review does not include data from the group that received intraperitoneal normal saline and a pulmonary maneuver in the Tsai et al.⁹ study. Incisional infiltration of local anesthesia was utilized in one study in both control and treatment groups.

Table 1
Pulmonary Recruitment Maneuver Evidence Matrix

Author	Sample (n) & Population	Variables	Results
Cakmakkaya et al. ³ , 2008	n=100 n=46 control n=54 treatment Elective gynecologic laparoscopic surgery	<u>Dependent</u> Shoulder pain incidence Shoulder pain severity PONV Meperidine consumption <u>Independent</u> 5 manual pulmonary inflations at a pressure of 60cmH ₂ O, 5th held for 5s	<ul style="list-style-type: none"> • Shoulder pain incidence less in treatment group at 12, 24, 36h • Shoulder pain severity less in treatment group at 12, 24, 36h • PONV lower in treatment group • No difference in postoperative analgesic requirements between groups • No difference in pain at discharge between groups
Sharami et al. ⁴ , 2010	n=131 n=64 control n=67 treatment Minor gynecological laparoscopy	<u>Dependent</u> Shoulder pain incidence Shoulder pain intensity Diclofenac consumption <u>Independent</u> 5 manual pulmonary inflations at a pressure of 40cmH ₂ O, 5th held for 5s	<ul style="list-style-type: none"> • Shoulder pain incidence and intensity less in treatment group at 4, 12, 24, 48h • Greater postoperative use of diclofenac in control group • No difference in pain within first 4h between groups

Author	Sample (n) & Population	Variables	Results
Tsai et al. ⁹ , 2011	n=104 n=51 control n=53 treatment Laparoscopic surgery for benign gynecologic lesions	<u>Dependent</u> Shoulder pain incidence Shoulder pain severity Upper abdominal pain Lower abdominal pain PONV Meperidine consumption <u>Independent</u> 5 manual pulmonary inflations at a pressure of 60cmH ₂ O, 5th held for 5s	<ul style="list-style-type: none"> • No difference in shoulder pain incidence or severity between groups • Upper abdominal pain severity and incidence less in treatment group at 12 and 24h • No difference in lower abdominal pain incidence or severity between groups • No difference in PONV or postoperative meperidine consumption between groups
Tsai et al. ¹ , 2013	n=100 n=50 control n=50 treatment Laparoscopic surgery for benign gynecologic lesions	<u>Dependent</u> Shoulder pain incidence Shoulder pain severity Upper abdominal pain Surgical pain PONV Meperidine consumption <u>Independent</u> Normal saline (15-20mL/kg) instilled in upper abdominal cavity followed by 5 manual pulmonary inflations at a pressure of 60cm H ₂ O	<ul style="list-style-type: none"> • Shoulder pain incidence and severity lower in treatment group at 12, 24, 48h • Upper abdominal pain incidence lower in treatment group at 12, 24, 48h • Upper abdominal pain severity lower in treatment group at 12, 24h • No difference in surgical pain incidence or severity between groups • No difference in PONV between groups • No difference in meperidine consumption between groups
Khanna et al. ⁸ , 2013	n=76 n=39 control n=37 treatment Laparoscopic cholecystectomy or transabdominal preperitoneal inguinal hernia repair	<u>Dependent</u> Postoperative pain, includes abdominal and shoulder tip <u>Independent</u> 2 manual pulmonary inflations at a pressure of 60cmH ₂ O, each inflation held for 5s	<ul style="list-style-type: none"> • Pain severity less in treatment group at 12 and 24h

Author	Sample (n) & Population	Variables	Results
Liu et al. ⁵ , 2014	n=60 n=30 control n=30 treatment Diagnostic hysteroscopy and laparoscopy	<u>Dependent</u> Shoulder pain incidence Shoulder pain severity Dynamic pain Static pain PONV Tramadol consumption <u>Independent</u> 5 manual pulmonary inflations at a pressure of 40cmH ₂ O, 5th inflation held for 5s	<ul style="list-style-type: none"> • Lower overall incidence of shoulder pain in treatment group • No difference in R or L shoulder pain between groups • No difference in shoulder pain severity between groups • Dynamic pain decreased in treatment group at 0, 4, 24h • Static pain decreased in treatment group at 0, 2, 24h • No difference in PONV between groups • Tramadol use less in treatment group

Note. All results are statistically significant at $p < 0.05$. Treatment groups received pulmonary recruitment maneuvers for all studies. Control groups did not receive a pulmonary recruitment maneuver.

Conclusions

The reviewed studies shared similar methodologies with minor variations in the pulmonary recruitment maneuver technique and the quantity of data collected. Pulmonary recruitment maneuvers expose patients to a low risk of pneumothorax because of the increased intrapulmonary pressure. However, coughing and sneezing can increase intrapulmonary pressures to 80-130 cmH₂O³ so it is unlikely manual pulmonary inflations with a pressure of 60 cmH₂O would cause a pneumothorax in the absence of underlying pulmonary disease. Pulmonary recruitment maneuvers may be contraindicated if a pneumothorax is suspected or the patient has risk factors for pneumothorax.

The pulmonary recruitment maneuver may decrease cardiac output secondary to decreased venous return caused by increased intrathoracic pressure. However, the Trendelenburg position may offset the decrease in venous return. It may be prudent to avoid this maneuver if the patient has cardiovascular disease and does not tolerate a decrease in venous return. Furthermore, use good clinical judgement and proceed with caution in the hypotensive patient as this maneuver can potentiate hypotension. None of the study subjects in this literature review suffered complications related to the pulmonary recruitment maneuver.

A key limitation in this review is the relative homogeneity of the population examined. The results may only be generalized to females with an ASA physical status of I or II undergoing laparoscopic gynecologic procedures. A pulmonary recruitment maneuver in Trendelenburg position (30°) at the end of laparoscopic surgery appears to be a safe, effective technique that may reduce PONV, postoperative pain, and postoperative analgesic consumption in healthy

females undergoing gynecological surgery. Patients who qualify for pulmonary recruitment are healthy patients undergoing laparoscopic surgery without coexisting cardiopulmonary disease. Despite the homogenous sample of females, the intervention may have an affect on the general surgical population. The proposed explanation of exsufflation of the abdomen is independent of gender. Further research is necessary to support the findings in other patient populations.

Surgeon compliance is required for implementation of the intervention. Anesthesia practitioners are recommended to utilize a multimodal approach to postoperative pain. A pulmonary recruitment maneuver at the end of laparoscopic surgery in healthy patients seems to be a low risk nonpharmacological intervention that may reduce PONV, postoperative pain, and postoperative analgesic requirements.

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Mentor: Shannon Pecka, CRNA, PhD

Editorial

Spread the word!

After more than 14 years since the inaugural issue of the International Student Journal of Nurse Anesthesia (ISJNA) was published it remains relatively unknown among many nurse anesthesia students, faculty, and clinical preceptors. It is even less known among practicing CRNAs. The ISJNA provides a great forum for nurse anesthesia students to combine didactic and clinical effort to develop writing and publication skills. The Journal offers professional development and learning opportunities to students, faculty, preceptors, and practitioners by participating as readers, authors, mentors, reviewers, and/or editors.

For those of you already familiar with the ISJNA, take the opportunity familiarize others with the publication, which includes case reports, evidence based practice analyses, and research abstracts. For students, when you are rotating to clinical sites with students from other programs or attending review courses or other meetings, encourage other students to contribute to the ISJNA. For faculty, when you are attending the Assembly of School Faculty or other meetings attended by faculty from other programs, encourage them to incorporate assignments into the curriculum that can be submitted to the ISJNA. For anyone who knows about the Journal, encourage your co-workers to read and participate in the Journal. Let them know how they can easily access the Journal on the AANA website. Spread the word!

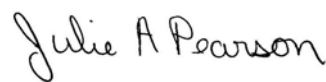
Many thanks to those of you who have contributed over the years as authors, mentors, reviewers, and editors. We would like to recognize and extend our sincere gratitude to Dennis Spence, CRNA, PhD, a 7 year member of the editorial board. Dr. Spence has been promoted to Senior Nurse Executive (Chief Nursing Officer) for Naval Hospital Guantanamo Bay, and elected to the NBCRNA Board of Directors. In light of these added responsibilities Dr. Spence is stepping down from the editorial board, but in keeping with his dedication to the ISJNA he has mentored a colleague to take his place. Please join us in welcoming Ryan L. Nations, CRNA, PhD of Uniformed Services University!

We hope you enjoyed this issue of the ISJNA – remember to spread the word!

Sincerely,



Vicki C. Coopmans, CRNA, PhD
Editor



Julie A. Pearson, CRNA, PhD
Associate 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 °C, pressure in mm Hg or cm H₂O.
 - c. In general, first use of pulmonary/respiratory abbreviations should be expanded, with the following exceptions: O₂, CO₂, PCO₂, PaCO₂, PO₂, PaO₂. Please use SpO₂ 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 one 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 patient 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 patient was intubated and put on a ventilator.* “The trachea was intubated and respiration was controlled by a mechanical ventilator.
 - d. *The patient had been on Motrin for three days.* “The patient had taken ibuprofen for three days.”
 - e. Avoid the term “MAC” when referring to a sedation technique - the term sedation (light, moderate, heavy, unconscious) sedation may be used. Since all anesthesia administration is monitored, the editors prefer to use specific pharmacology terminology rather than reimbursement terminology.
6. Use the words “anesthesia professionals” or “anesthesia practitioners” when discussing all persons who administer anesthesia (avoid the reimbursement term “anesthesia providers”)
7. References
 - a. Again, the **AMA Manual of Style** must be adhered to for reference formatting.
 - b. All should be within the past 8 years, except for seminal works essential to the topic being presented.
 - c. Primary sources are preferred.
 - d. All items cited must be from peer-reviewed sources – use of internet sources must be carefully considered in this regard.
 - e. Numbering should be positioned at the one-inch margin – text should begin at 1.25”.
8. See each item for additional information.
9. **Heading** for each item (Case Report, Abstract, EBPA Report) must adhere to the following format:

Title (bold, centered, 70 characters or less)

[space]

Author Name (centered, include academic credentials only)

Name of Nurse Anesthesia Program (centered)

[space]

Anticipated date of graduation (italics, centered, will be removed prior to publication)

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

[space, left-justify from this point forward]

Keywords: (‘Keywords:’ in bold, followed by keywords (normal font) that can be used to identify the report in an internet search.)

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>

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

Journals

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

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

Journal, 6 or fewer authors:

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

Journal, more than 6 authors:

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

Texts

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

Text:

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

Chapter from a text:

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

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

Electronic references

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

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

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

Examples:

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

Gupta A, Aggarwal N, Sharma D. Ultrasound guided ilioinguinal block. *The Internet Journal of Anesthesiology*. 2011;29(1).

http://www.ispub.com/journal/the_internet_journal_of_anesthesiology/volume_29_number_1/article/ultrasound-guided-ilioinguinal-block.html. Accessed August 1, 2011.

ACADEMIC INTEGRITY

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

"Plagiarism is defined as the act of passing off as one's own the ideas, writings, or statements of another. Any act of plagiarism is a serious breach of academic standards, and is considered an offense against the University subject to disciplinary action. Any quotation from another source, whether written, spoken, or electronic, must be bound by quotation marks and properly cited. Any paraphrase (a recapitulation of another source's statement or idea in one's

own words) or summary (a more concise restatement of another's ideas) must be properly cited.”
http://grad.georgetown.edu/pages/reg_7.cfm

HOW TO SUBMIT AN ITEM

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

REVIEW AND PUBLICATION

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

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

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

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

PHOTOS

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

Vicki C. Coopmans, CRNA, PhD
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.
- Reference numbers are sequenced beginning with one and superscripted.
- References are from anesthesia and other current primary source literature.
- All inclusive pages are cited, texts as well as journals.
- Journal titles are abbreviated as they appear in the PubMed Journals Database.
- Number of references adheres to specific item guidelines.
- Internet sources are currently accessible, reputable, and peer reviewed.

Transmission

- The article is sent as a attachment to **INTSJNA@AOL.COM**
- The file name is correctly formatted (e.g. PedsPain_Smyth_GU_Pearson_5.19.09)
- It is submitted by the mentor with cc to the student author
- The words "Submission to Student Journal" are in the subject heading.