Volume 11 Number 3 Fall 2012

The International Student Journal of Nurse Anesthesia

TOPICS IN THIS ISSUE

INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA
Vol. 11 No. 3 Fall 2012

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Front Cover:

Jack Davenport, BSN, a graduate student enrolled in the University of Southern California Program of Nurse Anesthesia, mask ventilates his patient in preparation for laryngoscopy and intubation as clinical instructor Robert Olson, CRNA, MS observes.

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Case Reports

Blood on the Spine: Neuraxial Anesthetic Considerations

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Keywords: neuraxial anesthesia, spinal anesthesia, epidural hematoma, thrombotic therapy, anticoagulant therapy

The number of Americans on anticoagulant and antiplatelet therapies for atrial fibrillation is estimated at 2.3 million and expected to increase to 4 million by the year 2030.[1] The rate of neuropathy following either spinal or epidural anesthesia occurs approximately 3.78 times in 10,000 neuraxial procedures.[2] There is a 33% higher incidence of hemorrhage following neuraxial anesthesia with hypercoagulable states, especially if two or more antithrombotic agents are being used at once.[3-5] If a patient develops a spinal or epidural hematoma, there is an increased risk of developing cauda equina syndrome, neuropathies, paralysis, or death.[3,6]

Case report

A 59 year old Caucasian male presented for a left below the knee amputation due to non-healing osteomyelitis. The patient was 180 cm in height, weighed 147 kg, and had no known drug allergies. Based on the American Society of Anesthesiologists guidelines, this patient was designated a physical status classification of IV. The patient's medical history included diabetes mellitus, coronary artery disease with drug eluting stent placement, symptomatic chest pain with new onset pericarditis, moderate pulmonary hypertension, chronic obstructive pulmonary disease (COPD), bilateral pleural effusions, obstructive sleep apnea (OSA), chronic renal insufficiency with new onset acute renal failure and hemodialysis as well as multiple other co-morbidities. The patient's airway assessment included a Mallampati score of 3, a thyromental distance of 5 cm, oral aperture of 4 centimeters, and neck full range of motion. He had a full beard, and natural dentition. The patient's medications included: fluticasone propionate, tiotropium bromide inhalation powder, albuterol, insulin glargine, insulin aspart, glipizide, metoprolol, furosemide, amlodipine, losartan, labetalol, gabapentin, aspirin, metoclopramide, omeprazole, sitagliptin, lisinopril, ezetimibe, iron polysaccharide, indapamide, and fluticasone. Clopidogrel was administered the morning of surgery.

Standard monitors were applied to the patient, O_2 was administered via face mask, and an arterial line was placed in the regional anesthesia holding room. The patient received a lumbar 2-3 interspace spinal anesthetic with the injection of 0.5% isobaric bupivacaine 15.5 mg, epinephrine 200 mcg, and fentanyl 20 mcg. Cerebral spinal fluid was obtained following the single insertion of a 25 gauge Whitacre spinal needle. No evidence of blood was detected during the procedure. Absent detection of pain and motor activity in the lower extremities verified spinal anesthetic efficacy. The patient was transported to the operating room and positioned supine on the table. Standard monitoring modalities were applied. The arterial line systolic blood pressure correlated within 10 mmHg of the non-invasive blood pressure cuff measurement Cefazolin 2 gm was administered intravenously. The patient was draped for surgery and a tourniquet was

placed on the thigh of the left lower extremity. The patient's vital signs remained within 20% of the preoperative measurements throughout the surgery. When the procedure was completed, a negative pressure wound therapy dressing was applied. The patient was transported to the recovery room. Sensory and motor function returned to baseline in the patient's lower extremities and he was able to return to his hospital room with adequate analgesia after 160 minutes of postoperative monitoring.

Discussion

Neuraxial anesthesia is the administration of a local anesthetic or analgesic drug injected in either the subarachnoid or epidural space. Neuraxial techniques have been attributed to improving patient outcome following surgery and reducing patient mortality and major morbidity.[7]An epidural hematoma is the collection of blood outside the dura mater of the spinal cord. An epidural puncture that causes blood to be aspirated is referred to as a bloody tap and increases the chance of hemorrhage, regardless of the patient's anticoagulant status.[4] Consideration of neuraxial anesthetic risks, benefits, and the anesthesia practitioner's skill should be weighed against other anesthetic techniques.

Patient management recommendations are based on expert opinion and case reports, rather than case studies, since neuraxial complications are rare.[4,5] Epidural hematomas occur an estimated 1:150,000 in patients with normal coagulation hemostasis, which makes performing random controlled trials difficult.[2,7] The increase in incidence of hematomas vary depending on the antithrombotic medication and number of anticoagulants used.[2] New antiplatelet and anticoagulant therapies are being introduced into clinical practice and have varying therapeutic ranges, duration of actions and reversibility.[1,2] Age, gender, spinal anatomy, pregnancy and type of surgery also influence the risk of developing neuraxial complications.[2]

This patient's history of COPD, obstructive sleep apnea (OSA), bilateral pleural effusions and a new onset respiratory infection increased his risk of remaining intubated postoperatively and further compromising his respiratory function. The analgesia and akinesis of a spinal anesthetic provides optimal surgical conditions for this procedure and attenuates the tourniquet pain. The patient had tolerated receiving regional anesthesia in the past without the need of sedation. By limiting the number and types of medications administered to the patient we could decrease the possibility of an adverse cardiac or respiratory event perioperatively. The patient had a pericardial effusion and had been experiencing chest pain at rest. His troponin levels were normal and a 12-lead ECG remained unchanged from prior studies. The cardiologist instructed the patient to remain on his clopidogrel to prevent coronary stent occlusion. The ventilation and intubation of this patient was predicted to be challenging. The patient had a full beard, OSA and obesity which could make ventilation difficult. The patient was likely to desaturate rapidly due to his respiratory infection, bilateral pleural effusions and COPD. A fiberoptic scope and difficult intubation equipment was in the operating room should the patient have required intubation. The clinical judgment of the anesthesia team was to proceed with neuraxial anesthesia following the complete disclosure of risks to the patient and the placement of an arterial line.

The patient had an increased risk for an epidural hemorrhage because of the

clopidogrel, decreased renal clearance of the medication and iron deficiency anemia. Clopidogrel is an antiplatelet agent which inhibits the adenosine disphosphate (ADP) induced pathway in the platelet activation cascade and thus prevents platelets from aggregating. Fortunately, the patient did not have a history of a bleeding disorder, active signs of bleeding nor abnormal spinal anatomy. An epidural hematoma was of the greatest concern in this patient because of the epidural venous plexus which can be injured during the spinal anesthetic placement.[8] Subarachnoid hematomas are more rare because the cerebral spinal fluid dilutes the blood collection in the subarachnoid space, which does not contain major blood vessels.[8] When a subarachnoid hematoma does occur it is thought to be caused by puncture of the radicular vessels found along the nerve roots.[8] An early sign of an epidural hemorrhage may include severe back pain in the region of the puncture site.[2] The patient can have spinal cord compression that can cause immediate or delayed motor and sensory deficits which may lead to dysfunction of the bowel or bladder.[2,3] Magnetic resonance imagining is the gold standard for diagnosing an epidural hematoma and it should be performed within 4 hours if neurological symptoms have not resolved.[2] It is best for a laminectomy and hematoma evacuation to be performed within 8 to 12 hours of the onset of symptoms for the best chance at neurological recovery.[2,3] The spontaneous resolution of an epidural hematoma is rare.[3] Diagnosing an epidural hematoma in this patient could be challenging due to his history of diabetes, lower extremity neuropathy and because a urinary catheter was placed intraoperatively.

Because of the epidural hematoma risk, the recovery room nurses monitored the patient until sensation and motor control returned to the patient's lower extremities. Fortunately, this patient did not develop an epidural hematoma postoperatively. Thorough preoperative assessment and consideration of the benefits versus the risks of administering neuraxial anesthesia is essential in maintaining the safety of patients. If possible, the discontinuation of antithrombotic therapy according to the American Society of Regional Anesthesia guidelines is recommended to further minimize the risk of a neuraxial hematoma.[7] With the increased use of NSAIDs, herbal remedies and antithrombotic therapies, the anesthesia provider must have a sound understanding of the pharmacokinetics of these medications in order to minimize the risk of neuraxial complications in these patient populations.

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Anesthetic Management of a Pregnant Patient for Non-Obstetric Surgery

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Keywords: non-obstetric surgery, obstetric anesthesia, parturient, pregnancy, pregnant.

Significant consideration is required when planning the anesthetic management of a pregnant patient undergoing non-obstetric surgery. In developed countries, approximately 0.75%-2% of pregnant women will undergo anesthesia for non-obstetric related surgery.[1] It is estimated that 42% of these surgeries occur in the first trimester, 35% in the second trimester, and 23% in the third trimester.[2] Consequently, to ensure anesthetic safety for the mother and fetus, it is paramount to be knowledgeable in the physiological and pharmacological changes that occur starting in the first trimester of pregnancy.

Case Report

A 17 year old female presented for an exploratory laparotomy with right ovarian cystectomy after being diagnosed with an 18 cm adnexal mass. The patient was 66 kilograms in weight and 154 centimeters in height, was positive for an intrauterine pregnancy at 15 weeks (second trimester), and had no known allergies. The patient denied any symptoms from her adnexal mass, such as pain or discomfort, and had no significant past medical, social, or surgical history.

The patient's medications consisted of a daily pre-natal vitamin. Laboratory values of note included an elevated white blood cell count of 11,700 and hemoglobin and hematocrit of 10.7 g/dL and 30.6%, respectively. Abnormally elevated levels of carcinoembryonic antigen at 16 ng/mL and cancer antigen at 173 U/ml were also noted. All other laboratory values were within normal limits. Ultrasound showed a normal fetal heart rate (FHR) of 145 beats/min. Physical assessment was unremarkable for any incongruities as was the airway exam.

Preoperative medications included intravenous (IV) metoclopramide 10 mg and famotidine 20 mg. Upon transfer to the operating room table, the patient was placed in the left lateral decubitus position and preoxygenated with 100% oxygen for 5 min while standard monitors were applied. A rapid sequence induction with cricoid pressure was performed and consisted of fentanyl 100 mcg, lidocaine 70 mg, propofol 100 mg, and succinylcholine 60 mg. A grade I view of the vocal cords was obtained

utilizing a GlideScope (Verathon Inc., Bothell, WA) video laryngoscope. The trachea was intubated with a size 7.0 endotracheal tube without trauma or difficulty. Positive end-tidal CO_2 and bilateral breath sounds were confirmed. Ventilation was accomplished with a tidal volume of 475 ml and a ventilatory rate of 12 and general anesthesia was maintained with sevoflurane 2.5% inspired concentration within a mixture of oxygen and air at 1L/min each. After the return of train-of-four twitches via a peripheral nerve stimulator, vecuronium 5 mg was administered followed by cefoxitin 1 gram. A bispectral index monitor (BIS) (Covidien, Boulder, CO), esophageal temperature probe, warming blanket, and IV fluid warmer were implemented followed by the insertion of a second 18 gauge peripheral IV catheter. All pressure points were padded and surgery ensued.

Anesthetic depth was maintained to a BIS reading of 40-50. Neuromuscular blockade was maintained using vecuronium titrated to a peripheral nerve stimulator result of 1-2 twitches out of 4. The surgical procedure lasted three hours and was uneventful. The mean arterial pressure was maintained at ≥ 60 mmHg in order to maintain organ and placental perfusion. Neuromuscular blockade was antagonized at the end of the surgical procedure with emergence and extubation being unremarkable for any significant events. Postoperative evaluation the following day revealed no anesthetic complications and the patient was discharged on postoperative day two.

Discussion

Anesthetic assessment of a parturient undergoing non-obstetric surgery should always include a pre- and post-operative ultrasound assessment of the fetus. It is important for the obstetrician to establish a baseline examination to determine if any fetal injuries occurred during the surgical procedure. The assessments in this case were normal.

Competent management of respiratory physiological changes in parturients is paramount as these patients are extremely susceptible to hypoxemia and rapid desaturation during periods of apnea.[3] Minute ventilation increases by 45-70% compared to prepregnant values at full term to meet an increased demand for oxygen and functional residual capacity is decreased by 20% due to restrictive effects of the gravid uterus.[4,5] Airway changes in the parturient are complicated by anatomical changes consisting of weight gain, airway edema, and increased vascularization of the upper airway.[1] Since failed intubation is the leading cause of maternal death attributed to anesthesia for cesarean delivery, a GlideScope (Verathon, Bothell, WA) was utilized for optimal visualization and efficiency during intubation.[6]

Cardiovascular changes include a gradual increase in cardiac output starting at 8 weeks gestation with a maximal increase of 50% by the end of the second trimester.[4] Aortocaval compression from the gravid uterus is a major concern and can result in a significant reduction in preload which consequently can lead to decreased cardiac output and decreased placental perfusion. Aortocaval compression is apparent in the supine position starting at the second trimester and is relieved by positioning the patient in the left lateral decubitus position, which was performed before induction and maintained throughout the surgical procedure.

Gastrointestinal changes include a reduction in the barrier pressures of the lower

esophageal sphincter from 15 weeks onward which predisposes the patient to aspiration.[5] Due to this, an aspiration prophylaxis regime congruent with literature was given to our patient which consisted of an H_2 antagonist (famotidine) and a pro-kinetic (metoproclamide) along with the use of cricoid pressure during rapid sequence induction.[3,4]

The teratogenicity of anesthetic agents is of primary concern during surgery for parturients. It has been shown that both inhaled and intravenous anesthetic medications can affect DNA synthesis, cell signaling, and mitosis depending on the dose, route, and timing of exposure in relation to fetal development.[1] The most susceptible period is during the third and eighth weeks of gestation when organogenesis occurs.[2] Since our patient was in the fifteenth gestational week, she was considered safe from this critical period. In addition, nitrous oxide has been shown to inhibit methionine synthetase which can affect DNA synthesis in the fetus and has also been shown as a teratogen in rodent studies.[2] Consequently, nitrous oxide has been associated with unwanted outcomes such as miscarriage.[2] Animal studies show a correlation between the use of benzodiazepines and cleft palate but the association in humans is controversial as a single dose has not been associated with teratogenicity.[1] Ketamine is known to increase uterine tone which decreases blood supply to the placenta and can lead to fetal asphyxia.[2] In this case, the use of these agents was avoided.

Although most anesthetic medications are known teratogens, several human studies show that the use of these agents is safe when used in the appropriate dose, route, and timing of administration. The largest known retrospective study of surgical and anesthetic exposure to inhaled anesthetics for appendectomy during pregnancy was conducted by Mazze and Källén[7]. The study involved 5405 subjects and concluded that there was no difference between non-surgical and surgical parturients in regards to the overall incidence of stillbirths and congenital abnormalities. Cohen-Karem et al[8] concluded that with modern surgical and anesthetic techniques, the risk of maternal death is extremely low and occurs in less than 1 in 10,000 patients. Cohen-Karem et al[8] also concluded that the occurrence of spontaneous abortion is extremely low and that non-obstetric surgical procedures do not increase the risk of fetal birth defects.[8]

Light general anesthesia is associated with a catecholamine surge which leads to impaired uteroplacental perfusion due to vascular constriction.[4] This can lead to fetal morbidity and/or mortality so intraoperative anesthetic depth was titrated to a BIS reading between 40 and 50. Also, a mean arterial pressure of $\geq$60 mmHg was maintained using IV fluid administration along with the use of both the direct and indirect vasoactive agents phenylephrine and ephedrine, respectively. Older studies favored the use of ephedrine; however, more recent studies show superior efficacy of phenylephrine as it produces better fetal acid-base balance.[5]

Fetal heart rate monitoring is possible once the fetus is 18-22 weeks gestation with FHR variability is detectable at 25 weeks.[2] Since the patient was at 15 weeks gestation, perioperative FHR monitoring was not indicated and was not performed outside of the pre- and post-operative ultrasound examinations.

It has been shown that parturients undergoing non-obstetric surgery can have robust outcomes with proper anesthetic

management. Competency of the anesthesia practitioner to recognize both the physical and pharmacological changes in the parturient is indispensible. Consequently, our plan for an efficacious and safe anesthetic focused on a safe intubation, maintenance of uteroplacental perfusion, avoidance of detrimental agents, and a proper fetal examination.

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Mentor: Terrie Norris, CRNA, EdD

Anesthesia for Arrhythmia Ablations

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Keywords: radiofrequency catheter ablation, tachyarrhythmia, electrophysiology, arrhythmogenicity

In the United States, cardiac arrhythmias account for greater than 881,000 hospitalizations, 40,700 deaths per year, and have an estimated prevalence of 14.4 million patients.[1] With a shift in emphasis from pharmacologic to non-pharmacologic therapy of tachyarrhythmias, anesthesia professionals are seeing a considerable increase in the volume of radiofrequency catheter ablations being performed in cardiology suites. Patients treated in electrophysiology (EP) labs have multiple significant comorbidities requiring full monitoring and anesthesia professional vigilance.[2] Continued growth in EP lab procedures will require an increased presence of anesthesia personnel to provide services. In addition to providing a comfortable and safe anesthetic for the patient, anesthesia professionals must acknowledge the effects of their pharmacologic plan to ensure hemodynamic stability while allowing the induction of abnormal heart rhythms during catheter ablations.

Case Report:

This case involved an 81-year-old, 51kg female with a history of paroxysmal atrial tachycardia (PAT), palpitations, hypertension, hyperlipidemia, coronary artery disease, status post 3-vessel coronary artery bypass graft (CABG), and a 10 pack-year smoking history 20 years prior. She was referred to an electrophysiologist with complaints of frequent palpitations, dizziness, and nervousness. A 24-hour Holter monitor revealed frequent premature ventricular contractions (PVC) and PAT. A 2-D echocardiograph indicated she had mild to moderate mitral regurgitation, abnormal diastolic relaxation, and a left ventricular ejection fraction of 64%. A basic metabolic panel and complete blood count showed normal values. The patient had been prescribed diltiazem extended-release 120 mg by mouth once daily but stated she was not taking the medication. She reported having a long history of medication intolerances and wished to proceed with a non-pharmacological approach. She was therefore consented for a PVC/PAT ablation.

On the day of her procedure, the patient was identified in the electrophysiology (EP) holding room. The anesthesia plan of care was discussed with the patient and her daughter. Once all questions were answered, a 20-gauge peripheral intravenous catheter was placed in the patient's left hand. The patient was then assisted to the EP lab table, standard monitors were applied, and defibrillator pads were placed on her chest. The patient was noted to be in sinus bradycardia, heart rate 58, with frequent PVCs and premature atrial contractions (PAC). She was preoxygenated with 100% oxygen. General anesthesia was induced with 120 mg of propofol. Successful mask ventilation was confirmed and muscle relaxation was achieved with 50 mg of rocuronium. Direct laryngoscopy with a Mac 3 blade provided a grade I glottic view and a 6.5 mm cuffed oral endotracheal tube (ETT) was inserted between the vocal cords, placement was confirmed, and mechanical ventilation was initiated. Phenylephrine 120 mcg was administered after induction and a total of six bolus doses of 120 mcg aliquots were given as needed throughout the case to maintain a mean arterial pressure of 80 mmHg. Anesthesia was maintained with 0.7% isoflurane. A bispectral index (BIS) monitor was placed on the patient and maintained between 50-60 throughout the case. A 20 gauge right radial arterial line was placed for continuous monitoring of blood pressure. One hour after starting the case the spontaneous PVCs and PACs subsided and the physician was unable to stimulate the arrhythmias. After a discussion with the EP physician, isoflurane was discontinued and total intravenous anesthesia (TIVA) was initiated with infusions of propofol 40 mcg/kg/min and remifentanil 0.05 mcg/kg/min. Within 45 minutes of discontinuing the volatile agent, the physician was able to stimulate and ablate the patient's arrhythmias.

The ablation was successfully completed, neuromuscular blockade was antagonized with neostigmine 3 mg and glycopyrrolate 0.4 mg, and the propofol and remifentanil infusions were discontinued. The oropharynx was suctioned, a return of spontaneous respirations was noted, and when the patient was able to follow commands, she was extubated with positive pressure. The patient was transported to the post-anesthesia care unit (PACU) with full monitoring and supplemental oxygen. Overnight telemetry monitoring revealed a marked reduction in the number of PVCs and no PACs were noted. The patient was discharged home the next day after a chest

x-ray and echocardiograph confirmed no complications from the procedure.

Discussion:

The goal of catheter ablation therapy is to stimulate an arrhythmia, identify the site of origin, and deliver radiofrequency energy to destroy it.[3] In this situation, the goals of anesthesia management must be altered to allow for the induction of arrhythmias and to avoid administering medications that may interfere with the cardiac conduction system.

A current literature review revealed a differing of opinions regarding the most appropriate anesthetic plan for patients undergoing an EP study or catheter ablation. Most publications contain information gained from studies assessing the effects of anesthetic agents on children and animals. The intent of this article is to highlight data regarding the use of volatile agents, opioid infusions, and propofol infusions in the adult population undergoing EP studies or catheter ablations.

A study assessing the effects of volatile anesthetics on the normal atrioventricular (AV) conduction system and accessory pathways in Wolff-Parkinson White Syndrome suggest that because they increase refractoriness and abolish conduction within the accessory and atrioventricular pathways, volatile anesthetics are not advisable as their effects would result in a difficult or unsuccessful ablation.[4] In their article comparing the EP effects of propofol and isoflurane-based anesthetics in children undergoing catheter ablation for supraventricular tachycardia (SVT), Erb et al. referenced earlier studies which reinforce the notion that mapping of and inducing SVT is very difficult when using isoflurane.[5] Contrastingly, in their own findings, Erb et al. noted that isoflurane and propofol were equally suitable anesthetic agents for catheter ablation specifically when drug administration was titrated to maintain the (BIS) score between 50-60.[5] Concerning epicardial ablation of ventricular tachycardia, Mandel et al. found the disadvantage of general anesthesia using volatile agents is the potential to suppress re-entrant ventricular tachyarrhythmias. In addition, they noted general anesthesia exacerbates the hypotension associated with arrhythmia induction.[6]

Another possible consideration could be the use of an opioid infusion. A study published in 2010 assessing the effects of remifentanil in children undergoing catheter ablation for SVT noted remifentanil would not be the optimal agent, especially in instances when AV nodal function needed to be preserved and closely monitored, as it slows both sinus and AV nodal function.[7] Mandel et al. do agree remifentanil is known to produce bradycardia but report it has comparatively little effect on arrythmogenicity. Additionally, they noted that in their experience, remifentanil has not interfered with the ability to induce ventricular tachyarrhythmias proving its usefulness during the ablation of SVT.[6] Likewise, in studying the utilization of opioid infusions, Sharpe et al. found that a sufentanil infusion, along with benzodiazepines, would provide hemodynamic stability and unimpeded physiologic induction of the aberrant pathway.[4]

A final anesthetic option for a patient undergoing ablation therapy includes the use of a propofol infusion. A case report written by Nora et al. described the use of a total intravenous anesthesia with target-controlled infusions of remifentanil and propofol for ablation of atrial fibrillation.[8] They noted in their research that while enflurane, halothane, and isoflurane seem to increase

the refractory period of anomalous pathways, as well as normal atrioventricular conduction pathways, intravenous anesthetics, such as sufentanil, alfentanil, midazolam, and propofol did not seem to change the electrophysiological properties of either pathway.[8] Using target-controlled infusions, propofol was titrated to maintain a BIS between 40 and 50 and remifentanil was titrated to maintain a maximal 20% variation in mean arterial pressure.[8] They suggest that not only does this method of anesthesia avoid interfering with electrical pathways, but also, by being able to titrate down the amount of medication given, may reduce cost, allow a fast awakening to facilitate immediate neurologic evaluation, and facilitate the maintenance of a satisfactory level of anesthesia.[8]

Much consideration should be given to the balance between the need to maintain hemodynamic stability and the ability of the electrophysiologist to safely and effectively achieve a therapeutic result.[5] In the case report mentioned here, isoflurane did not allow for the stimulation of arrhythmias so an alternative plan was formulated to accomplish all goals for the patient's care. It becomes vital for the anesthesia professional to maintain vigilance in assessing the effects of their selected anesthetic plan and demonstrate flexibility in altering the plan as needed to ensure patient comfort and safety. Further studies comparing the effects of volatile agents, opioid infusions, and propofol infusions on adults with arrhythmias would provide a greater understanding and perhaps reveal practices that are conducive to both the physicians' goal of initiating arrhythmias and the anesthesia providers' goal to ensure hemodynamic stability.

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Hemorrhagic Hereditary Telangiectasia and Arteriovenous Malformations

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Keywords: anesthesia, pulmonary arteriovenous malformation, hemorrhagic hereditary telangiectasia, Osler-Weber-Rendu disorder

Hemorrhagic hereditary telangiectasia (HHT) is an autosomal dominant disorder that affects approximately 1 out of 5,000 to 8,000 people.[1,2] Hemorrhagic hereditary telangiectasia, also known as Osler-Weber-Rendu disorder, is related to endoglin and/or ALK-1 gene mutations, which hinder the production of proteins involved in vascular endothelial cells.[2] Manifestations of HHT include telangiectasias of the mucous membrane, epistaxis, gastrointestinal (GI) bleeding, and arteriovenous malformations (AVMs).[1,2] Pulmonary AVMs (PAVMs) can be especially problematic and require the anesthesia professional to understand the various PAVM-related anesthesia implications.

Case Report

A 48 year-old, 45 kg female presented for embolization of a right lung, middle lobe PAVM. Medical history included chronic abdominal pain, gastroesophageal reflux disease, and suspected HHT. At the time of the procedure endoglin and ALK-1 genetic testing was pending to confirm HHT diagnosis. Past surgical history included a left lingular PAVM embolization nine months prior to the current hospital visit. Due to the patient's complaint of continued fatigue and dyspnea on exertion, embolization of the right middle PAVM was scheduled.

Physical assessment, vital signs, and EKG findings were all within normal limits. Computed tomography (CT) scan revealed the right lower lobe PAVM as measuring 1.3 x 0.6 cm. Pulse oximetry on room air was 98%, and the patient denied any shortness of breath (SOB). Prior to the left lingular PAVM embolization, the arterial blood gas (ABG) showed a shunt fraction of 18%; the post-procedure shunt-fraction was 13%. A general anesthetic with endotracheal intubation was planned. A thorough airway assessment revealed a Mallampati I airway with no visible telangiectasias in the oropharynx. A size 37F double-lumen endotracheal tube (ETT) and bronchial blocker were available. Preoperative medications included metoclopramide 10 mg PO, famotidine 30 mg PO, and sodium bicitra 30 mL. Fentanyl 50 mcg IV was given while in transit to the interventional radiology suite.

After 5 min of denitrogenation, general anesthesia was induced via rapid sequence with fentanyl 100 mcg, lidocaine 50 mg, propofol 100 mg, and rocuronium 50 mg. Under direct laryngoscopy, the vocal cords were visualized and the endotracheal tube size 7.0 mm ID was inserted into the

trachea. Endotracheal tube placement was verified. Anesthesia was maintained with 2% sevoflurane with oxygen 1 L/min and air 1 L/min. An invasive arterial catheter was placed in the left radial artery and a large bore intravenous catheter was placed in the right forearm. No intraoperative antibiotics were ordered by the surgeon.

The patient's vital signs remained stable throughout the case. Fentanyl 50 mcg was given upon incision, and embolization of the PAVM progressed without incident. At the conclusion of the surgery 2 hours later, train-of-four (TOF) 4/4 was observed, and neuromuscular blockade was antagonized with neostigmine 3 mg and glycopyrrolate 0.4 mg. The patient emerged from the anesthesia with tidal volumes of 300-400 mL. Standard extubation criteria were met before the endotracheal tube was removed without difficulty.

Vital signs were within normal limits, and the patient denied pain and dyspnea after waking. The patient was then transferred to the recovery room, and she was discharged home in stable condition later that day.

Discussion

Clinical diagnosis of HHT is commonly established using the Curacao criteria. Definitive HHT diagnosis is made if at least three of the four Curacao criteria are present. These criteria include: (1) spontaneous and recurrent epistaxis; (2) telangiectasia at typical sites e.g. oral mucosa or tongue, lips, fingertips; (3) visceral involvement e.g. pulmonary, hepatic, or cerebral AVMs; (4) a first-degree family member affected with the aforementioned condition. Visceral AVMs are very rare within the general population, therefore clinicians tend to presume the HHT diagnosis even in the absence of the other clinical criteria or genetic tests (e.g. epistaxis[1,5]). Therefore, the anesthesia team created a plan for this patient based on a probable HHT diagnosis.

Screening tests for PAVMs include transthoracic contrast echocardiography with agitated saline and CT scans.[1] It is important for the anesthesia professional to consider the results of these diagnostic studies in order to identify any other PAVMs and anticipate related complications. AVMs in other areas e.g. brain, GI system, liver, may be revealed, and therefore help the anesthesia professional determine if certain interventions should be done or avoided (e.g. avoiding orogastric tube placement secondary to GI AVM).

Because HHT can also contribute to telangiectasias or small dilated blood vessels within the mucous membranes, a thorough airway assessment is especially critical in order to decrease the risk of airway trauma and bleeding.[5] Telangiectasias have been found on the epiglottis, larynx, trachea, and bronchi.[5] Upon clinical examination, the patient in question did not have any noticeable telangiectasias within the oropharynx, and a single laryngoscopy was performed in order to decrease the risk of airway trauma.

Embolization is the most efficacious treatment of PAVMs.[3,6] Thoracotomy and lung resection are alternative treatment options for PAVMs.[5] Although rare, a potential complication of embolization is rupture and bleeding of the AVM. Because of the risk of rupture, preparations were made for an emergency thoracotomy and a 37F double-lumen tube and bronchial blocker were immediately available.[4] However, since PAVM-related hemorrhage during an embolization is rare blood was not typed or crossmatched. Although the literature does not specifically suggest

performing a type-and-screen or type-and-cross for PAVM embolization procedures, dialogue between the anesthesia team and interventional radiologist may be prudent in determining if a patient is considered at higher risk for bleeding or rupture complications.

PAVMs can contribute to right-to-left intrapulmonary shunting.[5] Depending on the degree of shunting, uptake of volatile anesthetics may be prolonged and induction via intravenous agents may be accelerated.[4] The IV induction of this patient did not seem especially rapid. This may be due to the small shunt fraction in this patient. Additionally, the literature does not provide recommendations favoring the use of specific kinds of medications e.g. rocuronium vs. succinylcholine.

Because no capillary bed exists within a PAVM, the pulmonary capillary loses its "filtering" capacity. Thus, emboli or thrombi from the vein can simply travel to the arterial side, and place patients with PAVMs at a higher risk for paradoxical emboli of air, bacteria, and plaque to the brain and other vital organs.[1,3] Neurological complications including stroke and cerebral abscess may therefore occur.[1] Thus, a primary treatment goal of PAVM is to prevent these neurological complications. Because these complications appear to be related to PAVMs with feeding arteries that are 3 mm or greater in diameter, it is at this size that standard treatment for PAVMs begin.[3,5] Using an in-line IV filter and ensuring that no bubbles are present within the IV tubing are also recommended.[1,3] Prophylactic antibiotics, while not typically administered for interventional radiological procedures, may be beneficial in decreasing the risk of cerebral infection and abscess secondary to the potential translocation of bacteria.[1]

While definite recommendations are not available, invasive monitoring via an arterial line may be warranted due to the hemodynamic changes expected with induction and emergence from general anesthesia. Considerations like the size of the AVM and its potential for rupture would be specific reasons for choosing invasive monitoring. For example, larger and untreated AVMs may be at higher risk for rupture and therefore hemodynamic instability is to be avoided.[5]

An arterial line may also be valuable for ABG assessment. One case study described a patient with known bilateral PAVMs who underwent general anesthesia. This patient experienced mixed venous desaturation and subsequent arterial desaturation that was attributed to increased pulmonary vascular resistance due to positive pressure ventilation.[7] Adjusting ventilation settings to improve oxygen saturation would therefore be facilitated with ABG assessments.

Although the literature does not recommend specific ventilation modes to use in patients with PAVMs, positive pressure ventilation via general anesthesia may increase the right-to-left shunt and contribute to desaturation.[7,8] One case study described a clear relationship between removal of positive pressure ventilation and improvement in oxygenation. The authors also observed that adding PEEP may have also increased shunting and subsequent desaturation.[8]

Regarding management of this patient, placement of an in-line IV filter would have been an additional safety measure for minimizing the risk of paradoxical air emboli. Second, discussion with the interventional radiologist regarding the benefits of antibiotic administration in decreasing the risk of cerebral abscess may

have been valuable. In conclusion, the anesthesia practitioner must consider numerous factors when formulating a plan of care for a patient with HHT, tailoring it to address the varied and problematic manifestations of the disease.

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Analgesia for a Parturient with Chronic Regional Pain Syndrome

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Keywords: epidural analgesia, spinal analgesia, regional anesthesia, neuraxial analgesia, chronic regional pain syndrome, remifentanil, and patient controlled analgesia, parturient.

Each year in the United States, over 4 million vaginal deliveries are managed in a hospital setting.[1] It is estimated that 61% of these deliveries will be assisted by the placement of epidural or spinal anesthesia.[1] The pain associated with labor can be severe and has been compared to the pain experienced from chronic regional pain syndrome (CRPS) or even digital amputation.[2] Patients in which regional anesthesia is contraindicated can present a unique challenge to the anesthesia practitioner in the delivery of labor analgesia.

Case Report

A 40-year-old Caucasian, gravida 6 para 3 at 40 weeks gestation, presented to triage with complaints of painful uterine contractions, a

headache rated at 5/10, and a blood pressure of 133/96 mmHg. The patient was 165 cm tall, weighed 83 kg and listed an allergy to ultram. The patient's medical history was significant for CRPS, anxiety and depression. Pertinent surgical history included spinal cord stimulator placement for chronic low back pain following a motor vehicle accident. Current medications included oxycodone sustained release three times daily, oxycodone PRN, zolpidem, buproprion, and pre-natal vitamins. Fetal monitoring was initiated and a laboratory panel and urine sample was sent to the lab. Initial laboratory values were normal with the exception of the urinalysis that indicated the presence of 30mg/dl of urine protein. The patient was diagnosed with pre-eclampsia and admitted for augmentation of labor. Anesthesia was consulted to provide labor analgesia.

The patient was examined and interviewed and anesthesia consent was obtained. Physical exam was remarkable for lower back and hip pain and the presence of an implanted spinal cord stimulator. The pain clinic was consulted regarding options for pain control during labor. The pain clinic recommended the use of IV opioids though it was noted that the patients history of chronic opioid use might require higher than normal doses to achieve adequate analgesia. The elective placement of a CLE for labor analgesia was not recommended since stimulator lead location could not be verified. After discussion with the obstetrician and attending anesthesiologist, a remifentanil PCA was initiated with no basal rate, demand only at 20 mcg every 2 min. The option of utilizing ketamine boluses for breakthrough pain was also discussed. The patient's pain remained well controlled throughout the first stage of labor with reported pain scores of 4/10 to 6/10 during contractions. Analgesia for the second stage of labor was augmented by the placement of a pudendal block by the staff obstetrician and by one bolus dose of ketamine 20 mg IV. The PCA was secured with the start of active pushing. Total remifentanil use was 3040 mcg during approximately 11 hours of labor. Delivery of a healthy female occurred 9 minutes after PCA was discontinued. The infant had reported 1 and 5-minute APGAR scores of 9 and 9 respectively. The patient and infant had an unremarkable post-partum recovery and were discharged home.

Discussion

Labor pain can be severe in healthy parturient women. , Co-existing disease states, such as CRPS, can exacerbate labor pain and render adequate pain management a difficult task. [2,3] While pain in parturient women is not considered life threatening, the psychological harm can be far reaching, and has been linked to post partum depression and even post traumatic stress syndrome.[2] The American Society of Anesthesiologists in conjunction with the American College of Obstetricians has determined that no patient should suffer severe pain while under care of a physician and that maternal request is a sufficient medial indication for providing pain relief during labor.[2] For the majority of parturient, continuous lumbar epidural (CLE) anesthesia provides safe and adequate pain control.[2] When CLE placement is contraindicated, providers often rely on PCA delivered opioids such as fentanyl or remifentanil for labor analgesia.[4-7]

The ideal intravenous opioid for labor analgesia would have a rapid onset of action, rapid elimination by both mother and fetus, and have good analgesic efficacy.[5] Remifentanil meets these three requirements.[4,5] As a potent, short acting U-opioid agonist, remifentanil differs from

other opioids in its rapid onset of action of approximately one minute and rapid clearance by tissue and plasma esterases.[7] With a reported context sensitive half-life of approximately 3 minutes prolonged administration does not cause accumulation in the mother or the fetus.[4,5]

Remifentanil is becoming an increasingly popular adjunct in the treatment of labor pain in the parturient for which regional anesthesia is contraindicated. Studies remain limited and case studies remain the primary source of information regarding its use.[4] One major concern for providers in implementing a remifentanil PCA is what dose to use to provide adequate analgesia while limiting unwanted side effects such as maternal and infant respiratory depression.[4-6]

One study by Hill examined various remifentanil PCA dosing regimens used for labor analgesia among 8 different studies. Bolus dosing ranged from 0.2 to 0.93 mcg/kg, with lockout rates ranging from 1 to 3 minutes.[5] The most widely used dose was 0.5 mcg/kg. Timing of the dosage was an important factor in analgesic efficacy. A two minute lockout was reported to be the most effective as it pairs well with the timing of uterine contractions.[5] Only one of the eight studies added a background infusion of remifentanil in addition to the patient on demand delivery. This study supported that the background infusion did not improve analgesia, but increased the incidence of maternal oxygen desaturation and maternal sedation.[5] A demand only delivery system without a basal rate reported minimal maternal and fetal side effects.[5] Transient maternal oxygen desaturation was the most common complication associated with labor pain managed by remifentanil. Cases of maternal desaturation were easily treated with prompt delivery of oxygen to the mother via nasal cannula.[5] All articles reviewed recommend continuous pulse oxygenation monitoring of the parturient receiving remifentanil.[4,5,7] Additional recommendations include: one on one nursing supervision and the monitoring of maternal respiratory rate and sedation levels every 30 minutes.[7]

The dosage and timing of remifentanil administered in this case is within the limits of current published dosing regimens. The administered dose of 0.20 ug/kg, while at the low end of published literature, provided adequate pain relief for this patient. Given the patients history of CRPS that can cause hypersensitivity to pain[3] along with a history of chronic narcotic use, this low dosage should be carefully considered. There were no adverse maternal effects noted, and the patient reported a high level of satisfaction with her pain control. The effects to the fetus in this case were minimal with no effects noted on fetal monitoring. Additionally, naloxone administration or fetal resuscitation were not required after delivery. This appears to support the pharmacologic metabolism and redistribution of remifentanil in the neonate.[5,7] While the one on one supervision required for this patient placed additional strain on the Labor and Delivery nursing staff, the lack of adverse outcomes demonstrate the importance of close supervision.

This case has supported that remifentanil can be a safe and efficacious treatment modality for pain associated with labor. As large-scale research is limited[4-6], and dosing regimens vary greatly among reports, it can be difficult to determine the correct dosing for patients.[4-6] In this case the dosing was a collaborative effort between many services. The outcomes for both mother and baby

seem to confirm that this collaborative effort was a good approach.

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Elevated Carbon Dioxide Levels Caused By Expiratory Valve Malfunction

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Keywords: expiratory valve malfunction, elevated carbon dioxide, increased $FICO_2$, incompetent expiratory valve, modified pressure decline method

An elevation in the level of CO_2 in the body is a potentially dangerous condition for a patient undergoing anesthesia. Fraction of inspired CO_2 ($FICO_2$), end-tidal CO_2 ($ETCO_2$), and capnography patterns provide information about cardiopulmonary status.[1] There are numerous causes of elevated $FICO_2$ and $ETCO_2$, and their effects can be detrimental.[1] Observation of changes in $FICO_2$ and $ETCO_2$ levels, as well as CO_2 waveform analysis, may facilitate detection of anesthesia equipment dysfunction and possible dangers to patients.[1] A thorough pre-anesthesia machine check may also prevent some of the causes of iatrogenic increases in $FICO_2$ and $ETCO_2$.[2,3]

Case Report

A 68 year old female with renal cancer was scheduled to have a robotic left nephrectomy. She measured 160 cm in height and weighed 56 kg. Her history was significant for hypertension and kidney stones. Laboratory blood analysis was within normal values except for blood urea nitrogen of 22 mg/dL and a calculated glomerular filtration rate of 62 ml/min. A complete anesthesia machine check was performed on an Aestiva 5 (Datex-Ohmeda, Madison, WI) anesthesia workstation before

the scheduled surgery. The patient received midazolam 2mg intravenously. The patient was preoxygenated using O_2 at 10 L/min, and standard monitors were applied. Anesthesia was induced intravenously with fentanyl 50mcg, lidocaine 60mg, propofol 100mg, and rocuronium 30mg. The trachea was then intubated with an oral Mallinckrodt 7.0 mm internal diameter endotracheal tube and it was secured at 21cm at the patient's lips. Mechanical ventilation was initiated with a tidal volume of 600 ml and a rate of 7 breaths/minute. Anesthesia was maintained with oxygen at 1 L/min; air at 1 L/min; and desflurane at 6.8%. Vital signs were stable during induction and initiation of general endotracheal anesthesia. The patient was placed in the right lateral decubitus position.

After positioning, the $ETCO_2$ was elevated at 55-60 mmHg and $FICO_2$ values were elevated at 20 mm Hg. The baseline of the capnograph waveform was elevated and remained elevated without returning to baseline between ventilation cycles. Although no color change was present to indicate absorbent exhaustion, the CO_2 absorbent was changed. The CO_2 sidestream sample line was changed and recalibrated. There was no drop in the $ETCO_2$ or in the $FICO_2$ values following these interventions. Gas flows of oxygen and air were increased to 5 L/min each in an attempt to prevent rebreathing of CO_2. $ETCO_2$ decreased by 1-3 mm Hg and $FICO_2$ values decreased from 20 mmHg to 18 mmHg. It was observed that the expiratory unidirectional valve was stuck in the open position and moisture could be visualized around the valve disc. The patient was manually ventilated via the endotracheal tube using an ambu bag and oxygen tank, while the anesthetic was maintained with a propofol infusion at 150 µg/kg/min. The anesthesia workstation was then exchanged for an identical workstation with a properly functioning expiratory valve. $ETCO_2$ then decreased to 33 mm Hg and $FICO_2$ quickly decreased to 0 mmHg. The CO_2 waveform returned to zero. The patient remained stable during this time.

The patient had an otherwise uneventful intra-operative course, and emerged from anesthesia with no complications. The patient spent 1.5 hours in the post-operative recovery room, followed by a 24 hour hospital stay before being discharged home with no further complications. The malfunctioning expiratory valve was opened to air and allowed to dry over a few hours. Later in the day, the same machine was used successfully, and no rebreathing of CO_2 occurred.

Discussion

The causes of increased $ETCO_2$ can be divided into two categories of increased production and decreased excretion.[2] The causes of increased $ETCO_2$ are many and can make finding the etiology of increased $ETCO_2$ difficult. Since there are fewer causes of increased $FICO_2$, e.g. exhausted CO_2 absorbent, channeling through CO_2 absorbent, a rebreathing system, a blocked scavenging system, or a circle system with a malfunctioning expiratory valve, the first investigative step for a patient with elevated $ETCO_2$ and $FICO_2$ would be to look for possible etiologies of increased $FICO_2$.[1,2,4] Once $FICO_2$ is corrected, it is likely that $ETCO_2$ will also decrease. If $ETCO_2$ remains elevated, then the practitioner can troubleshoot causes of $ETCO_2$ specifically.[2]

The expiratory valve may malfunction due to dislodgment or as a result of water vapor condensation.[4] An expiratory valve that remains open throughout the respiratory cycle causes rebreathing of CO_2 that is displayed as an elevated baseline on capnography and a high $FICO_2$ value on

capnometry. In this case, the increase in $FICO_2$ was caused by an expiratory valve that was fixed in the open position due to an abundance of moisture around the valve.

The 1993 Food and Drug Administration anesthesia workstation checklist includes verification of CO_2 absorbent adequacy and visual inspection of proper action of unidirectional valves.[2] A 2007 follow-up by the American Society of Anesthesiologists recommends the same, and in addition, recommends checking the CO_2 absorbent and unidirectional valves between each case.[2] Item 13 of the 2007 update states that the breathing circuit should be observed for proper gas flow during both inspiration and expiration.[2] It also states that although the presence of unidirectional valves can be assessed visually, proper function of these valves cannot be visually assessed because subtle valve incompetence may not be detected.[2] A standard FDA machine check was performed for this case, including visual inspection of the unidirectional valves with no noted malfunction. Utilization of the modified pressure decline method (MPDM) to detect unidirectional valve malfunction is a potential solution that could assist in detection of a faulty valve since visual inspection alone may be inadequate.[2,3,5] The MPDM is an independently developed technique by Drs. Wade Weigel and W. Bosseau Murray; it is a modified version of a previously described technique called the "pressure decline method."[5]

In addition to the standard FDA machine check, it may be helpful to use the MPDM to detect subtle valve malfunction.[5] The MPDM takes about 1 minute to complete and should be performed after the system leak checks have been completed. To perform the MPDM, an extra 3 liter reservoir bag is placed at the inspiratory port. (Fig. 1) The adjustable pressure limiting valve (APL) is closed and fresh gas flows minimized to 75 ml/min. The circle system is pressurized using the O_2 flush valve to 30 cm H_2O. The user should see both reservoir bags inflate. (Fig. 2) The reservoir bag at the bag mount site is observed for signs of deflation such as creases or wrinkles for 20 seconds. If wrinkles are seen, this is indicative of deflation secondary to expiratory valve incompetence. (Fig. 3) A stuck inspiratory valve would prevent inflation of the extra reservoir bag at the inspiratory port. The user should open the APL valve to release pressure from the circle system. The extra reservoir bag is observed for 20 seconds at the inspiratory port. If wrinkles are seen in the bag, this indicates deflation due to inspiratory valve incompetence. The drawback of the MPDM is that it tests both the inspiratory and expiratory valve for incompetence (leaking), but tests only the inspiratory valve for sticking (closed).[5] A visual inspection of wrinkles in the reservoir bag has shown sensitivity equal to an incompetent valve with a leak of 0.6L/min.[5] In the case presented in this report, this would likely have been a valuable preoperative test since it involved an expiratory valve that was incompetent.

Although the patient in this case report suffered no complications related to elevated CO_2 levels, it is important to be aware of the causes of increased $FICO_2$ and the potential dangers for the patient. Potential detriments of an elevated $FICO_2$ include: hypertension, tachycardia, pulmonary hypertension, arrhythmias, increased cerebral blood flow, seizures, respiratory arrest, and death.[1,2] If $FICO_2$ is elevated, one should immediately increase total gas flows to decrease the amount of rebreathing, check the expiratory unidirectional valve for normal functioning, and check the CO_2 absorbent. It is also of paramount importance for anesthesia

professionals to implement a full anesthesia workstation checkout prior to the first procedure each day. The MPDM shows promise as a quick and easy tool in detecting unidirectional valve malfunctions and should be considered by anesthesia professionals as a valuable addition to the required checklist. Performing these tasks could help prevent serious patient complications from elevated $FICO_2$ levels.

Figure 1. Illustration of the first step of the MPDM; place an extra bag on the inspiratory port.

Figure 2. Depiction of both bags inflated during the MPDM test.

Figure 3. Illustration of a wrinkled bag; here, the bag at the bag mount site has wrinkles, indicating expiratory valve incompetence.

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Total Lung Lavage in a patient with Alveolar Proteinosis

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Keywords: alveolar proteinosis, alveolar recruitment, pulmonary proteinosis, whole lung lavage

Alveolar proteinosis is a rare disease in which surfactant lipids and proteins accumulate in pulmonary alveolar macrophages and alveoli, resulting in respiratory insufficiency and in severe cases, respiratory failure.[1] This idiopathic condition, now thought to be an autoimmune disorder, causes progressive exertional dyspnea.[2] Anesthetic management requires proficiency and diligence on the part of the anesthetic practitioners to prevent hypoxemia and respiratory failure throughout the duration of whole lung lavage. The case report presented will detail a patient with alveolar proteinosis who presented for whole lung lavage.

Case Report

A 54 year old, 117.9 kg, 165.1 cm African American female presented for whole lung lavage. Her past medical history included hypertension controlled with carvedilol. Surgical history included a total abdominal hysterectomy and two previous procedures involving whole lung lavage. No food or drug allergies were noted. All laboratory values were unremarkable. Her chest radiography report revealed dense opacities throughout bilateral lower lung fields. Physical examination revealed diminished lungs sounds bilaterally with significant work of breathing noted during the preoperative interview. No cardiovascular abnormalities were identified.

Upon arrival to the operating room, nonivasive monitors were applied. The patients SpO_2 on room air was 89%. With the patient positioned in a slight head up

position, the patient was pre-oxygenated with 100% oxygen for five minutes which increased her SpO_2 to 97%. Immediately following pre-oxygenation, intravenous (IV) induction was achieved using 200mg of propofol, 100mcg of fentanyl, and 60mg of rocuronium. Her trachea was intubated with a 39 french dual lumen endotracheal tube and both cuffs were inflated. Tube positioning was confirmed utilizing fiberoptic bronchoscopic inspection. Neuromuscular blockade was maintained throughout the procedure, and anesthesia was maintained with sevoflurane.

The right lung was isolated first. The surgeon then began an infusion of warmed normal saline into the lung, external percussion on the lung field was initiated, and the normal saline was then removed by gravity drainage. This procedure was interrupted intermittently at which time the lung was inflated manually with positive pressure at 30 mmHg and held for 45 seconds. After the lung lavage returned clear, the procedure was repeated on the opposite lung. Lasting a total of 6 hours, 14 liters of normal saline were lavaged through the lungs.

After the procedure was completed the dual lumen endotracheal tube was exchanged utilizing a cook exchanger for an 8.0 french endotracheal tube, and ventilatory support was maintained with mechanical ventilation with 15mmHg of positive end expiratory pressure (PEEP). The patient was transferred to the post anesthesia care unit where mechanical ventilation was weaned. PEEP was weaned in increments of 5 mmHg every 30 minutes, and assessment of inspiratory volumes were considered before further reduction in mechanical ventilation was done. Alveolar recruitment was optimized with the utilization of PEEP prior to the patient's trachea being extubated.

Discussion

Pulmonary alveolar proteinosis (PAP) is a disorder of unknown etiology and variable natural course.[2] It is characterized by the accumulation of lipoproteinaceous material within the alveoli secondary to abnormal processing of surfactant by macrophages.[2,4] It is associated with increased work of breathing, dyspena, dry cough, fever, and malaise, leading to respiratory failure. The prevalence of acquired pulmonary alveolar proteinosis has been estimated to be 0.37 per 100,000 persons.[3]

Recent studies have shown some improvement in the disease process with the administration of human granulocyte-macrophage colony-stimulating factor, however whole lung lavage remains the most effective treatment of PAP.[4] Whole lung lavage (WLL) involves the induction of general anesthesia, the isolation of the lungs with a double-lumen endotracheal tube and in the presence of single lung ventilation, normal saline lavages are performed on the nonventilated lung.[5] This is a means to wash out the proteinaceous material and reestablish effective oxygenation and ventilation.[5]

In a patient with compromised respiratory status, the isolation and ventilation of only one lung can cause severe hypoxemia and is very challenging for the anesthesia practitioner.[4] Maintaining adequate oxygenation and normocarbia are the anesthesia provider's primary goals. For this reason it is suggested that the lavage begin with suspected worst lung so as to prevent possible hypoxemia and hypercarbia and the cardiovascular effects that follow.[2,4,5]

Several methods of WLL have been suggested but recommendations to perform WLL under continuous positive airway

pressure (CPAP) ventilation have resulted in a decreased incidence of hypoxemia during one lung ventilation.[4] One case report demonstrated that a modified CPAP system used on the lung receiving the lavage fluid resulted in a noted improvement in oxygenation during the WWL.[4] This type of CPAP was not used for the case presented, and there were no periods of hypoxemia throughout the case.

As WLL involves large amounts of warmed saline (4-8 liters per lung in increments of 1 liter per lavage) being instilled into the nonventilated lung with the patient in the supine position. Chest physiotherapy is performed for 4-5 minutes and the lavage fluid is then allowed to drain passively from the lung. Drainage of the effluent can be aided by patient positioning in the trendelenburg position as well as the lateral position. It is recommended that with frequent positioning changes the patient be properly secured to the operating table with safety straps and be adequately padded at sites such as the elbows, heals, and sacrum in the supine position; and the axilla, knee, ankle, ear, and eye in the lateral position.[7]

Accurate documentation of the infused and drainage fluid will help avoid the most common complication of WLL which are: pneumothorax, pleural effusions, and a hydropneumothorax, through avoidance of increased intra-pleural pressures and subsequent barotrauma.[5] It is suggested the discrepancy of fluid should not be more than a few hundred milliliters.[5] Fluid discrepancies during this case were within these defined limits. Furthermore, anesthesia for WLL is undoubtedly hazardous: the use of one-lung ventilation for the instillation and drainage of large volumes of fluid in a patient with pre-existing respiratory failure increase the risk of hypoxemia as well as flooding the ventilated lung.[7]

Recommendations for volume versus pressure ventilation were not noted in the literature reviewed, as this seems to remain a preference of the pulmonologist; however inspiratory pressures should be kept within normal ranges to prevent barotrauma.[2,5]

Whole lung lavage has been successfully used in patients with PAP since the 1960's and can provide improvement which lasts for 15-24 months.[8] In the case presented the patient remained stable throughout the procedure and during the post operative period and was successfully treated for the 3rd time with WLL. Experience suggest that good teamwork with the respiratory physician throughout this prolonged procedure is necessary for safe WLL.[7] Therefore, it remains imperative that the anesthesia practitioner work diligently to maintain adequate ventilation in the patient undergoing WLL, through proper education regarding the procedure, attention to ventilation status and hemodynamic changes.

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Mentor: Christine Langer CRNA, MS, MSEd, MSN

Malignant Hyperthermia during Third Exposure to Volatile Anesthetic Agents

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Keywords: Malignant hyperthermia, sevoflurane, isoflurane, pediatric anesthesia, dantrolene.

Malignant hyperthermia (MH) is an inherited disorder that can be precipitated by volatile anesthetic agents and depolarizing muscle relaxants. It is associated with mutations within the gene encoding the skeletal muscle ryanodine receptor. This receptor causes the release of Ca^{2+} from the sarcoplasmic reticulum leading to muscle contraction and other metabolic activities.[1] The estimated incidence of MH episodes per million of hospital discharges increased from 10.2 in 2000 to 13.3 in 2005.[2] Symptoms of MH include increased $ETCO_2$, tachycardia, hyperthermia, acidosis, and muscle rigidity. This case report describes the manifestations and management of MH in an otherwise healthy pediatric patient who had undergone previous uneventful surgical procedures.

Case Report

A 9 year old, 33 kg, 139 cm African American male with no comorbidities was scheduled for the second application of a circular external fixator to treat a right fibula hemimelia with leg length discrepancy. Preoperative vital signs were: BP 115/67 mmHg, HR 58 beats/min, RR 18 breaths/min, temperature 36.6° C, and 98% SpO_2 while breathing room air. He received midazolam 15 mg by mouth. The initial intraoperative vital signs were: BP 107/71 mmHg, HR 74 beats/min, RR 33 breaths/min, and temperature 36.5° C. General anesthesia was induced with nitrous oxide (N_2O) 7 L/min, oxygen (O_2) 3 L/min, and a gradual increase of sevoflurane. A size 3 laryngeal mask airway (LMA, LMA North America, Inc., San Diego, CA) was placed, positive $ETCO_2$ and bilateral chest rise were noted, and spontaneous ventilations resumed. The sevoflurane was replaced with isoflurane for the length of the procedure. General anesthesia was maintained with 2-3% isoflurane inspired concentration in a

mixture of oxygen 1 L/min and air 1 L/min. Additional medications administered during the initial phase of the procedure were hydromorphone 0.3 mg IV and dexamethasone 6 mg IV.

Two hours after initiation of the surgical procedure, the patient's $ETCO_2$ increased from 66 mmHg to 115 mmHg, HR from 108 to 147 BPM, and temperature from 37.5° to 40.37° C. Despite efforts to decrease the $ETCO_2$ with manual hyperventilation, it continued to rise. Malignant hyperthermia was suspected, the halogenated volatile agent was discontinued, and 100% O_2 was administered. The surgeon was notified, and sterile drapes were placed over the surgical site.

The LMA was removed, and a 6.0 cuffed endotracheal tube was placed. The patient's jaw was noted as "very tight." A propofol infusion was initiated at a rate of 150 mcg/kg/min to maintain adequate sedation, and midazolam 2 mg was given. After arterial cannulation, the initial ABG was: pH 7.13, $PaCO_2$ 86.6 mmHg, PaO_2 490 mmHg, BE -2 mmol/L, HCO_3 28.8 mmol/L, and SpO_2 100%. The Bair Hugger cooling system (Arizant Healthcare Inc., Eden Prairie, MN) was used, and ice packs were placed in the patient's axillae and around the upper and lower extremities to achieve a temperature of <39° C. A Foley catheter was inserted to assess urine output.

An initial dose of premixed dantrolene with mannitol 40 mg was administered IV. Calcium chloride (CaCl) 300 mg IV was given to counteract physiologic effects of the potassium level of 5.4 mEq/L. Sodium bicarbonate 5 mEq/kg IV was given to treat acidosis. Three additional doses of dantrolene 20 mg IV were given to treat persistent hyperthermia, rigidity, and rising $ETCO_2$. After the fourth dose of dantrolene, the patient's $ETCO_2$, HR, and temperature were stabilized. The final ABG in the surgical suite was: pH 7.5, $PaCO_2$ 57.8 mmHg, PaO_2 576 mmHg, BE 23 mmol/L, HCO_3 48.1 mmol/L, and SpO_2 100%. The patient was transported to the pediatric intensive care unit in stable condition for further observation. Postoperative diagnostic studies obtained were additional ABGs, a CBC, chemistry panel, coagulation studies, and serial concentrations of creatine kinase (CK). All diagnostic studies were within a normal range, and the patient continued to improve.

Discussion

In 1981, Shulman et al. documented the first reports of MH triggered by sevoflurane in swine.[3] Compared to isoflurane, sevoflurane has been described as a less potent trigger for MH.[3] Chen et al. reported cases of MH after subsequent exposure to volatile agents in a five year old male undergoing an orthopedic procedure.[4] During the second exposure to sevoflurane, after 90 min of uneventful anesthesia, the patient developed MH. Two days prior to this anesthesia, he had undergone fixation for fractures with casting, using general inhalation anesthesia via mask with sevoflurane and O_2. The patient recovered without any complication as an aftermath. No research is available that explains the occurrence of MH in patients after a second or third exposure.

In our case, sevoflurane and N_2O were used for induction and isoflurane for the maintenance phase of anesthesia. The patient presented with MH manifestations approximately 2 hr after the procedure began. He was exposed to both sevoflurane and isoflurane. It could not be determined which agent precipitated MH, or if the combination of anesthetic agents was the cause. The patient was healthy with no

family history of muscular dystrophy. He had received general anesthesia with sevoflurane in 2009 and 2010 for orthopedic surgical procedures without complications. Muscle biopsy or genetic testing are the best methods for predicting MH. Genetic testing determines if there are abnormalities or changes to the RYR1 gene. Studies have shown that 50% of patients who are MH susceptible will exhibit a change in the RYR1 gene.[5] Malignant hyperthermia is an autosomal dominant genetic condition. If one parent carries the gene a child has a fifty percent chance of inheriting the gene and is then predisposed to developing MH.

The clinical manifestations of MH vary among patients. MH can be misdiagnosed as hypoxia, hypercarbia, infection or sepsis, thyrotoxicosis, pheochromocytoma, or neuroleptic malignant syndrome.[6] The MH clinical grading scale (MH CGS) estimates the qualitative likelihood of MH in a given patient without the use of specialized diagnostic testing and is based on points assigned for specific abnormal signs and laboratory findings (i.e., rigidity, muscle breakdown, respiratory and metabolic acidosis, temperature increase, and cardiac involvement) observed during an acute anesthetic reaction.[7] For the patient presented in the case report, a diagnosis was made based on his clinical presentation, and the MH CGS was not used to aid in the diagnosis.

Immediate recognition of an MH emergency is crucial to prevent further complications and avoid death. Early detection of MH in this case was due to diligent patient assessment. Once the irregularities in the patient's vital signs were noted, the MH diagnosis was determined 12 minutes later. Aggressive resuscitation commenced. The halogenated agent was discontinued and 100% O_2 was administered with the existing circuit. Once the temperature is < 38^o C the Malignant Hyperthermia Association of the United Stated (MHAUS) suggests cessation of the cooling process to prevent a drift to < 36^o C.[8] The suggested treatments for a MH crisis are the following: [8]

- Seek help from the anesthesia team for immediate MH resuscitation
- Notify the surgeon
- Discontinue the triggering agent(s)
- Give dantrolene 2.5 mg/kg rapid infusion through large bore IV
- Hyperventilate with 100% O_2 at flows of 10 L or more
- For the sake of time do not change the circuit and CO_2 absorbent
- Cool the patient with lavage, ice packs, and cooling blanket

During the post-acute phase constant monitoring is critical. The care team must be aware of recurrent signs. Dantrolene 1 mg/kg IV every 4-6 hr should be administered, and continued for 24-48 hr after an episode of MH. Additionally, the family should be referred to a biopsy center for discussion of testing options to determine MH susceptibility.[8] Until data suggest otherwise, these patients should be managed as MH susceptible with a non-triggering anesthetic technique, and relatives should be asked about anesthesia problems or neuromuscular disorders.[8] In conclusion, the anesthesia practitioner should not rule out the possibility of MH if a patient has undergone prior uneventful anesthetics.

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Mentor: Chris Hulin, CRNA, DNP

Editorial

Happy holidays to everyone! During this time we tend to reflect on what we are thankful for – regarding the ISJNA, I am deeply grateful for all of the volunteer CRNAs who make this journal a reality. Their efforts show true dedication to the education of future nurse anesthetists and support of our profession, and the ISJNA simply would not exist without them. So, scroll back up to the beginning and browse through the list of editors and reviewers – if you recognize a name, shoot them a quick 'Thank you for the ISJNA!' email.

Best wishes for a wonderful New Year,

Vicki C. Coopmans, CRNA, PhD
Editor

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

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INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA
GUIDE FOR AUTHORS

MISSION STATEMENT

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

ITEMS ACCEPTED FOR PUBLICATION

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

ITEM PREPARATION & SUBMISSION

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

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

PEER REVIEW

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

General guidelines

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

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

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

<u>Case Reports</u>

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

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

Heading (see #9 above in General Guidelines)

[space]

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

[space]

Case Report (bold, 400-500 words)

[space]

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

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

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

[space]

Discussion (bold, 600-800 words)

[space]

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

[space]

References (bold)

[space]

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

[space]

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

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

Research Abstracts

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

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

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

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

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

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

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

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

AMA MANUAL OF STYLE

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

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

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

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

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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:

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

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

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

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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.

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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.

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___ **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.
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___ 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.

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EBPA Report
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___ A critical evaluation of a practice pattern in the form of a precise clinical question about a specific intervention and population is presented.
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