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TOPICS IN THIS ISSUE

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Front Cover:

Ruth Jahn, BSN, a graduate student enrolled in the Northeastern University Nurse Anesthesia Program, delivers anesthesia to a patient undergoing ureteral reimplantation and obstetric fistula repair during a global health rotation in Kigali, Rwanda with the International Organization for Women and Development. Ms. Jahn is using the Glostavent Anaesthesia Machine (Diamedica, Devon, UK), which is designed for use in low resource areas. The Glostavent component gas driven ventilator and oxygen concentrator continue to function without interruption, even if central oxygen and power systems fail.

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Case Reports

Coagulation and Anti-coagulation for Combined Heart Liver Transplant

Jessica Holigan, MS
Northeastern University

Keywords: coagulation, anti-coagulation, combined heart liver transplant, hemostasis, CHLT

The first combined heart liver transplant (CHLT) was performed in 1984. [1] Based on data from the Organ Procurement and Transplantation Network, as of November 10, 2013, 139 CHLTs have been performed in the United States. Maintaining hemostasis during this procedure can be a particularly challenging and multi-factorial task. These patients usually present with a degree of underlying coagulopathy from pre-existing liver disease. There are also inherent factors in the process of cardiac surgery that leave patients at a greater risk for developing additional coagulopathies. This case report describes the anesthesia care of a CHLT with a focus on coagulation management.

Case Report

A 45-year-old male with a history of right atrial mass complicated by recurrent pulmonary emboli and deep vein thrombosis presented in end stage hepatic and cardiac failure. Fourteen years previously, the patient's atrial tumor was excised and a tricuspid valvuloplasty was performed. Subsequently, he developed tricuspid regurgitation, which eventually led to right-sided heart failure and hepatic cirrhosis. After 6 months on the recipient transplant list, a suitable donor became available and the patient was brought to the operating room.

A right radial arterial line and 18 gauge peripheral IV were placed, and anesthesia was induced by rapid sequence using etomidate 20 mg, rocuronium 90 mg, and fentanyl 100 mcg. During induction of anesthesia, his hemodynamics were maintained with dobutamine 1 mcg/kg/min, milrinone 0.1 mcg/kg/min and phenylephrine 0.15 mcg/min. In preparation for anticipated blood loss, large bore vascular access was obtained with an 8 Fr rapid infusion catheter inserted into the right antecubital vein and a 9 Fr double lumen central catheter in the right internal jugular vein. A rapid infuser, with the capability of infusing 1000 mL/min was attached to the peripheral line.

After adequate access had been established, the surgeon performed a median sternotomy and an IV bolus of heparin 350 units/kg and aminocaproic acid 10 mg were given to prepare for induction of the cardiopulmonary bypass pump. Infusions of heparin 100 units/kg/hr and aminocaproic acid 2000 mg/hr were then maintained throughout the procedure. Cardiopulmonary bypass was initiated once adequate systemic heparinization had been achieved and the patient was then cooled to a core temperature of 32^{o}C. The heart was transplanted uneventfully and the patient was rewarmed to a core temperature of 35^{o}C before attempting to wean cardiopulmonary bypass. Hemodynamics were initially stable and the grafted heart appeared to be adequately functioning by echocardiography. At that time, protamine sulfate 250 mg was given to reverse the remaining circulating heparin.

Approximately 30 min after cessation of bypass, his right ventricular function declined in the face of diffuse bleeding without an obvious source. Hemodynamic stability was maintained with infusions of epinephrine, norepinephrine, vasopressin and milrinone at varying dosages. While attempting to achieve control of the bleeding in the surgical field, the patient received 7 units fresh frozen plasma (FFP), 3 units packed red blood cells, 6 units of platelets and 600 mL of cell saver blood. The coagulopathy improved and hemodynamics were stabilized with pharmacologic support. At that point, the decision was made to proceed with the liver transplant. The aortic cannula was removed while the venous cannulas were left in place for veno-venous bypass during the anhepatic period. Additional blood products were given throughout the case to maintain hemostasis for a total of: 10 units cryoprecipitate, 14 units packed red blood cells, 30 units platelets, 20 units FFP, and 5,200 mL of cell saver volume. Total operating room time was 21.5 hours. When the liver was transplanted and once hemostasis was achieved and hemodynamics were adequately controlled on vasopressors, the patient was transferred to the cardiac surgical intensive care unit.

Discussion

The difficulty in maintaining hemostasis in a patient undergoing CHLT surgery is multifactorial. End stage liver disease is associated with coagulation abnormalities that are caused by reduced synthesis of clotting factors, fibrinolysis and thrombocytopenia. The need for cardiopulmonary bypass during cardiac transplant also adds factors that make maintaining hemostasis complex. This includes induced the need for systemic anticoagulation, dilutional coagulopathy, the effects of induced hypothermia, fibrinolysis and platelet abnormalities.

Taken together, the aforementioned factors increase the risk of bleeding. The ability to recognize and treat these conditions properly is essential. Patients undergoing CHLT may have massive transfusion requirements intra-operatively; therefore it is critical before the procedure begins to secure large bore vascular access and to communicate with the blood bank to confirm they have the products available should massive transfusion be required. It is common practice to use a rapid infusion system that is capable of infusing products at a rate that is above surgical loss.

Fibrinolysis is a common problem in both liver failure and cardiac surgical patients. It is caused by activation of endothelial plasminogen in response to fibrin formation. In this case, the antifibrinolytic agent aminocaproic acid was given in an attempt to inhibit excessive fibrinolysis. There is controversy surrounding the use of antifibrinolytics during liver transplant surgery due to the risk of thrombosis, although benefits have been clearly demonstrated during cardiac surgery, especially when used prophylactically. [2, 3]

Patients with liver disease often present with pre-existing thrombocytopenia. Additionally, the use of veno-venous bypass for liver transplantation and cardiopulmonary bypass for heart transplantation requires blood to come in contact with extracorporeal surfaces. This contact renders platelets inactive or leaves them with reduced function. Platelet dysfunction is further aggravated by induced hypothermia, and by the use of heparin and protamine. [2] Thromboelastography (TEG) can be useful for determining platelet dysfunction, factor depletion and the presence of fibrinolysis. Although standard activated partial thromboplastin time (aPTT) and prothrombin time (PT) testing was used in this case, the TEG has been shown to correlate

better with clinical bleeding, leading to a reduction in the use of blood products.[4] Management of systemic anticoagulation with appropriately dosed heparin and its neutralization with protamine improves intra and post-operative bleeding.[4,2]

The standard dose of heparin given before initiation of cardio-pulmonary bypass is 300-400 units/kg. Once adequate cardiac function is established and bypass successfully weaned protamine is given to neutralize the remaining circulating heparin. The usual dosing regimen is a protamine to heparin ratio of 1-1.2:1. One problem with this ratio method is that it does not take into account the rate of heparin clearance over the length of bypass. Excess protamine administration has been shown to cause an increase in post-operative bleeding because it decreases platelet function and weakens clot structure.[5]

Transfusion of blood products during CHLT is likely unavoidable. Standard risks associated with transfusion therapy include, but are not limited to: infections, organ dysfunction and increased mortality. [6] It is therefore essential to treat patients based not solely on lab values, but also to assess clinical evidence of coagulopathies.[2] Correction of aPTT, INR and fibrinogen with fresh frozen plasma or cryoprecipitate is warranted when there is evidence of bleeding. It is recommended that 15 mL/kg FFP be given for an INR greater than 2 or a PTT 1.5 times control.[2] If clinical coagulopathy persists after a second dose of FFP, recombinant factor VIIa can be considered. A risk benefit analysis should first be performed, because, in addition to its significant cost, factor VIIa has been associated with hypercoagulation states. [2] Transfusion of platelets is recommended when platelet counts are less than 100 K/μL with evidence of surgical bleeding. [2] The problem with using the platelet count alone is that this number assumes circulating platelets are functioning properly. A better approach would be to evaluate both the number of platelets and their level of function with the TEG analysis. [4] Desmopressin acetate may also be used to improve platelet count and function by its ability to increase circulating levels of factor VIII and Von Willebrand factor. It has been shown to decrease the amount of blood transfusion required when there is an excessive amount of surgical bleeding. [7]

The cardiopulmonary bypass circuit is prepared by priming it with 1800 mL of crystalloid solution; additionally patients receive approximately 2500 mL crystalloid intravenously. This infusion of crystalloids, along with red cell transfusion, can dilute the coagulation factors of a patient who has hepatic dysfunction and may already be up to 50% deficient. [8] Dilutional coagulopathy can ultimately contribute to the amount of blood products that need to be replaced by allogenic transfusion.

Managing coagulation and anticoagulation in CHLT patients poses numerous challenges due to the inherent issues of coagulopathy in liver failure and the requirement of anticoagulation during cardiopulmonary bypass. Performing a successful CHLT takes a combined effort of many specialized clinicians.

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Mentor: Janet A. Dewan, CRNA, PhD

Microvascular Decompression of the Facial Nerve

Anne Hughes, MSN
University of Pennsylvania

Keywords: hemifacial spasm, left retromastoid craniectomy, microvascular decompression, cranial nerve VII, brain auditory evoked potentials (BAEP)

Hemifacial spasm (HFS) is a disorder described as involuntary, repetitive twitching of muscles innervated by the facial nerve (cranial nerve VII).[1] Evidence shows that in many patients, the underlying physiologic mechanism of HFS is related to vascular compression of the facial nerve at its root exit zone (REZ).[2] The compressing vessel is often the posteroinferior cerebellar artery; the second most common is the vertebrobasilar artery.[3] Microvascular decompression (MVD) via retromastoid craniectomy has been the only proven method to provide a long term cure for HFS; but even this has only been proven effective in curing about 90% of patients.[1]

Case Report

A 45-year-old, 84 kg, 173 cm male presented for a left retromastoid craniectomy and microvascular decompression of cranial nerve VII following a diagnosis of hemifacial spasm. His medical history was significant for left hemifacial spasm. Current medications were a daily multivitamin, which the patient had stopped one week prior to surgery. The patient's past surgical history was a septoplasty. The patient was allergic to penicillin. A preoperative airway evaluation revealed a Mallampati classification of III, a thyromental distance greater than 6 cm, an estimated 4 cm mouth opening, and full range of motion in the cervical spine.

Preoperative vital signs were as follows: blood pressure 119/77 mm Hg, heart rate 59/min, respiratory rate 18/min, and SpO2 96% on room air. A peripheral 16 gauge intravenous (IV) line was placed in the preoperative area and midazolam 2 mg IV was given during transport to the operating room. A scopolamine patch (1.5 mg) was placed behind his right ear to prevent post-operative nausea and vomiting (PONV). In the operating room, noninvasive monitors were applied; preoxygenation was completed via facemask using an oxygen flow of 10 L/min for 5 minutes. Intravenous induction was performed with fentanyl 50 mcg and propofol 200 mg. Successful mask ventilation was verified and vecuronium 5 mg IV was administered. Direct laryngoscopy using a Glide Scope (Verathon Inc., Bothwell WA) was performed to secure quick access to the airway, and a grade I view of the glottis was noted. An 8.0 mm endotracheal tube was placed through the glottis without difficulty, and placement was confirmed with positive end-tidal carbon dioxide ($ETCO_2$) and bilateral breath sounds. A radial arterial line was placed on the patient's right arm using sterile technique.

The patient was placed in the Mayfield headrest in the right lateral decubitus position. Before incision, decadron 10 mg and mannitol 100 mg were administered IV. General anesthesia was maintained with total intravenous anesthesia (TIVA) to facilitate sensory-evoked potential monitoring, with a propofol infusion ranging from 120-150 mcg/kg/min. Brainstem auditory evoked potentials (BAEP) were also monitored. Fentanyl was titrated for pain management throughout the case for a total of 750 mcg. The surgeon decompressed the artery that was believed to be causing the hemifacial spasm, but the lateral spread of the electromyography (EMG) response remained unimproved. The patient was given ondansetron 8 mg IV for PONV prophylaxis, and neuromuscular blockade was antagonized with IV neostigmine 4 mg and glycopyrolate 0.6 mg. The endotracheal tube was removed without complication and the patient was transferred to the post anesthesia care unit.

Discussion

Hemifacial spasm can be a severe and disabling condition that greatly affects quality of life.[4] Diagnosis of primary HFS requires these three criteria, the spasm is: 1) not a sequela of ipsilateral facial palsy, 2) chronic in evolution and 3) self-limiting.[5] Spontaneous recovery is unlikely, so two treatments options are currently available: botulinum toxin injections and MVD.[3] Microvascular decompression for primary HFS is based on the hypothesis that neuro-vascular compression is the cause of the spasm; in 98% of patients with primary HFS, an arterial loop is found to be compressing the facial nerve at its exit from the brainstem.[5]

Due to the hyperexcitability of the facial nerve, the stimulation of one branch of the facial nerve will activate facial muscles that are innervated by other branches of the facial nerve, which produces abnormal muscle responses.[2] This abnormal muscle response is known as the lateral spread response (LSR) and is observed from one muscle innervated by the superior branch of the facial nerve when the inferior branch is stimulated (or vice versa). The LSR can disappear for the majority of patients when the offending vessel is moved off the facial nerve, thus monitoring the abnormal muscle response is used to guide the surgeon during a MVD.[2]

During most MVD surgeries, LSR disappears immediately after the offending vessel is moved off of the facial nerve. However, in some cases LSR does not resolve immediately.[2] One study found the facial spasm to have resolved in 88% of patients within 24 hours of the operation, and 90% of patients' facial spasm resolved by the day of discharge. Although LSR monitoring isn't statistically associated with long term reported outcomes, postoperative LSR resolution is predictive of long-term spasm relief.[1] Thus, a challenge with MVD is that the results of the operation are not always immediate, and while intraoperative monitoring devices may help, it is not always indicative of HFS resolution.

Anesthetic agents are purposefully designed to temporarily decrease neurologic function, so monitoring a patient's neurologic function during surgery can be challenging.[6] Intravenous anesthetics generally affect somatosensory evoked potential (SSEP) monitoring less than inhalational agents, so a TIVA anesthetic was selected for this procedure.[6] In general, opioids have minimal effects on SSEP as well.[6] Brainstem auditory evoked potentials are used during procedures involving or near the eighth cranial nerve and are derived similarly to SSEP.[6] Both types of evoked potentials have characteristic patterns of evoked peaks that are generated corresponding with synapses that occur between the eighth nerve and cortex.[6] Upon reviewing the anesthetic management for this case, one could have considered administering a narcotic infusion to achieve a steady state of analgesia throughout the procedure rather than intermittent bolusing. Remifentanil would have been an appropriate alternative as it is an ultra-short acting narcotic. One of the priorities of anesthetic management during evoked potential cases is to provide a safe anesthetic while not interfering with the neurophysiological monitoring. It is equally important that the anesthetic is reversed quickly at the case conclusion in order to perform a thorough neurologic assessment as soon as possible.

Anesthesia practitioners directly control two main factors affecting neurological monitoring; these include anesthetic depth and physiologic factors. Anesthetic depth is affected by medications/inhalational agents administered during the case.[6] Physiologic factors include temperature (both hypothermia and hyperthermia), systemic blood pressure, $PaCO_2$ (affecting cerebral blood flow), and PaO_2 (oxygen delivery).[6] Accurate neurophysiological monitoring is a crucial component of this surgery, and anesthesia practitioners can have a vital role in maintaining its accuracy. The reliability of the evoked potential monitoring depends on maintaining a constant anesthetic level while maintaining adequate nerve tissue perfusion.[6]

Hemifacial spasm is a potentially devastating condition to patients. It is frustrating for patients to emerge from surgery with ongoing spasm. However, evidence suggests that HFS may resolve over the next 24 hours. Although there are significant risks involved with this procedure, such as hearing loss and other damage to the nervous system, the risk/benefit ratio is different for every patient and is evaluated on an individual basis.[2]

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Mentor: Kelly L. Wiltse Nicely, CRNA, PhD

Alpha-2 Agonists: Valuable Adjuvants in Neuroanesthesia

Darren E. Aiken, MSN
Wake Forest Baptist Health

Keywords: alpha-2 agonists, clonidine, dexmedetomidine, neuroanesthesia, craniotomy

Alpha-2 agonists, such as clonidine and dexmedetomidine, possess useful properties that make them effective adjuvants in neuroanesthesia.[1] These agents have been credited with an array of desirable effects such as: sedation and anxiolysis, improved hemodynamic stability, reduction in intraoperative anesthetic and analgesic requirements, decreased post-operative pain, decreased incidence of post-operative shivering, as well as decreased incidence of post-operative nausea and vomiting (PONV).[2,3] Many anesthesia professionals avoid using alpha-2 agonists due to the potential adverse effects of bradycardia, hypotension, and prolonged recovery.[4] When seeking to deliver a balanced-anesthetic, the anesthesia professional should consider the incorporation of alpha-2 agonists into the neuroanesthesia plan.

Case Report

A 56 year old male (height: 180.3 cm, weight: 95.2 kg) presented with complaints of headache, fatigue, and inability to focus. Computed tomography revealed a tumor in the left medial temporal region. The appearance of the lesion was consistent with a glioblastoma. The anesthetic evaluation yielded the following assessment: Mallampati class II, thyromental distance 6 cm, maximal mouth opening 4 cm, and no limitation in neck extension. Preoperative vital signs were: blood pressure 123/56 mm Hg, heart rate 79/min, S_pO_2 95%, and respiratory rate of 18. No medications were administered in the preoperative area

Upon arrival to the operating room, standard monitoring was instituted and pre-oxygenation commenced with 10 L/min oxygen delivered via facemask. The patient was given lidocaine 80

mg and fentanyl 150 mcg intravenously (IV). General anesthesia was induced with propofol 150 mg. Mask ventilation was initiated and rocuronium 70 mg was administered. Direct laryngoscopy was performed utilizing a Macintosh 3 blade yielding a grade 1 view. The trachea was intubated with a 7.5 mm cuffed oral endotracheal tube, placement confirmed, and mechanical ventilation initiated. A remifentanil infusion was started at 0.1 mcg/kg/min. The left radial artery was cannulated and invasive blood pressure monitoring initiated. An additional large bore peripheral intravenous catheter was placed in the right arm. Prior to the cranial pins being placed, esmolol 50 mg was given. A baseline arterial blood gas showed a gradient of 8 mm Hg between end-tidal carbon dioxide ($ETCO_2$) and the partial pressure of arterial carbon dioxide ($PaCO_2$). The patient's $ETCO_2$ was kept at approximately 27 mm Hg intraoperatively. Anesthesia was maintained with 0.6% isoflurane, 1 L/min nitrous oxide, 1 L/min oxygen and remifentanil 0.1 mcg/kg/min. Neuromuscular blockade was maintained with intermittent intravenous vecuronium administration. Intraoperative pathology confirmed that the tumor was a high-grade glioma.

The tumor was excised and isoflurane was discontinued 20 minutes prior to completion of closure. After the cranial pins were removed, the remifentanil infusion was discontinued and glycopyrrolate 0.6 mg and neostigmine 5 mg were given. After the surgical dressing was applied, the nitrous oxide was discontinued and oxygen was increased to 10 L/min. Though moderately agitated and not following commands, the patient was moving all extremities and demonstrated purposeful movement by reaching for the endotracheal tube. He was taking 500 milliliter tidal volumes at a rate of 12/min. He was extubated using positive pressure, taken to the post-anesthesia care unit (PACU) on 8 L/min oxygen via facemask, where he subsequently followed commands.

Discussion

Alpha-2 agonists are effective adjuvants that can be administered throughout the perioperative period. As a premedication, clonidine has been shown to produce equivocal sedation and anxiolysis as compared to midazolam.[2] In the same study, heart rate and adrenocorticotropic hormone was found to be lower in the clonidine group in the preoperative period suggesting improved attenuation of the stress response.[2] Only one third of patients in another study were satisfied with clonidine as a premedication versus the majority being satisfied in the midazolam group.[4]

Hemodynamic stability is of paramount concern in neuroanesthesia. Induction of anesthesia can prove to be a treacherous time. Alpha-2 agonists have been shown to reduce the amount of induction agent which can decrease hemodynamic collapse.[2,4,5] After induction of anesthesia, the anesthesia professional should attempt to blunt the hemodynamic and neuroendocrinal responses of laryngoscopy, intubation, and cranial pin placement. These nociceptive stimuli lead to sympathetic activation and subsequent elevation in systemic arterial pressure with concomitant increase in cerebral blood flow as well as intracranial pressure (ICP). This response could prove detrimental for patients with altered cerebral compliance, impaired auto regulation, or an intracranial aneurysm.[1,6] Both clonidine and dexmedetomidine have been shown to attenuate the hypertensive response to laryngoscopy, intubation, and cranial pin application.[1,6] Uyar et al. found that a single intravenous dose of dexmedetomidine at 1 mcg/kg prior to induction of

anesthesia attenuated the hemodynamic response and resulted in lower plasma cortisol levels as compared to the control group.[6]

In addition to the benefits of alpha-2 agonists during the induction period, there are many desirable effects during the intraoperative period. In their study, Soliman et al. explored the role of a continuous infusion of dexmedetomidine in patients with supratentorial tumors undergoing craniotomy.[1] Compared with the control group, patients in the dexmedetomidine group maintained greater hemodynamic stability, had reduced volatile anesthetic and intraoperative opioid requirements, lower ICP, as well as improved intraoperative urine output.[1]In addition to increased intraoperative urine output, another study found that postoperative creatinine levels were lower in those patients pretreated with clonidine.[5] In a study concerning myocardial contractility in isolated rat hearts, it was found that treatment with clonidine improved the myocardial oxygen supply/demand curve during isoflurane administration.[7] This finding may be consistent with the fact that clonidine has been shown to reduce the risk of perioperative cardiac mortality in patients with or at risk for coronary artery disease undergoing non-cardiac surgery.[2]

Alpha-2 agonists have many alluring properties pertinent to the post-operative period as well. A concern over utilization of alpha-2 agonists is a potential delay in awakening. There are conflicting studies regarding this concern. Smith reported a study in which dexmedetomidine was found to result in a modest delay in awakening but no delay in regards to discharge from the recovery room or to home.[4] Other studies report no prolongation in emergence times with the use of alpha-2 agonists.[2,8] Clonidine has been shown to result in less emergence agitation than midazolam.[4] One study reported a 57% decrease in the incidence of emergence agitation after 2 mcg/kg clonidine was given pre-operatively.[4] Reduction in the incidence of PONV is especially important for the neurosurgical patient because vomiting can result in a marked elevation in ICP.[3] Many studies have noted a correlation between use of an alpha-2 agonist and reduced incidence of PONV.[2,4,8] The physiology in reduction of PONV is unclear but may be due to a reduction in anesthetic and analgesic requirements as well as a reduction in the levels of circulating catecholamines.[8]Alpha-2 agonists can reduce analgesic requirements in the post-operative period. Blaudszun et al. reported the analgesic-sparing effects in terms of morphine equivalents. On average, they found that at 24 hours post-surgery that preoperative clonidine use resulted in a 4.1 mg decrease in morphine use, while preoperative dexmedetomidineuse resulted in the equivalent decrease of 14.5 mg of morphine.[8] It should be noted that the analgesic-sparing effects with alpha-2 agonists is weaker than that reported with ketamine or toradol.[8]

While alpha-2 agonists can be beneficial adjuvants in a variety of anesthetic plans, they have been proven particularly useful in neuroanesthesia. Alpha-2 agonists assist the anesthesia professional in mitigating the hemodynamic and neuroendocrinal responses that can have deleterious effects in the neurosurgical patient. In the case described above, an alpha-2 agonist was not used. Preoperative low-dose clonidine could have provided some measure of anxiolysis and contributed to improved perioperative hemodynamic stability. The reduction in anesthetic and analgesic requirements that alpha-2 agonists confer may have contributed to a more lucid and less agitated emergence of the patient, leading to an improved neurologic examination in the operating room.

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Mentor: Nancy Curll, CRNA, MSN, CDR, NC, USN (Ret.)

Bloodborne Pathogens and Maintaining a Safe Provider and Patient Care Environment

Jessica E. Rose, MSN
Wake Forest Baptist Health

Keywords: needlestick injury, bloodborne pathogens, hepatitis, healthcare professionals

Anesthesia practitioners face a variety of work-related challenges; yet a rising concern involving proper needle handling has such severe implications that improper techniques can lead to patient or provider injury as well as litigation issues.[1] Although prevention tools and safety methods exist, occupational exposure and needlestick injuries frequently occur.[1] Needle use and blood sample collection occur frequently during the intraoperative period and appropriate techniques must be utilized in order to ensure a safe patient and provider environment. It is of extreme importance to maintain standard precautions and use protective measures, especially when in contact with blood and bodily fluids.

Case Report

A 46-year-old male presented for thoracic spinal stabilization at levels T5-T10 with lysis of dural adhesions. The patient was 180.3 cm in height, weighed 79.4 kg, and had no known drug allergies. The patient's medicinal regimen included oxycodone-acetaminophen tablets three times per day as needed for pain control. The patient's past medical and surgical history included: daily tobacco use, kyphosis, hepatitis C, syringomyelia, thoracic myeolopathy, thoracic laminectomy, and intramedullary nailing of the femur. Despite prior surgical intervention (T7 laminectomy for excision of arachnoid cyst) the patient reported worsening pain and increased symptomatology. Symptoms included: right leg spasm and tenderness, left arm pain/tingling/burning, poor balance, and difficulty moving at times due to thoracic pain. The patient had no significant laboratory results. His most recent MRI displayed tethering of the mid-thoracic spinal cord and myelomalacia.

On the day of surgery, the patient presented with a normal physical and neurological examination, however the patient reported persistent back pain and anxiety. An 18-gauge peripheral intravenous (IV) catheter was inserted. The patient was given midazolam 2 mg IV prior to transportation to the operating room (OR). The patient reported his pain at 8/10 on the numeric pain scale and fentanyl 50 mcg was administered IV.

In the OR, monitors were applied and the patient was pre-oxygenated with O_2 10 L/min via facemask. General anesthesia was induced with the following IV medications: propofol 200 mg, lidocaine 100 mg, succinylcholine 140 mg, rocuronium 50 mg, and fentanyl 200 mcg. Direct video laryngoscopy was used to place and secure a 7.5 endotracheal tube (ETT). Positive $ETCO_2$ was confirmed and bilateral breath sounds were auscultated. Respiration was controlled by mechanical ventilation. A left radial arterial line was inserted and used for hemodynamic monitoring and blood sampling. The patient was positioned prone on the OR table and appropriately prepared and draped for surgery.

Due to the projected duration of the case as well as the anticipated blood loss, blood samples were drawn intermittently to assess the patient's overall hemodynamic status. The patient's hemoglobin and hematocrit were monitored closely. In addition, a type and screen sent so compatible blood transfusion products could be obtained if necessary. The equipment cart contained lab vacutainers, syringes, and needles. No needleless blood transfer devices were readily available for use.

General anesthesia was maintained with isoflurane and nitrous oxide, analgesia was provided with ketamine, fentanyl and sufentanil infusion, and akinesia was maintained with vecuronium. A systolic blood pressure of $\geq$ 90 mm Hg was maintained using IV fluid administration along with the use of a phenylephrine infusion. At the completion of the procedure, neuromuscular blockade was pharmacologically antagonized and the patient was repositioned supine. The ETT was successfully removed and the patient was transported to the recovery area with O_2 4 L/min via facemask, where handoff report was given to the receiving nurse.

Discussion

Healthcare professionals are at risk for infection, illness, disability, and death from bloodborne pathogens due to needlestick injuries. Needlestick injuries are defined as lacerations or punctures from needles or sharp instruments with contaminated blood or bodily fluids.[2] It is estimated that more than three million healthcare workers experience percutaneous injury from contaminated sharps each year and of these exposures, approximately 385,000 occur in the United States.[3] The surgical environment is blood-intensive, contains large amounts of sharp instruments, and has been identified as second only to patient rooms with regards to location of highest frequency of reported injuries.[1] Accidental needlesticks continue to remain at epidemic levels in healthcare workers despite current legislative acts and prevention practices.[4] Lack of experience, insufficient training, work overload and fatigue are probable causes that lead to sharp injuries.[3] Current preventative measures include hepatitis B vaccinations, HIV exposure prophylaxis, standard precautions, and safety devices; however, safer technologies and enforcement of occupational safety and health regulations would greatly reduce needlestick injury rates.[2]

Common bloodborne pathogens include hepatitis B virus (HBV), hepatitis C virus (HCV), human immunodeficiency virus (HIV), cytomegalovirus, herpes simplex virus and parvovirus. Bloodborne pathogens exist within all patient populations and the rising number of carriers poses a significant occupational health hazard to providers.[2] Blood is the most common vehicle of transmission and HIV, HBV, and HCV are the most common causes of occupational-related infections.[3] Furthermore, high rates of exposure and potential risk for infection transmission exist when caring for infected patients. Risk of transmission depends on factors such as individual infectivity, clinical context, technical skill, and hospital environment.[5]

In this particular case report, the patient was known to have hepatitis C. Hepatitis is a disease of the liver most often due to a virus, but can also be caused by drugs or toxins. Hepatitis C is primarily transmitted via parenteral route and often results in chronic hepatitis and cirrhosis.[6] The Center for Disease Control (CDC) reports that 3.9 million individuals are infected with HCV.[5] According to the 2002 World Health Report, 40% of hepatitis C cases among healthcare workers were the result of occupational exposure.[3] Opportunities for exposure existed within this particular case, including IV placement, arterial line insertion, blood draws, and surgical manipulation. The patient had an arterial line available for blood draws yet no safe blood transfer devices were readily available for use when obtaining blood samples due to common practice with use of needles for blood draw transfer and absence of needleless safety devices as routine stock items within the supply carts. Although no incidents occurred during the case it was noted that a risk for needlestick injury and bloodborne pathogen exposure existed due to the patient's noted hepatitis status and the lack of safety device equipment available.

According to the American Association of Nurse Anesthetists (AANA) all CRNAs must uphold and adhere to ethical standards, ensure that the care they render reduces risks posed to patients and themselves from infectious agents, and take precautions to minimize the risk of infection to the patient, the CRNA, and other healthcare providers.[7] Ample literature exists reviewing and identifying needlestick injuries amongst healthcare workers. Needle usage and bloodborne pathogen exposure poses harm to patients and anesthesia practitioners during anesthesia care.

Various legislative acts have been enacted to address the issue of needlestick injuries and the importance of protection against bloodborne pathogens. The CDC initiated a Sharps Injury Action Plan in 2005, Congress enacted the Needlestick Safety and Prevention Act in 2000, and OSHA insisted that tools must be safe and reliable.[4] As a result of these initiatives, multiple companies have produced newer technologies to prevent needlestick injuries, yet individuals continue to suffer from accidental sticks due to shortcomings in safety device designs.

The importance and effectiveness of safety-engineered needles has been demonstrated to reduce sharps injuries and bloodborne pathogen exposure; however, there remain areas of weakness and poor compliance. Surgeons and anesthesiologists generally have been resistant to new safety equipment and the surgical setting is least likely to adopt safety-engineered devices.[8] Although sharp injuries have decreased since the initiation of safety acts, no such decreases have occurred in the surgical setting due to unresponsiveness to the adoption of safety measures.[1] Although safety devices would not completely eradicate needlestick injuries, they would aid in decreasing the frequency.[2] Occupational exposure management is very costly when considering the need for follow-up tests, prophylaxis after exposure, counseling, treatment, ethical and legal issues; therefore, prevention practices are not only safe and necessary but cost-effective.[5]

Utilizing appropriate equipment and implementing protective measures when obtaining blood samples is essential in ensuring a safe environment of care. It is of utmost importance to treat all blood samples with the same attention and safety measures when caring for patients. After use of a needle, the needle should never be used again to repuncture any item, including lab sample tubes, medication vials, intravenous tubing, or other patient related equipment. All needles should be appropriately handled and disposed after single use.

Healthcare workers, especially those in surgical settings, including CRNAs, continue to remain at the highest risk for exposure and contamination of bloodborne pathogens due to the nature of their work. A combination of increased awareness, proper education, strict regulations, and improved technologies must occur in order to decrease needlestick injury incidence and protect healthcare professionals against bloodborne pathogens.

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Mentor: Michael Rieker, CRNA, DNP, FAAN

Total Knee Arthroplasty: A Case Review of Unimodal Therapy

Carlos Camara, MSN
University of Southern California

Keywords: multimodal, total knee arthroplasty, opioids, degenerative knee disease, pain

Substantial medical advances in total knee arthroplasty (TKA) surgery have led to great benefits for degenerative knee disease.[1-2] Nevertheless, perioperative pain control remains suboptimal. Historically, patients' perioperative pain has been managed primarily with opioids.[1-7] Large opioid doses often correlate with untoward side effects such as somnolence, respiratory depression, nausea, and vomiting. Opioid related side effects might delay recovery thereby increasing the risk of medical complications and hospital length of stay.[1-7] Furthermore, suboptimal pain control and increased incidence of nausea and vomiting leads to patient dissatisfaction. This case report is an example of unimodal therapy where opioids are the only medication used for perioperative analgesia.

Case Report

A 60-year-old, 165.1 cm, 97 kg male with left knee osteoarthritis was scheduled for a left TKA. The patient's past medical history included Klinefelter's syndrome, obstructive sleep apnea, hyperlipidemia, hypertension, anxiety, and erectile disorder. He denied drug allergies and his current medications included testosterone 200 mg intramuscular every two weeks, simvastatin 40 mg by mouth every night, hydrochlorothiazide 25 mg/triamterene 37.5 mg by mouth every morning, and ibuprofen 800 mg by mouth every evening with the last dose 72 hours prior to surgery. The patient's surgical history included breast reduction for gynecomastia, appendectomy, multiple penile prostheses, and scrotal prosthesis. The physical exam revealed a Mallampati class II airway and nearly full neck range of motion. Preoperative vital signs and laboratory data were unremarkable. Intravenous (IV) midazolam 2 mg was administered in the preoperative area and the patient was transported to the operating room.

Standard noninvasive monitors were placed while the patient received oxygen at 10 L/min via facemask for 5 minutes. Hydromorphone 0.8 mg IV was administered. An IV induction was performed using lidocaine 50 mg propofol 150 mg, hydromorphone 0.4 mg, and succinylcholine 100 mg. Upon the loss of eyelash reflexes, the eyes were taped and the patient's trachea was intubated with a 7.5 cm endotracheal tube via direct laryngoscopy. Positive end tidal carbon dioxide ($ETCO_2$) and equal bilateral breath sounds confirmed the endotracheal tube placement. The endotracheal tube was secured at 22 cm at the teeth and respirations were resumed via mechanical ventilation (volume control at 10/min, tidal volume 600 mL, positive end expiratory pressure of 4).

General anesthesia was maintained with isoflurane end tidal 0.55% to 0.80% in a combination of oxygen and nitrous oxide, both at 1 L/min. In addition, four hydromorphone 0.4 mg IV boluses were administered for acute pain throughout the intraoperative period noted by hemodynamic changes of SBP ≥145 mm Hg accompanied by an increased HR ≥ 80/min. In order to minimize blood loss, tranexamic acid 1000 mg IV was administered and a left leg surgical tourniquet was inflated (total tourniquet time 22 minutes at 250 mm Hg pressure) prior to incision. Intravenous crystalloid fluids totaling 2200 mL were administered during the surgical procedure with an estimated blood loss of 550 mL and urine output of 125 mL. At the completion of the procedure the patient met extubation criteria and the endotracheal tube was removed from the trachea. Supplemental oxygen 8L/min via facemask was provided to the patient who was transported to the post-anesthesia care unit (PACU) in stable condition. The patient remained in the PACU for 150 minutes where he was somnolent, but easily aroused by voice command. The patient's pain level was 5/10 and he received a one-time dose of morphine 2 mg IV while in PACU reducing the pain level to 0/10. No reports of nausea and vomiting were noted.

Discussion

Total knee arthroplasty is a painful surgery. Adequate pain control allows for increased patient satisfaction, faster rehabilitation and decreased postoperative complications.[1-4] Unimodal opioid therapy is frequently used for pain control following TKA; however, the use of opioids alone generally results in suboptimal pain control. Often, large doses of opioids are required to provide adequate pain control leading to increased incidence of untoward opioid side-effects such as somnolence, respiratory depression, nausea, and vomiting. The literature review for this case report does not contraindicate the use of opioids for pain management for TKA; however, the data reveal that multimodal therapy is a more effective approach for pain control.[1-7] Multimodal therapy utilizes the additive and synergistic effects of distinct classes of pain medications to attain adequate pain control. Furthermore, multimodal analgesia decreases opioid related side effects, improves patient satisfaction, decreases hospital length of stay and leads to a faster recovery.[1-7]

A study by Lamplot et al. divided patients undergoing TKA into two groups. Group 1 patients received a multimodal pain management including a peri-articular injection of 0.5% bupivacaine 30 mL, morphine sulfate 10 mg, and ketorolac 15 mg before skin closure. They also received tramadol 50 mg orally every 6 hours, ketorolac 15 mg IV every 12 hours, and oxycodone 10 mg orally every 12 hours for the first 48 hours. For breakthrough pain, the patients received hydrocodone 5 mg orally or hydromorphone 1 mg IV via patient controlled analgesia (PCA),

both as needed. Group 2 patients received a hydromorphone PCA and hydromorphone 1 mg bolus IV as needed. The multimodal group showed a significantly decreased visual analog pain scale at rest ($p < 0.0004$), decreased hospital length of stay (mean of 1.9 days in multimodal group vs. 2.3 days in PCA group), increased patient satisfaction ($p < 0.05$), decreased opioid consumption (66.2 morphine equivalents (ME) ± 12.8 for the multimodal group vs 150.4 ME ± 35.81 for the PCA group, $p < 0.0004$), decreased opioid related adverse effects (16% incidence in the multimodal group vs. 94% for the PCA group), and decreased time to physical therapy milestone achievement (100% of multimodal patients were able to get out of bed on postoperative day 0 vs. 69% of PCA patients).[6]

Xiao et al. divided osteoarthritis patients undergoing unilateral TKA into two groups. The control group received oral placebo one day prior to surgery and continuing for one month after the surgery. They also received an intraoperative intra-articular placebo injection. The trial group received oral celecoxib 200 mg and tramadol 0.1 mg, both twice daily, starting one day prior to surgery and continuing for one month after surgery. They also received an intra-articular injection of morphine 5mg, ropivacaine 150 mg (7.5:1,000), epinephrine (1:1,000) 0.5mL, and betamethasone 1 mL intraoperative. Both groups received morphine PCA with a 0.5 mg bolus, 6-min lock-out, and a maximum rate of 5 mg/h for 48 hours after the surgery. The trial group had significantly lower morphine consumption up to 48 hours post-surgery with a significantly lower incidence of nausea and vomiting ($p<0.05$ for all comparisons). Furthermore, the trial group indicated significantly lower pain levels at rest and with activity on the visual analog scale ($p<0.05$ for all comparisons)..[7]

Sinatra et al. studied patients undergoing total hip arthroplasty or TKA. The patients were randomly divided into three postoperative groups: group 1 received acetaminophen 1 g IV every 6 hours for 24 hours; group 2 received proparacetamol (prodrug) 2 g IV every 6 hours for 24 hours; group 3 received placebo every 6 hours for 24 hours. All groups were also provided morphine PCA and bolus morphine as needed for analgesia. When compared to the control group, the acetaminophen group and the proparacetamol group exhibited significantly superior pain control and lower requirement for morphine (33% reduction in morphine consumption for the acetaminophen group and 29% reduction in morphine consumption for proparacetamol group).[8]

The patient in this case received unimodal opioid therapy for perioperative and postoperative pain control. A high dose of opioids was required to control this patient' pain. Intraoperatively, the high dose and frequency of opioid requirement indicates a suboptimal pain control for this patient; however, postoperatively the patient's pain level was well controlled after one dose of opioid. The literature review shows the use of a multimodal pain management approach following TKA provides more effective pain control with fewer side effects than the unimodal pain management used in this case. According to the literature review a multimodal pain management approach would have provided a superior pain regimen for the patient, avoid untoward opioid side effects, and improve patient satisfaction. Patients can benefit from the use of additive and synergistic properties of anesthetics and analgesics and the anesthesia practitioner should use these modalities in order to provide the highest quality, most efficient, and safest anesthetic to patients.

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Mentor: Terrie Norris, CRNA, EdD

The Anticoagulated Patient with Antiphospholipid Syndrome

Margaret Melissa Loayza, MN
Louisiana State University Health Sciences Center

Keywords: antiphospholipid syndrome, Hughes syndrome, prothrombin complex concentrate, tubal ligation, endometrial ablation, retroperitoneal hematoma

The autoimmune disorder antiphospholipid syndrome (APLS), also known as Hughes syndrome, is characterized by a hypercoaguable state manifesting as arterial and venous thromboses.[1] Antiphospholipid syndrome is diagnosed by the presence of specific clinical and laboratory criteria.[1] Antiphospholipid syndrome treatment includes anticoagulation and corticosteroids, as well as immunosuppressants if necessary.[2] Surgery in an anticoagulated patient with APLS presents significant hematological and surgical concerns to the anesthesia professional. Knowledge of the use of human prothrombin complex concentrate for the acute reversal of warfarin is essential.

Case Report

A 30-year-old, 63 kg, 163 cm, female patient presented for emergent exploratory laparoscopy on her third postoperative day following tubal ligation and hydrothermablation. Medical history

included: antiphospholipid syndrome, lupus anticoagulant positive and deep vein thrombosis during pregnancy four years prior to this surgical event. Laboratory values included: hemoglobin 9.1 g/dL, hematocrit 26.3 %, white blood cells 10.6 x 10^3/µL, platelets 162,000/µL and chemistries within normal limits. Coagulation studies were: international normalized ratio (INR) 6.4, prothrombin time 75.9 seconds, partial thromboplastin time 47.7 seconds. Her electrocardiogram showed sinus tachycardia with non-specific ST changes. Computed tomography of the abdomen and pelvis showed a 12 x 12 cm hematoma. She complained of shortness of breath since POD 1 which she related to pain. Physical examination revealed regular heart tones with a rate of 140/min. Her abdomen was moderately distended. Inpatient medications included: ampicillin-sulbactam sodium, methylprednisolone sodium succinate, hydrocodone-acetaminophen, famotidine, and warfarin. Outpatient medications included the last three of previously mentioned medications. Peripheral intravenous access was obtained with two 16 gauge catheters.

In the holding area the patient received a one-time dose of vitamin K 2 mg intravenously (IV) and four units of fresh frozen plasma (FFP). Thirty minutes later a one-time dose of a non-activated factor form of prothrombin complex concentrate (PCC) 2,148 units was administered IV for a reduction in the INR value to 2. The patient was given midazolam 3 mg and fentanyl 100 mcg IV in divided doses. Methylprednisolone sodium succinate 125 mg was administered IV.

Upon arrival to the operating room, standard monitors were applied. Initial vital signs included a room air oxygen saturation (SpO2) of 98%, blood pressure 115/85 mm Hg, heart rate 130/min and core temperature 38.1^{O} C. The patient was preoxygenated and a rapid sequence induction with cricoid pressure was initiated using propofol 100 mg, lidocaine 60 mg, and succinylcholine 100 mg IV. Successful atraumatic laryngoscopy was accomplished using a Miller 2 blade and a 7.0 mm internal diameter endotracheal tube was inserted into the trachea. A 2.2% end-expired sevoflurane concentration was used to maintain general anesthesia and rocuronium 20 mg was used to maintain neuromuscular blockade. Steroids had been given thirty minutes prior and were not redosed. Four units of packed red blood cells and 1.7 L of crystalloids were administered during the procedure. Fentanyl 150 mcg was administered IV in divided doses. Post tranfusion hemoglobin and hematocrit were 32.3 % and 11.9 g/dL respectively. Ondansetron 4 mg was given IV.

The surgeons evacuated the hematoma, but found no active source of bleeding. At the conclusion of the procedure, the neuromuscular blockade was antagonized with neostigmine 3 mg and glycopyrolate 0.4 mg IV. The patient's oropharynx was suctioned and after extubation criteria were met the endotracheal tube was removed. She was taken to the post anesthesia recovery unit (PACU) in stable condition.

Discussion

Antiphospholipid syndrome is an autoimmune disorder that affects 2% of the population, 70% of which are female.[3, 4] It was first described in 1983 by Graham R.V. Hughes of London Hammersmith Hospital.[5] Since that time, the pathogenesis of APLS and the antibodies involved are still not well understood.[3] Antiphospholipid syndrome is characterized by at least one and

often numerous thromboses in veins and arteries.[5] Various molecular routes are involved in APLS clot formation such as defective fibrinolysis and obstruction of anticoagulant pathways.[2] In APLS, certain antiphospholipid antibodies are present, such as: Lupus anticoagulant (LA), anticardiolipin (aCL), or anti-beta 2 glycoprotein-I (anti-β_2GPI).[5] These antiphospholipid antibodies create heterogeneous interactions with phagocytic white blood cells, thrombocytes and endothelial cells.[2]

Antiphospholipid syndrome can occur as a primary condition or it can occur secondary to another autoimmune disease such as systemic lupus erythematosus, infections, medications, or malignancies.[5] Antiphospholipid syndrome is known to be associated with certain conditions such as injury to heart valves, antiphospholipid autoantibody-associated nephropathy, thrombocytopenia, livedo reticularis, and central nervous system symptoms.[1, 3, 5]A minimum of one clinical criterion and one laboratory criterion must be present for the diagnosis of APLS.[1] Clinical criteria include: vascular thrombosis or pregnancy morbidity such as unexplained fetal death or prematurity. Laboratory criteria include the presence of LA, aCL, or anti-β_2GPI on at least two separate occasions with a minimum of 12 or more weeks between laboratory blood testing.[1] This patient received the diagnosis of APLS after presenting with a DVT in her lower extremity during pregnancy and positive LA blood analysis. She remains LA positive.

A retrospective analysis[6] of APLS patients showed an increased risk of future thrombotic events in those who tested positive for LA, aCL, and anti-β_2GPI. Although oral anticoagulant therapy can greatly diminish the risk of thromboembolism in these patients, the recurrence of thrombotic events remains high.[6] Current literature suggests there is a high probability of recurrent arterial thrombotic events.[2] This is especially true for patients with anti-β_2GPI which has clinically demonstrated a strong correlation with thrombosis.[1, 2] Conventional therapy to induce anticoagulation is with warfarin and it should be continued indefinitely with a target INR of 2.0-3.0 if administered with acetylsalicylic acid and higher if not. Low molecular weight heparin is also an option, but heparin-induced thrombocytopenia is a possible side effect. Newer oral anticoagulants, such as rivaroxaban, are being evaluated for efficacy in prophylactic thrombotic therapy in APLS patients.[2] Corticosteroids and intravenous immunoglobulin may also be used in the treatment of APLS.[2] This patient was not taking daily corticosteroids, however methylprednisolone sodium succinate 125 mg IV was administered empirically thirty minutes prior to surgery. She was on daily warfarin with an INR goal of 2.5 to 3.5. She presented to the preoperative holding area on postoperative day (POD) 3 after having a bilateral tubal ligation with endometrial ablation.

With an INR of 6.4 and internal bleeding, the warfarin had to be urgently reversed in order to safely take the patient to surgery. Two reversal therapies were used for this patient. Four units of FFP as well as a non-activated four-factor form of PCC was administered IV to reverse the warfarin. The PCC used contained coagulation factors II, VII, IX, and X, and antithrombotic proteins C and S.[7] The administration of PCC quickly increased those factors and reversed the anticoagulation process. Vitamin K was administered with PCC so that Vitamin K coagulation factors would be sustained after PCC's effects decreased.[7] It is important to note that an excessive dose of vitamin K should be avoided, so that when warfarin is restarted it is not refractory.[8] Of importance to this case was attention to atraumatic direct laryngoscopy.

It is essential for the anesthesia provider to understand risks, complications, and benefits associated with the administration of PCC. A patient with APLS is at risk for a thromboembolic event if their anticoagulation is reversed. Therefore, there is an inherent risk for a fatal or non-fatal thromboembolism with the use of PCC. During this particular case, blood products and crystalloids were administered, the patient was kept normothermic and sequential compression devices were used. Awareness for signs of pulmonary embolism, such as sudden loss of end-tidal CO_2, tachycardia, or sudden hypotension were employed.

A benefit to using PCC versus FFP is decreased volume, cross-matching is not required, it is virally inactivated and can be administered quickly. Studies have demonstrated thirty minutes after initiating PCC, the INR decreases from above 3 to 1.2 verses 2.4 with FFP.[7] However, there is evidence that PCC has a higher incidence of thromboembolic events compared to FFP.[7] The literature suggests there is a decreased risk for thrombosis with inactivated clotting factor concentrates.[7] In this particular case, an inactivated form of PCC was used. Other precautions with PCC are hypersensitivity reactions such as nausea, vomiting, dyspnea, bronchospasm, pulmonary edema, hypotension, and tachypnea.[7] It is also important to note that although PCC is virally inactivated, there is still a risk of transmitting infectious material since it is made from human blood products.[7]

Since the description of APLS in the early 1980s, it has become a more frequently diagnosed autoimmune disease.[3, 5] It is important for the anesthesia practitioner to be familiar with APLS due to the risks of the disease, the anticoagulation treatment and reversal of this treatment. Vigilant monitoring for bleeding or thromboembolic events must be employed, as well as extreme care during potentially traumatic procedures such as laryngoscopy. It is also essential for anesthesia practitioners to be knowledgeable of the dosing and administration of PCC, since it is possible to have a patient with this syndrome at some point during one's career.

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Mentor: David M. Kalil CRNA, DNAP

Propofol, Alfentanil, and Lidocaine in Outpatient Cataract Surgery

Janiece Miller, MSN
University of Southern California

Keywords: propofol, alfentanil, lidocaine, sedation, cataract, outpatient

Cataract surgery, the most common procedure in ophthalmic surgery, can be performed using a variety of anesthetic techniques.[1] Although topical anesthesia during cataract surgery is becoming increasingly popular, it is not suitable for use on all patients. Patients with corneal opacity, complex cataracts, and surgeon preference are a few reasons warranting the use of a regional nerve block in combination with intravenous (IV) sedation for cataract surgery.[2] This case study focuses on the administration of propofol, alfentanil, and lidocaine together (referred to as the 6-2-2 mixture) to provide anxiolysis, amnesia, and analgesia during retrobulbar block placement for cataract surgery.

Case Report

A 75-year-old, 108 kg, 183 cm male with a body mass index (BMI) of 32 presented for right eye cataract surgery. The past medical history included hypertension (HTN), coronary artery disease, a myocardial infarction in 1999, ischemic cardiomyopathy, single chamber automatic implantable cardioverter-defibrillator device (AICD), hyperlipidemia, pneumonia, poorly controlled type 2 diabetes mellitus, gastroesophageal reflux disease, diverticulitis, obesity, obstructive sleep apnea (OSA) requiring continuous positive airway pressure (CPAP), and lumbar back pain. The surgical history included a 4-vessel coronary artery bypass graft, cholelithotomy, placement of an AICD, and perforated colon repair. The patient had no known drug allergies. Medications consisted of amlodipine, aspirin, atorvastatin, bisoprolol, cyclobenzaprine, docusate, fenofibrate micronized, hydrocodone-acetaminophen, insulin glargine, insulin lispro, insulin NPH, regular insulin, ketorolac, potassium chloride, and valsartan-hydrochlorothiazide.

An electrocardiogram showed sinus rhythm with a rate of 66/min, left axis deviation, and an old inferior infarct. The echocardiogram identified a left ventricular ejection fraction of 51%, pulmonary pressure of 37 mm Hg, mild concentric hypertrophy, mildly decreased left ventricular systolic function with mild hypokinetic inferior wall motion, left ventricular grade I diastolic dysfunction, a mildly dilated left atrium, mild aortic insufficiency, and mild mitral and tricuspid regurgitation. A thallium stress test findings included wall motion abnormalities in the inferior wall segments. The chest x-ray showed moderate cardiomegaly and no acute cardiopulmonary disease. All laboratory values were within normal limits with the exception of hemoglobin A1c at 8.4% and glucose at 132mg/dL.

The anesthetic plan consisted of monitored anesthesia care with a retrobulbar block. The preoperative vital signs included a heart rate of 66/min, blood pressure 156/83 mm Hg, respiratory rate 20/min, SpO2 98% on room air, and temperature 98º F. The physical examination was unremarkable.

On arrival to the operating room, standard monitors were placed on the patient and oxygen was administered via nasal cannula at 3 L/min. End-tidal CO_2 was measured through the nasal cannula. Three minutes prior to the retrobulbar block placement, a mixture of propofol 30 mg, alfentanil 500 mcg, and lidocaine 10 mg (totaling 5 ml) was administered IV. During this time, a conversation was maintained with the patient to assess his level of sedation. Two minutes later, another bolus mixture of propofol 6 mg, alfentanil 100 mcg, and lidocaine 2 mg (totaling 1 ml) was administered. Once the patient's speech became slurred and he appeared relaxed, the retrobulbar block, (4 mL of 2% lidocaine), was placed by the surgeon. The patient tolerated the block placement well with a chin lift to prevent airway obstruction and verbal reminders to take a deep breath. No patient movement during the block was noted. Four subsequent 1 ml boluses of propofol 6 mg, alfentanil 100 mcg, and lidocaine 2 mg were administered IV every 3-4 minutes. Vital signs remained stable within 20% of baseline throughout this time.

The total surgical procedure time was 21 minutes. Upon transport to the post anesthesia care unit, the patient was alert, oriented, and stable on room air. Fluid administration included 250 mL crystalloid with no blood loss. Vital signs remained within normal limits. The patient reported no nausea, vomiting, or postoperative pain prior to being discharged home that afternoon.

Discussion

As cataract surgery has advanced in recent years due to the introduction of phacoemulsification and foldable intraocular lens, so has anesthesia for cataract surgery.[1-3] Once performed under general anesthesia, cataract surgery has moved to local and topical anesthetic techniques over the past 25 years. Although newer surgical techniques have lessened the need for ocular immobility, patient co-morbidities, complex cataracts, complicated ocular co-morbidities, patient preference, and surgeon experience and preference are important factors for consideration when deciding on an anesthetic plan.[2,3] With a high majority of cataract surgeries occurring in the geriatric population and the outpatient setting, early recovery and rehabilitation are important. Catecholamine release from pain and anxiety can lead to HTN, tachycardia, and hyperventilation, which can negatively affect a geriatric patient with multiple co-morbidities and declining organ function leading to poor outcomes, longer PACU stays, and higher costs.[1,4] In this case study, numerous factors necessitated the need for placement of a retrobulbar block combined with IV sedation including an extensive cardiac history, a complex cataract, and surgeon preference.

While placement of a retrobulbar block allows for complete ocular immobility during cataract surgery, placement of the block can cause serious risks ranging from optic nerve damage and retrobulbar hemorrhage to total spinal blockade, cardiopulmonary arrest, and death.[1,2,5] Any movement made by the patient during block placement can increase these risks. If the anesthetic

plan consists of a retrobulbar block, patients must be given sedation prior to block placement to provide anxiolysis, amnesia, and analgesia to ensure patient comfort and minimize the risks associated with block placement.[4,6]

The ideal sedative agent for outpatient ophthalmic surgeries should have predictability with a rapid onset and offset, minimal side effects, allow for early recovery and rehabilitation, and be cost effective.[4] No ideal agent for sedation exists and no sedation regimen has proven to be superior.[1,3] However, the successful use of propofol and alfentanil for sedation during cataract surgery has been identified for many years. In a randomized control trial of 40 patients, those patients that received both propofol and alfentanil, administered separately, experienced amnesia and analgesia during retrobulbar block placement and had higher satisfaction rates, whereas patients that received only alfentanil experienced recall of the block.[5] In this same study, the addition of alfentanil to propofol also decreased patient movement in a dose-dependent manner during retrobulbar block placement. However, doses of alfentanil exceeding 15mcg/kg caused significant hypoventilation.[5] A more recent study also successfully demonstrated the use of propofol and alfentanil administered together, along with lidocaine to provide anxiolysis, amnesia, and analgesia during retrobulbar block placement.[6] Adding lidocaine to the mixture serves to diminish the pain associated with propofol administration. The safety of the 6-2-2 drug combination was established in this clinical study.[6]

This 6-2-2 mixture is pre-mixed to a 10 ml total volume of the three medications. This includes propofol 6 ml (10 mg/ml for 60 mg), alfentanil 2 ml (0.5 mg/ml for 1 mg), and 1% lidocaine 2 ml (10 mg/ml for 20 mg). Therefore, each 1 mL of the 6-2-2 mixture in the 10 ml total volume is equal to: propofol 6 mg, alfentanil 100 mcg, and lidocaine 2 mg. Dosing of the 6-2-2 mixture is based on the alfentanil concentration and is administered according to the patient's age. Patients younger than 45 years of age receive a bolus dose of 9 mcg/kg of alfentanil. The dose is decreased by 1 mcg/kg per 10-year increase in the patient's age with the lowest dose at 5 mcg/kg of alfentanil for patients greater than 75 years of age. The initial alfentanil dose for this 75 year-old male was 5 mcg/kg, equaling 500 mcg of alfentanil or a 5 ml bolus of the 6-2-2 mixture (which would include propofol 30 mg and lidocaine 10 mg) given prior to retrobulbar block placement. Subsequent doses were titrated to the patient response to provide optimal conditions for retrobulbar block placement, which include slurred or absent speech, closure of eyes, and immobility during the block.

Alfentanil, a fentanyl analogue that is 5 times less potent, has quick onset and offset pharmacokinetics, and can cause significant hypoventilation, especially in the geriatric population.[1] Considering the pharmacologic activity and evaluating the patient (BMI 32, history of OSA, and 75 years of age) the decision to proceed with the 6-2-2 procedure was based on familiarity with the technique and former success. Chin lift and verbal reminders were effective in this case, and may be necessary in other cases using this technique. Alternative sedative techniques include dexmedetomidine, an alpha 2-receptor agonist. This drug has proven to be an effective sedative agent in cataract surgery and could have been administered to this patient as alternative therapy. Dexmedetomidine can provide anxiolysis, sedation, and analgesia, while preserving respiratory drive, which are the primary goals to achieve during sedation for retrobulbar block placement.[7] Prolonged sedation leading to longer PACU stays and hypotension are among the adverse effects of dexmedetomidine.[1,4]

When placing a retrobulbar block for cataract surgery, selection and titration of sedative agents for each patient must be evaluated and performed to achieve optimal conditions including anxiolysis, analgesia, and immobility. At the same time, avoiding or minimizing the potential adverse effects, specifically hypoventilation, must be achieved. The 6-2-2 mixture is one regimen that can be utilized for sedation during retrobulbar block placement.

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Mentor: Michele Gold, CRNA, PhD

Naloxone for Postoperative Opioid-induced Respiratory Depression

Melanie Maraghy, MSN
University of Southern California

Keywords: Opioid-induced respiratory depression, antagonist, naloxone, receptor

Opioids are widely utilized in the perioperative period to prevent and treat pain. They are highly efficacious in this regard, yet may present associated side effects, of which opioid-induced respiratory depression (OIRD) is particularly concerning for the anesthesia practitioner. Certain patient groups are at a higher risk for this, including the morbidly obese, patients who suffer from sleep apnea, patients with specific neuromuscular diseases, the very young, the very old, and the critically ill.[1] In the event of significant postoperative OIRD, the opioid receptor antagonist naloxone is the definitive treatment for this potentially life-threatening complication.

Case Report

A 50-year-old male (60 kg, 163 cm) presented for a right lower extremity below-knee amputation secondary to a severe right foot infection. The past medical history was significant

for hypertension and poorly controlled type 2 diabetes mellitus, both diagnosed 12 years prior. Medications for this patient included metformin, regular insulin infusion, piperacillin sodium/tazobactam sodium, vancomycin hydrochloride, clindamycin hydrochloride, and hydralazine, as needed. The patient had no known drug allergies and denied any tobacco, alcohol, or illicit drug use. The patient had no previous surgeries or any known family history of anesthesia related complications.

After the anesthesia equipment was verified and anesthesia machine checkout performed, the patient was identified, interviewed, and assessed for anesthesia care. The patient was brought to the operating room and assisted to the operating table, standard monitors were applied and oxygen was delivered at 8 L/min via face mask to the patient. A one-liter bag of lactated ringers was hung and attached to the patient's existing 18 gauge peripheral intravenous (IV) catheter. Midazolam 2 mg was administered to aid in anxiolysis. Anesthesia was induced, easy mask ventilation was established and a direct laryngoscopy performed. A 7.0-mm endotracheal tube was easily placed in the trachea, just beyond the vocal cords and secured at 21 cm at the lip. Respirations were controlled by mechanical ventilation. Positioning of the tube was confirmed by auscultation of bilateral breath sounds and positive end tidal carbon dioxide.

The patient was positioned supine, with bilateral arms on arm boards, and a warming blanket placed on the patient's upper body. A tourniquet was applied to the right lower extremity, which was in place for 12 minutes at 250 mm Hg pressure. Throughout the approximately 3 hour-case, the patient received midazolam 4 mg, hydromorphone 2 mg, lidocaine 50 mg, propofol 140 mg, rocuronium 50 mg, phenylephrine 160 mg, ondansetron 4 mg, neostigmine 2 mg, glycopyrrolate 0.4 mg, 1 liter lactated ringers, and 500 mL 5% albumin. The hydromorphone was administered, in part, early on during the case, as a narcotic loading dose. Once the patient was beginning to resume breathing during the case, the remainder was titrated in for a respiratory rate goal of less than 20 breaths per minute towards the end of the surgery in 0.2-0.4 mg doses at a time. Urine output during the case was 700 mL and estimated blood loss was 20 mL.

At surgery end, the patient had received antagonism of the remaining neuromuscular blockade and exhibited sustained tetany for 5 seconds, however was no longer breathing spontaneously. With assistance to initiate a breath via gentle squeezing of the the anesthesia machine bag reservoir, the patient would occasionally then draw in additional air to complete an initiated breath with an adequate tidal volume. With this assistance in initiating breaths, the patient would complete 4-6 breaths per minute with tidal volumes over 1,000 mL. The oxygen saturation dropped slightly during this time, never falling below 94%. Verbal and physical stimulation attempts were made with no movement or increased ventilatory response from the patient. Naloxone 0.04 mg was administered and the patient began initiating his own breaths without assistance and achieved adequate tidal volumes of 6-8 mL/kg within 5 minutes. The trachea was extubated successfully and the patient maintained adequate respiratory rate and tidal volume throughout transport to the intensive care unit and sign off of anesthesia care, without the need for additional doses of naloxone. The patient denied any pain during this time.

Discussion

Opioids can induce respiratory depression through a centrally mediated decrease in the involuntary respiratory rate with a potential decrease in oxygen saturation. In OIRD, the respiratory rate is decreased which, in severe cases, can lead to low oxygen saturation, high arterial $PaCO_2$ values, and decreased ventilatory response to hypoxia and hypercapnea. The oxygen saturation may not decrease in spite of a decreased respiratory rate, due to a corresponding increase in tidal volume with conservation of minute ventilation. In severe cases, this compensatory mechanism fails and the patient can develop respiratory failure with a decreased respiratory rate and a subsequent decrease in blood oxygenation.[2]

When faced with postoperative OIRD, the first consideration should be whether the patient can be managed by conservative measures such as rousing through verbal and physical stimulation and oxygenation.[2] This technique was attempted, although the patient did not respond effectively, and the opioid antagonist, naloxone, was administered. Naloxone is a competitive antagonist of all three opioid receptors (mu, kappa and delta).[3] Mu receptors include two subtypes, mu1 and mu2. Mu1 receptors cause analgesia, euphoria, and serenity, while mu2 receptors relate to respiratory depression, pruritus, prolactin release, dependence, anorexia, and sedation.[4] Kappa receptors cause analgesia, sedation, dyspnea, dependence, dysphoria, and respiratory depression, while delta receptors are believed to cause psychomimetic and dysphoric effects.[4] Naloxone's greatest affinity is for mu receptors and the antagonistic effect is highly efficacious; small doses may reliably reverse or prevent the effects of opioid agonists.[3] If naloxone is needed despite conservative measures, as with this patient, it should be titrated on an individual basis to obtain an optimal respiratory response while maintaining adequate analgesia and do not titrate to pain or consciousness level, as acute reversal of opioid analgesia can be detrimental to the patient.[2] Respiratory depression occurs at higher receptor occupancy than some degree of analgesia, and analgesia is not compromised when titrating naloxone to respiratory effect.[1] This phenomenon was exhibited in this patient, since following a single administration of low dose naloxone (0.04 mg), the OIRD was adequately reversed, yet he still denied postoperative pain.

There is little evidence-based research that supports an optimal naloxone dose to be given in order to reverse OIRD, while still maintaining analgesia. Bailey et al conducted a double-blind, randomized controlled trial which showed that, when titrated in small doses, naloxone is effective as a postoperative antagonist of respiratory depression. They found that prior studies reporting serious complications associated with naloxone (hypertension, pulmonary edema, cardiac arrest) may have been due to the larger doses administered in those instances, since the recommended dose for naloxone was initially 0.4-0.8 mg.[5] In this study, they gave doses of naloxone of 0.08, 0.16, or, at most, 0.24 mg, which they found to be all that was necessary for naloxone reversal of opioid-induced respiratory depression. The 0.16 and 0.24 mg doses were given as 0.08mg doses, x2 or x3 respectively, titrated based on the reassessment of respiratory status every 2 min postoperatively, due to renarcotization. They concluded that naloxone is efficacious and safe, yet there remains a need to observe for and treat renarcotization.[5]

After giving the initial dose of naloxone, this patient was monitored closely to assess if additional doses would be required, of which were none. The onset of the antagonist effect of

naloxone is very rapid, but the duration of action is quite brief, frequently shorter than that of the opioids it is used to antagonize. Small incremental doses (0.04 mg in an adult, every 3 minutes) can be given IV, usually with dramatic improvements.[3]

Opioid reversal can cause increases in systemic blood pressure, heart rate, and plasma levels of catecholamines. This may be due to a sudden onset of pain, but these effects have been reproduced experimentally in the absence of painful stimuli.[6] There exists a potential for sudden pulmonary edema, dysrhythmias, and even death in young, previously healthy individuals given naloxone, yet this is rare and the etiology of this remains largely unknown.[7] When naloxone is given to a patient who is hypovolemic, hypotensive, or previously in severe pain or stress, high-dose naloxone and/or rapidly infused naloxone (not titrated) can cause catecholamine-mediated cardiac arrhythmias and vasoconstriction, resulting in pulmonary edema. When naloxone is titrated to effect, it is generally safe. The possibility of complications and recurrence of sedation and respiratory depression should always be anticipated when treating a patient with naloxone.[1] This patient's vital signs remained stable from time of emergence of anesthesia through the transfer of care.

Following emergence of anesthesia, an essential goal is for effective pain relief without complications. As OIRD is a potentially fatal complication of opioid administration, it can usually be treated safely and efficiently with careful titration of the opioid antagonist, naloxone, followed by vigilant monitoring for effectiveness and possible adverse reactions, although more research is particularly warranted regarding an optimal dose.

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Mentor: Michele E. Gold, CRNA, PhD

The Use of Tranexamic Acid in Orthopedic Surgery

Amanda Jernigan, MSN
University of Southern California

Keywords: total knee arthroplasty, tranexamic acid, blood loss sparing techniques, anti-fibrinolytic therapy

Approximately 700,000 knee replacement procedures are performed annually in the United States and these operations are projected to increase by 390% to 3.48 million annually by 2030.[1] Total knee arthroplasty (TKA) procedures are associated with perioperative bleeding and an increased probability of blood transfusions which can pose an increased risk to patients. Tranexamic acid (TXA) is an anti-fibrinolytic drug therapy used in orthopedic procedures that has been shown to reduce blood loss. This case study evaluates the use of tranexamic acid in orthopedic knee surgeries and its effect on patient outcome based on total blood loss, transfusion risk and patient safety.

Case Report

A 72-year-old, 112 kg, 185 cm male presented for a right total knee arthroplasty secondary to knee osteoarthritis. Past medical history included a 10-year history of hypertension, coronary artery disease, and atrial fibrillation, as well as type two diabetes mellitus and sleep apnea for 5 years. He had a myocardial infarction two years ago and a drug eluding stent was placed in his right coronary artery. The patient was on sitaglipton 100 mg, carvedilol 6.25 mg, losartan 25 mg, simvastatin 40 mg, glyburide 2.5 mg, furosemide 80 mg, regular insulin sliding protocol, multi-vitamins, and warfarin 3 mg. He had taken losartan and simvastatin the morning of the procedure and stopped warfarin 5 days prior.

Pertinent lab information included a hematocrit of 43.7%, hemoglobin 14.1g/dL, INR 1.2, creatinine1.52mg/dL, and serum glucose of 94mg/dL. The transthoracic echocardiogram four months previously demonstrated a left ventricular ejection fraction of 50% and the stress echocardiogram completed three days prior was negative for ischemia. On physical exam, the patient was noted to be in atrial fibrillation with a heart rate of 110 and his other vital signs were unremarkable.

In the preoperative area, an intravenous catheter was started. Midazolam 2mg IV was administered prior to prepping for a peripheral block. The pain team fellow placed a right femoral nerve 20g indwelling catheter in the adductor canal under sterile fashion. It was activated in the preoperative area with ropivicaine 0.5% 20 ml. In the operating room, standard monitors were placed and the patient was assisted to a seated position. The patient's back was sterilely prepped and draped and a cutting bevel spinal needle was inserted in the L3-L4 interspace. After the spinal needle placement was confirmed by free flow of cerebral spinal fluid, lack of blood and negative parasthesia, bupivacaine 0.5% 1.5 ml was administered. The patient was placed supine and pre-oxygenated with the anesthesia circuit mask for five minutes. The IV induction consisted of lidocaine 50 mg, propofol 150 mg and succinylcholine 80 mg. A size 8-cuffed oral endotracheal tube (ETT) was inserted through the vocal cords under direct

laryngoscopy and secured after placement was confirmed. Anesthesia was maintained with rocuronium and sevoflurane 1.2%-1.5% inspired concentration in a mixture of oxygen and air, both at 1 L/min.

Tranexamic acid 1 g IV was administered five minutes after surgical incision and two minutes prior to tourniquet inflation. The tourniquet was inflated to 275 mm Hg on the right thigh for 72 minutes total time. The patient was hemodynamically stable throughout the case. Intraoperative arterial blood gas results were pH 7.43, pCO_2 38, pO_2 197, HCO_3 20, BE -5.9. The patient received a total of 2500 ml of crystalloid and 500 ml of albumin 5%. Estimated blood loss during surgery was 50 ml.

Upon emergence, the ETT was removed when extubation criteria were met. The patient was transported to the recovery room where he remained stable and was subsequently transferred to the floor after one hour. He had a total of 185ml of blood loss into a closed system exudate drain over 24 hours, and his hematocrit was 37% the morning after surgery.

Discussion

Many orthopedic procedures are associated with a large blood loss. Blood transfusions increase the patient's risk of immunological reactions and contracting blood born infections. Techniques to avoid allogenic blood transfusions in orthopedic surgery include tourniquets, regional anesthetic techniques, normovolemic hemodilution, and autologous blood donation. The blood loss for TKA procedures is primarily in the postoperative phase because of hyper-fibrinolysis.[2] Postoperative blood loss, even with conservative therapies, can average 1000-1800ml.[3] Therefore, anti-fibrinolytic medications are being used as adjuncts to decrease blood loss.[2]

Tranexamic acid is an anti-fibrinolytic that is inexpensive, widely available and has been shown to reduce blood loss in orthopedic procedures.[4] Tranexamic acid is a synthetic amino acid that competitively inhibits activation of plasmin by blocking the lysine binding sites of plasminogen, which in turn, stops the lysis of polymerized fibrin.[4] A concern regarding the use of TXA is the theoretical increased risk of systemic thrombosis, although no studies have demonstrated a statistically significant increase in detrimental pro-thrombotic effects for the lysine analogue. Tranexamic acid crosses the blood brain barrier and studies have reported that high doses (>100mg/kg) are an independent risk factor for postoperative seizures in all patients. In laboratory mice, the drug has been shown to be a competitive antagonist of glycine and this could account for the seizures; however, the deleterious side effects have not been shown to be associated with low dose regimens as used in orthopedic cases. [4]

In a 2014 meta-analysis by Huang et al, researchers reviewed 46 randomized controlled trials involving 2925 patients. Twenty-one studies evaluated low dose TXA (<15mg/kg) and 18 studies used a high dose (>15mg/kg) regimen. A single bolus was utilized in 20 studies and repeated doses were used in 26 studies. Twenty-one of the studies were specific to knee procedures. According to the meta-analysis, TXA reduced total blood loss by a mean volume of 408.33ml ($p<0.0001$) and decreased the probability of a transfusion by 49% ($p< 0.00001$). In addition, there was a positive effect whether TXA was delivered in single or multiple doses and

regardless of the type of orthopedic surgery. There was no statistically significant increase in thromboembolic events with the use of tranexamic acid.[5]

A 2009 meta-analysis reviewed 29 trials with almost 2000 patients undergoing hip and knee arthroplasty procedures with anti-fibrinolytic therapy. The studies reviewed had a lack of homologous regimens for type of drug, dosage and timing of administration. Regardless of that fact, results showed patients who received anti-fibrolytics, including TXA, e- aminacroproic acid, and/or aprotinin, had decreased blood loss between 393-639 ml and decreased transfusion requirements by 52% ($p<0.00001$). TXA specifically reduced blood loss by 393 ml (95% CI, -442 to -345). The results showed no increase in venous thrombosis risk.

Tranexamic acid can be administered orally, intravenously, via topical administration at the joint, or injected into a surgical drain. Due to possible systemic side effects with intravenous administration, topical application to target the source of bleeding is being investigated in several studies. A systematic review by Panteli and associates summarized seven trials on the use of topical TXA during knee arthroplasty procedures. Four papers had relevant data pertaining to blood loss. Within those four papers, there was a mean postoperative blood loss reduction of 220 ml ($p< 0.00001$) and decreased hemoglobin drop by a mean of 1 g/dL ($p< 0.00001$). Various dosing regimens were used from 250 mg to 3 g, but results suggested that a higher dose (>2g) was more effective for topical TXA administration.[7]

There is no consensus on single versus multiple dose regimens or routes of administration. According to Huang et al, a one-time IV bolus of tranexamic acid has been shown to be effective in reducing blood loss.[5] Ker et al argue that a total IV dose of 1g per administration appears sufficient in most procedures and there is no evidence to support higher doses.[8] Studies also suggest that the first bolus of TXA is beneficial if given prior to release of the tourniquet.[3] The plasma half-life of TXA is 2 hours and a second dose could further reduce blood loss within the postoperative time when most bleeding occurs.[2]

In this case study, TXA 1g was administered prior to tourniquet inflation as part of the surgical protocol. The combination of a regional anesthetic, tourniquet and tranexamic acid could have contributed to the patient's blood loss being significantly lower than the average blood loss for TKA surgeries.

Repeated studies show that tranexamic acid reduces blood loss and transfusions in orthopedic knee procedures without an increased risk of thrombosis. At this time, however, it is still considered an off-label use of the drug. The literature review did not identify specific contraindications for administration used during the studies. Lexicomp lists defective color vision, active intravascular clotting, and subarachnoid hemorrhage as contraindications for injection. TXA should be used cautiously in patients with thromboembolic disease, uncorrected vascular disease, disseminated intravascular coagulation, a history of seizures and renal disease or ureteral obstruction.[9] More studies need to be completed to evaluate if there is an ideal route or dosing regimen of TXA administration that maximizes the benefit to risk ratio for the patient.

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Mentor: Teresa Norris CRNA, EdD

High Fidelity Human Simulation in Nurse Anesthesia Education

M. Kellar Lambert, MS, MSN
Wake Forest Baptist Health

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High fidelity human simulators (HFHS) are complex machines used in healthcare education to build knowledge and skill through experiential learning. HFHS mannequins are placed in an authentic clinical environment, and integrated computer software produces lifelike and real-time physiological and pharmacological responses.[1-3] The effective use of high fidelity simulation for education and training in nurse anesthesia programs has the potential to improve student learning and performance without risk to patients. However, in order to maximize the benefits of this complex technology, there is a need for the development of standardized simulation implementation and assessment tools, along with research on cost-effectiveness and the impact of HFHS on patient safety and outcomes.

Case Report

Three first-semester graduate students enrolled in a nurse anesthesia program were scheduled to provide anesthesia in a simulated adult surgical case. Prior to the simulation, students were given time to familiarize themselves with the mock operating room, check equipment, and discuss the roles for the upcoming scenario. Students were also instructed to verbalize their assessments and actions throughout the scenario.

The students were briefed that their patient would be an 18-year-old male scheduled for an arthroscopic anterior cruciate ligament (ACL) repair following a traumatic football injury. No additional information was provided.

The simulation began when the mannequin opened his eyes and asked when the surgery would begin. The students quickly placed standard monitoring devices on the "patient," while interviewing him and performing a brief physical assessment. The patient had no known allergies and unremarkable medical and surgical histories, with the exception of well-controlled asthma. The attending anesthesiologist, played by program faculty, was called to the room and the anesthetic plan was discussed.

The anesthetic plan was approved, the physician left the room, and the students proceeded with induction. After intubation, the patient quickly became tachycardic, with a heart rate above 130/min and hypotensive with a blood pressure of 85/40 mm Hg. Esmolol and phenylephrine were administered. The patient's blood pressure continued to rapidly decline. One of the students believed she was unable to palpate a pulse and began chest compressions.

During resuscitation efforts, the other two students had difficulties ventilating the patient. It was decided that the patient may be having a bronchospasm because of high peak airway pressures. Epinephrine was administered, and the attending anesthesiologist was called back to the room. He quickly determined that the anesthesia machine had never been turned on, the endotracheal tube was in the esophagus and the patient in fact did have a pulse. The anesthesia machine was turned on, the patient re-intubated, and chest compressions suspended. The patient's vital signs stabilized and the attending physician left the room.

Shortly after the patient was stabilized, the scenario was fast-forwarded to emergence. Immediately following removal of the endotracheal tube the patient vomited, was re-intubated, and CPR was again commenced as his vitals deteriorated again. The case ended with preparation for transport of an intubated patient to the intensive care unit for follow up care.

At the conclusion of the case, the students participated in a debriefing session with instructors. The discussion centered on decision making, clinical judgment, and communication, and offered students the opportunity to self-assess each of these elements throughout the debriefing process.

Discussion

The preceding case report illustrates both the advantages and potential challenges in the use of HFHS for education and training in anesthesia. The use of high fidelity computer-controlled

simulators originated from the military and aviation industries, both of which had successfully used this approach as an instructional tool since the 1960s.[1,4] In the mid-1980s, the development of several mannequin-based patient simulators was inspired by research findings on error and human performance factors in anesthesia and supported by advances in computing technology.[4] Grounded in efforts to improve patient safety, HFHS has gained increasing acceptance as a sustainable mode of teaching, assessment, and research with over 1500 mannequin simulation centers world-wide.[3] A survey of nurse anesthesia program directors in 2008 by Turcato et al.[5] indicated that half of the respondents incorporated HFHS into their curriculum.

One of the primary benefits of patient simulation is the ability to practice critical thinking and decision-making as well as technical, cognitive, and interpersonal skills without fear of causing harm to a patient.[1,2] Errors, which would require immediate supervisor intervention in an actual clinical setting, can be allowed to occur during a simulation. Additionally, a simulation does not have the time constraints of actual patient interactions - it can be stopped, restarted or fast forwarded in order to highlight or explore learning in a particular situation.[1] Furthermore, critique and reflection of performance are a part of the simulation experience, allowing students structured time to gain awareness of strengths, limitations, and areas for improvement.[5]

Though simulators are designed to portray reality as closely as possible, there is inherent and inevitable variability in the authenticity of a simulation experience that has the ability to affect student performance. Hypervigilance is a common stress response for simulation participants, whose anxiety over the perception of impending disaster can cause negative behavioral and performance responses.[6] While the degree of physical, environmental, and behavioral fidelity of a simulator can also have an impact on learning and performance, the relevance of an exercise appears to have a greater influence than absolute realism.[1] It has been shown that student satisfaction and confidence are most highly associated with student attributes, clear objectives, adequate introduction and familiarization to the simulator, and instructor experience at debriefing techniques. [1,7]

As the use of HFHS in nurse anesthesia curricula increases, the development of reliable, validated tools is crucial for the objective assessment of both technical and nontechnical skills performance during simulations. Though there has been an increase in research designed to develop these instruments,[8] there is still a conspicuous lack of a standardized and accepted framework for evaluation. The systematic development, organization, and testing of performance assessments designed for the simulation environment will improve both evaluation and training methodology.[8]

While HFHS technology certainly has the potential to positively impact student learning, the question remains as to whether the benefits that can be achieved justify the expenses incurred. The cost of simulation hardware, clinical equipment, physical space, training, and organizational factors can easily result in six-figure price-tags.[1,5,7] In a literature review comparing improvements in performance with the use of high and low fidelity simulators (LFS)[9], there was little evidence showing an advantage of HFHS over LFS. Skeptics of high fidelity simulation also cite a lack of definitive research effectively proving performance transfer, or linking this teaching tool to direct improvements in student learning or patient outcomes. [2-4,10]

The presence of high fidelity human simulation in nurse anesthesia curricula is the subject of some debate. However, it is not likely that the use of this technology will diminish as researchers begin to explore additional applications of its use in certification, continuing education, and even program admission. Despite questions regarding its cost and utility as an educational tool, a large and increasing body of evidence exists indicating its importance as a resource for training future anesthetists to provide safe and efficient care for patients. With the development of consistent metrics for performance and improved training for educators and students alike, HFHS is a powerful instrument that can positively impact patient care.

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Mentor: Courtney Brown, CRNA, PhD

Lactated Ringer's Versus Normal Saline in Renal Transplantation

Ebou M.A. Cham, MSN
Georgetown University

Keywords: lactated ringer's, normal saline, renal transplantation, hyperkalemia

Approximately 16,000 patients undergo kidney transplant surgery annually.[1] These patients are typically dialysis-dependent and often have comorbidities such as heart disease, hypertension, and diabetes.[2] Perioperative management of these patients provides numerous anesthetic challenges.[3] Selecting the appropriate crystalloid solution for fluid management can be a major challenge.[2,3] Normal Saline solution (NS) has been commonly used perioperatively instead of Lactated Ringer's solution (LR) as LR may be associated with hyperkalemia in renal failure patients.[2-5] However, recent literature indicates that NS is associated with an increased incidence of hyperkalemia and metabolic acidosis in comparison to LR, which may contribute to poor outcomes following renal transplantation.[4,5]

Case Report

A 64-year-old, 51 kg, 157 cm female presented for cadaveric renal transplantation due to non-diabetic anuric end-stage renal disease (ESRD). The patient received hemodialysis three times a week for over 6 years. Her last dialysis treatment was the day prior to surgery. Post dialysis laboratory results were as follows: hemoglobin 11.4 mg/dl, hematocrit 35%, white blood cell count 5,200 cells/mcl, platelets 137 x 1000 mm^3, sodium 135 mEq/L, potassium 4.8 mEq/L, blood urea nitrogen/creatinine 31/5.74 mg/dl, and blood glucose 83 mg/dl. The patient's past medical history included hypertension, osteoarthritis and systemic lupus erythematosus. Home medication regimen included amlodipine, diltiazem, prednisone, and sevelamer. Patient allergies included lisinopril and cefazolin. Her past surgical history included a left lower extremity arteriovenous fistula insertion with no reports of anesthesia complications. A recent chest x-ray showed mild cardiomegaly, electrocardiogram showed sinus rhythm, and an echocardiogram revealed an ejection fraction of 65%. The patient's airway assessment revealed a Mallampati class III airway.

A peripheral intravenous (IV) catheter was inserted preoperatively and an NS infusion was initiated. Vancomycin 1 g IV was administered and infused over 60 minutes. Alemtuzumab 30 mg IV was also administered and infused over 4 hours.

In the operating room, the patient was premedicated with midazolam 1 mg IV and fentanyl 50 mcg IV. A noninvasive blood pressure cuff, pulse oximeter, and a 5-lead electrocardiogram were applied as standard monitors. The patient was preoxygenated with 100% Fi02 via facemask and induced with lidocaine 60 mg IV, propofol 120 mg IV, and cisatracurium 10 mg IV. A Glidescope (Verathon Inc., Bothell, WA) was used to intubate using a size 7.0 mm endotracheal tube. Maintenance of general anesthesia was achieved with a combination of desflurane 6% inspired concentration in oxygen 0.4 L/min and air 0.4 L/min. A left radial arterial line and a double-lumen left subclavian central catheter were inserted and transduced.

After 1L of NS was infused, fluid management was maintained with LR for the remainder of the procedure. A total of 4.5 L was infused over four hours. In addition, 1 L of 5% serum albumin was given IV over 30 min. Dopamine was infused at a rate of 3 mcg/kg/min to help maintain renal perfusion. Mannitol 50 g IV was infused over 60 minutes prior to reperfusion. In addition, a furosemide 100 mg bolus was administered and a continuous infusion at 20 mg/hr was initiated prior to reperfusion. Methylprednisolone sodium succinate 500 mg IV was given following vascular clamp removal. Central venous pressure (CVP) was maintained at 12 to 13 mm Hg with fluid administration. Intra-operative urine output was measured at 125 ml. Intraoperative arterial blood gases showed serum potassium range of 3.8 - 4.1 mEq/L and pH of 7.39 and 7.30 respectively.

At procedure's end, the patient was transported to the post anesthesia care unit (PACU) and was successfully extubated two hours later. Serum pH increased to 7.33 in the PACU. Potassium values were 4.4, 4.1, and 3.7 mEq/L on post-operative days 2, 3, and 9 respectively. Creatinine levels progressively dropped to 3.47 mg/dl, 2.59 mg/dl, and 1.41 mg/dl on post-operative days 1, 2, and 9 respectively. The patient did not receive postoperative dialysis and no lab findings were abnormal, including coagulation studies.

Discussion

Patients with ESRD are at increased risk for electrolyte abnormalities, notably hyperkalemia, due to the progressive decline in the diseased kidney's capacity to excrete potassium.[2] For most patients, survival is dependent upon regular hemo or peritoneal dialysis to rid the body of metabolic waste and excess elements in order to avoid hyperkalemia-induced cardiac arrhythmias and potential death. The definitive treatment for ESRD is renal transplantation.[2,6] However, due to the disproportionately high demand and low supply of donor kidneys[7], only one in four ESRD patients on the transplant waiting list will be privileged a new kidney.[1] Therefore, intraoperative optimization of these patients is especially vital.

As ESRD patients are already at a high risk for hyperkalemia by virtue of their failed kidneys, NS became the recommended crystalloid of choice for fluid management during kidney transplantation due to its lack of potassium as a constituent. [2-5] A nationwide survey of 49 hospitals revealed that in 90% of renal transplant cases, NS is used in order to avoid hyperkalemia that may be associated with potassium-containing fluids such as LR.[2] However, in the first known randomized, double-blind prospective study to compare NS and LR during kidney transplant, O'Malley et al revealed that although NS contains no potassium, it caused higher rates of both hyperkalemia and metabolic acidosis than LR in chronic renal failure patients. [2]

The above study showed that the patients in the NS group had higher intraoperative potassium levels than the LR cohort.[4] Additionally, within the NS group, patients who received treatment for metabolic acidosis were reported to have higher graft function in the forms of increased urine output and decreased creatinine levels postoperatively compared to other patients in the NS group who had untreated metabolic acidosis.[4] The above findings parallel the clinical course of this case report; her perioperative potassium levels consistently stayed within 3.7 mEq/L to 4.8 mEq/L while receiving mostly LR intra-operatively. The patient's urine output also improved as

early as in the PACU, and her serum creatinine levels progressively decreased from 5.41 mg/dl to 1.41 mg/dl by postoperative day 9 without any need for dialysis.

The metabolic acidosis associated with high volumes of NS is thought to be due to the high chloride content in NS leading to hyperchloremic metabolic acidosis and subsequent hyperkalemia.[2-6] NS contains equal amounts of sodium (154 mEq/L) and chloride (154 mEq/L).[2,6] However, the chloride content in NS is much higher than that in normal serum. Chloride is an anion like bicarbonate (HCO3-), which is the main buffer of hydrogen ions in the body. Unlike HCO3- however, chloride lacks any buffering ability and in effect, it not only dilutes the serum HCO3-, but its anionic presence also decreases reabsorption of HCO3- in the kidneys.[2,6] Perhaps in a patient with oliguric ESRD, chloride exerts more of a dilutional effect on HCO3- than it does decreasing its reabsorption. In either case, the presence of chloride will decrease the buffering ability of HCO3-, increasing serum hydrogen ions and decreasing pH. As serum pH decreases, there is an intracellular shift of serum hydrogen ions which causes intracellular potassium to shift outward into the serum, leading to hyperkalemia.[2,4,6]

Lactated ringers contains 131 mEq/L of sodium, 4 mEq/L of potassium, 112 mEq/L of chloride, 28 mEq/L of lactate, and 2.7 mEq/L of calcium.[2,5] The lower amount of chloride in LR compared to NS suggests that acidosis from hyperchloremia with LR infusion is much less likely. The lactate component of LR is converted to HCO3- in the body, which strengthens bicarbonate's buffering capacity against acidosis.[2,5] Furthermore, because of its potassium content, LR may reduce the intracellular-extracellular concentration gradient of potassium and hence limit the degree of extracellular potassium shift.

The use of LR in renal transplantation has gained more popularity than that of NS in recent years.[2,4,5] However, individual ESRD patients may be at different stages of the disease process and present with various comorbid conditions that can all impact anesthetic management.[2,4,5] Therefore, fluid management for each ESRD patient undergoing renal transplantation should be individualized, guided by the patient's perioperative hemodynamic condition, as well as diligent and timely analysis of laboratory findings.[5] Hyperkalemia is still possible with LR use, especially if it is infused at rapid rates and in significantly large volumes.[4,5] This suggests that the volume and rate of fluid administration is equally as important a consideration as the choice of crystalloid used.[4,5]

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Mentor: Catherine Horvath, CRNA, MSN

Right Upper Lobectomy in the Patient with Coccidioidomycosis

Andrew J. Roylance, MS
Westminster College

Keywords: Coccidioidomycosis, one lung ventilation, pulmonary nodules, lobectomy, thoracic epidural

Coccidioidomycosis, also known as Valley Fever, is a systemic fungal disease with a wide variety of manifestations. Coccidioidomycosis is caused by inhalation of Coccidioides fungi spores, which reside in the soil of regions where coccidioidomycosis is endemic, including the southwestern United States.[1] The majority of infections are asymptomatic or result in community required pneumonia. However, approximately 0.5% of infected persons develop nodules, cavities, or miliary disease.[2] In this case report the patient developed a new right upper lobe nodule that required surgical intervention. This case report describes the high risk anesthetic that was provided.

Case Report

A 70-year-old, 182 cm, 83 kg male presented with chronic obstructive pulmonary disease (COPD) and lung nodules likely due to past coccidioidomycosis infection. His medical history was significant for non-insulin dependent diabetes mellitus, tobacco use, multiple nodules of the lung, hypoxemia, coccidioidomycosis infection, and gastroesophageal reflux disease. His current medications included glipizide, metformin, tiotropium, metoprolol, albuterol, omeprazole, and mometasone. A preoperative pulmonary function study demonstrated severe obstructive airway disease with moderately reduced forced expiratory volume over 1 s (FEV1) of 1.85 L, 55% of predicted, severely reduced FEV1/forced vital capacity (FVC) percent ratio of 37%, severely reduced flow rates, and no improvement post bronchodilator therapy.

The patient was informed of anesthetic risks and concerns, and the anesthesia consent was signed. A thoracic epidural was placed in the T6-T7 interspace without complication in the preoperative holding area. Upon arrival to the operating room, noninvasive monitors were applied and O_2 8 L/min was delivered via face mask. An intravenous induction was achieved with lidocaine 80 mg, propofol 120 mg, and rocuronium 50 mg. The left mainstem bronchus was intubated using a Macintosh 4 blade, a 41 French dual lumen endobronchial tube (DLT) was placed to a depth of 29 cm. Placement of the DLT was verified using a flexible fiberoptic

bronchoscope and the carina was viewed with the endobronchial cuff just below the tracheal carina. Volume controlled mechanical ventilation was initiated with a tidal volume (TV) of 600 mL and respiratory rate of 10/min. A left radial arterial line was then placed. After successful induction of anesthesia the thoracic epidural was connected to an epidural infusion pump and preservative free bupivacaine 0.25% with fentanyl 2 mcg/mL was infused into the epidural catheter at a rate of 10 mL per hour. This rate continued throughout the postoperative period.

Anesthetic maintenance included sevoflurane 2% end tidal concentration in O_2 2 L/min. The patient was then placed in left lateral decubitus position and DLT placement was verified. One lung ventilation (OLV) was initiated to improve surgical exposure. The patient's SpO_2 decreased to 82%; 5 cm H_2O of continuous positive airway pressure (CPAP) was applied to the nondependent lung and 5 cm H_2O positive end expiratory pressure (PEEP) was applied to the dependent lung to maintain adequate oxygen saturation. After surgical exposure of the non-dependent lung the SpO_2 decreased to 79%. The patient was removed from mechanical ventilation and ventilated manually. This revealed low TV and high peak airway pressures. The DLT was assessed at this time and the fiberoptic scope demonstrated that the DLT had advanced distally into the left upper lobe bronchus. The tube was withdrawn approximately 2 cm and the endobronchial cuff was inflated. The breath sounds were auscultated and even throughout the left lung, and SpO_2 returned to 92%. The surgeon was able to successfully remove the right upper lobe. At the completion of the surgery the patients SpO_2 was 91% and after discussion with the surgeon it was decided to keep the patient intubated. The patient remained intubated and was transferred to the intensive care unit (ICU) postoperatively.

The patient was revaluated upon arrival in the ICU and was hemodynamically stable. Care was assumed by the nursing staff upon ICU arrival. The ICU pulmonologist determined the appropriate parameters to wean the patient from the mechanical ventilator. The patient remained intubated and sedated for 24 hours and then was successfully extubated. The patient remained in the ICU for three days and was discharged on postoperative day seven.

Discussion

Coccidioidomycosis is a fungal disease endemic to the southwestern United States. This systemic infection is caused by inhalation of airborne spores that are contained in the soil from a fungus called Coccidioides immitis.[2] A variety of risk factors have been identified including race; African American, Hispanic, and Filipino people are at higher risk.[1-2] People, who are elderly, have diabetes, are pregnant, smoking, outdoor occupations, and low socioeconomic class have all been implicated in increased incidence of the disease.[1-2] Severe forms of the disease may result in meningitis, community acquired pneumonia, and pulmonary disease that results in nodules, cavities, or miliary disease.[1-2] Approximately 8% of hospitalized patients die due to complications from coccidioidomycosis infections.[1]

The results of pulmonary function tests and arterial blood gases can be useful for predicting pulmonary function following lung resection, but they do not reliably predict the likelihood of postoperative pulmonary complications after nonthoracic surgery.[3] Even patients defined as high risk by spirometry (FEV1< 70% of predicted, FEV1/FVC ratio < 65%) or arterial blood gases ($PaCO_2$>45 mm Hg) can undergo surgery including lung resection with an acceptable risk of

postoperative pulmonary complications.[3] These pulmonary function tests should be viewed as a management tool to optimize preoperative pulmonary function but not as a means to predict risk.[3] This patient had no improvement of pulmonary function test post bronchodilator therapy and it was determined that the patient's preoperative pulmonary function was optimized.

Postoperative pain control is one of the most important factors affecting patient morbidity. Thoracotomy is considered one of the most painful surgeries, and higher pain scores of 4-6 (on a 0-10 scale) are expected. Adequate postoperative pain control is necessary for recovery and this should be evaluated on a case-by-case basis due to varying pain tolerance. Acute uncontrolled pain can lead to respiratory and cardiovascular complications, pneumonia, and atelectasis in the postoperative period.[4-5] Preemptive analgesia is the concept that pain therapy is more effective if given before the surgical incision and noxious stimulus.[4] During the preoperative period it was determined by the patient that his pain was adequately controlled if his pain score was less than 5. During the postoperative period he rated his pain at 4 and it was determined that the thoracic epidural provided adequate pain control.

Dual lumen tubes consist of two bonded catheters, each with its own lumen; one lumen is used for ventilating the trachea and the other for ventilating the bronchus. For laryngoscopy, the lubricated DLT is advanced with the distal curve concave anteriorly until the vocal cords are passed. The stylet is usually removed at this point. The fiberoptic bronchoscope is used to verify correct placement of the endobronchial tube. The fiberoptic bronchoscope is placed down the right lumen to determine precise left sided DLT position. The endoscopist should see a clear view of the tracheal carina, and the left lumen going off into the left mainstem bronchus with the cuff just below the tracheal carina.[5]

After surgical exposure of the non-dependent lung, distal migration of the DLT resulted in a decrease in the patient's SpO2. The fiberoptic scope revealed that the endobronchial lumen was now in the left upper lobe bronchus. This resulted in only the left upper lobe being ventilated/oxygenated. This problem rapidly resolved after the DLT was repositioned.

Absolute indications for OLV include isolation of one lung from the other to avoid infection, massive hemorrhage, control of distribution of ventilation, and bronchopulmonary lavage.[5] Surgical exposure is a relative indication for OLV.[5] The primary goal during OLV is maintaining adequate arterial oxygenation while providing a surgical field favorable for visualization and manipulation of the operative lung.[5] Should hypoxia occur during OLV, the anesthetist should assess for physiologic causes or DLT malpositioning. Once DLT positioning has been confirmed, several interventions can help attenuate hypoxia during OLV. First, CPAP to the nondependent lung is almost 100% efficacious in increasing the PaO_2, however this can inflate the lung to some degree and interfere with surgical field exposure. Second, PEEP applied to the dependent, ventilated lung acts to increase PaO_2 by recruiting collapsed airways, increasing compliance of the lung, and increasing functional residual capacity.[5] CPAP to the nondependent lung is utilized first because this allows for both lungs to participate in oxygen exchange. PEEP to the dependent lung is only initiated after CPAP because PEEP can exert pressure on small pulmonary vessels, causing more shunt to the nondependent lung.[5] It is generally recommended that one uses a FiO_2 of 1.0 during OLV.[6]

Providing care for the right upper lobectomy patient poses many challenges for the anesthesia practitioner, especially when the patient has severe COPD. These challenges include pneumonia, pain, pneumothorax, hemorrhage, infection, hypoxemia, respiratory failure, and inability to wean from the ventilator. It was determined that this patient was high risk for this surgery due to his poor lung function tests. However the risk of the surgery was justified to prevent metastatic disease. Our primary concern postoperatively was the inability to wean from the ventilator due to poor diffusion and high oxygen concentration requirements. This was avoided and the patient was able to discharge to home with home O_2. Postoperative concerns such as respiratory failure, and uncontrolled pain influenced our decision to keep the patient intubated at the end of the surgery, and to insert a thoracic epidural preoperatively. This case report has significant educational value to the student nurse anesthetist. During OLV hypoxia can be a common problem that needs to be judicially treated to prevent patient harm. When the patient becomes hypoxic the first thing that should be assessed is the placement of the DLT. Another concern during the management of OLV is monitoring for DLT migration distally to the left upper lobe bronchus. The DLT can appear to be positioned correctly because the bifurcation of the left mainstem bronchus can appear to look like the carina.

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Mentor: Art Shimata, CRNA, MAE, MSNA

Pneumocephalus after Epidural Anesthesia

Nora Reimbold, MSNA
Texas Wesleyan University

Keywords: pneumocephalus, epidural, blood patch, dural puncture

Epidural anesthesia is widely considered the most effective analgesic available for laboring parturients.[1] Epidural placement is associated with complications including intravascular catheter insertion, inadvertent dural puncture, and post dural puncture headache.[2] Pneumocephalus is a

rare complication that has been documented following the administration of epidural anesthesia, particularly in the instance of inadvertent dural puncture resulting in an injection of air into the subarachnoid or subdural space.[3] Clinical presentation includes headache, nausea, vomiting, seizures, dizziness, and depressed neurological status.[3] Sequelae due to post dural puncture pneumocephalus is considered rare with exact incidence unknown, but has been associated with the loss of resistance to air technique used to access the epidural space.[2]

Case Report

A 20-year-old, 66 kg, 168 cm, gravida-2, para-0 parturient was admitted for a post date induction of labor. The patient reported an uncomplicated term pregnancy. Past medical history included one prior spontaneous abortion, poorly controlled asthma, anxiety, and gastric reflux. Daily medications included albuterol, fluticasone, ranitidine, and pre-natal vitamins. Vital signs and routine laboratory values were normal.

Upon labor epidural consent, procedure site was prepped with chlorhexidine, and skin localized with lidocaine 1%. An 18-gauge epidural needle and loss of resistance to saline (LORS) technique was used to locate the epidural space at lumbar (L) space 2-3. An inadvertent dural puncture was identified by cerebrospinal fluid. Using LORS, a second attempt at L3-4 successfully located the epidural space. An epidural catheter was threaded and secured at 12 cm. A continuous infusion was initiated with 0.1% bupivacaine and fentanyl 2 mcg/mL at 12 mL/h. Patient stated adequate pain control. Following an uneventful vaginal delivery, the epidural catheter was removed.

Twelve hours after epidural catheter placement the patient reported photophobia, neck pain, and a severe bi-frontal headache exacerbated by sitting upright. Approximately fourteen hours post epidural catheter placement, the patient consented to an epidural blood patch. Venous blood was drawn from the patient's arm. Simultaneously, epidural space L2-3 was located using LORS and 20 mL of venous blood was injected. The patient did not report immediate relief. However, 8 h following the blood patch, the patient reported minimal headache, decreased neck pain, and moderate inter-scapular pain.

Approximately 38 h after the epidural blood patch procedure, the patient developed lower back pain and received oxycodone 5 mg orally without relief. The patient reported pain radiating from her lower back to the occiput, including inter-scapular and post auricular areas. Additional symptoms included frontal sinus pressure, transient diplopia, and severe nausea and vomiting. Forty-six hours after the epidural blood patch procedure the anesthesia team was consulted a second time. Following a neurology consult, a non-contrast head computed tomography (CT) scan revealed intraventricular pneumocephalus with gas present in the frontal horns of the lateral ventricles, body, and occipital horn of the left lateral ventricle. A regimen of ketorolac 30 mg intravenously (IV) every six hours, diphenhydramine 25 mg IV every six hours, prochlorperazine 5 mg IV every six hours, and acetazolamide 250 mg IV every twelve hours was started with no pain relief reported. Five days after the initial epidural placement the patient's symptoms spontaneously resolved.

Discussion

The mission of the Anesthesia Patient Safety Foundation (APSF) is to continually improve the safety of patients during anesthesia care. APSF advocates safety research and education, patient safety programs and campaigns, and national and international exchange of information to foster safe clinical anesthesia practice. This allows for the frequent comparison, critique, and debate of the safety of current anesthesia techniques. . The safety and effectiveness of epidural analgesia are contingent upon precise needle placement using the loss of resistance technique.[2] Although the safety of the loss of resistance to air (LORA) technique has been debated, LORA and LORS techniques to access the epidural space are equally accepted practices by anesthesia professionals.[1] Case reports of intracranial air following epidural anesthesia warrant a discussion on whether using LORA technique is indeed a safe practice. This pneumocephalus case report intends to highlight an infrequent and devastating complication that may occur following one of the most common regional anesthesia procedures provided for labor analgesia.

This case report led to a literature search about pneumocephalus following epidural anesthesia. The primary literature exploration was conducted to determine the safety of LORA technique and to discover evidence warranting a recommendation in anesthesia practice. Secondarily, reports of pneumocephalus were reviewed and compared to this report. Evidence from randomized controlled trials, meta-analyses, and case reports were reviewed. Specifically, the literature revealed multiple case reports confirming pneumocephalus following epidural anesthesia using LORA technique.[1,3, 4-7] With the exception of this case report, there were no documented reports of pneumocephalus using LORS technique found.

Pneumocephalus is a relatively rare complication of inadvertent dural puncture that is well-described within the literature.[7] Diagnosing pneumocephalus can be difficult due to similarities in symptomology of a pneumocephalus headache and a post-dural puncture headache. Therefore, it is theorized that pneumocephalus cases may often be undiagnosed, unconfirmed, and unreported. Signs and symptoms of pneumocephalus include a severe sudden onset headache, diplopia, and varying neurological deficits depending on the distribution and amount of intracranial air.[3,7] As little as 2 ml of air has been reported to cause symptoms.[4] Standard of care for the treatment of pneumocephalus is conservative therapy with supportive care of symptoms and hospitalization for observation.[6] High concentration of inspired oxygen facilitates denitrogenation and reabsorption of the air collection, and has been used as an adjunct to conservative therapy.[6] Symptomology and radiographic evidence of pneumocephalus usually resolve spontaneously within one week of onset.[6]

Randomized controlled trials have attempted to determine whether there is a difference in complication rates between the LORA and LORS techniques. A meta-analysis from 2009 comparing the different LOR techniques found that the risk for adverse outcomes was not statistically significant in the obstetric population[2]. A 2006 randomized controlled trial studied outcomes between LORA and LORS, reporting that there was insufficient data to analyze adverse effects and concluded that air and saline are equally safe. Furthermore, a 2013 systematic review comparing LORA and LORS techniques reported non-conclusive evidence in determining a superior technique for analgesia while suggesting the need for further studies[1]. Recommendations were made for anesthesia professionals to consider the possibility of

documented complications associated with the LORA technique[1]. Multiple randomized controlled trials have hypothesized the LORA technique is associated with complications, but to date these hypotheses have remained null. Larger, randomized controlled trials may be necessary to accurately document and correlate the LORA technique and pneumocephalus. The multiple case reports of pneumocephalus following LORA present strong arguments against the technique and supports discussion about clinical practice. However, at this time the case reports do not provide enough evidence-based data to inspire a large-scale change in practice. This unique case report inspires the need for further clinical research trials related to epidural anesthesia techniques and pneumocephalus.

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Mentor: Dion A. Gabaldon, CRNA, DHA

Anesthetic Management of an Adult Patient with Dandy-Walker Syndrome

Tori Woodward, MSN
Westminster College

Keywords: central nervous system malformation, craniofacial malformation, Dandy-Walker Syndrome, hydrocephalus, ventriculoperitoneal shunt

Dandy-Walker Syndrome (DWS) is a rare congenital disease of the central nervous system (CNS) occurring only once in 25,000-35,000 infants.[1-2,5] This disease consists of anatomical malformations of the cerebellum, fourth ventricle, posterior fossa, and foramina which often

results in hydrocephalus and upward displacement of the lateral sinuses.[1-5] Such malformations may cause increased intracranial pressure (ICP) and craniofacial anomalies such as cleft lip/palate, high-arched palate, retrognathia (receded jaw), and poor dentition.[1-5] Anesthetic management of patients presenting with Dandy-Walker Syndrome requires careful evaluation of the airway, preparation for a difficult airway, intraoperative ICP control, and attentive post operative care monitoring.[1,3-5]

Case Report

A 40-year-old, 157 cm, 58 kg Caucasian female presented for a laparoscopic excision of abdominal lesions. The patient's past medical history was significant for DWS with hydrocephalus and ventriculoperitoneal (VP) shunt placement at 9 months old, VP shunt revision at ages 4, 21, and 35, seizures as an infant, seasonal allergies, and depression. Previous anesthetics had resulted in no adverse events. Assessment of the airway revealed a Mallampati class II airway, small mouth opening, recessed chin, and a thyromental distance of 4 cm with full range of motion of the neck. The patient's medications included montelukast 10 mg once a day, bupropion 100 mg twice a day, and a multivitamin once a day. Additionally, the patient reported no current subjective signs of increased ICP.

In the preoperative area, a 20 gauge peripheral intravenous (IV) catheter was inserted in the left hand and midazolam 2 mg was administered. Standard monitors were applied upon entry to the operating room. The patient was pre-oxygenated with 100% O_2 for five minutes and induction was performed using fentanyl 150 mcg IV, lidocaine 60 mg IV, and propofol 150 mg IV. After absence of eyelash reflex was confirmed, the patient's eyes were taped closed, and a size 8 oral airway was inserted. Rocuronium was administered after easy mask ventilation was confirmed. Direct laryngoscopy was performed utilizing a Miller 2 blade resulting in a Cormack-Lehane grade II view of the vocal cords. The patient was easily intubated with a 7.0 endotracheal tube and mechanical ventilation was initiated.

Isoflurane 1.3% inspired concentration in oxygen was used to maintain anesthesia with fentanyl 50 mcg IV boluses for pain control titrated to heart rate and blood pressure. Ondansetron 4 mg IV and dexamethasone 8 mg IV were given for nausea and vomiting prophylaxis. End tidal carbon dioxide was maintained at 30-35 mm Hg. At the end of the case, neostigmine 3 mg IV and glycopyrrolate 0.6 mg IV were given after four of four twitches were confirmed with a peripheral nerve stimulator. Mechanical ventilation was changed to pressure support mode, the respiratory rate was decreased to 4 breaths per minute, and the patient began to exhibit spontaneous respiratory effort. Mechanical ventilation was discontinued and the patient maintained adequate spontaneous tidal volumes and respiratory rate with confirmatory sustained tetanus response to neuromuscular monitoring. The volatile agent was discontinued, oral airway was re-inserted, the patient was suctioned, and when able to open eyes to command, the endotracheal tube was removed. The patient had an uneventful recovery from anesthesia and was discharged from the hospital later that day.

Discussion

Although cases of patients who first present with signs and symptoms of DWS when they are adolescents or adults exist, most patients are diagnosed when they are infants.[5] All DWS patients, regardless of treatment for hydrocephalus, retain high probability for the presence of craniofacial abnormalities such as micrognathia, macrocephalus, cleft lip/palate, high-arched palate, retrognathia, anteriorly placed larynx, and poor dentition.[1-2,4] Potentially difficult airways require anesthetists to prepare the proper equipment.[6] In the present case, the patient's small mouth opening and recessed chin signified potential for a difficult airway.[6] Availability of difficult airway equipment at the bedside, such as a GlideScope (Verathon Inc., Bothell, WA) and endotracheal tube introducer (Bougie) (SunMed Healthcare, Largo, Florida), may help the anesthetist in the event airway difficulties are encountered and therefore were part of the preparation for the patient in the case study.[6] Placement of an oral airway occurred before attempted bag-mask ventilation. Paralytics were administered only after confirmation of easy bag-mask ventilation to increase the probability of avoidance of a cannot intubate/ventilate scenario.[6]

An obstruction of the fourth ventricle results in hydrocephalus and increased ICP in patients with DWS.[1-5] Volatile anesthesia agents vasodilate cerebral vasculature and thus increase ICP.[6] Although the patient in the present case did not exhibit signs of increased ICP most likely due to a functional VP shunt, cerebral spinal fluid volume and ICP maintenance measures were utilized throughout the case.[7] Isoflurane minimally affects cerebral blood flow and facilitates cerebral spinal fluid absorption and therefore was chosen as the volatile agent in the case study.[6-7] Jang et al. reported the use of propofol and remifentanil for induction and maintenance of anesthesia to reduce ICP in a patient with hydrocephalus induced increased ICP.[4] The present case included propofol and fentanyl in the induction sequence to prevent an acute increase in ICP.[6] In addition to these pharmacological measures, maintenance of $ETCO_2$ levels in the low normal range contribute to the maintenance of normal ICP.[4,6-7] The patient's $ETCO_2$ remained less than 45 mm Hg even during emergence.

In addition to the fourth ventricle and cerebellum abnormalities, partial or complete absence of the corpus callosum commonly exists in DWS patients thus increasing the likelihood of apnea and respiratory failure.[1,3] Kusumoto and Shinozuka, describe the successful initial extubation of a DWS patient after a dental extraction but go on to explain that the patient later needed reintubation due to hypoxia and pneumonia.[1] The authors recommend monitoring DWS patients in the intensive care unit postoperatively.[1] Not only are these patients at risk for postoperative respiratory decompensation, but they are also at risk for sudden and unexpected death due to the CNS malformations.[2] Precautions taken to confirm adequate ventilation and oxygenation included peripheral nerve stimulation monitoring, confirmation of adequate reversal of muscle relaxation, and documentation of spontaneous breathing tidal volumes between 400ml-500ml before removal of the endotracheal tube. Monitoring was continuous for 2 hours in the post-operative care unit and then another hour of monitoring in the recovery room occurred before discharge. A routine surgery and uncomplicated anesthesia recovery allowed for discharge that same day.

Due to the increased likelihood of craniofacial abnormalities, increased ICP, and potential need for postoperative ventilation in patients with DWS, a thorough preoperative assessment and anesthesia plan of care along with vigilant monitoring is necessary.[1,4] Regardless of the age of diagnosis of DWS, the anesthesia plan includes a thorough airway evaluation, difficult airway preparation, utilization of techniques to prevent the increase of ICP, diligent monitoring of muscle relaxation, and attentive post-operative care monitoring.[4]

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Mentor: Manardie Shimata, CRNA, MAE

Nitrous Oxide with Cochlear Implantation

Kristen M. Bettis, BSN
Wake Forest Baptist Health

Keywords: nitrous oxide, cochlear implant, middle ear pressure, ear procedure, sensorineural hearing loss

Nitrous oxide is an inhaled anesthetic with quick onset and offset, but there are situations for which it is contraindicated. When using nitrous oxide, an air-filled cavity has the risk of increasing pressure within the cavity quickly. Clinically, the use of nitrous oxide should be avoided in patients with possible pneumothorax or recent surgeries which might have created an air-filled cavity, such as eye surgeries and craniotomies.[1] Contraindications to nitrous oxide with ear procedures are not as straightforward as those with a pneumothorax, ileus, intracranial procedures, or laparoscopic procedures.[2] There are otological procedures when nitrous oxide should also be avoided, but when is nitrous oxide contraindicated in ear surgery?

Case Report

A 50-year-old, 88 kg, 170 cm, Caucasian male presented for a left cochlear implant. The patient stated he had loss of hearing for most of his life, but in the past five years he had lost hearing completely in his left ear. His past medical history included alcohol abuse, deviated nasal septum, bilateral sensorineural hearing loss, hypertension, and mild asthma. No past surgical history or family history of malignant hyperthermia. His current medication consisted of benazepril 10 mg daily that he took the morning of surgery. His preoperative vital signs and lab results were within normal limits.

The patient was pre-medicated with midazolam 2 mg intravenously via a 20-gauge intravenous catheter placed in the right antecubital vein. In the operating room, non-invasive monitors were placed on the patient and fentanyl 100 mcg was given. Oxygen 10 L/min was administered by face mask for 5 minutes. General anesthesia was induced with lidocaine 30 mg, propofol 200 mg, and succinylcholine 140 mg. Successful mask ventilation was followed by direct laryngoscopy with a Miller-2 blade and insertion of a bougie due to the patient's anterior glottis. A 7.5 endotracheal tube (ETT) was placed over the bougie with no difficulty. Placement of the ETT was confirmed with chest rise, positive end-tidal carbon dioxide and equal bilateral breath sounds. The ETT was secured at the lips at 22 cm. Mechanical ventilation was initiated with volume control to keep end-tidal carbon dioxide within normal limits and a positive end-expiratory pressure at 5 cm H_2O. Cefazolin 2 g was administered before the incision. General anesthesia was maintained with isoflurane 0.45% expired concentration in O_2 1L/min and N_2O 1L, and a remifentanil intravenous infusion at 0.2 mcg/kg/min. Additional neuromuscular antagonists were not administered due to facial nerve monitoring.

The cochlear implant was completed in approximately two hours. Ondansteron 4 mg was administered intravenously. The trachea was extubated after the patient was breathing spontaneously with adequate tidal volumes and following commands before coughing ensued. Oxygen 10 L/min was administered via face mask on the way to the post anesthesia care unit (PACU). Report was given to the PACU nurse. He had an uneventful recovery and was discharged the next day in stable condition

Discussion

The middle ear has a relatively fixed volume of air when temperature is stable.[3] The pressure in this cavity is proportional to the contained gas molecules.[3] Therefore, the rate and trajectory of the middle ear pressure is determined by the net loss or gain of physiological gases.[3] The cavity within the middle ear exchanges gas with four compartments including the inner ear, ambient environment, local blood flow, and the nasopharynx.[3] Caution must be taken if using nitrous oxide when one of these routes is blocked or if any increase in pressure is detrimental considering the surgical procedure and the patient's condition. Pressure can increase quickly due to the diffusion of nitrous oxide from the blood into the space 34 times faster than the nitrogen can leave the cavity into the blood.[4] Nitrous oxide diffusion to the ear may present complications for certain procedures. For example, increased middle ear pressure may actually dislodge a tympanoplasty graft.[4] It is also important to note any interference with the gas transfer to rebalance middle ear and ambient pressure.[3]

A cochlear implant is indicated for sensorineural hearing loss and involves placement of electrodes by the surgeon using anatomical landmarks of the cochlea and its scalae to place electrodes in the middle ear surrounding the round window.[5] Sensorineural hearing loss occurs when there is damage to the cochlea or damage to the nerves in the inner ear that travel to the brain.[6] The device is implanted through a retroauricular incision and a mastoidectomy is performed to place the device in the inner ear. The electrode wire attached to the stimulator placed behind the ear is threaded through the cochlea to stimulate the cochlear nerve directly. Usually, the device is activated two weeks later and allows the patient to hear a representation of sounds to help them understand speech.[7]

Akinesis was needed for this procedure without the use of paralytics to allow for facial nerve monitoring.[7] Isoflurane, nitrous oxide, and remifentanil were used together to prevent movement and allow for facial nerve monitoring. Any movement of the patient could be detrimental due to the microdissection approach of the procedure.[7]

The risk of using nitrous oxide in this case involves the possibility of obstructing the outlet used for normal diffusion of nitrous oxide. This risk could potentially cause damage to the patient's tympanic membrane. Electrode placement does have the risk of traumatic damage, but is often not to the point of obstructing the gas transfer.[2] Compared to the tympanoplasty graft, this procedure does not contain risk for damage to the cochlear implantation from the increased pressures that nitrous oxide would cause.

Another risk to consider is post-operative nausea and vomiting. A study conducted in Scandinavia showed that the number one reported reason of decreased use of nitrous oxide was due to the side effect of post-operative nausea and vomiting.[2] Surgeries involving the middle ear also have increased risk of post-operative nausea, vomiting, and vertigo.[7] Therefore, introducing nitrous oxide with a high risk post-operative nausea and vomiting procedure may not be the most beneficial option for the patient if other means of effective anesthesia are available. This is especially true if vertigo is present pre-operatively. The patient did not have vertigo pre-operatively therefore we used it for its additive effect to allow less use of isoflurane after discussing with surgeon.

The nitrous oxide was beneficial in this situation due to the quick lung elimination, allowing the use of less isoflurane and a short wake up time. This permitted the surgeon to perform an assessment of the facial nerve in the operating room. The post-anesthetic assessment note did not mention any problems with post-operative nausea and vomiting. No surgical problems were noted. Therefore, retrospectively, the anesthetic management was appropriate and requires no changes. The anesthesia professional must understand the surgical procedure of the ear and the implications of the use of nitrous oxide in order to safely use the agent.

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Mentor: Barbara Brown, CRNA, MSN

Pulmonary Artery and Vena Caval Embolectomy using the AngioVac Circuit

Katie R. Tonniges, BSN
Bryan College of Health Sciences

Keywords: AngioVac circuit, embolectomy therapy, suction thrombectomy, catheter assisted embolectomy, endovascular therapy for pulmonary embolism

The AngioVac (AngioDynamics, Latham, NY) cannula and circuit is a novel minimally invasive endovenous treatment option for removal of undesirable intravascular material. The AngioVac is designed to extract vascular materials including thrombus, tumors, vegetations, and foreign bodies from within the central venous system, right heart chambers, and pulmonary arteries, without the complications of traditional approaches.[1] Previously, central venous and pulmonary artery emboli (PE) management included therapeutic anticoagulation, systemic fibrinolysis, catheter directed thrombolysis, mechanical embolectomy, and surgical embolectomy.[2] Associated contraindications, risks of severe bleeding complications, mechanical trauma, and high mortality limit previous management approaches.[2,3]

Case Report

A 41-year-old female presented for AngioVac suction embolectomy of a large supra-renal inferior vena cava (IVC) mass and large central saddle PE with heavy clot burden to the right lung. Significant medical history included hypertension, chest pain with shortness of breath for two weeks, obesity, history of smoking, and bi-weekly heavy alcohol consumption. Cardiac evaluation revealed non-ischemic disease by heart catheterization and right ventricular hypokinesis per echocardiogram. The patient reported increased abdominal girth, pitting ankle edema, increased cough, and severe fatigue. Diagnostic testing revealed a suspicious left pelvic mass, an IVC mass originating from the left renal vein, saddle PE, and right lower lobe emboli. Preoperatively, the patient was hemodynamically stable with room-air $SpO_2 \geq 94\%$. Generalized

anasarca, pitting edema to lower extremities, ascites, and a prominent systolic murmur were noted. Electrocardiogram revealed normal sinus rhythm. Abnormal laboratory values include: hemoglobin 10.2 g/dL, hematocrit 32%, albumin <1.0 g/dL, and antithrombin activity 65%.

Preoperatively, a left radial arterial line and left internal jugular vein (IJ) 9-French quad-lumen access catheter were placed after versed sedation. Induction and intubation were uneventful with fentanyl, propofol, cisatracurium, and standard 8.0 mm endotracheal tube (ETT). General anesthesia was maintained with sevoflurane 1.3-2% inspired concentrations in a mixture of oxygen 1 L/min and air 1 L/min. Neuromuscular blockade was maintained at one twitch using train of four assessments with intermittent boluses of cisatracurium and vecuronium.

Surgical cut-downs were performed to place 24-French sheaths in both the right IJ and common femoral veins. The patient was anticoagulated with intravenous (IV) boluses of unfractionated heparin to maintain the activated clotting time (ACT) greater than 250 seconds. Veno-venous extracorporeal circulation was initiated after confirmation of adequate ACT, with flow rates up to 2 L/min during periods of cannula aspiration. The perfusionist drew ACT and arterial blood gases (ABGs) every 15 minutes. Aspirated blood was filtered and returned to the opposite cannulation site. Fluoroscopy and continuous transesophageal echocardiogram (TEE) were used to guide cannula advancement through the right heart and monitor for emboli. Initially, the AngioVac cannula was introduced via the femoral vein with moderate success producing clot like material in the circuit filter from the IVC. Secondary attempts via the right IJ to access the pulmonary artery embolism proved difficult to advance through anatomic structures. Efforts were aborted due to minimal debris filtered and firmness of the pulmonary artery thrombus. Total procedure time was 4 hours 14 minutes with 53 minutes of extracorporeal circulation. Blood loss was estimated at 300 mL. Minimal hemodynamic effect was noted with aspiration periods, requiring only 100 mcg of neosynephrine IV to maintain a mean arterial pressure greater than 65 mm Hg. Initial ABGs revealed hemoglobin 8.8 g/dL and hematocrit of 27%. Two units of packed red blood cells were transfused. Upon cessation of extracorporeal circulation, anticoagulation was antagonized with protamine sulfate 90 mg. The patient was taken to the cardiac intensive care unit for recovery on the ventilator with versed and propofol sedation.

Discussion

The AngioVac system includes the AngioVac circuit, extracorporeal bypass tubing, and the AngioVac cannula. The cannula has a balloon-actuated funnel-shaped tip that accommodates high flow aspiration and engagement of large intravascular material while performing extracorporeal circulation.[3] The AngioVac suction embolectomy system has been found to be a beneficial minimally invasive treatment option for patients that present with large pulmonary, right heart, and venocaval thrombus, masses, tumors, and vegetations.[1-3] The AngioVac was a treatment option for this patient due to the locations of the emboli, the option of a minimally invasive approach, and the unknown time span for the PE and IVC thrombus pathology.

Other management strategies for emboli of the pulmonary arteries and central venous system have accompanying risks, contraindications, limitations, and high morbidity/mortality. Therapeutic anticoagulation and systemic fibrinolysis are contraindicated in up to 50% of patients.[4] Anticoagulation and systemic fibrinolysis were not considered first for this patient due

to multiple locations and the large clot burden present. Surgical embolectomy is highly invasive and associated with high morbidity and mortality rates of up to 40%.[3,4] Vegetations on pacemaker leads removed via standard percutaneous methods have a 34-55% risk of septic pulmonary embolism and subsequent risk of pulmonary abscess formation, and refractory sepsis. The AngioVac significantly reduces the risk of septic emboli when leads are extracted.[5] Advantages of the AngioVac procedure include the ability to remove a large thrombus without thrombolysis as well as pathological debris from the right heart and central venous system. Additionally, it has the advantage of rapid initiation in many medical centers with interventional radiology or endovascular suites. Finally, unstable patients with multiple co-morbidities who may be limited by other treatment options better tolerate the AngioVac procedure. [6]

Indications for an endovascular approach include evidence of right heart strain on echocardiogram or angiography, patient preference on treatment, or contraindications to other treatment routines. This patient demonstrated evidence of right ventricular strain per echocardiogram and desired a minimally invasive approach. The goal of the procedure is to improve perfusion and decrease pulmonary artery pressures by removal or debulking of pathological material.[5,7] The primary complication is vessel dissection and hematoma due to the large bore cannulas.[1] Additional complications include bleeding, severe hemodynamic instability, arrhythmias, rapid onset pericardial tamponade, perforation of vessels and arteries, and perforation of the right atria or ventricle.[5,6]

General anesthesia is recommended for AngioVac procedures, especially when cannula placement is achieved via surgical cut-down and TEE will be performed. The procedure can be done under deep sedation, however patient movement is not recommended.[2]

Considering there are potential life-threatening complications associated with AngioVac procedures, anesthesia practitioners need to understand and plan for them. Central venous line and arterial access are essential and provide a means to rapidly assess hemodynamic instability. Additionally, TEE has become quite standard for these procedures. The patient should be type and cross-matched for blood products. Anesthesia practitioners need to plan for, assess, and intervene with symptomatic arrhythmias, significant blood loss, changes in pulmonary compliance, blood in the ETT or sputum, and changes in laboratory and ABG analyses.

Hemodynamic deterioration may be associated with high flow rates during aspiration periods and can be effectively treated with slowing the extracorporeal circulation flow rate, fluid boluses, inotropes, and vasopressors. Atrial or ventricular perforation should be suspected if there is a sudden decrease or loss of arterial line pressure, symptoms of tamponade, and/or evidence of fluid around the heart demonstrated by TEE.[4,5] Tamponade can develop rapidly with the presence of a few hundred milliliters of blood with symptoms including Beck's triad (hypotension, jugular vein distention, and distant muffled heart tones) and pulsus paradoxus. The TEE is the most sensitive tool for diagnosis of tamponade allowing for early intervention.[8] Management includes fluid and blood resuscitation, positive inotropic agents, vasopressors, pericardiocentesis, and possible emergent cardiopulmonary bypass and sternotomy. Ketamine has sympathomimetic effects and may be beneficial in the management and maintenance of anesthesia during an episode of acute tamponade.[8] Arrhythmias may be transient or sustained as the cannula and wires pass through the right heart. Blood loss should be minimal and any large

losses should be rapidly evaluated and replaced depending on the source of bleeding and hemodynamic status.

Sudden changes in pulmonary compliance or hemoptysis are signs of a perforated pulmonary artery. Treatments may include placement of a dual lumen ETT to perform lung isolation and emergent thoracotomy or sternotomy to repair the artery. Post-procedure extracorporeal membrane oxygenation support may also be needed to support heart function or for inability to wean from bypass.[4-8]

This case demonstrates the need for anesthesia providers to be knowledgeable and prepared for potential complications of the AngioVac procedure. Recommendations for future cases include having a double lumen ETT, fiberoptic bronchoscope, and multiple vasopressors and inotropic drips available for implementation if needed.

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Mentor: Shannon Pecka, CRNA, PhD

Bilateral Lingual Nerve Injury Following Use of a Laryngeal Mask Airway

Kellyn Nieland, BSN
Bryan College of Health Sciences

Keywords: lingual nerve, laryngeal mask airway, taste, sensation, general anesthesia

The laryngeal mask airway (LMA; Teleflex Inc., San Diego, CA) is used in nearly one-fourth of general anesthetics performed in the USA.[1] Several cases of nerve injury have been documented following endotracheal intubation. Although previously thought as a rare occurrence, lingual nerve damage following the use of the LMA is becoming more common.[2-5] To date there have been only 12 documented cases of lingual nerve injury following LMA use, with only one utilizing the LMA Supreme (Teleflex Inc., San Diego, CA).[3]

Case Report

A 54-year-old, 84 kg, 172 cm male presented for a left open inguinal hernia repair under general anesthesia. Medical history was significant for hypercholesterolemia. Simvastatin was prescribed but the patient was noncompliant. The patient did not take any daily medications, had no known medication allergies, and consumed one alcoholic beverage per week. Basic metabolic laboratory values were all within normal range.

Standard monitors were applied and included electrocardiogram, pulse oximetry, non-invasive blood pressure, and capnography. Induction consisted of intravenous midazolam 2 mg, fentanyl 50 mcg, and propofol 150 mg. A small amount of water based lubricant was placed on the posterior surface of a number 4 LMA Supreme. The device was placed without difficulty on the first attempt utilizing a tongue blade. Placement required light chin lift without jaw thrust with the head in neutral position. The cuff was then inflated with 45 mL of air to achieve minimal occlusive pressure. Proper positioning was confirmed by capnography and bilateral breath sounds to auscultation. Audible leak was present during ventilation with 20 cm H_2O pressure using the adjustable pressure-limiting valve.

Anesthesia was maintained with sevoflurane and oxygen using pressure support ventilation for the first 15 minutes followed by pressure control ventilation for 10 minutes. At this time, a loss of airway pressure and decreased end tidal inhalation agent was noted with SpO_2 remaining greater than 96%. Bag mask ventilation through the LMA was initiated and the patient moved slightly. Intravenous midazolam 2 mg was administered and the LMA was removed without difficulty. Bag mask ventilation resumed for the duration of the procedure (approximately 15 minutes). Total LMA time was 25 minutes. The patient was hemodynamically stable throughout the procedure and transferred to recovery without complications.

On postoperative day one, the patient reported numbness on the anterior two-thirds of the tongue. Bilateral bruising was noted on the lateral region of the tongue accompanied by slight difficulty chewing, swallowing, and talking secondary to numbness. Diagnosis following consultation with an ear, nose, and throat specialist was bilateral lingual nerve injury with minimal hypoglossal nerve involvement. Oropharyngeal assessment eight days postoperatively revealed resolution of

swelling and bruising and continued loss of taste and numbness of the anterior two-thirds of the tongue. No motor deficit was noted. Eighteen days postoperatively, taste returned and tongue numbness was partially resolved.

Discussion

Nerve injury is a rare occurrence after use of the LMA.[1] Nerves at risk for insult include the recurrent laryngeal, hypoglossal, and lingual. Hypoglossal injury leads to difficulty swallowing and recurrent laryngeal nerve injury leads to postoperative dysarthria, stridor and aspiration.[1,2] Lingual nerve damage is associated with loss of sensation and taste in the anterior region of the tongue. This patient presented with both loss of taste and sensation which was consistent with lingual nerve injury. Lingual nerve damage with the LMA Classic (Teleflex Inc., San Diego, CA) has been reported, yet only one case has been associated with the LMA Supreme.[3] It is possible that the low incidence of nerve injury reports may be attributed to lack of patient reporting or provider detection.[4]

The lingual nerve is a branch of the mandibular nerve (CN V3), which is a branch of the trigeminal nerve. The lingual nerve supplies sensation and taste to the anterior two-thirds of the tongue via the chorda tympani branch of the facial nerve and crosses the lateral edge of the tongue base near the medial aspect of the mandible. Here the lingual nerve lies close to the third molar where it is most susceptible to injury.[3] Placement of the LMA makes nerves and blood vessels in this anatomical region vulnerable to compression.

Potential factors responsible for lingual nerve injury include: 1) difficult LMA insertion; 2) operator inexperience; 3) excessive balloon inflation; 4) improper LMA sizing; 5) prolonged anterior displacement of the mandible during jaw thrust; 6) cricoid pressure during LMA placement; 7) perioperative LMA manipulation; 8) use of nitrous oxide; 9) lubrication with lidocaine jelly; 10) extreme head rotation during LMA placement or during the procedure; 11) alternating insertion techniques; 12) inadequate depth of anesthesia; and 13) excessive intra-cuff pressure.[1,2] If an LMA is too small, the provider may compensate by over inflating the cuff causing increased pressure on the nerves. Cricoid pressure and jaw thrust has been shown to displace the tongue anteriorly and stretch lingual nerves.[4,5] Nitrous oxide was not used in this case but increases in cuff pressures and volumes have been demonstrated with its use.[2,6,7] Size 4 and 5 LMAs are routinely used for adult males. A size 4 was used in this case and adequate ventilation was confirmed by capnography and audible air leak with 20 cm H_2O pressure. The cuff was inflated according to device manual for an 85 kg patient to 45 mL. The cuff pressure used was consistent with the manufacturer recommended optimum pressure of 60 cm H_2O.[6]

The likely mechanism of lingual injury for this patient was mechanical compression of the lingual nerve along the inner aspect of the mandible where it is only covered by mucus membranes. Pharyngeal musculature relaxation during general anesthesia may precipitate this compression.[5] Another potential cause is pressure from the LMA Supreme in the buccal cavity. If no predisposing factors are present and manufacturer recommendations are followed, lingual nerve injury can still occur.[3] Over-inflation of the cuff may have been a predisposing factor in this case because cuff pressure monitoring was not used. This problem can be avoided by inflating the cuff with the minimum volume of air that effectively prevents air leak around the

mask. Another explanation for this patient's lingual nerve injury is inadequate depth of anesthesia. Patient movement can cause increased pressure on the lingual nerve by the LMA. Intra-cuff pressure measurement can reduce the incidence of LMA induced nerve injury. Cuff inflation volume without use of manometry is variable and can exceed the manufacturer's recommended limit by more than two times.[8] Over-inflation of LMA cuffs impedes pharyngeal mucosal perfusion increasing risk of nerve injury. Limiting LMA intra-cuff pressure to less than 44 mm Hg or 60 cm H_2O has been shown to reduce pharyngolaryngeal adverse events in ambulatory surgical patients by 70%.[8]

Adherence to device manual recommendations can aid in proper sizing and placement and may attenuate nerve injury. Consideration of patient characteristics including anatomy, physiology, and weight can also ensure proper sizing. The LMA device can be aligned with anatomical landmarks such as the lips and mandible to ensure proper sizing.[6] Correct placement produces a leak-free seal against the glottis.[6] The quantity of air used to inflate the cuff is weight-based with the goal of achieving an intra-cuff pressure of 60 cm H_2O or less. Lingual injury is an infrequent neurovascular event following use of the LMA. Conservative treatment yields a good prognosis with symptoms typically resolving in two weeks to six months.[1,3] In this case, symptoms began to subside 18 days postoperatively and resolved completely within three weeks.

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Mentor: Shannon Pecka, CRNA, PhD

A Patient in Labor with Marfan Syndrome

Aubrey Ballard, BSN
Missouri State University

Keywords: Marfan syndrome, pregnancy, aortic dissection

Marfan syndrome (MFS) is an autosomal dominant connective tissue disorder cause by a mutation in the fibrillin-1 gene. Around 30% of patients with MFS developed de novo mutations, where neither parent carried the defect. This syndrome affects approximately 1 in every 3,000 to 5,000 people in the United States.[1] Marfan syndrome characteristically changes the cardiovascular, skeletal and ocular systems. During pregnancy, symptoms of MFS can be exacerbated, especially within the cardiovascular system. This places both mother and baby at increased risk for morbidity and mortality. Knowing how to assess and treat the complications associated with MFS can improve patient outcomes.

Case Report

Anesthesia was consulted for the placement of a labor epidural for a 22-year-old, 172 cm, 85 kg female. The patient was 38 weeks pregnant and the cervix was 6 cm dilated. She had arrived one hour prior via emergency medical services (EMS) with a blood pressure (BP) of 175/115 mm Hg and was "coming off the bed" in severe pain, rated 10 on a 1-10 scale. A magnesium infusion at 2 mg/hr was initiated on arrival to the hospital; the patient's BP decreased to 150/100 mm Hg. The patient had been incarcerated for the majority of her pregnancy. She had received minimal prenatal care. She told EMS a physician had diagnosed her with pre-eclampsia there had been no follow up care. The patient stated she had stopped using methamphetamines 4 months ago; her urine drug screen was negative.

Upon initial assessment the patient appeared extremely uncomfortable, with a nurse on one side and the patient's mother on the other, holding her down. During the interview the patient had difficulty maintaining attention. She denied any health problems. At the end of the interview the patient's mother stated the patient was diagnosed with Marfan syndrome but had not been evaluated since age 18. At that point, the patient was asked to more clearly describe the pain. The patient described the pain as very sharp in her upper back and did not correlate with uterine contractions. The obstetrician was immediately notified of the possibility of a dissecting aorta and the decision was made to proceed with an emergent cesarean section.

The patient was taken to the operating room. She was unable to lie down flat due to increased pain and dyspnea. Oxygen 10 L/min was administered via facemask with the patient in sitting position. A rapid sequence induction was performed with propofol 150 mcg and succinylcholine 100 mg with the patient in semi-fowlers position. Once the patient lost consciousness she was placed supine for laryngoscopy, the trachea was intubated, and endotracheal tube placement was confirmed. General anesthesia was maintained with sevoflurane 1.9% inspired concentration in oxygen 2 L/min. The baby was delivered 1 minute later and after clamping of the umbilical cord, fentanyl 250 mcg was administered, and sevoflurane was decreased to 1.2% inspired concentration in a mixture of O_2 2 L/min and N_2O 2 L/min. Upon induction, the patient's BP

was 155/105 mm Hg, and after delivery it decreased to 92/68 mm Hg. An oxytocin infusion was started after delivery of the placenta. Cardiology was consulted by the surgeon upon completion of the procedure.

The endotracheal tube was removed without difficulty and the patient was transferred to post anesthesia care unit. When fully awake, the patient was taken for a computed tomography scan of the chest and abdomen. The results showed a dissecting thoracoabdominal aortic aneurysm. The patient was transferred to the intensive care unit, and after evaluation by a cardiologist she was transferred to a larger facility for further treatment.

Discussion

Marfan syndrome can cause structural changes to multiple body systems of which the anesthetist should be aware. Cardiovascular, skeletal, pulmonary and central nervous system changes can influence what type of anesthesia is best for the patient. The cardiovascular changes associated with MFS are a leading cause of morbidity and mortality in these patients and 15% of pregnant patients with MFS will exhibit a major cardiovascular complication.[2] Cardiac problems associated with MFS include aortic root dilation, aortic valve regurgitation, mitral valve regurgitation, mitral valve prolapse, arrhythmias, cardiomyopathy and congestive heart failure.[1].Before open heart surgery, the average life expectancy of patients with MFS was 45 years.[3]

A parturient with MFS should always be considered “high-risk” and care should be taken to reduce the risk of aortic dissection. Recent studies have shown the rate of aortic dissection in patients with MFS range from 1-10%.[4] Fifty percent of dissections occur in the third trimester and 33% occur post-partum.[1] Vaginal versus cesarean delivery should be chosen based on the leas physiologically stressful method. For vaginal deliveries, epidural placement should occur early to decrease the physiologic sympathetic response to labor pain, like increased blood pressure and heart rate. Vasodilation can also be beneficial in decreasing pressure in the aorta. Standard dosing of epidurals may need to be increased due to the possibility of dural ectasia in the lumbosacral area which occurs in 70% of patients with MFS.[4]

Spinal anesthesia can be performed for caesarean delivery, although placement may be difficult from kyphoscoliosis.[5] General anesthesia for cesarean delivery should be performed in the presence of any indication unrelated to MFS, or with significant aortic dilation. MFS is associated with high-arched palate , micrognathia and retrognathia, which may make intubation difficult. Careful attention to proper patient positioning is essential due to possible joint laxity however, atlantoaxial dislocation is rare. Due to chest wall deformities, tidal volumes may need to be lowered and peak pressure closely monitored to avoid possible spontaneous pneumothoraces.[5] Sympathetic response to laryngoscopy should be minimized pharmacologically. General anesthesia offers the benefit of a protected airway and the ability for immediate cardiac surgery, should a dissection or rupture occur.[1] There is no definitive evidence of one method of delivery being superior to the other.

In this case study, the symptoms of aortic dissection were recognized quickly. The most distinctive symptom of aortic dissection is intense chest or back pain that is maximal and abrupt

in onset. Pain can be described as sharp, knifelike, or tearing that can radiate to the back, between the shoulder blades, tracking the progression of the dissection. Other symptoms may include aortic murmur, unequal peripheral pulses in the absence of pulmonary edema.[5] When aortic dissection is suspected, BP management is top priority with the goal of decreasing sheer forces on the aortic wall. Systolic blood pressure should be maintained between 100-120 mmHg and stabilized.[6] An emergent cesarean section under general anesthesia was planned due to the patient's inability to cooperate for spinal or epidural placement. Better BP management could have occurred pre-operatively since BP was consistently over 150/110 mmHg even after the start of the magnesium drip. An arterial line for BP monitoring could have been placed intraoperatively for more accurate measurements. Cardiology should have been notified as soon as aortic dissection was suspected and time would have been saved by going to CT directly from the OR. Admission to the ICU also delayed transfer to a tertiary care facility.

Communication, assessment and knowledge of the major complications associated with different diseases or syndromes are important. Sometimes the anesthesia professional is one of the first providers to interact with the patient and should be able to recognize these complications. In this case, quick identification of the symptoms of aortic dissection allowed for prompt surgical management patient and follow up care.

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Mentor: Adriane Fain, CRNA, MSNA

Multifactorial Acute Pulmonary Edema after TAVR

Louisa Dasher Martin, BSN
Northeastern University

Keywords: cardiogenic pulmonary edema; noncardiogenic pulmonary edema; aortic stenosis; airway obstruction; Trans Catheter Aortic Valve Replacement (TAVR)

Postoperative pulmonary complications occur in 5 – 80% of the surgical population.[1] Acute pulmonary edema is a serious postoperative complication that can strike any patient undergoing general anesthesia (GA), including patients presenting for Transcatheter Aortic Valve Replacement (TAVR). Pulmonary edema can severely impair gas exchange and oxygenation and the anesthesia provider must be able to correctly diagnose and intervene expeditiously if this occurs. Most TAVR patients are advanced in age and have multiple comorbidities, making them complex and challenging to safely anesthetize.[2] Moreover, severe aortic stenosis (AS) coupled with rapid intraoperative hemodynamic fluctuations, increases the risk for postoperative pulmonary complications.[2]

Case Report

An 80-year-old, 103 kg male with severe AS presented for TAVR. His medical history included obstructive sleep apnea (OSA), congestive heart failure (CHF), hypertension (HTN), cerebrovascular accident, chronic renal insufficiency, diabetes mellitus, obesity, dementia and anxiety. Echocardiogram revealed left ventricular (LV) hypertrophy with an ejection fraction (EF) of 65%, systolic anterior motion with outflow obstruction, severe AS (valve area 0.81 cm) and mild mitral regurgitation (MR). Medications included aspirin, lorazepam, furosemide, metformin, zolpidem, simvastatin, insulin and amlodipine.

General anesthesia was induced with the following intravenous (IV) medications: fentanyl 100 mcg, lidocaine 80 mg, propofol 120 mg, phenylephrine 100 mcg and rocuronium 30 mg. Using a Miller 2 blade for direct laryngoscopy, the trachea was intubated with a 7.5 mm oral endotracheal tube (OETT). Equal, bilateral lung sounds were auscultated with the OETT secured at 22 cm at the level of the teeth.

General anesthesia was maintained with sevoflurane 1.8% inspired concentration in O_2 2 L/min. Intraoperative transesophageal echocardiography (TEE) revealed low LV volume, therefore, aggressive IV fluid resuscitation was administered. The patient received 500 mL of 5% albumin and 2,500 mL of crystalloid solution IV. Hemodynamics were maintained with titrated doses of phenylephrine and norepinephrine IV. At the end of the case, the patient's SpO_2 decreased from 100% to 95%, and his heart rate (HR) increased from 50 to 80 beats/min. The results of an arterial blood gas revealed that his PaO_2 on O_2 FiO_2 1.0 was 91 mm Hg. It was then noted that the OETT's 24 cm mark was at the level of the teeth. The OETT was withdrawn 2 cm, and the 22 cm mark was again at the teeth. Equal, bilateral lung sounds were auscultated and ventilatory recruitment maneuvers were performed. Within minutes, the SpO_2 increased to 100%. The PaO_2 on repeat ABG was 204 mm Hg.

Neuromuscular monitor stimulation showed sustained tetany. Neuromuscular blockade was antagonized with neostigmine 3 mg IV with glycopyrrolate 0.6 mg IV. Sevoflurane was discontinued and the O_2 flow was increased to 10 L/min. Although the patient spontaneously generated tidal volumes of 200 – 400 mL at a rate of 12 – 16 breaths/min, he was difficult to arouse. Once he did respond to voice command, the OETT was removed and 100% O_2 with 5 mmHg continuous positive airway pressure was administered via facemask.

After extubation, the patient's tidal volumes decreased, and his airway was briefly obstructed. The patient was immediately assisted with ventilation. Despite continuous vocal and physical stimulation, he did not respond. Additional neostigmine 2 mg IV and glycopyrrolate 0.4 mg IV were administered. The patient's respiratory status did not improve, and the trachea was re-intubated. Frothy sputum was immediately visible in the OETT. The OETT was suctioned, and furosemide 20 mg IV was administered. The patient was transferred to the intensive care unit, where he remained for several days.

Discussion

Pulmonary edema occurs when there is fluid overload within the alveoli and interstitial tissue.[3] Pulmonary edema can be described as cardiogenic, noncardiogenic or some form of both.[1] The development of pulmonary edema involves an abnormal rise in pulmonary capillary hydrostatic pressure and an increase in the permeability of the alveolarcapillary membrane.[3] This is coupled with a decrease in colloid oncotic pressure within the intravascular space, and the net result is movement of fluid out of the plasma and into the interstitial lung space.[3] The pathophysiology can be complex, especially in elderly surgical patients with numerous comorbidities, such as the patient in this case report.

Cardiogenic pulmonary edema is the result of cardiac impairment, usually a failure of the LV to adequately pump and promote forward flow.[3] As volume and pressure increase in the LV, volume and pressure also increase upstream in the pulmonary system. Pulmonary capillary pressures greater than 20 – 25 mm Hg cause the rate of fluid entering the interstitial space to be greater than lymphatic drainage capacity, and this results in fluid overload within the alveoli.[3] Specific risk factors for excessive pulmonary intravascular pressures include disease in the coronary arteries or muscle, HTN, MR and mitral stenosis.[3] OSA is also a cause of increased pulmonary artery pressure.[4] This patient's risk factors for increased pulmonary pressures and therefore, pulmonary edema, included MR, HTN, a history of CHF and emergence hypercarbia, causing increased pulmonary vascular resistance for which this patient could not compensate.

Severe AS and the TAVR procedure warranted consideration of pulmonary risks. Aortic stenosis mandates specific hemodynamic management in order to avoid decreases in cardiac output, increases in pulmonary pressure and cardiopulmonary volume overload. It is ideal to keep patients with severe AS in a normal sinus rhythm (NSR) at a relatively slow rate because tachycardia decreases both filling and emptying time.[5] During the aortic valvuloplasty and valve implantation that occurs in a TAVR, the heart is put into ventricular fibrillation (VF). In VF there is a lack of forward flow and a state of unopposed aortic insufficiency, potentially resulting in pulmonary overload. Other than these periods of intentional arrhythmia and tachycardia, the patient remained in NSR with a rate in the 50s.

Regarding volume status, AS patients should be kept in a euvolemic state.[5] It is essential that afterload, preload and euvolemia are maintained.[5] The fixed and narrow diameter of the aortic opening impairs the left ventricle's ability to increase stroke volume, and any decrease in systemic vascular resistance or venous return can bring about intractable decreases in cardiac output. At the same time, it is imperative to not volume overload these patients.[5] If there is excessive end diastolic volume, forward flow through the stenotic valve may not be sufficient to prevent blood from backing up into the right atrium and lungs. It is in this way that hypervolemia can ultimately give rise to pulmonary edema. Intraoperative TEE indicated significant hypovolemia, leading to a response of liberal IV administration of crystalloid and colloid.

Negative pressure pulmonary edema (NPPE) is an example of noncardiogenic pulmonary edema and is of particular importance to anesthesia because it usually results from post extubation airway obstruction. The incidence of NPPE occurring after oral endotracheal intubation and GA is 0.05 – 0.1%.[1] This patient's baseline risk factors for obstruction included having a short neck, obesity and OSA.[1] Many etiological elements can contribute to the development of NPPE, but it most often occurs when a patient attempts to inspire air against closed vocal cords, causing excessive negative pressure to develop within the thorax.[1] The subsequent increase in venous return to the right heart, leads to pulmonary vascular vasodilation and increasingly negative pressure within the capillary interstitial space, and an increase in fluid leaving the vasculature and entering the pulmonary interstitial tissue.[1] Impaired gas exchange results in hypoxemia, and catecholamine release gives rise to systemic and pulmonary hypertension.[1] Afterload is increased and this further exacerbates the edema within the lungs.[1]

The etiology of this patient's acute pulmonary edema could not be attributed to a single factor. His high risk cardiac status coupled with the infusion of a large amount of IV fluid led to the presumptive diagnosis of cardiogenic pulmonary edema. Because his airway was obstructed for at least 2 – 3 breath attempts immediately after extubation, it is also reasonable to describe the pulmonary edema as noncardiogenic. Ultimately, complex and multifactorial cases such as this reinforce the importance of assessing the entire clinical and procedural picture and then anticipating all perioperative pulmonary complications and treating adverse events quickly.

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Mentor: Connie L. Lorette, CRNA, MSN, PhD

Inhalational Induction for Difficult Intubation

Kate M. Saftner, BSN
Wake Forest Baptist Health

Keywords: intubation, inhaled, induction, difficult, fiberoptic

Through a thorough preoperative assessment, a safe induction and intubation plan can be made. Some characteristics, such as thyromental distance, mallampati score, and previous difficult intubations may help the anesthesia provider identify a more difficult airway.[1] Although the gold standard of management for this population is an awake fiberoptic intubation, the performance of an anesthetized fiberoptic intubation has shown similar success rates.[2] In terms of safety, during intubation with an awake fiberoptic, spontaneous ventilation may provide a sense of security to the anesthesia professional. Through inhalational induction prior to fiberoptic intubation, spontaneous ventilation can be maintained while mask ventilation is assessed and the patient remains unaware. If mask ventilation were not possible, the patient could be awakened without loss of spontaneous breathing and the approach to intubation may be reevaluated. If mask ventilation is indeed possible, then intravenous induction agents could be given and an anesthetized fiberoptic intubation could be safely performed on the difficult airway.

Case Report

An 81-year-old, 63 kg, 165 cm female presented for partial tonsillectomy and partial glossectomy for a right oropharyngeal lesion. She had a medical history that included hypertension, osteoarthritis, gastroesophageal reflux disease, and esophageal stricture related to radiation therapy for esophageal cancer. Her surgical history consisted of percutaneous endoscopic gastrostomy tube placement and appendectomy. The patient had a history of difficult intubations. Previous surgical notes indicated that multiple failed attempts of direct laryngoscopy and fiberoptic intubations had taken place before successful intubation with glidescope occurred. It was documented that mask ventilation was not difficult with prior procedures. An airway assessment revealed a limited range of motion. The patient had a Mallampati IV airway and numerous mouth sores were visualized. No other anesthesia complications were noted and all other physical assessments were unremarkable.

The patient was transferred to the operating room, moved to the surgical table, connected to standard monitors, and preoxygenated with 100% Fi02 at 10 L/min. Inhalation induction was

accomplished using oxygen and nitrous oxide at 5 L/min each, along with sevoflurane at 1%. Sevoflurane was increased by 1% every two respirations. The patient was appropriately anesthetized after 30 seconds with the sevoflurane at 6%. Mask ventilation was assessed over the patient's spontaneous breathing and was done without difficulty. Intravenous induction was then completed with lidocaine 80 mg, propofol 70 mg, and rocuronium 40 mg. Fiberoptic intubation with a 5.0 double cuffed omniguide carbon dioxide laser endotracheal tube was successful. Placement of the endotracheal tube was confirmed with equal bilateral breath sounds and end-tidal carbon dioxide.

Following induction, sevoflurane was turned off and anesthesia was maintained with isoflurane. Nitrous oxide was discontinued, oxygen was decreased to 0.8 L/min, and air was added at 0.8 L/min. The bed was turned 180 degrees and correct endotracheal tube placement was confirmed. Throughout the case, her blood pressure was maintained within 20% of her baseline with esmolol 100 mg, metoprolol 5 mg, labetalol 25 mg, and hydralazine 4 mg. Increased blood pressure and heart rate due to surgical stimulation was managed with fentanyl 150 mcg. Before emergence, ondansetron 4 mg was administered. Neostigmine 3 mg and atropine 0.8 mg were given intravenously to antagonize the neuromuscular blockade. The patient began breathing spontaneously and appropriately followed commands, and following oropharyngeal suctioning, she was extubated. Oxygen at 10 L/min was administered by face mask until the patient was moved to the stretcher. Oxygenation at 6 L/min was resumed via a Mapleson C circuit and the patient was then transported to the post anesthesia unit in stable condition.

Discussion

Awake intubation is considered the gold standard when a difficult intubation is anticipated. The American Society of Anesthesiologists (ASA) Difficult Airway Algorithm guides the anesthetist to intubate the patient awake when the preoperative assessment results in a predictive difficult intubation. However, the algorithm also stresses that the anesthesia professional should intubate using measures in which they are most proficient.[3] In this case, there were no contraindications for an awake intubation, but the gold standard was not utilized due to the anesthesia professional's preference.

Studies have shown that awake fiberoptic intubation is successful 88%-100% of the time in patients who have been identified as a difficult intubation.[2] However, inhalational induction is a technique that can be useful in the management of a difficult airway by allowing the fiberoptic guided intubation to be done on a fully anesthetized patient. By using fiberoptic guided intubation in this manner, successful intubations have been reported 87%-100% of the time.[2] With minimal difference in success rates between awake fiberoptic and fully anesthetized fiberoptic intubations, both approaches are acceptable in the management of a difficult intubation. Some benefits of anesthetized fiberoptic intubation with inhalation induction may outweigh the gold standard of awake fiberoptic intubation.

The benefits of inhalational induction are that the patient maintains spontaneous ventilation during induction, while remaining comfortable and unaware. A rapid inhalational induction can be safely completed with the use of nitrous oxide and a volatile anesthetic. The high vapor pressure of nitrous oxide allows it to quickly move into the blood, resulting in a higher

concentration of the volatile anesthetic.[4] Through inhalation induction the patient will continue to spontaneously ventilate, which allows the provider to safely assess mask ventilation.[5] By starting with a low concentration of sevoflurane and gradually increasing, the patient is less likely to lose spontaneous ventilation. Immediate high concentrations of sevoflurane may have a faster onset, but if spontaneous ventilation is lost the benefit of inhaled induction is negated.[6] If previous documentation was inaccurate and mask ventilation was not possible for this patient, the sevoflurane could be turned off. The discontinuation of sevoflurane would result in rapid emergence, all while the patient maintained spontaneous ventilation. With awake fiberoptic the anesthetist is unable to assess mask ventilation, which may result in a more conservative wake up and extubation. Despite the benefits of an anesthetized fiberoptic intubation after inhalation induction, there are associated risks.

In this particular case, once the ability to mask ventilate was confirmed, intravenous induction agents were given and fiberoptic intubation was attempted. However, no approach comes without risk. Due to the use of intravenous induction agents, a failed intubation would require mask ventilation until the patient awakened and the neuromuscular blockade was antagonized. Prolonged mask ventilation poses an increased risk for aspiration, especially in a patient with a history of gastroesophageal reflux disease and esophageal strictures. Although this patient's reflux was well controlled on a daily proton pump inhibitor, aspiration is a serious threat. This risk is specific to the anesthetized fiberoptic intubation and would be less of an issue in the awake fiberoptic, where mask ventilation is not a part of the induction sequence.

A fully anesthetized fiberoptic intubation with inhaled induction was successful for this anticipated difficult intubation. Although literature and current practice supports awake fiberoptic intubation as the preferred method of intubation for difficult airways, the anesthesia professional's preference must be taken into account. Inhalation induction is another way to deal with patients who present with positive identifiers of difficult intubation. By using sevoflurane, mask ventilation can be assessed while maintaining patient comfort and respiratory drive. A negative aspect of using inhaled induction followed by intravenous induction for difficult intubation cases is that if intubation were unsuccessful the patient must be mask ventilated until the muscle relaxants are completely antagonized. With this comes increased risk of aspiration. The combination of an inhalation and intravenous induction was conducted without any complications in this case and proved to be another route that can be utilized when difficult intubations are identified.

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Mentor: Joe Joyce, CRNA

Does High Fidelity Simulation have an Impact on Student Registered Nurse Anesthetists' Critical Thinking Skills?

Amber Bradford, MSN and Elaine Cook, MSN
Lincoln Memorial University

Keywords: simulation, high fidelity simulation, student registered nurse anesthetist, critical thinking

Introduction

High fidelity simulation is used in the military, aviation, and healthcare fields to acquire skills needed to perform a variety of tasks from the everyday to crisis situations. These skills are necessary to reduce mortality and morbidity. Promoting patient safety has been brought to the forefront by follow-up reports from the 1999 landmark publication To Err is Human from the Institute of Medicine.[1] More than 47,00 CRNAs provide anesthesia to over 34 million patients in the United States per year[2]. With this large number of anesthetic cases being performed, it is imperative that newly graduated CRNAs are prepared to provide safe and effective patient care. The use of high-fidelity simulation to develop critical thinking skills can improve the quality of student registered nurse anesthetist (SRNA) education and provide preparation for clinical practice. This research explores high-fidelity simulation as it influences critical thinking skills in SRNAs. Our null hypothesis is there will be no change in critical thinking skills after high-fidelity simulation experience.

Methods

This quasi-experimental research design used quantitative methods guided by Bloom's Taxonomy theory. The study was conducted in the high fidelity simulation laboratory at Lincoln Memorial University (LMU). Research subjects included 16 SRNAs scheduled to graduate in 2015. The study included nine females and seven males. Demographical averages include: age 30 years, critical care nursing experience 1.6 years, graduate record examination score 1006, and undergraduate grade point average 3.6. Previous nurse anesthesia coursework included anatomy, physiology, equipment and airway management. Instrumentation consisted of a pre-simulation and post-simulation questionnaire of multiple choice questions pertaining to a specific, structured

simulation scenario that required critical thinking. Validity and reliability of the questionnaire were established. Data was compared using a two-tailed t-test and a p value of less than or equal to 0.05 was regarded as significant.

Results

Variables included pre-simulation and post-simulation test scores. Average pre-simulation test score was 7.4 points out of a possible 15 points. Average post-simulation test score was 9.8 points. The two-tailed p-value was 0.007297. The null hypothesis, which stated critical thinking will not improve after high-fidelity simulation, was rejected when the two-tailed t-test resulted in a p-value less than 0.05.

Discussion

The results of this study suggest high fidelity simulation does increase critical thinking skills in SRNAs. However, there are several weaknesses to this study. The sample size consisted of only 0.8% of the total population of SRNAs in the United States and was based on convenience sampling. Due to the small sample size, it is impossible to determine if prior nursing experience influenced the results. Another weakness was the lack of clinical experience in this SRNA study sample. Strengths of the study included the consistency of non-faculty personnel operating the high-fidelity simulator to promote a low-stress learning environment. Future studies should be constructed to control for previous nursing experience and nurse anesthesia didactic knowledge. The use of a larger cohort from multiple nurse anesthesia programs may be appropriate in future studies.

Anesthesia practice requires a high degree of critical thinking. Utilizing simulation to provide realistic scenarios during the training of SRNAs is gaining popularity. Approximately 50% of nurse anesthesia programs employ this teaching method to prepare students for the challenges of the operating room.[3] Although more research needs to be completed, this study strongly suggests that critical thinking skills can be developed through the use of high-fidelity simulation.

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Mentor: Crystal Odle, CRNA, DNAP
Acknowledgement: Joy Lewis, CRNA, MSN

Is the use of desflurane associated with higher airway resistance and decreased lung compliance when compared to sevoflurane?

LT Nathan C. Dickerson, DNP
Texas Christian University

Keywords: Desflurane; sevoflurane; volatile anesthetic; general anesthesia; airway resistance; pulmonary, compliance, resistance; endotracheal anesthesia; bronchodilator; bronchoconstriction.

Introduction

Practitioners began use of third-generation volatile anesthetics, such as desflurane and sevoflurane, in the 1990's.[1] Jakobsson found that over the course of two decades the use of desflurane by practioners has evolved into an extremely beneficial volatile agent, when used in appropriate cases.[1] Over the course of the last twenty years many questioned if desflurane, like sevoflurane, was a true bronchodilator or if its irritant properties cause bronchiole constriction that lead to increased airway resistance and decreased lung compliance. Anesthesia professionals may base their opinion of desflurane's irritant properties on acquired clinical observations instead of clinical research. The following literature review aims to analyze the following question: in patients undergoing general anesthesia with a volatile anesthetic, is the use of desflurane associated with higher airway resistance and decreased lung compliance when compared to sevoflurane?

Methodology

An in depth literature search was performed on multiple databases, to include EBSCOhost, Embase, CINAHL, and MEDLINE. Original key words utilized on all databases were "desflurane" and "sevoflurane" yielding a total of 1,894 articles. Additional search terms "bronchodilation OR resistance" were included to further reduce the search to 133 articles. Any article that did not focus specifically on the airway was excluded. Inclusion criteria were English text, dates ranges 2000 to 2013 and studies that induced with intravenous medications prior to administration of volatile anesthetics. Studies involving children were excluded. All were reviewed, to include articles addressing bronchodilation and/or bronchoconstriction. A combination of evidence based reviews, randomized control trials and literature reviews were utilized to investigate this topic; five articles were retrieved overall. Each articles reference list was examined and yielded an additional 3 articles, bringing the total of reviewed articles to 8.

Literature Review

In 2006, Nyktari et al. used a two-chamber test lung that simulated a human lung, with a fixed resistance, to try to determine the physical properties of isoflurane, sevoflurane, and desflurane.[2] Nyktari et al. found when desflurane is delivered at 1 MAC it increases pulmonary resistance 44.5% from baseline; sevoflurane at 1 MAC increased it 5.7% from baseline. When both gases are delivered at 4%, desflurane increased pulmonary resistance by 25.8% and sevoflurane increased it by 28.9%. Desflurane is a more dense gas (5.44 kg/m^3) than sevoflurane (6.12

kg/m^3), which becomes significant when discussing higher MAC.[2] These findings indicate that desflurane increases pulmonary resistance more than sevoflurane based on the understanding that higher concentrations required to achieve therapeutic doses of desflurane lend to higher density of gas in the lungs/airway. The main focus was to determine if different concentrations of each gas would cause varying pulmonary resistance. The lung model provided a constant fixed resistance of 20 cm H_2O for each gas concentration comparison.[2] This hinders the application of this study to clinical practice because human lungs have varied compliance, i.e. a health lung verses a diseased lung. The test lung does not have biological receptors to react to each volatile anesthetic gas. A human lung's irritant gas receptors react with the noxious stimuli caused by the pungency of desflurane, where the test lung will not have these reactions[2]. Strengths of this study are that it looks at the minimum alveolar concentration (MAC) of each gas and concentration percentages to allow for accurate comparison. In Nyktari et al. they used desflurane 1 MAC as 6.6%, compared to sevoflurane 1 MAC as 1.8%.[2] This lends a slightly skewed view of each gas given that 1 MAC of desflurane is generally 6% and sevoflurane is generally 2.1%.[9] It is unclear as to why the researchers went high on desflurane MAC, as compared to the generally accepted MAC dosing, and lower on sevoflurane MAC. Desflurane is less potent than sevoflurane, meaning that in order to achieve the same efficacy it must be delivered at a higher concentration.

A follow on study by Nyktari et al applied the laboratory based findings from their previous study and examined inspiratory resistance of desflurane, sevoflurane, and isoflurane in healthy adults undergoing elective surgical procedures. A MAC of 1 and 1.5 was used for desflurane, isoflurane, and sevoflurane then findings for both MAC sets were compared.[3] In a healthy human lung at 1.5 MAC desflurane caused an increase in pulmonary resistance compared to sevoflurane at 1.5 MAC. With the evidence from the 2006 study, it was concluded that desflurane causes dose dependent increase in pulmonary resistance directly related to the higher gas density when a MAC over 1.0 is used. There is an initial rapid increase in airway resistance when desflurane was administered at 1.5 MAC. Over the course of the 30-minute administration at 1.5 MAC there was a slow decline in resistance that rapidly returned to baseline when desflurane was stopped. The abrupt decrease in airway resistance once the gas is discontinued indicates that desflurane does not cause bronchoconstriction. Instead this evidence supports that the density of desflurane plays a role in increased airway resistance: at 1.5 MAC airway resistance is seen, opposed to no airway resistance at 1 MAC. Decreasing resistance throughout the administration of desflurane at 1.5 MAC indicates it causes bronchodilation after an abrupt peak upon administration (plateau effect).[3] This plateau effect is attributed to increased resistance from high gas density that is slowly attenuated by desflurane's ability to cause bronchodilation in a dose-dependent manner. A limitation to this study is that it may only be applicable to those without reactive airway diseases or chronic obstructive pulmonary disease

Dikmen et al. performed a similar clinical trial to Nyktari et al to test the dose dependent bronchoconstriction of desflurane. The study compared the changes caused by desflurane, sevoflurane, and isoflurane on respiratory mechanics with increasing MAC doses, after total intravenous anesthetic induction had been achieved on ASA I-II patients undergoing gastrointestinal surgery. Dikmen et al. found identical results to Nyktari et al.: desflurane when administered at 1 MAC exhibits bronchodilation effects, similar to sevofluranes effects at 1 MAC; at 2 MAC increased airway resistance with desflurane is seen due to the increased density of the gas.[4] This study excluded patients with a history of smoking, COPD or those taking

corticosteroids or sympathomimetics. Measurements of respiratory resistance, dynamic compliance and peak inspiratory pressures were measured at the following time points: 1) before administration of anesthetic gases after 3 inspirations on mechanical ventilation, 2) after 5 minutes of a gas at 1 MAC, and 3) after 5 minutes of a gas at 2 MAC. This study revealed that at 1 MAC both desflurane and sevoflurane have bronchodilatory effects: both cause equivalent decrease in respiratory resistance and increase in pulmonary compliance.[4] At 2 MAC sevoflurane continued to exhibit the same bronchodilatory properties. Desflurane at 2 MAC caused the opposite effect: increase in airway resistance and a decrease in compliance. A weakness in this study is that measurements were only recorded for 5 minutes, so it is assumed that desflurane does not cause bronchodilation at higher MAC and the plateau effect that Nyktari et al. described was not acheived.[3]

In contrast to the previous designs, Goff et al. modeled a randomized clinical trial that included subjects with a documented smoking history.[5] Patients received 1 MAC of sevoflurane, desflurane or a weight-based dose of thiopental. At baseline respiratory system resistance (Rrs) was not different between the desflurane, sevoflurane, and thiopental groups. However, over time the groups were significantly different. As previous studies demonstrated desflurane at 1 MAC does not produce a significant difference in pulmonary resistance when compared to sevoflurane.[3] There are no measurable differences between desflurane an sevoflurane in regards to pulmonary resistance when using a health, non-smoking lung. It is significant to note that this data is true in those with healthy lungs and no history of smoking.[3] Those with a past or present smoking history had an extreme increase in Rrs at 1 MAC desflurane.[5] Sevoflurane had a decrease in Rrs in both smokers & non-smokers at 1 MAC. Data was recorded for a 10-minute period, compared to 30-minutes or more in all other studies.[2-7] Figure 1 of Goff et al. shows at the 7.5-minute mark Rrs in the desflurane smoking group is starting to decrease towards baseline. Indicating a decreased compliance existed in this group before the administration of desflurane and the higher density caused an additional increase in Rrs from baseline.[5] The bronchodilatory effect of desflurane is slowly attenuating the airway resistance caused by gas density at the 7.5-minute mark. Given the short time frame under study and the trend back to baseline at 7.5 minutes, one cannot deduce that desflurane is a constricting volatile anesthetic. Goff et al. attribute the increased resistance in smokers to the fact that the airway of a smoker is sensitive to exogenous stimuli from the pungent odor of desflurane, leading to bronchial smooth muscle constriction.[5]

To further explore the phenomena behind if desflurane causes increased airway resistance and decreased lug compliance Satoh et al., in 2 different studies, examined the pungent effects of desflurane on tachykinin pathways in guinea pigs.[6,7] The original laboratory trial was focused on a possible cause of increased resistance being linked to vagal nerve reflexes.[6] 40% of subjects were pretreated in numerous ways prior to either desflurane or sevoflurane administration: atropine or a vagotomy, to determine parasympathetic relation; either tachykinin selective antagonist neurokinin 1 (NK_1) sendide or tachykinin selective antagonist neurokinin 2 (NK_2) MEN-10376, to determine antidromic reflexes; depletion of tachykinins in the inflammatory cells of sensory afferent nerves of the smooth airway with capsaicin (C-fibers that are activated via noxious stimuli, i.e. the pungency of desflurane); the remaining 60% were untreated and exposed to different MAC (0-2.0) of desflurane and sevoflurane as a comparison.[6] All pretreated subjects were exposed to 2.0 MAC of either desflurane or sevoflurane. Untreated subjects

displayed findings parallel to previously discussed studies: desflurane increased total lung resistance and decreased lung compliance in a dose dependent fashion. Pretreatment with atropine or a vagotomy showed no effect, indicating there is no connection between the cholinergic efferent pathways in the increase lung resistance caused by desflurane. The other 2 pretreated groups showed a significant reduction in total lung resistance and increased lung compliance when exposed to desflurane at 2.0 MAC.[6] The study findings indicate that the increased density of dose dependent desflurane induces airway constriction. Considerable evidence relates this constriction to the antidromic activation of afferent C-fibers, leading to a subsequent release of inflammatory tachykinins, substance P or NKA. Satoh et al. link the possibility of desflurane causing increased lung resistance to this inflammatory pathway through airway edema and increased secretions.[6]

Satoh and Yamakage utilized the previous study findings and explored the potential for specific receptors to provoke decreased airway diameter via an antidromic inflammatory response that leads to airway constriction, focusing on A1 receptors of sensory C-fibers in the airway.[7] Guinea pigs were again used as test subjects and pretreated with receptor antagonizing agents. Afferent C-fiber receptors: the irritant gas receptors transient receptor potential A1 (TRPA1) and the capsaicin receptors transient receptor potential V1 (TRPV1) were antagonized by the drugs HC030031 or BCTC. Results showed that antagonizing TRPA1 irritant gas receptors significantly diminished the effects on lung resistance and compliance caused by desflurane. Antagonizing TRPV1 receptors had no effect on outcomes. The guinea pig studies indicate that the pungent odor, in combination with the density of desflurane, directly activates the irritant gas receptors of afferent C-fibers, leading to an inflammatory tachykinin release that results in airway constriction. [7]

Satoh et al. have increased validity and strengthened their primary study based on the number of subjects (100), the diversity in pathways examined and the specificity of receptors narrowed down to the airway in the second study.[6,7] Both studies of guinea pigs have a draw back in that they are animal studies, not human studies. The animals were pretreated with urethane for basal anesthesia prior to gas administration. Urethane has been shown to increase bronchoconstrictive effects, especially acetylcholine; this possibly skewed the results of the parasympathetic subject group.[6,7]

A literature review done in 2012 by Jakobson discussed the findings and appropriate clinical applications of desflurane to practice over the past decade.[1] Numerous conclusions about desfluranes metabolism, safety, quick emergence, use in the elderly and obese, and the possible cardio protective properties are discussed. The most applicable finding to the topic at hand is a discussion of the synergistic effect of opioids, like remifentanil or fentanyl, when higher MAC of desflurane is needed to achieve/maintain general anesthesia. This adjunct to desflurane has been shown to blunt the possible sympathetic effects caused from a rapid increase in MAC, allowing for fewer symptoms like coughing or breath holding. Jakobson discusses many positive uses of desflurane in this review. While informative and a road map to numerous sources of information, this study shows bias leaning toward only the positive uses of desflurane. Many sources were discussed but the levels of evidence vary, 40% being level I control studies and 60% being lower level V expert opinions or qualitative studies. [1,8]

Numerous clinical and laboratory studies evaluated the pulmonary effects of desflurane versus sevoflurane have been discussed in this narrative review. Following a review of the literature, 8 articles reviewed, two primary conclusions were obtained regarding the pulmonary effects of desflurane: 1) bronchodilatory effects are dose dependent and 2) irritant receptors play a significant role in moderating increases in airway resistance.[3-7] Desflurane, when delivered at 1 MAC, causes equivalent bronchodilation to that of sevoflurane in patients with no known smoking history or chronic lung/airway disease.[3-5] One MAC desflurane caused significant airway resistance in patients with a history of smoking or chronic lung/airway disease.[5] This effect was attributed to the understanding that smokers or those with chronic lung diseases have an increased sensitivity of irritant lung receptors and decreased compliance prior to anesthetic administration.[5] Desflurane at 2 MAC shows a decrease in respiratory compliance and an increase in airway resistance in the first 10 minutes of administration among all populations tested.[2-7] This dose dependent effect is directly correlated with the higher gas density property of Desflurane and often slower to achieve equipotent end alveolar gas concentration.[2] Desflurane is less potent than sevoflurane and requires a higher percentage to reach 1 MAC, 6%, versus sevoflurane 2.1%.[4] An increased density and higher administration percentages due to the decreased potency are the main reasons an increase in resistance and decrease in compliance is seen with higher MAC of desflurane.[2] With desflurane administration above 1 MAC, there is an immediate increase in airway resistance followed by a decrease in pulmonary compliance that levels off (7 to 10 minute plateau effect). There is a subsequent decrease in airway resistance and increase in pulmonary compliance that takes place on the descending side of the plateau with administration at a MAC greater than 1 in both smokers & non-smokers.[3,5] This trend is due to the slower bronchodilatory effect of desflurane versus the immediate resistance from high gas density.[5]

Increased airway resistance is also linked to the pungency of desflurane when administered in doses above 1 MAC, an effect not exhibited by increased MAC of sevoflurane. Increased airway resistance is directly related to the release of tachykinins substance P and NKA from irritant gas receptors in the smooth airway muscle.[6,7] This increased constriction is caused by the release of tachykinins or sympathetic catecholamine's that lead to airway inflammation, i.e. edema and/or secretions. These inflammatory agents can be blunted by administration of TRPA1 selective antagonist HC030031, NKA antagonist MEN-10376, or opiods.[1,6,7] This conclusion has only been tested in animals, thus limiting its application to clinical practice.

Overall, desflurane does have equivalent bronchodilatory effects to that of sevoflurane when administered at 1 MAC in majority of patients. Clinically, anesthesia practitioner should be aware that even at therapeutic doses, 1 MAC, desflurane may cause an initial increased airway resistance and decreased pulmonary compliance that starts to return to baseline around 7-8 minutes in patients with a known smoking history or a chronic lung/airway pathophysiology. Anesthesia practitioner should consider this when administering desflurane to current or past smokers. If a dose above 1 MAC of desflurane is needed to achieve adequate sedation it is important to understand the plateau effect and that bronchodilation will take slightly longer, approximately 7-10 minutes, to achieve with desflurane versus sevoflurane.[3,5]

Author, Date	Design/ Purpose	Sample & Data Collection	Primary Outcome Variables	Results/Comments
(Nyktari, Papaioannou, Volakakis, Lappa, Margaritsanaki & Askitopoulou, 2006, 2011)	Randomized clinical trial to investigate the effects of 1 & 1.5 MAC desflurane on inspiratory resistance and its components during 30 minute administration in patients with healthy lung lungs undergoing general anesthesia	71 patients, age 18-75 years old, undergoing non-thoracic, non-abdominal elective surgery Group D: n=25 Group S: n=24 Group I: n=22 R_{rs} – collected by the inspiratory hold maneuver Screen pneumotachgraph: measure flow & tidal volume Pressure transducer: measure inspiratory pressures	Baseline (after intubation, before volatile agents) & every 5 min after volatile agents: 30 min - 1MAC 30 min - 1.5MAC Total inspiratory resistance (R_{rs}) Minimal resistance (R_{min}) Effective resistance (D_{Rrs})	1 MAC: No significant difference was shown between agents 1.5 MAC: Desflurane showed a significant increase in all effective parameters; Sevoflurane & Isoflurane had no significant change Possible explanations as to why desflurane increases airway resistance at higher MAC: the density of the gas at higher volumes or airway receptors agonize bronchoconstriction
(Jakobsson, 2011)	Review article of 71 RCTs - an update of clinical experience w/3rd generation anesthetics, focus on the efficacy and safety of desflurane	Multiple topics & samples from various RCTs/case studies described: Potency & MAC, pharmacokinetics, dynamics & cost, drug metabolism, obese & elderly patients	None listed	Combines many studies, majority from 1991-present, that exhibit positive clinical uses for desflurane and evidence to support this This article contains a wealth of information on RCTs with desflurane; it seems to have a biased author opinion towards the positive implications of desflurane
(Dikmen, Eminoglu, Salihoglu, Demiroluk, 2003)	Randomized clinical trial to compare the changes in respiratory mechanics occurring during MAC of desflurane, sevoflurane & isoflurane, following intravenous induction & tracheal intubation.	67 patients with an ASA of I-II having gastrointestinal surgery Group D: n=22 Group S: n=23 Group I: n=22 VenTrak respiratory measured respiratory mechanics Flow sensor to measure respiratory resistance (Rr), dynamic compliance (Cdyn) and peak respiratory airway pressure (PIP)	T1- Baseline T2 – following 5 min of inhalation of 1 of the 3 gases at 1 MAC T3 – following 5 min of inhalation of 1 of the 3 gases at 2 MAC 1 MAC concentration: Isoflurane 1.15% Sevoflurane 2% Desflurane 6% 2 MAC concentration: Isoflurane 2.3% Sevoflurane 4% Desflurane 12%	T2 - all gases caused increase in Cdyn & decrease in PIP & Rr T3 - Group S: decrease in Rr Group I: no significant change Group D: PIP & Rr increased, Cdyn Desflurane has a bronchodilator effect at 1 MAC. At 2 MAC desflurane is seen to cause increased airway resistance. Desflurane has dose dependent effects

Author, Date	Design/ Purpose	Sample & Data Collection	Primary Outcome Variables	Results/Comments
(Satoh, Yamakage, Kobayashi, Tohse, Watannabe & Namiki, 2009, 2009)	Randomized control trial to clarify the increasing mechanism of desflurane on lung resistance by examining the vagal nerve reflexes in guinea pigs	99 four week old, male, pathogen free Hartley guinea pigs 60 non-treated divided into 6 groups of 10 (n=6 each) Desflurane at 0, 0.5, 1.0, 1.5 or 2.0 MAC Sevoflurane at 0, 0.5, 1.0, 1.5 or 2.0 MAC 24 treated (n = 6 each group) then exposed to desflurane 15 treated (n = 3 each group) then exposed to sevoflurane Transpulmonary pressure (P_{TP}): Statham differential transducer Flow Rate (V): Fleishc pneumotachograph & Statham transducer Total lung resistance (R_L) & Dynamic lung compliance (C_{Dyn}): equation of motion of the respiratory system- continuously analyzed by online breathe-by-breathe analysis	None listed	**Untreated**: no change in R_L or C_{Dyn} seen until MAC over 1.0. *Desflurane* at MAC over 1.0 increased R_L and decreased C_{Dyn}. *Sevoflurane* increased C_{Dyn} at 2.0 MAC **Atropine**: no significant changes **Vagotomy**: 30% inhibited first peak, but not second peak **Tachykinin antagonist**: Desflurane at 2.0 MAC did not have an effect **Capsaicin**: Desflurane at 2.0 MAC had no effect on R_L & only a small effect on C_{Dyn} Pretreatment with atropine had no effect, while pretreatment with tachykinin receptor antagonist inhibited the unwanted side effects of desflurane at higher MAC. Implying that desflurane stimulates more of the sensory afferent C-fibre pathways than the parasympathetic efferent pathway.

Conclusion

The research question explored in this review leads to the understanding that desflurane is a bronchodilating agent as is sevoflurane. When delivered at a MAC greater than 1.0 desflurane will cause decreased pulmonary compliance and increased airway resistance due to its high density, in comparison to sevoflurane.[2] This increased airway resistance is due to a property of the gas affecting flow. Baxter Healthcare Corporation, manufactures of the inhalation agent desflurane, recommend that it not be used by itself as a sole induction agent in specific populations due to its pulmonary effects, i.e. history of airway damage/disease or in pediatric patients.[9] Baxter recommends the conjugate use of opiods when desflurane induction is being performed to help attenuate the sympathetic response to the high density of this volatile anesthetic, thus allowing for a lower MAC delivery of desflurane. [9]

In summary desflurane, in comparison to sevoflurane, has been shown to cause higher airway resistance and decreased pulmonary compliance in a dose dependent manner with varied effects among specific patient populations. We can incorporate this understanding into clinical practice of the physical properties of a high-density gas and the pathophysiologic effects on the airway. Desflurane is an excellent volatile anesthetic with numerous benefits to patients, i.e. quick emergence due to low blood and tissue solubility, reduced chance of delayed extubation, rapid

intraoperative hemodynamic control, fast return of swallow and gag reflex post operatively, a low accumulation in adipose tissue benefiting anesthesia in the obese population, etc.[1]

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

Esmolol Infusion: An Opioid-Sparing Technique for Outpatient Surgery

Adam Farber, MSN
University of Southern California

Keywords: esmolol, infusion, opioid, sparing, perioperative

With more procedures being performed on an outpatient basis, anesthetists are challenged to provide anesthesia that results in rapid recovery and control of acute postoperative pain.[1,2] While opioids remain an important medication for pain control in any setting, overuse in the outpatient setting may lead to increased side effects and delayed discharge. Thus, multimodal and opioid-sparing techniques are beginning to gain traction, especially in outpatient surgery.

Opioids are a mainstay of anesthesia practice, although they cause an array of adverse effects. Side effects include nausea, vomiting, sedation, urinary retention and respiratory depression which decrease patient satisfaction and can lead to delayed discharge times and increased costs during ambulatory surgery.[1] Despite the heavy reliance on opioids by practitioners in outpatient surgery, many patients continue to experience postoperative pain.[1,3] Opioid-sparing techniques are becoming the standard of care in ambulatory surgery to decrease medication side effects and increase patient satisfaction.[1]

Medications that block the autonomic response and provide intraoperative hemodynamic stability have been shown to reduce opioid consumption and postoperative nausea and vomiting (PONV).[2,4] For these reasons, α_2-agonists such as dexmedetomidine and clonidine have become reasonable anesthetic adjuncts during outpatient procedures.[2,4] The success of α_2-agonists in maintaining intraoperative hemodynamic stability while improving perioperative outcomes and pain management during ambulatory surgery has led to research of other sympatholytic medications as possible anesthetic adjuncts.[2,4] Research over the past decade has shown that additional categories of sympatholytic medications, mainly β-receptor blockers, have similar advantages in outpatient procedures.[2]

Esmolol is a cardioselective, ultra-short-acting β_1-receptor blocker. The ability of esmolol to rapidly decrease heart rate (HR) and blood pressure (BP) for short durations of time has led to multiple uses in the perioperative setting.[5] Recent studies have shown that esmolol administered as an infusion has the ability to decrease intraoperative and postoperative opioid requirements.[6-11] The purpose of this evidenced-based analysis is to investigate the opioid-sparing advantages of esmolol for laparoscopic outpatient procedures.

Methodology

A population, intervention, comparison, and outcome (PICO) question was developed to guide a review of current literature. The PICO question posed was: "In adult patients undergoing laparoscopic procedures (P), does the use of an esmolol infusion intraoperatively (I), compared with no esmolol administration intraoperatively (C), result in decreased intraoperative or postoperative opioid consumption (O)?"

Current literature was identified using 3 electronic databases: MEDLINE/Pubmed, The Cochrane Library, and Google Scholar. Using a date filter, returned searches included studies from August 2007 to April 2014. The following keywords were used individually or in combination: *esmolol*, *infusion*, *analgesia*, *intraoperative*, *postoperative*, *opiate*, and *sparing*.

Retrieved studies were included for review if an esmolol infusion was administered intraoperatively during a laparoscopic procedure. Studies were included if results were published regarding either intraoperative or postoperative opioid consumption, even if this was not the primary interest of the original study. Searches yielded 6 studies that met inclusion criteria; all of which were randomized controlled trials (RCT) (Level II evidence).[6-11]

Literature Review

Six single-center studies were reviewed to determine if an intraoperative esmolol infusion decreased opioid consumption in the intraoperative or postoperative period.[6-11] Two studies specifically had a primary interest in opioid consumption in the post anesthesia care unit (PACU),[6,7] while one study researched remifentanil consumption intraoperatively when esmolol was administered as an infusion.[8] Two studies identified effects of intraoperative esmolol infusions on PONV.[9,10] The remaining study had a primary interest in sevoflurane concentrations during maintenance of general anesthesia when esmolol was administered as an infusion intraoperatively.[11] For studies that did not have intraoperative or PACU opioid consumption as a primary interest, opioid consumption was included as a secondary point of interest.[9-11]

Three studies involved laparoscopic cholecystectomy patients.[6,7,10] Two included laparoscopic gynecologic procedures,[8,11] and one included laparoscopic appendectomies.[9] The studies with patients undergoing cholecystectomy and appendectomies included men and women.[6,7,9,10] Five studies included patients with an American Society of Anesthesiologist (ASA) physical status of I or II.[6-9,11] The research by Ozturk et al[10] utilized ASA II patients with controlled hypertension.

Two studies reported significantly reduced opioid consumption intraoperatively in patients who received an esmolol infusion, but did not report postoperative opioid consumption.[9,10] One study reported significantly decreased intraoperative and postoperative opioid consumption in patients who received esmolol as an infusion intraoperatively.[8] Due to primary interest and study protocol, the remaining three studies did not report intraoperative opioid consumption, but found a significant decrease in opioid requirements throughout the postoperative period in patients who received an intraoperative esmolol infusion.[6,7,11] In total, three studies reported decreased intraoperative opioid consumption,[8-10] while four studies found decreased postoperative opioid consumption with the administration of esmolol intraoperatively.[6-8,11]

The observer-blinded RCT by Collard et al[6] enrolled 90 patients, 18 to 85 years of age, undergoing elective laparoscopic cholecystectomy. Participants were randomized into three groups consisting of a control (fentanyl), esmolol, or remifentanil cohort. The participants received fentanyl 1 mcg/kg, esmolol 1 mg/kg, or remifentanil 1 mcg/kg IV bolus prior to induction based on group allocation. Induction was standardized between groups. Following induction all participants received acetaminophen 1.3 g per rectum and dexamethasone 8 mg IV. The control group received fentanyl 50 mcg IV bolus every 30 minutes throughout surgery. The esmolol group received esmolol 5-15 mcg/kg/min and the remifentanil group received remifentanil 0.1-0.5 mcg/kg/min. Esmolol, or remifentanil was titrated to maintain HR within 20% of baseline. Anesthesia was maintained with desflurane at 4-8% end-tidal concentration and titrated to keep bispectral index (BIS, Aspects Medical Systems Inc., Norwood, MA) values less than 60 and systolic blood pressure (SBP) within 20% of preoperative baseline. Fifteen minutes before closure all patients received ketorolac 30 mg and droperidol 0.625 mg IV. Bupivacaine 0.25% with epinephrine 1:200,000 (10 mL) was infiltrated at incision sites prior to suturing. The esmolol or remifentanil infusions were discontinued at the end of the procedure. Pain was treated in PACU according to study protocol. Registered nurses (RNs) in the PACU were not aware of the study hypothesis and the anesthesia record was not provided to RNs or research fellows. The esmolol group required the least amount of fentanyl (91.5 +/- 42.7 mcg) in the PACU (*P* =

0.0001). The remifentanil group required almost two and a half times the amount of fentanyl (237.8 +/- 54.7 mcg) than the esmolol group ($P < 0.0001$). The control group had significantly higher fentanyl (168.1 +/- 96.8 mcg) requirements in the PACU when compared to the esmolol group ($P = 0.001$). Postoperatively, 30% of patients reported nausea in the esmolol group versus 66.7% in the control group and 67.9% in the remifentanil group ($P = 0.004$) and patients from the esmolol group were discharged 45-60 minutes earlier from PACU ($P = 0.003$) compared with the other two groups.

Hwang et al[8] enrolled 56 patients in a double-blinded, placebo-controlled RCT. The study included women aged 20-60 years old undergoing various laparoscopic gynecologic procedures lasting less than 2 hours. Participants were randomized into a control (saline) or esmolol group. The anesthesiologist and participants were blinded to group allocation. Induction was standardized between groups. Anesthesia was maintained with sevoflurane 2% inspired concentration in a mixture of oxygen 1 L/min and air 3 L/min. Remifentanil 2 ng/mL was administered via a computer-assisted (Orchestra Base Primea; Fresenius Vial, Grenoble, France) target-controlled infusion. Remifentanil was titrated to maintain adequate anesthetic depth 30 minutes after abdominal insufflation. Hwang et al defined adequate anesthesia depth by maintaining BIS values between 50-60 and SBP within 15% of baseline measured every minute for three consecutive recordings. Remifentanil effect-site concentration to maintain this adequate depth of anesthesia resulted in a before-concentration dose. Differences in before-concentration doses of remifentanil between groups were not statistically significant ($P = 0.588$). After the before-concentration dose was determined, the esmolol group received esmolol 0.5 mg/kg IV bolus followed by an esmolol infusion of 30 mcg/kg/min. The control group received the same volume of NS. Remifentanil was titrated every 5 minutes throughout the procedure by 0.2 ng/mL to maintain an adequate depth of anesthesia. Sevoflurane, remifentanil, and esmolol were discontinued at the time of trocar removal. Adjusted remifentanil concentrations throughout surgery were averaged and resulted in the after-concentration dose. After-concentration doses were compared to before-concentration doses. The esmolol group required 33% less remifentanil than the control group ($P < 0.005$) to maintain an adequate depth of anesthesia as defined by Hwang et al. The esmolol group also received a significantly lower total dose of remifentanil (0.09 +/- 0.01 mcg/kg/min) than the control group (0.14 +/- 0.03 mcg/kg/min, $P = 0.031$). All patients received ondansetron 4 mg IV 30 minutes prior to surgery end. A certified registered nurse anesthetist (CRNA) blinded to group allocation assessed patients in the PACU. Pain was treated per study protocol. The two groups did not differ significantly in the number of patients that required opioids in the PACU ($P = 0.096$). Median PACU fentanyl consumption was reported. The esmolol group required significantly lower total fentanyl consumption compared to the control group (25 (25-50) mcg versus 50 (25-75) mcg respectively, $P = 0.008$). No significant differences were found between the two groups with regard to PONV ($P = 0.626$), number of patients requiring antiemetics ($P = 0.5$), or PACU length of stay ($P = 0.635$).

Lee et al[9] conducted a placebo-controlled RCT with 60 patients undergoing laparoscopic appendectomies. Patients ranged from 18-45 years old. Participants were randomized into a control group and an esmolol group. Prior to induction, the esmolol group received esmolol 1 mg/kg IV while the control group received the same volume of NS. Induction was standardized between groups. Anesthesia was maintained with remifentanil 0.2-0.5 mcg/kg/min and propofol 75-85 mcg/kg/min. Remifentanil was titrated to maintain BIS levels of 40-60. Esmolol 10

mcg/kg/min was administered to the esmolol group after induction and the control group received the same volume of NS. Prior to extubation the esmolol group received esmolol 1 mg/kg IV and the control group received the same volume of NS. The esmolol or control infusion was discontinued after extubation. Total doses of remifentanil were recorded and it was found that the esmolol group had significantly lower remifentanil consumption intraoperatively compared to the control group (191.2 +/- 58.8 mg versus 285.4 +/- 68.2 mg respectively, P = 0.035). Pain was treated in the PACU according to study protocol. Patients received diclofenac intramuscularly (IM) and no patients required opioids in either group. In the PACU patients in the esmolol group, when compared to the control group, reported lower visual analogue pain scores (0-100) at 30 minutes (45.1 +/- 2.4 versus 60.2 +/- 4.2 respectively, P = 0.014), consumed less diclofenac (120 +/- 30 mg versus 180 +/- 75 mg respectively, $P < 0.05$), a higher rate of experiencing no PONV at 30 minutes (77% versus 43% respectively, P = 0.025), and required less antiemetic medication (20% versus 37 % respectively, P = 0.04).

López-Álvarez et al[7] studied 60 patients ages of 18-85 years old undergoing laparoscopic cholecystectomy. Participants were randomized into an esmolol or remifentanil-ketamine group. During induction, patients in the esmolol group received esmolol 0.5 mg/kg IV bolus followed by an esmolol infusion ranging from 5-15 mcg/kg/min. Induction for the remifentanil-ketamine group consisted of ketamine 0.5 mg/kg IV followed by a remifentanil infusion ranging from 0.1-0.5 mcg/kg/min. Other induction agents were standardized per protocol. After induction, all patients received paracetamol 1 g IV and dexamethasone 8 mg IV. Sevoflurane was titrated to 1-1.5% mean alveolar concentration in a mixture of oxygen 2 L/min and air 2 L/min. Both groups received dexketoprofen 50 mg IV, a non-steroidal anti-inflammatory, and ondansetron 4 mg IV 30 minutes prior to surgery end. At the end of the procedure levobupivacaine 0.5% was infiltrated at incision sites. The anesthesiologist who administered anesthesia was not involved in patient care in the PACU. The RNs caring for the patient in PACU did not receive the anesthesia record and were blinded to the allocated group, as were patients. Pain was treated in the PACU according to study protocol. Median PACU morphine consumption was reported. The remifentanil-ketamine group received 5 (4-6) mg of morphine IV, while the esmolol group received 0 (0-2) mg IV ($P < 0.001$). In the remifentanil-ketamine group, 83% of patients received morphine versus 23% in the esmolol group (60%, 95% confidence interval, 36 to 75) in the PACU. There were no significant differences in PONV between the remifentanil-ketamine group (26%) and the esmolol group (20%).

Fifty-four patients were enrolled in the randomized, double-blind, placebo-controlled study by Moon et al.[11] The participants were randomized into a control (saline) group or an esmolol group. The study included women aged 20-60 years old undergoing various laparoscopic gynecological procedures that lasted less than 2 hours. An anesthesiologist not involved in anesthetic management prepared esmolol and placebo solutions with the labels covered. The anesthesiologist providing anesthesia and the participants were blinded to group allocation. Anesthesia was induced per protocol and maintained with remifentanil 1 ng/mL delivered by target-controlled infusion and sevoflurane in a mixture of oxygen 1 L/min and air 3 L/min. Sevoflurane was titrated to a BIS value of 50-60 and protocol target hemodynamics. When target values were met, the percent volume noted on the vaporizer resulted in a before-concentration dose. The esmolol group received esmolol 0.5 mg/kg IV loading dose followed by an esmolol infusion at 30 mcg/kg/min. The control group received the same volume of NS. Sevoflurane was

titrated to protocol target hemodynamics throughout the procedure. Sevoflurane, remifentanil, and esmolol was discontinued at the time of trocar removal. The after-concentration of sevoflurane was calculated using mean inspired concentrations. All participants received ondansetron 4 mg IV 30 minutes prior to surgery end. Those patients receiving an esmolol infusion intraoperatively required 18.2% less inspired concentration of sevoflurane compared to the control group ($P < 0.01$). On arrival to the PACU, patients were assessed by a CRNA who was blinded to group allocation. In the PACU, 89% of the control group required fentanyl compared to 56% of the esmolol group ($P < 0.05$). Median PACU fentanyl consumption was reported. The esmolol group required significantly lower total fentanyl consumption compared to the control group (25 (0-25) mcg versus 50 (50-50) mcg respectively, $P < 0.005$). There were no differences between groups in regards to PONV or antiemetic doses in the PACU. The control group had significantly prolonged discharge times from PACU compared to the esmolol group (40.9 +/- 17.2 min versus 30.6 +/- 9.6 respectively, $P < 0.05$).

In yet another study, Ozturk et al.[10] conducted a double-blind RCT with 40 patients undergoing laparoscopic cholecystectomy. The study included patients with controlled hypertension ages 44-70 years old. Only patients taking angiontensin-converting enzyme inhibitors with or without concomitant diuretic therapy were enrolled in the study. Inclusion criteria were a preoperative blood pressure less than 140 mmHg systolic and 90 mmHg diastolic. Patients and the anesthesiologist providing anesthesia were blinded to group allocation. All participants were randomized into an esmolol group or a placebo group. Prior to surgery all patients were premedicated with diclofenac 75 mg IM, dexamethasone 4 mg IV, and metoclopramide 10 mg IV. Immediately prior to a standardized induction, the esmolol group received esmolol 1 mg/kg IV over 3 minutes. The placebo group received the same volume of lactated ringer's (LR) over the same time period. Following induction, the esmolol group received an esmolol infusion 5-10 mcg/kg/min titrated to a target HR of 65-75 beat per minute. The placebo group received the same volume of LR. Anesthesia was maintained with desflurane 2.5% inspired concentration in 65% nitrous oxide (N_2O) and oxygen. All participants received alfentanil 0.5-3 mcg/kg/min IV which was titrated to a mean arterial pressure within 20% of baseline. At removal of the laparoscope, the desflurane and N_2O were discontinued. Surgeons infiltrated lidocaine 1% and bupivacaine 0.25% (10 mL) at incision sites prior to closure. The esmolol or LR solution was discontinued after extubation. The esmolol group required significantly less alfentanil compared to the placebo group (4.7 +/- 0.8 mg versus 5.5 +/- 0.9 mg respectively, $P = 0.001$). Observers collecting PACU data were blinded to group allocation. Pain was treated based on study protocol. Tramadol 100-400 mg IV infusion and diclofenac 75 mg IM were used to treat pain in the PACU. The esmolol group required less postoperative analgesic ($P = 0.003$). Participants in the esmolol group required no doses of postoperative analgesics (35%) or one dose per protocol (60%). The placebo participants required one dose (70%) or two or more doses (30%) of analgesics. Participants in the esmolol group had less incidence of PONV compared to the placebo group ($P < 0.001$) with 8 participants in the esmolol group requiring antiemetic treatment versus all participants in the placebo group receiving at least one dose of antiemetic ($P = 0.003$).

Methods varied between studies, which may indicate a weakness of the literature review. There were differences between the use of esmolol in regards to bolus dose, timing of dose, and rates of infusion. Multiple studies used intraoperative adjunct analgesics such as acetaminophen,

Source	Sample (n)	Esmolol bolus/infusion rate	Intraoperative opioids	PACU opioids
Collard,[6] 2007	• Men and women aged 18-85 • ASA physical status I-II • Elective outpatient laparoscopic cholecystectomy • (n = 90)	• Esmolol 1 mg/kg IV bolus immediately prior to induction • Post-intubation infusion at 5-15 mcg/kg/min IV • Esmolol discontinued at the end of the procedure	• Control (fentanyl) group received fentanyl 200.5 +/- 54.9 mcg IV • Remifentanil group received remifentanil 1.3 +/- 0.4 mcg/kg IV • Esmolol group did not receive opioids intraoperatively	• ↓ fentanyl consumption in esmolol group compared to other groups ($P = 0.0001$) • Esmolol group received fentanyl 91.5 +/- 42.7 mcg IV • Control (fentanyl) group received fentanyl 168.1 +/- 96.8 mcg IV • Remifentanil group received fentanyl 237.8 +/- 54.7 mcg IV
Hwang et al,[8] 2013	• Women aged 20-60 • ASA physical status I-II • Laparoscopic gynecologic surgery of < 2 h duration • (n = 56)	• Esmolol 0.5 mg/kg IV bolus followed by an infusion of 30 mcg/kg/min IV after intubation and establishment of remifentanil effect-site concentration • Esmolol discontinued with removal of trocar	• Remifentanil (mcg/kg/min IV) in control received 0.14 +/- 0.03 vs 0.09 +/- 0.01 in esmolol group ($P = 0.031$) • Total remifentanil before-concentration (ng/mL IV) in control 2.9 +/- 1.3 vs 2.7 +/- 0.9 ($P = 0.588$) • Total remifentanil after-concentration (ng/mL IV) in control 3.2 +/- 1 vs 1.8 +/- 0.6 ($P < 0.005$)	• ↓ fentanyl dose as rescue analgesic in PACU in esmolol group ($P = 0.008$) • Esmolol group received fentanyl 25 (25-50) mcg IV* • Control group received fentanyl 50 (25-75) mcg IV *
Lee et al,[9] 2010	• Men and women aged 18-45 • ASA physical status I-II • Laparoscopic appendectomy • (n = 60)	• Esmolol 1 mg/kg IV bolus prior to intubation • Esmolol infusion of 10 mcg/kg/min • Emolol 1 mg/kg IV bolus prior to extubation • After extubation esmolol discontinued	• Total remifentanil (mcg IV) in control received 285.4 +/- 68.2 vs 191.2 +/- 58.8 ($P = 0.035$)	• N/A study administered diclofenac IM in PACU
López-Álvarez et al,[7] 2012	• Men and women aged 18-85 • ASA physical status I-II • Laparoscopic cholecystectomy • (n = 60)	• Esmolol 0.5 mg/kg IV bolus at induction followed by infusion of esmolol at 5-15 mcg/kg/min • Does not report when esmolol was discontinued	• Remifentanil-ketamine group received remifentanil (mcg/kg IV) 21 (standard deviation 7) • Esmolol group did not receive opioids intraoperatively	• ↓ morphine requirement in PACU in esmolol group ($P < 0.001$) • Esmolol group received morphine 0 (0-2) mg IV* • Remifentanil-ketamine group received morphine 5 (4-6) mg IV* • 83% of patients in remifentanil-ketamine group required morphine vs 23% in esmolol group (difference 60%; 95% CI, 36 to 75)
Moon et al,[11] 2011	• Women aged 20-60 • ASA physical status I-II • Elective laparoscopic gynecological surgery of < 2 h duration • (n = 54)	• Following intubation and establishment of sevoflurane concentration, esmolol 0.5 mg/kg IV bolus followed by an infusion of 30 mcg/kg/min • Esmolol discontinued at the time of trocar removal	• Remifentanil effect-site concentration 1 ng/mL to both groups	• ↓ doses of PACU rescue analgesics in esmolol group ($P < 0.05$) • ↓ fentanyl dose as rescue analgesic in PACU in esmolol group ($P = 0.005$) • Esmolol group received fentanyl 25 (0-25) mcg IV* • Placebo group received fentanyl 50 (50-50) mcg IV*
Ozturk et al,[10] 2008	• Men and women aged 44-70 • ASA physical status II • Hypertensive patients with BP < 140/90 preoperatively • Laparoscopic cholecystectomy • (n = 40)	• Esmolol 1 mg/kg IV bolus immediately prior to induction • Following induction, esmolol infusion was started and maintained at 5-10 mcg/kg/min • Esmolol discontinued after extubation	• ↓ total dose of alfentanil in esmolol group ($P = 0.001$) • Placebo group received alfentanil (mg IV) 5.5 (0.9) vs 4.7 (0.8) in esmolol group	• Tramadol 100-400 mg IV and diclofenac 75 mg IM were used to treat pain in PACU • ↓ number of patients requiring one or more doses of PACU analgesics ($P = 0.0032$, Fisher's exact test)

Key: ASA = American Society of Anesthesiologists; CI = confidence interval; IV = intravenous; IM = intramuscular; PACU = post-anesthesia care unit; ↓ = decreased; ↑ = increased
*Data presented by authors as median (interquartile range)

dexketoprofen, diclofenac, ketorolac, ketamine or local anesthesia while some omitted adjuncts completely. Differing results were found between studies in regards to PACU discharge times, incidence of PONV, and number of antiemetics given, which could be attributed to study methodology.

Although there were differences in study design and methodology, all of the studies reviewed concluded that opioid requirements were decreased in the intraoperative or postoperative periods when esmolol was administered as an infusion intraoperatively. Despite differing methodologies, the evidence that esmolol decreases opioid consumption is reinforced by each study. Other strengths of the current review include that no study reported adverse events, increased incidence of PONV, or delayed discharge times in patients who received an esmolol infusion compared to the control group.

It is important to note that esmolol is not appropriate for all patients. Esmolol is contraindicated in the parturient, in patients with sinus bradycardia, heart blocks greater than first degree, heart failure, pulmonary hypertension, and should be used cautiously in patients with renal failure.[5] Adverse effects of esmolol include hypotension and bradycardia.[5] In the studies reviewed, the protocols for treatment of decreases in HR and BP included the use of atropine, phenylephrine, or ephedrine. Esmolol infusions were not discontinued in any study; only one study reported the need to use atropine or ephedrine for treatment of decreased HR or BP.[7]

Conclusion

The ability to provide cost-effective care and expedient outpatient discharges without compromising patient safety or satisfaction is a major concern in anesthesia practice. Decreasing the administration of opioids has been shown to decrease PONV, discharge times, and costs.[1,2,6] The ability to maintain hemodynamic stability with intraoperative sympatholytic medications has proven to be a reasonable opioid-sparing technique.[2] The goal of this evidence-based practice analysis was to determine if intraoperative esmolol infusion would decrease opioid consumption in the intraoperative or postoperative period. Of the six studies reviewed, each concluded that an intraoperative esmolol infusion used as an adjunct during general anesthesia reduced intraoperative or postoperative opioid requirements, and thus, opioid related adverse effects. While more research in this area is necessary, the current review provides promising evidence that intraoperative esmolol infusions decrease opioid requirements during intraoperative and postoperative periods in ASA physical status I-II patients between the ages of 18-83 undergoing laparoscopic outpatient surgery.

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Mentor: Kären Kim Embrey CRNA, EdD

Editorial

Welcome to the 14th volume of the International Student Journal of Nurse Anesthesia (ISJNA) and our largest issue yet. In this edition you will find 20 case reports, one abstract, and two evidence-based practice analysis reports from 12 different nurse anesthesia programs. If you have read past issues you will also note a change in appearance from two columns to one, for ease of reading and to provide a better example of desired format for future authors. This decision was made at the most recent meeting of the student journal at the Assembly of School Faculty (AoSF) in New Orleans this past February, and many more ideas and where shared and discussed as well. I've mentioned a revision to the author guidelines is in progress – I hope to release the update sometime this summer. If anyone has any comments or suggestions please email me at intsjna@aol.com!

I would also like to officially introduce and welcome two new members of the Editorial Board:

MAJ Sarah Bellenger, CRNA, MSN – Darnall Army Medical Center; Fort Hood, TX

Connie L. Lorette, CRNA, PhD – Northeastern University

Shannon Pecka, CRNA, PhD – Bryan College of Health Sciences

Thank you for volunteering your time and talent to the student journal! I also appreciate the hard work of all of the individuals that serve as reviewers for the ISJNA – another thanks to all the CRNAs that offered to join the ranks at AoSF. The journal is thriving because of all of you!

Sincerely,

Vicki C. Coopmans, CRNA, PhD
Editor

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

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INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA
GUIDE FOR AUTHORS

MISSION STATEMENT

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

ITEMS ACCEPTED FOR PUBLICATION

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

ITEM PREPARATION & SUBMISSION

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

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

PEER REVIEW

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

General guidelines

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

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

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

Case Reports

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

Heading (see #9 above in General Guidelines)

[space]

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

[space]

Case Report (bold, 400-500 words)

[space]

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

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

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

[space]

Discussion (bold, 600-800 words)

[space]

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

[space]

References (bold)

[space]

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

[space]

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

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

Research Abstracts

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

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

EBP Analysis Reports

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

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

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

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

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

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

AMA MANUAL OF STYLE

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

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

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

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

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

Journal, 6 or fewer authors:

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

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

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

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

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

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

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

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

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

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

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

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

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

HOW TO SUBMIT AN ITEM

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

REVIEW AND PUBLICATION

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

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

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

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

PHOTOS

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

Vicki C. Coopmans, CRNA, PhD
Webster University
470 E. Lockwood Ave. Suite 15
St. Louis, MO 63119

SUBMISSION CHECK LIST

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

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

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

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

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

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

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