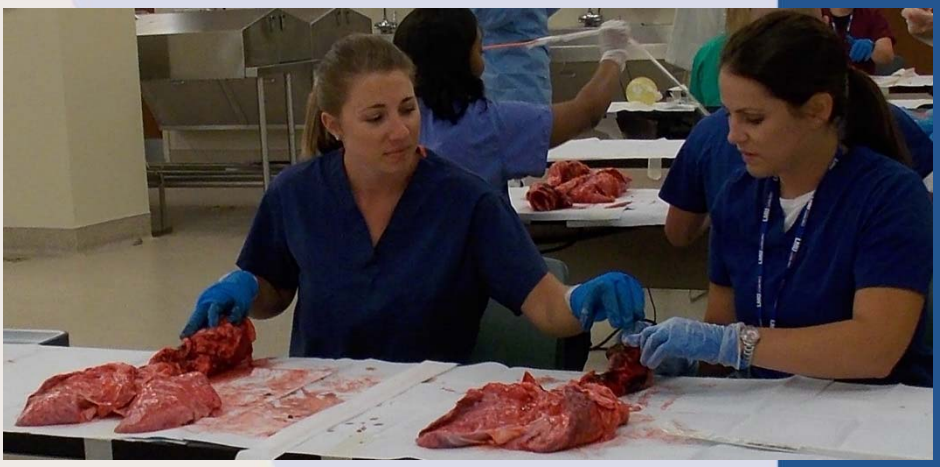
Volume 13 Number 3 Fall 2014

The International Student Journal of Nurse Anesthesia



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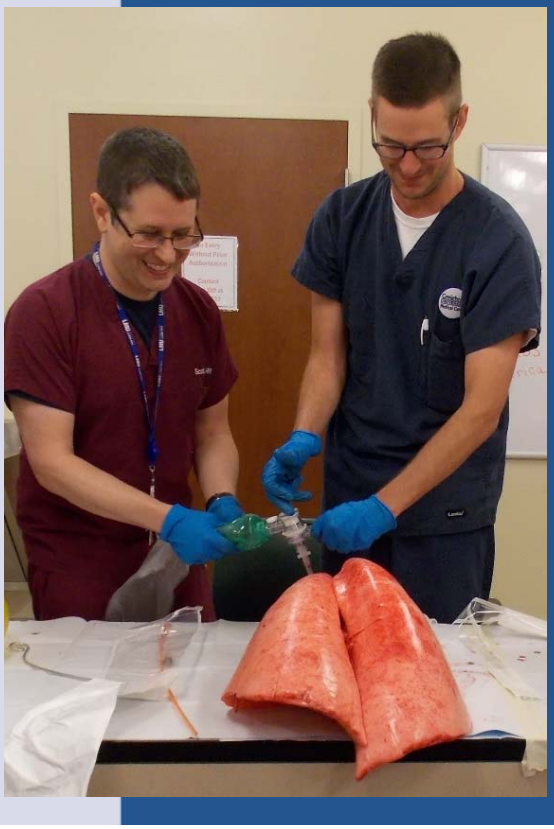

INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA
Vol. 13 No. 3 Fall 2014

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Front Cover:

Hailey Mullinax BSN and Lindsay Pate BSN, graduate students enrolled in the Lincoln Memorial University Nurse Anesthesia Program prepare to practice cricothryotomy skills on pig tracheas during an emergency airway laboratory session. The second image shows successful cricothyrotomy and lung ventilation by fellow students Scott Cantrell, BSN and Nicholas Quinton, BSN.

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Acute Adrenal Insufficiency after an Induction Dose of Etomidate

Kathleen Black, MS
Webster University

Keywords: etomidate, adrenal suppression, refractory hypotension

Etomidate is an induction agent often used for its hemodynamic stability. However, the administration of an induction dose of etomidate can result in acute adrenal insufficiency: a medical emergency. Etomidate has a known history of inhibiting 11-beta-hydroxylase, preventing the cortisol synthesis. The inhibition of this enzyme prevents cortisol from responding to surgical stress and triggers a state of adrenal insufficiency with refractory hypotension leading to hemodynamic instability and eventual cardiovascular collapse.[1,2]

Case Report

A 71-year-old, 93 kg, female patient presented for a closed reduction of the left shoulder. Significant medical history included hypertension, chronic obstructive pulmonary disease and hypothyroidism. Pertinent surgical history included a mandibular surgery from the 1980s. The patient had no known drug allergies and denied complications from previous anesthesia. The plan was to perform general anesthesia with an awake fiberoptic placement of an endotracheal tube (ETT) due to previous mandibular surgery. In the pre-operative area, glycopyrrolate 0.2 mg was administered intravenously (IV) and a dexmedetomidine bolus was initiated at 1mcg/kg and infused over 10 min. In addition, a 4% lidocaine nebulizer was administered for topical anesthesia.

In the operating room, standard noninvasive monitors were applied. Oxygen was delivered at 10 L/min via face mask. A total of midazolam 5 mg and alfentanil 1000mcg were given IV during the fiberoptic intubation in divided doses. Placement of ETT was confirmed. Ventilation mode was spontaneous intermittent mandatory ventilation. General anesthesia was induced with etomidate 20 mg and maintained with sevoflurane 1% with a mixture of 1 L/min of oxygen and 1 L/min of nitrous oxide. Dexamethasone 8mg was administered to decrease airway edema from the fiberoptic intubation and for post-operative nausea prophylaxis.

The patient was placed in the beach chair position and the procedure initiated. Ten minutes into the procedure, the patient's blood pressure began to fluctuate. Hypotension and bradycardia ensued with systolic blood pressure (SBP) falling to 70 mm Hg and heart rate (HR) in the 50s. Ephedrine 20 mg was administered and an IV fluid bolus was initiated. The blood pressure was unresponsive and additional doses of ephedrine were administered. Due to the lack of response from the ephedrine, vasopressin 1unit IV boluses were administered every 3 min. With SBP in the 80s and HR in the 60s, a vasopressin drip was initiated 0.05 units/min and titrated to effect. A second intravenous catheter was placed and colloid 250 mL was administered.

The case continued and control of the blood pressure was maintained with the vasopressin drip. At the conclusion of the procedure, the patient was spontaneously breathing with tidal volumes >5 ml/kg, minute ventilation <10 L/min and the patient was following commands. The trachea was

safely extubated and the patient was taken to the post anesthesia care unit. Thirty minutes after arrival in the PACU, ST changes were noted on the patient's EKG. An arterial line with cardiac output monitoring capabilities was placed and a cardiology consult called. Patient's SBP remained in the 60s and HR in the 50s despite vasopressors and IV fluid administration. An echocardiogram was performed at bedside. Results revealed the patient was intravascularly depleted despite fluid resuscitation totaling 5 L of crystalloid and 1 liter of colloid. Intravenous hydrocortisone 100 mg was administered. Within 30 min, the patient's hemodynamics became responsive to vasopressors resulting in SBP in the 100s. Acute adrenal insufficiency was diagnosed on the basis of failure to respond to fluid and vasopressor therapy while having a positive response after steroid injection after administration of etomidate. The patient was admitted to the surgical unit for observation and further treatment.

Discussion

Etomidate is a caboxylated imidazole-containing hypnotic induction agent commonly used in the hemodynamically unstable patient.[1] Etomidate was first introduced into clinical practice in 1972. It has been used for induction and maintenance of anesthesia and as a sedative in intensive care units (ICU).[2] Its hypnotic characteristics result from targeting γ-aminobutyric acid type A receptor subtypes ($GABA_A$) thus enhancing the affinity of the neurotransmitter GABA for these receptors.[2] Its pharmacokinetics and pharmacodynamics make it an appealing choice for induction of general anesthesia. Etomidate has a peak onset time of 1 minute after IV injection and fast redistribution from the target site of the brain to inactive tissues. The fast wake up time is due to its large volume of distribution. It is rapidly metabolized by hepatic enzymes and plasma esterases via hydrolysis with an elimination half-time of 2 to 5 hr. The hemodynamic stability etomidate provides, separates it from other induction agents. It does not suppress sympathetic tone nor myocardial function.[3] At a dose of 0. 3mg/kg, there are minimal changes in heart rate, stroke volume or cardiac output.[1,2]

During the 1980s, etomidate was first linked to adrenocortical suppression. Watt and Ledingham did a retrospective study in 1984 evaluating the mortality rate of ICU patients treated with etomidate versus morphine and benzodiazepines. They noted a decrease in mortality from 47% to 25% after cessation of etomidate infusions.[2] In a prospective randomized study done by Hildreth et al, the use of etomidate for rapid sequence inductions (RSI) in trauma patients was evaluated. Serum cortisol levels were assessed prior to administration of an induction dose of etomidate, and again at an interval of 4 to 6 hours after the RSI. The group who received etomidate experienced lower cortisol levels after the RSI, required more IV fluids and packed red blood cells transfusions, and had longer ICU stays than the non-etomidate group. The findings of these authors suggest the suppression of the adrenocortical function caused by etomidate leads to hemodynamic instability and greater resuscitation requirements.[4]

The current use of etomidate is now limited due to its known adrenal cortex suppression. It has been found to suppress the normal increased secretion of cortisol and aldosterone in response to surgical stress. This suppression has been found to last in patients for 6-8 hr after a single dose.[3] The mechanism of action of this suppression is the result of etomidate inhibiting the adrenal steroid synthesis by blocking the activity of

11-beta-hydroxylase.[3] The block occurs at the rate limiting step, preventing conversion of cholesterol to dihydrocholesterol. It causes a dose dependent reversible inhibition of 11-beta-hydroxylase. This enzyme is necessary for the synthesis of cortisol, aldosterone, 17-hydroxprogesterone, and corticosterone. Therefore, there is no increase in cortisol or aldosterone in response to the adrenocorticotropic hormone (ACTH).[5]

During a normal stress response, glucocorticoids play an important role. Cortisol increases the sensitivity of the blood vessels to circulating catecholamines, resulting in an increased heart rate and blood pressure. Gluconeogenesis, glycogenolysis, lipolysis and hepatic glucose secretion are also increased due to the elevated levels of circulating catecholamines and cortisol. Cortisol also has anti-inflammatory characteristics, decreasing histamine release and preventing lysosomal damage to healthy tissues.[6]

Manifestations of acute adrenal insufficiency include hypotension, decreased consciousness, and refractory distributive shock due to decreased systemic vascular resistance.[7] Adrenal insufficiency is related to catcholamine-resistant hemodynamic instability and should be suspected if refractory hypotension is present without a definitive cause.[8] It is considered a medical emergency and is associated with increased mortality. Diagnosis is usually made upon clinical presentation, however a random cortisol level <20 mg/dl is also definitive. If time allows in the clinical setting, adrenocorticotropin hormone simulation testing may also be performed.[2]

Etomidate was chosen as the anesthetic induction agent for this case after reviewing a previous anesthesia record. It was noted the patient had become very hypotensive after induction with propofol and several vasopressors were required to support her blood pressure. The patient had no history of adrenal insufficiency or prior steroid use. Etomidate was selected in order to maintain hemodynamic stability throughout induction and limit the need for vasopressor support. Adrenal insufficiency was suspected after a failure to respond to fluid and vasopressor therapy in conjunction with the previous administration of a single induction dose of etomidate 0.3 mg/kg. Hydrocortisone 100 mg was administered and the patient's hemodynamics became more stable within 30 min of administration. Cortisol levels were checked after the administration of hydrocortisone and therefore could not be used as an accurate assessment of adrenal insufficiency. After reviewing the literature, in the future, etomidate may not always be considered a first choice medication for patients with hemodynamic instability. Alternative induction places for cardiovascularly unstable patients could also include the use of high opioid and low dose propofol administration, as well as a combination of ketamine and propofol in order to prevent post-induction hypotension.

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Mentor: Martina Steed, CRNA, BSN, MS

One-Lung Ventilation for Total Pneumonectomy

Elizabeth M. Broome, MSN
Samford University

Keywords: Double-lumen tube, pneumonectomy, one-lung ventilation, thoracic surgery

Optimal operating conditions for cardiothoracic procedures, including total pneumonectomy, often require one-lung ventilation (OLV). Anesthesia practitioners must understand the physiologic changes that occur during OLV, which can make maintaining adequate oxygenation, ventilation, and hemodynamic conditions challenging. OLV is achieved by lung separation using a double-lumen bronchial tube (DLT), single-lumen tracheal tube with bronchial blocker, or single-lumen bronchial tube. DLTs are the most frequently used technique for OLV.[1] Correct positioning of the DLT by the anesthesia practitioner is essential, as a misplaced tube can alter one-lung ventilation and jeopardize the surgical procedure.[2]

Case Report

A 67-year-old, 183 cm, 97 kg male presented for a right total pneumonectomy following diagnosis of right lung bronchoalveolar carcinoma. The patient had a medical history of smoking, osteoarthritis, obstructive sleep apnea, gastroesophageal reflux, and hypertension. The patient's past surgical history included a right upper lung lobectomy. The patient denied previous anesthetic complications. The patient had no known drug allergies and current medications included colesevelam, aspirin, ketoconazole, meloxicam, metoprolol, omeprazole, tramadol, and valsartan. Preoperative vital signs were blood pressure 138/83 mm Hg, heart rate 68 beats/min, respiratory rate 14 breaths/min, and SpO2 98% on room air. Upon assessment, the patient had clear breath sounds except slightly diminished breath sounds in the right lower lobe. A right radial arterial line

and two left arm 18 gauge peripheral IVs were inserted prior to induction. Midazolam 2 mg IV was administered preoperatively.

On arrival to the operating room, the patient was preoxygenated with 10 L/min of oxygen via facemask and standard monitors applied. Anesthesia was induced using fentanyl 150 mcg, lidocaine 100 mg, and propofol 200 mg. After mask ventilation was verified, vecuronium 10 mg was administered. Direct laryngoscopy was performed and a 39 French left-sided double-lumen tube (DLT) was inserted into the trachea without difficulty. Correct placement of the DLT in the left bronchus was verified by passing a fiberoptic bronchoscope (FOB) down the tracheal lumen and observing the top portion of the blue bronchial cuff in the left bronchus. After the patient was positioned in left lateral decubitus position, correct placement of the DLT was again verified using the FOB. General anesthesia was maintained with sevoflurane 2.5-3% inspired concentration in a mixture of oxygen 1 L/min and air 1 L/min. Controlled ventilation was achieved with a tidal volume of 700 mL at a respiratory rate of 10 breaths/min. An end tidal CO_2 of 32-34 mmHg was maintained. Once the patient was prepped and draped, the tracheal lumen of the DLT was clamped and right lung deflated. The left lung was ventilated with a tidal volume of 500 mL at a respiratory rate of 14 breaths/min.

A baseline arterial blood gas (ABG) showed the following results on room air: pH 7.38, $PaCO_2$ 39, PaO_2 95, SaO2 98%. Results of an ABG drawn 20 minutes after one-lung ventilation (OLV) was initiated with 100% oxygen were: pH 7.43, $PaCO_2$ 42, PaO_2 397, SaO_2 100%. During the surgical procedure the patient received fentanyl 450 mcg, acetaminophen 1 gm, ketorolac 30 mg, and ondansetron 4 mg intravenously. OLV was implemented for a total of 123 minutes. At the end of the procedure neuromuscular blockade was antagonized and the patient returned to spontaneous ventilation. Once extubation criteria were met, the DLT was removed from the trachea and oxygen was administered by facemask. Upon extubation, the patient had a respiratory rate of 20 breaths/min with no respiratory distress and maintained SpO2 greater than 95% on 6 L/min O2 via face mask. The patient was transported to the PACU. Vital signs in the PACU were blood pressure 141/89, heart rate 72 beats/min, respiratory rate 12 breaths/min, and SpO_2 99% on 6 L/min O_2.

Discussion

Double-lumen endotracheal tubes are the most commonly used endotracheal tubes for lung isolation.[3] In this case, lung isolation was accomplished by placing a 39-french left-sided DLT in the patient's left bronchus. A curve should be placed at the end of the DLT by using the stylet.[3] As Foley and Slinger suggest, after the tip of the tube is through the patient's vocal cords, the stylet is removed and the anesthesia professional rotates the tube 90 degrees to the left, advancing the tube to a depth of 30 cm.[3] After inflating both cuffs, and confirming placement by auscultation, correct placement of the DLT is best confirmed by visualization using a fiberoptic bronchoscope (FOB). One study determined that despite positive clinical confirmation of DLT placement by auscultation and clamping maneuvers, fiberoptic bronchoscopy demonstrated a misplaced DLT that required repositioning in 37% of cases.[2] In this case, a fiberoptic bronchoscope was used to confirm correct placement after initial placement and after the patient was repositioned in the lateral decubitus position.

Risks associated with OLV include hypoxemia, acute lung injury (ALI), and barotrauma.[1] To minimize these risks, the anesthesia professional must understand important physiologic changes that occur during OLV. One factor influencing adequate ventilation and perfusion of the lungs during thoracic surgery is the patient's position. This patient was positioned in the left lateral decubitus position. A spontaneously breathing, upright patient has the greatest perfusion at the base of the lungs. The dependent alveoli are more compliant and receive the majority of the tidal breath. Gas exchange is most efficient in this position.[4] When the awake patient is placed in a lateral position, the dependent lung receives most of the tidal volume and perfusion and gas exchange remains efficient. However, in the anesthetized, spontaneously breathing patient in the lateral position, functional residual capacity is decreased, especially in the dependent lung.[4] This results in less compliant alveoli in the dependent lung, which results in the majority of ventilation going to the nondependent lung. Blood flow remains gravity-dependent and is distributed primarily to the dependent lung, resulting in a mismatch between ventilation and perfusion.[4] Once neuromuscular blockade occurs, loss of the contracting diaphragm results in ventilation shifting more to the nondependent lung, and an increased ventilation-perfusion mismatch.[4] Furthermore, when one-lung ventilation is required, a right-to-left intrapulmonary shunting (20-30%) occurs, due to the surgical lung being perfused but poorly ventilated.[1] This shunting often results in hypoxemia.

Blood flow to the non-ventilated lung is decreased due to hypoxic vasoconstriction (HPV), gravity, and surgical compression of non-dependent lung tissue and vessels. Anesthesia practitioners must be aware that drugs common in anesthesia inhibit HPV.[1] Halogenated inhalational agents have minimal effects on HPV if kept less than 1.5 minimum alveolar concentration (MAC).[4] In this case, sevoflurane was used at a MAC of 1.25-1.5 by closely monitoring the patient's end-tidal sevoflurane concentration. Factors that decrease blood flow to the ventilated lung also counteract HPV by diverting blood flow to the nonventilated lung. These include high PEEP in the ventilated lung, the development of HPV in the ventilated lung due to low inspired oxygen, and intrinsic PEEP resulting from inadequate expiratory times.[1] While many vasodilating drugs can inhibit the effect of HPV, drugs such as epinephrine and phenylephrine can also work in opposition to HPV by constricting oxygenated pulmonary vessels and thus reestablishing shunt flow.[4]

If hypoxemia is persistent and is not from a reversible cause (i.e. tube malpositioning), there are several strategies that increase oxygenation by decreasing shunt. One is administering oxygen, with or without continuous positive end-expiratory pressure (CPAP) of 5-10 cm H_2O to the nondependent lung. CPAP may also reduce injury to the nondependent lung by helping to prevent reperfusion injury.[5,6] Adding positive end expiratory pressure (PEEP) to the dependent, ventilated lung, or combining this with CPAP in the nondependent lung, are methods of improving oxygenation. In this case, 5 cm H_2O of PEEP was utilized with OLV to prevent alveolar collapse and the development of atelectasis. CPAP may also be used in the nondependent lung to increase oxygenation by decreasing shunting.[1] Another option to reduce hypoxemia is early ligation of the pulmonary artery. In pneumonectomy patients this may be used to improve oxygenation when other strategies do not

reverse hypoxemia.[4] Over distention of alveoli may reduce perfusion to the well-ventilated lung. It is important to maintain moderate tidal volumes (6 mL/kg) during OLV.[4] One study showed that higher intraoperative tidal volumes, 8.3 vs. 6.7 mL/kg, were associated with post-pneumonectomy respiratory failure.[7] This patient's OLV tidal volumes were decreased to approximately 5 mL/kg.

On post-operative day one, this patient remained extubated, was breathing room air, and demonstrated no signs of respiratory distress. A thorough understanding of the special equipment used in OLV, respiratory physiology and the changes that occur during OLV, as well as the properties of the anesthetic drugs used during this case can ensure that similar patients receive high quality, safe care.

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Mentor: Terri Cahoon, CRNA, DNP

Anesthesia and Postoperative Delirium in the Elderly

Jon F. Faigle, MSN
Wake Forest Baptist Health

Keywords: anesthesia, cognitive, delirium, postoperative, elderly

By the year 2030 the number of individuals 65 years and older is projected to be 71.5 million, up from 40.2 million in 2010.[1] With the number of older citizens increasing in the United States, the cognitive effects of anesthesia on elderly patients must be considered. Postoperative delirium (PD) is likely to develop in 15% to 50% of older surgical patients, resulting in increased morbidity, mortality, extended hospital stays, and increased nursing home placement.[1,2] Understanding the disorder is important to the anesthesia practitioner in order to limit the severity and incidence of PD.

Case Report

A 70-year-old, 160 cm, 87.5 kg female patient presented for a laparoscopic lower anterior rectum resection for a diagnosis of rectal cancer. The patient's past medical history included gastric esophageal reflux disease, diverticulitis, colon polyps, hypertension, anemia, and emphysema. Her past surgical history consisted of two colonoscopies and a tubal ligation. The patient reported problems with lethargy after her colonoscopy one month earlier; specifically she "felt out of it for days," was slow to wake up, and had a prolonged PACU stay. Her preoperative blood pressure was 120/78 mm Hg, heart rate was 93/min and $Sp0_2$ 97% on room air. Her preoperative electrocardiogram revealed normal sinus rhythm. Preoperative labs included a complete blood count with differential and a basic metabolic panel with no abnormal values.

In the preoperative holding room a 22 gauge peripheral intravenous (PIV) catheter was placed in the left hand and maintenance infusion a lactated ringer (LR) was started. Patient identity, consents, procedures and allergies were verified, and the patient was transported to the operating room without preoperative sedation. In the operating room the patient was assisted onto the operating table in a supine position. Standard monitors were applied and pre-oxygenation was initiated with 10 L/min of flow via facemask. General anesthesia was induced intravenously (IV) with lidocaine 90 mg, fentanyl 100 mcg, propofol 170 mg and rocuronium 50 mg. Direct laryngoscopy was performed and the trachea was intubated with a cuffed 7.0 mm endotracheal tube (ETT). End tidal CO2 ($EtCO_2$), and bilateral breath sounds (BBS) were confirmed, and ventilation was controlled mechanically. The volume control setting was used to provide 500 ml per breath, delivered 10 times per minute, and 5 cm H_2O of peep was applied.

General anesthesia was maintained with desflurane end tidal concentrations of 3%-4% and nitrous oxide 0.5 L/min blended with 1.5 L/min of oxygen. Intraoperative pain was managed with fentanyl totaling 150 mcg and methadone 4 mg IV. After induction and intubation a 20 gauge PIV was placed in the patient's left forearm and an additional infusion of LR was initiated. The patient was positioned in lithotomy with neutral alignment, and pressure points were checked and padded appropriately. Then, a moderate trendelenburg with right side declination was applied, and BBS were reconfirmed. The surgery was uneventful with minimal fluctuations in vital signs.

Upon completion of the case, the adductor pollicis muscle train-of-four revealed 3 of 4 twitches. Neuromuscular blockade was then reversed using 4 mg of neostigmine combined with 0.6 mg of glycopyrrolate. The Volatile agent was weaned off and the antiemetic ondansetron 4 mg was administered. After the anticholinesterase had time to take effect, the patient's neuromuscular blockade was reassessed finding 4 of 4 twitches, and sustained tetanus at 50Hz for 5 seconds without fade. The patient began spontaneously breathing at a rate of 10-14 breaths/min with tidal volumes of 300-350 mL, and began to follow commands. The ETT was then removed and oxygen was administered through a facemask at 8 L/min. The patient had an estimated blood loss of 300 mL, a urine output of 235 mL, and 1,700 ml of IV LR. The patient was transported to the post anesthesia care unit (PACU), report was provided to the PACU nurse, and transfer of care was completed.

Discussion

Postoperative delirium is an acute change in consciousness usually lasting between 24-72 hours, and effects focus, perception, memory and language.[3] It can be expressed as hyperactivity to agitation, hypoactivity to somnolence, or a mix switching from one extreme to the other.[3] The patient described above had a mental status/level of consciousness change after a previous surgery, which indicated she might have suffered from PD. This anesthetic complication, and the patient's advanced age made her susceptible to developing PD after her colon resection. Considering these attributes, the anesthesia practitioners created a plan to minimize risk for postoperative delirium.

PD occurs in 15% to 50% of elderly patients receiving major surgeries, but the practitioner can attenuate some anesthetic risks.[2] There are several methods the anesthesia practitioner can reduce patient risk for PD including: manipulating inhaled and intravenous anesthetics, limiting the depth of sedation, controlling postoperative pain levels, and prescreening for cognitive impairments.[4] A combination of desflurane and nitrous oxide was used for this case. Desflurane was chosen for its rapid elimination and paired with nitrous oxide to reduce desflurane's alveolar concentration.[1] This mixture also allowed for rapid elimination of the volatile anesthetic and afforded a faster emergence. A current review of literature failed to populate information regarding this practice to reduce PD. It should also be noted that nitrous oxide is not typically used in abdominal surgeries.[1] Therefore, we were ready to abort that plan if necessary.

To decrease the incidence and severity of PD, literature supports avoiding the use of certain sedatives, narcotics, hypnotics and anticholinergics.[1,2,5] Sedatives, mainly benzodiazepines, increase the incidence and severity of delirium in the elderly postoperative patient.[6] As previously stated, the patient was not sedated prior to surgery. As a result she became anxious, began crying and expressed fear upon entering the operating room. As an alternative to benzodiazepines, alpha 2 agonists dexmedetomidine or clonidine could have attenuated this stress. In multiple studies, alpha 2 agonists have been shown to provide sedation equivalent to propofol and midazolam, with lower frequency and severity of delirium.[2,5] Clonidine and dexmedetomidine can also be effective pain management adjuncts, thereby reducing narcotic administration[7].

Narcotics such as opioids are associated with PD, therefore, limiting their use is helpful in reducing the incidence and severity of PD.[2,5] The opioid remifentanil is associated a lower risk for PD and may be beneficial in high-risk patients.[1,2,5] However, remifentanil offers little to no postoperative pain control, which is another important risk factor in PD.[1,2,5] Our patient received fentanyl and methadone for surgical pain. Methadone has a long duration of action, a multimodal mechanism of action, and has been shown to reduce the use of opioids in the perioperative period.[8] The drug is a strong mu receptor agonist, and it also acts as a N-methyl-D-asparate antagonist, thereby by inhibiting additional pain pathways and reducing narcotic consumption.[8] Non-narcotic adjuncts like the anticonvulsant gabapentin, the NMDA receptor antagonist ketamine, and the cyclooxygenase pathway inhibitor ketorolac may also be beneficial in limiting PD by controlling postoperative pain and reducing opioid use.[2,5,6,8] Many anesthetics that reduce the incidence and severity of PD may

be costly and unnecessary if age is the only risk factor. Therefore, it is important for the anesthesia practitioner to prescreen elderly patients for unrecognized cognitive dysfunction before creating their anesthetic plan.

The most predictive non-modifiable risk factor for PD is a patient history of dementia or other cognitive impairments.[3-5] Our patient did not have a known history of dementia or pre-existing cognitive dysfunction, and a specific cognitive impairment prescreen was not performed. Had she failed to mention her slow recovery, we would not have altered the anesthetic plan. Therefore, prescreening the elderly for unrecognized cognitive dysfunction and/or dementia would be a helpful tool in limiting PD. A prescreening tool like 'The Sweet 16' can help detect unrecognized conditions.[9] It is a brief assessment tool that takes approximately 2 minutes to perform and can detect subtle cognitive impairments without the use of paper, pencil or props.[9] 'The Sweet 16' instrument scores patients from 0 – 16 and includes items related to orientation, registration, 4 digit spans, and recall.[9] A score of 16 is the best and not related to cognitive dysfunction, while a score of 13 or less indicates the existence of a previously unrecognized cognitive dysfunction.[9] The test has been shown to be as, or more reliable than existing assays.[9] Pre-screening the patient for the most predictive non-modifiable risk factor can lead to modifications in the anesthetic planning and potentially reduce the occurrence of PD.

The anesthetic plan for this case was altered, but there were limits to attenuating anesthetic risk factors. This included the necessity for a deep level of sedation, and significant opioid use. During the postoperative period the patient was somnolent, but arousable and appropriate. She was "groggy" the next morning after taking a hydrocodone 7.5mg for pain, but she left the hospital for home after her anticipated overnight observation, and no further follow up was obtained. An anesthetic plan can be adjusted for elderly patients with un-modifiable PD risk factors in order to limit the likelihood and severity of PD. However, the practitioner must be able to recognize risk factors and have a working knowledge of how to limit anesthetic risks. This is important to patient outcomes and can be achieved by performing preoperative assessments, limiting the use of benzodiazepines and narcotics, replacing these agents safer sedatives and pain adjuncts, reducing the depth of anesthesia, and managing postoperative pain. This is a tall order, but vigilance and planning can shorten hospital stays, reduce costs, and limit the severity of PD in a the most rapidly growing age group in the United States.

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Mentor: Courtney Brown, CRNA, PhD

Hepatic Trauma and Massive Transfusion

Jennifer Hults, BSN
Goldfarb School of Nursing

Keywords: Massive transfusion, transesophageal echocardiogram, liver trauma, hemorrhage

Hemorrhagic shock and exsanguination account for more than 80% of mortality within the operating room following a traumatic event.[1] Proper preparation and planning for the use of large amounts of blood products is imperative for the survival of the patient. Although trauma patients cannot be enrolled in randomized controlled trials to evaluate massive transfusion protocols, data supports the use of a high ratio of plasma and platelets. Current studies have shown that ratios of 1:1:1 platelets to plasma to PRBCs will result in higher survival.[2,3] The following case study illustrates the use of the massive transfusion protocol following hepatic trauma.

Case Report

A 62-year-old male with no known medical history presented with shortness of breath. Upon clinical workup, he was found to have new onset atrial fibrillation and an ejection fraction of 30%. During his clinical course a new diagnosis of Hodgkin's Lymphoma with nonischemic cardiomyopathy was made. Over the next few days, he was admitted to the intensive care unit, intubated and placed on vasopressors. Further cardiovascular deterioration warranted an extracorpeal membranous oxygenator (ECMO). After fourteen days of therapy, ECMO was removed, and an intraaortic

balloon pump was inserted. That evening's chest radiograph indicated a small right chest pleural effusion that necessitated the placement of a chest tube, and chest tube insertion took place early the next morning. Throughout the day the patient became hemodynamically unstable with a distended abdomen. The trauma service was consulted and the patient was brought to the operating room for an emergent exploratory laparotomy.

Preoperative preparation included initiation of the massive transfusion protocol, set up of the level one rapid infuser, securement of appropriate pharmaceutical agents, and confirmation of adequate intravenous access. The patient arrived in the operating room intubated and on norepinephrine 0.4 mcg/kg/min, epinephrine 0.15 mcg/kg/min, and vasopressin 0.08 units/min. Peak airway pressures were high secondary to the hemoperitoneum. Initial boluses of fentanyl 250 mcg, midazolam 5 mg, and rocuronium 100 mg were given. The rapid infuser was immediately initiated and PRBCs, plasma, and platelets were administered in anticipation of the opening of the abdominal fascia and the release of the hemoperitoneum tamponade. Upon opening the fascia and the evacuation of the hemoperitoneum, blood pressure instability ensued. The retroperitoneum was packed and inspected by quadrant for the source of the bleeding. A 1cm laceration was noted on the hepatic parenchyma secondary to the chest tube insertion that took place earlier in the day.

Over the course of the repair, serial blood work was followed and treated. A total of nine ampules of sodium bicarbonate 50 mEq and calcium gluconate 12 grams were given. With the continuous use of the rapid infuser and the massive transfusion protocol, 13 units of packed red blood cells, 10 units of fresh frozen plasma, and 2 units of platelets were given over the course of the laparotomy and hepatic repair. A transesophageal echocardiogram (TEE) was utilized continuously to closely monitor heart function and fluid status. The final intraoperative TEE evaluation revealed severely decreased right ventricular function, global hypokinesis, an estimated left ventricular ejection fraction of 20% and moderate to severe mitral regurgitation. Upon completion of the repair, the patient was noted to have pink froth within the endotracheal tube suggesting significant pulmonary edema. The pulmonary edema was treated with suctioning and positive end expiratory pressure. Furosemide was not given due to hemodynamic instability, and further management took place within the intensive care unit.

After the hepatic repair, the abdomen was packed with laparotomy sponges, left open, and covered with an ioban dressing. The patient was returned to the cardiothoracic intensive care unit in critical, but stable condition.

Discussion

A massive transfusion protocol(MTP) is initiated within a hospital setting when an estimated requirement of at least 10 units of PRBCs will be given to a patient short term. It can be initiated for acute or imminent blood loss or in the setting of class IV hypovolemic shock. In this particular case study, the massive transfusion protocol was initiated before surgery in anticipation of rapid blood loss. In the absence of a predefined protocol for mass transfusion, significant delays may be encountered in receiving appropriate and sufficient amounts of blood products.[1] These delays may significantly increase mortality in the trauma patient.

When the massive transfusion protocol is initiated for a trauma patient, coagulopathy of trauma shock must be considered. The current approach to the management of this early coagulopathy involves the implementation of damage control resuscitation(DCR). DCR has three basic components: permissive hypotension, limiting crystalloids, and delivering plasma and platelets in ratios similar to that of whole blood.[4] Whole blood has a hematocrit of 38-50%, 150-400 thousand platelets, 100% coagulation activity, 1500mg fibrinogen, and is exactly what the patient is losing.[2] When solely administering PRBCs, the patient will not be receiving the coagulation factors that they are losing. Studies show that patients receiving a high plasma and platelet to red blood cell ratio had a significantly higher 24 hour and 30 day survival.[3,4] Ratios of 1:1:1 platelets to plasma to red cells must be initiated early in the resuscitation to optimize patient outcomes.[3] This approach has proven that postoperatively patients are less edematous and coagulopathic. Large volumes of crystalloid have been associated with an increased risk of acute respiratory distress and a higher mortality.[4]

Multiple studies have demonstrated that infusing a 1:1:1 ratio of fresh frozen plasma, red blood cells and platelets increases survival and has since become a common resuscitative strategy.[3,4] Few studies exist that evaluate the best hematocrit range to strive for when mass transfusion had been initiated, and an intraoperative goal can better direct patient care towards positive outcomes. A retrospective analysis was done on patients with intra-abdominal injuries requiring greater than 10 units PRBCs. The study revealed that a post-massive transfusion protocol (MTP) hematocrit of 29.1-39 percent led to an overall survival advantage.[5] These findings demonstrate the need for judicious blood product administration during a massive transfusion for increased potential positive outcomes.

Fluid overload, pulmonary edema, and acute respiratory distress syndrome have been associated with massive transfusion. Concurrent utilization of TEE with massive transfusion can lead to fluid optimization and potentially reduce morbidity and mortality. Intraoperative TEE was utilized with this case to assess cardiac function and fluid status. The probe was inserted prior to surgical incision and was utilized throughout the operative period. The use of real time images and the ability to monitor cardiac responsiveness to interventions solidify the usefulness of TEE within patients at high risk for hemodynamic instability. Studies have shown that objective TEE measurements can be useful for the management of high-risk patients in noncardiac surgery and can be used as a substitute for a pulmonary artery catheter.[6,7] Based on TEE findings, management changes can be made including administration of fluids, vasopressors, vasodilators, or beta blockers. The current case utilized TEE in conjunction with blood pressure and lab values to guide fluid and blood product administration. Based on the TEE findings of right ventricular dysfunction and global hypokinesis, the epinephrine infusion was increased to 0.2 mcg/kg/min for transfer back to the cardiothoracic intensive care unit.

Hemorrhage accounts for up to one-third of traumatic injury deaths within 24 hours of insult.[8] Early utilization of a mass transfusion protocol can aid in resuscitating a critically ill patient in a timely manner. From an anesthesia standpoint, proper preparation and knowledge of the massive transfusion protocol can significantly impact the mortality of trauma patients. In addition

to understanding individual institution protocols related to blood products, anesthesia providers must be aware of potential complications related to mass transfusion and appropriate monitoring techniques.

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Mentor: Bernadette Henrichs PhD, CRNA, CCRN

Anesthetic Implications of Kartagener's Syndrome

Amy Elizabeth O'Donnell, MSN
University of Pennsylvania

Keywords: Kartagener's syndrome, primary ciliary dyskinesia, Afzelius syndrome, dextrocardia-bronchiectasis-sinusitis, situs inversus.

Kartagener's Syndrome is an autosomal recessive genetic disorder, occurring in 1:16,000 births. It is characterized by ciliary dyskinesia, situs inversus and bronchiectasis.[1] Ciliary dyskinesia results in cilia that are motionless or absent, preventing the necessary clearance of mucus and cellular debris in the airways and spinal cord. Situs inversus is the reversal of all major visceral organs, including the heart, lungs, stomach and liver. Bronchiectasis is the irreversible destruction and dilation of the airway, leading to mucus buildup and infection. Acknowledging the complexities of Kartagener's syndrome can aid in

management of intraoperative and postoperative complications.

Case Report

A 6-month-old, 8.9 kg, 69 cm, male presented to the Emergency Department with a two day history of increasing irritability and poor oral intake. Upon physical examination, it was noted the patient had a bulging anterior fontanel and sun setting eyes. The diagnosis of hydrocephalus was made and the patient was scheduled for an emergency ventriculoperitoneal shunt. Oral intake of infant formula was noted within the previous hour. His past medical history included Kartagener's syndrome with situs inversus. His home medications included amoxicillin, hyper saline nebulizers and albuterol nebulizers.

The patient was brought to the operating room where a three lead electrocardiogram, pulse oximeter and blood pressure cuff were applied. He was preoxygenated with oxygen at 8 liters per minute via facemask. A modified rapid sequence induction (RSI) was initiated and cricoid pressure was held. Lidocaine 12mg, propofol 22 mg and rocuronium 9 mg were given intravenously (IV). Mask-bag ventilation with peak inspiratory pressures less than 20cm H_2O and cricoid pressure was continued. Direct laryngoscopy using a Miller 2 blade was performed and a grade I view of the airway was noted. A 4.0 mm uncuffed endotracheal tube (ETT) was inserted into the trachea without difficulty. Positive bilateral breath sounds and end-tidal carbon dioxide ($ETCO_2$) were noted. Immediately following intubation, an orogastric tube was placed into the stomach and gastric contents were removed via suction. Dexamethasone 4 mg and cefazolin 220 mg were administered IV. Pressure control ventilation was utilized throughout the procedure to maintain an ETCO2 of 28-33mmHg. Fentanyl 25mcg was administered prior to incision. Surgical anesthesia was maintained with desflurane 5-6%, oxygen 1 l/min and medical air 1 l/min.

Upon conclusion of the surgical procedure, the neuromuscular blockade was assessed with 4 out of 4 train of four twitches present. Neuromuscular blockade was antagonized using neostigmine 0.63 mg and glycopyrrolate 0.1mg. Prior to extubation, an albuterol inhaler of 10 puffs was given via the ETT and the patient was suctioned with return of minimal clear secretions. The patient was extubated awake and transported to post anesthesia care unit with oxygen 4 liters per minute via face mask.

Discussion

Kartagener's syndrome was first diagnosed by Manes Kartagener in 1933.[1,2] The diagnosis of Kartagener's syndrome is one of exclusion. Definitive diagnosis is made after a nasal or bronchial biopsy or examination of immotile sperm.[2] The clinical presentation of ciliary dyskinesia, situs inversus and bronchiectasis are hallmarks of the disease.
Cilia are found in the lining of the respiratory tract, fallopian tubes, brain and spinal cord. Synchronized vacillating movement of cilia is necessary for normal function of many organs. Communicating hydrocephalus is present due to cilia dysfunction within the brain preventing CSF reabsorption.[1] Inadequate efflux of CSF can lead to increased intracranial pressure. With situs inversus, a small percentage of patients also have various cardiac anomalies including atrial septal defect, right pulmonary artery hypoplasia, and transposition of great vessels.

Symptoms, including upper airway obstruction and increased work of breathing, are noted soon after birth in infants born with Kartagener's syndrome. Ciliary movement in the respiratory tract normally sweeps bacteria, fluid and cellular debris toward larger airways where they can be expelled via coughing. With impaired ciliary function, the accumulation of bacteria leads to respiratory infections, pneumonia and advancement of bronchiectasis. Children may develop chronic rhinitis and otitis media with possible associated hearing loss. Nasal inflammation and polyps with olfactory impairment are frequent findings.

Medical and surgical treatments are aimed at symptom improvement. Medical treatment consists of antibiotics to treat upper and lower respiratory infections. Bronchiectasis is treated with daily chest physiotherapy, inhaled bronchodilators, and mucolytics. [3] Bilateral myringotomy tubes may be inserted to reduce infections and prevent hearing loss as well as functional endoscopic sinus surgery to treat chronic rhinitis and minimize the risk of airway infection. [3] Considering hemophilus influenza and staphylococcus aureus commonly cause infections, immunizations should be encouraged.

Obtaining accurate diagnostic imaging and laboratory data including arterial blood gases and pulmonary function tests are important components of the preoperative assessment.[4] The preoperative period should focus on optimization of respiratory status and treatment of active infection by such means as chest physiotherapy, postural drainage, antibiotics, bronchodilators and incentive spirometry.[1]

Knowledge of the anatomical distortion associated with the syndrome is paramount. Precordial assessment will produce absent heart sounds on the left and electrocardiogram leads and external defibrillation pads should be placed in reverse fashion. Central line placement will be less difficult when approached from the left side.[4] In the parturient, right uterine displacement is essential.[5] Endotracheal intubation does not pose any unusual challenge, however, careful placement of the ETT is crucial as the main stem bronchus is reversed. As such, double lumen endotracheal tubes should be selected with pulmonary inversion in mind.[5] Nasal intubation or nasal airways are contraindicated in the presence of nasal polyps and chronic sinusitis.[6]
Regional or local anesthesia is favored over general anesthesia as spontaneous ventilation is maintained and airway manipulation avoided. [1,4,5,6] If regional anesthesia is contraindicated, a general anesthetic should be performed.

Volatile anesthetics produce bronchodilation and are rapidly eliminated by the lungs. Inspired gases should be humidified to keep the airway moist and thin respiratory secretions. Large tidal volume of 10-15 ml/kg combined with a slow respiratory rate of 6-10 breaths per minute with an extended expiratory phase is ideal. This combination minimizes the turbulent airflow through the airways, maintains optimal ventilation to perfusion match, allows sufficient time for venous return to the heart and time for complete exhalation.[5]

Frequent suctioning via the ETT will be essential as thick secretions may be present. Administration of a bronchodilator prior to extubation is advisable as was done is this case. Dosing of short acting opioids will decrease postoperative respiratory depression.[6] Extubation at the end of the procedure should be expected, but is

dependent upon preoperative respiratory status and extent of the disease.

Recognizing the anatomical alterations associated with Kartagener's syndrome is the first step to successful management. Proactive albuterol treatments, suctioning and ventilatory vigilance were substantial factors in this patient's favorable outcome. The potential to optimize the patient's pre-operative respiratory status was impaired by the emergent scheduling of the procedure. However, the opportunity to ascertain further knowledge of the patient's respiratory status would have been advantageous. While the idiosyncrasies of Kartagener's syndrome are complex, the anesthetic management can be tailored to permit a successful conclusion.

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Mentor: Kelly L. Wiltse Nicely, CRNA, PhD

Anesthetic considerations a for patient with Down syndrome

Michael Huynh, MSN
University of Southern California

Keywords: Anesthetic considerations for Down syndrome, obesity, obstructive sleep apnea (OSA), airway anomalies, cervical subluxation, hypothyroidism, laryngeal mask airway (LMA) for obese patients

Also known as trisomy 21, Down syndrome is the most common chromosomal abnormality occurring in 1:800 births.[1] There are multiple systemic effects of this disorder and the implications involved when providing anesthesia for cataract extractions are critical to assess. Of highest priority are the adverse affects on the respiratory and cardiac systems, but the gastrointestinal, musculoskeletal and endocrine systems must also be evaluated judiciously prior to the development of an anesthetic plan. A detailed summary of these anesthetic implications will be discussed.

Case Study

A 36-year-old female with senile nuclear sclerosis was scheduled for a left eye cataract extraction and prosthetic lens insertion. The patient weighed 86 kg, was 147 cm tall, and calculated body mass index (BMI) was 39.7 kg/m^2. The patient's medical history included hypothyroidism, obesity, and Down syndrome. She was able to walk up multiple flights of stairs without chest pain or shortness of breath and denied symptoms of gastroesophageal reflux disease (GERD). The laboratory values (basic metabolic panel, thyroid function studies, CBC and coagulation time) were within normal limits. The electrocardiogram showed normal sinus rhythm and lungs were clear bilaterally upon auscultation. Although she was able to prognath her lower jaw and extend her neck without difficulty, an airway assessment revealed she had micrognathia and macroglossia. Her thyromental distance was < 3 fingerbreadths and the sub-mental space was <2.5 fingerbreadths. Midazolam 2 mg intravenously (IV) was administered in the preoperative area.

Following transport to the operating room, pulse oximetry, blood pressure cuff, and electrocardiogram leads were placed, and the patient was preoxygenated with 100% oxygen for 5 minutes. General anesthesia was induced with propofol 150mg and lidocaine 100mg. A laryngeal mask airway (LMA) Unique (Teleflex Inc.,Wayne PA) was inserted deep into the hypopharynx until the tip of the cuff was positioned at the upper esophageal sphincter. The patient's head was in a neutral position during placement but air leakage was noted. The head position was changed with more extension and the LMA advanced another centimeter to correct the leakage. Spontaneous ventilation was established and the patient's respiratory rate (RR) of 35-40/min and tidal volume (TV) of 220-250 ml were noted. To improve patient ventilation (high RR and low TV), the patient was placed in slight reverse Trendelenburg to decrease the intrathoracic pressure and a propofol infusion was started at 100mcg/kg/min. The patient was placed on pressure support ventilation (PSV) at 6 cm H_2O to improve the TV to 350-400 ml, and the propofol infusion was increased to 130 mcg/kg/min. The RR subsequently decreased to 20-28/min.

General anesthesia was maintained with sevoflurane 1.9% expiratory concentration in oxygen 2 L/min and the propofol infusion. Nadir systolic blood pressure was 64 mmHg and was effectively treated with phenylephrine 80 mcg IV and ephedrine 10 mg IV. The systolic blood pressure increased to 98 mm Hg. Acetaminophen 1g IV was administered at incision and ondansetron 4 mg IV was administered 30 min before the end of the case. The procedure lasted one hour and the sevoflurane was discontinued 10 minutes prior to the end of the procedure. At the end of the case, the patient was spontaneously ventilating (TV 450 ml; RR 18/min) but did not respond to verbal commands or rigorous stimulation. Full patient awakening was noted 10 minutes after the end tidal sevoflurane concentration was 0%. The LMA was removed without complication as the patient's eyes opened. Mild snoring was noted and a nasal trumpet was placed to improve ventilation. The patient was taken to the post anesthesia recovery unit where vital signs (106/70 mm Hg, 105/min, 18/min, SpO_2 99% on 6 L/min facemask) and 0/10 pain score were noted. The nasal trumpet was removed at this time. One hour later, the patient was discharged home with family without further complications.

Discussion

The etiology of Down syndrome may include advanced maternal age, exposure to anesthesia, electromagnetic fields, pesticides, and the consumption of alcohol/caffeine during pregnancy.[1] Down syndrome is characterized by an extra copy of chromosome 21, where each cell in the body has 3 copies of chromosome 21. This extra copy changes the development of the fetus' body and brain, resulting in central nervous system and physical disorders.[1] These patients may present with intellectual disability, hypotonia, subglottic stenosis, upper airway obstruction, micrognathia, macroglossia, obesity, OSA, congenital heart disease, GERD, atlantoaxial instability, thyroid disorders, and low catecholamine levels amongst other complications.[1] Not all patients with Down syndrome present with all of these disorders, but the potential implications involving the neurological, cardiac and respiratory systems require consideration.

Due to the stress of surgery and the developmental status of these patients, preoperative intravenous anxiolytics are provided if deemed appropriate. This patient was premedicated with midazolam for anxiety. In a retrospective study conducted by Yoshikawa et al., male patients <21-years-old with Down syndrome given midazolam (>0.032 mg/kg) for dental procedures exhibited prolonged awakening times likely due to midazolam administration.[2] Although this female patient was >21-years-old and was administered midazolam 0.023 mg/kg, this dose of midazolam possibly contributed to the delayed emergence and titration of midazolam may have prevented the delayed emergence.

Cataract surgery requires patient cooperation and immobility during the surgical procedure. A general anesthetic with LMA ventilation was selected to obviate intraoperative problems that might arise from the patient's developmental delay. Cheon et al suggested the use of LMAs for surgeries (duration 2 hours or less) under general anesthesia, but complications such as insertion and ventilation can increase by 2.5 fold in obese patients (BMI >30) due to larger tongue, smaller mouth opening, and excess soft tissue in the airway.[3] Immediately post induction in this case, the LMA insertion necessitated airway manipulation techniques such as jaw thrust and the tongue being manually swept aside. Although the patient had no known subluxation of the cervical spine or any neurological symptoms of nerve impingement, precautions such as careful extension and flexion of the head and neck were taken during LMA placement and positioning. Tokunaga et al demonstrated that atlantoaxial subluxation is aggravated with flexion and reduced with extension and symptoms may go unnoticed under general anesthesia.[4]

Following induction and securement of the airway, the patient exhibited shallow, rapid breathing. Slight reverse Trendelenburg was initiated to improve the patient's TV although Benedik et al reported this to be ineffective for overweight and mild to moderately obese patients as compared to leaner body habitus.[5] Ventilatory support in the PSV mode was initiated to improve TV. In a review conducted by Chmielewski et al, PPV with LMA was not only safe and effective, but also demonstrated improved ventilation and oxygenation compared to spontaneous ventilation alone.[6] Concurrently, the blood pressure decreased and this was treated with incremental doses of phenylephrine (80 mcg) and ephedrine

(10 mg) IV. O'Driscoll et al. evaluated children with Down syndrome and demonstrated hemodynamic instability may be related to lowered catecholamine levels associated with Down syndrome as evidenced by low levels of urinary noradrenaline, adrenaline, and dopamine in this study group.[7] This patient was administered propofol 150 mg IV for induction. This may have been responsible for the hypotension and incremental doses of propofol and additional fluid volume administered could have maintained the systolic blood pressure above 90 mm Hg.

This patient was at risk for obstructive sleep apnea (OSA) secondary to macroglossia, micrognathia, and obesity, although the patient's family denied any symptoms of OSA to include snoring, apnea, and wakefulness. Parents of Down syndrome children often under report OSA and miss the signs and symptoms because they assume it is characteristic of the genetic disorder.[8] Due to the minimal level of postoperative pain in cataract surgery, acetaminophen was sufficient for analgesia and eliminated the need for narcotics, which exaggerate the hypoventilation seen in OSA or, as in this patient, with noted macroglossia, micrognathia, and obesity.[1]

Patients with Down syndrome present with multisystem involvement of this genetic disease to include delayed developmental status, airway anomalies, congenital heart defects, endocrine disorders, and musculoskeletal deformities. The specific concerns noted for this patient involved attention to the airway throughout the intraoperative course and the prolonged emergence. Use of acetaminophen obviated postoperative hypoventilation. Hypotension may have been prevented with propofol titration and fluid volume. The use of PSV to increase the TV could have also contributed to the hypotension by causing impedance to the venous return. There are many complications related to obesity including cardiovascular, respiratory disease and OSA. Patient's with Down syndrome may suffer from OSA without knowing and view symptoms of this disorder, such as snoring, as benign. When presented with a patient with Down syndrome, the anesthesia practitioner is to tailor the anesthetic plan to the needs of this unique population.

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Mentor: Michele E. Gold, CRNA, PhD

T-wave Inversion after Posterior Spine Surgery

Chris K. Beck, MSN
Wake Forest Baptist Health, University of North Carolina at Greensboro

Keywords: anesthesia, perioperative complications, ECG changes, T-wave inversion

Cardiovascular diseases are the medical illnesses most frequently encountered in anesthetic practice and represent a major cause of perioperative morbidity and mortality.[1] Moreover, the prevalence of cardiovascular disease increases with advancing age, which is significant considering the number of patients over 65 is expected to increase by 25-35% over the next 20 years, and cardiovascular complications account for 25-50% of deaths following non-cardiac surgery.[1,2] Consequently, as the subsequent case demonstrates, there is growing value in efficient and valid ECG interpretation and using these findings to direct anesthetic care in the perioperative period.[3]

Case Report

A 76-year-old, 181 cm, 105 kg Caucasian male patient presented for a lumbar laminectomy of L1-L5, L2-L3 discectomy and L4-L5 fusion with instrumentation and bone grafting. His past medical history included chronic back pain, chronic renal insufficiency, degenerative disc disease, diabetes mellitus type II, hypertension, hyperlipidemia, hypothyroidism, lumbosacral spondylosis, and stroke (no residual affects per patient report and per assessment). His preoperative blood pressure was 155/73, heart rate 59/min, and SpO_2 was 98% on room air. His preoperative 12-lead electrocardiogram showed sinus bradycardia. Preoperative labs and testing included a complete blood count (CBC), a basic metabolic panel (BMP), HgA1C and electrocardiogram (ECG) with all values within normal limits.

An 18-gauge peripheral intravenous catheter was placed in the holding room with a second placed intraoperatively. In the operating room, standard monitors were applied and pre-oxygenation initiated via facemask. An intravenous induction was performed with lidocaine, fentanyl, propofol, and rocuronium. Direct laryngoscopy was performed using a Macintosh 3 blade with a grade II view. The trachea was intubated with a cuffed 7.5 mm oral endotracheal tube. End tidal CO_2 ($EtCO_2$) was confirmed and bilateral breath sounds (BBS) were auscultated. The patient was positioned onto the Jackson table in a prone position with all extremities and head secured and pressure points padded. General anesthesia was maintained with 0.5% isoflurane, one liter of nitrous oxide with one liter of oxygen, and a remifentanil infusion. Neuromonitoring was utilized, and no deficits were noted throughout the case. The patient maintained normal sinus rhythm

on the ECG during the case without ectopy, and the pulse oximeter measure 100% throughout the case.

Upon completion of the case, the patient was repositioned onto the transport stretcher in the supine position. After resuming spontaneous ventilation with adequate tidal volumes and respiratory rate and following commands, the patient was extubated and placed on 100% oxygen by facemask. The patient's heart rate was noted to be elevated from intraop values of 60s bpm to 80s bpm after extubation and pulse oximeter measured 100%. Overall, the patient required the following fluid replacement: 4200 mL of normal saline, 500 mL hetastarch, 1000 mL 5% albumin, and 246 mL intraoperative cell salvage system blood. Total fluid replacement was 5946 mL with an estimated blood loss of 450 mL and total urine output of 739 mL.

The patient was transferred to the recovery room where he was noted to have tachycardia with inverted t-waves in leads II and V5; however, blood pressure, SpO_2, and temperature were stable. Patient denied chest discomfort or dyspnea. A 12-lead ECG was ordered, which demonstrated t-wave inversions in leads V3-V6, I and II, which were all new compared to baseline ECG. Aspirin, metoprolol, cardiac enzymes, CBC, BMP and arterial blood gas were ordered, and a cardiology consultation was requested.

Discussion

This case highlights ECG changes and the significance of t-wave inversions after non-cardiac surgery. A review of the literature concerning t-wave inversions reveals several contributing factors to this phenomenon. The t-wave of an ECG is representative of ventricular repolarization, and it is typically a positive deflection in leads I, II, aVL, aVF, and V4 through V6. According to the American Journal of Critical Care, the nonspecific finding of t-wave inversion warrants further investigation via diagnostic testing in order to distinguish between pathological and benign conditions. Considering 13 million Americans have known coronary artery disease and even more exhibit risk factors for heart disease, it's not surprising that perioperative recommendations for surgical risk screening suggest preoperative ECG for men 45 years or older, women 55 years or older, and patients with known CAD.[3,4] A baseline ECG is invaluable for comparison in the event of perioperative complications that suggest myocardial ischemia. T-wave inversions that represent a change from the baseline may indicate the presence of an underlying pathological condition or that a cardiac event has occurred.[5]

It is important to distinguish between primary and secondary T-wave inversions. Primary t-wave inversions can be caused by: myocardial ischemia or infarction, pericarditis, electrolyte abnormalities, anemia, pH changes, drug effects, pulmonary embolism, left or right ventricular overload, cerebrovascular injury, post-tachycardia t-wave pattern, hypertrophic cardiomyopathy, dilated and restrictive cardiomyopathies, arrhythmogenic right ventricular cardiomyopathy (ARVC). Primary inversions can be idiopathic or normal variant in origin. Secondary t-wave inversions include left bundle branch block, right bundle branch block, Wolff-Parkinson-White patterns, and ventricular ectopic beats.[4,6] Aro et al. notes that t-wave inversion in right precordial leads V1 to V3 is a relatively common finding on a 12-lead ECG of children and adolescents. However, this finding is infrequently found in healthy adults, and it can be the first presentation of

ARVC. Aro et al. also found right precordial t-wave inversions did not predict increased mortality, yet inverted t-waves in leads other than V1 to V3 were associated with an increased risk of cardiac and arrhythmic death. Aro et al. concluded that incidentally observed t-wave change in an asymptomatic subject poses a clinical problem, because t-wave inversions can be the expression of an underlying cardiac disease capable of causing sudden cardiac death.[6]

Choi and Park that found t-wave inversion in precordial leads are most frequently observed in patients with acute pulmonary embolism (APE), another adverse event of the perioperative period. Their study found that sinus tachycardia and t-wave inversion were the most frequently observed ECG changes in patients with APE. Also, t-wave inversion was significantly more frequent in patients with right ventricular dysfunction. The pathophysiological ECG changes of tachycardia and t-wave inversions in APE were thought to be due to myocardial ischemia, chemical mediators such as catecholamines and histamine, and altered pathways of electrical repolarization.[5]

Undoubtedly, most providers would desire a preoperative ECG and/or other noninvasive cardiac function tests as a baseline for evaluation. It is prudent to follow the American College of Cardiology/American Heart Association Task Force Report recommendations for preoperative noninvasive cardiac testing. These guidelines state noninvasive cardiac testing should be conducted prior to surgery if any of the following two factors are present: 1) intermediate clinical predictors are present (Canadian class I or II angina, prior MI based on history or pathological Q waves, compensated or prior heart failure, or diabetes); 2) poor functional capacity (less than 4 METs); and 3) high surgical risk procedure (emergency major operations, aortic repair or peripheral vascular disease, prolonged surgical procedures with large fluid shifts or blood loss).[6] Using these guidelines will aid in identifying patients who will benefit from preoperative noninvasive cardiac testing.

The patient undergoing posterior lumbar laminectomy depicted in the case report is at risk for primary causes of t-wave inversions described in the literature. APE is listed as a complication of any orthopedic surgery, especially those involving the spine.[1] The patient's past medical history demonstrates several risk factors for coronary artery disease, most importantly advanced age, diabetes mellitus, hypertension, and hyperlipidemia. These findings predispose the patient to an adverse perioperative cardiac event.[2] Although exposure to general anesthesia and surgical manipulation can precipitate many of the factors contributing to primary t-wave inversions, it is highly suspected in this particular case that the patient has suffered from mild myocardial ischemia secondary to increased oxygen demand due to stress of the perioperative period and postoperative tachycardia.

In the case reported, the patient was noted to have t-wave inversions in leads II and V5 on the two-lead ECG monitor. In addition, a 12-lead ECG demonstrated T wave inversions in leads V3-V6, I and II. When compared to baseline, the cardiologist noted the ECG suggested ST and T wave abnormality suspicious for inferior and anterolateral ischemia. An echocardiogram revealed a left ventricle of normal size with mild concentric hypertrophy and normal function, an ejection fraction of 55-60%, impaired left ventricular filling pattern, and mild tricuspid regurgitation. Supporting the suspicion of ischemia, serial troponins were elevated seventeen hours after surgery. A

follow up cardiology visit three months postoperatively revealed no permanent adverse cardiac changes, as evidenced by echocardiogram.

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Mentor: Courtney Brown, CRNA, PhD

Anesthetic Management of Total Thyroidectomy with Sternotomy

Kelsey M. Merrick, BSN
Texas Christian University

Keywords: thyroidectomy, sternotomy, anesthesia, mediastinal goiter, substernal goiter, thyroid, recurrent laryngeal nerve injury, airway management

Thyroid surgery is traditionally performed under general anesthesia via a low transverse collar incision with intraoperative recurrent laryngeal nerve (RLN) monitoring.[1] Neuromuscular blocking agents (NMBAs) are contraindicated with intraoperative nerve monitoring, as they interfere with electromyography (EMG) readings. Therefore, surgical removal of a mediastinal goiter via a median sternotomy approach presents unique considerations to the anesthesia practitioner. These cases require constant communication and precise planning between the anesthesia practitioner(s) and the surgeon(s) to ensure a positive outcome.

Case Report

A 93-year-old, 74 kg, 170 cm Caucasian male presented to the emergency department (ED) with dysphagia, dysphonia, and tracheal deviation. A chest x-ray revealed a large mediastinal mass with tracheal deviation. The patient's medical history was significant for: advanced age, hyperthyroidism, hypertension, hyperlipidemia, gastroesophageal reflux disease, benign prostatic hypertrophy, and chronic obstructive pulmonary disease. The patient had been under the care of an endocrinologist for his hyperthyroidism. Until his recent ED admission, the patient had opted for medical management only.

His surgical history included an inguinal hernia repair and a hemorrhoidectomy. Current medication regimen included amlodipine, finasteride, losartan, pantoprazole, tamsulosin, methimazole, and labetalol. The patient reported having an allergy to atorvastatin.

Laboratory studies were significant for a hemoglobin of 10.9 g/dL and a hematocrit (Hct) of 33.6%. A twelve lead EKG indicated sinus rhythm with a right bundle branch block. Physical exam was significant for tracheal deviation; however, there was no palpable or visual neck mass. Assessment of the patient's airway revealed a Mallampati score of two, normal cervical spine range of motion, mouth opening, and thyromental distance. The patient was given an ASA Physical Status of IV due to his acute presentation and significant past medical history. Preoperative vital signs were as follows: blood pressure 155/78 mm Hg, heart rate 79/min, respirations 18/min, and SpO_2 100% on room air.

Preoperatively, a right radial 20 gauge arterial catheter was inserted for invasive blood pressure monitoring. The patient's airway was anesthetized with 3 mL of 4% nebulized lidocaine and 3 mL of 5% topical lidocaine jelly in preparation for an awake intubation with a video laryngoscope. The patient was then transferred to the operating room (OR) where he positioned himself on the OR table in the supine position. The standard ASA monitors were applied, and oxygen was administered at 10 L/min via facemask for five minutes. Video laryngoscopy was used to perform the awake intubation, and a 7.0 mm nerve integrity monitoring (NIM) endotracheal tube (ETT) (Medtronic Xomed, Jacksonville, FL) was successfully placed. Endotracheal intubation was confirmed with positive end-tidal CO_2, equal bilateral breath sounds, equal chest rise, and condensation in the ETT. After the ETT was secured at 21 cm at the lip, general anesthesia was induced with etomidate 10 mg, propofol 40 mg, fentanyl 100 mcg, and midazolam 1 mg. Sevoflurane 1.5% expired concentration with oxygen at 2 L/min was used to maintain general anesthesia, and a mechanical ventilator controlled respirations. A left femoral central venous catheter was placed for large bore venous access. Ephedrine 10 mg and phenylephrine 100 mcg were used to treat episodes of hypotension during maintenance of general anesthesia.

It was determined intraoperatively that the patient's goiter was too large to be safely resected via a traditional incision. A cardiothoracic surgeon had been consulted and was present to perform a full sternotomy to facilitate removal of the patient's goiter. Two units of packed red blood cells were infused per the surgeon's request to maintain a Hct level greater than 35% and replace the estimated blood loss of 600 mL. After completion of the surgery, the patient was transferred to the intensive care unit where he remained intubated overnight. The patient was extubated the following day and no anesthetic complications were noted. The patient was discharged to a cardiac rehab facility after a seven day admission to the hospital.

Discussion

First described in the 18th century,[2] mediastinal goiters have a reported occurrence of 2.6% to 30.4%, with up to 10% requiring a sternotomy for surgical resection.[3] If left untreated, goiters can continue to grow and may eventually pass through the thoracic inlet and expand in the mediastinum.[2] Mediastinal, or substernal, goiters pose a great risk to the patient

because of the risk of impingement on vital nearby structures.[2] Because of their tendency for slow growth, patients may remain asymptomatic for many years.[3] Common presenting symptoms include cervical mass, compressive symptoms, dyspnea, orthopnea, cough, dysphagia, chronic adrenergic symptoms, dysphonia, superior vena cava syndrome, and impingement on nearby structures.[2,3] Preoperative computed tomography (CT) scan is the gold standard for diagnosis of mediastinal goiters[4] and is highly recommended for any patient who has a suspected mediastinal goiter. It is generally agreed that substernal goiters require surgical resection for definitive treatment.[3] Predictive risk factors for sternotomy include recurrent goiter, malignancy, large thyroid gland volume, and goiter location in the mediastinum.[2-4] In these cases, the anesthesia practitioner must communicate with the surgeon to develop an appropriate and safe plan of care that facilitates resection of the mass and minimizes risks to the patient.

In patients with large thyroid masses, a thorough preoperative airway assessment is crucial.[1] The anesthesia practitioner must assess for symptoms of airway compromise, including dyspnea, orthopnea, tracheal deviation, and tracheomalacia.[1] Patients with a thyroid goiter, perceived difficult airway, or altered airway anatomy may require awake intubation with topical anesthesia to secure the airway.[1] Administration of sedatives and narcotics should be avoided in the preoperative period because of the risk of respiratory depression, and they should not be administered until the patient's airway is secured.[5] Airway management and the risk of airway compromise also presents a major challenge to the anesthetist during emergence from anesthesia, as underlying tracheomalacia may be revealed following thyroid resection causing airway collapse following extubation.[5] The anesthetist should have emergency airway equipment available should airway collapse occur after extubation.[5]

Other important intraoperative considerations include the use of invasive hemodynamic monitoring, prevention of sympathetic nervous system (SNS) stimulation, and selection of adjunct medications. The need for invasive monitoring is determined on an individual basis.[5] Continuous monitoring of the patient's blood pressure via an arterial catheter is suggested, especially if the patient is not in a euthyroid state.[1] The hyperthyroid patient is hypermetabolic and may experience tachycardia, dysrhythmias, hypertension, and anxiety.[1] In addition, hyperthyroidism is associated with an increased risk for perioperative hemodynamic complications.[1] Therefore, it is extremely important to make sure the patient is adequately anesthetized to prevent an exaggerated SNS response to laryngoscopy and surgical stimulation.[1,5] It is also prudent to avoid drugs that stimulate the SNS such as ketamine, atropine, ephedrine, epinephrine.[5] However, hypotension that is refractory to phenylephrine or hypotension in the presence of bradycardia should be treated with ephedrine or epinephrine. General endotracheal anesthesia is the anesthetic technique of choice for thyroidectomy, although there is no evidence to support one preferential anesthetic technique or agent.[5]

A major complication of thyroidectomy is unilateral or bilateral RLN damage, which can result in temporary or permanent hoarseness and airway obstruction.[1,5] Research has shown that thyroidectomy with sternotomy has an increased risk of complications, especially RLN damage.[3] To

minimize this risk, many surgeons monitor RLN activity with a NIM tube (Medtronic Xomed, Jacksonville, FL).[1,2] Electrodes in the NIM tube are connected to an EMG monitoring device, and RLN activity is recorded with nerve stimulation by the surgeon.[1] The anesthesia practitioner must remember that NMBAs inhibit EMG readings, and are, therefore, not routinely used during thyroidectomies.[1] Instead, general anesthesia is maintained with volatile anesthetics.[1] This presents a unique challenge to the anesthetist, as paralytics are routinely used in surgeries requiring a sternotomy. It is imperative that the patient is adequately anesthetized prior to the sternal split to prevent a hyperdynamic response and movement.[1] In this particular case, the anesthesia care team consulted with the surgeons prior to performing the sternotomy and a decision was made to proceed without the use of NMBAs. Consequently, the end-tidal percentage of sevoflurane was increased 2.4% to deepen the patient's anesthetic depth prior to sternotomy.

This case highlights the importance of a proper and thorough preoperative assessment and the development of an individually tailored anesthetic plan. It also demonstrates the importance of diligent communication between the surgeon and the anesthesia practitioner. Despite the preoperative concerns regarding airway management, intraoperative hemodynamic stability, and postoperative complications, this case went very smoothly. The patient's airway was secured without difficulty. The patient's hemodynamic status remained stable throughout the critical aspects of the case. Finally, and perhaps most importantly, this elderly patient experienced no postoperative or anesthetic complications and was discharged from the hospital without incident.

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Mentor: Ross Castille, CRNA, DNP

The Challenges of Anesthesia and Trisomy 13

Edwin George, MSN
Louisiana State University Health Sciences Center

Keywords: difficult airway, atrial septal defect (ASD), trisomy 13

Trisomy 13 (Patau's Syndrome) was first described by Patau and colleagues in 1960.[1-3] It is a rare autosomal disorder with an incidence that ranges from 1 in 5,000 to 12,000 live births.[1-3] A variety of elements may compose the syndrome including cleft lip and palate, holoprosencephaly, polydactyly, micropthalmia, microcephaly, rockerbottom feet, severe mental retardation, and genitourinary abnormalities.[1,4] Cardiovascular defects may include patent ductus arteriosus, septal defects, valve abnormalities, and dextrocardia.[1,4] Medical complications can include apnea, seizures, developmental delay, failure to thrive, gastroesophageal reflux, and renal insufficiency.[4] Although life expectancy has improved, overall prognosis is poor, with only 6-12% of children living beyond a year.[5]

Case report

A 3-year-old, 13.6 kg, 89 cm female presented for dental restoration, with a diagnosis of Mosaic Trisomy 13 with Robertsonian translocation between chromosomes 13 and 14 and duplication of 22q11.21. Her physical appearance was normocephalic with dysmorphic features including low set ears, a broad, wide set nose, and micrognathia. Airway evaluation revealed a Mallampati score of IV. Surgical history included eye muscle repair and bilateral polydactyly repair. Medical history included unrepaired moderate to large secundum atrial septal defect (ASD), vesicouretal reflux, hypothyroidism (resolved), seizure disorder, developmental delay, spontaneously closed muscular ventricular septal defect, pulmonary hypertension (resolved), and hydronephrosis. Hemangiomas were noted on her wrists bilaterally. Normal sinus rhythm was present and echocardiogram revealed an atrial septal defect of 5 to 6mm with left to right flow, no atrioventricular valve insufficiency, no right heart enlargement, good biventricular contractility, and no pericardial effusion. Cardiology deemed that no subacute bacterial endocarditis prophylaxis was warranted. No allergies were reported. Daily medication included levetiracetam 500 mg twice daily.

The patient was premedicated with 5 mg oral midazolam and taken to the operating suite. A Glidescope (Verathon, Inc., Bothell, WA) was available and noninvasive monitors were applied. Inhalation induction consisted of N_2O 7 L/min, O_2 3 L/min, and sevoflurane 8% by face mask. The N_2O was discontinued after intravenous (IV) placement and O_2 was increased to 10 L/min. Oxymetazoline HCl was given intranasally, followed by fentanyl 10 mcg and rocuronium 7 mg intravenously. Direct laryngoscopy was performed with a Wis-Hipple 1.5 blade revealing a grade 2 Cochrane Lehane view. A 5.0 uncuffed nasal RAE with a lubricated 16 french red rubber catheter covering the tip was introduced to the right nare, and passed into the oropharynx without difficulty. The red rubber catheter was visualized and retrieved with McGill forceps and removed from the end of the nasal RAE. The vocal cords were then re-exposed by direct laryngoscopy and

the nasal RAE was placed in the airway with the assistance of the McGill forceps. Bilateral breath sounds were auscultated and respirations were controlled by mechanical ventilation using a volume control mode. Desflurane was the volatile agent selected. Four extractions were performed. Neuromuscular blockade was antagonized with neostigmine and glycopyrrolate and the nasotracheal tube was removed after spontaneous respirations and eye opening. She was monitored for apnea postoperatively and discharged home the next day.

Discussion

Trisomy 13 presents with an array of challenges. The most formidable aspect is the presence of cardiac anomalies in concert with a potentially difficult airway.[2] Cardiac anomalies are present in 80% of patients, and are frequently multiple in nature (dextrocardia, atrial and ventricular septal defects, patent ductus arteriosus).[2,6] Renal abnormalities are present 60% of the time which demands careful monitoring of fluid administration and blood loss.[2] Other potential complexities include malrotation of the bowel, umbilical hernia, Meckel's diverticulum, omphalocoele, pancreatic dysplasia, and elevated levels of fetal hemoglobin persisting into late childhood.[2] Should the child survive into early childhood they may develop thoracic kyphoscoliosis or pectus carinatum which may result in decreased lung volumes.[2] The incidence of apneic episodes can be as high as 50% which may be worsened by opioid administration.[2] It is recommended that mechanical ventilation be available in the post-operative period.[2]

The diagnosis of trisomy 13 can be challenging to a family. Often the diagnosis of trisomy 13 alone is used as a justification for not providing many interventions.[5] Parents are often presented with dire predictions about the brevity quality of their child's life.[5] While the truncation of length of life is frequently accurate, the lack of quality is not necessarily assessed from the family's perspective.[5] The subjective nature of quality of life must be taken into consideration when speaking with families.

This patient's potentially difficult airway demanded thorough preoperative assessment and preparation. A Glidescope was available for a potentially difficult airway. Covering the end of the nasal RAE with the lubricated red rubber catheter serves to prevent intranasal trauma with the introduction of the nasal RAE. Trauma could result in a bloody field obscuring visualization and potentially resulting in laryngospasm. Elective fiberoptic intubation was another option. This could be accomplished safely with a ketamine induction to maintain spontaneous respiration, intranasal oxymetazoline to prevent epistaxis, nasal airway, laryngotracheal topical anesthetic for supraglottic and glottic anesthesia, and a pediatric fiberoptic bronchoscope for visualization of the airway.[1] Desflurane was chosen due to its smaller blood/gas partition coefficient.
Seizure disorders require a knowledge of the child's medication and schedule as well as potential interactions with anesthetic drugs.[1] Here the patient's seizure disorder was reportedly well-controlled by levetiracetam, a drug known to have no influence on the induction or inhibition of drug metabolism.[7] Seizure threshold can be lowered by the stress of surgery and it is necessary to continue the antiepileptic therapy up until surgery.[1] Blood levels may need to be monitored preoperatively, and intra-operative blood loss may lead to considerable changes in therapeutic effect.[1] Drugs known to lower seizure threshold

should be avoided.[1] Drugs known to induce benign myoclonic activity should not be confused with epileptiform behaviors.[1]

A secundum atrial septal defect (ASD) is a result of a deficiency in the septum secundum found near the fossa ovalis, and is the most common ASD accounting for 85% of the four types.[1,8] This results in a left to right shunt allowing some oxygenated blood back to the pulmonary circulation preventing initial cyanosis.[8] Complications develop as increased blood flow from the left to right results in increased pulmonary vascular resistance leading to pulmonary arterial hypertension which reverses the direction of blood flow back through the defect (Eisenmenger syndrome).[8] This cyanotic state can lead to arrhythmia, valve regurgitation, and heart failure.[8] Unresolved ASD may present as the child grows older with shortness of breath, fatigue, and lethargy, with more severe cases presenting with atrial dysrhythmias, and congestive heart failure as early as one year.[8]

Additional challenges include hemangiomas and vesicouretal reflux. Hemangiomas noted on the head or face, especially near the eyes, should raise suspicion of airway hemangioma and be further evaluated.[1] Vesicouretal reflux allows urine to pass up the ureter in retrograde fashion potentially leading to pyelonephritis and impaired renal function which can affect drug elimination.[1]

There is a paucity of information regarding anesthesia and trisomy 13 in a review of the literature. As children with the disorder rarely live beyond a year, and the incidence is so uncommon, experiential data is moderated by infrequency. As trisomy 13 may exhibit so many variations and degrees of severity, it is incumbent upon the anesthesia provider to be as knowledgeable as possible regarding the specific pathologies and complications with which the elective patient may present. A variety of anesthetic and social considerations must be taken into account to insure a safe and comprehensive plan of care.

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Mentor: David M. Kalil, CRNA, DNAP

Suspected Pseudocholinesterase Deficiency

Jenna Kelly, MS
Louisiana State University Health Sciences Center

Keywords: pseudocholinesterase deficiency, prolonged ventilation, succinylcholine, prolonged neuromuscular blockade, dibucaine number

Pseudocholinesterase is an enzyme required to metabolize esters, such as succinylcholine, mivacurium, and ester-linked local anesthetics.[1] A deficiency of this enzyme can go undetected in affected individuals until exposed to one of the forementioned drugs. Unfortunately, an undiagnosed pseudocholinesterase deficiency can lead to adverse outcomes, such as prolonged paralysis in the post-operative period. It is imperative to increase awareness in providers who administer these medications in order to increase patient safety.

Case Report

A 73-year-old, 66.2 kg, 155 cm Caucasian female presented for open reduction and internal fixation of her proximal right fifth metatarsal. Her medical history was significant for hypothyroidism, hypertension, and hyperlipidemia. Her past surgical history included a tonsillectomy and adenoidectomy, and a right shoulder arthroplasty, both without a documented history of anesthesia-related complications. Medications taken by mouth on the morning of surgery included levothyroxine sodium 75 mcg, metoprolol 50 mg, and atorvastatin calcium 20 mg. A recent echocardiogram showed a normal left ventricular ejection fraction of 65%. The electrocardiogram showed sinus rhythm with a rate of 76/min and chest x-ray reading was within normal limits. Preoperative laboratory values included hemoglobin 14.1 g/dL, hematocrit 42.8%, platelets 268x1000/mm3, potassium 4.0 mEq/L, and creatinine 1.0 mg/dL. The patient was classified as an American Society of Anesthesiologists (ASA) II physical status. Physical examination revealed full neck range of motion, mouth opening greater than 3 cm, and a thyromental distance greater than 6 cm.

The original anesthesia plan included an intravenous (IV) induction and the use of a size 4 laryngeal mask airway. The patient was premedicated with midazolam 1mg IV and transported to the operating room. She was preoxygenated by circuit face mask with oxygen at 10 L/min. Intravenous anesthetic induction medications included fentanyl 50 mcg, lidocaine 40 mg, and propofol 150 mg. After several unsuccessful attempts at laryngeal mask airway insertion, a decision was made to perform a direct laryngoscopy. Mask ventilation was started and succinylcholine 100 mg IV was administered along with inhalational sevoflurane at 2%. The patient's trachea was intubated with a 7.0 endotracheal tube using a miller 2 blade and respirations were controlled by a mechanical ventilator. The case remained uneventful during the maintenance phase, which lasted 40 min. During emergence, the patient did not show any spontaneous respiratory effort. Flumazenil 0.2 mg and naloxone 0.2 mg IV were administered, which had no effect on the patient's respiratory efforts. A peripheral nerve stimulator was used to assess neuromuscular blockade, revealing no evoked response. The patient was taken to the recovery room with the endotracheal tube left in place and respirations controlled by mechanical ventilation. She was placed on a propofol infusion 30 mcg/kg/min for

sedation. Although the endotracheal tube was safely removed 3 hours later, she was admitted to the hospital for overnight monitoring. The following morning, she was discharged home.

Discussion

The use of succinylcholine to facilitate endotracheal tube placement remains popular among anesthesia professionals due to its rapid onset and short duration of action. Therefore, it is imperative to understand the pharmacology of this medication in order to provide optimal patient care. Recovery from succinylcholine generally occurs within 2-3 min due to its rapid metabolism by pseudocholinesterase.[2] When there are deficient levels of pseudocholinesterase in the plasma, more of the drug is able to reach the neuromuscular junction resulting in a prolonged neuromuscular blockade.[3] If a patient exhibits prolonged neuromuscular blockade, as evidenced by apnea and lack of an evoked response from a peripheral nerve stimulator, a pseudocholinesterase deficiency should be suspected.[3] These patients should remain intubated with ventilatory support, sedated, and treated for pain postoperatively until adequate spontaneous respirations return, as there is no antagonist for succinylcholine.[4] Spontaneous breathing efforts may not be regained for hours and in some cases days until diffusion away from the nicotinic receptor occurs, as evidenced in multiple case studies.[2,3,5]

One case report[5] stated that a pseudocholinesterase deficiency can either be acquired or inherited. It is not until an individual gets tested for a pseudocholinesterase abnormality, that it can be determined if the abnormality is due to a decreased enzyme level or decreased activity of the enzyme.[3] Therefore, a serum pseudocholinesterase level during the post-operative period should be considered in order to determine if the prolonged paralysis is due to a decrease in the enzyme level.[5] Since multiple physiologic, pharmacologic, and pathologicfactors can influence the amount and/or activity of this enzyme, one must also rule out whether the cause was genetic in origin.[3] Therefore, a dibucaine inhibition test should be performed.[4] This test will determine if the individual is homozygous normal, homozygous atypical, or heterozygous.[3] Unfortunately in this case study, neither test was considered post-operatively. The patient and family were counseled regarding the atypical prolonged effects of succinylcholine. The case was properly documented in the patient's records to prevent prolonged neuromuscular blockade in the future.

Although there is no antagonist for succinylcholine, there are interventions that may be useful in speeding the recovery from prolonged paralysis. Transfusions of fresh frozen plasma or packed red blood cells have been used in an attempt to reverse paralysis.[1] One case report[2] stated that transfusions may not be the preferred management plan due to the quantity required to adequately reverse the paralysis.[2] Unless there is an emergent need to check neurological function, the risk of transfusion transmitted infections and transfusion reactions may outweigh the benefit. Cholinesterase inhibitors, used for the antagonism of nondepolarizing neuromuscular blockers, should not be administered because their effects can further exaggerate the paralysis and worsen patient outcomes.[3] Additionally, antagonists may lead to an increase in undesirable muscarinic side effects, such as bradycardia and an increase in pulmonary secretions.[2]

Abnormalities in electrolytes, such as potassium, magnesium, and calcium, can contribute to the compromised function of pseudocholinesterase and should be corrected.[2] Finally, human serum cholinesterase has been cited in the literature to hasten recovery times, but is not currently available in the United States.[1] In this case report, no attempts at antagonizing the effects of succinylcholine were attempted. Regardless of whether or not an intervention is attempted to reverse the paralysis, the primary concern is to protect the airway and maintain mechanical ventilation until spontaneous respiratory muscle function has returned.

Managing the patient postoperatively who is experiencing prolonged neuromuscular blockade secondary to a pseudocholinesterase deficiency is a team effort. Awareness while paralyzed is a risk factor in the post-operative period.[2] Vital signs should be closely monitored for an increase in sympathetic activity, such as increases in heart rate and blood pressure, as these could be a sign of pain and anxiety.[2] Immobility should not be mistaken for comfort. Narcotics and sedatives should be continued until spontaneous respiratory efforts return.[2] In this case study, the patient was sedated using a propofol infusion. Additionally, every attempt should be made to prevent complications associated with prolonged paralysis and mechanical ventilation such as corneal abrasions, pressure ulcers, and infection.[2] It is also the responsibility of the anesthesia team to correct any electrolyte abnormalities in the pre, intra, and post-operative period, as this can contribute to the compromised function of pseudocholinesterase.[2]

The patient in this case study had no history of succinylcholine administration during her previous surgeries, which resulted in no documentation of anesthesia complications. Routine genetic testing for pseudocholinesterase deficiency is not the standard of practice prior to general anesthesia.[2] Therefore, a deficiency usually goes undetected until the patient is exposed to a medication hydrolyzed by psuedocholinesterase. Once diagnosed, it is the responsibility of the healthcare team to educate the patient and family, encourage them to get tested, and communicate this information to future healthcare practitioners.

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Mentor: David M. Kalil, CRNA, DNAP

Cardiac Stent Occlusion following Discontinuation of Pradaxa

Tamara Edie, MSN
Louisiana State University Health Sciences Center

Keywords: Oral anticoagulation, intraoperative myocardial infarction, cardiac stents, direct thrombin inhibitor, stent thrombosis

Many patients presenting for non-cardiac surgery have cardiac stents which require anticoagulant therapy to prevent stent thrombosis and sequelae such as myocardial infarction. Anesthesia practitioners are often responsible for completing the preoperative evaluation, which includes advising patients to continue or discontinue certain medications that may have a deleterious impact on bleeding, blood pressure or heart rate in the perioperative period. Dabigatran etexilate (Pradaxa) is a new anticoagulant agent which anesthesia practitioners need to be aware of.

Case Report

A 63-year-old, 93 kg, 180 cm male presented for right distal ureterectomy secondary to a ureteral tumor. His medical history was significant for smoking, hypertension, coronary artery disease, myocardial infarction, diastolic dysfunction, atrial fibrillation and ureter cancer. His surgical history included thyroid surgery, Achilles tendon repair, failed cardiac ablation, and a coronary angioplasty with stent placement to the right coronary artery (RCA) and left anterior descending artery (LAD). Current medications consisted of acetylsalicylic acid, atorvastatin, dabigatran etexilate, metoprolol, and methotrexate. The acetylsalicylic acid was discontinued 5 days and the dabigatran etexilate 2 weeks prior to surgery as instructed. Noteworthy preoperative diagnostic procedures included an echocardiogram and a heart catheterization which revealed a left ventricular ejection fraction (EF) of 55% with blockages of previously inserted stents (50 % RCA, 30% LAD). Serum laboratory values were within normal limits. Coagulation studies were not included in the lab tests ordered. Preoperative vital signs were within normal limits with a heart rate ranging between 80 to 110/min.

Upon arrival to the operating room, the patient was preoxygenated with oxygen 10L/min via face mask and standard noninvasive monitors applied. Anesthesia was induced with intravenous (IV) administration of midazolam 2 mg, fentanyl 100 mcg, lidocaine 60 mg, propofol 150 mg and rocuronium 50 mg. The trachea was intubated with an 8.0mm ID endotracheal tube under direct laryngoscopy and respirations controlled by mechanical ventilator. Anesthesia was maintained using end tidal concentrations of desflurane 6-7 %, oxygen 1 L/min, and air 1 L/min.

One hour post-induction, the patient became tachycardic, with a heart rate of approximately 140/min. Esmolol 10 mg and metoprolol 1 mg were administered IV to control the heart rate. The patient's cardiac rate returned to his prior range of 80 to 110/min. Approximately 20 min after this intervention, the pulse was noted to be 65/min and decreasing. Glycopyrolate 0.2 mg IV was given, followed by atropine 1mg IV. Despite the administration of these medications, the heart rate continued to decrease steadily. A left radial arterial pressure line and a right internal jugular central venous catheter were inserted. The patient became hypotensive due to the bradycardia but maintained a palpable

carotid pulse. Epinephrine 400 mcg and ephedrine 50 mg IV were administered and chest compressions were initiated to circulate the medications. A trans-esophageal echocardiogram was performed and it was revealed that the patient's EF was 10% (down from 55%). It was also noted that only the apex of his left ventricle appeared to be contracting. Epinephrine and nitroglycerine drips were initiated and titrated to maintain a mean arterial pressure of at least 60 mm Hg. The surgeons were able to quickly complete the procedure and the patient was transferred to interventional radiology where it was discovered that his cardiac stents were occluded (proximal RCA 100%, mid RCA 60% and LAD 70%). Revascularization via angioplasty was achieved and the epinephrine and nitroglycerine infusions were titrated to off. Post procedure echocardiogram revealed an EF of 50%. The patient was transferred to the cardiac care unit and the endotracheal tube was removed without complication.

Discussion

The American Heart Association's data estimates that 15.4 million Americans over the age of 20 have Coronary Heart Disease. Revascularization and pharmacologic therapy have become a part of mainstream medical treatment for coronary heart disease. In 2010, at least 454,000 Americans underwent coronary stenting.[1] After cardiac stenting, antiplatelet and anticoagulant medications are commonly utilized to prevent thrombus formation from occluding the stents. Occlusion of stents may lead to ischemia and adverse cardiac events, such as myocardial infarction and often death.[2] However, these medications may have a significant negative impact on bleeding in the perioperative period, contributing to hemorrhage and hematoma formation. Often times, patients are encouraged to wait at least one year before having elective surgery to minimize the interruption of anti-platelet therapy. Even after this time period, thrombus formation may occur if anticoagulant medication is halted.[3]

It is important that the healthcare community remain educated about the current guidelines and recommendations regarding anticoagulant medications for the patient with cardiac stents in the perioperative period.[3] As such, a thorough preoperative evaluation and knowledge of care of the patient with coronary stents is crucial to the safe anesthetic care of these patients.

Acetylsalicylic acid, warfarin, and clopidogrel are commonly prescribed antiplatelet agents. Healthcare personnel are more familiar with recommendations for the perioperative management of these medications. Dabigatran etexilate, a direct thrombin inhibitor, is a newer type of oral anticoagulant medication. The mechanism of action of this agent is to prevent the following: 1) conversion of fibrinogen to fibrin, 2) positive feedback amplification of coagulation activation 3) cross-linking of fibrin monomers 4) platelet activation, and 5) inhibition of fibrinolysis.[4] The onset of anticoagulant activity is fairly rapid (0.5-2 hours) with an elimination half-life of 12-14 hours and a duration of action of 4 hours. It undergoes renal excretion, so it may have an extended duration of action in patients with decreased renal function. There is no antidote or reversal for this medication; however it may be removed via dialysis in an emergent situation.

There are guidelines for discontinuation of dabigatran prior to invasive procedures, with recommendations for procedures with low and high risk bleeding. The half-life of dabigatran can be used to estimate the length of time that the patient will have impaired

coagulation. Because dabigatran is excreted via the kidneys, the creatinine clearance may be used to estimate the half-life of dabigatran. This would potentially aid in approximating the amount of time that the patient will have impaired coagulation, thereby assisting in the guidance of recommendations for discontinuation in the perioperative period. If the creatinine clearance is elevated, one may assume that the dabigatran will be effective longer; conversely if the creatinine clearance is normal then one may assume the half-life of dabigatran is typical (12-14 hours).[1] Common coagulation tests such as the prothrombin time and activated partial thromboplastin time are often not useful or recommended in evaluating the coagulation status of a patient taking dabigatran because they are not able to sufficiently quantify the drug levels present.[1,2] Thrombin clotting time and ecarin clotting time results are the most sensitive assays to evaluate anticoagulant status, but these tests may not be readily obtained in many facilities.[4,5]

Literature regarding perioperative management of the patient with coronary stents suggests that the risk of stent thrombus formation should be compared to the risk of hemorrhagic complications associated with the proposed surgery.[6] The goal is to minimize the length of time that the patient is without the beneficial effects of the anticoagulant medication thus minimizing the risk of an adverse thrombotic event.[4]

Although the recommendation given to this patient was to discontinue his dabigatran 14 days prior to surgery, this was not in accordance with the literature available regarding stent thrombosis or dabigatran.[1, 4] Retrospectively, the patient should have stopped taking his dabigatran 5 days prior to surgery at the same point that he stopped taking aspirin. While the surgical ureterectomy posed a high risk for hemorrhagic complications, the risk for a thrombotic event was also extremely high given that the patient's renal clearance was normal. Because the surgeon was adamant regarding the time frame that the dabigatran be withheld, perhaps bridge therapy should have been recommended to protect the patient from adverse thromboembolic complications.[3] Also, if there was any doubt regarding the patient's coagulation status, lab testing to obtain a dabigatran level might have been ordered in an effort to make an educated decision regarding his medication regimen.[1]

There are many different antiplatelet and anticoagulant medications on the market. The incidence of myocardial infarction that occurs from stent occlusion when perioperative management of anticoagulant medications are inappropriately administered has been well documented.[4, 5] Anesthesia practitioners should be vigilant in the preoperative evaluation of patients with cardiac stents and be alert to the possible complications which may arise due to mismanagement of preoperative antiplatelet and anticoagulant medications.

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Mentor: David M. Kalil, CRNA, DNAP

Unilateral Hypoglossal Nerve Palsy Following a Lightwand Intubation

LT Cody R. Gremore BSN
Uniformed Services University

Keywords: hypoglossal, nerve palsy, general anesthesia, endotracheal intubation, neurapraxia, unilateral, cranial nerve XII

The hypoglossal nerve (cranial nerve XII) is a pure motor nerve that supplies the infrahyoid muscle in the tongue and controls swallowing as well as speech.[1,2] While damage to the hypoglossal nerve remains a rare complication following general anesthesia,[3] nerve damage is a significant source of anesthesia-related legal claims.[2] Typical manifestations include dysarthria, dysphagia, or respiratory distress. Treatment for hypoglossal nerve palsy is typically supportive in nature and may include fluid replacement, systemic steroids, or vitamin B12.[4] This case report presents a unilateral hypoglossal nerve palsy following a lightwand intubation for a laparoscopic ileocecectomy.

Case Report

A 30-year-old male with a history of Crohn's disease and asthma was scheduled for a laparoscopic ileocecectomy for a Crohn's stricture refractive to treatment with infliximab. Past surgical history consisted of a fistulectomy 4 years previously with no anesthetic complications. Upon examination, the patient had normal body habitus with a body mass index of 24 kg/m^2, a Mallampati score of 2, and a thyromental distance greater than 6 cm.

The surgery was performed under general anesthesia in the supine position. After induction, the second-year nurse anesthesia student electively attempted intubation utilizing a lightwand and was unsuccessful in passing the 7.5 mm endotracheal tube (ETT) into the trachea. The staff anesthesiologist attempted intubation using the same method and was able to successfully intubate the patient. Bilateral breath sounds were auscultated and the ETT was taped 24 cm at the teeth to the right of midline. The entire event took less than 3 min and there was no identifiable oropharyngeal trauma. An 18 french orogastric tube (OGT) was then passed to decompress the stomach for the operation. The procedure was uneventful, lasting

approximately 3.5 hours. The patient was placed in the left airplane position briefly for surgical access and blankets were used to maintain the head and neck in a neutral position. An oral airway was placed during emergence, prior to extubation, to prevent occlusion of the ETT and was removed prior to proceeding to the post-anesthesia care unit (PACU).

During the post-operative visit the following day, the patient reported difficulty with mobility of his tongue and slurred speech. When asked to protrude his tongue, a deviation to the left was noted. The staff anesthesiologist was consulted and together a brief neurologic exam was conducted and cranial nerves II-XI appeared grossly intact. Neurology was consulted and a computed tomography (CT) scan of the head and neck was performed to rule out a cerebral vascular accident. The CT scan noted no acute intracranial process but was significant for swelling on the base of the tongue that was more prominent on the left side. A swallow study was also conducted, which showed normal function and the patient's diet was advanced as tolerated. The patient was diagnosed with cranial nerve XII injury of unknown etiology. Follow-up was conducted on a daily basis throughout the week noting consistent improvement and speech returning to baseline. The patient was discharged home on post-operative day 6 with only trace evidence of the signs and symptoms previously described. Follow-up via a phone call was attempted on two separate occasions but unsuccessful.

Discussion

Hypoglossal nerve palsy is most often associated with malignant tumors, but may occur as a result of trauma, stroke, multiple sclerosis, Guillain-Barre neuropathy, infection, or surgery.[2] Very few cases exist in the literature describing unilateral hypoglossal nerve palsy following oral intubation, making it a rare complication associated with general anesthesia.[5] Suspected mechanisms of injury following general anesthesia include compression of the hypoglossal nerve against the hyoid bone by excessive cuff pressure in an ETT or laryngeal mask airway (LMA), compression of the base of the tongue by a laryngoscope blade or ETT, hyperextension of the neck, and direct trauma.[2,3,5,6]

The hypoglossal nerve courses on the lateral prominence of the transverse process of the first cervical vertebrae and hyperextension of this joint could theoretically compress the nerve against the prominence.[6] Excessive cuff pressure in an LMA has been cited as a direct source of nerve compression against the hyoid bone,[3] while excessive ETT cuff pressure in the presence of throat packing has also been postulated as a similar mechanism of nerve compression resulting in hypoglossal nerve palsy.[6]

In a literature review conducted by Dziewas and Lüdemann, the majority of reported cases of hypoglossal nerve palsy followed rhinopharyngological surgery with the use of direct laryngoscopy such as a tonsillectomy or various vocal cord procedures.[3] They also identified some prognostic data following a suspected hypoglossal nerve injury. Out of 20 cases mentioning some form of recovery, 13 of them achieved complete recovery within 1 week to 4 months, only partial recovery was documented in 3 of the cases, 4 of the cases exhibited no improvement of the nerve palsy.[3] Age and gender did not appear to be correlated with the incidence of hypoglossal nerve palsy in the data gathered by Dziewas and Lüdemann.

In this particular case, intubation was achieved with the use of a lightwand. During intubation, the head was maintained in a neutral position and direct laryngoscopy was not performed at any point during the process. Furthermore, there was no identifiable trauma following intubation and the surgical site was far from the site of injury. In this instance, hypoglossal nerve palsy is suspected to be the result of the ETT compressing the base of the tongue as evidence by swelling of the base of the tongue on the CT scan. Without the use of direct laryngoscopy, it is difficult to determine the position of the ETT beyond the oral cavity and this may have been the source of unidentified compression of the tongue. It is also possible that the OGT was the source of nerve compression, however the diameter of the tube was much smaller than that of the ETT and it was not secured in place, which would allow it to move freely in the oral cavity, eliminating any significant source of direct pressure. Steroid and/or vitamin therapy were not prescribed, as evidence regarding their efficacy is lacking.[2] However, consistent follow-up indicated almost complete spontaneous recovery within one week post-operatively.

In conclusion, it appears that while hypoglossal nerve palsy remains a rare complication following general anesthesia, it is a definite risk associated with endotracheal intubation. The degree to which this complication is preventable remains unidentified, but it would be prudent to avoid hyperextension of the neck during intubation, monitor ETT and LMA cuff pressures (particularly when using nitrous oxide), and in this instance, assess the position of the endotracheal tube after intubation to ensure that excessive compression is alleviated. Duration of direct laryngoscopy appears to be a more significant risk factor than the duration of the procedure itself, as evidenced by the overwhelming number of cases being reported after the use of direct laryngoscopy for surgical visualization.[3] Furthermore, manufacturers recommend that the use of a LMA be limited to less than 3 hours while ensuring that cuff pressures do not exceed 60 cm H_2O pressure, although there have been documented LMA cases for up to 24 hours without any adverse events.[7] While most documented cases of hypoglossal nerve palsy following general anesthesia result in full recovery,[3] any degree of neurologic deficit in the immediate post-operative period remains a concern, so awareness and vigilance regarding this complication cannot be overemphasized.

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Mentor: LTC Paul M. Johnson, CRNA, DNP

Visual Evoked Potentials and Anesthesia

Aaron Champagne, MS
Louisiana State University Health Sciences Center

Introduction

Visual evoked potentials (VEPs) are one type of evoked potential used for neurophysiologic monitoring in the surgical arena. Other, more familiar, evoked potentials include somatosensory, motor, and brainstem auditory evoked potentials. Visual evoked potentials are employed to assess any disruption in the pathway of the visual system from eye to occipital cortex.[1] The patient wears goggles that elicit light-emitting diode (LED) flashing lights that stimulate the retina and optic nerve pathways. Through electrodes on the scalp, activity in the visual cortex is recorded. The recorded waveforms produced by the VEPs have both amplitude and latency. Amplitude refers to the size of the positive or negative deflection of the waveform from baseline and is measured in relationship to the preceding waveform.[1] Latency describes the time from start of a stimulus to the start of a response.[1] Various anesthetic drugs can have differing effects on amplitude and latency. Although an infrequent event for anesthesia providers, the effects of anesthetic drugs used while VEPs are being conducted need to be deliberated so that a reliable and valid test can be accomplished. A review of the current literature was undertaken on the effects common anesthetic drugs have on VEPs to improve anesthetic plans in order to minimize influence on VEP monitoring.

Methodology

Evidence-based Practice Model
The PICO format was used to create a clinical question that would guide the search criteria. The PICO parameters were P (patient population) for patients who underwent VEPs, I (current intervention) for anesthetic drugs that affect amplitude and latency during VEPs, C (contrasting intervention) for anesthetic drugs that do not or minimally affect amplitude and latency during VEPs, and O (outcome of interest) for a valid and reliable VEP test.

Purpose
The purpose of this review was to determine how certain anesthetic drugs impact the amplitude and latency of the waveforms in VEPs, thus affecting the test's validity and reliability. The clinical questions used for the search included: (1) Which anesthetic drugs are used most commonly during VEPs? and (2) How are the amplitude and latency of the VEP waveform impacted by the more commonly used anesthetic drugs?,

and (3) Which anesthetic drugs are a better choice to use during VEPs?

Search Terms
Visual evoked potentials, evoked potentials, anesthesia, dexmedetomidine, sevoflurane, desflurane, propofol, midazolam, fentanyl, neuromonitoring

Search Models
An electronic database search using Cochrane library, Medline via OVID and PubMed, CINAHL and Google Scholar from the years 2008-2014

Levels of Evidence
Studies found and deemed appropriate for review were assessed for its level of evidence based on its design. The rank of I being randomized control trials or meta-analysis, rank of II being non-randomized control trials such as retrospective studies, rank of III being correlation or comparative studies such as those with an experimental design, and rank of IV being opinion pieces. There were four studies with experimental designs that provided Level III evidence.

Literature Review

The majority of the literature available regarding VEPs is not current. Visual evoked potentials are not commonplace and so the current peer reviewed literature is scant, especially literature that considers the effect of anesthesia drugs on VEPs. The current literature includes several studies which are animal based, and additional studies that involved measuring VEPs directly from the visual cortex as opposed to measuring from electrodes placed onto the scalp.

Kuroda et al used a rat model to measure the effects of certain opioids on VEPs. Anti-nociceptive doses of morphine, pentazocine, fentanyl, and butorphanol were administered subcutaneously to a rat that underwent VEP testing. An antagonizing dose of naloxone was administered intraperitoneally to the rat and VEPs retested. The number of rats tested was not reported. Amplitude and latency during early VEP components and during late VEP components were compared. This study demonstrated that some narcotics do influence the amplitude of VEPs. Early VEP components were modified due to the effects of morphine.[2] However, fentanyl, pentazocine, and butorphanol caused no variation in early VEP components.[2] Late VEP components were influenced by morphine, fentanyl, and pentazocine.[2] Butorphanol caused no change in late VEP components.[2] Naloxone was shown to have had significant antagonism to the VEPs components caused by morphine and fentanyl. Moreover, the authors inferred that a relationship existed between early and late VEP components and opioid receptors.[2]

In 2010, Sloan et al published their findings from an experimental study that looked at the use of volatile anesthetics in a baboon model during evoked potential testing.[3] Ten baboons, five male and five female, were given general endotracheal anesthesia with varying combinations of isoflurane with nitrous oxide and varying combinations of isoflurane with halothane. Sensory evoked potentials, auditory brainstem responses, and VEPs were recorded during the general anesthetic and again one month later.[3] The authors stated that analysis of the data was performed by a blinded observer. The amplitude and latency of the results were compared after normalizing the data to the average recorded at 0.8% isoflurane for that dataset.[3] The authors concluded that the decreases in amplitude and increases in latency during the evoked potentials were attributable to the synergistic effects of isoflurane and nitrous oxide and additive

effects of isoflurane with halothane. In addition, the authors noted that limitations to their study included a lack of literature that compared combination volatile agents to pure volatile agents during sensory evoked responses. The authors stated their results may not be applicable in humans because the MAC of nitrous oxide is lower in the human than in the baboon.

In an experimental study by Ota et al in 2010 the usefulness of VEPs during neurosurgery was evaluated. It was found that VEPs were a useful adjunct to monitor the visual integrity of the posterior visual pathway during neurosurgery.[4] Visual evoked potentials were performed in 17 patients that had neurologic microsurgical procedures near posterior visual pathways. General anesthesia was induced with sevoflurane or propofol. An unnamed muscle relaxant was also administered. This study recorded VEPs directly from the visual cortex instead of the scalp. No statistical analysis was mentioned by the authors. Given the controversy regarding whether or not VEPs provide reliable, useful information, this study was able to consistently produce detectable VEPs in 82% of the patients.[4] Visual evoked potentials were stable and detectable despite the patient being anesthetized with sevoflurane or propofol.[4] The findings of this study suggest that sevoflurane and propofol may be used to anesthetize a patient while preserving the ability to record and obtain VEPs in a manner that is both consistent and reliable when the VEPs are measured directly from the visual cortex instead of the scalp.

Hudetz et al conducted an experimental study that examined the effect of desflurane on resting and visual evoked unit activity in a rat model.[5] Varying concentrations of the volatile agent were administered to eight adult rats during a general anesthetic. Visual evoked potentials were recorded per the animal's left eye at the different concentrations. Results demonstrated that VEPs were stable but the long-latency component was attenuated in a concentration-dependent manner.[5] No change in early response firing rate with increases in anesthetic concentration was noted by the authors. In addition, the authors pointed out that an increase in desflurane concentration significantly reduced late response. Moreover, the authors laid caution to the notion of comparing intracortical VEPs in a rat model to scalp recorded VEPs in humans.[5]

Articles	**Description**	**Types of anesthesia**	**Results**
Kuroda et al., 2008[1]	Experimental design. Subjects were male Sprague Dawley rats (quantity not specified). Anesthetized with pentobarbital and then stereotaxic apparatus was surgically implanted into the occipital cortex. Morphine, pentazocine, fentanyl, butorphanol and naloxone were administered and then VEPs were recorded to see if/ how the drugs affected amplitude and latency.	Morphine Pentazocine Fentanyl Butorphanol Naloxone	Pupillary size has an effect on amplitude and latency of VEPs. Thus any drug that causes mydriasis or miosis will impact VEP results. These include opioids and anticholinergic agents.

Sloan, Sloan, and Rogers, 2010[2]	Experimental design. Subjects were five male and female baboons. Ketamine and lidocaine given to facilitate tracheal intubation. The effect of isoflurane in combination with nitrous oxide on visual, auditory and somatosensory evoked potentials was evaluated. Then isoflurane combined with halothane was administered and the effect on visual, auditory and somatosensory evoked potentials was evaluated.	Isoflurane (Iso) and Nitrous Oxide (N2O) combinations: *0.8% Iso (No N2O) *0.6% Iso with 20% N2O *0.4% Iso with 40% N2O *0.2% Iso with 60% N2O *79% N2O (No Iso) Isoflurane (Iso) and Halothane (Hal) combinations: *0.8% Iso (No Hal) *0.6% Iso (No Hal) *0.6% Iso with 0.15% Hal *0.4% Iso with 0.3% Hal *0.2% Iso with 0.45% Hal	The mixtures of isoflurane and halothane affected the measurements to an extent that the effect of the mixtures was greater than the agents alone. The combination of isoflurane and nitrous oxide had synergistic effects on sensory evoked responses.
Ota et al., 2010[3]	Experimental design. 17 subjects (10 male and 7 female). Evaluation of cortical VEPs instead of scalp-recorded VEPs. Electrodes placed directly to the visual cortex. VEPs recorded while general anesthesia was maintained with sevoflurane or propofol	Sevoflurane (11 patients) or propofol (5 patients) or a combination of sevoflurane and propofol (1 patient).	Results suggest that recording directly from the cortex produces reliable VEPs that are unaffected by sevoflurane and propofol.
Hudetz, Vizuete, and Imas, 2009[4]	Experimental design. Subjects were 8 adult male Sprague-Dawley rats. Studied the effect of desflurane on resting and visual evoked potential activity in vivo. VEPs were recorded directly from the visual cortex.	Desflurane concentration ranges from 2%-8%	VEPs were stable and able to be recorded, but the long-latency component is affected in a dose-dependent manner which may be related to loss of consciousness and a loss of cortico-cortical feedback.

Table 1. Recent literature regarding anesthesia drugs and sensory evoked potentials

Conclusions

Neurophysiologic monitoring, such as SSEPs, MEPs, and VEPs, during a surgical procedure are an issue that anesthesia providers must take into account when developing an anesthetic plan. Knowing which anesthetic drugs have the least amount of impact on amplitude and latency of evoked potential waveforms will guide the anesthesia provider in this decision making process ensuring a valid and reliable test. The manner in which the VEPs are conducted, whether scalp-recorded or

recorded directly from the visual cortex, according to recently reviewed literature, have an impact on the information obtained. The anesthesia provider should communicate with the neuromonitoring technician and inquire to the manner in which VEPs will be recorded. In vivo cortical recordings seem not to be affected by volatile anesthetics or propofol. However, current literature suggests that medications that have an effect on pupillary size should be used judiciously as they did have an impact on cortically recorded VEP waveforms. Randomized control trials using volatile anesthetics, opioids, and sedative hypnotics need to be conducted to garner more evidence and further this discussion.

References

1. Odom J, Bach M, Brigell M, et al. ISCEV standard for clinical visual evoked potentials (2009 update). *Doc Ophthalmol*. 2010;120:111-119.
2. Kuroda K, Fujiwara A, Takeda Y, et al. Effects of narcotics, including morphine, on visual evoked potential in rats. *Eur J Pharmacol.* 2009;602:294-297.
3. Sloan T, Sloan H, Rogers J. Nitrous oxide and isoflurane are synergistic with respect to amplitude and latency effects on sensory evoked potentials. *J Clin Monitor Comp.* 2010;24:113-123.
4. Ota T, Kawai K, Kamada K, et al. Intraoperative monitoring of cortically recorded visual response for posterior visual pathway. *J Neurosurg*. 2010;112:273-284.
5. Hudetz A, Vizuete J, Imas O. Desflurane selectively suppresses long-latency cortical neuronal response to flash in the rat. *Anesthesiology*. 2009;111:231-239.

Mentor: Jennifer E. Badeaux, CRNA, DNP

Editorial

I would like to dedicate this issue in memory of Russell Lynn, CRNA, MSN. Russ started as a reviewer for the ISJNA in the summer of 2006, and served as a cherished member of our editorial board for the past six years. Russ was truly dedicated to the education of nurse anesthetists as an educator, scholar, and practitioner. He led by example as a professional and lifelong learner through his active involvement at the state and national level, and by pursuing his PhD at the University of Pennsylvania. He will truly be missed.

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.**"

To access prior issues of the ISJNA visit the following link:
www.aana.com/studentjournal

INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA
GUIDE FOR AUTHORS

MISSION STATEMENT

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

ITEMS ACCEPTED FOR PUBLICATION

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

ITEM PREPARATION & SUBMISSION

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

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

PEER REVIEW

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

General guidelines

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

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

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

<u>Case Reports</u>

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

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

Heading (see #9 above in General Guidelines)

[space]

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

[space]

Case Report (bold, 400-500 words)

[space]

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

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

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

[space]

Discussion (bold, 600-800 words)

[space]

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

[space]

References (bold)

[space]

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

[space]

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

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

Research Abstracts

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

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

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

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

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

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

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

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

AMA MANUAL OF STYLE

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Vicki C. Coopmans, CRNA, PhD
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.