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TOPICS IN THIS ISSUE

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Front Cover:

William Gafford, BSN, RN and Newton Tinsley, BSN, RN, graduate students enrolled in the Samford University nurse anesthesia program, instruct an Unless U student on proper hand hygiene. Unless U is a

non-profit organization serving individuals with disabilities through continuing education, life skills, and social skills. Mr. Gafford and Mr. Tinsley were named Albert Schweitzer Fellows - Schweitzer Fellows develop and implement service projects that address the root causes of health disparities in under-resourced communities - each project is implemented in collaboration with a community-based health and/or social service organization. The goals of Mr. Gafford and Mr. Tinsley's project were to implement preventative measures to optimize the physical well-being of students, including developmentally appropriate teaching on physical fitness, nutrition, and hygiene, and prepare teachers and families for medical emergencies that may be encountered by this vulnerable population. They succeeded in getting an AED donated and trained the teachers. In the cover photos they are teaching the students at Unless U proper hand hygiene using an engaging and hands-on tool called Glow-Germ to highlight the most important aspects of proper hand washing. After successful teaching, along with other aspects of their partnership with the school, they were able to report zero flu cases for this past flu season!

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Spinal Cord Perfusion during Cervical Discectomy Fusion

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Keywords: spinal cord perfusion, invasive hemodynamic monitoring, cervical surgery

When blood flow to the spinal cord is compromised, prompt intervention is critical to avert suboptimal patient outcomes. In the clinical setting, a mean arterial pressure (MAP) of 60 mm Hg is generally considered the minimum to maintain organ perfusion. Falling below 55 mm Hg has been associated with kidney and myocardial injury.[1] In patients with spondylitic myelopathy, ensuring adequate spinal perfusion pressure is critical to reduce the risk of spinal cord infarction. Invasive blood pressure monitoring is recommended during anterior cervical spine surgery for this reason.[2] This case report questions the sufficiency of current monitoring practice in spinal procedures.

Case Report

A 50-year-old, 89.8 kg, 165 cm female with a diagnosis of cervical spondylosis with myelopathy presented for a cervical five-six (C5-C6) anterior cervical decompression discectomy fusion. Magnetic resonance imaging (MRI) of the neck revealed spondylosis with a large disc protrusion at C5-C6, causing narrowing of the diameter of the dural sac.
The patient's health history included anemia, non-insulin dependent type II diabetes, Grave's disease, migraine, muscle pain, pituitary microadenoma, polycystic ovarian syndrome, and sleep disorder. The patient's medications included a multivitamin, methocarbamol, tramadol, zolpidem, ferrous sulfate, spironolactone, metformin, meclizine, and magnesium. All laboratory values were normal.

Upon assessment, the patient complained of severe left-sided neck pain with facial numbness. Any attempt at movement from the right lateral position, with the neck in a rightward flexion was not tolerated. Prior to transfer to the operating room (OR), midazolam 2 mg and fentanyl 50 mcg were administered into a 20-gauge intravenous (IV) catheter.

Upon arrival to the OR standard noninvasive monitors applied and fentanyl 50 mcg was administered. The patient was allowed to remain in the semi-lateral position. Oxygen 12 L/min was administered via facemask while the patient took deep breaths to ensure adequate preoxygenation. General anesthesia was induced on the stretcher using lidocaine 50 mg, propofol 200 mg, and succinylcholine 100 mg. Modified in-line stabilization of the neck was performed by one practitioner while a second performed laryngoscopy using a video laryngoscope. After securing the airway with a 7.0 mm endotracheal tube, a second IV and a radial arterial line were placed.

General anesthesia was initiated with desflurane 6% with oxygen at 0.5 L/min and air at 0.5 L/min. After placement of neuromuscular monitoring needles, the desflurane was replaced with a propofol infusion at 200 mcg/kg/min. Supplemental boluses of dexmedetomidine 10 mg were administered twice. A MAP goal of greater than 70 mm Hg was maintained using a

phenylephrine infusion, titrated as needed. Throughout the surgery, somatosensory evoked potential (SSEP) and motor evoked potential (MEP) monitoring was utilized.

The case proceeded without incident through emergence. A total of fentanyl 600 mcg was administered throughout the surgery. Upon closing of the surgical wound, desflurane 6% was initiated to replace the propofol infusion. At completion of the procedure, after return of spontaneous ventilation and awakening of the patient, the endotracheal tube was removed and O_2 10 L/min administered to the patient via facemask. Transfer to the post anesthesia care unit (PACU) was uneventful. The patient followed commands equally with all four extremities, reported alleviation of pain, and tolerated an aligned semi-fowler's position.

Discussion

The effects of blood pressure on patient outcomes in spinal surgery cannot be overemphasized. Patients such as the one presented in this case study are at risk for microinfarction of the cord due to chronic compression at the injured area.[2] For that reason, perfusion of the spinal cord is a critical consideration. A MAP of greater than 85-90 mm Hg to ensure spinal cord perfusion is recommended for patients with acute spinal cord injuries.[3] Current recommendations for anterior cervical neurosurgical procedures include maintaining MAP > 80 mmHg, along with the use of arterial blood pressure monitoring.[2,4] This is intended to protect circulation to the anterior two-thirds of the spinal cord that is perfused by the anterior spinal artery. In this case, the surgeon indicated that MAP should be maintained at >70 mm Hg. An arterial line was placed and MAP maintained as ordered, but not necessarily as high as recommended for acutely injured spinal cord patients.

A study by Saadoun, Chen, and Papadopoulos sought to validate blood pressure management by monitoring intraspinal pressure (ISP) and spinal cord perfusion pressure (SCPP) at the site injury of spinal cord injured patients.[5] In this study, probes were tunneled to the spinal cord surface to measure ISP. Arterial blood pressure was measured with a radial artery catheter. Results were measured in neurological outcome at 9-12 months after surgery. The key finding of this study was that ISP and SCPP measurements were predictive of neurological improvement over time. The best outcomes were observed in patients with a low ISP (<10 mm Hg) and SCPP > 90 mm Hg. Low SCPP and high ISP were found to be associated with ischemia at the site of injury.[5] The researchers concluded that MAP does not predict recovery because it is neither a direct measurement of ISP or SCPP. Further investigation of a correlation between recovery and initiation of interventions that reduce ISP or increase SCPP were recommended. However, no suggestion was made for alteration of current practice to improve monitoring.

Neurological monitoring such as SSEP and MEP is frequently used to continuously assess for damage to the spinal cord during surgery. The sensory pathway is located in the dorsal columns of the spinal cord. The posterior spinal artery and pial arterial plexus, an anastomosing network of vessels originating from the anterior and posterior spinal arteries,[6] provide blood supply to this portion of the spinal cord. Motor evoked potential monitoring was developed after recognition that SSEP fails to assess portions of the spinal cord that are perfused via the anterior spinal artery, which provides circulation to the anterior two-thirds of the spinal cord.[7] Use of this intraoperative monitoring impacts anesthetic plan and associated monitoring. Coordination

among the anesthesia provider, surgeon, and electrophysiological monitoring representatives is an important consideration in preparation for an anterior cervical surgery. Intraoperative monitoring with SSEP assesses sensory pathways at the site of surgery and has considerably reduced the risk of injury.[7]

The use of electrophysiological monitoring requires an anesthetic plan devoid of long-acting neuromuscular blockade. In the case study presented, the anesthesia practitioners collaborated with the surgeon and electrophysiological monitoring representatives. Halogenated agents can be used at concentrations of less than 0.5 MAC; however, a propofol infusion anesthetic provides optimal conditions for both SSEP and MEP assessment. The anesthesia practitioners administered drugs previously detailed that would not interfere with intraoperative monitoring of neurological function. Opioids, local anesthetics, and ketamine are common adjuncts, with dexmedetomidine added in this case. Because minor changes in blood pressure can alter readings on cortical evoked responses, use of invasive blood pressure monitoring is indicated when electrophysiological monitoring is performed.[7] A radial arterial line was used to monitor the patient and previously listed vasoactive drugs to maintain blood pressure.

The best indication of neurological function available during this case was electrophysiological monitoring data paired with arterial line blood pressure, as changes in SSEP or MEP due to changes in blood pressure could be immediately identified. While outcomes were positive for this patient and no adverse events occurred, it is impossible to determine if adequate SCPP was maintained throughout the case, except by examining the end result. As ISP monitoring is not a common practice, the anesthesia practitioners maximized the use of available equipment and standards of care to improve patient safety. It would be valuable in the future to study ISP monitoring during spinal procedures to determine if a more sensitive measurement of spinal cord perfusion could improve patient outcomes.

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Anterior Cervical Discectomy and Fusion with Neurophysiologic Monitoring

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Keywords: anterior cervical discectomy, intraoperative neurophysiologic monitoring, somatosensory evoked potential, motor evoked potential, intraoperative electroencephalography

Anterior cervical discectomy and fusion (ACDF) is a common procedure with a relatively low complication rate. However, any surgical manipulation of the spine can potentially lead to significant irreversible neurologic damage. Intraoperative neurophysiologic monitoring (IONM) can be routinely used to evaluate neuronal integrity by trending neurophysiologic signals to assess for ischemia and potential impending nerve injury. Anesthetic agents can alter the neurotransmission of these signals to varying degrees, so anesthetic technique must be adapted to minimize interference. The successful anesthetic management of a patient undergoing an ACDF with IONM is described here.

Case Report

A 58-year old female, weighing 79 kg and 158 cm in height, presented with chronic neck pain and worsening paresthesia in the upper extremities, more acutely on the left and without myelopathy. After computerized tomography and magnetic resonance imaging, the patient was diagnosed with cervical spine spondylosis and radiculopathy. Conservative noninvasive management did not yield any meaningful improvements, so the patient was scheduled for a two level ACDF including C5-6 and C6-7. The past medical history included moderate obesity, hyperlipidemia, hypertension and obstructive sleep apnea requiring continuous positive airway pressure at night. Her medications included atenolol, hydrochlorothiazide, atorvastatin, prochlorperazine and hydrocodone with acetaminophen. Preoperative laboratory data was unremarkable. The airway assessment revealed limited cervical range of motion with a sternomental distance of less than 12 cm and a Mallampati classification of III.

Preoperatively, the risks and benefits of general endotracheal anesthesia with IONM were discussed with the patient and informed consent was obtained. Before induction, midazolam 2 mg was given intravenously (IV) and pulse oximetry, noninvasive blood pressure and electrocardiogram monitors were applied. Following preoxygenation with O_2 10 L/min by facemask for 5 minutes, anesthesia was induced with IV doses of fentanyl 100 mcg, lidocaine 60

mg, propofol 130 mg and succinylcholine 120 mg. A GlideScope (Verathon Inc., Bothell, WA) provided a grade 1 view and the trachea was intubated with a 7.0 mm endotracheal tube (ETT) while maintaining neutral cervical spine alignment. Respirations were controlled with synchronized intermittent mandatory ventilation with pressure support providing tidal volumes of 550 mL, respiratory rate of 10/min, positive end expired pressure of 5 cm H_2O, and pressure support of 8 cm H_2O.

Somatosensory evoked potentials (SSEP), motor evoked potentials (MEP) and electroencephalography (EEG) electrodes were placed by the electroneurodiagnostic technician and baseline data documented. Maintenance of anesthesia included end-tidal sevoflurane 0.7 – 0.9%, propofol 80 – 100 mcg/kg/min and remifentanil 0.08 – 1 mcg/kg/min. Agents were titrated to EEG effect balancing adequate anesthetic depth and cerebral perfusion with minimal IONM interference. Multi-channel MEP and SSEP signals were strong throughout the procedure. Cefazolin 1 gm IV was given for antibiotic prophylaxis. Dexamethasone 4 mg IV and ondansetron 4 mg IV were given for antiemetic therapy. Intermittent hypotension was treated with a total of phenylephrine 400 mcg IV. At closure, hydromorphone 1 mg IV was administered for postoperative analgesia and IONM, propofol and remifentanil were discontinued. Throughout the procedure, the patient received lactated ringers 1,700 mL. On emergence, the ETT was removed with the patient awake; airway patency was maintained without assistance. Postoperatively, the patient moved all extremities with equal strength.

Discussion

Anterior cervical discectomy and fusion is indicated when disc compression of the spinal cord or spinal nerves cause radiculopathy. The meticulous surgical manipulation in close proximity to the spinal cord is associated with the potential for significant neurologic compromise and injury. Intraoperative neurophysiologic monitoring evaluates nervous system integrity by stimulating a series of neurophysiologic signals over the continuum of the surgical procedure. Any deviation from the trend of signals can alert the surgeon of impending neurovascular damage. Although it is considered a standard for many spinal procedures and despite its prospective prognostic advantage, there is an ongoing debate regarding the benefits of routine IONM with ACDFs considering its already low complication rate.[1] Currently, only 6.9% of ACDF procedures in the United States are performed with IONM.[2,3] For those anesthetics, a significant implication is the tailoring of anesthetic technique to be compatible and not interfere with clinically useful IONM.

Multimodal IONM simultaneously utilizes SSEPs, MEPs, EEG, and/or other modalities to monitor the parallel, but separate ascending and descending spinal pathways.[2] The combination of SSEPs and MEPs decreases the risk of paraplegia by 60% compared to historical methods such as wake up tests.[4] Somatosensory evoked potentials monitor dorsal column (sensory) integrity with peripheral stimulation below the surgical site and measurement of the subsequent filtered neurotransmission to the brain. A limitation of SSEPs is the inability to monitor the anterolateral spinal cord; therefore, motor deficits may occur without any trend changes in SSEP signaling.[5] Motor evoked potentials monitor anterior corticospinal tract (motor) integrity with either transcranial or direct spinal cord stimulation above the surgical site and measurement of consequent motor response in the peripherally innervated muscles. Controversy exists regarding the accuracy of MEPs, but multi-channel MEPs, which monitor at least 8 or more muscles,

enhance sensitivity and increase detection of motor deficits.[3] Electroencephalography assesses depth of anesthesia and cerebral perfusion by measuring the summation of excitatory and inhibitory neurotransmissions in the cerebral cortex.[3]

A 50% decrease in amplitude and/or a 10% increase in latency of evoked potential signals suggests ischemia.[3,5] However, these changes can also be attributed to fluctuations in anesthetic depth, hypoxia, hypercarbia, hypotension, hypothermia or equipment malfunction.[5] Consequently, it is essential to maintain a consistent anesthetic depth and regulate hemodynamics with non-anesthetic agents to prevent ambiguity and misinterpretation of the source of waveform changes.[4] Electroencephalography tracings assist in anesthetic agent titration to sustain theta and delta wave activity indicative of general anesthesia and to avoid burst suppression. Frequent, clear communication with the electroneurodiagnostic technician is crucial for appropriate anesthetic titration and signal interpretation.

Volatile anesthetics cause a dose-dependent decrease in amplitude and increase in latency that can mimic ischemic changes. Researchers have reported that a minimal alveolar concentration of 0.5 or less of volatile agent permits adequate IONM conditions, but anesthetic depth must be supplemented with other anesthetic agents to reach a therapeutic level for surgery.[5] Propofol causes a dose-dependent decrease in amplitude and increase in latency, but to a lesser extent than volatile agents. When anesthetic depth significantly interferes with SSEP or MEP signal transmission, the volatile agent can be discontinued and a total intravenous anesthetic technique implemented with minimal alteration of the neurotransmissions.[5] Opioids only marginally decrease amplitude and increase latency. Propofol and remifentanil infusions are valued for their short context-sensitive half-life, which allows for rapid emergence and early postoperative neurological assessment. Due to the quick offset of analgesic effect, longer-acting analgesic agents should be given prior to the discontinuation of remifentanil. A limitation of remifentanil is the opioid-induced hyperalgesia that increases postoperative analgesic requirements, although there is evidence that this phenomenon is dose-dependent and may be diminished with slow discontinuation.[5] The hyperalgesic effect of remifentanil can be avoided with a sufentanil infusion but because of its longer context-sensitive half-life, sufentanil should be discontinued earlier to avoid prolonged emergence. Neuromuscular blocking (NMB) agents prevent signal transmission during transcranial MEP stimulation; however, direct spinal cord MEP stimulation is unaffected by NMBs and muscle relaxation is even recommended to prevent gross motor movements.[4]

Despite the low complication rate of ACDFs, the gravity of potential neurological deficits might warrant a standard of neuronal integrity monitoring. Multimodal IONM with SSEPs and multi-channel MEPs provides greater sensitivity to ischemia than either modality alone and significantly decreases the risk of postoperative neurologic deficits and complications. For IONM to be clinically useful, anesthetic techniques must be carefully modified to reduce interference and provide a consistent anesthetic depth to ensure validity of signals.

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Foreign Body Stapling during Roux-en-Y Gastric Bypass Anastomosis

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Keywords: anastomosis, laparoscopic Roux-en-Y gastric bypass, complication, bariatric

Laparoscopic Roux-en-Y gastric bypass (LRYGB) is the gold standard in bariatric surgery and accounts for 70% of all bariatric procedures worldwide.[1] With obesity rates rising, LRYGB operations are increasing and education about avoidable complications is necessary. Stapling of a foreign body into the anastomotic line is a potentially fatal complication and often goes unreported.[2-4] This complication increases intraoperative time, length of hospital stay, and risks of postoperative complications.[2,3] This case report is an attempt to educate the anesthesia practitioner about this complication, propose solutions, and help to minimize its occurrence.

Case Report

A 67-year-old, 105 kg, 157 cm female with a history of morbid obesity presented for LRYGB. Pre-operative evaluation revealed hypertension, obstructive sleep apnea, dyslipidemia, gastroesophageal reflux disorder, hypothyroidism, depression, and anxiety. Surgical history included bilateral total knee arthroplasty, rhinoplasty, breast augmentation, bilateral tubal ligation, colonoscopy, and liposuction. The patient denied anesthetic complications and reported an allergy to lisinopril. Her medication regimen included levothyroxine, bumetanide, oxybutynin, carvedilol, alprazolam, ranitidine, vitamin B-12, calcium citrate, amlodipine, and citalopram.

Upon entering the operating room (OR) the patient was introduced to the OR staff and transferred to the OR table in the supine position. Standard monitors were applied, preoxygenation was administered as was 100 mcg of fentanyl. After preoxygenation, general anesthesia was accomplished with a rapid sequence induction including: 5 mg defasciculating

dose of rocuronium, 100 mg lidocaine, 150 mg Propofol and 120 mg succinylcholine. Atraumatic tracheal intubation was completed with a McGrath laryngoscope (Medtronic, Minneapolis, MN). Additionally, a 16 french orogastric tube (OGT) was placed for evacuation and decompression and a nasopharyngeal temperature probe was inserted. General anesthesia was maintained throughout the case with approximately desflurane 6% expired concentration in O_2 0.5 L/min and air 0.5 L/min.

After surgical exposure of the gastroesophageal junction, the anesthesia practitioner removed the previously placed OGT. Upon surgeon request a gastric-sizing balloon was placed, and inflated with 15 mL of air. While assessing position on the laparoscopy screen, the anesthesia practitioner maneuvered the gastric sizing balloon adjacent to the gastro-esophageal junction. The surgeon used four firings of the Gold Echelon 60 (Ethicon US LLC., Somerville, NJ) stapler around the balloon. The anesthesia practitioner removed the sizing balloon and inserted an OrVil Anvil (Medtronic, Minneapolis, MN) with a circular Peri-Strip (Baxter Healthcare Corporation, DeerField, IL) transorally and extruded it through a gastrotomy on the proximal gastric pouch staple line. With the anvil in place, the tubing was detached, discarded, and the anastomosis was completed.

When the anesthesia practitioner attempted to remove the nasopharyngeal temperature probe (NTP), he encountered resistance to its removal. With further investigation to the cause of this resistance it was noted that the NTP had migrated past the oropharynx. A gastroscope confirmed that during passage of the OrVil Anvil, the NTP had become intertwined and consequently stapled into the gastrojejunal anastomosis. Using the gastroscope and laparoscopy, the team located the site where the NTP was stapled. With a harmonic scalpel, the anastomosis was reopened and the NTP was removed with a small section of the jejunum. In the process of removing the entrapped NTP, approximately 60% of the gastrojejunal anastomosis and 50% of the stomach pouch staple line was opened. The surgical team completed closure of the open anastomotic line with an endo stitch. A leak test was performed, with a leak noted on the posterior aspect of the suture line where it met the staple line. The OGT was removed and sutures were placed to reinforce this area. A second OGT was reinserted and a methylene blue leak test was performed with no leak noted. The OGT was removed and replaced with a nasogastric tube (NGT) to low intermittent suction. The patient emerged from general anesthesia in stable condition, the endotracheal tube was removed, and she was transferred to the post anesthesia care unit.

Discussion

Obesity is a major health concern, with rates of extreme obesity increasing by more than 400% in the last two decades across the United States (U.S.). It is estimated that 64% of adults are overweight and 34% are classified as obese.[5] Furthermore, it is estimated that more than 15 million Americans have a body mass index of 40kg/m^2 or higher.[6] Several studies have shown that the risk of physical disability and early mortality increases with increasing body mass index. The World Health Organization reports that obesity accounts for more than 400,000 deaths yearly, second only to tobacco related disease, as a cause of preventable and premature death.[6] The rise in obesity has led to an increased number of LRYGB operations as treatment, increasing from 16,000 to 103,000 annually in the last 11 years.[3] Postoperative complications from LRYGB

are broadly grouped into early and late complications.[1,4] Early complications occur during the first two postoperative weeks, while late complications arise after the second postoperative week.[1]

The actual incidence of foreign body stapling into the anastomosis during LRYGB is difficult to quantify, as it often goes unreported.[2-4] In a study, Stanford Medical Center reviewed 727 LRYGB cases; 1.2% of patients were identified with the complication of foreign body stapling into the anastomosis.[3] All nine of these cases necessitated anastomotic revision; two of which were converted to open procedures, with one developing an anastomotic leak requiring re-operation and repair.[3] In a multicenter retrospective analysis performed in Israel, eight surgeons reported on 17 cases in which foreign body stapling occurred including eight NGT/OGT, six temperature probes, and three bougies.[2] While this complication occurs in just a small percentage of cases, it is 100% avoidable, increases operative time, length of hospital stay, and need for diagnostic imaging.[3] Lack of recognition intraoperatively can lead to severe complications, including repeat operation, anastomotic leak, anastomotic stricture, and possibly death.[2,3] Anastomotic leak is the strongest independent risk factor for postoperative death and is associated with a mortality rate of nearly 50%.[1,6]

Management of this complication is dependent upon the situation and often includes some form of anastomotic correction, placement of a NGT, possible further diagnostic imaging, and maintaining the patient on an NPO status with lengthened in-hospital monitoring.[2,3] In the case presented, the operative time was extended by three hours, hospital stay was lengthened by two days, and additional postoperative swallow studies were performed. This patient population carries a higher incidence of post-operative complications with any surgical procedure. Due to the increased length of hospital stay, patients incur higher medical costs and a greater risk of postoperative medical complications, including deep vein thrombosis, pulmonary embolism, small bowel obstruction, and myocardial infarction.[1,5] Absolute vigilance on the part of the anesthesia practitioner is recommended to ensure optimal patient outcome and to avoid any incidental complications to provide for the best chance for full recovery.[6]

Current practices require multiple OGTs and operative devices to be introduced and removed throughout the procedure, increasing the anesthesia practitioners responsibilities in these cases. These added responsibilities, make the anesthesia practitioner vulnerable to contributing to iatrogenic complications. Adverse events must lead to education and prevention strategies. Poor communication among the anesthesia and surgical team at times of stapling, lack of proper equipment use for monitoring, patient position, and body habitus all increase the risk of this avoidable complication.[2-4] Studies have shown prevention strategies should include closed loop communication from the surgeon to the anesthesia practitioner when stapling and the use of large bore OGT to aide in visualization by the surgeon.[2,3] Focus should be on methods of prevention of such complications, not early recognition of them.[2] One should employ alternative temperature monitoring probes (i.e. Foley temperature probe) that are impossible to become inadvertently intertwined with operative devices being passed transorally throughout the procedure.[2,3] Implementation of systematic protocols, communication guides, and operative guides can aide the anesthesia and surgical team in prevention strategies and should be available where LRYGB is practiced to ensure optimal patient outcomes and avoid preventable complications.[2]

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Mentor: James Stimpson, DNP, CRNA

Lidocaine Infusion for Abdominal Hysterectomy

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Keywords: intravenous lidocaine, lidocaine infusion, lidocaine for postoperative pain, perioperative pain, multi-modal analgesia

Intravenous (IV) lidocaine has analgesic, antihyperalgesic, and anti-inflammatory properties, making its use beneficial in a multi-modal pain control approach. Its use can decrease opioid requirements, leading to secondary benefits for patients. While evidence does support the use of lidocaine infusions for abdominal surgery, more evidence is needed to show effective dosing and length of administration and benefits with other types of procedures.[1]

Case Report

A 54-year-old female patient presented to the hospital for a total abdominal hysterectomy and bilateral salpingo-oophorectomy with a diagnosis of a complex ovarian cyst suspicious for malignancy. Her past medical history included hypercholesteremia, benign thyroid goiter, osteopenia, and vitamin D deficiency. Her past surgical history included laparoscopic cholecystectomy and appendectomy, tubal ligation, and colonoscopy. Pre-operative labs were within normal limits. After a thorough pre-operative assessment by the anesthesia team (anesthesiologist, nurse anesthetist, and student nurse anesthetist), it was decided that the patient would benefit from a multi-modal pain management approach for this surgery.

Preoperatively, acetaminophen 1000mg, celecoxib 400mg, and pregabalin 150mg were administered by mouth (PO). In route to the operating room (OR), midazolam 2mg IV was given. Standard monitors were applied, preoxygenation was initiated, and general anesthesia was induced using fentanyl 100mcg IV, lidocaine 30mg IV, propofol 120mg IV, and vecuronium 6mg IV. Endotracheal intubation was achieved without difficulty and a lidocaine infusion was started at 2mg/min IV. Prior to surgical incision, dexamethasone 8mg IV and ketamine 20mg IV were administered. Ketamine and fentanyl boluses were given throughout the surgery totaling an additional 30mg IV and 100mcg IV respectively.

Hydromorphone 0.4mg IV was administered during surgical closure and the lidocaine infusion was decreased to 1mg/min. Emergence from anesthesia was uneventful, and extubation criteria was met as the patient was hemodynamically stable, adequately strong with 4 train-of-four twitches and sustained tetany, able to maintain airway reflexes, and spontaneously ventilating with adequate tidal volumes and respirations. The trachea was extubated, and the patient was transferred to the post anesthesia care unit (PACU) with the lidocaine infusion. The initial post-operative assessment revealed that the patient was hemodynamically stable, sedated but easily arousable, and without report of pain from the patient. The lidocaine infusion was discontinued 30 minutes after arrival to the PACU. A patient controlled analgesia (PCA) was initiated upon transfer to the inpatient floor.

Discussion

Perioperative pain occurs due to tissue and nerve injury. This leads to inflammation and release of pro-inflammatory mediators that produce sensitivity to stimuli in both the local/peripheral and central nervous system.[2] A multi-modal pain control approach can be used to target multiple receptors and decrease the need and side effects of narcotics. The use of a multi-modal pain approach should be determined based on the individual patient, type of procedure, and side effects of medications.[3] A lidocaine infusion can be an analgesic adjunct to aid in perioperative pain relief. Because of its analgesic properties, the use of a lidocaine infusion can lower narcotic requirements. Reducing narcotic requirements can accelerate the return of bowel function and reduce postoperative nausea and vomiting (PONV), which potentially decreases length of hospital stay.

Along with its basic analgesic mechanism, lidocaine has been shown to decrease upregulation of pro-inflammatory mediators and induce secretion of anti-inflammatory mediators.[2] Having effect on inflammatory mediators, lidocaine acts as an anti-hyperalgesic and anti-inflammatory agent. Lidocaine's multiple properties act synergistically to decrease perioperative and postoperative pain. In the immediate postoperative period and up to 24 hours post-surgery, patients that received intraoperative lidocaine infusions have been shown to have lower pain scores as opposed to patients that did not receive lidocaine.[1,4] Some studies have shown analgesic benefits up to 48 hours postoperatively.[5] However, throughout the literature, the length of infusion time and dosage of the lidocaine infusion vary widely in studies.[1] The lidocaine infusion in the case presented was started at 2mg/min and then decreased to 1mg/min and continued for 30 minutes postoperatively in the PACU. Due to the lack of consistency and limited evidence, there is no established time period or effective dosage of continuous lidocaine infusions that has been shown to have optimal analgesic benefit.

There are multiple secondary benefits of a lidocaine infusion. Since lidocaine provides analgesia to patients, opioid requirements can be reduced. In a systematic review of the literature by Kranke et al., opioid consumption was significantly reduced in the lidocaine infusion group when compared with the control in 32 trials.[1] Additional benefits include a decrease in PONV and earlier return of bowel function.[1,4] The incidence of PONV has been shown to be lower in the early postoperative period as well as greater than 24 hours postoperatively when a lidocaine infusion is administered.[1] The use of a lidocaine infusion significantly shortens the time to first flatus and bowel movement/sound; however, it does not significantly shorten time to first defecation postoperatively.[1]

In a study by Terkawi et al., patients that received a lidocaine infusion as opposed to epidural anesthesia had a higher opioid consumption postoperatively. However, patients that received lidocaine infusions had decreased incidence of hypotension, lower PONV, earlier urinary catheter removal, and quicker return of gastrointestinal function than patients that received epidural anesthesia.[6] It is not known if the patient in the case presented had any additional secondary benefits from the use of the lidocaine infusion.

While studies have shown that there are multiple benefits from the use of lidocaine infusions, evidence is limited, scarce, and at times conflicting in regards to lidocaine reducing pain and providing secondary benefits.[1,7] Evidence points to lidocaine infusions having positive outcomes for abdominal surgery, whereas its beneficial use in other types of surgery varies .[1,5] For example, in a study of patients undergoing posterior spinal arthrodesis, patients receiving lidocaine infusions did not have improvement of postoperative morphine consumption, PONV, or return of intestinal function.[8] Also, the evidence lacks reports of adverse outcomes of lidocaine infusions as well as optimal dosing and length of administration.[1]

Research indicates that lidocaine infusions have positive outcomes for patients who have had abdominal surgery, however, more studies are necessary to understand its effectiveness, dosing, length of administration, and clinical side effects. With a current focus on identifying multi-modal methods to reduce opioid use, lidocaine infusions are an adjunct with multiple benefits and minimal side effects for patients undergoing abdominal surgery.

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Prolonged Peripheral Nerve Blockade in a Patient with Preexisting Neuropathy

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Keywords: peripheral nerve blockade, peripheral nerve injury, prolonged nerve blockade, neuropathy, popliteal nerve block, adductor canal block

Peripheral nerve blocks (PNB) are useful tools in modern-day anesthesia and analgesia. Nerve stimulation and ultrasound imaging have improved the safety profile of PNBs. PNBs decrease intraoperative anesthetic and opioid requirements, which potentially leads to fewer symptoms commonly associated with anesthetics including postoperative nausea and vomiting (PONV) and hemodynamic instability.[1] The increased safety provided by technologic advances and decreased pharmacologic requirements make PNBs an attractive option for ambulatory procedures and patients with preexisting comorbidities. However, these procedures are not devoid of risk, as complications such as nerve injury, infection, and local anesthetic toxicity are possible.[2]

Case Report

A 52-year-old, 74 kg, 64 in female with left foot metatarsalgia and hallux valgus presented for a left foot bunionectomy, first metatarsal osteotomy with internal fixation, second metatarsal capsulorrhaphy, and removal of fifth metatarsal hardware. Her past medical history included hypothyroidism, L4-L5 lumbar disc degeneration with L5 radiculopathy, celiac disease, anxiety, and migraines. The patient endorsed preoperative left lateral calf and foot paresthesias. Her past surgical history included a left foot bunionectomy, L4-L5 lumbar fusion, and a hysterectomy. The patient experienced PONV after all general anesthetics. Medications included levothyroxine, estradiol, calcium carbonate, sumatriptan, diazepam, and ibuprofen. Her reported

allergies were penicillin, morphine, gluten, and bananas. Preoperative white blood cell count was 8,500 cells per microliter.

Preoperatively, standard monitors were attached to the patient. Oxygen was administered via nasal cannula at 2 L/min. Midazolam 1 mg and ketamine 10 mg were administered intravenously (IV) as procedural premedication. The anesthesia practitioner donned head cover, surgical mask, and sterile gloves and cleansed the PNB sites using an up and down scrubbing motion with ChloraPrep (Becton Dickinson and Company, Franklin Lakes, NJ) for 30 seconds prior to block performance. The PNB site was allowed to dry for one minute. 1.5 mL 1% lidocaine was injected subcutaneously to anesthetize the block sites. Popliteal nerve block and adductor canal blocks were performed using 20 mL and 10 mL of 0.5% ropivacaine with 1: 400,000 epinephrine, respectively. PNB effectiveness was confirmed by decreased sensory perception prior to proceeding to the operating room.

Upon arrival to the operating room, standard monitors and oxygen via nasal cannula were reapplied. Midazolam 1 mg was administered IV, and a propofol infusion was initiated at 100 mcg/kg/min and titrated to moderate sedation. Dexamethasone 8 mg was administered IV for PONV and PNB prolongation. The procedure lasted for approximately two hours. The patient tolerated surgical stimulation and did not require opioids during the intraoperative or postoperative periods.

Forty-eight hours after the PNB was placed, the patient (a medical professional) verbally quantified her sensory deficit in the triceps surae muscle region as a 20% decrease from her baseline without motor deficit. A 5% sensory deficit was reported by the patient on postoperative day four. The patient underwent a postoperative exam with the surgeon on postoperative day seven, during which she reported a complete return of sensation. The surgeon noted cellulitis, erythema, and vesicles to the operative foot. Postoperative white blood cell count was 15.3 cells per microliter. The patient was prescribed Clindamycin 300 mg orally four times a day for postoperative infection. Resolution of the symptoms related to postoperative infection was noted 12 days postoperatively.

Discussion

Peripheral nerve injury (PNI) occurs in approximately 0.5-1% of PNBs.[2] Nerve injury can be transient or permanent, with the clinical manifestations of injury dictating the severity of the injury. “Because the primary determinant of prognosis is the residual integrity of the axons, PNI severity is typically classified according to the relative degree of axonal disruption.”[3] Proximal axonal lesions are deemed more severe than distal lesions based on the reinnervation and recovery is more likely to occur in nerves with the least distance between the area of axonal injury and the target tissue.[3] PNIs can are further classified using the Seddon anatomical scale, which categorizes nerve injuries as either a neuropraxia, axonotmesis, or a neurotmesis. A neuropraxia, which is the mildest and most common anatomical injury, involves damage to the myelin sheath without subsequent damage to the axon caused by stretching or compression.[3] Prognosis is good and recovery occurs within weeks to months.[3] An axonotmesis is an injury associated with axonal discontinuity amid an intact endoneurium and is caused by fascicular impalement, nerve crush injury, or toxic injury.[3] Recovery from an axonotmesis injury can take

months to a year, with the length of recovery depending upon the relationship between the distance of the injury and the corresponding muscle.[3] A neurotmesis involves a complete transection of the axon, endometrium, perineurium and connective tissue that classically requires surgical intervention and has a poor prognosis for function recovery.[3]

Preexisting neuropathy theoretically increases the predisposition to PNIs and prolonged sensory blockade following PNBs.[2,3] Patients with preoperative entrapment neuropathies, particularly carpel syndrome, brachial plexopathy, and ulnar neuropathy syndrome demonstrate a higher risk of developing a PNI post-PNB.[4] Lumbar stenosis causes nerve impingement and subsequent neuropathy via a similar mechanism, thus it is reasonable to assume that preexisting neuropathies caused by lumbar stenosis also leads to a higher risk of post-PNB neural dysfunction. However, the etiology of this increased risk is not well defined in current literature.[2,3]

Another possible causative agent of PNI and prolonged peripheral nerve sensory blockade is local anesthetic adjuvants. Regional anesthesia with local anesthetics (LA) can provide superb postoperative analgesia, but in their standard preparations local LAs have very limited durations of action. Adjuvants such as epinephrine and dexamethasone can be utilized in conjunction with LAs to prolong their duration of action. Epinephrine, a potent alpha- and beta-agonist, prolongs LA duration of action by producing vasoconstriction and decreasing systemic uptake of LA.[2,3] While this mechanism of action allows LA to remain in the nerve sheath for extended periods, it can also decrease blood flow to the nerve and local tissues.[2] Dexamethasone, when either added directly to ropivacaine and injected perineurally or given intravascularly, can prolong the duration of sensory blockade by as many as 11 hours.[5] While dexamethasone's exact role in the prolongation of PNB is unknown, one conceivable theory is that the glucocorticoid's inherent vasoconstrictive properties increase the LAs duration of action by decreasing systemic absorption.[5] Another plausible theory is that dexamethasone acts via the glucorticoid receptors on nociceptive c-fibers to increase the activity of inhibitory potassium channels on the fibers, resulting in a decrease in c-fiber activity.[5] Once injected perineurally, dexamethasone is eventually absorbed into systemic circulation and continues to exert its analgesic effects after the sensory blockade provided by LA ceases.[5]

Postsurgical infection cannot be discounted as another potential causative agent of prolonged PNB. Current literature clearly demonstrates that injection of LA into actively infected tissue results in slower onset due to a decreased extracellular base-to-acid ratio.[6] However, the consequences regarding infection that develops after LA has been injected are not well defined. Inflammation can result in compression neuropathy and trigger the inflammatory-immune response to inappropriately target peripheral nerves.[3] Thus, it is conceivable that this patient's transient neuropathy with sciatic distribution was either caused or exacerbated by surgical site inflammation. Given that the postoperative infection developed in the patient's foot and the LA injection sites were in the lateral thigh and popliteal regions, it is most likely that the distal portion of femoral and sciatic nerves were affected by surgical-site infection and edema.

Peripheral nerve block placement for this patient was uneventful using ultrasound guidance. The patient did not endorse paresthesias with needle placement or LA injection, making nerve injury via neural puncture or mechanical nerve compression unlikely. She also previously had a PNB for a similar procedure on the same extremity with no block-associated complications. These

factors, coupled with the relatively low incidence of PNI in patients with baseline entrapment neuropathies, make it unlikely that the prolonged blockade was due to her preexisting radiculopathy. Though the patient's predisposition to PNI presented only relative contraindications to PNB, it reasonably justified the avoidance of block-prolonging agents. For this case, outcomes were favorable, but displays the importance of thorough evaluation of baseline neuropathies prior to PNB to predict and prevent PNI.

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Pneumothorax Following Implantable Venous Access Port Placement

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Keywords: port catheters, port-a-cath, vascular access ports, vascular access devices, pneumothorax

Implantable venous access ports (IAVP) are surgically placed to provide long-term intravenous therapy. These ports enter the vasculature at the left or right subclavian vein, very near to either lung, and unintended puncture of the lung's parietal pleura is known to occur in approximately 3% of these procedures.[1] Puncture of the lung can cause air to move into the potential space between the visceral and parietal pleurae, resulting in respiratory distress, tension pneumothorax, or death. Being aware of the potential for pneumothorax during IVAP placement, anesthesia practitioners should be able to quickly identify and effectively treat this infrequent but serious complication.

Case Report

A 44-year-old female presented to outpatient surgery for placement of an IVAP for chemotherapy. She had received a diagnosis of metastatic colon cancer two weeks prior to this procedure following an exploratory laparotomy. Medical history was significant for colon cancer with metastasis to the liver. The patient was taking the following medications: oxycodone hydrochloride, ibuprofen, docusate calcium, daily multivitamin, and probiotics. She denied having any allergies to medications, food, or latex. Her surgical history was significant for left knee surgery, tonsillectomy and hemicolectomy with colostomy. She denied having complications with previous anesthetic administration, and she also denied any family history of anesthetic related problems. A complete blood count and a complete metabolic panel were performed prior to surgery, and all values were within normal limits. Her airway examination was normal, breath sounds were normal to auscultation, and she had normal heart sounds.

An 18-gauge peripheral intravenous (IV) catheter was inserted into the patient's right hand, and a continuous infusion of lactated ringers was initiated. Midazolam 2 mg IV was administered in the preoperative holding area. After arriving in the main operating room, standard non-invasive monitors (blood pressure, SpO_2, and 3-lead ECG) were applied. Pre-oxygenation and de-nitrogenation was achieved O_2 10 L/min via facemask for an expired O_2 concentration of approximately 88%. General anesthesia was induced with lidocaine 100 mg and propofol 170 mg IV. Once the patient was apneic and demonstrated loss of lid reflex, the eyes were taped and a size four supraglottic airway was inserted. Spontaneous ventilation without pressure support resumed after induction, and 2% sevoflurane was administered for maintenance of anesthesia.

The surgeon prepared the surgical field and began placing the implantable access port utilizing anatomical landmarks. Multiple unsuccessful attempts were made to access the subclavian vein with inadvertent arterial puncture noted on one attempt. Another surgeon arrived in the room to assist with placement and the port was successfully placed under fluoroscopic guidance. Vital signs and respiratory function remained stable throughout the procedure. After successful placement of the IVAP, general anesthesia was discontinued, and the supraglottic airway was removed without incident. The patient demonstrated no shortness of breath, no oxygen desaturation, no tachypnea, and vital signs were within 20% of baseline parameters. The patient was transported to the recovery unit and report was given to the recovery nurse.

Shortly after arriving in the recovery unit, the patient became tachycardic with, left-sided chest pain, difficulty breathing and a decrease in oxygen saturation. The surgeon ordered an immediate chest x-ray that demonstrated a small left-sided pneumothorax. A chest tube was inserted, placed to water seal, and the patient was admitted to the intensive care unit (ICU) for further observation. On hospital day three the chest tube was removed. A repeat chest x-ray on hospital day four showed a significant worsening of the left-sided pneumothorax, and a repeat thoracostomy with chest tube placement was completed. The pneumothorax persisted through postoperative day five, and the medical team arranged for transfer to a higher acuity facility for further medical management. She was discharged home 5 days later on postoperative day 10.

Discussion

Pneumothorax, although uncommon, occurs in approximately 0-6% of all IVAP placements, and it is more likely to occur when the surgeon chooses the subclavian approach.[2] The incidence of pneumothorax also increases significantly if the surgeon fails to access the subclavian vein on the first attempt and has to make multiple passes. One study demonstrated that mechanical complications related to subclavian access increased from 1.6% on the first pass, to 43.2% with three or more attempts.[3] Although studies have demonstrated that ultrasound or fluoroscopic guidance may substantially decrease the risk for pneumothorax during IVAP placement, the routine use of such adjuncts has not been universally adopted.[2] The anesthesia practitioner must be aware that pneumothorax is a risk associated with IVAP procedures, and he/she must be able to identify the symptoms when puncture is suspected.

Small pneumothoraxes may resolve without intervention, but early recognition of associated signs and symptoms can prevent the development of a life-threatening tension pneumothorax. Bacon et al., 2005 described the role of various monitors, and clinical observations in detection of pneumothorax. Pulse oximetry, blood pressure, and pulse were considered non-specific monitors, as most patients will not demonstrate immediate changes in vital signs. However, if the pneumothorax is large, desaturation, hypotension and heart rate changes are likely to occur. Clinical observations described by Bacon and colleagues included: unilateral chest wall expansion, abdominal distension, distended neck veins, tracheal deviation, and absence of breath sounds on the affected side. Identification of these clinical signs may be difficult due to surgical draping, and again, they may not be present in smaller pneumothoraces.[4] Anesthesia practitioners should have a high suspicion of pneumothorax if any of the clinical signs or symptoms develop during IVAP placement, and immediate action should be taken to prevent further injury.

Treatment of pneumothorax varies with the severity of lung collapse. Smaller pneumothoraxes may resolve spontaneously and may only require avoidance of positive pressure ventilation and administration of supplemental oxygen to hasten recovery.[2] A larger, symptomatic, pneumothorax will require emergency actions and surgical intervention to prevent patient deterioration. Initial treatment involves simple aspiration by placing a 16-guage needle into the second intercostal space at the mid-clavicular line.[5] If aspiration is unsuccessful or reoccurrence of pneumothorax is likely, a tube thoracostomy should be performed. A surgical incision is made at the fifth intercostal space at the mid-axillary line, and a chest tube is inserted into the pleural space. The chest tube is then attached to a water-seal drainage system (suction may be applied to the system after the first 24 hours) in order to remove all air and allow the lung to expand.[5] The patient should be admitted for observation, and serial radiographic studies should be completed to confirm resolution of pneumothorax.

In the case presented, the patient did not demonstrate signs or symptoms of a pneumothorax until she arrived in the recovery unit. However, the anesthesia professional's suspicion for pneumothorax should have been piqued due to the surgical approach and the multiple failed attempts. Fortunately, recovery unit nursing staff and the surgeon were able to recognize that the patient was experiencing a respiratory event, and appropriate action was taken to treat her collapsing lung. In future cases, the anesthesia practitioner may recommend the use of

fluoroscopy or ultrasound to assist with IVAP placement, as this technique has been shown to greatly reduce the incidence of pneumothorax.[6]

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Management of Congenital Heart Disease for Non-Cardiac Surgery

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The anesthetic considerations for small infants with congenital heart disease, in various stages of surgical palliation, who present for non-cardiac surgery can be complex. A thorough understanding of the post-surgical anatomy of the heart is essential to properly augment the systemic and pulmonary circulation. These post-surgical changes can also have effects on hemodynamic stability such as blood pressure, volume management and oximetry. This case report describes refractory hypotension in an infant with altered congenital and post-surgical systemic blood flow. The understanding of how these alterations can affect blood pressure and blood pressure monitoring proved to be important in the anesthetic management of this patient.

Case Report

An 8-month old female weighing 7.1 kg with a past medical history of hypoplastic left heart syndrome (HLHS), atrial flutter, hydronephrosis, recurrent urinary tract infection (UTI), and

gastro-jejunostomy (G-J) tube dependence presented for embolization of a leaking thoracic duct causing chylothorax. The patient was status-post stage 1 Norwood procedure and stage 2 bi-directional Glenn shunt procedure. The echocardiogram obtained the week prior showed trivial tricuspid regurgitation, pulmonic valve gradient of 6 mm Hg and "good right ventricle function" without numeric estimation. Kidney function was within normal limits. She presented from the intensive care unit (ICU) with tachypnea and labored breathing on O_2 2L NC with an SpO_2 of 92%. She underwent the same procedure a week prior but it was aborted after the surgical team was unable to gain access to the thoracic duct. There were no major anesthetic complications and the patient was intubated using a Wis-Hipple 1 laryngoscope blade and had a Cormack-Lehane Grade I. The patient was assessed pre-operatively by cardiac anesthesia who recommended IV hydration and normocapnia.

The anesthetic plan for the patient was a combination inhaled sevoflurane and IV induction as the patient presented with a left saphenous peripherally inserted central catheter (PICC) line. The patient was induced with 10 mg IV ketamine, 5 mcg Fentanyl and 0.7 mg of vecuronium in combination with inhaled sevoflurane. The trachea was intubated with a 3.5 mm cuffed ETT and sevoflurane was delivered as anesthesia maintenance. The patient's mean arterial pressure (MAP) decreased to 32 mm Hg after induction. The patient was treated with a 50 mL IV bolus of isotonic crystalloids with minimal effect, expired sevoflurane concentration was titrated to 0.8% and intermittent boluses of 2.5 mg to 5 mg of ephedrine were given. The patient was given several more fluid boluses totaling 150 mL. A dopamine infusion was started at 5 mcg/kg/min and quickly titrated to 10mcg/kg/min with minimal effect. Surgical stimulation raised the MAP to 40-45 mm Hg. After the procedure started, the patient became hypotensive again and the decision was made to place an arterial line. The procedure was interrupted while the blood pressure cuff was changed to the left arm to place and an arterial line was inserted into the right radial artery under ultrasound guidance. The initial left arm blood pressure was elevated with a MAP of 80 mm Hg and the arterial line was aborted.

After the patient became hypotensive, a focus was placed on delivering IV fluids and subsequently inotropes such as dopamine and ephedrine. The anesthesia team was unaware that this was an inaccurate blood pressure reading. Sevoflurane was reduced and the patient became more tachycardic. After moving the blood pressure cuff we searched for an explanation. The patient's initial Stage 1 Norwood procedure operative note was closely examined. According to the report, the patient presented with an aberrant right subclavian artery. The aberrant artery was divided and a common-carotid – subclavian bypass graft was created during the first operation. This surgical intervention and the unique flow changes to the right upper extremity helps explain the unreliable non-invasive blood pressure measurement.

The differential diagnosis for hypotension in this patient included right ventricle (RV) failure, hypovolemia, cannulation of the aorta and anesthetic drug factors. Preload in the form of isotonic IV fluids were administered and contractility was augmented with inotropes. The patient was paralyzed and anesthetic gases were reduced. After these interventions, a new monitor of blood pressure was discussed and planned, resulting in an accurate blood pressure measurement. The surgical team was again unable to cannulate the thoracic duct and the procedure was eventually aborted.

Discussion

Hypoplastic Left Heart Syndrome (HLHS) is a severe congenital heart disease that presents as a spectrum of disorders. The cause for HLHS is unknown and there are no known risk factors for the disease. The syndrome presents as an underdeveloped left ventricle, aorta, aortic arch as well as mitral atresia or stenosis. The incidence of HLHS has been reported to occur in approximately 0.016 to 0.036% of all live births.[1] The primary diagnostic tool is fetal echocardiogram and can been seen between 18 and 22 weeks' gestation. Children who suffer from the disease require surgery as neonates as part of a various staged palliation process. Systemic perfusion relies on a patent ductus arteriosis (PDA) and pulmonary flow relies on an atrial-septal defect (ASD). As the PDA closes over about 11 days, the babies decompensate. The Stage 1 Norwood involves an atrial septectomy, connecting the right ventricle (RV) to aorta with an aortic-pulmonary shunt to provide pulmonary flow while pulmonary vascular resistance is high. After pulmonary vascular resistance falls, the aorto-pulmonary shunt is removed and the SVC is connected to the main pulmonary artery (PA) with a bidirectional shunt, known as a Glenn procedure. The Fontan Procedure is the final in the series and it involves connecting the inferior vena cava to the PA allowing the lungs to receive 100% of the venous return.

An aberrant right subclavian artery (arteria lusoria) is the most common aortic arch anomaly with an incidence between 0.5-2.5%.[2] The patients are usually asymptomatic and present for evaluation for unrelated medical problems. The most common symptom of arteria lusoria is dysphagia, an asymptomatic disease which often does not require surgical intervention. Various surgical approaches have been described in the literature. The restoration of flow to the right subclavian artery can be accomplished with a graft or with direct translocation to the right common carotid artery.[3]

Kranenberg et al. performed a cross-sectional study examining patients with vascular disease and inter-arm blood pressure discrepancies. A cohort of 7344 patients with vascular disease or risk thereof who had a large inter-arm systolic blood pressure difference was examined for determinants and future risk. The average inter-arm systolic difference was 7m Hg and 16% had an inter-arm systolic difference of greater than 15m Hg. The study found that higher age, higher systolic blood pressure, diabetes mellitus, peripheral artery disease, and carotid artery stenosis were related to large (>15m Hg) inter-arm systolic pressure differences. The results of the analysis showed that for every 5m Hg increase in inter-arm systolic BP resulted in a 12% higher risk of vascular events in patients without manifest vascular disease.[4] The random placement of invasive monitoring such as arterial lines can lead to a risk of clinical error. The blood pressure should be measured with flow-directed fluid optimization systems in the arm that is most likely to reflect systemic blood pressure.[5] The selection the significantly lower pressure extremity can lead to end-organ damage and the data recommends monitoring pressure in the higher reading extremity.[6] It is important to document any inter-arm blood pressure discrepancy in the pre-operative clinical area and to follow-up with primary care physicians to manage the long-term risks associated with the finding.

The patient's post-surgical anatomy played a pivotal role in the accuracy of blood pressure measurement. Planning for a case such as this in an infant with congenital heart abnormalities warrants a focused surgical history including any shunting or re-routing of vessels because of the

pathology. In this case, previous surgical interventions had a profound impact on an essential anesthetic monitor.

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Awake Craniotomy for Intracranial Tumor Resection

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Keywords: awake craniotomy, conscious sedation, neuroanesthesia, cortical mapping, intracranial tumor

Awake craniotomy (AC) can preserve and assess a patient's cognitive and motor functions intraoperatively necessary for cortical mapping and surgical planning. With the continued development of neurosurgical technology and physiologic maintenance, AC has become an increasingly common tool for lesions located close to functional regions.[1] Mapping in these regions can help assess risk associated with the extensive tissue resection to preserve eloquent cortex.[2] The following report reveals anesthetic challenges of an awake craniotomy, as the level of sedation and analgesia must be carefully balanced with respiratory and hemodynamic stability in a patient who must remain cooperative for intraoperative neurological assessments.

Case Report

A 64-year-old, 185 cm, 77 kg, left hand dominant male presented for right sided craniotomy for tumor resection with language and motor mapping. Past medical history was significant for hypertension, hyperlipidemia, diverticulitis, arthralgia of lower extremities, non-small cell lung

cancer that metastasized to the liver and brain, complicated by pulmonary embolism and pericardial effusion with tamponade, both of which he had been treated and had recovered. Home medications comprised of acetaminophen, dexamethasone, gabapentin, dilaudid, levetiracetam, lisinopril, metoprolol, ranitidine, and sertraline. Allergies included levofloxacin and simvastatin. Past surgical history included a rotator cuff repair with no reported anesthetic complications. Serum laboratory values were normal. Vital signs were normal (BP 126/75 mm Hg). MRI revealed a 4 cm temporal-parietal mass with surrounding edema. Physical examination revealed left sided facial droop as well as left upper extremity and left lower extremity weakness. The proposed procedure endeavored to improve the patient's quality of life and was therefore palliative in nature.

Following informed consent, a 22-gauge peripheral intravenous (PIV) cannulation was performed and a sodium chloride 0.9% infusion started. Ondansetron 4 mg and midazolam 1 mg were administered prior to meperidine 12.5 mg. The patient was taken to the operating suite where standard noninvasive monitoring was applied. Oxygen 3 L/min was administered via nasal cannula. Dexmedetomidine bolus of 1 mcg/kg was administered over 10 minutes followed by a continuous infusion at 0.5 mcg/kg/hr for maintenance of sedation. A 16-gauge PIV was inserted. Following subcutaneous injection of 1% lidocaine, a left radial arterial line was inserted. Additional doses of meperidine totaling 37.5 mg and propofol 20 mg were administered, in addition to local infiltration of 2% lidocaine by the surgeon, for placement of Mayfield pins. Intravenous levetiracetam 1500 mg and cefazolin 2000 mg were administered as the patient was positioned in a slight left lateral decubitus position with careful attention to pad pressure points and secure extremities.

A total of labetalol 25 mg and hydralazine 10 mg was administered for systolic blood pressures (SBP) sustained in the 150-159 mm Hg range, with a SBP reduction to 130 mm Hg. A dexmedetomidine infusion was continued until language and motor mapping interventions, when the infusion was turned off. At that point, the patient was fully conscious and following instructions. The patient was asked to perform several language tasks which included identifying pictures of objects, repeating phrases, and answering yes/no questions, which were done without difficulty. However, during this phase of the operation, seizure activity was noted three separate times on electroencephalography, with no apparent changes in the patient's consciousness or physical state beyond a slight delay in language response. Cold saline was immediately placed on the cortex with each episode of seizure activity. One additional dose of levetiracetam 500 mg was administered. When language and motor mapping were complete, the tumor was resected and the patient was then re-sedated with low dose dexmedetomidine 0.3 mcg/kg/hr until the conclusion of the procedure. Ofirmev 1000 mg was administered for mild headache. Vital signs remained stable throughout the procedure. Postoperative neurological exam was consistent with baseline exam. The patient was taken to the intensive care unit for close monitoring and was discharged from hospital on his second post-operative day.

Discussion

AC was indicated because of the tumor location in the temporal-parietal junction. This required neurocognitive mapping to ensure cognitive functions were spared following resection. Preservation of motor and language capabilities was particularly important, as the patient is left

handed and had experienced left extremity weakness as well as left facial droop secondary to vasogenic edema from the tumor. Real time mapping involves application of electrical stimulation to the cortical regions responsible for language.[1] Broca's area, located in Brodmann's area 44, is responsible for primary motor and expressive language,[2] while Wernicke's area, located in Brodmann's area 22, is responsible for receptive language.[2] While performing real time mapping, two possible responses may result from stimulation: a 'positive response' where brain function is induced by electrical stimulation, or a 'negative response' where brain function is suppressed by stimulation.[1] In between high frequency cortical stimulations (50-60 Hz)[1], language tasks are assessed, including: object identification, repetition of phrases, and auditory comprehension. The technique allows for determination of tumor borders resulting in a precise removal of the lesion and not healthy brain tissue. At times in cortical mapping, the patient experienced a language delay, categorized as a 'negative response' which identified a cortical region to avoid during surgical excision.

AC may be performed by using one of three anesthetic techniques: the asleep-awake-asleep (AAA) method, moderate sedation, or the awake-awake-awake approach. AAA involves general anesthesia (GA) prior to and following brain mapping.[3] Moderate sedation may be achieved with any combination of: propofol, dexmedetomidine, and opioid infusions. For this case, we chose moderate sedation using a dexmedetomidine infusion with small boluses of meperidine and propofol for stimulating portions of the operation. Compared to craniotomies performed under GA, procedures with dexmedetomidine and propofol have had better hemodynamic reactions to pinning and incision, less narcotic use, and decreased intraoperative vasopressor use.[3,5] In addition, AC provides a better neurological outcome and a shorter hospital length of stay when compared to GA.[8] The awake-awake-awake approach involves selectively blocking the sensory branches of the trigeminal nerve.[3] Careful patient selection is required, as no sedatives are administered.

Dexmedetomidine is a highly selective alpha-2 adrenoreceptor agonist and produces sedation by decreasing sympathetic nervous system activity and decreasing the level of arousal.[3] The result is dose dependent sedation, anxiolytic effects, and analgesia while preserving respiratory stability[5] in a patient who can be easily aroused to full consciousness.[3] The pharmacokinetics and pharmacodynamics of dexmeditomidine make it well-suited for AC.[7] Anesthesia publications describe dexmedetomidine used independently, as well as with continuous propofol and/or short acting opioid infusions. Exercise caution if using both dexmedetomidine and remifentanil infusions as severe bradycardia and cardiac arrest may occur.[3]

Hemodynamic stability is critical throughout AC. Maintenance of a mean arterial pressure (MAP) to produce adequate cerebral perfusion pressure (CPP) must be balanced with a MAP that will increase cerebral blood flow to the surgical site and increase bleeding risk. To abate fluctuations in blood pressure during exceedingly stimulating portions of the case, administer agents in doses that will allow maintenance of spontaneous ventilation whilst providing adequate analgesia or sedation. In the present case, meperidine 12.5 mg doses as well as propofol 20 mg and beta adrenergic antagonists (labetalol and esmolol) maintained respiratory status, however, a sustained SBP of 155 mmHg indicated that the patient required additional analgesia. A low dose remifentanil infusion at 0.02 – 0.05 mgc/kg/min has been used successfully in previous reports and could have been considered for this case. Remifentanil's short context-sensitive half-life,

even after prolonged infusion, and its ability to maintain spontaneous respirations make it ideally suited agent for AC.[3,4]

Seizure is another common complication observed during AC.[5] The incidence of seizures during the intraoperative period is 3-10%. Most seizures occur with cortical stimulation, at which point immediate cessation of stimulation and direct cortical irrigation with cold saline solution should occur.[5] Careful selection of preoperative and intraoperative drugs must consider the effect of the central nervous system. In this case, meperidine was employed for premedication as well as during stimulating portions of the procedure. Meperidine is a synthetic opioid agonist on the mu and kappa opioid receptors and provides perioperative analgesia, euphoria, dysphoria, and sedation. However, at high doses, the active metabolite, normeperidine, may accumulate in the plasma and produce excitation in the central nervous system that can result in a range of effects from irritability to agitation to seizure activity.[6]

Technological advancements in neurosurgery have made AC a growing technique that allows for preservation of eloquent cortex. AC can be performed safely and successfully with careful planning, effective communication with the neurosurgical team, appropriate patient selection, and thorough patient education.

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Enhanced Recovery after Surgery and Opioid-sparing Techniques

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Keywords: ERAS, enhanced recovery, opioid-sparing, anesthesia, colorectal

Enhanced recovery after surgery (ERAS) encompasses multidisciplinary pathways implemented by healthcare teams with a goal of decreasing a patients response to surgical stress, maintaining their baseline physiologic function, and accelerating surgical recovery.[1,2] The routine opioid use and misuse have led to an increase in morbidity, mortality, along with delaying surgical recovery.[1] With the recent success of ERAS pathways, the use of opioid-sparing techniques have quickly gained popularity among anesthesia professionals. Therefore, familiarization with current guidelines utilizing opioid-sparing and ERAS techniques is beneficial when considering successful patient outcomes with enhanced recovery.

Case Report

A 44-year-old, 67 kg, 157 cm, Hispanic female was admitted for stricture of her sigmoid colon and a submucosal endometrial tumor of the proximal sigmoid. A sigmoidectomy, ileoscolostomy, loop ileostomy, colovaginal fistula repair, and reversal of loop ileostomy were planned. Her past medical history included sigmoid colon stenosis from a pelvic mass, mucous fistula, rectovaginal fistula, & hypertension. Her surgical history included cesarean section, hernia repair, cystoscopy with urethral stent placement, colonoscopy, exploratory laparotomy, oophorectomy, and colostomy placement. Her current medications include medroxyprogesterone 5 mg, benazepril 40 mg, and hydrocodone-acetaminophen 5-325 mg. She reports using hydrocodone-acetaminophen 5-325 mg three to four times daily as needed.

Preoperative vital signs, laboratory findings, and physical examination were unremarkable. A preoperative ERAS preparation was not implemented by the healthcare team, however, intraoperative anesthetic opioid-sparing techniques were applied, as further discussed below.

In the operating room, standard monitors were applied. The patient was preoxygenated until the end-tidal O_2 concentration was >80 %. Intravenous induction was initiated using fentanyl 50 mcg, 2% lidocaine 100 mg, propofol 150 mg, and rocuronium 50 mg. Direct laryngoscopy was executed and a grade II view was visualized. A 7.5 mm oral endotracheal tube (ETT) was inserted and secured without incident.

In anticipation of fluid shifts and blood loss, an arterial line and second 18-gauge intravenous catheter were placed. A nasogastric tube was inserted for gastric decompression. Sevoflurane 2% with 2 L/min of O_2 was used during the maintenance phase. Neuromuscular blockade was maintained with rocuronium. An infusion containing ketamine 50 mg, magnesium 1 g, and 2% lidocaine 200 mg in 100 mL of normal saline was initiated at 50 mL/hr for opioid-sparing analgesia.

The surgery lasted 5.5 h and the intraoperative surgical course was uneventful. A total of 2,000 mL of lactated ringers along with 500 mL of 5% albumin was administered. The estimated blood loss was 800 mL. The ketamine, magnesium, and lidocaine infusion was discontinued approximately 30 minutes before surgical closure. Upon conclusion of the surgery, a total of 150 mg ketamine, 2.5 g magnesium, and 600 mg lidocaine was administered.

Bilateral surgical site wound infiltration was administered by the surgical team using 150 mg of 0.25% bupivacaine. The nasogastric tube was removed. Neuromuscular blockade was antagonized with sugammadex 2 mg/kg. After successful extubation, the patient was transitioned to O_2 8 L/min via simple mask and transferred to the post-anesthesia care unit (PACU).

In the PACU, the patient exhibited pain by moaning, restlessness, tachycardia, and hypertension. The patient was given morphine 2 mg x 3 doses over 10 minutes based on a pain rating of 10, 8, and 8 respectively on a 1-10 pain scale. In order to achieve an adequate pain control rating of 3, she required two additional boluses of fentanyl 50 mcg prior to discharge from the PACU. Postoperatively, a fentanyl containing patient-controlled analgesia (PCA) pump was initiated which provided sufficient analgesic control. The patient was subsequently discharged home on postoperative day 8 with oral opioids.

Discussion

This case study demonstrates suboptimal pain control despite implementation of some ERAS techniques. Although ERAS techniques have shown their effectiveness, barriers still exist that hinder their effectiveness; one of them being the use of a multidisciplinary approach. A lack of a multimodal approach to ERAS may have played a role in the patient's increased postoperative analgesic requirement. A consideration must also be made regarding the patient's chronic opioid use, unknown postoperative opioid requirements, and opioid-sparing technique.

A basic ERAS protocol incorporates pre-hospital, preoperative, postoperative, and post-discharge phases. Counseling and education of staff and patients hold significance as ERAS protocols start before and extend beyond the intraoperative period. The pre-hospital phase identifies realistic expectations for the patient and resolves fear and anxiety associated with the surgery.[3] With a goal of preoperative optimization, identification of existing deconditioning issues such as anemia, malnutrition, hypertension, diabetes mellitus (DM), smoking, and alcohol consumption help decrease morbidity and mortality.[3]

The patient in this case study began fasting the midnight priot to her surgery. Historically preoperative fasting is thought to decrease the incidence of pulmonary aspiration during surgery. The intention is to reduce gastric content and increase gastric pH. However, there is strong evidence that fasting from solid food for ≥6 h and clear liquids up to 2 h before surgery has the same benefit as fasting after midnight.[3] In order to maintain a patient's baseline physiologic state perioperatively, ERAS protocols include providing clear fluid containing complex carbohydrates up until 2 h before surgery. This recommendation decreases a patient's thirst, hunger, and anxiety preoperatively and decreases insulin resistance postoperatively.[3]

The use of mechanical bowel preparation (MBP) preoperatively should be avoided in the majority of bowel surgeries and have been shown to have no clinical benefit. This is especially the case with the elderly, as MBP is associated with dehydration and electrolyte disturbances.[3] The patient in this case study received a MBP and may have benefited from omission of a MBP.

The use of a multimodal analgesic approach with long acting analgesics such as acetaminophen, gabapentin, and pregabalin preoperatively have shown to decrease postoperative opioid use.[1] In this case report, an established ERAS protocol was not yet established at the facility and therefore the use of preoperative multimodal analgesics were not employed or discussed with the surgical team.

Intravenous lidocaine is beneficial due to its analgesic and anti-inflammatory properties. An induction dose of 1.5 mg/kg and infusion of 2 mg/kg/h follow current guidelines.[3,4] The patient received suboptimal lidocaine dosing. The recommended ketamine dosing includes a bolus dose of 0.5 mg/kg then a 0.5 mcg/kg/h infusion.[5] A loading dose of ketamine was not administered, and the infusion dosing was likely inadequate. Magnesium dosing was within recommended infusion dosing range of 6 mg/kg/hr. Although, a recommended bolus dose of 30 mg/kg was not administered.[6]

The appropriate amount of intraoperative fluid is debatable. What is known is excessive fluid administration is associated with negative outcomes.[3] The anesthesia professionals in this case study chose to administer conservative fluid resuscitation with crystalloids and colloids. Although the best indicator for adequate O_2 delivery and hemodynamic status is awareness of the patient's cardiac output.[3] It is unclear whether optimal cardiac output was achieved in this case study as no measurement tool was employed.

Many bowel procedures are performed in the trendelenberg position and place the patient at increased risk of atelectasis. It is important to maintain adequate tidal volumes with reduction of peak pressures. Maintaining an inspired O_2 concentration >80 % decreases the risk of surgical sight infections, nausea, and does not increase the risk of postoperative atelectasis.[3,7] Maintaining perfusion to the splanchnic vasculature may be achieved by monitoring mean arterial pressure (MAP), cardiac output (CO), and the use of vasopressors to avoid hypotension.[3]

The routine use of nasogastric tube insertion for gastric decompression postoperatively is no longer indicated, and it is shown to have association with fever, atelectasis, pneumonia, gastroesophageal reflux, and delayed return to bowel function. Thus, nasogastric tubes should be discontinued before emergence from anesthesia as done in this case study.[3]

Many ERAS strategies are geared towards minimizing the stress response associated with surgery and were not included in the plan of care of this case report. Minimizing perioperative stress can be accomplished using established ERAS protocols. In order to improve patient outcomes, increased focused should be placed on the use of multidisciplinary ERAS pathways. Although there are several effective guidelines pertaining to ERAS management for bowel surgeries, a patient-centered multidisciplinary team approach seems to be an important factor for successful patient recovery.

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Anesthetic Management for patient with Leigh Syndrome

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Keywords: Leigh syndrome, mitochondrial dysfunction, dystonia, malignant hyperthermia, propofol infusion syndrome

Leigh syndrome (LS) is a hereditary and fatal neurodegenerative disease first identified in 1951 by Denis Leigh.[1,2] As one of the most common mitochondrial myopathies, it is defined by three features: mitochondrial dysfunction, a neurodegenerative phenotype and bilateral basal ganglia or brainstem lesions.[1,2] Despite strikingly similar brain changes in all patients, clinical manifestations of LS are heterogeneous and may include dystonia, cardiomegaly, dyspnea, epilepsy and cranial nerve palsies.[1] This case report addresses the physiologic alterations and complex perioperative scenario that accompanies LS so that anesthesia practitioners may deliver optimal care to these patients.

Case Report

An 18.6 kg, 128 cm, 20-year-old female with an allergy to adhesive tape presented for a tracheostomy tube change, removal of tracheal granulation tissue and tracheal bronchoscopy due to respiratory failure. Her past medical history was significant for cerebral palsy, Wolff-Parkinson-White syndrome, intractable juvenile myoclonic seizures with epilepsy, cardiomyopathy, bicuspid aortic valve with aortic stenosis, and severe kyphoscoliosis. The patient presented with tachypnea (respiratory rate of 25), low grade pyrexia of 38°C, and tachycardia (110 bpm). In addition, she exhibited nystagmus, muscular atrophy with dystonia and severe kyphoscoliosis. Her current medication regimen included zonisamide, acetaminophen, lorazepam, tizanidine, levocarnitine, acetylcysteine, polyethylene glycol, albuterol, baclofen, glycopyrrolate, gabapentin, ethinyl estradiol and levonorgestrel, scopolamine, carvedilol, and torsemide.

Due to the existence of mitochondrial myopathy and generalized dystonia in this patient, an anesthetic technique was chosen that avoided both MH triggering and LS affecting anesthetic agents. The patient was scheduled as the first case of the day and the anesthesia machine was primed with a 60 minute oxygen flush at 10 L/min. Vaporizers were removed, a new circuit applied and the carbon dioxide absorbent was replaced.

The patient was premedicated with midazolam 2 mg intravenous (IV). A #4 cuffed Shiley (Covidien, Boulder, CO) tracheostomy tube was in place and attached to the anesthesia circuit with oxygen at 1 L/min administered. General anesthesia was induced using fentanyl 50 mcg, lidocaine 40 mg, glycopyrrolate 0.2 mg, and ketamine 50 mg IV. Neuromuscular antagonists were avoided during induction and throughout the procedure. The trachea was intubated using video laryngoscopy. Once the view of the vocal cords was obtained with a GlideScope (Verathon Inc., Bothell, WA), the surgeon removed the current tracheostomy tube while a 5.0 cuffed endotracheal tube (ETT) was passed orally into the trachea. General anesthesia was maintained using continuous IV infusions of remifentanil 0.2 mcg/kg/min and dexmedetomidine 0.5 mcg/kg/hr. Fresh gas flow consisting of O_2 0.4 L/min and air 0.6 L/min was delivered during the case. No volatile anesthetics were used.

Once the removal of tracheal granulation tissue and tracheal bronchoscopy were completed, the surgeon placed a 4.0 cuffless Shiley (Covidien, Boulder, CO) tracheostomy tube while the anesthesia practitioner removed the 5.0 ETT. At the conclusion of surgery, the patient was transported to the intensive care unit and respiration was controlled by a mechanical ventilator. On post-operative day one the patient was assessed as stable with no anesthesia related complications.

Discussion

Patients with LS pose a particular challenge to anesthesia professionals due to an increased susceptibility to malignant hyperthermia (MH) and rhabdomyolysis, drug induced mitochondrial depression, propofol infusion syndrome (PRIS), respiratory complications and metabolic acidosis.[2-5,7] Given the varied clinical presentation of the disease, anesthetic management of the patient with LS must include a comprehensive preoperative assessment with attention given to

the potential presence of chronic metabolic acidosis, cardiomyopathy and cardiac dysrthymias, dystonia, gastrointestinal dysmotility and seizures.[2] Secondary to mitochondrial dysfunction, these patients have compromised glucose regulation and lactic acidemia which makes them more vulnerable to hypoglycemia and metabolic acidosis.[2] For this reason prolonged fasting times and dehydration should be avoided in these patients.[2,4] Given their inability to easily mobilize alternative substrates for metabolism, preoperative dextrose/ saline containing IV fluids should be administered.[4] Solutions containing lactate as a buffer should be avoided because these patients lack the ability to metabolize lactate.[2] Other conditions that increases metabolic burden on the patient such as hypovolemia, protracted tourniquet times, hypothermia and postoperative nausea and vomiting should also be avoided.[4]

Respiratory abnormalities are common in LS due to basal ganglia and brainstem involvement.[2] During the preoperative evaluation, the anesthesia professional should assess for the presence of hypoventilation, irregular respiration, apnea, obstructive symptoms like stridor and underlying respiratory infections, all of which may compromise lung function and lead to prolonged ventilator requirements following general anesthesia.[2] Long acting opioids should be avoided in these patients due to the risk of postoperative respiratory depression.[2,7] Although limited research with newer opioids like remifentanil in patients with LS is available, remifentanil was selected in the present case due to its brief duration of action, stable pharmacokinetics across age spectrums and organ-independent metabolism.[2]

Given that all general anesthetics cause some degree of mitochondrial depression, the ideal anesthetic regimen in these patients remains controversial.[3-5] The safety of administering halogenated agents in this patient population is debated owing to a widespread assumption that children with neuromuscular disorders are at increased risk for malignant hyperthermia. Recently published data concerning the incidence of MH and rhabdomyolysis following general anesthesia for muscle biopsy in children with mitochondrial disorders found no evidence to support avoiding volatile agents in this population.[5] Despite a lack of reports of MH in patients with LS, it may be prudent to avoid MH triggers in patients with myopathic conditions who may be misdiagnosed with LS or other mitochondrial disorders.[7] To minimize further potential complications, the patient in the present case study was treated as being MH susceptible.

Additional concerns with administering volatile agents in this population relate to their ability to induce mitochondrial depression.[4] Volatile anesthetics are known to inhibit complex 1, an enzyme linked to the generation of adenosine triphosphate (ATP) via oxidative phosphorylation.[4,6] Propofol, like the volatile agents, is associated with depressed mitochondrial function at complex I and IV and despite anecdotal cases of anesthesia safely provided with volatile agents and propofol in patients with LS, it may be judicious to avoid these agents.[4,6] The decreased ATP production associated with anesthetic induced mitochondrial depression leads to increased glycolysis and glycogenolysis, decreased gluconeogenesis, and increased oxygen consumption.[6] An increase in glycolysis increases lactate and pyruvate production eventually causing PRIS, a syndrome characterized by metabolic acidosis, hyperlipidemia, acute refractory bradycardia and rhabdomyolysis.[2,5,6]

Additional drugs to avoid in this population include succinylcholine and neuromuscular blocking agents. Since many patients with LS display myopathies and dystonia, succinylcholine should be

avoided due to the potential risk of rhabdomyolysis and hyperkalemia.[6] Neuromuscular blocking agents should be used with caution as the myopathy may cause prolonged or unpredictable recovery times in this population.[5] Local anesthetics must also be administered with extreme caution in this population or not at all. Both human and rate studies have demonstrated bupivacaine causes structural alterations in mitochondria including decreasing the transmembrane electrical potential and ATP synthesis rate.[5] Ropivacaine and tetracaine have also been shown to decrease mitochondrial membrane potential.[4,5]

Dexmedetomidine, a selective α_2 agonist, is an appropriate alternative to propofol when performing a total intravenous anesthetic (TIVA) in this patient population.[7] Studies performed on ischemic rats have even demonstrated a beneficial effect of dexmedetomidine on the mitochondrial membrane, thus making this drug a much safer substitute for propofol.[7]

Given the varied presentation of LS and limited reports regarding anesthetic care of patients with LS, it is challenging to provide evidence–based recommendations on best practice. Instead thorough individual assessment combined with an understanding of the physiology of LS should guide anesthetic care of these patients. Despite newer evidence that the use of volatile agents in these patients may be entirely safe, the fact that they do cause a degree of mitochondrial depression should prompt anesthesia professionals to consider using TIVA. The current case demonstrated the efficacy of a remifentanil and dexmedetomidine based anesthetic avoiding the use of propofol and other agents that exacerbate mitochondrial dysfunction.

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Management of Obstetrical Disseminated Intravascular Coagulation

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Keywords: DIC, coagulation, obstetric, pregnant

Disseminated Intravascular Coagulation (DIC) is an acquired syndrome which results from a cascade of events that lead to the formation of excessive amounts of thrombin, depletion of coagulation factors, activation of the fibrinolytic system, and hemorrhage.[1] The diagnosis of DIC is made through laboratory results and clinical assessment of bleeding or bruising. Definitive treatment for DIC is achieved by managing the precipitating cause, including acute fatty liver, sepsis, and several obstetrical complications.[1] In the obstetric patient, a vaginal delivery can be attempted if the mother is hemodynamically stable and not actively hemorrhaging. If this is not feasible, cesarean delivery may be required.[1]

Case Report

An 18-year-old, 158 cm, 75.5 kg, primigravida female at 36 weeks gestation presented with decreased fetal movement and intraabdominal pain. An abdominal ultrasound revealed intrauterine fetal demise. The patient's prenatal course was significant for intrauterine growth restriction, and recent influenza. Her medical history included well controlled asthma. The patient was admitted for induction of labor with a consultation to anesthesia for an epidural placement. However, laboratory results consisted of a prothrombin time (PT) of 26.6 seconds, an international normalized ratio (INR) of 2.45, and a partial thromboplastin time (PTT) of 37.4 seconds. Epidural placement was contraindicated and pain was controlled with fentanyl 50mcg intravenously (IV). The patient's hemoglobin (hgb) was 10.3 g/dL, hematocrit (hct) 30.8%, platelet count 112 K/cumm, blood urea nitrogen 16 mg/dL, and creatinine 2.23 mg/dL. Transfusion of fresh frozen plasma (FFP) was initiated. Vital signs were blood pressure (BP) 142/76 mm Hg, heart rate (HR) 54/min, respiratory rate 16/min, temperature 37.2^{o}C, and SpO2 100%.

With the transfusion of FFP, the patient's laboratory values began to correct. However, her IV sites began to ooze and sanguineous vaginal discharge was noted. The patient's hgb, hct, and platelets decreased while BUN and creatinine increased with minimal urine output. Despite the transfusion of 2 units of packed red blood cells (PRBC), hgb and hct continued to decrease to 5.3 g/dL and 15.5%. The patient's fibrinogen was less than 60mg/dL and D-dimer was greater than 9,999 ng/mL. A massive transfusion protocol was initiated which consists of a plasma to PRBC ratio of 1:1 and a platelet to PRBC ratio of 1:6 starting with the 7th PRBC unit.

The patient was transferred to the labor and delivery operating room for delivery of the fetus, with the aim of avoiding laparotomy. A right internal jugular Cordis (Cardinal Health, Miami, FL) and a left radial arterial line were placed. The patient was positioned in dorsal lithotomy and after 3 pushes delivered a non-viable female fetus along with copious amounts of blood with clots. Rectal cytotec 1,000 mcg and pitocin 60 units IV were administered. The use of

methergine and hemabate were contraindicated due to high blood pressure and asthma history, respectively. Vital signs consisted of BPs in the 150s/90s mmHg, HR between 120-160/min, temperature 36.8 ºC, and SpO2 in the high 90s on O_2 4 liters via nasal cannula.

Due to a retrouterine hemorrhage, the patient's estimated blood loss was 4.5 L. A total of 10 units PRBCs, 11 units FFP, 1,700 mL of platelets, and 2 units of cryoprecipitate were transfused. Consequently, the patient began experiencing shortness of breath, hypoxia, bibasilar rales, and pink, frothy sputum production. Furosemide 40mg and propofol 150mg were administered and the trachea was intubated via direct laryngoscopy with a 7.5 mm endotracheal tube. The patient was transferred to the intensive care unit (ICU) with normal coagulation studies, but with acute kidney injury (AKI) and elevated liver enzymes. Within the next several days, kidney and liver enzymes down trended to normal limits. The patient remained intubated for a total of 5 days with substantial improvement of her pulmonary edema. She was then transferred to the medical surgical unit without any signs of bleeding, and subsequently discharged home.

Discussion

Cunningham and Nelson[2] discuss the pathogenesis by which fetal death and delayed delivery cause DIC. This is thought to be mediated by the slow release of tissue factor or thromboplastin from fetal demise and degradation of the placenta. This syndrome is usually encountered in women with a spontaneous abortion missed for several weeks, especially in twin or triplet pregnancies where there is loss of one of the fetuses.[2]

A retrospective study by Rattray et al[3], sought to determine precipitating factors including morbidity and mortality associated with DIC in a tertiary maternity hospital. Over a period of 30 years, there were 49 cases of DIC in a total of 151,678 deliveries (3 in 10,000). Contributing causes included placental abruption (37%) postpartum hemorrhage (29%), preeclampsia (14%), acute fatty liver (8%), and sepsis (6%). The associated maternal morbidity included blood transfusion of at least 5 units (59%), ICU admission (41%), hysterectomy (18%), and dialysis (6%). Three maternal deaths were reported of the 49 cases of DIC (1 in 16). Perinatal outcomes included neonatal ICU admission (72.5%), stillbirth (25%), and neonatal death (5%). This study concluded that obstetrical DIC is an uncommon condition with high morbidity and mortality.[3]

A review article by Erez et al[4], discussed the diagnostic tools and treatment modalities of DIC. Efforts have been made to create scoring systems to identify patients at high risk of developing DIC. The first such scoring system was proposed by the International Society on Thrombosis and Hemostasis (ISTH) in 2001, which takes into account platelet count (0-2 points), increase in fibrin markers (0-3 points), PTT (0-2 points), and fibrinogen level (0-1 point), assigning points to each based on severity of abnormality.[4] They showed a correlation between a score of 5-8 points and higher probability of DIC, thus necessitating daily monitoring of scores. In the order of relative importance, the hallmarks of DIC are decreasing platelet count, prolonged PT, increasing fibrin-related marker, and decreasing fibrinogen.[4] The obstetrical patient however presents a unique challenge to the accurate diagnosis of DIC as changes in coagulation parameters are inherent to the normal state of pregnancy. The gestational thrombocytopenia of the third trimester for example, may be confused with DIC. The PT and PTT are also considerably shorter in pregnancy; therefore, these values may appear to be within normal range in a pregnant patient

with DIC. Values may not be prolonged in obstetrical patients until the underlying condition, whether it be placental abruption, preeclampsia, or postpartum hemorrhage, has progressed considerably, thus delaying early diagnosis and treatment.[4] Low fibrinogen concentration is also a part of the diagnostic algorithm for DIC, however, it is very uncommon for fibrinogen levels to be low in pregnant women unless in the setting of massive postpartum hemorrhage. D-dimer is another laboratory marker that may already be elevated in pregnant patients. Taking this into consideration, Erez et al proposed a modified version of the ISTH DIC score, which omits D-dimer as a parameter. Based on points assigned to platelet count, fibrinogen concentration and PT alone, they were able to show a sensitivity of 88% and specificity of 96% for the diagnosis of DIC.[4] Nonetheless, with each of these scoring systems, consideration of the trends of these coagulation values, rather than their absolute values is more essential.

Point-of-care testing using devices like thromboelastography (TEG), which tests the efficiency of blood coagulation, can also be useful in these patients to achieve rapid results and decide intervention.[5] TEG assesses platelet function, clot speed, and clot strength via proprietary computer generated calculations.[5] However, data on the use of TEG in pregnant women is limited, largely due to poor familiarity and availability of the technique, and lack of large clinical studies.[5]

Prompt treatment of DIC is crucial, especially in obstetrics where more than one life is at risk. Treatment of the underlying disorder such as placental abruption, preeclampsia, and postpartum hemorrhage is imperative. In the case of retained fetal or placental material, evacuation of the uterus with concurrent rapid volume replacement is the treatment of choice.[6] The repletion of coagulation factors by blood component transfusion remains a cornerstone in the treatment of obstetrical DIC. Mechanical ventilator support may be required in the setting of massive blood product transfusion due to volume overload, pulmonary edema, and subsequent hypoxemic respiratory failure.[6] Many other treatment options for DIC, including administration of antithrombin concentrate, activated protein C, fibrinolytic inhibitors, and prohemostatic options have not been fully studied in the obstetrics population and each carry their own risks.[6]

After exploration and interpretation of available clinical research regarding obstetrical DIC, the identification and management of this patient's disorder was appropriate and timely. The patient was diagnosed promptly, laboratory trends were followed, a massive transfusion protocol was implemented, and respiratory support initiated. The patient's pulmonary edema, AKI, and elevated liver enzymes were treated promptly. She was further monitored in the ICU until she recovered and was subsequently discharged from the hospital.

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Prophylactic Ondansetron to Attenuate the Bezold-Jarisch Reflex

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Keywords: Bezold-Jarisch reflex, bradycardia, cesarean delivery, hypotension, ondansetron, spinal anesthesia

Spinal anesthesia, although associated with hypotension and bradycardia, is the preferred method of anesthesia for cesarean deliveries.[1] These side effects can cause detrimental events to both the mother and fetus, such as maternal unconsciousness, pulmonary aspiration, and placental hypoperfusion leading to fetal hypoxia, acidosis and neurological injury.[2] Maternal hypotension following spinal anesthesia has an incidence of 50-80%.[3] Anesthesia professionals need to be diligent in both treating and preventing these adverse events. Intravenous (IV) ondansetron can attenuate the hypotensive and bradycardic events associated with spinal anesthesia by interfering with the serotonin-induced Bezold-Jarisch reflex (BJR).[1,4]

Case Report

A 32-year-old gravida 1, para 0 female presented for a scheduled cesarean delivery for a breech presentation of the fetus. The pregnancy was full term at 39 weeks and 4 days gestation. The patient's medical history was pertinent for gestational diabetes, well controlled with intermediate-acting insulin. No other significant medical history or medications were noted. Laboratory values were unremarkable, with a white blood cell count of 7000 cells/mcL, hemoglobin 11.3 g/dL, hematocrit 34.9%, mean corpuscular volume 94.3 fL, red blood cell distribution width 13.8%, and a platelet count of 309,000 cells/mcL. She had known allergies to shellfish and latex, both of which caused hives. Baseline vital signs were: blood pressure (BP) 129/89 mm Hg, heart rate (HR) 84/min, SpO2 99%, respiratory rate 15/min, and a temperature of 36.6ºC.

An 18-gauge IV catheter was inserted in the preoperative area, and ondansetron 4 mg IV was administered before transport to the operating room (OR). Once in the OR, the patient was placed in a sitting position on the OR table, and standard monitors were applied including electrocardiogram, non-invasive blood pressure cuff, and pulse oximetry. A 1 L bag of lactated ringers IV solution was hung, and a fluid bolus was initiated. Prior to spinal anesthetic

administration, vital signs were: BP 135/90 mm Hg, HR 91/min. Spinal anesthesia was administered using sterile technique between lumbar vertebrae 3 and 4 in the subarachnoid space with 0.75% bupivacaine in dextrose 8.25%, 1.8 mL, fentanyl 20 mcg, and morphine 0.15 mg. The patient was immediately repositioned supine with left uterine displacement and a continuous IV infusion of phenylephrine at 35 mcg/min was initiated by the attending anesthesiologist. Vital signs were assessed every 2 minutes from the start of the spinal anesthetic for 30 minutes, with the lowest BP reading of 128/89 mm Hg and lowest HR of 69/min. Vital signs (VS) were then assessed every 5 minutes until the end of the surgery. Additional IV medications were administered: cefazolin 2 g, metoclopramide 10 mg, and famotidine 20 mg. The spinal anesthetic block was assessed for laterality, density, and height and an adequate surgical anesthetic level was achieved up to the T4 dermatome.

The 65-minute surgical procedure proceeded without complications or need for additional anesthetic or hemodynamic agents. Other medications used included oxytocin 40 units IV and methylergonovine 0.2 mg intramuscular for uterine atony. The total estimated blood loss was 600 mL, and the patient received a total of 2,500 mL lactated ringer's solution. Throughout the procedure, all VS remained stable, with the lowest BP reading at 110/62 mm Hg and the lowest HR at 61/min. The postoperative period was uneventful.

Discussion

The mechanism by which spinal anesthesia leads to hypotension and bradycardia is believed to be attributed to sympathectomy 2-6 dermatomes cephalad from the initial block placement, a decreased physiological response to the baroreceptor reflex, vagal overactivity, increased parasympathetic tone, and most prominently, activation of the BJR.[4] The BJR is a cardio-inhibitory reflex elicited by noxious stimuli in the left ventricular wall, sensed by both mechanoreceptors and chemoreceptors.[4] The activated receptors send signals along unmyelinated, cardiopulmonary vagal afferent C-fibers, which blunt the efferent sympathetic fibers to peripheral vessels and heart tissue, leading to hypotension, bradycardia, and cardiovascular collapse.[5]

The sympathectomy that occurs during spinal anesthesia leads to a decrease in vascular resistance, both venous and arterial.[1,4] The venous system contains 75% of total blood volume. Therefore, venodilation and its associated venous pooling lead to a significantly decreased preload. The reduced pressure in the arteries is sensed by the arterial baroreceptors, and the typical physiologic response is tachycardia. The tachycardia leads to increased noxious stimuli, which are detected by the mechanoreceptors in the volume depleted left ventricle, eliciting the BJR. The decreased vascular resistance also leads to catecholamine release, which then causes platelet activation. Activated platelets release stored serotonin, which binds to serotonin type 3 (5-HT_3) chemoreceptors located in the left ventricle. Activated 5-HT3 receptors within the left ventricle are believed to be the leading cause in eliciting the BJR.[1,4]

Sahoo et al. hypothesized spinal-induced hypotension and bradycardia could be attenuated by use of a 5-HT_3 antagonist prior to induction of spinal anesthesia in non-laboring obstetric patients undergoing a cesarean section, to decrease the BJR.[1] Results of the double-blind, randomized, placebo-controlled trial showed administering 4 mg ondansetron, a highly

efficacious and specific 5-HT_3 antagonist, 5 minutes before the administration of spinal anesthesia, resulted in a decreased drop in systolic blood pressure (SBP) and mean arterial pressure.[1] Wang et al. performed a similar randomized controlled trial, using the same dose and administration timing of ondansetron, and found ondansetron preloading significantly reduced hypotension.[6] The optimal dose of ondansetron to attenuate the BJR and mitigate maternal hypotension was studied in a double-blinded controlled trial, which found ondansetron 4 mg IV, also given 5 minutes prior to spinal anesthesia for elective cesarean delivery, to be the optimal preloading dose.[7] Although these studies did not find statistically significant decreases in the incidence of bradycardic events, a systematic review and meta-analysis of 13 randomized controlled trials found IV ondansetron, administered 5 minutes prior to a spinal anesthetic, strongly attenuates both hypotension and bradycardia.[4]

The anesthetic plan for this patient addressed the known complications of hypotension and bradycardia associated with spinal anesthesia by attempting to blunt the BJR. Measures were taken to avoid the BJR, not merely treat its sequelae. Five minutes prior to induction of spinal anesthesia, the patient received ondansetron 4 mg IV with the intention to antagonize the $5HT_3$ receptors located in the left ventricle of the heart, minimize the impact of serotonin-induced BJR, suppressing its effects of bradycardia, vasodilation, and hypotension. Although there is no universal definition of hypotension, most authors agree that hypotension is present when SBP drops by 20-25% from baseline, or the SBP is less than 80-90 mm Hg.[3] Bradycardia also lacks a universal definition, but most authors define bradycardia as less than 40-60/min or a 30% drop from baseline.[2,4] In this case, the patient's SBP and HR did not decrease to meet these definitions of hypotension and bradycardia. The lowest SBP recorded was 110 mm Hg, far from a 20% drop from baseline, and a HR of 61/min. It is postulated the patient did not experience a hypotensive or bradycardic event during spinal anesthesia, in part from suppression of the BJR by pre-administration of ondansetron.

Administering IV ondansetron prior to spinal anesthesia in cesarean delivery is a simple, efficient way to help prevent the BJR and provide a safer and more pleasant experience for the patient. Not only is hypotension and bradycardia deleterious for maternal and fetal health, it is also associated with maternal nausea and vomiting. The timed administration of ondansetron with attenuation of the BJR and decreased incidence of nausea and vomiting can result in improved maternal and neonate postoperative outcomes.

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Anesthetic Management for Free Flap Surgeries of the Head and Neck

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Keywords: free flap failure, head and neck surgery, free tissue transfer, microvascular flaps, intraoperative vasopressors, anesthesia management, intraoperative fluid management

Microvascular free flap surgery has become the mainstay of surgical treatment in head and neck cancer, trauma, and burns.[1] Since the introduction of free tissue transfer for head and neck reconstruction in 1958, the success rate has improved from 40-50% up to 99% amongst experienced surgeons.[1,2] The intraoperative anesthesia management of these patients, with respect to hemodynamic control and regional blood flow via intravenous fluid administration and vasopressor use, is believed to be an important and determining factor in the success of microvascular flaps.[2,3]

Case Report

A 70-year-old, 86 kg, 185 cm male was scheduled for a radical neck dissection, complete glossectomy, surgical tracheostomy and free flap. His medical history included two months of significant weight loss, lethargy, tongue pain, with resultant diagnosis of tongue cancer, status post radiation treatment and gastrostomy tube. His past medical history was insignificant for smoking, alcohol use, or recreational drugs. Other medical conditions included hypertension, and idiopathic cardiomyopathy with ejection fraction of 42%. An automatic internal cardiac defibrillator (AICD) was present and recently interrogated during a preoperative visit; no history of AICD discharge. His surgical history included knee replacement, inguinal hernia repair, and gastrostomy tube placement. Current medications included metoprolol 50 mg daily (taken day of surgery), lisinopril 10 mg daily (held day of surgery), oxycodone 50 mg/day, and aspirin 81 mg daily.

Preoperative airway assessment revealed Mallampati 4 with limited mouth opening. Thyromental distance of three fingerbreadths, limited neck range of motion, edentulous, and a beard were noted. No prior anesthesia records were available for review, and patient denied prior difficulty with intubation. A 20-gauge right forearm peripheral intravenous line was in situ. The patient received midazolam 2mg prior to going to the operating room.

On arrival to the operating room, standard monitors were applied, including 5-lead ECG, non-invasive blood pressure monitoring, axillary thermometer, and pulse-oximetry. A magnet was placed over the AICD and defibrillator pads were optimally placed out of the surgical field. The patient was pre-oxygenated with O_2 10 L/min for 3 minutes. Propofol 100 mg, ketamine 30 mg, and remifentanil 100 mcg were administered. Bag mask ventilation was established with an oral airway. Succinylcholine 100 mg was then administered. Video laryngoscopy with a Glidescope (Verathon Inc., Seattle, WA) was used and intubation with a 7.0 cuffed oral endotracheal tube was uneventful. Mechanical ventilation was initiated. A second peripheral intravenous line was placed after induction with normal saline infusing through a fluid warmer. A lower body warmer was placed over the torso and right leg to allow access to left leg surgical site. Hemodynamic management during induction was maintained with titration of sevoflurane and administration of a 500 mL intravenous fluid bolus of crystalloid.

Maintenance of anesthesia during the tracheostomy, neck dissection, and full thickness grafting was established with sevoflurane 1.4% expired concentration in O_2 0.5 L/min and air 2.5 L/min. Continuous infusions included remifentanil at 0.15 mcg/kg/min and ketamine at 70 mcg/kg/min. During flap anastomosis and microvascular portion of surgery, remifentanil was decreased to 0.07 mcg/kg/min and sevoflurane to 1.0% expired concentration in mixture of nitrous oxide 2 L/min and O_2 2 L/min. Intravenous crystalloid and Albumin 5% were used to maintain mean arterial blood pressure between 70-90 mm Hg for a total of 2500 mL and 1500 mL, respectively. Vasopressors were avoided throughout the entire procedure. Urine output averaged 0.5mL/kg/hr. The ketamine infusion was discontinued one hour prior to end of case and hydromorphone 0.4 mg (0.005 mg/kg) administered at that time.

At surgical completion, remifentanil infusion was discontinued and spontaneous ventilation established with sevoflurane 1.0% expired concentration in O_2 10L/min. Dexmedetomidine 20 mcg and hydromorphone 0.6 mg was titrated according to respiratory rate and hemodynamics. The patient was disconnected from the anesthesia machine and a tracheostomy mask delivering O_2 6 L/min was placed over the tracheostomy for transfer. Vital signs were stable upon arrival to the post anesthesia care unit.

Discussion

Microvascular free tissue transfer (FTT) is one of the preferred reconstructive options for patients with head and neck defects.[4] Total or partial flap failure is extremely rare, but remains a significant post operative risk. Postoperative complications can result in serious morbidity as well as increased hospital costs due to longer hospital stays and additional surgical or medical interventions.[3] Meticulous perioperative anesthetic management with attention to how the use of vasopressors, fluid therapy, analgesia, and temperature control affect the flap, may improve the outcome of this challenging population.[1]

Pre-operative assessment and investigation plays an important role in risk stratification and development of the anesthesia plan.[1] The typical patient presenting for head and neck cancer surgery is greater than 60 years of age and often has had many years of heavy tobacco and alcohol use. Common coexisting medical conditions include chronic obstructive pulmonary disease, coronary artery disease, hypertension, diabetes, alcoholism, and malnutrition.[5] The case study is a somewhat atypical presentation of a patient requiring FTT, with age of 70 years and history of hypertension as the only common coexisting conditions. Many of these patients, like the one presented, have undergone radiation, which can result in dry mouth, airway swelling, dysphagia, poor oral intake, and dehydration.[6] Dehydration, in addition to anti-hypertensive medications, may predispose these patients to hypotension during induction, thus one may consider a fluid bolus prior to induction and/or slow titrated induction technique. Radiation may also make tracheal intubation and mask ventilation difficult by causing fibrosed, immobilized, and distorted airway structures.[5] Video laryngoscopy or fiberoptic intubation should be utilized without hesitation. For this patient, the anesthesia plan included intubation with Glidescope (Verathon Inc., Seattle, WA) and a smaller than normal endotracheal tube in response to initial airway assessment and history of radiation therapy.

The plan for anesthesia management for head and neck surgery requires especially close coordination with the surgeon. It is not uncommon for microvascular surgeons to observe a variety of fiercely contended dogmas regarding the best care for flap patients as a result of their particular training, mentor's beliefs, anecdotal experience, and institutional practice.[7] The surgeon may ask for avoidance of neuromuscular blockers during portions of the surgery to identify nerves by direct stimulation and to preserve them.[5] Remifentanil was utilized in this case as it provides adequate perioperative analgesia, rapid control of blood pressures, vasodilation, and reduces the need for intraoperative muscle relaxant, allowing excellent conditions in microvascular surgery.[2] The airway will be shared, and a tracheostomy is common practice for many head and neck procedures.[5] Because large mobilizations of fluids may occur, large bore intravenous access is essential, and location must be discussed with the surgeon as to not influence the harvesting location.[2] Active heating should be initiated as soon as possible to maintain normothermia and prevent increased blood viscosity; the forced hot air blanket should cover the greatest possible surface area, without interfering with surgical sites.[2] With use of both an intravenous fluid warmer and forced hot air blanket, the patient in this case study maintained axillary temperatures of 35.7 to 36.2 degrees Celsius.

Blood pressure control and fluid management are at the center of anesthesia management in microvascular head and neck surgery. Moderate controlled hypotension may be helpful during dissection to improve surgical conditions and limit blood loss.[2,5] This was done to a limited extent, keeping mean arterial pressure 70-90mmHg, given the patient's baseline hypertension. Following reanastamosis of a microvascular flap, avoidance of hypotension is a key factor in maintaining good perfusion of the flap.[1,5] Agents, such as sevoflurane, resulting in the decrease of systemic vascular resistance and arterial pressure can cause severe reductions in blood flow to free flaps and must be used with caution.[7] The intraoperative use of vasoactive medications is one of the most fiercely contended dogmas of microvascular surgery.[7] Several animal models have shown that phenylephrine administered directly into the feeding artery of the free flap can decrease blood flow through increasing systemic vascular resistance, cause hypersensitivity to

alpha-adrenergic stimulation, and result in vasospasm of the flap.[3] However, recently published prospective and retrospective human studies suggest that the intraoperative use of vasopressors does not appear to negatively influence surgical success.[1,3] A balanced anesthetic with short acting and titratable agents such ketamine, remifentanil, and low concentrations of sevoflurane mixed with nitrous oxide was administered in this case to optimize hemodynamics and maintain adequate flap perfusion while avoiding vasopressors.

In an attempt to avoid vasopressor use, patients will often receive generous amounts of intravenous fluid to compensate for periods of hypotension.[3] Both perioperative fluid volume and choice of crystalloid versus colloid is a major contributory factor in flap survival.[1] Traditionally, hypervolemic hemodilution has been used during anesthesia for this type of surgery.[2] However, free flap vasculature is likely to have been exposed to endothelial damage leading to enhanced capillary permeability.[1] Free flaps do not have lymphatic drainage and, thus, excessive fluid can lead to edema and be deleterious.[2] The current literature suggests that more than 7 liters of crystalloid given during initial FTT is associated with higher rates of graft failure and complications.[3] It is suggested that the use of crystalloids can cause a hypercoagulable state, especially when administered rapidly compared to colloids.[7] The combination of crystalloid and colloid is generally appropriate, guided by signs of adequate perfusion (i.e. urine output > 0.5ml/kg/hr).[2] Furthermore, transfusion should be judicious, as increased hematocrit is associated with increased viscosity, risk of micro-embolism, and decreased perfusion in the microvasculature.[1] Fluid management for the case study patient was guided by these recommendations. Blood transfusion was unnecessary, he received a combination of crystalloid and albumin 5% totaling 4 L, and urine output was adequate throughout.

Free flap failure is increasingly uncommon, yet perioperative management for head and neck FTT is inconsistent.[1] During microvascular head and neck surgery, anesthesia providers should be aware of the risk factors for free flap failure, and the optimization of physiological conditions that are noted to increase complications. Excessive fluid administration, significant medical comorbidities, and prolonged operative time are associated with increased risk of free flap failure.[7] There is limited evidence that vasopressors, hypotension, colloids, anticoagulants, nitrous gas use, or obesity negatively affect free flap outcomes.[7] High quality prospective outcome studies are needed to identify best practice, thus the development of guidelines and protocols for perioperative management of microvascular free flap surgery.[1]

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Propofol, Ketamine, and Ketofol Use for Procedural Sedation

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Keywords: ketamine, propofol, ketofol, procedural sedation, analgesia

Introduction

Procedural sedation is used frequently for a variety of noxious procedures.[1] Several different sedative and analgesic drugs are administered in order to achieve patient comfort for these procedures, such as propofol, ketamine, and ketofol.[1] The anesthetic goal is to provide adequate sedation and analgesia, limit patient recall, minimize side effects, and allow for a quick recovery from the anesthetic agents.[2] Despite the frequent use of these drugs, the consensus regarding which anesthetic best achieves these outcomes is unclear.

Propofol is a short-acting sedative hypnotic that interacts with the inhibitory neurotransmitter, γ-aminobutyric acid (GABA), and directly stimulates $GABA_A$ receptors to produce its amnestic action.[3,4] Propofol is useful for producing rapid loss of consciousness, quick emergence, anxiolysis, amnesia, and preventing nausea. However, propofol can produce side effects, such as hypotension, bradycardia, a widened QRS complex, respiratory depression, apnea, and reduced cerebral blood flow.[4] Ketamine is a dissociative anesthetic that acts by *N*-methyl-*D*-aspartate amino acid (NMDA) receptor antagonism, provides amnestic and analgesic effects, causes minimal respiratory depression, and is a potent bronchodilator. Ketamine is useful in hemodynamically unstable patients as the drug minimizes cardiodepression. Similar to propofol, ketamine has unwanted side effects, such as producing delirium, increased intracranial pressure, greater production of oral secretions, and increased heart rate or blood pressure, which can be detrimental in certain patients.[4]

The combination of propofol and ketamine, commonly described as ketofol, allows for a decrease in the dose of each individual drug needed for producing sedation and lessens the

adverse effects that can occur when administering each drug alone.[3] Ketofol has the potential to provide less nausea and vomiting, deeper anesthesia and analgesia, and greater hemodynamic stability.[5] A review of current literature describing use of these anesthetic drugs for procedural sedation was conducted and summarized.

Methodology

Purpose and Evidence-Based Practice Model

The purpose of this literature review was to determine whether ketofol, propofol or ketamine is more effective and advantageous in reducing respiratory complications for procedural sedation. The PICOT format was used to develop the following clinical question for this review: For adult patients undergoing procedural sedation (P), does administering ketofol (I) versus propofol or ketamine alone (C) reduce adverse respiratory events (O) throughout the perioperative period (T)?

Search Terms and Databases

Search terms used included ketamine, propofol, ketofol, procedural sedation, and analgesia. Databases used in the search include PubMed, CINAHL, ProQuest Nursing and Allied Health Source, and Google Scholar. Articles that were reviewed were between the years of 2011-2016 and involved procedural sedation for adult subjects.

Levels of Evidence and Inclusion Criteria

The evidence cited in this review is a combination of randomized control trials (RCTs), systematic reviews, and meta-analyses that meet level I and level II evidence criteria. Studies that had pediatric patients, low levels of evidence or that were non-procedural sedation cases were excluded from this review.

Literature Review

Propofol, ketamine, and ketofol are all used as anesthetic options in procedural sedation. Ferguson et al.[1] performed a randomized double-blinded clinical trial on 573 emergency department patients that were recruited from three different hospitals. The study occurred from April 2013 until April 2015 and evaluated three outcomes. The main objective was to compare use of propofol as an individual agent and use of propofol and ketamine combined in a 1:1 ratio to determine which agent had the least amount of respiratory adverse events. The secondary objective was to determine which agent resulted in more hypotension and greater patient satisfaction. Study participants had to be 18 years of age or older and undergoing deep procedural sedation. Several criteria were used to exclude patients from this study, such as allergy to ketamine or eggs, hypertension > 160/90 mm Hg, American Society of Anesthesiologists (ASA) class IV or greater, and many other criteria. Of the 573 patients, 292 patients were randomized to the propofol group whereas 281 were randomized to the ketofol group. Respiratory adverse events requiring an intervention occurred in 5% of patients in the propofol group and 3% of patients in the ketofol group, which indicated similar occurrence rates. Hypotension occurred more frequently in the propofol group (8%) as compared to the ketofol group (1%). Both groups reported high patient satisfaction levels, whereas patients that received ketofol had a greater incidence of emergence delirium than those that received propofol.

Strengths of the study were the large sample size and that patients were randomized to the groups. Weaknesses of the study included the possibility of sample selection bias and that confounders could have altered results. These confounders included pre-procedural opiate and oxygen use.[1]

Miner et al.[6] also performed a randomized, double-blinded clinical trial. In that study, 271 patients from Hennepin County Medical Center emergency department were included. Of those 271 patients, 90 patients were randomized to the propofol group, 85 patients were randomized to the 1:1 propofol: ketamine group, and 96 were randomized to the 4:1 propofol: ketamine group. Patients received deep procedural sedation and had age greater than 18 years. Patients that were excluded from the study were ASA greater than II, those not able to consent, were pregnant or intoxicated, and any patient with a hypersensitivity to propofol or ketamine. The main objective of this study was to determine whether one of the groups experienced more airway or respiratory events that required an intervention. Several other outcomes were measured such as depth of sedation, time to recovery, recall, patient satisfaction, and pain. The researchers found that not only were airway and respiratory adverse events that required an intervention similar between all three groups, the other outcomes were as well. Strengths of this study included the large sample size and that patients were randomized to all groups. Conversely, one of the weaknesses of the study was that patients were sampled from one facility. Also, adverse events were transient, and clinicians performed interventions before adverse events happened, which could have altered results.[6]

Yan et al.[5] performed a systematic review and meta-analysis of six RCTs containing 932 patients. Studies reviewed were less than six years old and compared outcomes of both adult and pediatric patients. This high level of evidence, large sample size, and use of recent literature are also the strengths of this study. Literature was found through searches of MEDLINE, Embase Classic plus Embase, CINAHL, and the Cochrane Central Register of Controlled Trials. Initially, 1,688 citations were found. Studies were eliminated from the search if they were not human research, in a language other than English, not RCTs, were duplicate sources, and did not meet certain criteria set by the researchers, such as taking place in an emergency department setting. After filtering out those studies, six RCTs met the study criteria. The primary outcome being evaluated in this systematic review and meta-analysis was whether a ketamine-propofol combination had a reduced number of respiratory adverse events compared to propofol. Secondary outcomes and areas measured were overall adverse events, sedation time, procedure time, and recovery time. Results were statistically significant and found that ketamine-propofol had a lower incidence of adverse respiratory events when compared to propofol alone. Also, no difference was found between the groups for overall adverse events. Other secondary outcome results for sedation time, procedure time, and recovery time could not be pooled due to reporting of medians rather than means, which is one of the study's weaknesses. Another weakness is the fact that definitions of specific outcomes, such as adverse events, sedation, and recovery time, were different between RCTs and made comparison between RCTs more challenging.[5]

Jalili et al.[7] conducted a systematic review and meta-analysis of 18 RCTs that compared ketofol and propofol use for procedural sedation. The primary outcome reviewed was complications associated with administration of ketofol versus propofol. Secondary outcomes evaluated were psychomimetic complications, muscle rigidity, and nausea and vomiting. The results of this

systematic review were very similar to the systematic review conducted by Yan and colleagues. Ketofol was found to be a more effective choice than propofol for reducing respiratory adverse events, and this result was statistically significant. Ketofol also reduced cardiovascular complications, such as bradycardia and hypotension. Secondary outcomes were not statistically significant. Strengths of this study included the level I evidence and pooled results of the RCTs. Weaknesses of this study were that the RCTs that were compared had different procedures performed, settings, and doses of ketofol. This could have altered the conclusions that the researchers formed from all the RCTs.[7]

Author, Date & Level of Evidence	**Patient Groups**	**Study Outcomes**	**Key Results**	**Strengths & Weaknesses**
Ferguson et al, 2016[1] Randomized double-blinded clinical trial, level II evidence	573 adult patients (emergency room) randomized in 2 groups (propofol group = 292, ketofol group = 281) propofol group median dose: 1.3 mg/kg ketofol group median doses: propofol 0.675 mg/kg ketamine 0.675 mg/kg	Primary outcome: respiratory adverse event occurrences that required an intervention Secondary outcome: hypotension, patient satisfaction, and emergence delirium	Both groups had similar occurrences of adverse respiratory events that required an intervention: propofol group (5%) and ketofol group (3%). Both groups had high levels of patient satisfaction (median 10, range 0-10). Propofol led to more hypotension (n=24, 8%) than ketofol (n=3, 1%); statistically significant. Ketofol group experienced more emergence delirium (5%) versus propofol group (2%).	Strengths: large sample size and randomization Weaknesses: possible sample selection bias; confounders may have affected results, such as pre-procedural opiate and oxygen use
Miner et al, 2015[6] Randomized double-blinded clinical trial, level II evidence	271 patients randomized to 3 groups: propofol group = 90, 1:1 propofol: ketamine (p:k) = 85, 4:1 p:k= 96 Initial sedative bolus 0.1 ml/kg for all 3 groups. Median total bolus doses: propofol group 1.5mg/kg 1:1 p:k 0.75 mg/kg	Primary outcome: airway and respiratory adverse events that required an intervention Secondary outcomes: depth of sedation, time to recovery, recall, patient satisfaction, and	Airway and respiratory adverse events that required an intervention were similar between all three groups: propofol group (29%), 1:1 propofol and ketamine (19%), and 4:1 propofol and ketamine (32%). Secondary outcomes were also similar between all groups.	Strengths: large sample size and randomization Weaknesses: transient adverse events; interventions performed before adverse events happened, which could have altered results; patients sampled from one facility

	ea 4:1 p:k - propofol 1.28 mg/kg with ketamine 0.32 mg/kg	pain		
Yan et al, 2015[5] Systematic review and meta-analysis of relevant RCTs, level I evidence	Six RCTs containing 932 patients (520 patients in the ketamine-propofol group and 412 patients in the propofol group)	Primary outcome: respiratory adverse events Secondary outcome: compared ketamine-propofol and propofol alone for overall adverse events, sedation time, procedure time, and recovery time	Ketamine-propofol had lower incidence of adverse respiratory events when compared to propofol alone (29% vs 35.4%); statistically significant. No difference found between the groups for overall adverse events (ketamine-propofol group 38.8% vs propofol 42.5%). Sedation time, procedure time, and recovery time results could not be pooled due to reporting of medians and not means.	Strengths: level I evidence and pooled results of the RCTs; studies were less than six years old Weaknesses: summaries of some outcomes could not be combined; definitions of specific outcomes were different between RCTs; individual trials had small sample sizes; methodology had heterogeneity in patient populations, procedures, and study settings
Jalili et al, 2016[7] Systematic review and meta-analysis of relevant RCTs, level I evidence	18 RCTs comparing ketofol versus propofol administration	Primary outcome: administration complications, such as respiratory or cardiovascular complications Secondary outcomes: Psycho-mimetic complications, muscle rigidity, and nausea and vomiting.	Ketofol was more effective than propofol in reducing respiratory adverse events (RR 0.31, p=0.001) and cardiovascular complications, such as bradycardia (RR 0.47, p=0.008) and hypotension (RR 0.11, p=0.04). Psychomimetic complications, muscle rigidity, and nausea and vomiting were not statistically significant.	Strengths: Study provided level I evidence and pooled results of the RCTs; all trials had a high quality report score (3 or higher) on Jadad scale Weaknesses: methodology had heterogeneity in individual trials and compared different procedures performed, settings, and doses of ketofol used.

Conclusions

Propofol, ketamine, and ketofol are all used during procedural sedation. The goal of this evidence-based review was to determine which of these anesthetic options best reduces respiratory complications. This factor was used as the primary determinant for which anesthetic provided the most ideal anesthetic for procedural sedation. Other factors assessed in the literature were cardiovascular complications, nausea and vomiting, recovery time, and surgical satisfaction. In the studies conducted by Ferguson et al.[1] and Miner et al.[6], both sets of researchers found that there was no statistical difference in the adverse respiratory events between the propofol and ketofol groups. Ferguson et al.[1] also found that propofol led to more hypotension. In the meta-analysis conducted by Yan et al.[5] and Jalili et al.[7], the researchers did find statistical significance with ketofol having a lower incidence of respiratory adverse events. These results differ from the reviews done by Ferguson et al. and Miner et al. While many of these studies met criteria for the highest level and quality of evidence, the review of the literature suggests that not one of these anesthetics has been consistently shown to be better than the other anesthetics for use during procedural sedation. Each of these anesthetics has benefits and negative side effects. Therefore, anesthesia professionals should choose the anesthetic drug for procedural sedation that is patient specific and based on an individual's health history, offers the best health outcome and surgical experience, and avoids the potential negative drug side effects for that patient.

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The Mallampati Test versus the Upper Lip Bite Test in Predicting Difficult Intubation

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Keywords: Airway assessment, Mallampati test, upper lip bite test, difficult intubation

Introduction

The accurate assessment of the airway is one of the most important skills an anesthesia practitioner should perfect. A difficult intubation is often defined as a Cormack Lehane (CL) grade of III or IV in adult patients. The reported frequency of difficult intubation or CL grade III and IV is 12.35% and 9% in those undergoing a general anesthetic.[1] Several assessments and scoring systems are used by anesthesia providers with the goal of identifying a potentially difficult intubation.

The modified Mallampati test (MMT) is the most widely used tool for the assessment and prediction of the difficult intubation.[2] The oropharyngeal structures as well as space in the mouth are assessed and graded based on the view with the mouth opened maximally. A wide variability in the predictive value of the MMT in identifying a difficult intubation has been found.[1]

The upper lip bite test (ULBT) is a simple maneuver that may be used as an alternative for predicting difficult intubation. The patients' ability to extend their temporomandibular joint is measured by comparing the position of the lower incisors and upper lip.[2] The grades of the ULBT are easily identifiable and distinguishable from one another.

Reliable preoperative airway assessments are necessary to identify patients at risk of difficult or failed intubation. Failure to anticipate a difficult intubation can have serious consequences. The purpose of this evidence-based practice analysis is to compare the accuracy of the MMT with the ULBT in predicting difficult intubations in adult surgical patients undergoing general endotracheal anesthesia.

Methodology

The clinical question was expressed through the use of a PICO format. In adult patients undergoing general endotracheal anesthesia (P), does the ULBT (I) compared to the MMT (C), more accurately predict a CL grade III or IV(O)?

An electronic database search was conducted using EBSCO, PubMed, Google Scholar, Cochrane Database, and Cumulative Index to Nursing & Allied Health Literature (CINAHL). Peer-reviewed journals published in English between 2009 and 2017 were searched. Search Terms included: Airway assessment, Mallampati test, upper lip bite test, difficult intubation, difficult laryngoscopy, difficult endotracheal intubation, and difficult airway. All studies reviewed included adult patients undergoing general endotracheal anesthesia.

Fifteen studies met the inclusion criteria with six studies excluded from analysis because the ULBT and MMT were not compared. One study that examined the obstetric population was also excluded. The eight remaining studies consisted of male and female adult patients that were 18 years of age or older. All included studies had an observational prospective design and were level III evidence in the Joanna Bringgs Institute hierarchy of evidence. The study participants had an American Society of Anesthesiologists (ASA) Physical Status classification of either I and II or I, II and III.

Literature Review

All studies used the CL classification for defining the ease of intubation. A CL grade I was defined as a full view of the glottis, a grade II was a partial view of the glottis, grade III was assigned when only the epiglottis was visible, and grade IV was assigned when the epiglottis was not visible. A CL grade I or II was considered easy, and a CL grade of III or IV was considered a difficult intubation in all studies.

The MMT in all studies was assessed with the patient in the sitting position, mouth wide open with the tongue fully protruded and without phonation. The patient was assigned a MMT class I if the pillars, uvula, soft palate, and hard palate could be seen; class II if all but the pillars could be seen; class III if the soft palate and hard palate only could be seen; and class IV if the only the hard palate was seen. The ULBT was done by asking patients to bite their upper lip with the lower incisors as high as possible in the sitting position with the head in neutral position. The ULBT was rated as class I if the lower incisors could bite the upper lip above the vermilion line, class II if the lower incisors could bite the upper lip below the vermilion line, and class III if the lower incisors could not bite the upper lip.

All of the studies rated a MMT score of I or II as a potentially easy intubation and a MMT score of III or IV as a potentially difficult intubation. Six of the eight studies rated an ULBT score of I or II as a potentially easy intubation and an ULBT score of III as a potentially difficult intubation. Honarmand et al[6] and Haq and Ullah[2] determined that an ULBT score of I was considered a potentially easy intubation and a score of II or III was a potentially difficult intubation. Both the ULBT and MMT were obtained simultaneously by a single trained observer not involved in the clinical care. The results of the evaluations were not available to the provider performing the tracheal intubation.

All study subjects were scheduled for a general endotracheal anesthetic for an elective procedure and had routine monitors applied upon arrival to the operating room. Subjects were then placed in a sniffing position and asked to breathe 100% oxygen. An induction dose of anesthesia medications was given intravenously as well as a muscle relaxant. After full muscle paralysis, intubation was performed without applying external laryngeal pressure. Those performing the intubation had at least one year or more experience and were blinded to the results of the ULBT and MMT. All intubations were performed with a Macintosh laryngoscope blade size III or IV with a CL grade immediately recorded.

Ali et al[3] evaluated 324 adult patients in a prospective cross-sectional study. Excluded patients were those not able to perform either of the tests, edentulous patients, or those requiring a rapid

sequence induction. No patients were rated a CL grade IV upon intubation while 56 patients (17.3%) were rated a CL grade III and a difficult intubation. Eleven (19.6%) of the 56 difficult intubations were rated a MMT grade III or IV compared to 49 (87.5%) rated an ULBT grade III.[4] The sensitivity, or the percentage of difficult intubations which were correctly predicted to be difficult intubations, was 87.5% with the ULBT and 19.6% with the MMT. Specificity, or the percentage of correctly predicted easy intubations as a proportion of all predicted easy intubations, was 92.9% for the ULBT and was very similar to the MMT specificity of 91.8%. The positive predictive value (PPV), or the number of predicted difficult intubations which were difficult, was 71.6% with the ULBT and 33.3% with the MMT. The negative predictive value (NPV), or the number of predicted easy intubations which were easy, was 97.3% for the ULBT and 84.8% for the MMT. The ULBT had a significantly higher sensitivity ($p<0.05$) compared to the MMT, and specificity was high for both the MMT and ULBT.

In a single-blinded prospective observational study Badheka et al[4] evaluated 170 adult patients. Excluded patients were those with head or neck abnormalities, who were edentulous, or who were scheduled for a cesarean section. Three had a CL grade IV and 48 had a CL grade III for a total of 51 (30%) difficult intubations. The sensitivity of the ULBT was 96.64% compared to 78.99% for the MMT. Specificity for the ULBT was 82.65% compared to 68.63% for the MMT. The ULBT PPV was 92.7% and the MMT PPV was 85.45% while the NPV for the ULBT was 91.3% and the MMT was 58.3%.[5] Sensitivity and specificity were both higher with the ULBT when compared to the MMT.

A prospective double-blinded study of 402 adult surgical patients was conducted by Wajekar et al.[5] Excluded patients were those with previous surgery, burns, tumors/masses in the head or neck region, patients with restricted neck mobility, edentulous patients, pregnant patients, and those with a BMI>26kg/m2. Forty-six (11.4%) patients were a difficult intubation with a CL of III or IV. The sensitivity of the ULBT was 98.6% compared to 90.4% for the MMT. Specificity for the ULBT was 8.7% compared to 30.4% for the MMT. The ULBT PPV was 89.3% and the MMT PPV was 91%. The NPV for the ULBT was 44.4% and the MMT was 29.2%. [6] A high sensitivity of the ULBT demonstrates that when it predicted a difficult intubation a high percentage were difficult, however, both the ULBT and MMT were highly sensitive in the study. The ULBT had a much lower specificity (8.7%) meaning many predicted easy intubations were difficult. The MMT also had a low (30.4%) specificity in the study.

Honarmand et al[6] conducted a prospective observational study of 525 adult patients. Excluded patients were those with previous surgery, burns, tumors/masses in the head or neck region, patients with restricted neck mobility, edentulous patients, those unable to sit, and those that required an awake intubation.[3] An ULBT of I was considered to predict an easy intubation, and a ULBT of II-III was considered to predicted a difficult intubation. The incidence of difficult intubation was found to be 9.7% or 51 patients. The ULBT sensitivity was 90.2% compared to the MMT of 68.6%. The specificity for the ULBT was 59.4% and 52.8% for the MMT. The ULBT PPV was 19.3%, and the MMT PPV was 13.6%. The low PPVs indicate that many predicted difficult intubations were easy. The NPV for the ULBT was 98.3% compared to the MMT NPV of 94.0%.

Another prospective observational study by Safavi et al[7] evaluated 467 adult patients. Excluded patients were those not able to cooperate, those with a burn or trauma to the airway, a tumor or mass in the head or neck region, restricted mobility of the neck or mandible, and morbidly obese patients.[7] The incidence of difficult intubation was 6.9% with 25 patients with a CL grade III (5.3%), and 8 patients with a CL grade IV (1.8%). The ULBT sensitivity was 75.7% and the MMT was 63.6%. The ULBT specificity was 80.8% compared to the MMT of 46.95%. The ULBT PPV was 22.7% and MMT was 8.2%. The ULBT NPP was 97.8% and a MMT NPV of 94.5%. The sensitivity of the ULBT was higher than the MMT while both had a low PPV.

A prospective observational design was also used by Safavi et al[8] in evaluating 603 patients. Excluded patients were those with previous surgery, burns, tumors/masses in the head or neck region, patients with restricted neck mobility, edentulous patients, those unable to sit, and those that required an awake intubation.[8] There were 41 (6.8%) patients with a CL grade of III or IV, or a difficult intubation. Sensitivity of the ULBT was 66.01% compared to 87.37% for the MMT. Specificity of the UBLT was 73.1% compared to 14.63% for the MMT. The PPV of the ULBT was 97.1% compared to 93.3% for the MMT. The ULBT NPV was 13.6% compared to a 7.8% NPV for the MMT. The area under the curve (AUC) for the ULBT was 0.82, significantly greater ($p<0.0001$) compared to the MMT AUC of 0.56. Although the sensitivity of the ULBT was lower than the MMT, the ULBT had a higher AUC.

A single-blinded observational study by Shah et al[9] evaluated 480 adult patients. Excluded patients were those not able to perform either of the tests, those with any malformation of the airway head or neck, and pregnant patients. There were 67 (13.95%) difficult intubations including 65 patients with a CL grade III and 2 patients with a CL grade IV. The sensitivity of the ULBT was 74.63% compared to 70.15% for the MMT. The specificity of the ULBT was 91.53% compared to 61.02% for the MMT. The ULBT PPV was 58.82% compared to 22.6% for the MMT PPV. The ULBT NPV was 95.70% compared to the MMT NPV of 92.65%. The ULBT had a slightly higher percentage of predicted difficult intubations, which were difficult when compared to the MMT; however, the ULBT had a significantly higher percentage of predicted easy intubation, which were easy ($P<0.0001$).

Haq and Ullah[2] conducted a prospective observational study of 760 adult patients. This study determined that an ULBT of I predicted an easy intubation while a ULBT of II-III predicted a difficult intubation. Patients not able to perform either of the tests, or who were edentulous, pregnant, had a BMI>28kg/m2, or deformities of the head or neck were excluded from the study. There were 17.9% that had a CL III or IV. The UBLT sensitivity was 95.8% compared to 27.0% for the MMT. The specificity of the ULBT was 88.4% compared to 95.7% for the MMT. The ULBT PPV was 70.5%, compared to 64.7% PPV for the MMT. The ULBT NPV was 98.5% compared to an 82.0% NPV for the MMT. The ULBT had a statistically significant ($P<0.05$) higher percentage of correctly predicted difficult intubations compared to the MMT.

Reference	Sample/ Design	Airway Assessment	Sensitivity/Specificity	Conclusion
Ali et al., 2012[3]	Cross-sectional study N=324 Age>18yr ASA Not given	**E** **D** **MMT** I-II III-IV **ULBT** I-II III E=Easy D=Difficult	**MMT** **ULBT** **Sens** 19.6% 87.5% **PPV** 33.3% 71.6% **Spec** 91.8% 92.9% **NPV** 84.8% 97.3% Sens=Sensitivity Spec=Specificity	The ULBT showed a higher percentage of correctly predicted difficult intubations when compared to the MMT (P<0.05). Both ULBT and MMT were similar in correctly predicting easy intubations.
Badheka et al., 2016[4]	Single blinded prospective observational study N=170 Age 20-70yrs ASA I-III	**E** **D** **MMT** I-II III-IV **ULBT** I-II III	**MMT** **ULBT** **Sens** 78.9% 96.6% **PPV** 85.4% 92.7% **Spec** 68.6% 82.3% **NPV** 58.3% 91.3%	The ULBT had a higher percentage of predicting difficult intubations as well as easy intubations when compared to the MMT.
Wajekar et al., 2015[5]	Prospective comparative, double-blind study N=402 Age>18yrs ASA I-II	**E** **D** **MMT** I-II III-IV **ULBT** I-II III	**MMT** **ULBT** **Sens** 90.4% 98.6% **PPV** 91.0% 89.3% **Spec** 30.4% 8.7% **NPV** 29.2% 44.4%	The ULBT was slightly more specific in predicting difficult intubations. A high number of predicted easy intubations were difficult.
Honarmand et al., 2014[6]	Prospective observational blind study N=525 Age >18yrs ASA I-III	**E** **D** **MMT** I-II III-IV **ULBT** I II-III	**MMT** **ULBT** **Sens** 68.6% 90.2% **PPV** 13.6% 19.3% **Spec** 52.8% 59.4% **NPV** 94.0% 98.3% **AUC** 0.611 0.831	The ULBT was found to be more clinically accurate (P=0.0001). Many predicted difficult intubations were found to be easy for both the MMT and ULBT. The ULBT AUC was significantly more compared to the MMT AUC.
Safavi et al., 2014[7]	Randomized prospective double-blind study N=467 Age >18yrs ASA I-III	**E** **D** **MMT** I-II III-IV **ULBT** I-II III	**MMT** **ULBT** **Sens** 63.6% 75.7% **PPV** 8.2% 22.7% **Spec** 46.9% 80.8% **NPV** 94.4% 97.8% **AUC** 0.569 0.820	The ULBT was found to be more clinically accurate. The ULBT AUC was significantly more compared to the MMT AUC.

Safavi et al., 2011[8]	Prospective observational study N=603 Age >18yrs ASA I-III		**E**	**D**		**MMT**	**ULBT**	A high number of intubations MMT predicted easy, were difficult. The ULBT AUC was significantly more compared to the MMT AUC.
		MMT	I-II	III-IV	**Sens**	87.3%	66.0%	
		ULBT	I-II	III	**PPV**	93.3%	97.1%	
					Spec	14.6%	73.1%	
					NPV	7.8%	13.6%	
					AUC	0.511	0.709	
Shah et al., 2013[9]	Prospective observational study N=480 Age >18yrs ASA I-II		**E**	**D**		**MMT**	**ULBT**	The ULBT had a slightly higher predictability of difficult intubations, however, it had a significantly higher percentage of identifying easy intubations. A high number of MMT predicted difficult, were easy.
		MMT	I-II	III-IV	**Sens**	70.1%	74.6%	
		ULBT	I-II	III	**PPV**	22.6%	58.8%	
					Spec	61.0%	91.5%	
					NPV	92.6%	95.7%	
Haq & Ullah, 2013[2]	Prospective observational study N=760 Age >18yrs ASA I-III		**E**	**D**		**MMT**	**ULBT**	The ULBT had a higher percentage of correctly predicted difficult intubations compared to the MMT, which was statistically significant (P <0.05).
		MMT	I-II	III-IV	**Sens**	27.0 %	95.8%	
		ULBT	I	II-III	**PPV**	64.7%	70.5%	
					Spec	95.7%	88.4%	
					NPV	82.0%	98.5%	

MMT= Modified Mallampati Test
ULBT= Upper Lip Bite Test
Sensitivity= The percentage of difficult intubations which were predicted to be difficult.
PPV= Positive predictive value. The percentage of predicted difficult intubations which were difficult intubations.
Specificity= The percentage of easy intubations which were predicted to be easy.
NPV= Negative predictive value. The percentage of predicted easy intubations which were easy intubations.
AUC= Area under the curve. An area of 1 represents a perfect test, or 100% accuracy; an area of 0.5 represents a worthless test, or accuracy no greater than chance.

Conclusion

Seven of the eight studies found the ULBT to have a higher sensitivity indicating that when the ULBT predicted a difficult intubation, a higher number were difficult. Safavi et al[8] did not find a higher sensitivity, however, the ULBT AUC was significantly greater (P<0.0001) than the MMT. Three studies included an AUC, which represents both the sensitivity and specificity and is considered a measure of accuracy. All found that the ULBT AUC was significantly more than the MMT AUC.[6,7,8] There was a wide variation in the specificity or number of predicted easy

intubations which were easy, 92.9% to 8.7% for the ULBT. In the Wajeker et al[5] study, the ULBT specificity was low at 8.7% with a large number of predicted easy intubations that were actually difficult. The study was an outlier with the next lowest specificity reported at 59% for the ULBT. The lowest specificity for the MMT was 14.6% in the Safavi et al[8] study, whereas the Wajeker et al[5] study reported a MMT specificity of 30.4%.

Some limitations of this evidence-based practice analysis are that all studies included were performed in countries outside the United States (US). This may not take into account the general population and ethnicities found in the US. Another limitation was that one person in each study conducted all assessments. The results could be skewed if the individual did one or both of the assessments poorly. The ULBT also has limitations; it may not be performed in all patients such as an edentulous and non-cooperative patient or patients unable to move their jaw. The ULBT is not a perfect tool to correctly identify 100% of patients with a difficult intubation; however, the review of studies reported a higher level of accuracy when compared to the MMT in predicting difficult intubation.

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Mentor: Sharon Hadenfeldt, PhD, CRNA

Increasing Use of Sugammadex among Anesthesia Providers

Sarah Niemann, DNP
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Keywords: Sugammadex, neostigmine, glycopyrrolate, neuromuscular blockade, reversal, rocuronium, vecuronium

Introduction

Residual neuromuscular blockade (NMB) persists in up to 41% in patients after receiving an anticholinesterase inhibitor.[1] Anticholinesterase inhibitors competitively antagonize acetylcholinesterase to inhibit breakdown of acetylcholine in the synaptic cleft, achieving antagonism of neuromuscular blockade that is often incomplete, and not without potential significant adverse effects.[1] Sugammadex, approved by the U.S. Food and Drug Administration in 2015, acts by binding with rocuronium or vecuronium to terminate NMB.[2] An internal record review at a large academic medical center revealed that anesthesia practitioners had not yet optimized use of sugammadex. A survey of anesthesia practitioners revealed barriers to sugammadex use including knowledge about the drug, cost, and access to the drug. This evidence-based practice project investigates whether sugammadex use by anesthesia practitioners increased after implementation of an education session and dosing guide.

Methods

Institutional Review Board determination as not human subjects research was obtained. A thirty-minute education presentation on sugammadex was given for anesthesia practitioners on August 2, 2017. Anesthesia practitioners were instructed on sugammadex dosing, institutional pricing, and administration criteria in special populations extracted from the literature. The project team collaborated with pharmacy administration to expand access to sugammadex to all operating room Pyxis medication dispensing stations. A sugammadex dosing guide was developed and made available in each operating room.

A pre and post-intervention record review was conducted on patients age 18 or greater, ASA Class I-IV, who underwent a surgical procedure requiring rocuronium or vecuronium between the dates of July 1-August 1, 2017 (pre-intervention group) and August 2-31, 2017 (post-intervention group). Data collected included the following variables: type of NMBA administered; the type of surgical procedure; special populations including neuromuscular disease, COPD, OSA, nutritionally poor or frail, and a BMI > 40; if the encounter was a “can’t

intubate/can't ventilate" situation; whether or not sugammadex was administered; surgery stop time; and out of room time. A McNemar's test was performed to assess statistical significance. SPSS software (SPSS Version 20; SPSS Inc., Chicago, IL) was used to analyze the data on sugammadex utilization.

Outcome

There were 170 patients included in the pre-intervention group. Of these patients, 140 received NMB reversal. Of the 111 patients meeting criteria for sugammadex, only 8 (7.2%) received it. In the post-intervention group, there were 175 patients. Of these patients, 145 received NMB reversal. Fourteen patients (12.7%) received sugammadex despite 110 patients meeting criteria for sugammadex administration. A McNemar's test of the data using a 0.05 significance level resulted in a p-value of 0.286.

Conclusions

Use of sugammadex increased by 75% post-education, however, this was not statistically significant ($p = 0.286$). Sugammadex did not become available in all operating rooms until after the chart review was completed, which is postulated to be a barrier to success of this project. Other barriers included direction from the pharmacy to limit use of sugammadex due to cost. Acquisition costs for sugammadex are higher, but sugammadex administration may result in reduced indirect costs associated with residual NMB. Residual NMB is associated with substantial morbidity and mortality due to inadequate airway protection, aspiration, pneumonia, airway obstruction, hypoxia, and blockade of the diaphragm and pharyngeal muscles.[3,4] The project team has continued education with the pharmacy on the value of sugammadex.

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Mentor: Kelli Pryor, DNP, CRNA

Editorial

This summer went by particularly fast, and its end signals the release of another issue of the ISJNA. This larger issue features a wide variety of interesting topics, and the first Evidence-Based Practice Project Abstract. I would also like to bring attention to the compelling story that accompanies the cover art for this issue. William Gafford and Newton Tinsley are among 16 graduate students in Alabama, and 240 nationally who spent the past year learning to effectively address the social factors that impact health and developing lifelong leadership skills. Mr. Gafford and Mr. Tinsley developed and implemented their service project while also fulfilling their academic and clinical responsibilities as graduate students enrolled in Samford University's Nurse Anesthesia Program – no small task! I expect we will see and hear more of Mr. Gafford and Mr. Tinsley after they graduate as leaders within our profession.

Sincerely,

Vicki C. Coopmans, PhD, CRNA
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.**"

INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA

GUIDE FOR AUTHORS

MISSION STATEMENT

The International Student Journal of Nurse Anesthesia (ISJNA) 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.

ITEM PREPARATION & SUBMISSION

Case reports, research abstracts, evidence-based practice (EBP) analysis reports, evidence-based practice project abstracts, 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). Case and EBP analysis reports must be single-authored, while abstracts may have multiple authors. Submissions may list only one mentor. **Mentors should take an active role** in reviewing the item to ensure appropriate content, writing style, and format prior to submission. 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. Authors and mentors should critically evaluate the topic and quality of the writing – multiple reviews of the item by the mentor, faculty, and peers (fellow graduate students) prior to submission is recommended. If the topic and 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*.

The journal is committed to publishing the work of nurse anesthesia students. The review process is always initiated with the following rare exceptions. We are conservative in accepting reports where the patient has expired, realizing that you can do everything right and still have a negative outcome. Submissions that report a case demonstrating failure to meet the standard of care (by any practitioner involved in the case) will not be accepted. Unfortunately, while the experiences in these cases can offer valuable insight, these submissions will not be accepted for review due to potential legal risks to the author, journal, and anyone else involved in evaluating the report.

It is the intent of this journal 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** (4-6 months recommended) to the author's date of graduation. 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 use the following format: ISJNA Submission_submission type_author last name_mentor last name. 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 PROCESS

Items submitted for publication are initially reviewed by the chief editor. If the chief editor does not acknowledge receipt of the item within two weeks, please inquire to ensure receipt. Upon receipt, the chief editor will review the submission for compliance with the Guide to Authors. If proper format has not been followed, 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.

All accepted submissions undergo a formal process of blind review by at least two reviewers. After review, items may be accepted without revision, accepted with revision, or rejected with comments. Once the item has been accepted for review the chief editor will send a blinded copy to an editor, who will then coordinate a blinded review by two reviewers who are not affiliated with the originating program. The editor will return the item to the chief editor, who will return it to the mentor for appropriate action. Every effort is made to complete the process in an efficient, timely matter. Again, the goal is for all articles submitted by students to be published while the author is still a student. If an item is not ready for publication within 6 months after the student author has graduated it will no longer be eligible for publication. Mentors 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. Please contact the chief editor at intsjna@aol.com to submit photos for consideration. Only digital photos of high quality will be accepted. If the photo is accepted, consent forms must be completed and returned by all identifiable individuals in the photo, and the individual who took the photo.

ACADEMIC INTEGRITY

Issues of academic integrity are the responsibility of the author and mentor. Accurate and appropriate acknowledgement of sources is expected. The two most common breaches of academic integrity that have been identified in submissions to this journal are (AMA 10th ed., p. 158):

1. Direct plagiarism: verbatim lifting of passages without enclosing the borrowed material in quotation marks and crediting the original author.
2. Paraphrase: restating a phrase or passage, providing the same meaning but in a different form without attribution to the original author.

Please note that changing one or two words in a reference source passage (e.g. 'of' for 'in', or 'classified' for 'categorized') and then citing it as a paraphrase or summary is also not appropriate, and still falls within the definition of direct plagiarism. If plagiarism in any form is identified, review of the item will be suspended and it will be returned to the mentor. Repeated instances of plagiarism will result in rejection of the item.

Plagiarism detection software (TurnItIn, PlagScan, SafeAssign, etc . . .) can be used to analyze the document prior to submission to ensure proper citation and referencing, but is not required.

"Plagiarism is the presentation of someone else's ideas, writings, or statements as one's own. Plagiarism is a serious breach of academic integrity, and anyone who is found to have committed plagiarism will be subject to disciplinary action.
Paraphrase is the act of putting someone else's ideas into one's own words. The use of paraphrase can be an acceptable practice under some circumstances if it is used sparingly and if the original text is properly acknowledged. Unacknowledged paraphrase, like plagiarism, is a serious breach of academic integrity. Any improper use of sources may constitute plagiarism. Every quotation from another source, whether written, spoken, or electronic, must be bound by quotation marks and be properly cited. Mere citation alone is not sufficient when a scholar has used another person's words. Similarly, every paraphrase or summary (a more concise restatement of another's ideas) must be properly cited."
https://sites.google.com/a/georgetown.edu/gsas-graduate-bulletin/vi-academic-integrity-policies-procedures

GENERAL GUIDELINES

Items for publication **must adhere to the *American Medical Association Manual of Style*** (AMA 10th ed., the same guide utilized by the *AANA Journal* and such prominent textbooks as *Nurse Anesthesia* by Nagelhout and Plaus). Page numbers are provided for easy reference in the AMA Manual of Style throughout this document. The review process will not be initiated on items submitted with incorrect formatting and will be returned to the mentor for revision. Please note the following:

1. Use complete sentences.
2. Acronyms/Initialisms (p. 379) - spell out with first use, do not capitalize the words from which the acronym/initialism is derived unless it is a proper noun or official name. If you are using the phrase only once, do not list the acronym/initialism at all. Avoid beginning sentences with acronym/initialisms.
3. Abbreviations (p. 441)
4. Use *Index Medicus* journal title abbreviations (p. 472, http://www.ncbi.nlm.nih.gov/nlmcatalog/journals)
5. Always provide units of measure (p. 521 & 795). 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. Report height in cm, weight in kg, temperature in ^{o}C, pressure in mm Hg or cm H_2O. Report heart and respiratory rate as X/min (e.g. the patient's heart rate increased to 145/min).
6. 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, $EtCO_2$, N_2O. Please use SpO_2 for oxygen saturation as measured by pulse oximetry.

7. Use the nonproprietary (generic) name of drugs (p. 568) - avoid proprietary (brand) names. Type generic names in lowercase. When discussing dosages state the name of the drug, *then* the dosage (midazolam 2 mg).
8. 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 (p. 583, e.g. a GlideScope (Verathon Inc., Bothell, WA) was used) Please note, TM and ® symbols are not used per the AMA manual.
9. Infusion rates and gas flow rates:
    a. Use mcg/kg/min or mg/kg/min for infusion rates. 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. Report gas flow of O_2, N_2O and Air in L/min (not %) and volatile agents in % as inspired or expired concentration (e.g. General anesthesia was maintained with sevoflurane 3% inspired concentration in a mixture of O_2 1 L/min and air 1 L/min.)
10. 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. 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.
11. Do not use Endnotes or similar referencing software – any embedded formatting must be removed prior to submission.
12. Remove all hyperlinks within the text.
13. Avoid jargon and slang terms. Use professional, scholarly, scientific language.
    a. *'The patient was reversed'* - Did you physically turn the patient around and point him in the opposite direction? "Neuromuscular blockade was antagonized."
    b. *The patient was put on oxygen.* "Oxygen 2 L/min was administered via face mask."
    c. *The patient was intubated and put on a ventilator*. "The trachea was intubated and mechanical ventilation was initiated.
    d. *An IV drip was started.* "An intravenous infusion was initiated."
    e. Avoid the term "MAC" when referring to a sedation technique - the term sedation (light, moderate, heavy, unconscious) may be used. Since all anesthesia administration is monitored, pharmacologic, rather than reimbursement, terminology should be used.
14. Direct quotes are discouraged for reports of this length – please express in your own words.
15. Use the words "anesthesia professionals" or "anesthesia practitioners" when discussing all persons who administer anesthesia (avoid the reimbursement term "anesthesia providers").
16. Do not include ASA Physical Status unless it is germane to the report.
17. Do not use the phrase "ASA standard monitors were applied". Instead, "standard noninvasive monitors" is acceptable – additional monitoring can be detailed as needed.
18. References
    a. The **AMA Manual of Style must be adhered to** for reference formatting.
    b. All sources should be published within the past 8 years. Seminal works essential to the topic being presented will be considered.
    c. Primary sources are preferred.
    d. **A maximum of one textbook (must be most recent edition available) may be used as reference for case report submissions only**.
    e. All items cited must be from peer-reviewed sources – use of sources found on the internet must be carefully considered in this regard. URLs must be current and take the reader directly to the referenced source.

Heading – for all submission types (Case Report, Abstract, EBPA Report) use the following format.

1. **Title** is bolded, centered, 70 characters (including spaces) or less
2. Author name (academic credentials only) and NAP are centered, normal font,.
3. *Graduation date and email address* are centered, italicized, and will be removed prior to publication)
4. **Keywords** is left-justified, bolded – list keywords that can be used to identify the report in an internet search

Title

Author Name
Name of Nurse Anesthesia Program
Anticipated date of graduation
E-mail address

Keywords: keyword one, keyword two, etc . . .

Case Reports - The student author must have had a significant role in the conduct of the case. The total word count should be between 1200 – 1400 words (references not counted). 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 above)

A brief introductory paragraph of less than 100 words to focus the reader's attention and interest them to continue reading. 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.
[space]
Case Report (bold, 400-600 words)
[space]
This portion discusses the case performed 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. 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 heart of your paper which is the discussion and teaching/learning derived from the case.

- 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 of measure after the values (eg. Mmol/L or mg/dL).
- Physical examination/pre-anesthesia evaluation - **significant** findings only.
- Anesthetic management (patient preparation, induction, maintenance, emergence, post-operative recovery).

[space]
Discussion (bold, 600-800 words)
[space]
Describe the ***anesthesia*** implications of the focus of the case report citing current literature. Describe the rationale for your actions and risk/benefits of any options you may have had. This section is not merely a pathophysiology review that can be found in textbooks. *Relate the anesthesia literature with the conduct of your case noting how and why your case was the same or different from what is known in the literature.* Photographs are discouraged unless they are essential to the article. Photos with identifiable persons must have a signed consent by the person photographed forwarded to the editor via first class mail. Diagrams must have permission from original author. This is the most important part of the article. In terms of space and word count this should be longer than the case presentation. End the discussion with a summary lesson you learned from the case, perhaps what you would do differently if you had it to do over again.
[space]
References (bold)
[space]
A minimum of 5 references is recommended, with a maximum of 8 allowed. One textbook may be used as a reference – it must be the most recent edition. All references should be no older than 8 years, except for seminal works essential to the topic. This is also an exercise in searching for and evaluating current literature.
[space]
Mentor: (bold, followed by mentor name and credentials in normal text)
E-mail address: (normal text, 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, population, and outcome. 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. Textbooks and non-peer reviewed internet sources may not be used, and sources of reference should be less than 8 years old unless they are seminal works specifically related to your topic of inquiry. A maximum of 16 references is allowed.

Heading

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]
Methods (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]
Analyze and critique the literature relevant to your question, determining scientific credibility and limitations of studies reviewed. Your synthesis table is 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, 16 maximum)
[space]
Mentor: (bold, followed by mentor name and credentials in normal text)
E-mail address: (normal text, will be removed prior to publication)

Evidence Based Practice Project Abstracts - Evidence-based practice project abstracts are limited to 600 words. References do not impact the word count - a maximum of 5 are allowed. Note that the abstract is different from a project proposal. The following format should be used:

Heading

Introduction (bold)
[space]
A brief introductory paragraph including purpose (what change is intended) and rationale (why change is needed/evidence to support the change) here.

[space]
Design and Methods (bold)
[space]
Include population, intervention, and measures
[space]
Outcome (bold)
[space]
Present results from statistical analysis – do not justify or discuss here.
[space]
Conclusion (bold)
[space]
Discuss results (implications). Optionally include limitations, suggestions for future projects/research.
[space]
References (bold, 5 maximum)
[space]
Mentor: (bold, followed by mentor name and credentials in normal text)
E-mail address: (normal text, will be removed prior to publication)

Research Abstracts - Research abstracts are limited to 600 words. References do not impact the word count - a maximum of 5 are allowed. Note that the abstract is different from a research proposal. The following format should be used:

Heading

Introduction (bold)
[space]
A brief introductory paragraph including purpose and hypotheses.
[space]
Methods (bold)
[space]
Include sample and research design
[space]
Results (bold)
[space]
Present results from statistical analysis – do not justify or discuss here.
[space]
Discussion (bold)
[space]
Discuss results (implications, limitations, suggestions for future research)
[space]
References (bold, 5 maximum)
[space]
Mentor: (bold, followed by mentor name and credentials in normal text)
E-mail address: (normal text, will be removed prior to publication)

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. Some helpful websites are listed below:

https://guides.nyu.edu/amastyle
https://owl.english.purdue.edu/owl/resource/1017/01/

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.

Journals - A comma is placed 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). Page numbers are inclusive - **do not omit digits** (note - some online journals do not use page numbers). Some journals 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:
Han B, Liu Y, Zhang X, Wang J. Three-dimensional printing as an aid to airway evaluation after tracheotomy in a patient with laryngeal carcinoma. BMC Anesthesiol. 2016;16(6). doi:10.1186/s12871-015-0170-1.

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.

Elayi CS, Biasse L, Bai R, et al. Administration of isoproterenol and adenosine to guide supplemental ablation after pulmonary vein antrum isolation. *J Cardiovasc Electrophysiol*. 2013;24(11):1199-1206. doi: 10.1111/jce.12252.

Electronic references - Only established, peer-reviewed sources may be referenced. Please do not reference brochures, fact sheets, or informational websites where a peer-review process cannot be confirmed. The URL must be functional and take the reader directly to the source of the information cited. The accessed date may be the only date available.

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

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

Howlader N, Noone AM, Krapcho M, Garshell J, Miller D, et al. SEER Cancer statistics review, 1975-2012. National Cancer Institute. http://seer.cancer.gov/csr/1975_2012/. Published April 2015. Updated November 18, 2015. Accessed February 29, 2016.

Textbooks - There are two types of books – 1) those that are fully authored by one or more individuals, and 2) those that are edited by one or more individuals, with chapters authored by different individuals. Edited textbooks give primary credit to the chapter authors, who are listed first, and the inclusive page numbers of the entire chapter are provided at the end. Textbooks that are authored do not have different chapter authors and the chapter titles are not listed, but the inclusive page numbers where the information was found are provided, unless the entire book is cited.

Authored text:
Shubert D, Leyba J, Niemann S. *Chemistry and Physics for Nurse Anesthesia*. 3rd ed. New York, NY: Springer; 2017:405-430.

Chapter from an edited text:
Pellegrini JE. Regional anesthesia. In Nagelhout JJ, Elisha S, eds. *Nurse Anesthesia*. 6th ed. St. Louis:Elsevier; 2017:1015-1041.

SUBMISSION CHECK LIST

___ **Adheres to AMA Manual of Style and all other format instructions**
___ Total word count not exceeded (1400 for case report, 600 for abstracts, 3000 for EBPA report)
___ The item is one continuous Word document without artificially created page breaks
___ All matters that are not common knowledge to the author are referenced appropriately
___ 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/slang is absent

Heading
___ Concise title less than 70 characters long
___ Author name, credentials, nurse anesthesia program, graduation date and email are included
___ Three to 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 400-600 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

Abstracts
___ The 600 word count maximum is not exceeded
___ Appropriate format used depending on type of abstract (research vs. EBP project)

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, population, and outcome is presented
___ A focused foreground question following either the PICO or SPICE format is used
___ Includes Introduction, Methodology, Literature Analysis (with synthesis table), and Conclusion sections

References
___ Adheres to AMA Style format
___ Reference numbers are sequenced beginning with 1 and superscripted
___ References are from anesthesia and other current (within past 8 years) primary source literature
___ Journal titles are abbreviated as they appear in the PubMed Journals Database
___ Number of references adheres to specific item guidelines (1 textbook allowed for case reports only)
___ 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)
___ Item is submitted by the mentor
___ Subject heading format - ISJNA Submission_submission type_author last name_mentor last name