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TOPICS IN THIS ISSUE

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Front Cover:
The front cover depicts doctoral students enrolled in the Missouri State University (MSU) nurse anesthesia program engaging in various activities. On the left, Jace Corray, BSN, RN practices epidural placement on a task trainer. On the bottom right, from left to right, Roman Davidyuk, BSN, RN, James Cadle, BSN, RN, Andrew Lacek, BS, RN, and Steve Chambers, BSN, RN participate in a clinical scenario in MSUs simulated operating room. At the top right, Thomas Nance, BSN, RN prepares his set-up for a clinical day. Mr. Nance has a case report published in this issue of the ISJNA.

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Anesthetic Management of a Pediatric Patient with CHARGE Syndrome

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Keywords: CHARGE syndrome, choanal atresia, tracheomalacia

CHARGE syndrome is a rare genetic disorder with autosomal dominance involving the mutation of gene CHD7 on chromosome 8.[1] CHARGE is an acronym for **c**oloboma, **h**eart disease, **a**tresia choanae, **r**etardation of growth, **g**enitourinary malformation, and **e**ar abnormalities.[2] Patients with CHARGE syndrome have multiple congenital anomalies requiring them to undergo many surgeries in the neonatal period and throughout childhood. Delivery of general anesthesia can be challenging due to the structural and functional anatomical abnormalities that frequently accompany CHARGE syndrome.[2]

Case Report

A 4-year-old male presented to the operating room (OR) for bronchoscopy with bronchoalveolar lavage, nasal endoscopy, and laryngoscopy for excision of granuloma. Diagnoses warranting the procedure were choanal atresia, tracheomalacia, and chronic respiratory failure, all conditions related to CHARGE syndrome. He was also diagnosed with choroid coloboma and microphallus, other anomalies commonly associated with CHARGE syndrome. The patient had undergone this procedure multiple times and had an extensive surgical history to correct and manage his airway anomalies. Assessment of the airway revealed a size 4 cuffed tracheostomy tube which was placed at 2 months of age, along with a gastrostomy tube. The patient had a history of difficult intubation prior to placement of the tracheostomy. Cardiac assessment revealed normal sinus rhythm with expected left ventricular systolic function and no evidence of pulmonary hypertension. Neurologically, he had cognitive developmental delay with a reported improvement in mentation. While sleeping, the patient was dependent on continuous positive airway pressure (CPAP) with synchronized intermittent ventilation pressure-controlled ventilation (SIMV-PCV) and positive end-expiratory pressure (PEEP).

The patient arrived to the operating room in a transport crib from the pediatric intensive care unit (PICU) with a speaking valve attached to the tracheostomy tube. He had a significant amount of white frothy nasal secretions which were cleared before induction of anesthesia. The patient was positioned in the supine position on the OR table and standard noninvasive monitors were applied. Initial vital signs were stable with heart rate 85/min, SpO_2 98% on room air, and blood pressure 87/51 mmHg. The ECG displayed normal sinus rhythm. Anesthesia was induced with propofol 100 mg and fentanyl 15 mcg IV. After the patient became apneic, the circuit was attached to his tracheostomy tube and manual bag ventilation was initiated utilizing sevoflurane and O_2 at 2 L/min.

The bronchoscopy and bronchoalveolar lavage were performed by a pediatric pulmonologist. The bronchoscope was introduced through the tracheostomy to the tracheobronchial tree and bronchoalveolar lavage was performed to the middle lobe of the right lung. The bronchoscope occupied the airway and obstructed ventilation, resulting in brief episodes of desaturation as low

as 80%. This was anticipated and treated by reconnecting the circuit to the tracheostomy tube and manually ventilating the patient with 100% oxygen until an oxygen saturation of 100% was achieved. The nasal endoscopy and laryngoscopy with excision of granuloma was then performed by the otolaryngologist. There was marked improvement in the patency of the choanocytes from a recent surgical repair of his choanal atresia. However, a suprastomal granuloma just above the indwelling tracheostomy tube was noted. Excision of the mass required intermittent removal of the tracheostomy tube. The surgeon decannulated the patient to remove the mass but only for as long as was tolerated, between 1-2 minutes. To treat periods of desaturation, the patient was intermittently recannulated, reconnected to the circuit, and manually ventilated with O_2 10 L/min until his SpO_2 returned to 100%. At the end of the procedure, the patient was suctioned through the tracheostomy with a soft suction catheter. Vital signs were stable at baseline values. The patient was breathing independently without difficulty and emerged from general anesthesia without incident. The tracheostomy was capped with the patient's speaking valve and he was transported back to the PICU.

Discussion

The incidence of CHARGE syndrome is presumed to be 0.1-1.2/10,000 births.[2] Coloboma and other ocular abnormalities are seen in up 90% of these patients. Cardiac defects have been reported in 80% of cases, while choanal atresia is reported in 65% off cases. Up to 50% of CHARGE syndrome patients require tracheotomy for a variety of airway abnormalities, chronic aspiration, and swallowing disorders.[1,2]

Choanal atresia is the complete blockage of the posterior nasal opening due to enlargement of the pterygoid plate during fetal development. Nasal breathing is essential to newborns, but because of the complete nasal obstruction from the malformed pterygoid plate, respiratory problems may develop after birth.[1,4] Patients may require intubation or tracheotomy to manage their obstructed airway. The most common clinical symptoms of choanal atresia are unilateral thick mucoid nasal discharge or unilateral sinusitis.[4] Upon arrival to the OR, this patient had thick mucoid secretions draining from the nares. Patients with choanal atresia are considered at high risk for experiencing difficulty with airway management during general anesthesia.[4] Patients typically are given a grade 3 or 4 Cormack-Lehane classification during direct laryngoscopy (DL), indicating that either only the epiglottis is visible or the epiglottis was not visible at all during DL.[4] This patient had a tracheostomy tube and thus laryngoscopy was not required.

The presence of choanal atresia along with other upper airway and oral cavity anomalies can cause dysphagia, frequently leading to feeding problems. Additionally, most children with CHARGE syndrome have cranial nerve abnormalities: the glossopharyngeal and vagus nerves in particular.[1] These nerves are responsible for sensory and motor innervation to the pharynx respectively, which allow swallowing to occur.[3] Additionally, the vagus nerve supplies motor innervation to the larynx. For this reason, patients with CHARGE syndrome are at risk for complications during anesthesia due to the high risk for regurgitation and aspiration from difficulty in protecting their airway.[3] This patient had a history of severe dysphagia which necessitated the insertion of a gastric tube for effective feeding.

Tracheomalacia results from a weakness of the supporting structures of the trachea. This stems from atrophy of the elastic fibers and impaired cartilage integrity leading to tracheal collapse, particularly during inspiration.[5] Similarly, laryngomalacia is an impaired structural support of the larynx.[4] These are both common in CHARGE syndrome, contributing to airway collapse during anesthesia. This may lead to the required use of CPAP, particularly during light anesthesia. This patient's procedural diagnosis was tracheomalacia, but the presence of a tracheostomy tube and provision of positive pressure ventilation throughout the procedure prevented airway collapse.

Eighty percent of patients with CHARGE syndrome have congenital heart defects.[6] A variety of cardiac anomalies have been reported in these patients. A frequently associated cardiac defect is tetralogy of Fallot, which is found in 33% of CHARGE patients.[6] Sevoflurane has been shown to be well tolerated in these patients as it does not cause airway irritation or tachycardia. Additionally, sevoflurane is preferred because it has low blood-gas solubility and provides a fast and smooth induction.[4] Propofol is the most commonly used induction agent.[4] However, these anesthetics must be used with high caution in CHARGE syndrome patients with tetralogy of Fallot. A decrease in systemic vascular resistance resulting from the vasodilating properties of sevoflurane and propofol can be detrimental. The patient presented in this case report did not have a medical history of cardiac defects. Sevoflurane was used as the primary anesthetic and propofol was used for induction. The patient tolerated both anesthetic agents well.

When preparing an anesthetic for a patient with CHARGE syndrome, one should consider the following steps. Anticipate that patients with CHARGE syndrome will have difficult airways. Preparation is important because it will make the intubation process easier and reduce complications that might arise.[1,4] Prepare and have available endotracheal tubes (ETTs) of different types and sizes, supraglottic airways, and a video laryngoscope. Prepare the ETT with a hockey stick-like J-curvature at the tip. This has been shown to be more advantageous for placing the tip of the ETT in the glottis.[2] Because the pharynx and larynx of CHARGE syndrome patients can be smaller than expected, prepare a smaller sized laryngeal mask airway to be used first.[4] Have a fiberoptic bronchoscope ready and prepare for a surgical tracheostomy for patients that do not already have one in place. Equipment needed for cardiopulmonary resuscitation should be made available. Anticipate critical events such as laryngospasm, bronchospasm, and reintubation. Although these events are less likely related to CHARGE syndrome, a knowledge of the disorder and associated conditions is necessary for the safe anesthetic care of these fragile patients.[4]

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Case Study of a Patient with Subclavian Steal Syndrome

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Keywords: Subclavian steal syndrome, carotid subclavian bypass, thoracic aortic ring, general anesthesia

Subclavian steal phenomenon (SSP) refers to the stenosis or occlusion of the subclavian artery proximal to its origin from the vertebral artery.[1] Presence of SSP facilitates retrograde flow in the ipsilateral vertebral artery towards the upper arm.[1,2] Patients are usually asymptomatic, developing collateral blood supply from the head, neck and shoulder.[2] Subclavian steal syndrome (SSS) is the presence of neurological symptoms, occurring when there is vertebrobasilar insufficiency from increased compensatory blood flow to the subclavian artery from the vertebral artery.[2] Anesthetic management requires general anesthesia with the avoidance of hypotension to promote cerebral blood flow to the circle of Willis.[3]

Case Report

The patient was a 21-year-old male with a clinical history of subclavian steal syndrome of the right subclavian artery, repair of a vascular ring from a left aortic arch with aberrant right subclavian artery in childhood, ligation and occlusion of the proximal replaced right subclavian artery, recurrent syncopal episodes for 10 months, acrocyanosis, systemic lupus erythematosis, juvenile asthma, hyperlipidemia and childhood developmental delay. He weighed 122 kg and his height was 191.77 cm, with a body mass index of 34.17 kg/m^2. He had no known drug allergies. The patient was scheduled for a right carotid subclavian bypass. All laboratory values, including prothrombin time (PT), thromboplastin time (PTT), and international normalized ratio (INR), were within normal limits. An echocardiogram showed no significant findings and an ejection fraction of 67%. Cardiology, neurology, epilepsy, vascular, autonomic, endocrine, allergy, immunology, and sleep studies were conducted, and all returned as negative. The patient also reported numbness to the right upper extremity and was noted to have a weak right radial pulse compared to the left radial pulse. The patient was one of four male siblings with the recurring syncopal episodes and genetic testing was in process.

The presumptive diagnosis of subclavian steal syndrome was inferred from a documented history of cauterization of a vascular ring from an aberrant right subclavian artery in infancy. The anesthetic plan was devised given the nature of the procedure and the disorder of the patient. The patient was placed on standard noninvasive monitors prior to induction of general anesthesia. Bispectral index (BIS) and somatosensory evoked potential (SSEP) monitoring was used. He had

a smooth intravenous (IV) induction with midazolam 2 mg, fentanyl 100 mcg, lidocaine 40 mg, propofol 180 mg, rocuronium 40 mg and hydrocortisone 50 mg. The patient was taking fludrocortisone 0.2 mg daily for greater than 6 months for his lupus. A stress dose of hydrocortisone 50 mg was given IV prophylactically, as maintenance of hemodynamic stability was crucial to maintain collateral circulation. The patient was an easy mask ventilation, followed by endotracheal intubation and subsequent mechanical ventilation. A left radial arterial line was placed after induction for continuous blood pressure monitoring. Because the patient also reported experiencing syncopal episodes when overheated, a lower body Bair hugger was placed on the patient but was not turned on due to a core temperature of 36º C. Heparin 3000 U was given IV prior to incision as requested by the surgeon. Anesthesia was maintained with a propofol infusion of 100 mcg/kg/min and a remifentanil infusion at 0.2 mcg/kg/min. Boluses of ephedrine 5 mg and phenylephrine 100 mcg were given incrementally to support hemodynamic stability. A phenylephrine infusion of 0.2 mcg/kg/min was initiated and titrated to effect. Glycopyrrolate 0.2 mg IV was given for a heart rate of 50/min. Heparin 15,000 U IV was given prior to clamping of the carotid artery. After a sudden 400 mL blood loss, 25% albumin 100 mL was infused over 30 minutes. The phenylephrine infusion was adjusted to maintain mean arterial pressure (MAP) between 80 to 110 mm Hg in response to the blood loss. After cross-clamp removal, protamine 120 mg IV was given. Acetaminophen 1 g and hydromorphone 1 mg were given IV for analgesia and ondansetron 4 mg IV was given for antiemesis. Neuromuscular blockade was antagonized with sugammadex 250 mg IV. The patient received a total of 3,700 mL of lactated Ringers and 450 mL of 0.9% normal saline with an estimated blood loss of 1,600 mL. All anesthetics and vasopressors were weaned off and spontaneous ventilation was resumed with the patient following commands prior to extubation. He was transported to the post anesthesia care unit (PACU) with stable vital signs and then transferred to the surgical intensive care unit (SICU) for further observation prior to discharge to home the next day.

Discussion

Subclavian steal syndrome is a term that was first used in 1961 to encompass the neurological symptoms that would present during or immediately after exertion of the ipsilateral arm.[3] Subclavian steal phenomenon is the result of a steno-occlusive disease of the subclavian artery proximal to the vertebral artery, leading to the reversal of flow in the vertebral artery on the ipsilateral side.[4] The phenomena is mostly observed in patients older than 55 years of age and in males at a 2:1 ratio. [1,2] Subclavian steal syndrome occurs on the left side more often at a 4:1 ratio.[1,2] Left-sided subclavian steal is thought to be more common due to the acute angle of origin of the left subclavian artery which increases turbulent flow and catalyzes atherosclerosis at the subclavian-aortic junction.[1] The risk factors for SSS are increased with smoking, hyperlipidemia, hypertension, diabetes mellitus, family history and age.[1]

Due to collateral circulation from the shoulder, patients with subclavian steal are usually asymptomatic.[3] Another clinical presentation is upper extremity ischemia that presents more often than neurologic symptoms.[4] The etiology of upper limb ischemia can originate from acute and chronic conditions.[3] Chronic large vessel disease, particularly arteriolosclerosis, is the most common cause of both subclavian steal and SSS.[3] Chronic small vessel diseases such as systemic lupus erythematosis and rheumatoid arthritis can cause limb ischemia.[3] Upper extremity ischemia manifests as exercise induced arm pain, fatigue, coolness, paresthesia, or

numbness.[2] A difference in blood pressure in both arms greater than 40 mmHg and a weakened radial pulse may also be present and indicate the need for intervention.[1,3,4] However, the gold standards for diagnosis of SSS are computed tomography or magnetic resonance angiography.[5]

Other documented causes of SSS, particularly of the right subclavian, include arteritis, trauma, tumor thrombus, previous surgical procedures such as Blalock-Taussig shunt and congenital aortic interruption.[6] Congenital vascular diseases such as vascular rings including a right aortic arch with an isolated left subclavian artery and more rarely, a normal left aortic arch with an isolated right subclavian artery have been noted to cause SSS.[6] The term vascular ring encompasses any congenital vascular anomalies of the aortic arch that cause a compression of the trachea and/or esophagus leading to respiratory and gastrointestinal issues.[7] A normal left aortic arch with origin of the right subclavian artery from the descending thoracic aorta is one of the more common vascular ring abnormalities.[7] Once diagnosed, surgical intervention is highly recommended and involves relocation of the right subclavian artery which can subsequently lead to SSS from ligation and occlusion of the proximally replaced right subclavian artery.[7] Typically in infancy, a right common carotid subclavian artery transposition precedes a cauterization or ligation of an aberrant subclavian artery.[7] However, this was not the case for this patient, possibly due to the presence of adequate collateral circulation.

Ischemia of the vertebrobasilar artery (which supplies both the peripheral and central auditory and vestibular systems) is uncommon, unless there are other cerebrovascular lesions.[2,4] Neurologic symptoms, including vertigo (most common), visual disturbances such as double vision and visual loss, and transient loss of consciousness may occur due to ischemia in the posterior cerebral circulation and stenosis or occlusion of the proximal vertebral artery.[3] Exertion of the affected upper extremity can precipitate neurologic symptoms, though the development of collateral circulation may prevent symptoms.[4]

Surgical interventions for SSS include carotid-subclavian bypass, axillo-axillary bypass, or percutaneous transluminal angioplasty of the subclavian artery with stent placement.[2] The goals of these surgical procedures are to restore the antegrade vertebral artery flow, alleviate cerebral hypoperfusion, and improve arterial perfusion to the upper extremity.[2] For patients with steno-occlusive subclavian disease, carotid-subclavian bypass surgery has been used successfully with patency rates reported as high as 95% at 10 years and no mortality.[1]
It can be presumed that patients diagnosed with SSS would likely have the development of collateral circulation and anesthesia should be managed to maintain this circulation. The use of general anesthesia is usually the preferred anesthetic technique and should include a large bore IV and continuous hemodynamic monitoring. The major risk associated with the use of general anesthesia is the potential for intraoperative hemodynamic instability.[8]
It has been reported that even brief periods of hypotension can adversely affect end-organ function; therefore, blood pressure should be maintained within 20% of the patient's preoperative baseline prior to clamping of the carotid artery.[5] The use of continuous blood pressure monitoring modalities (such as a contralateral arterial line) is recommended to reduce periods of hypotension and decrease intervention time with vasoactive medications.[3,5]
Maintaining a MAP 20% above baseline or systolic blood pressure above 150 mmHg during clamping of the carotid artery is needed for controlled permissive hypertension.[9] Permissive hypertension can be achieved with the use of an ephedrine or phenylephrine infusion to promote

collateral cerebral circulation through the circle of Willis after clamping of the carotid artery.[9] The use of cerebral perfusion monitors, such as SSEP and cerebral oximetry, is also encouraged to assess the adequacy of cerebral perfusion.[9]

Patients with SSS require certain anesthetic precautions during their perioperative period. Maintaining hemodynamic stability is key in the management of these patients. Continuous hemodynamic monitoring is essential for assessing and maintaining adequate cerebral perfusion through collateral cerebral circulation. The patient in this case did have continuous blood pressure monitoring with a left radial arterial line and promotion of controlled hypertension with a phenylephrine infusion. These anesthetic additions helped to aid anesthesia professionals in the maintenance of cerebral perfusion, rapid response to abrupt intraoperative bleeding, and facilitation of a positive outcome for this patient.

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Opioid-Sparing Anesthetic for Robot-Assisted Total Prostatectomy

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Keywords: opioid sparing, multimodal analgesia, enhanced recovery, pain management, prostatectomy, robot-assisted

Opioids are the most prevalently used means to achieve analgesia in the perioperative setting.[1] There are, however, many undesirable side effects that can lead to increased cost to the patient, and an increased length of hospital stay. Multimodal, opioid-sparing analgesic regimens have been shown to decrease perioperative and postoperative pain, as well as enhance recovery of the patient. The following case report describes the use of an opioid-sparing analgesic process utilized to prevent postoperative nausea and vomiting in a patient with a documented history of this opioid-related adverse side effect.

Case Report

A 72-year-old, 177 cm, 82 kg male with allergies to sulfonamides, fentanyl, and morphine presented for a robot-assisted simple prostatectomy. His preoperative diagnoses included cancer of prostate and enlarged prostate with lower urinary tract symptoms. His overall health history included: obstructive sleep apnea, arthritis, back pain, and actinic keratosis. The patient also reported a history of severe postoperative nausea and vomiting (PONV) associated with all prior surgical procedures. Preoperative laboratory values were all within normal limits, which included a chemistry panel, a complete blood count with differential, and coagulation studies.

Preoperatively an 18-gauge intravenous catheter was placed in the patient's right forearm. Intravenous acetaminophen 1 g was administered, and the patient was transported via stretcher to the operating room and assisted in transferring from the stretcher to the operating room table. After the application of standard monitors and preoxygenation with O_2 10 L/min, anesthesia was induced with IV administrations of lidocaine 80 mg, esmolol 30 mg, propofol 200 mg, rocuronium 5 mg, and succinylcholine 120 mg prior to oral intubation. A Miller-2 blade was used for direct laryngoscopy providing a Cormack and Lehane grade-2 view. The laryngoscopy allowed for the placement of a 7.5 mm cuffed endotracheal tube. Respirations were controlled via mechanical ventilation using a volume control setting. A second 18-gauge IV was placed in his left hand.

General anesthesia was maintained with sevoflurane 1% inspired concentration in a mixture of O_2 1 L/min and N_2O 1 L/min, and intravenous infusions of: lidocaine 2 mg/kg/hr, ketamine 5 mcg/kg/min, magnesium sulfate 10 mg/kg/hr, and dexmedetomidine 0.4 mcg/kg/hr. Cefazolin 1 g was administered via IV for infection prophylaxis and re-dosed after four hours. Dexamethasone 8 mg IV was administered prophylactically for post-operative nausea and vomiting. Prior to incision rocuronium 50 mg IV was administered.

The case proceeded as planned without complications. Intravenous anesthetic infusions were stopped concurrently with the beginning of surgical incision closure. At conclusion, O_2 flow was

increased to 12 L/min and anesthetic gases turned off. Sugammadex 200 mg IV was administered for antagonization of neuromuscular blockade. Ondansetron 8 mg and a second dose of acetaminophen 1 g IV were administered. The patient was extubated in the operating room and transferred to the post anesthesia care unit (PACU). At the time of discharge from the PACU the patient had reported no pain or nausea. His total anesthesia time was approximately 6 hours. Estimated blood loss was 300 mL.

Discussion

Opioids are a very effective treatment modality for the alleviation of operative and post-operative pain. Opioid use, however, is associated with longer hospital stays due to dose-related negative side effects including respiratory depression, prolonged sedation, PONV, urinary retention, and ileus.[2] A retrospective study utilizing a large, national hospital database to assess the impact of opioid-related adverse events (ORADE) on patient outcomes following selected surgical procedures known to require postoperative pain control found that patients with documented opioid-related adverse events had higher adjusted mean costs and increased hospital readmission rates.[2] Multimodal analgesia has become a very important part of enhanced recovery after surgery (ERAS) pathways. Multimodal analgesia is defined as using more than one modality of pain control to achieve effective analgesia while reducing opioid-related side effects.[2]

Multimodal analgesia includes both pharmacologic and non-pharmacologic modalities.[2] This case study focuses primarily on a multimodal approach to systemic pharmacological analgesia utilizing acetaminophen, lidocaine, esmolol, ketamine, dexmedetomidine, and magnesium. Use of this combination of pharmacological agents allowed for an opioid-sparing anesthetic in a patient undergoing robot-assisted total prostatectomy who had a documented history of severe PONV following prior anesthetics where opioids were utilized.

Esmolol and other selective beta-1-adrenoreceptor antagonists can be used to attenuate the sympathetic response to nociception.[2] Esmolol was utilized in the case as a 30 mg bolus to attenuate the sympathetic response to direct laryngoscopy, effectively removing the need for fentanyl during induction. Beta-1-adrenoreceptor antagonists have also been shown to reduce both intraoperative and postoperative opioid requirements.[2] Esmolol continuous IV infusion has also been proposed as an intraoperative alternative to continuous remifentanil infusion, and has been used successfully in ambulatory laparoscopic cholecystectomy, resulting in shorter length of stay, reduced PONV, and decreased postoperative pain.[2]

Acetaminophen is a cyclooxygenase inhibitor with centrally acting analgesic effects.[3] The use of acetaminophen can reduce opioid consumption as much as 30% when administered as an opioid adjunct.[2] A recent meta-analysis found that the prophylactic administration of acetaminophen 1 g IV as part of a multimodal analgesic regimen before surgery or at PACU arrival reduces nausea.[2] The long duration of this case provided the opportunity for both a preoperative 1 g IV dose for perioperative analgesia and a postoperative 1 g IV dose for continued analgesia throughout the recovery period. Studies have also shown that intravenous acetaminophen's opioid-sparing effects allow for rapid emergence from general anesthesia along with a reduction in opioid side-effects such as nausea, vomiting, and sedation.[3]

Lidocaine was employed during this case as a continuous IV infusion at a rate of 2 mg/kg/hr to aid in analgesia in both the perioperative and postoperative periods. The administration of systemic lidocaine is a well-known technique that when used as an intraoperative continuous infusion, can reduce opioid consumption, pain intensity, PONV and improves the quality of recovery and patient satisfaction.[4] Lower concentration systemic lidocaine produces its analgesic effects by increasing acetylcholine concentration at the spinal level by activation of both muscarinic and nicotinic receptors, thus prolonging the pain threshold.[4] Controlled trials found that intravenous lidocaine infusion reduces acute postoperative pain for up to six hours, reduced postoperative morphine requirement, and reduced opioid-related side effects.[2]

Ketamine and magnesium sulfate are useful multimodal adjuvants that work by antagonism of the N-methyl-D-aspartate (NMDA) receptor.[2] By blocking NMDA receptors in the central and peripheral nervous systems, ketamine and magnesium sulfate can possibly reduce hyperalgesia and postoperative acute and chronic pain.[5] Clinical trials have shown the administration of magnesium sulfate and ketamine during spine surgery demonstrate a reduction in perioperative opioid use, as well as a reduction in opioid use and pain score one month after surgery.[5] Continuous infusions of ketamine 5 mcg/kg/min and magnesium sulfate 10 mg/kg/hr were utilized as part of the multimodal approach to analgesia for this case.

Dexmedetomidine in the form of a continuous IV infusion at a rate of 0.4 mcg/kg/hr was also put to use in this case as an adjunct to both analgesia and the attenuation of the sympathetic response to surgical stimulation. An alpha-2 adrenergic agonist, dexmedetomidine produces an analgesic effect by stimulating both central and peripheral alpha-2 receptors.[2] Dexmedetomidine has anxiolytic, sympatholytic, and analgesic properties, and like lidocaine it has been shown to lower postoperative scores.[1]

There are many approaches and techniques to multimodal opioid-sparing analgesic regimens. A common theme in multimodal analgesia is the reduction in opioid use. By avoiding opioids, the associated adverse events that accompany their administration, including nausea, constipation, pruritus, sedation, hormonal and immunological dysfunction, respiratory depression, and even death, can be effectively prevented.[1]

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Spinal Cord Injury and Severe Autonomic Dysreflexia

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Keywords: spinal cord injury, autonomic dysreflexia

Autonomic dysreflexia (AD) is a clinical emergency described as an exaggerated sympathetic response to a noxious stimulus, below the level of the spinal cord injury (SCI).[1-5] The incidence and severity of AD depends on the SCI level, complete versus incomplete injury, and age of injury.[1] The prevalence of AD has been reported between 20% and 70%, and greater than 90% in patients with a SCI in the cervical or upper thoracic region.[2] AD most commonly occurs in chronic stages of SCIs of at least one year, but can also occur within the first month of the initial injury.[1] Moreover, 10% of those with a SCI above T6 experience AD within the first year.[1]

Case Report

A 58-year-old male with a C7 chronic SCI and history of severe AD presented for a cystoscopy to obtain a bladder biopsy. The patient's history included: neurogenic bowel and bladder with a suprapubic urinary catheter in situ, recurrent urinary tract infections, benign prostatic hyperplasia, and bladder tumors. Surgical history included two cystoscopies with suprapubic catheter exchange in 2010 and 2012, a C7-C8 spinal fixation in 1983, a urinary sphincterotomy in 1985, and a transurethral prostate resection in 1999. The American Spinal Injury Association (ASIA) classification of this patient's SCI was ASIA B, describing an incomplete injury with preserved sensory function but loss of motor function, below the neurological level of injury.[3] This patient's SCI occurred during a swimming pool accident in 1983. He experienced AD approximately once per week in response to bladder stimulation, such as a catheter exchange, for which he received transdermal nitroglycerin. His AD symptoms presented as clamminess and hypertension.

The preoperative anesthesia interview revealed an intact sensory and weak motor function of the patient's upper extremities, and very little sensory with no motor function of his lower extremities. Although this patient's SCI lesion was incomplete, the risk of AD occurring during surgery was very high due to his lesion above T6, and weekly AD episodes with bladder stimulation. Prior anesthetic records indicated that a spinal anesthesia technique was placed for his two previous cystoscopies with minimal or absent perioperative symptoms of AD.

Once the patient arrived in the operating room, midazolam 1mg was administered to allay anxiety, and the patient was placed in a sitting position for spinal placement on the operating room table. To achieve a T8 spinal anesthetic, a lidocaine infiltration was administered followed by 0.5% hyperbaric bupivacaine 15 mg (3 mL) with a 22-gauge cutting-point needle inserted between the L3 and L4 interspace. After spinal administration, the patient was positioned supine.

Fifteen minutes post spinal placement, an additional dose of midazolam 1mg was administered. The patient was placed in the lithotomy position and surgery began. The patient's blood pressure was maintained within 20% of baseline with administration of ephedrine boluses as needed, while intravenous lactated ringer's maintenance fluids continuously infused. The patient remained comfortable throughout the procedure and did not require additional sedation.

Throughout the 20-minute surgery, the patient did not experience AD, and the patient was safely transported to the post anesthesia recovery unit. Upon postoperative follow-up, the patient remained asymptomatic of AD as the spinal block resolved.

Discussion

Autonomic dysreflexia is a physiologic but inappropriate autonomic response due to a stimulus below the level of SCI.[4] This stimulus causes a sympathetic response with profound hypertension.[4] Baroreceptors sense the hypertension and send signals to the brain which can cause bradycardia.[1]

Several noxious stimuli which can cause AD include surgery or distention of the bladder or bowel.[4] Patients with both complete and incomplete SCIs can experience AD.[2] Complete SCIs above the level of T6 have the highest risk for AD because injuries above this level involve the splanchnic circulation, which results in more severe symptoms.[1] A SCI above T6 and history of AD are the two major risk factors for intraoperative AD.[1] While these two major risk factors were present in this patient, no intraoperative AD occurred.

Anesthetic management of patients at risk for AD begins with prevention. Both spinal and general anesthesia are equally effective in preventing AD in SCI patients.[5] Epidural anesthesia is not considered as effective because it spares the sacral segments and provides a lesser block density.[5] The patient presented here underwent two prior urological procedures utilizing intrathecal anesthesia without AD; thus, the safest anesthetic choice for this patient was spinal anesthesia with sedation. A standard dose of spinal anesthetic is reported to be an acceptable block in SCI patients.[5] A poor anesthetic choice would be to block surgical stimuli with topical local anesthetics in the urethra for cystoscopy procedures because it does not prevent AD,[5] the bladder muscle proprioceptors would be triggered by bladder distention instead of blocked.[5] Additionally, even without sensory function at the surgical site, it is important to provide anesthesia if stimulation is anticipated, in order to prevent and minimize AD triggers.[5]

For this patient, general anesthesia was the back-up plan; however, if warranted, it could have been used as an alternative primary plan of care. An adequate depth of general anesthesia (either volatile anesthetic or total intravenous anesthesia) must be attained to reduce the incidence of

AD.[1] For neuromuscular blockade, succinylcholine is avoided because it can cause severe hyperkalemia in patients with a SCI present for greater than 48 hours.[5] Because chronic SCI patients may be more sensitive to the vasodilatory and myocardial depressant properties of induction agents, hypotensive management for the patient with a SCI includes decreased medication doses, fluid boluses, and vasoactive medications.[5] Effective and safe vasoactive medications include phenylephrine, ephedrine, or glycopyrrolate.[5]

Management of this patient's potential hemodynamic changes were considered. With high risk for AD, arterial line placement was considered; however, due to the patient's prior successful outcomes with spinal anesthesia alone, an arterial line was deemed unwarranted. An intravenous maintenance fluid infusion was initiated in attempt to prevent occurrence of hypotension after spinal administration. Blood pressure within 20-25% of the patient's baseline is important to preserve spinal cord and myocardial perfusion.[5] In order to maintain the patient's blood pressure within this goal range, ephedrine was administered.

Additionally, SCI patients, especially with high injury levels, can have a greater sensitivity to sedative medication, which may be due to hypovolemia, decreased muscle mass, decreased baroreceptor response, and decreased sympathetic activity.[5] Sedation for this patient was administered sparingly throughout the perioperative course using midazolam, which was adequate to maintain patient comfort during the procedure.

An intraoperative management plan was in place if AD occurred. Based on the patient's prior history of AD, its presentation would likely include hypertension and clamminess. The first priority for treatment of AD is to stop the stimulus.[4,5] The second priority is to deepen the anesthetic if the patient is under general anesthesia.[5] The third priority is to administer 100% oxygen and elevate the head to attempt an orthostatic drop in blood pressure.[5] For pharmacological treatment, intravenous nitroglycerin, a short acting vasodilator, was readily available in the case of sudden and severe hypertension, which is supported by the literature.[5] Alternatively, intravenous sodium nitroprusside and nicardipine infusions are appropriate.[5] If persistent hypertension continues, a vasodilator infusion is warranted which can be given in conjunction with a longer acting medication such as intravenous hydralazine, labetalol or transdermal nitropaste.[5] Long acting vasodilators should be used cautiously to prevent hypotension upon AD resolution.[5]

Even if AD has not occurred intraoperatively, careful monitoring of the patient is important in the postoperative period as the risk of AD continues.[5] Postoperative AD commonly occurs due to bladder distention[4,5] and also because the anesthetic drug levels decline due to metabolism. In hindsight, assessment of the patient's suprapubic urinary catheter for patency upon arrival to the recovery unit would have helped ensure bladder distention did not occur. In summary, this case highlights the perioperative anesthetic considerations for a patient with a SCI, as well as the value and importance of reviewing past anesthesia records to guide future, safe administration of anesthesia.

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Multifocal Atrial Tachycardia and Emergent Surgery

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Keywords: Emergent surgery, multifocal atrial tachycardia, risk stratification

Multifocal atrial tachycardia (MAT) is defined as a heart rate of greater than 100/min with three or more distinct P wave morphologies and/or atrial activation patterns at varying rates and an irregular rhythm.[1,2] MAT is a rare dysrhythmia with an estimated incidence of 0.05% - 0.32% among hospitalized patients.[3] Perioperative risk stratification of a patient with multiple comorbidities undergoing emergent surgical intervention and management of multifocal atrial tachycardia are discussed.

Case Report

An 89-year-old female presented with an incarcerated ventral hernia and abdominal wall adhesions ventral hernia for emergent exploratory laparotomy, lysis of adhesions, and ventral hernia repair. The patient endorsed nausea, vomiting, and abdominal tenderness for 24 hours prior to admission, but no vomiting within the last 4 hours. The patient was allergic to penicillin and her past medical history included MAT, acute kidney injury (AKI) on chronic kidney disease (CKD), insulin dependent diabetes mellitus (IDDM) type II, morbid obesity (body mass index 38 kg/m^2), and hypertension treated with enalapril. Computed tomography findings further noted coronary artery disease and severe peripheral vascular disease. The patient's surgical history included umbilical hernia repair in 2011 and an appendectomy as a child.

Airway exam revealed a Mallampati III score, inadequate prognath, limited neck mobility, edentulousness, and a thyromental distance of 4 cm. The patient had a nasogastric tube (NGT) to her right naris. She stated an inability to walk 1 block without feeling short of breath prior to

admission and was receiving 2 L/min O_2 via nasal cannula and had an SpO_2 of 97%. Laboratory values were notable for creatinine of 2.1 mg/dL, potassium of 4.5mmol/L, hemoglobin of 13.6 g/dL, and glucose of 179 mg/dL. Chest x-ray was notable for atelectasis and left lower lobe opacity secondary to pneumonitis. Electrocardiogram (ECG) revealed MAT with irregular rate between 93 - 138/min and a left anterior fascicular block. The patient's baseline blood pressure (BP) was 147/95 mm Hg. The patient had a urinary catheter in place.

Prior to anesthesia induction, a ramp was placed behind the patient with the OR table in reverse Trendelenburg, the NGT was connected to low continuous suction, and standard noninvasive monitors were applied. Intravenous (IV) induction proceeded over 90 seconds. An anesthetized state was achieved after a total of fentanyl 100 mcg, propofol 80 mg, and ketamine 20 mg were administered. After bag mask ventilation was confirmed, cisatracurium 20 mg IV was administered. The patient's trachea was intubated using a video laryngoscope with a 7.0-mm endotracheal tube (ETT). Placement was verified by positive $ETCO_2$, and equal bilateral chest rise and breath sounds. Clindamycin 900 mg IV was administered over 60 minutes. Dexamethasone 4 mg was administered IV. A left radial arterial line was inserted, and after multiple failed attempts to establish large bore peripheral IV access, a right internal jugular catheter was placed. A nasopharyngeal temperature monitor was introduced. General anesthesia was maintained with sevoflurane 1-2% inspired concentration with $O_2$1 L/min and air 1 L/min. Bispectral index monitoring values averaged 40-60. Intravenous lactated Ringers was administered to maintain stroke volume variation < 12%.[1] Additionally, blood pressure was maintained within 20% of baseline with phenylephrine 40 mcg and/or ephedrine 5 mg IV boluses.

The NGT drained 2 L of gastric contents throughout the case and ondansetron 4 mg IV was administered. Neuromuscular blockade reversal was achieved with IV glycopyrrolate 1 mg and neostigmine 5 mg after a train of four count of 3/4 was measured. Immediately, the patient's heart rate increased to 170-180/min in a MAT rhythm on ECG and BP was 110/60 mm Hg; magnesium sulfate 1 g IV was administered, which reverted the patient's HR to 130-140/min and BP reading to 135/85 mm Hg. Sevoflurane was discontinued and the patient was preoxygenated with 100% O_2. The head of the bed was elevated and awake extubation was performed after the oropharynx was suctioned and the patient met extubation criteria of 6 ml/kg of tidal volume, and regular respiratory rate of 12/min. The patient vomited after extubation; her head was immediately turned to the left and oropharynx suctioned. The patient was awake and maintained an SpO_2 reading of $\geq$ 95% on 4 L/min of O_2 via simple face mask, a HR of 90-130/min in MAT rhythm, and a blood pressure of 150/95 mm Hg before being transported to the intensive care unit (ICU). Since the patient's MAT and blood pressure were stable, no further treatment was required. The patient was referred to medicine postoperatively for further evaluation.

Discussion

This patient's comorbidities combined with acute bowel obstruction posed unique challenges to the anesthesia team. Due to the emergent nature of this surgery, the patient did not undergo a full preoperative workup including echocardiography or pulmonary function tests for risk stratification. However, current evidence provides methods of assessing perioperative risk for non-cardiac surgery based on age, activity level, comorbidities, and type of surgery.

Age is an independent risk factor for increased cardiac risk.[4] Patients ≥ 75 years old are more likely to develop postoperative myocardial infarction than patients < 75 years old (9.5% vs 4.8%, respectively).[2] Additionally, patients ≥ 80 years old are also at increased risk of developing postoperative respiratory failure, AKI, and infection compared to patients < 80 years old (26.1% vs 15.1%, respectively).[4] This patient was unable to walk up 2 flights of stairs, which equates to a measurement of exercise tolerance before surgery of less than 4 units, which independently increases the risk of perioperative complications 2-fold.[4] CKD and IDDM each further increase the risk of perioperative complications three-fold.[4] Emergent surgery carries a significantly greater risk of surgical morbidity and mortality when compared with elective surgery (13.8% vs 6.7% morbidity and 3.7% vs 0.4% mortality, respectively).[4] Because this qualified as a high-risk surgery, the patient had a history of coronary artery disease, she relied on insulin therapy for diabetes, and her preoperative serum creatinine was greater than 2.0, her risk of myocardial infarction, cardiac arrest, or death at 30 days after noncardiac surgery was 15%.[5]

After accounting for increased risk, current guidelines state to proceed with emergent surgery as long as benefits outweigh risks and the patient does not have an absolute contraindication to noncardiac surgery including: acute coronary syndrome, acute decompensated heart failure, brady or tachyarrhythmias causing hypotension and requiring immediate medical intervention, and/or severe, symptomatic aortic stenosis.[4]

Multifocal atrial tachycardia is defined as a heart rate of greater than 100/min with three or more distinct P wave morphologies and/or atrial activation patterns at varying rates and an irregular rhythm.[1,2] MAT is a rare arrhythmia with an estimated incidence of 0.05% - 0.32% among hospitalized patients.[3] A 12 lead ECG is indicated to differentiate MAT from atrial fibrillation (AF), which will display a clear isoelectric segment between P-waves, a feature not found in AF.[1,2] The pathophysiology of MAT is not well understood but has been associated with pulmonary hypertension, coronary artery disease, pulmonary disease, valvular heart disease, theophylline therapy, and hypomagnesemia.[1,2]

The goal of treatment is to slow conduction at the atrioventricular node to reduce the heart rate and terminate the arrhythmia.[1,2] Appropriate treatment options for the management of unstable intraoperative MAT arrhythmias include metoprolol, a beta blocker (BB), verapamil, a calcium channel blocker (CCB), and/or magnesium therapy, even if the serum magnesium level is within normal limits.[1,2] Metoprolol and verapamil are also appropriate choices for long term management of MAT.[1,2] The greatest risk associated with BB and CCB therapy is hypotension resulting in hemodynamic instability and both should be avoided in patients with ventricular dysfunction, sinus node dysfunction, and/or atrioventricular block.[1,2] Additionally, BB therapy is avoided in patients with reactive airway disease due to increased risk of precipitating bronchospasm.[1,2] Cardioversion has not been shown to be effective in treating MAT.[1,2]

Postoperatively, this patient was referred to medicine and electrophysiology to assess the need for beta blocker or calcium channel blocker therapy to treat the MAT[1,2], anticoagulation and to further evaluate the degree of the patient's ischemic heart disease. The patient was discharged from the ICU within 48 hours after surgery. The patient was started on oral metoprolol 25 mg after diet advancement and was scheduled to follow up with cardiology on an outpatient basis

upon discharge from the hospital to a skilled nursing facility. There was no evidence of anesthesia complications throughout her hospitalization.

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Adenosine-Induced Asystole during Craniotomy Bleeding

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Keywords: adenosine, craniotomy, arteriovenous malformation, bleeding, asystole

Treatment of arteriovenous malformations (AVMs) found in the brain is aimed at preventing future hemorrhage and injury for the patient.[1] Any craniotomy is a serious procedure and presents the risk of surgical bleeding and other adverse outcomes to the patient.[2] Both the neurosurgical and anesthesia teams must have a plan to address any bleeding that occurs intraoperatively. Adenosine administration can produce transient asystole and hypotension during periods of craniotomy bleeding that allows for an optimal surgical field and the best opportunity for vessel repair.[3]

Case Report

A 35-year-old female presented for a scheduled craniotomy to resect a chronic, stable AVM. She was 160 cm and 66 kg with a calculated body mass index (BMI) of 25.8 kg/m^2. Her past medical history was significant for migraines. Past surgical history included a C2-C4 laminectomy in 2018. Preoperative vital signs were as follows: blood pressure 131/72 mm Hg, heart rate 81/min, respiratory rate 16/min, and SpO_2 of 100% on room air. Laboratory values indicated no abnormal results expected from her age group and gender. Her preoperative neurological exam showed no deficits. Extremities were strong and equal.

Upon entering the operating room, standard noninvasive monitors were applied. After preoxygenation with O_2 10 L/min, anesthesia was induced with lidocaine 100 mg, fentanyl 100 mcg, propofol 150 mg, and succinylcholine 100 mg. The trachea was intubated with a size 7.0 mm endotracheal tube using a GlideScope (Verathon Inc.) size 3 blade. Both induction and intubation were uneventful and atraumatic. Total intravenous anesthesia (TIVA) was then initiated with propofol 150 mcg/kg/min and remifentanil 0.2 mcg/kg/min to accommodate for neurophysiological monitoring during the case. Fresh gas flow with O_2 1 L/min and air 1 L/min was initiated and maintained throughout the case. An additional 16-gauge intravenous catheter was placed along with right radial arterial line for continuous blood pressure monitoring and arterial blood gas (ABG) sampling.

During the case, hourly ABG samples were drawn and reviewed. Elevated base deficit and lactate values were corrected with aggressive fluid resuscitation including albumin 5% and lactated ringers solution. Urine output (UO) increased to 400-750 mL/hr, and the patient was then treated with desmopressin 1 mcg. After desmopressin administration, UO decreased and no further administrations of desmopressin were required to maintain a stable UO and BP.

Approximately 9 hours into the procedure, the neurosurgeon alerted the anesthesia practitioners that severe vessel bleeding had been encountered in the brain. The surgeon requested adenosine 6 mg followed by a normal saline solution (NSS) 10 mL flush. Post adenosine administration, the patient's HR decreased from 80 to 50/min and BP decreased from 125/85 to 80/40 mm Hg. Within one minute, the HR increased to 110/min. After the systolic BP returned to baseline to approximately 120 mm Hg, the HR returned to 80/min. The surgeon reported bleeding was still occurring. Adenosine 24 mg followed by a NSS 10 mL flush was requested. This elicited the same results as the adenosine 6 mg administration. The surgeon then requested three more additional administrations of adenosine 24 mg with a NSS 10 mL flush. With each administration the patient experienced transient tachycardia following the initial bradycardic episode. On the fifth and final adenosine 24 mg administration, the patient's HR rate decreased to 20/min and the BP decreased to 50/20 mm Hg. These hemodynamic changes then provided an adequate amount of bloodless surgical exposure for the surgeon to successfully repair the hemorrhaging intracerebral vessel.

The surgical procedure subsequently ended after 18 hours. The patient remained intubated and was transferred to the intensive care unit (ICU) to be closely monitored. Reevaluation would then be completed during morning rounds by the neurosurgeon, intensivist, and ICU teams.

Discussion

A cerebral AVM is a tangle of blood vessels that connect arteries and veins.[4] This finding that is typically discovered in adults has an unclear cause and is extremely rare.[4] Common conditions associated with cerebral AVMs include headaches, seizures, cerebral hemorrhage, and cerebral ischemia.[6] Medical and interventional therapy is aimed at preventing stroke, damage, or death in people with this condition.[5] Interventional treatments for AVMs include surgical options such as surgical resection, endovascular embolization, or stereotactic radiosurgery.[5] Medical therapies are aimed at symptoms control such as headache and seizure suppression.[5]

During cranial resection of a cerebral AVM, a variety of risks are present. The greatest intraoperative risk during AVM resection is bleeding and hemorrhage.[6] Strict control of blood pressure is a necessity during craniotomy for an AVM to both maintain cerebral perfusion pressure (CPP) and prevent blood loss associated with hypertension and vessel damage.[6] Anesthesia practitioners must have blood products readily available during this procedure and have a plan of action if bleeding is experienced by the surgical team.[6]

In the case described above, the patient experienced the intraoperative complication of vessel bleeding. It was important for both the neurosurgical and anesthesia teams to collaborate in efforts to fix this issue. As the bleeding started, the surgeon requested adenosine administration to provide the best surgical field and opportunity to repair the vessel. Adenosine can produce a period of transient asystole and hypotension that will slow bleeding and provide the most optimal opportunity to repair a hemorrhaging vessel during both surgical and endovascular procedures.[3] Intravenously administered adenosine acts by slowing the conduction through the AV node.[7] Traditionally used as the drug of choice for treating supraventricular tachycardia (SVT) and restoring sinus rhythm, adenosine is typically cleared in approximately 10 seconds.[7] Along with the use of controlling and correcting arrhythmias, adenosine possesses the ability to act as an agent to control intraoperative bleeding during craniotomy.[3]

Transient adenosine-induced asystole is proven to be both a safe and effective means of producing hemostasis, minimizing blood loss, and providing a near bloodless surgical field for vessel repair.[8] Use of this technique is considered safe, and there has never been a reported death caused by adenosine-induced asystole for bleeding control.[8] During this technique, it has been reported that typical asystole periods range from 0-44 seconds.[8] Doses of adenosine range from 6 mg to 45 mg.[8] Administration of this medication has proven to provide a near-instant decrease in heart rate and blood pressure that allows for a decrease in blood loss, the best opportunity for vessel repair during hemorrhage, and the possibility to avoid the use of transfused blood products.[3] This case provided an example of how adenosine can be successfully and safely administered for this purpose.

Vigilant monitoring and timely treatment are crucial during craniotomy. It is also essential for the neurosurgical and anesthesia practitioners to effectively communicate during the procedure. During this case, desmopressin was administered after patient presentation suggested diabetes insipidus. Fluid administrated was also more aggressively administered after ABG analysis. When intraoperative bleeding was experienced during craniotomy for AVM resection, adenosine was successfully administered to provide an opportunity for vessel repair. Although adenosine is

primarily used for cardiac arrhythmias, it has an important use within neurosurgical procedures that can help control hemorrhage, prevent the transfusion of blood products, and improve patient outcomes.[3]

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Anesthetic Management of a Patient with Ehlers-Danlos Syndrome

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Keywords: Ehlers-Danlos syndrome, perioperative management, EDS, E-D syndrome

Ehlers-Danlos syndrome (EDS) is a heritable connective tissue disorder characterized by joint hypermobility and instability, skin texture anomalies, and vascular and soft tissue fragility.[1] The overall incidence is 1:10,000 to 1:25,000 in Northern America. In 2017, the International Classification for the Ehlers-Danlos Syndrome designated thirteen subtypes which are currently in use.[2] EDS subtype classification continues to evolve resulting in various classification systems

seen in the literature such as the original categorization system (EDSI-EDS XI) and the Villefranche nosology (EDS I-VI).[2] The tissue defects in EDS syndrome can manifest in many organs and has widespread implications for the anesthesia professional.

Case Report

A 22-year-old, 164 cm, 75 kg male presented for perianal abscess incision and drainage. The patient's past medical history included EDS type 8, Postural Orthostatic Hypotension (POTS), chronic joint pain, bleeding tendency, smoking, dysautonomia, vitamin deficiency, thoracolumbar scoliosis, anxiety, and depression. A current list of medications included metoprolol, midodrine, morphine, tramadol, buspirone, celecoxib, gabapentin, clonazepam, duloxetine, and omeprazole. The patient's past surgical history included two cystoscopies and a perianal abscess drainage with no anesthetic complications. An airway exam revealed good dentition and a Mallampati score of 1. The patient had good neck range of motion and MRI results did not show any abnormalities. Basic metabolic panel and complete blood count values were within normal limits. Preoperative vital signs were within normal limits. A 12-lead ECG showed sinus rhythm with nonspecific ST elevation.

Upon entrance to the operating room, standard noninvasive monitors were applied. The patient was preoxygenated with O_2 10 L/min via facemask for 5 minutes. General anesthesia was induced with midazolam 2 mg, fentanyl 100 mcg, lidocaine 80 mg, and propofol 150 mg IV. The airway was secured with a laryngeal mask airway (LMA) size 4. The patient received dexamethasone 4 mg and ondansetron 4 mg IV after induction.

General anesthesia was maintained with isoflurane 1.5% inspired concentration in O_2 1 L/min and air 1 L/min. The patient maintained spontaneous ventilation, adequate tidal volume and respiratory rate on spontaneous mode of ventilation. The patient was placed in lithotomy position for the procedure. Foam padding was placed around the upper and lower extremities. The patient's eyes were carefully taped shut and head maintained in neutral position. The patient's intraoperative course was uneventful. Isoflurane was discontinued at the end of the procedure. The LMA was removed, oropharynx suctioned, and O_2 8 L/min via closed face mask was administered. The patient was transferred to recovery area in stable condition.

Discussion

Patients with EDS require numerous surgeries due to vascular and joint fragility. Preoperatively, it is important to perform a thorough history and physical along with determining the subtype classification of the syndrome as each subtype involves unique clinical features. The patient presented in this report had EDS subtype 8 or kyphoscoliotic EDS which involves defects in collagen folding and cross linking.[2] These patients present with muscle hypotonia, congenital or early onset of kypho-scoliosis and generalized joint hypermobility resulting in shoulder, hip or knee dislocations. The patients can also have skin hypextensibility, easily bruised skin, osteoporosis, scleral fragility increasing the risk of white globe rupture of the eye and medium sized artery aneurysms.[2]

Chronic pain in EDS patients is common with up to 90% of patients reporting some form of chronic pain. Pain can result from joint dislocations, previous surgery, muscle weakness, proprioceptive disorders, and vertebral instability.[3] Patients may present with long-term opioid therapy necessitating careful assessment of pain and the use of multimodal analgesia during the perioperative period.[3] The decision to proceed with general or regional anesthesia should be made on an individual basis and consideration for patient specific risk factor such as EDS subtype, bleeding diathesis, and spinal pathology.[5] General anesthesia can be performed with volatile anesthetics, nitrous oxide or as total intravenous anesthesia. Succinylcholine can be safely used, however it should be avoided in immobilized patients. Neuromuscular blockade should be carefully assessed in patients with baseline muscular weakness.[1] The use of neuraxial blocks is controversial due to risk of spinal hematoma, especially in vascular type EDS.[5] If neuraxial anesthesia is desired, assess spine pathology, history of failed local anesthetics during dental procedures or previous regional anesthesia attempts.[1] A combined spinal-epidural technique can allow for administration of additional local anesthetic doses in case of local anesthetic resistance.[5] Wegener et al [5] reported a successful supraclavicular nerve block in a patient with EDS type III (hypermobility type) for arthroplasty of carpometacarpal joint. Regional anesthesia under ultrasound guidance is recommended to avoid injury to fragile vessels and to visualize spread of local anesthetic which can be hampered by tissue scarring.[1,5]

Intraoperatively, non-invasive monitoring should be performed, when possible, especially in patients with EDS subtypes involving vascular fragility. Due to increased risk of vascular rupture, central venous access and arterial puncture should be avoided. The use of ultrasound is recommended for all vascular access.[1] Patients with kyphoscoliotic EDS and brittle cornea syndrome can have ocular fragility. Patients with frail skin can develop bruising and hematoma from repetitive non-invasive blood pressure measurements. About 90% of all EDS subtype patients bruise easily, therefore care should be taken when transferring and positioning patients. Even minor shear force can result in tissue injury, highlighting the importance of proper padding and positioning.[1] During preoperative interview, the patient reported a history of incision dehiscence with the use of sutures which was relayed to the surgeon. The patient did not report any anesthetic or positioning specific complications.

Airway management considerations for this patient population includes avoiding temporomandibular joint luxation during mask ventilation and airway instrumentation. There is an increased risk of joint dislocation during intubation. Patients may have higher incidence of premature spondylosis, occipitalatlantoaxial instability and cervical spine instability.[1] A smaller endotracheal tube should be used to avoid mucosal damage. Upon reviewing the diagnostic studies, this patient was found to have history of neck pain, weakness and numbness of the left arm. The MRI showed preserved vertebral body and disc height. Neck range of motion was carefully assessed during the preoperative interview.

Bleeding abnormalities are common placing this patient population at an increased risk of intraoperative bleeding and hematoma formation. In patients with EDS subtypes involving the vasculature, the risk of hematoma, diffuse bleeding and compartment syndrome is high from the use of tourniquets. Laboratory results may be within normal range and do not accurately estimate

bleeding risk. Desmopressin has been shown to decrease bleeding and reduce transfusion requirements.[1]

Postural Orthostatic Hypotension is a common co-existing disease in patients with EDS, as was the case in the patient presented in this case report.[1] Orthostatic hypotension is thought to result from connective tissue defects in dependent blood vessels which distend excessively in response to ordinary hydrostatic pressures resulting in increased venous pooling and hemodynamic consequences. Patients should be advised to continue their preoperative medications. In addition, venous pooling can be reduced by lower limb elevation and abdominal or lower limb compression devices.[4] In symptomatic patients, preoperative crystalloid infusion and early use of vasopressors is recommended. The patient maintained hemodynamic stability throughout the procedure.

Patients presenting with EDS can be challenging as there is little evidence-based knowledge about this rare condition. Careful preoperative assessment along with determining the subtype can help facilitate safe anesthesia care for these patients.

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Perineural Dexmedetomidine as an Adjunct in Peripheral Nerve Blocks

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Keywords: dexmedetomidine, regional anesthesia, peripheral nerve blocks, supraclavicular or brachial plexus blocks

Up to 80% of surgical patients suffer from postoperative pain, necessitating a multimodal approach to analgesia.[1] Peripheral nerve blocks are considered an integral component in a multifaceted approach to decrease opioid use and related side effects; however, single shot nerve blocks are limited by their duration of action. Numerous adjuncts have been explored to prolong block duration. The optimal perineural adjunct is preservative-free (PF), decreases time to block onset, improves block quality, and does not significantly prolong motor block if movement is desirable postoperatively.[2]

Case Report

A 64-year-old female presented for an open reduction and internal fixation of a right distal radius fracture and open carpal tunnel release. Her past medical history was significant for hypertension, gastroesophageal reflux disease, Lyme disease with no residual neuropathies, Raynaud's syndrome, and postoperative nausea and vomiting (PONV). Her past surgical history included a pulmonary valve repair, anterior and posterior cervical discectomy, a transurethral resection of a bladder tumor, and a cardiac catheterization demonstrating normal coronary anatomy, normal left ventricle size and function, and the absence of valvular abnormalities. The patient had documented allergies to latex, adhesives, vancomycin, cephalosporins, clindamycin, and sulfonamides. Home medications included amlodipine, metoprolol succinate XL, oxycodone, acetaminophen, and risedronate. Upon physical examination, breath sounds were clear to auscultation, heart rate and rhythm were regular with no abnormalities, and an airway exam revealed a small mouth opening and the presence of a permanent bridge. After discussion with the surgeon, the decision was made to place a supraclavicular nerve block to provide surgical anesthesia and postoperative analgesia.

Preoperatively, oxygen was administered via a nasal cannula at a rate of 3 L/min and standard noninvasive monitoring was utilized. Once the patient was properly positioned with the head of the bed elevated and the patient's head turned to the left, the supraclavicular site was prepped with chlorhexidine. A sterile ultrasound probe was placed cephalad to the patient's right clavicle and was manipulated until a view of the brachial plexus and subclavian artery were obtained. After 1% lidocaine 3 mL was injected subcutaneously, a 21-gauge, 4 inch short-bevel insulated needle was inserted utilizing an in-plane technique to visualize local anesthetic spread at the level of the trunks under ultrasound guidance. After negative aspiration, dexmedetomidine (PF) 40 mcg mixed with 0.5% bupivacaine (PF) 25 mL was injected in 3-5 mL increments with no resistance noted and no complaints of paresthesia. The patient was subsequently taken to the operating room and received midazolam 2 mg and fentanyl 100 mcg and was maintained on a propofol infusion ranging from 50-75 mcg/kg/min throughout the procedure.

Intraoperatively, a tourniquet was placed on the right upper extremity and was inflated to 250 mm Hg. A total of ephedrine 15 mg was administered in 5 mg increments for intermittent episodes of hypotension. Additionally, dexamethasone 4 mg followed by ondansetron 4 mg were administered to prevent PONV. Postoperatively, the patient was hemodynamically stable and did not require any additional opioids or pain interventions while in the post anesthesia care unit (PACU). The patient was subsequently discharged home the same day once PACU discharge criteria was met.

Discussion

Dexmedetomidine, like its prototype clonidine, is a selective alpha-2 adrenoreceptor agonist that is not only used for its sedative properties, but also for its anxiolytic and analgesic properties.[1] There have been several studies demonstrating the effectiveness of the perineural use of dexmedetomidine to shorten time of block onset, prolong the duration of analgesia, and improve block quality.[1] While the exact perineural mechanism of action is not entirely understood considering the lack of alpha-2 receptors on peripheral nerve axons, it is thought that dexmedetomidine helps to reduce inflammation and cause vasoconstriction maintaining higher concentrations of local anesthetic in the targeted plexus.[2,3] In the case narrative above, the patient tolerated the surgery and tourniquet inflation with solely the supraclavicular block, sedation, and fentanyl 100 mcg intravenously. No additional opioids or antiemetics were needed in the postoperative period.

In a meta-analysis of 18 randomized controlled trials (RCTs), the use of perineural dexmedetomidine in brachial plexus blocks shortened the onset time by 3.2 minutes, prolonged sensory blockade by 261 minutes, and prolonged the duration of analgesia by 289 minutes compared to local anesthetic alone.[4] Similar clinical and statistical significance in sensory block duration prolongation was found in comparable meta-analyses.[2] In another meta-analysis of 43 RCTs, dexmedetomidine shortened the onset of sensory block duration by 9 minutes and motor block by 8 minutes.[2] In an increasingly fast-paced, high patient turnover perioperative environment, saving minutes for a block to adequately set for surgical anesthesia could have lasting impacts on productivity, enhancing work flow to minimize operating room delays, and ultimately patient safety by ensuring adequate block density prior to incision.

The most reported adverse events associated with dexmedetomidine are hypotension and bradycardia, experienced by 1% and 6% of patients respectively. [4,5] Of note, the bradycardic episodes reported were either transient or self-resolving, or easily treated with atropine. [4,5] Lastly, episodes of bradycardia increased in a dose-dependent fashion in cases associated with perineural doses of dexmedetomidine greater than 50 mcg.[4,5] In this case narrative, only 40 mcg of dexmedetomidine was administered, and the patient did not have any episodes of bradycardia. The few episodes of hypotension experienced intraoperatively were responsive to vasopressor support and could also be attributed to the propofol infusion or tourniquet deflation. A paired, blinded randomized trial demonstrated through bilateral adductor canal blocks that perineural dexmedetomidine also has some peripheral effects accounting for sedation.[6] While sedation could be an undesirable effect in some patients, sedation may be beneficial when using peripheral nerve blocks as a sole anesthetic, minimizing the use of volatile anesthetics and decreasing the dose of sedatives and benzodiazepines, such as in this case narrative. The use of

dexmedetomidine and a peripheral nerve block in this case narrative eliminated the need for volatile anesthetics and thus mitigated some of the risk for PONV in a high-risk patient. Additionally, the adequate pain control and decreased need for opioids facilitated a quick discharge from the PACU.

Perineural dexmedetomidine 50-60 mcg combined with bupivacaine, levobupivacaine, or ropivacaine demonstrated an increase in mean duration of analgesia by approximately 4.5 hours, with minimal hemodynamic side effects.[2,7] Of note, the majority of these studies were related to the efficacy of using perineural dexmedetomidine in peripheral nerve blocks, but dexmedetomidine also showed similar promise with prolonging motor and sensory block when used intrathecally as an adjunct to spinal anesthetics.[8] Despite data in its support and promising outcomes, it is worth noting that the use of perineural dexmedetomidine is still "off-label."[3] In the studies explored, there have been no neurological consequences reported with perineural dexmedetomidine use to date, and in vivo rat studies suggest a neuroprotective effect due to the inhibition of proinflammatory cytokine release and mast cell degranulation.[2] Since episodes of hypotension, bradycardia, or sedation may occur following perineural dexmedetomidine,[4,5,6] anesthesia providers must be vigilant and prepared to treat these conditions.

Managing postoperative pain remains a significant focus of anesthesia professionals to optimize patient outcomes, minimize opioid consumption, and enhance patient satisfaction. Since perineural dexmedetomidine may enhance duration of analgesia in peripheral nerve blocks, it represents a valuable treatment option to extend perioperative analgesia from local anesthetics well into the postoperative period. More studies are needed to evaluate the most effective perineural dose of dexmedetomidine for maximum and long-lasting analgesia, while minimizing side effects.

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Blood Loss During Liver Resection: A Case Report

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Keywords: liver resection, liver, blood loss, and central venous pressure

Liver resection is often indicated when colorectal cancer metastasizes to the liver and transfusion of blood products may be indicated.[1,2] Bleeding and transfusion needs liver resection may be attributed to the 25% of cardiac output that the liver receives, the complex vasculature, and an ability to reserve up to 1 L of blood.[2] Surgical intervention may confer a high risk for bleeding, especially when resections involve more than 3 lobes, because the liver stores blood and creates most clotting factors.[2] This report discusses initial assessment, intraoperative monitoring considerations, and recommended interventions for addressing blood loss in this population.

Case Report

A 65-year-old, 178 cm, 100 kg male with a 5.7 cm intrahepatic cholangiocarcinoma, was scheduled for a left liver lobectomy. Six years earlier, he had been treated non-surgically with chemotherapy and radiation for metastatic adenocarcinoma to the liver. He reported having no prior surgeries. His medical history was notable for asthma, hypertension, hyperlipidemia, hypothyroidism, diabetes mellitus, and esophageal reflux. He reported taking the following oral medications regularly, including the morning of surgery: amlodipine 5 mg, gabapentin 600 mg, levothyroxine 175 mg, lisinopril 10 mg, and pantoprazole 40 mg. An albuterol metered-dosed inhaler was used to manage asthmatic episodes as needed.

Preoperative vital signs were as follows: blood pressure 111/67 mm Hg, heart rate 69/min, respiratory rate 18/min, SpO_2 98% on room air, and temperature 36.1 degrees Celsius. Laboratory values were as follows: serum albumin 2.2 g/dL, prealbumin 6.0 mg/dL, international normalized ratio (INR) 1.3, prothrombin time (PT) 15.2 seconds, hemoglobin 11.3 g/dL, and hematocrit 34.6%. His electrocardiogram revealed a first-degree atrioventricular block.

Subcutaneous enoxaparin 40 mg and intravenous (IV) midazolam 2 mg were administered in the preoperative area. Standard monitors were placed, and the patient was preoxygenated with 100% oxygen. Induction of general anesthesia was initiated with IV fentanyl 200 mcg, lidocaine 50

mg, propofol 200 mg, and succinylcholine 100 mg. After endotracheal intubation and return of motor nerve function, IV rocuronium 50 mg was given. A radial arterial line was placed, and anesthesia was maintained with inspired sevoflurane 2.4%, in O_2 1 L/min and air 1 L/min. Ventilation was maintained using pressure control with volume guarantee, tidal volume 450 mL, pressure max 30 cm H_2O, respiratory rate 12/min, and positive end expiratory pressure 5 cm H_2O. Other medications administered in divided doses throughout the case included IV dexamethasone 10 mg, cefazolin 4 g, and fentanyl 300 mcg.

During liver tissue resection, the surgeon reported difficulty visualizing vessels through adipose tissue layers, and the hepatic vein was transected. The surgeon noted increased bleeding and an estimated 700 mL of blood loss. The surgeon intermittently employed the Pringle maneuver and requested placement of an internal jugular central venous catheter. The patient was placed into the Trendelenburg position and the central venous pressure (CVP) measured 10 mm Hg immediately following catheter placement; the arterial blood pressure was 74/34 mm Hg, heart rate 68/min, SpO_2 98%, and bladder temperature 35.5 degrees Celsius. The following medications were administered in divided doses to treat hypotension: phenylephrine 2.85 mg, ephedrine 10 mg, vasopressin 19 U, and albumin 5% 750 mL. Fluids administered included Plasmalyte 1 L, lactated ringers 3 L, and 1 unit of packed red blood cells. The blood pressure improved to 123/55 mm Hg with a heart rate of 77/min. An arterial blood gas demonstrated a pH 7.29, PO_2 219 mm Hg, HCO_3 19 mmol/L, base excess -7.1 mmol/L, hemoglobin 12.8 g/dL, and hematocrit 38.2%. No signs of cardiac ischemia were observed on electrocardiogram.

At surgical closure, the total estimated blood loss for the case was 1800 mL. The sevoflurane and air were turned off, and O_2 was increased to 15 L/min. Intravenous ondansetron 4 mg and sugammadex 200 mg were given. The patient emerged from anesthesia hemodynamically stable, was extubated, and transported to the post-anesthesia care unit (PACU). Laboratory values in the PACU revealed serum albumin 3.0 g/dL, prealbumin 6.1 mg/dL, international normalized ratio 1.2, prothrombin time 15.1 seconds, hemoglobin 12.8 g/dL, and hematocrit 38.3%.

Discussion

Liver anatomy is functionally divided into eight segments and can be removed by segment or by lobe.[2] This patient had a total left liver lobectomy, removing 3 segments, which is considered a major resection of the liver.[2] Major liver resection carries the risk of significant intraoperative blood loss.[1] Preoperative and intraoperative implementation of assessment, monitoring, and interventions exists to prevent significant blood loss.[1,2]

Anesthesia practitioners are responsible for the preoperative assessment of liver function to anticipate potential intraoperative complications such as hemodynamic lability and hemorrhage.[1,2] Before surgery the patient's hemoglobin and hematocrit were noted to be low; however, he was asymptomatic and there were no signs of acute bleeding, such as hypotension and tachycardia, and the operative team deemed no interventions were necessary. His serum albumin level was below 3.5 g/dL, which may have been a marker for poor liver function.[2,3] His INR was greater than 1.0, and his PT elevated beyond 12 to 15 seconds, indicating an increased risk for bleeding.[2] Given this risk, 2 units of crossmatched blood was made readily available before surgery. Coagulation testing is useful in assessing liver function because almost all

coagulation factors, except factor VIII, are produced in the liver.[3] After the preoperative assessment, the operative team, concluded it was safe to proceed to surgery.

The chosen surgical approach, laparoscopic versus open, may reduce intraoperative blood loss during liver resection.[4] For resection of tumors less than 3 cm, and involving less than 3 segments, the laparoscopic approach is preferred.[4] For surgical resection of tumors sized greater than 5 cm, and involving more than 3 segments, an open resection is the preferred approach.[4] The open approach was preferred in this case, given the patient's tumor size exceeded 5 cm, and resection required more than 3 segments.[4]

Administration of anesthetics can contribute to decreased intraoperative blood loss during liver resection.[5] Inhaled and intravenous anesthetics may vasodilate and decrease hepatic arterial and portal venous blood flow, mean arterial pressure, and cardiac output.[2,3,5] Sevoflurane and desflurane are less disruptive to hepatic artery and venous blood flow compared to other inhaled agents;[3] however, no anesthetic approach is superior in liver resection surgery.[2,3,5] Liver circulation depends on systemic pressure and blood flow through a complex venous and arterial system.[2] Hepatic veins dispersed within the liver tissue return filtered blood to the inferior vena cava.[2] The majority of oxygenated blood is supplied to the liver by the hepatic artery and hepatic portal vein.[2] The hepatic portal vein receives venous drainage from the gastrointestinal tract into a low-pressure system.[2] This low-pressure system allows the liver to act as a reservoir, holding as much as 1 L of blood.[2] If an injury to hepatic vessels occurs, the blood in the liver can shift to the central circulation, increasing overall blood loss.[2] A controversial strategy to minimize bleeding is to monitor and avoid elevations in CVP, leading some practitioners to require central venous catheters for these cases.[1,5]

Low CVP, defined as less than 5 mm Hg, may decrease blood loss during tissue dissection and improve visibility of the surgical field.[1,5] During high CVP states, the vessels within the liver are distended and are more prone to unintentional injury.[1,5] If an injury occurs, bleeding can be challenging to control due to vessel location and complex liver anatomy.[5] The Pringle maneuver was employed in this case after inadvertent transection of the hepatic vein. During this maneuver, a clamp occludes the liver's arterial and venous blood flow into the portal-triad.[1] Clamp time should not exceed 1 hour due to the increased risk of hepatocellular ischemia.[1] Anesthesia practitioners should be aware of increased risk for air embolism during clamp time.[1,3] Risk for embolism during the Pringle maneuver stems from the disruption of integrity to the hepatic vessels exposing them to air.[1,3] This risk is exacerbated under low CVP states due to a larger available vascular area serving as a negative pressure conduit that could entrain room air.[1,3] Risk for air embolism may be reduced by positioning the patient in Trendelenberg during Pringle maneuver implementation.[1] Some authors suggest the use of transesophageal echocardiography for monitoring cardiac function, fluid management, and assessing for air embolism during liver resection.[2,3] Anesthesia practitioners can attempt to decrease blood loss by initiation of low CVP which may be achieved by fluid restriction before liver tissue dissection and the use of vasodilators such as volatile anesthetics.[2,5] Unfortunately, maintaining a low CVP has not been shown to improve patient outcomes such as long-term survival or risk for intraoperative blood loss requiring transfusion.[6] In this report, CVP monitoring was not initiated until requested by the surgeon.

Management of patients undergoing major liver resection can be challenging, and improper management may contribute to excessive blood loss, coagulopathy, post-operative liver dysfunction, and cardiopulmonary impairment.[1,2] Careful consideration of the anesthetic plan may prevent hemodynamic instability.[2,5] As described in this report, implementation of a safe anesthetic plan can be achieved with a thorough initial assessment, vigilant intraoperative monitoring, and judicious implementation of intraoperative interventions.[1,2]

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Anesthetic Implications for Patients with Extensive Burn Injuries

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Keywords: Burns, inhalation injury, hypermetabolic, anesthesia management

Patients with burn injuries present a critical pathophysiology that must be diligently anticipated by the anesthesia practitioner. Between the years of 1988 and 2008, the National Inpatient Sample database reported burn injuries to be responsible for 506,628 hospital admissions.[1] Primary complications in burn victims include an initial shock phase followed by a hypermetabolic response to the injury.[2] Overall, the pathophysiologic changes related to these injuries pose life-threatening complications that often require surgical intervention, at which point the anesthesia practitioner becomes the leader of airway, ventilation resuscitation, analgesia, and anesthesia management.

Case Report

A 47-year-old male (106.1 kg, 183 cm, BMI 31.7 kg/m^2) presented for debridement and skin grafting of full-thickness burns to approximately 30% of his total body surface area (TBSA). Affected areas included the anterior chest, posterior back, face, neck, bilateral circumferential upper extremities, and posterior lower extremities. Surgical history included upper extremity escharotomies to prevent compartment syndrome. Pertinent laboratory findings included hemoglobin 10.6 g/dL, hematocrit 30.8%, platelets 181 x 10^9/L, and albumin 1.6 g/dL.

In the emergency department, the patient's trachea was intubated with a 7.0 mm cuffed endotracheal tube (ETT) due to inhalation injury. Subsequently, the patient was admitted to the intensive care unit (ICU) where he was sedated and received mechanical ventilation prior to arriving in the operating room (OR). Fentanyl 300 mcg/hr, dexmedetomidine 1.5 mcg/kg/min, and propofol 25 mcg/kg/min were infusing through a right femoral central venous line when anesthesia practitioners arrived to the ICU for transport to the OR. Enteral nutrition was infusing and allowed to continue. Prior to transport to the OR, 4 mg of midazolam was administered intravenously (IV), and neuromuscular blockade was induced with vecuronium 10 mg IV.

During transport, the patient was ventilated with O_2 8 L/min via a Mapleson D (Bain) circuit. In the OR, standard noninvasive anesthesia monitors were applied, and mechanical ventilation was initiated with a tidal volume of 5 mL/kg of ideal body weight, positive end expiratory pressure (PEEP) 10 cm H2O, a respiratory rate of 12/min, and FiO2 of 0.6. An existing right radial arterial line was zeroed and connected for monitoring. The patient's EtCO2 was maintained between 33 to 35 mm Hg and SpO_2 > 90%.

General anesthesia was maintained through total intravenous anesthesia (TIVA), which consisted of propofol 100 mcg/kg/min, dexmedetomidine 0.5 mcg/kg/min, and fentanyl 300 mcg/hr. Hydromorphone 2 mg IV was administered prior to supplementing a multimodal analgesic approach. The continuous propofol infusion was titrated to maintain bispectral index (BIS) between 40 and 60. Neuromuscular blockade was monitored via peripheral nerve stimulation of the orbicularis oculi muscle, and vecuronium 3 mg IV was administered as needed to maintain a train of four count < 2/4 twitches. Dexamethasone 8 mg was administered IV to decrease laryngeal edema.

The anesthesia practitioners and surgeon agreed on hemodynamic goals of hemoglobin > 7.0 mL/dL, mean arterial pressure > 65 mm Hg, and urine output > 1.0 mL/kg/hr. Intravascular volume was maintained with plasmalyte 2000 mL, albumin 5% 500 mL, 4 units packed red blood cells (PRBCs), and 2 units of fresh frozen plasma (FFP). Two units of PRBCs were transfused in response to calculated estimated blood loss. Intraoperative hemoglobin and hematocrit labs revealed 6.9 g/dL and 19.7%, respectively. Two additional units of PRBCs were transfused. Body temperature was maintained between 35.5°C to 37°C (esophageal) by increasing the OR temperature to 39°C. Tranexamic acid 1000 mg was administered IV to inhibit fibrinolysis. Total blood loss was estimated to be 1300 mL, and total urine output was 900 mL. Final hemoglobin was 9.0 g/dL and hematocrit was 27.2%.

The ETT was left in place and mechanical ventilation was continued at the conclusion of the procedure. The patient was transported back to the ICU with all sedation medications infused at preoperative rates. Neuromuscular blockade was not antagonized. During transport, manual ventilation was administered via Bain circuit with O_2 8 L/min. In the ICU, a hand-off report was given to the ICU nurse.

Discussion

Patients presenting to the hospital for burn injury must be immediately evaluated for inhalation injury if not previously evaluated or intubated prior to arrival.[3,4] It is estimated that 10-20% of burn victims suffer from inhalational injury, at which point the airway is compromised.[3] Facial burns, presence of soot in the mouth, shortness of breath, increasing TBSA percentage of burn, and entrapment in an enclosed space are several factors that indicate potential impending or current airway compromise.[3] Prophylactic intubation is recommended in these instances to prevent total airway collapse and subsequent cardiopulmonary arrest.

Burn victims suffer pathophysiologic changes that make standard monitoring and IV access difficult for the anesthesia practitioner.[4] Further, repeated attempts for invasive cannulation risk infection and exposure of bloodstream to pathogens.[4] Arterial cannulation is beneficial to monitor evolving fluid shifts, hemodynamic parameters, and provides means for repeated blood gas analysis.[4] For these reasons, it is recommended that the burn victim presenting for surgery has central IV access and invasive blood pressure monitoring.[4] Strict aseptic technique and careful selection of cannulation site must be considered to decrease the risk of central line-associated bloodstream infection.

The hypermetabolic phase of burn pathophysiology presents shortly after injury and can persist for more than 12 months.[2,4] This phase is characterized by a 50-fold increase in plasma catecholamines, cortisol, and inflammatory cells which leads to increased energy expenditure and multi-organ dysfunction.[2,4] Attenuation of this response through perioperative continuation of enteral feedings significantly reduces morbidity and mortality in this population.[2]

Neuromuscular blockade in patients with burn injury presents critical issues for the anesthesia practitioner. Depolarizing neuromuscular blocking agents (succinylcholine) should be avoided 48 hours after burn injury due to upregulation of acetylcholine receptors and the increased risk for hyperkalemia.[4] Further, resistance to nondepolarizing muscle relaxants may necessitate increased dose requirements.[4]

It is estimated that 60% of burn patients develop acute respiratory distress syndrome (ARDS).[5] Fluid resuscitation, direct pulmonary injury, and edema are some of the contributing factors.[3,4,5] Mechanical ventilation strategies in ARDS patients include low tidal-volume ventilation (6 mL/kg of predicted body weight) with application of PEEP and plateau airway pressures < 30 cm H_2O.[4,5] During the hypermetabolic phase, it is also recommended that increased respiratory rates are employed to offset increased carbon dioxide production.[4]

The hypermetabolic phase of burn injury leads to increased dose requirements for IV agents such as propofol and opioids.[4] Spinal cord changes lead to down-regulation of μ-opioid receptors

which also contributes to pharmacokinetic alterations in opioid efficacy.[4] Upregulation of N-methyl-D-aspartate receptors leads to increased dose requirements of ketamine.[4] However, the use of ketamine must be weighed against catecholamine depletion and the potential for exaggerated myocardial depression.

Due to the increased metabolism and dose requirements for IV anesthesia agents seen in burn patients, it is important to utilize measures to ensure adequate sedation while administering TIVA. BIS monitoring provides this information to anesthesia practitioners by interpretation of electroencephalogram signals.[6] Maintenance of BIS values between 40 and 60 provides optimal surgical anesthesia and has been evidenced to reduce recall by up to 80%.[6]

Immediately following burn injury, increased capillary permeability and release of inflammatory cytokines leads to loss of intravascular volume and widespread vasodilation, respectively.[4,7] It is estimated that every 1% of TBSA excised leads to an intraoperative blood loss of 9.2% of overall blood volume.[7] Considering these factors, it is critical to appropriately resuscitate burn victims to mitigate multi-organ dysfunction and prevent circulatory collapse. Resuscitation with colloid factors such as PRBCs, FFP, and albumin helps maintain intravascular oncotic pressure to a greater degree than crystalloids while simultaneously limiting the total amount of fluid administered IV.[8] Overall, this resuscitation strategy leads to less edema and decreased risks of compartment syndrome.[8]

Overall, this case demonstrates the importance of anesthesia practitioners appreciating the major physiologic changes and challenges that occur in the timeframe immediately following a burn injury. It is critical to understand the hypermetabolic phase of burn injury and the specific dosing modifications required to ensure adequate anesthesia. Inhalational anesthetics may provide some added benefit over TIVA in terms of documented bronchodilation effects, and avoidance of excessive IV infusions for prolonged periods. Fluid resuscitation should be meticulously calculated and diligently selected to avoid cardiovascular collapse and maintain intravascular volume. This method would simultaneously prevent compartment syndrome or further sequelae of over-resuscitation.

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Mentor: Jeanie Skibiski, DNAP, MHA, CRNA

Preserving Perioperative Cognitive Function: A Quality Improvement Project

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Keywords: postoperative delirium, geriatrics, bispectral index, postoperative cognitive dysfunction

Introduction

Postoperative delirium (POD) is the most common surgical complication experienced by older adults and is associated with increased morbidity and mortality, longer hospital stays, and delayed rehabilitation.[1] Anesthesia enhances inflammation and can have direct neurotoxic effects.[2-4] Anesthesia providers can help preserve perioperative cognitive function and prevent POD by implementing neuroprotective strategies aimed at risk stratification and reducing the stress of anesthesia and surgery. This project aimed to identify patients at high risk for developing POD through preoperative cognitive screening and improve cognitive outcomes by avoiding deep states of anesthesia through the use of bispectral index (BIS) monitoring. Standardized preoperative cognitive assessments can identify patients at high risk for developing POD, allowing for the optimization of their care.[1] Intraoperative monitoring of anesthetic depth and avoidance of deep states of anesthesia has been identified by multiple systemic reviews and randomized controlled trials as strategies for preventing POD.[2,3,5]

Methods

An in-person educational session was delivered to the Anesthesia Department staff on POD risk factors, screening tools employed in this project, and targeted monitoring of anesthetic depth using BIS to prevent deep states of anesthesia. Preoperative cognitive assessment was incorporated into the preanesthetic evaluation for patients over the age of 60 undergoing total knee arthroplasty (TKA) or total hip arthroplasty (THA). The Mini-Cog assessment tool was administered to patients during in-person preoperative assessments. Patients unable to be seen in-person were evaluated over the phone using the Blessed Orientation Memory Concentration (BOMC) assessment. Patients with a Mini-Cog < 4 or a BOMC > 4 or a preexisting diagnosis of

dementia or cognitive impairment were considered to have preoperative cognitive impairment. Anesthetic depth was monitored using BIS, targeted to an index range of 40–60. The primary outcome measures for this quality improvement project were implementation of preoperative cognitive screening and utilization of intraoperative BIS monitoring. A retrospective chart review determined if qualifying patients received a preoperative cognitive assessment and if BIS monitoring was utilized. BIS monitoring data was then analyzed to determine the percentage of the total anesthetic time, measured in minutes, that the index was maintained at or above 40. Project participants also received a postdischarge phone assessment using The Family Confusion Assessment Method (FAM-CAM) to determine if symptoms of delirium were present

Outcome

Sixty-four patients met the inclusion criteria. Fifty-two percent received a preoperative cognitive assessment (15% in the first month, 74% in the second month); 25% of patients who completed a preoperative cognitive assessment screened positive for cognitive impairment; 73% of patients were monitored using BIS, an 83% increase from preintervention data. Of the patients monitored with BIS, 77% remained above the target index of 40 for 95% or more of the case. Forty-nine percent spent no time below the target index, and 9% spent 50% or more of the case below the target index. Five percent of patients screened positive for delirium postdischarge. Survey results gathered before and after a staff education session demonstrated an 82% increase in anesthesia practitioners' rating for the importance of anesthesia care in preventing POD. The perceived importance of knowing a patient's baseline cognitive status increased by 10%, and the likelihood of monitoring anesthetic depth in older adults increased by 18% after the training.

Conclusion

Establishing a patient's baseline cognitive function is an essential element of the pre-anesthetic evaluation. Provider awareness of the potential for cognitive decline in the perioperative period can increase the use of BIS monitoring to avoid deep planes of anesthesia, thereby improving patient outcomes. Extending preoperative cognitive assessment and BIS monitoring to all surgical patients over 60 would provide a more holistic anesthetic approach. Incorporation of postoperative POD screening into the nursing electronic medical record would standardize and improve detection of POD in surgical patients. This data could be utilized to evolve current protocols and develop new interventions for prevention. To facilitate enhanced perioperative cognitive protection, future studies should seek to expand current understanding of anesthesia's impact on postoperative cognitive function and identify additional neuroprotective strategies.

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Mentor: Joanne Donnelly, DNP, APRN, CRNA

Peer Mentorship in a Graduate Nurse Anesthesia Program

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Keywords: nurse anesthesia, peer mentor, graduate student support, wellness, coping

Introduction

Nurse anesthesia students may experience high stress levels during all phases of anesthesia programs, but for some, the didactic phase is extremely stressful. The process to get into a program is demanding and for some students, the fear of failing out of the program is very real.[1] There is a significant amount of new knowledge covered in the didactic phase in a very short period of time. Students may have been out of school for many years and acquisition of efficient study skills can be challenging.

There are a limited number of nurse anesthesia programs currently using structured peer mentorship. The developed characteristics of stronger problem-solving abilities, leadership skills, professional advocacy, and positive coping mechanisms are invaluable to the professional organization of nurse anesthetists in addition to the individuals that are involved in mentorship.[2,3] A peer mentorship program could reduce stress for students during the didactic phase and assist in providing problem-solving and time management skills needed for student success. Peer mentorship can also provide encouragement, develop a sense of direction and goals, and increase motivation to persevere through the nurse anesthesia program.[3-5]

The purpose of this scholarly doctoral project was to implement a pilot peer mentorship program to determine student knowledge, satisfaction, compliance, and the feasibility of a formal peer mentorship program within the Nurse Anesthesia Program at Mount Marty University. The goal of this project was to see an increase in student knowledge, compliance, and satisfaction.

Design and Methods

The peer mentor program consisted of 34 students from the Class of 2022 and 32 students from the Class of 2023. Students from each cohort were initially grouped into primary clinical location, with further random pairing within that location. The role of mentor was assigned to the Class of 2022 while the role of mentee was assigned to the Class of 2023. An educational session was provided to each cohort with pre-implementation surveys provided prior to the session. The educational session provided education regarding the mentor-mentee relationship, guidelines for success, an optional discussion questionnaire, and the minimum requirements for the program. After a six-month period, post-implementation surveys were sent out to both cohorts. Both the pre-implementation and post-implementation surveys remained anonymous and consisted of 5-point Likert scale questions with areas for open-ended feedback post-implementation. Student compliance, student satisfaction, and student knowledge were measured from pre-implementation to post-implementation.

Outcome

A total of 38 post-implementation surveys were returned out of 65 total surveys distributed between both cohorts. Average knowledge scores increased by 18 and 13% for the 2022 and 2023 cohorts, respectively. Satisfaction scores increased by 6% for the 2022 cohort and declined by 9% for the 2023 cohort. Compliance was measured by a single question on the post-implementation survey. Forty-one percent of mentors' mentee reached out to a friend or other fellow student more than the paired mentor. Seventy-one percent of mentees stated that they reached out to a fellow student instead of their mentor. Open-ended feedback from both cohorts expressed overall satisfaction with the mentoring process, desire to be a mentor for future students, and suggestions for improvement.

Conclusion

Evidence shows that peer mentorship provides support for nurse anesthesia students, leading to the promotion of positive coping mechanisms, strengthening of critical thinking and problem-solving skills, and increasing student success.[1,2] While the body of evidence relates primarily to the clinical setting, the same principles of mentorship can be applied to the didactic phase of a nurse anesthesia program. Relationships created through mentorship could create long-lasting professional attributes that prove invaluable to the nurse anesthesia profession.

An increase in student knowledge after an educational session, as demonstrated in this doctoral project, has the potential to encourage the continuation of the peer mentor relationship not only in the clinical arena, but into the post-education arena as well. An increase in knowledge of the benefits of mentorship can encourage SRNAs and CRNAs to support their educational and professional peers. Further development of a mentorship program with an extended system for mentor and mentee pairing that considers similar backgrounds, hobbies, family structure, and prior student relationships, could contribute to overall increased student satisfaction. The extended system for pairing encompasses a detailed survey for both mentors and mentees, thus gathering extensive information and leading to the optimization of matching; this would also have the potential to increase student compliance within the mentor relationship.

The implementation of a pilot peer mentorship program at Mount Marty University and the measurement of student knowledge, satisfaction, and compliance, demonstrates the untapped potential of a formally structured mentorship program. The data from the scholarly project supports the potential benefits for the mentor and mentee involved and the profession of nurse anesthetists. Further research and implementation of peer mentorship would only prove to be beneficial and necessitates continued consideration by anesthesia programs, faculty, and students alike.

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Mentor: Taylor Rehfeldt, DNAP, CRNA

Editorial

Looking back: In the early 1900s, evolving surgical techniques and World War I prompted a need for trained anesthesia providers. Nurses were among those who were trained to administer anesthesia. Formalized anesthesia education programs, which included lectures on physiology, pharmacology, and anesthesia techniques, as well as supervised clinical, were established. In 1915, Agatha Hodgins established the Lakeside Hospital School of Anesthesia. Over the next decade programs were established at major hospitals across the country. It was not until 1935 that standards for the curriculum of programs of anesthesia education were set forth. The course of study was a minimum of six months in length. Plans to lengthen the time to one year were postponed by World War II. The first certification exam was given in 1945. Nurse anesthesia program accreditation was instituted in 1952 and programs were required to be at least one year in length. In 1955 the US Department of Health, Education and Welfare recognized the AANA as the accrediting agency for schools of nurse anesthesia. The accreditation function was transferred to the Council on Accreditation of Nurse Anesthesia Educational Programs/Schools in 1975 in response to a major revision of U.S. Office of Education criteria. Prior to 1998 nurse anesthesia programs granted certificates, bachelor's, master's, or doctoral degrees. Starting in 1998 all nurse anesthesia programs had to grant a minimum of a master's degree for entry into practice.

Looking forward: In June 2007, the American Association of Nurse Anesthetists (AANA) Board of Directors approved a recommendation to move entry to practice for nurse anesthetists to the doctoral level by 2025. As occurred in the 1990s when certificate and bachelor's programs were phased out, master's programs have been phased out over the last decade. Students entering programs in 2021 are the last that may graduate with master's degrees. All accredited entry-level nurse anesthesia programs matriculating students on or after January 1, 2022 must grant doctoral degrees.

The goal of the International Student Journal of Nurse Anesthesia 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". This aligns with the degree advancement of nurse anesthesia programs and provides a venue exclusively for entry-level nurse anesthesia students to publish their work.

Thank you to those student authors, editors, reviewers, mentors, preceptors, and educators who have worked so diligently over the past twenty years to make this journal a success.

Sincerely,

Julie A. Pearson, PhD, CRNA
Associate Editor

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 reports must be single-authored, while EBP analysis reports and 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 and author'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 assign a submission number and 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. Submissions are reviewed using the Track Changes function of Word. The editor will return the item to the chief editor, who will return it to the mentor for appropriate action. **The mentor should guide the author through the revision process. The revised copy must be returned clean (no comments or Track Changes) with the original submission number in the filename and subject line of the email.** 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 11th ed., 5.4.2):

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 (Scribbr, 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 11th ed., the same guide utilized by the *AANA Journal* and such prominent textbooks as *Nurse Anesthesia* by Nagelhout and Elisha). Section numbers from the online version 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.

Reference: Christiansen S, Iverson C, Flanagin A, et al. *AMA Manual of Style: A Guide for Authors and Editors*. 11th ed. Oxford University Press; 2020.

Please note the following:

1. Use complete sentences.
2. Acronyms/Initialisms (2.1.5, 10.6, 13.9) - 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 (13.0)
4. Use *Index Medicus* journal title abbreviations (3.11.2, http://www.ncbi.nlm.nih.gov/nlmcatalog/journals)
5. Always provide units of measure (17.0). 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 ºC, 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). The manual includes a complete list of SI units (17.1 – 17.5).
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 (2.1.3, 10.3.5) - 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 in parenthesis (e.g. a GlideScope (Verathon Inc.) was used) (14.5.1). 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. If referencing software is used (Endnote, Zotero, etc.), any embedded <u>formatting must be removed</u> 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 <u>patient</u> 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 **<u>AMA Manual of Style must be adhered to</u>** 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.

<u>Heading</u> – 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 pertaining to your topic.

Case Report (400-600 words)

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).

Discussion (600-800 words)

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.

References

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.

Mentor: mentor name, credentials

E-mail address: *(*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)

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.
Methods (bold)
Include the format used for formulating the specific question you seek to answer, search terms and methods used, and levels of evidence.
Literature Analysis (bold)
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. Please follow AMA formatting guidelines for your table (4.1.2, 10.2.3). Your review and discussion of the literature should logically lead to support a practice recommendation. Subheadings may be used if desired.
Conclusions (bold)
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)
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)
A brief introductory paragraph including purpose (what change is intended) and rationale (why change is needed/evidence to support the change) here.
Design and Methods (bold)
Include population, intervention, and measures
Outcome (bold)
Present results from statistical analysis – do not justify or discuss here.
Conclusion (bold)
Discuss results (implications). Optionally include limitations, suggestions for future projects/research.
References (bold, 5 maximum)
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)
A brief introductory paragraph including purpose and hypotheses.
Methods (bold)
Include sample and research design
Results (bold)
Present results from statistical analysis – do not justify or discuss here.
Discussion (bold)
Discuss results (implications, limitations, suggestions for future research)
References (bold, 5 maximum)
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.

Journal names should be in italics and abbreviated according to the listing in the PubMed Journals Database. PubMed can also be used to perform a search: 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 (3.11) - 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, 3.15.2) should be included (see examples 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 (3.15) - 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 accessed date may be the only date available. The URL must be functional and take the reader directly to the source of the information cited.

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

Examples:

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

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

Textbooks (3.12) - 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. Springer; 2017:405-430.

Chapter from an edited text (3.12.4)**:**

Pellegrini JE. Regional anesthesia. In Nagelhout JJ, Elisha S, eds. *Nurse Anesthesia*. 6th ed. 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 (including spaces)
___ 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 Word document 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