Volume 21 Issue 3 Fall 2022

The International Student Journal of Nurse Anesthesia

TOPICS IN THIS ISSUE

Postoperative Nausea and Vomiting
Anesthetic Technique and Cancer
Sphenopalatine Ganglion Block
Neurolytic Celiac Plexus Block
Von Hippel-Lindau Disease
Achondroplastic Dwarfism
Adult Fontan Physiology
Malignant Hyperthermia
Awake Tracheostomy

INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA
Vol. 21 No. 3 Fall 2022

Front Cover:
On the front cover, Lauren Hodson, BSN, RN and Erinn Eliason, BSN, RN, doctoral students enrolled in the Westminster College Nurse Anesthesia Program, practice video laryngoscopy under the guidance of Program Director Daniel Bunker, DNAP, MSNA, CRNA.

Publication Information:

The International Student Journal of Nurse Anesthesia (ISSN 2688-5263) is published three times a year in the spring, summer, and fall. Current and past issues, and the Guide for Authors of this free, open access, electronic journal can be found at:

www.aana.com - Member Center → Students → International Student Journal
https://www.aana.com/studentjournal
https://ifna.site/international-publications/international-student-journal-for-nurse-anesthesia/
Please note, the AANA maintains back issues for the past three years. The IFNA maintains back issues from 2009 on.

For additional information, please contact:

Vicki Callan, PhD, CRNA, CHSE, FAANA
Webster University Department of Nurse Anesthesia
Browning Hall, ISB 107
8274 Big Bend Blvd.
St. Louis, MO 63119
314-246-5928; Intsjna@aol.com

Table of Contents

Anesthetic Technique and Cancer

Eric Stradley, DNAP, BSN
Westminster College

Keywords: Cancer proliferation, anesthetic effect, agent choice, TIVA

Surgical intervention for cancer is a growing field of medicine, and likewise, a growing patient population for anesthesia care. The effect of surgical manipulation on cancer and the spread of cancer is a well-known and studied aspect of cancer proliferation. The effects of anesthesia and the agents used during a surgical procedure on cancer and its proliferation are drawing more attention and questions. The agents used for anesthesia have varying effects on immunity and may pose a risk to individuals with active cancer or who are in remission.[1,2]

Case Report

A 78-year-old, 51 kg, 162.6 female presented for an open biopsy of her left breast. The patient's diagnosis was inflammatory breast cancer and had been receiving intravenous chemotherapy for two months. The patient's medical history included chemotherapy-induced diabetes mellitus, gastric reflux, obstructive sleep apnea, tricuspid valve regurgitation, and hypertension. Upon examination, the patient's allergies were adhesive tape, aspirin, codeine, wheat, and citric acid. The patient's past surgical history included hysterectomy, bilateral oophorectomy, right mastectomy, and Port-a-Cath port placement. The patient had no history of anesthesia complications. An airway examination indicated a Mallampati score of 2, a thyromental distance of 6 cm, mouth opening of 5 cm, normal neck range of motion, complete set of dentures, and no previous difficulties with intubation. The patient's medications included: duloxetine, metformin, furosemide, rosuvastatin, enalapril, cyclobenzaprine, and dexamethasone. The patient received 1,000 mg of acetaminophen per os (PO) preoperatively, and an 18-gauge peripheral intravenous (IV) catheter was placed, and lactated ringers infused. Preoperative vital signs showed a blood pressure 142/69 mm Hg, heart rate 96/min, SpO_2 98% on room air, respiratory rate 18/min, and temperature 37.1°C.

Upon arriving in the operating room, standard noninvasive monitoring was applied, O_2 8 L/min was applied by mask, and dexmedetomidine 10 mcg was administered. General anesthesia was induced with 1% lidocaine 70 mg, ketamine 25 mg, esmolol 20 mg, propofol 150 mg, rocuronium 30 mg. Direct laryngoscopy was performed and endotracheal intubation was achieved with a 7.0 mm oral endotracheal tube upon observation of a Cormack Lehane grade one view of the glottic opening. Propofol 150 mcg/kg/min, dexmedetomidine 0.7 mcg/kg/h and magnesium 1 g/h infusions were initiated. A pectoral nerve (PECs 1) block was performed on the left side of the thorax using ultrasound guidance. Bupivacaine 0.25% 22.5 mg with dexamethasone 2 mg, and buprenorphine 0.15 mg was added for a total of 10 mL.

During the procedure, hypotension was treated with LR boluses of 250 mL and phenylephrine injections of 100 mcg aliquots. A lower body warmer was used on the patient to encourage normothermia. Nausea and vomiting prophylaxis was accomplished with dexamethasone 4 mg IV, given shortly after induction and ondansetron 4 mg IV at the end of the case. The patient's

hemodynamics remained stable throughout the procedure with a decrease in heart rate from 96 bpm to 71/min, likely attributed to the dexmedetomidine infusion. A total of 500 mL of LR was given as bolus and phenylephrine 100 mcg IV 3 times to maintain a mean arterial pressure of at least 65-70 mm Hg.

The procedure lasted 2 hours. Upon initiation of closing the surgical site, the IV propofol and dexmedetomidine were decreased by half and the patient's residual neuromuscular blockade was treated with sugammadex 100 mg. The propofol and dexmedetomidine infusions were stopped once the surgical incision was closed. The patient remained stable through emergence and endotracheal tube extubation. The patient was then transferred to the post-anesthesia care unit (PACU) on O_2 6 L/min via simple mask. Once in the PACU, the patient reported a pain scale of 0/10 and remained stable through recovery.

Discussion

Cancer is an increasing challenge and burden to the healthcare system and the general population. Cancer is the second leading cause of mortality globally and the incidence of cancer continuing to rise every year. More than 18 million new cancer diagnoses and over 9.5 million deaths were recorded in 2018.[3] Surgery and anesthesia stimulate the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic nervous system (SNS) to cause immunosuppression through several tumor-derived soluble factors.[1] Cancer-related mortality is often the result of a metastatic recurrence.

Different anesthetic techniques have varying effects on innate and cellular immunity, activation of adrenergic-inflammatory pathways, and activation of cancer-promoting cellular signaling pathways; these effects may translate into an influence of anesthetic technique on long-term cancer outcomes.[2] Volatile anesthetics produce a variety of effects on the immune system suppressing the body's immune function in a multitude of ways. Volatile anesthetics induce T-lymphocyte apoptosis, increase the expression of hypoxia-inducible factor 1a (HIF 1a), producing angiogenesis and cell proliferation. Volatile gases increase levels of pro-tumorigenic cytokines, increase levels of matrix metalloproteinases (MMP) aiding in the invasion of tumor cells to other areas of the body, and decrease lymphocyte and natural kill cell (NK cell) activity.[3]

Opioid analgesics use has shown a decrease in cellular immunity and activity. Morphine has been linked to inhibition of T-lymphocyte proliferation, the suppression of NK cell activity and the suppression of T cell differentiation.[1] Opioid-induced cell proliferation and cell death likely depend on the opioid concentration or exposure duration. It was shown that opioid analgesics promoted lymphocyte apoptosis and the decrease in Toll-like receptor 4 (TLR4) expression on macrophages.[1] These TLR4 allow the macrophages to recognize molecules produced by pathogens.[1] Fentanyl, sufentanil, remifentanil, and alfentanil also produce immunologic effects by decreasing NK cell activity, inhibition of leukocyte migration and proliferation, and increased regulatory T cells. Overexpression of the μ-opioid receptor (MOR), which promotes tumor growth and metastasis, is observed in several human cancers.[1]

Propofol total intravenous anesthesia (TIVA) appears to have a better effect on the immune system compared to volatile anesthetics and opioids. Propofol does not inhibit the growth of

cancer cells, but it does inhibit the cancer cell's invasion capability.[1] A protective effect on metastases has been found with propofol and inhibition of cyclooxygenase type 2 and prostaglandin E2 in tumor cells.[1] The risk of overall mortality was shown to be greater when sevoflurane was used as the primary anesthetic compared to propofol for 1-year and 5-year survival rates.[4]

Along with propofol-TIVA methods, the use of regional and local anesthetics has been shown to improve surgical outcomes and opioid usage postoperatively.[1] Regional anesthesia influences the long-term outcome of cancer surgery in three ways. First, regional anesthesia may attenuate the intrinsic immunosuppression from surgery. Second, patients who receive regional analgesia often do not need as much opioid treatment, and as a result, tend to avoid the immunosuppressive effects that accompany opioid treatment. Third, the combination therapy of regional and general anesthesia reduces the dose of inhalational anesthetic required. This decrease in the required dose can potentially affect long-term outcome from cancer-related surgery.[5] Local anesthetics suppress cancer proliferation and differentiation due to the abundance of sodium channels on the cancer cells, increase NK cell activity, decrease metastatic spread, and lidocaine and bupivacaine have shown apoptosis in breast cancer cells.[1]

This case was managed according to the most recent evidence-based studies and knowledge regarding anesthesia and cancer. The technique was well done, the regional block was appropriate for the surgery and placed correctly as visualized by ultrasound, and the patient-reported comfort postoperatively. Anesthesia technique can play a pivotal role in the mortality and outcomes of patients with cancer diagnoses. Research available today is showing the risks and benefits of techniques used. Knowledge about the effects of agents used in anesthesia on cancer is essential for providers to know and understand for the best patient outcomes. This case offered many learning points in the way that anesthesia providers can make more of a conscious effort to produce the best patient outcomes through unique, well-executed and individualized anesthetic plans. A key to success in this case and this technique was the choice of anesthetic agents used and a successful and effective regional block, allowing for a comprehensive decrease in immunosuppressive agents that carried the patient effectively through the procedure and the post-operative period.

References

1. Kim R. Effects of surgery and anesthetic choice on immunosuppression and cancer recurrence. *J Transl Med*. 2018;16(1). doi:10.1186/s12967-018-1389-7
2. Kaye AD, Patel N, Bueno FR, et al. Effect of opiates, anesthetic techniques, and other perioperative factors on surgical cancer patients. *Ochsner Journal*. 2014;14(2):216-228.
3. Edwards ZE, Kelliher LJ. Propofol-TIVA versus inhalational anesthesia for cancer surgery. *Dig Med Res*. 2020;3:15-15. doi:10.21037/dmr-20-58
4. Enlund M, Berglund A, Ahlstrand R, et al. Survival after primary breast cancer surgery following propofol or sevoflurane general anesthesia—A retrospective, multicenter, database analysis of 6305 Swedish patients. *Acta Anaesthesiol Scand*. 2020;64(8):1048-1054. doi:10.1111/aas.13644

5. Yap A, Lopez-Olivo MA, Dubowitz J, Hiller J, Riedel B. Anesthetic technique and cancer outcomes: a meta-analysis of total intravenous versus volatile anesthesia. *Can J Anaesth*. 2019;66(5):546-561. doi:10.1007/s12630-019-01330-x

Mentor: Art Shimata, DNAP, CRNA

Awake Tracheostomy following Ludwig's Angina

Kristian A. Hem, BSN
Barry University

Keywords: awake tracheostomy, Ludwig's angina, airway topicalization

Ludwig's angina (LA) is a form of gangrenous cellulitis, usually originating from dental infections which spread along the fascial planes of the floor of the mouth causing extensive swelling of the tongue, submandibular region, and the neck. While there is only a 10 percent incidence in at-risk populations, untreated LA is an urgent threat to airway patency and spontaneous ventilation. The impending airway obstruction with the presence of a difficult airway creates challenges for surgeons and anesthesia practitioners, necessitating multidisciplinary collaboration to attain the highest patient safety and best outcomes.

Case Report

A 29-year-old, 69 kg male patient with Ludwig's angina was scheduled for an elective awake tracheostomy (AT) and repeat incision and drainage (I&D) of recurrent abscesses despite pharmacologic intervention and multiple attempts of surgical decompression. Past medical history included dental caries accompanied by a dental infection. Initial outpatient treatment with amoxicillin and dexamethasone was unsuccessful which led to airway compromise and emergent intubation at an outside facility. Due to the nature of his condition, otorhinolaryngology (ENT) services were needed, warranting transfer to a larger facility. A bedside transoral and transcervical I&D of the submental abscess was performed upon admission to the medical intensive care unit (MICU). Worsening facial swelling and trismus ensued, warranting an awake fiberoptic nasal intubation, followed by multiple dental extractions and a subsequent I&D of the submandibular abscess under general anesthesia. Approximately 48 hours later, the patient required a third I&D and consented to an elective AT.

The preoperative evaluation revealed extensive facial, submandibular, and cervical edema, dysphonia and drooling, limited neck mobility, inter-incisor opening of < 1 fingerbreadth, multiple missing teeth due to prior extractions, and patent nares. Due to his limited mouth opening, an airway assessment and Mallampati score could not be accurately determined and, therefore, was deemed a difficult airway. Preoperative laboratory values reported leukocytosis (white blood cell count of 14.6 K/uL) and elevated lactate and procalcitonin levels, 2.1 mmol/L and 0.53 ng/mL, respectively. Computed tomography scan findings revealed extensive inflammation of the neck and floor of the mouth with abscess formation, consistent with LA, and reactive mucosal edema involving the hypopharynx and oral cavity. Current inpatient

medications included ampicillin/sulbactam 3 g, acetaminophen 650 mg, and chlorhexidine 0.12% solution.

Preoperatively, difficult airway equipment and a flexible fiberoptic bronchoscope were brought into the operating room (OR). Upon patient arrival to the OR, standard noninvasive monitors were applied, O_2 2 L/min was administered via nasal cannula, and capnography was confirmed through an $EtCO_2$ sampling line. Surgical landmarks of the lower airway were identified and marked by the surgeon. Midazolam 2 mg, glycopyrrolate 0.2 mg, and dexmedetomidine 8 mcg were administered IV, and a dexmedetomidine infusion was initiated at 2 mcg/kg/hr. The patient's SpO_2 measured 99%, and respiratory rate was 16/min. Oxymetazoline spray was administered in each nare. The patient's nasopharynx was then sprayed with 4% lidocaine laryngotracheal anesthesia (LTA), followed by insertion of a 28 Fr nasopharyngeal airway (NPA) lubricated with 5% lidocaine ointment. The surgeon infiltrated lidocaine HCl 2% with epinephrine 1:100,000 into the patient's neck for the "awake" portion of the procedure. Fentanyl 25 mcg IV and midazolam 2 mg IV were administered at the time of incision. A tracheal stoma was surgically created, and a 6.0 mm reinforced endotracheal tube (ETT) was inserted into the trachea. The ETT cuff was inflated, and after $EtCO_2$ confirmation, the patient underwent IV induction and general anesthesia by administration of propofol 200 mg, fentanyl 75 mcg, rocuronium 50 mg, ketamine 50 mg, and sevoflurane 1.8% inspired concentration in a mixture of O_2 1 L/min and air 1 L/min. Mechanical ventilation was initiated. At this point, the dexmedetomidine infusion was reduced to a rate of 1 mcg/kg/hr.

Following completion of his tracheostomy the I&D was performed and culture specimens of the submental, left submandibular, and left masseteric spaces were collected. Fentanyl was administered in 50 and 25 mcg increments, and the infusion rate of dexmedetomidine was titrated to 0.5 mcg/kg/hr after the patient became unconscious. Following the I&D, the affected spaces were irrigated with saline and Betadine. Penrose drains were left in place, and the neck incisions were approximated via suturing. Sevoflurane and dexmedetomidine infusion were discontinued. Before emergence, ondansetron 4 mg and sugammadex 200 mg IV were given. When the patient was spontaneously breathing and responsive to verbal commands, the surgeon exchanged the reinforced ETT for a 6 Shiley cuffed tracheostomy tube. A tracheostomy collar was immediately applied with blow-by O_2 delivered at 8 L/min. The patient was transferred to the postanesthesia care unit, where he remained stable with a patent airway. Per ENT orders, the patient was transferred back to the MICU following the recovery phase.

Discussion

The first description of LA dates to 1836, when Friedrich Wilhelm von Ludwig, a German surgeon, reported the unique criteria of this form of cellulitis. The Latin term "angere," contemporarily known as angina, means "to strangle."[2] Early references to Ludwig's description state LA is a gangrenous cellulitis affecting the soft tissues of the neck, most notably caused by odontogenic infections associated with the molar teeth. Other contributing factors include mandibular fractures, ear infections, oral lacerations, injuries to the floor of the mouth, and systemic illnesses, such as malnutrition, diabetes mellitus, alcoholism, and immune-compromising conditions.[2,3] Originating in the submandibular space along the mylohyoid ridge, LA is known for its abrupt extension to the floor of the mouth and deep cervical fascia and its

widespread edema to the submandibular and sublingual spaces. Molar teeth are linked with LA due to their roots near the mylohyoid ridge, which potentiates the ease of transmitting infection. Another observable characteristic of LA is the lack of lymphatic involvement.[1] As dental hygiene and antibiotic utilization have improved, LA's incidence has declined by 50% in the last 100 years.[2]

Although LA can present at any time, it typically occurs in middle-aged males. The risk of developing LA increases as socioeconomic status, preventive and routine dental care decreases.[2] Clinical presentation can include neck tenderness, bilateral cervical swelling or "bull neck," sore throat, drooling, pain in the floor of the mouth, malaise, dysphonia, dysphagia, limited range of neck movement, tongue swelling, and stridor.[2] Crepitus in the neck may be present due to gas production by bacteria. Because the infection spreads so rapidly, the tongue is often displaced posteriorly, risking obstruction of the oropharynx. Cervical edema can precipitate airway obstruction if compression of the laryngeal cavity occurs.[2] Treatment of LA includes intravenous antibiotics and potential surgical decompression. Research data indicate a lower incidence of airway compromise when patients are treated with I&D and intravenous antibiotics.[3]

Difficult mask ventilation should be anticipated in patients with LA. Patients will generally have a Mallampati classification III or IV and inadequate mouth opening (< 4cm), creating airway management challenges. Reviewing the preoperative imaging scans may help discern the severity of swelling before airway manipulation.[2] A review of the literature described no gold standard for LA when selecting an approach to manage the airway.[2-6] The intubation techniques used most frequently include awake blind nasal intubation, flexible fiber-optic oral or nasal intubation, and AT, which is implemented as a last resort to its alternatives.[2,4] Emergent tracheostomy or cricothyrotomy must be immediately performed when noninvasive methods fail to secure the airway.[2]

The patient suffered from worsening facial swelling and firmness of the floor of the mouth, despite previous interventions and intravenous antibiotic therapy. Considering the patient's clinical presentation, the otolaryngologist recommended an AT preceding his I&D. The surgeon was highly concerned about airway deviation and distortion of neck anatomy due to the amount of edematous and indurated tissue present. Repeated intubation attempts can lead to bleeding and further exacerbate oropharyngeal edema.[5] Thus, AT was deemed a safer option for intra- and postoperative airway protection.

It is essential to address patient discomfort and stress associated with AT.[4] In addition to selecting pharmacologic interventions, “verbal anesthesia” consisting of preoperative patient education and reassurance plays a critical role in successful outcomes. There is no standard approach for sedation and airway topicalization.[2,5,6] Sedation during an AT requires that the patient maintain spontaneous ventilation, hemodynamic stability, and the ability to respond to verbal commands while avoiding oversedation, apnea, aspiration, and hypoxia.[6] The strategy implemented included anxiolysis, amnesia, suppression of the cough and gag reflexes, and analgesia. Benzodiazepines, alpha-2 agonists, and opioids were titrated to effect without causing oversedation or respiratory depression. Numerous modalities can execute airway topicalization, including atomization, nebulization, the spray-as-you-go technique, transtracheal injection, regional anesthesia, and surgical infiltration.[2,6] In this case, 4% lidocaine LTA was utilized as an

effective and noninvasive method to topicalize the airway because the patient had limited mouth opening. Inserting an NPA coated in lidocaine jelly served as an additional safeguard to maintain spontaneous ventilation. When formulating an anesthetic plan, it is imperative to consider how the delivery device affects the absorption rate of local anesthetic to avoid high concentrations and local anesthetic systemic toxicity.[6]

The clinical presentation of LA and airway limitations can vary from patient to patient, preventing an established, universal technique for anesthesia professionals. Although the methods utilized in this case paralleled current literature recommendations, further exploration is needed to develop a definitive algorithm for anesthesia management during AT.

References

1. Tschiassny K. Ludwig's angina: An anatomic study of the role of the lower molar teeth in its pathogenesis. *Arch Otolaryngol* 1943; 38(5):485-496.
2. Dowdy REA, Emam HA, Cornelius BW. Ludwig's angina: Anesthetic management. *Anesth Prog* 2019; 66:103-110.
3. Edetanlen BE, Saheeb BD. Comparison of outcomes in conservative versus surgical treatments for Ludwig's angina. *Med Princ Pract* 2018; 27:362-366.
4. Sagiv D, Nachalon Y, Mansour J, et al. Awake tracheostomy: Indications, complications and outcome. *World J Surg* 2018; 42:2792-2799.
5. Lasala J, Guerra-Londono CE, Truong DT, Truong AT. Airway obstruction attributable to head and neck cancers: Awake fiberoptic intubation versus awake tracheostomy. *Anesthesiology* 2021; 134(6):937
6. Kostyk P, Francois K, Salik I. Airway anesthesia for awake tracheal intubation: A review of the literature. *Cureus* 2021; 13(7):16315.

Mentor: Jeanne M. Antolchick, Ph.D., APRN, CRNA

Anesthetic Considerations for the Achondroplastic Dwarf

Crystal Blackburn, MSNA
Westminster College

Keywords: achondroplasia, dwarfism, subglottic stenosis, anesthesia

Achondroplasia is the most common cause of dwarfism, affecting 1.5 of every 10,000 births.[1-6] This skeletal dysplasia is an inherited autosomal dominant trait that hinders cartilage formation, resulting in disproportionately short limbs compared with body size. [1-6.] Multiple organ systems can be affected by achondroplasia, [1,3,7] and anatomic abnormalities of the head, spine, and airway pose challenges for anesthesia practitioners caring for this population.[1-8] There is paucity of recent literature regarding anesthetic management of patients with achondroplasia. This case report summarizes anesthetic considerations and administration of deep sedation for an achondroplastic dwarf with subglottic stenosis.

Case Report

A 29-year-old male presented for direct laryngoscopy with the use of rigid endoscope to assess for tracheal stenosis. Significant details in his medical history included dwarfism and cholelithiasis with cholecystitis. During induction of anesthesia for the laparoscopic cholecystectomy, the vocal cords were visualized using video laryngoscopy. However, endotracheal intubation with a 5.0 endotracheal tube (ETT) was unsuccessful, requiring cancelation of the procedure. Postoperatively, the patient developed stridor requiring racemic epinephrine, steroid treatment, and an overnight stay in the intensive care unit. Consequently, the patient was referred to ear, nose, and throat (ENT) services to assess for tracheal narrowing. An ENT surgeon performed an office-based flexible fiberoptic examination. The assessment revealed a large amount of swelling and concern for possible unilateral vocal cord paralysis. This prompted the need for direct laryngoscopy in the operating room (OR). Preoperative testing included computerized tomography (CT) showing narrowing of the proximal trachea, and significant kyphosis of the upper dorsal spine from T2-T7. Cervical spine x-rays identified no gross abnormalities involving cervical or atlantoaxial instability. A chest x-ray was obtained; no evidence of acute cardiopulmonary disease was present.

Physical exam demonstrated a patient of short stature, 81 cm in height and 22 kg in weight, with disproportionally short limbs. Airway assessment revealed a Mallampati class I airway, thyromental distance >6 cm and full cervical neck range of motion. Notably, the patient had a gruff voice, which he stated was baseline. He denied problems with swallowing. The patient experienced shortness of breath with activity requiring the use of a wheelchair, resulting in a functional status of less than 4 metabolic equivalents. Lung sounds were clear to auscultation with normal heart tones.

In the preoperative phase, the patient received midazolam 2 mg intravenously (IV) prior to transfer to the OR. The OR team placed standard noninvasive monitors on the patient, including an appropriately sized blood pressure cuff. Oxygen 8 L/min was administered via adult sized facemask for at least 3 minutes prior to administering a mixture of N_2O 4 L/min and O_2 4 L/min. Ketamine 20 mg, fentanyl 25 mcg, lidocaine 30 mg, dexamethasone 10 mg and glycopyrrolate 0.1 mg were administered IV. After loss of consciousness, the patient maintained spontaneous respirations confirmed with $ETCO_2$. Maintenance anesthesia included sevoflurane 2% inspired concentration administered via circuit mask and propofol 150 mg IV given incrementally throughout the case. The ENT surgeon performed a direct laryngoscopy with a Miller 2 blade, avoiding neck manipulation to allow visualization of the true vocal cords. Lidocaine 4% 0.5 mL was sprayed onto the cords for topical anesthesia. After approximately 1 minute, the ENT surgeon again placed the laryngoscope and a 4mm rigid endoscope was used to visualize the supraglottic, glottic, and subglottic airway and the trachea, demonstrating significant narrowing at the cricoid. During airway manipulation, the patient's heart rate increased from 115 /min to 150/min and SpO_2 decreased from 100% to 88%. The heart rate slowed to 115/min after esmolol 5 mg IV, and the patient was allowed to breathe spontaneously with bag mask ventilation assistance until SpO_2 returned to 100%.

After completion of the examination, the patient awakened from anesthesia using O_2 10 L/min via anesthesia circuit. Subsequently, the patient developed significant stridor with chest wall

retractions and SpO_2 decreased to 87%. An oropharyngeal airway (OPA) was placed, epinephrine 1:10,000 2 mL was given via the OPA, and respirations were assisted using bag mask ventilation with O2 8 L/min until the SpO_2 was >95%. Stridor resolution was noted as the patient became more awake. When the patient was fully awake, no further stridor was heard. The patient was then transferred to the post-anesthesia care unit (PACU) on O_2 6 L/min via face mask. He was discharged 7 hours later without further sequelae.

Discussion

Achondroplasic dwarfs have anatomic alterations[1,3] such as a protruding forehead, depressed nasal bridge, mandibular enlargement, macroglossia, short neck, narrowed nasal passages, thickening of pharyngeal and laryngeal structures and laryngomalacia. These can make mask ventilation and laryngoscopy difficult.[1-6] Preparation is paramount in the management of a potentially difficult airway, including a variety of sizes of oral and nasal airways, endotracheal tubes, laryngeal mask airways (LMA) and emergency supplies.[1-4] All available previous records of airway management should be reviewed if possible.[1,2] During this case, the pediatric airway cart was placed in the OR with ETT sizes ranging from 3 to 6 mm, various sizes of LMAs, oropharyngeal airways, face masks, and emergency supplies. Adult and pediatric sized blood pressure cuffs were available to ensure appropriate sizing. Although guidelines for ETT sizing remain unclear for patients with achondroplasia, it is necessary to use a smaller ETT than those used in normal patients of similar age.[2-5] Using weight rather than age to anticipate the appropriate size of ETT is suggested.[2-4] Rigid endoscopic evaluation of the above patient determined that a size 3.0 mm ETT would be needed to successfully intubate the trachea, given the significant stenosis at the cricoid. Accurate blood pressure measurements require the use of an appropriately sized cuff, covering two thirds the length of the upper arm.[2]

Thorough evaluation of the cranio-cervical junction is necessary in patients with dwarfism.[1,3-7] Flexion-extension lateral cervical spine radiograph, magnetic resonance imaging (MRI) or CT should be considered due to the risk of atlantoaxial subluxation. Furthermore, foramen magnum stenosis may cause compression of the medulla and cervical spinal cord with extreme neck manipulation.[1-7] Maintaining in-line cervical stabilization may be required in patients who are high risk for cervicomedullary compression during laryngoscopy.[1-7] Awake fiberoptic intubation techniques are suggested as the safest method of securing the airway, especially in patients with spinal cord compression.[1-6] Premature fusion of the bones in the base of the skull can lead to limited neck extension and difficult tracheal intubation.[1-7] If the patient has full cervical motion without pain and has no history of neurological symptoms, gentle direct laryngoscopy with minimal neck manipulation can result in safe tracheal intubation.[4.]

Achondroplasia presents with altered thoracic anatomy including kyphosis, scoliosis, lumbar lordosis, and rib deformities.[1-3,6,7] These cause restrictive lung diseases, pulmonary hypertension and rarely, cor pulmonale.[1-3,7] Restrictive lung disease results in altered ventilation/perfusion (V/Q) matching and decreases in vital capacity and functional residual capacity.[1,3]
Joint deformities, laxity of skin and excess subcutaneous tissue can make IV access challenging.[2,4,5] Intravenous cannulation is recommended prior to induction given the potential for airway complications.[4] Central venous cannulation can also be challenging in this patient population given their short necks and limited mobility.[2,5] Ultrasound-guided techniques can

increase the chances of success with intravenous or central venous cannulation and should be considered if difficulty arises.[7]

There are limited recommendations in the literature regarding appropriate drug dosage selection in patients with achondroplasia.[3,8] The most definitive guideline is to calculate drug doses from body weight rather than age.[8] However, one case study documented failure to achieve appropriate rapid sequence intubation (RSI) conditions in an achondroplastic patient using weight-based dosing. The patient required two additional doses of thiopentone and rocuronium to obtain adequate intubating conditions.[8] The relative differences in organ mass may result in higher blood flow to the liver and central nervous system, thus suggesting that dwarfs may require drug doses similar to adults of normal size.[8] In cases where RSI induction is necessary, it may be advisable to administer drug dosages based off age vs. weight to facilitate expeditious intubation of the trachea.[3,8] Further investigation into the pharmacokinetics in achondroplasia is required to definitively determine appropriate drug dosing.[8]

In summary, achondroplasia presents unique challenges to the management of anesthesia. Alterations in airway anatomy, skeletal deformities, respiratory function, and pharmacokinetics require specific considerations. Meticulous pre-anesthetic evaluation including preoperative imaging and a thorough history and physical are key in anticipating difficulties during the perioperative period.[1-7] Planning is of utmost importance in patients with dwarfism. This should include access to difficult airway equipment, and multiple sizes of airway adjuncts.[1-4] Last, successful delivery of an anesthetic plan must involve careful selection of appropriate drug dosages tailored to specific anatomic alterations, associated comorbidities and clinical circumstances.[3,8]

References

1. Spiegel J, Hellman M. Achondroplasia: implications and management strategies for anesthesia. *Anesthesiology News.* 2015:63-66. Accessed date. August 12, 2021. https://www.anesthesiologynews.com/download/Dwarfism_ANSE15_WM.pdf
2. Berkowitz ID, Raja SN, Bender KS, Kopits SE. Dwarfs: pathophysiology and anesthetic implications. *Anesthesiology.* 1990;73(4):739-759. PMID:2221443
3. Dang SH, Samra A, Patel BV, Sanchez-Luege S. Problematic airway and anesthetic dilemmas for achondroplastic dwarfism in the acute care setting: A case report. *Cureus*. 2022;14(5):e25336. doi:10.7759/cureus.25336
4. Monedero P, Garcia-Pedrajas F, Coca I, Fernandez-Liesa JI, Panadero A, de los Rios J. Is management of anesthesia in achondroplastic dwarfs really a challenge? *J Clin Anesth.* 1997;9(3):208-212. doi:10.1016/s0952-8180(97)00033-0
5. Neisevilie N, Puneet K, Dhruv Jain. Anaesthetic management of an achondroplastic dwarf with difficult airway and spine for total hip replacement: a case report. *Gen Med.* 2016;4(2). doi:10.4172/2327-5146.1000227
6. Kim JH, Woodruff BC, Girshin M. Anesthetic considerations in patients with achondroplasia. *Cureus*. 2021 Jun 22;13(6):e15832. doi: 10.7759/cureus.15832
7. Jain A, Jain K, Makkar J, Mangal K. Case study: anesthetic management of an achondroplastic dwarf undergoing radical nephrectomy. *S African J of Anaesth Analg.* 2010;16(2):77-79.

8. Kaur J, Durga P, Jonnavithula N, Ramachandran G. Failed rapid sequence induction in an achondroplastic dwarf. *Indian J Anaesth*. 2011;55(3):296-298. doi:10.4103/0019-5049.82691

Mentor: Ted Morris, DNAP, CRNA

Von Hippel-Lindau Disease: Anesthesia for a Spinal Tumor

Juan Carhuas, MSNA, BSN
Westminster College

Keywords: Von Hippel-Lindau disease, hemangioblastoma, tumor resection, spinal tumor, genetic diseases.

Von Hippel-Lindau (VHL) disease is an autosomal dominant syndrome manifested by a variety of tumors including hemangioblastomas, retinal angiomas, renal carcinomas, pheochromocytomas, and reproductive tumors.[1] Hemangioblastomas are rare, highly vascular, slow growing tumors of the central nervous system.[1] These tumors account for 4 percent of all spinal cord lesions and only 1 percent are associated with VHL disease.[1,2] Symptoms are secondary to direct compression from the tumor.[1] Surgical resection is the definitive treatment for symptomatic spinal cord hemangioblastomas.[2,3] This case study focuses on the anesthetic management of a VHL disease patient undergoing a spinal hemangioblastoma resection.

Case Report

A 15-year-old, 157 cm, 61 kg female presented for cervical spinal hemangioblastoma resection. The patient was newly diagnosed with VHL disease. Presenting signs and symptoms included progressive bilateral lower extremity weakness, urinary and fecal incontinence, and neck pain. Previous anesthetic history included general anesthesia for magnetic resonance imaging (MRI), cerebral angiogram, spinal angiogram, and endovascular embolization. These procedures were completed during the current hospital admission either as a diagnostic procedure, or in preparation for the hemangioblastoma resection. Airway assessment included Mallampati class II, mandibular protrusion test class II, and limited neck mobility. Lab values were within normal limits. Blood type was A positive.

In the preoperative area, the patient was pre-medicated with midazolam 2 mg intravenously and then taken to the operating room. Standards noninvasive monitors were applied. Baseline vital signs were within normal limits. After three minutes of denitrogenation with O_2 at 10 L/min via face mask, intravenous induction was performed with fentanyl 100 mcg, lidocaine 100 mg, and propofol 200 mg. Intubation of the trachea was done using video laryngoscopy. A 6.5 mm endotracheal tube (ETT) was secured at 19 cm at the teeth. Tube placement was confirmed by visualization of the tube passing the vocal cords via the Glidescope (Verathon Inc.), positive end tidal CO_2, and bilateral auscultation of breath sounds. Mechanical ventilation was initiated. The settings included a volume control mode with a tidal volume (V_t) of 450 mL, respiratory rate of 12 breaths per minute, and a positive end expiratory pressure (PEEP) of 5 cm H_2O. An additional 16-gauge IV catheter and a 20 gauge right radial arterial line were placed.

Anesthesia was provided with a continuous propofol infusion of 150 mcg/kg/min and a continuous remifentanil infusion of 0.2 mcg/kg/min, which was guided by a bispectral index (BIS) monitor (Medtronic Inc.) with a goal value of 40-60. Intraoperative neurophysiological monitoring (IOM) consisting of motor evoke potentials (MEPs) and somatosensory evoke potentials (SEPs) were initiated after induction and remained constant throughout the case.

Forty-five minutes prior to the end of surgery, magnesium sulfate 1 gram and hydromorphone 0.5 mg were administered. The remifentanil infusion was turned off, and the propofol infusion was decreased to 100 mcg/kg/min. Shortly after these changes, the patient started triggering spontaneous ventilation. At this point, the ventilator mode was switched to a pressure support mode. Total fluid intake was 2,700 mL consisting of lactated ringers 2,450 mL and 5% albumin 250 mL. Total urine output was 300 mL, and total estimated blood loss was 250 mL.

At the end of the 7-hour procedure, the surgeon requested to keep the patient intubated overnight. The patient was transported to the pediatric intensive care unit (PICU) with the propofol infusion unchanged at 100 mcg/kg/min. Upon arrival to the PICU, patient appeared comfortable, vital signs were stable, and care was transferred to the attending intensivist and nursing staff. Postoperative recovery was uneventful, and the patient was discharged seven days after with significant improvement of initial signs and symptoms.

Discussion

The patient in this case study noticed onset of neck pain four months prior to surgery. Symptoms became progressive as time went on. Patient sought medical help after fecal and urinary incontinence developed. The specific neurological deficit from a hemangioblastoma compression depends upon tumor location in the spinal cord and can include motor weakness, sensory deficits, and incontinence problems.[1-4] The MRI showed a 12 mm x 18 mm, highly vascular, lesion located within a cyst inside of the cervical spinal cord at the C4-C5 level. The preferred diagnostic method is gadolinium-enhanced MRI.[2,3] The typical MRI feature for a hemangioblastoma is an intensified nodule associated with a cyst located either in the central nervous system or peripheral nervous system.[2,3]

The patient's hemangioblastoma blood supply originated from the anterior spinal artery and the right vertebral artery. She had two arterial approach embolization procedures three days prior to the hemangioblastoma resection. The goal of embolization is to destroy blood supply vessels to the lesion, thus preventing growth of the tumor and decreasing the risk of hemorrhage during surgery.[3] It can be used as a nonsurgical intervention for smaller tumors or as an adjunct with surgery for larger tumors.[3] Acute tumor-associated hemorrhage either preoperative or intraoperative can cause severe neurological deficits including quadriplegia.[1-4]

Hemangioblastomas are rare tumors that can arise sporadically or be associated with VHL disease, only about 25% of them are related to this genetic disease.[1-3] Sporadic tumors tend to be solitary in nature, whereas VHL disease-related lesions are accompanied by multiple lesions along the central nervous system.[1] This patient had four other smaller lesions located in the abdominal and pelvic plexuses. About 50% of hemangioblastomas in patients with VHL disease are located in the spinal cord.[4] Direct spinal cord compression from the hemangioblastoma can

cause neurological deficits including decreased neck mobility secondary to pain or mass.[1-4] Further compression can exacerbate symptoms leading to detrimental outcomes.[1-5] Patients with known cervical injury or pathologies such as tumors are at greater risk for developing devastating neurological consequences during intubation.[1-6] To provide safe and efficient care to these patients, the goal during airway management is to minimize cervical spine motion. Video laryngoscopy, including the Glidescope (Verathon Inc.), decreases cervical spine displacement due to acute angulation and less mouth opening, thus minimizing the risk of potential detrimental outcomes during intubation.[6]

The goal of using IOM for MEPs and SEPs is to maximize tumor resection and minimize neurological morbidity.[5] One typical anesthesia management technique that allows IOM consists of a constant infusion of propofol in a dose of about 100-150 mcg/kg/min and a remifentanil infusion at 0.01-0.2 mcg/kg/min.[5,7] Small adjustments to these infusion rates, within the ranges listed, were made throughout the procedure to maintain a BIS monitor (Medtronic Inc.) value between 40-60.

Whenever a patient with VHL disease undergoes surgical procedures, the possibility of an undiagnosed pheochromocytoma needs to be considered because of the high risk for potential anesthetic complications associated with sympathetic hyperactivity and hypertension.[8] About 30% of patients with VHL disease are diagnosed with pheochromocytoma, especially pediatric patients.[8] Fortunately, the patient described in this report did not experience any signs and symptoms that suggested the presence of an undiagnosed pheochromocytoma. However, emergency drugs for this condition were readily available in case it did occur.

This case study demonstrates the common anesthetic concerns, found in literature, for a VHL disease patient undergoing a spinal cord hemangioblastoma resection. Anesthesia practitioners need to recognize the importance of avoiding any anesthetics that can alter neurologic monitoring and the importance of minimizing neck mobility during airway manipulation. They also need to be prepared for potential intraoperative complications such as hemorrhage or occult pheochromocytoma. Understanding the pathophysiology of this condition and associated risks during the surgical process will aid the anesthesia provider in delivering the safest anesthetic technique and therefore improving patient outcomes.

References

1. Ganeshan D, Menias C, Pickhardt P, Sandrasegaran K, Lubner M, Ramalingan P, Bhalla S. Tumors in Von Hippel-Lindau syndrome: From head to toe-comprehensive state-of-the-art review. *Radiographics*. 2018;38(3): 849-866. doi: 10.1148/rg.2018170156
2. Wang H, Zhang L, Wang H, Nan Y, Ma Q. Spinal hemangioblastoma: surgical procedures, outcomes and review of the literature. *Acta Neurol Belg*. 2021;121(4):973-981. doi: 10.1007/s13760-020-01420-4.
3. Ampie L, Choy W, Khanna R, Smith Z, Dahdaleh N, Parsa A, Bloch O. Role of preoperative embolization for intradural spinal hemangioblastomas. *J Clin Neurosci*. 2016;24(1):83-87. doi:10.1016/j.jocn.2015.09.006
4. Aldape KD, Plate KH, Vortmeyer AO, et al. Hemangioblastoma. In *WHO Classification of Tumours of the Nervous System.* Lyon, IARC Press;2007:184-186.

5. Park J, Park Y. Intraoperative neurophysiological monitoring in spinal cord tumor surgery. Journal of Intraoperative Neurophysiology. 2021; 3(1): 10-15. doi:10.33523/join.2021.3.1.10
6. Krishnamoorthy V, Dagal A, Austin N. Airway management in cervical spine injury. *Int J Crit Illn Inj Sci*. 2014;4(1):50. doi:10.4103/2229-5151.128013
7. Hwang W, Lee J, Park J, Joo J. Dexmedetomidine versus remifentanil in postoperative pain control after SPINAL surgery: A randomized controlled study. *BMC Anesthesiol*. 2015;15(1). doi:10.1186/s12871-015-0004-1
8. Aufforth R, Ramakant P, Sadowski S, Mehta A, Trebska-McGowan K, Nilubol N, Pacak K, Kebebew E. Pheochromocytoma screening initiation and frequency in von Hippel-Lindau Syndrome. *J Clin Endocrinol Metab*. 2015;100(12):4498-4504. doi:10.1210/jc.2015-3045

Mentor: Daniel Bunker, DNAP, MSNA, CRNA

Suspected Malignant Hyperthermia in a Pediatric Patient

Kash Brown, DNAP
University of Kansas Medical Center

Keywords: malignant hyperthermia, ryanodine receptor, sevoflurane, pediatric, MHAUS

Malignant hyperthermia (MH) is an autosomal dominant disorder that impacts those with a mutation on the ryanodine receptor type 1 (RyR1) gene.[1] The incidence of MH in the United States is estimated at 1:100,000 adult anesthetics and about 1:30,000 with children.[2] This genetic mutation causes the abnormal excitation-contraction coupling in muscle that occurs following exposure to volatile anesthetic gases or the depolarizing skeletal muscle relaxant, succinylcholine.[1] If left untreated, the uncontrolled hypermetabolism of skeletal muscle can lead to increasing acidosis and vital organ failure.[3]

Case Report

A 2-year-old female (94cm, 14.1kg, BMI 15.9 kg/m^2, no known allergies) presented for a bilateral tonsillectomy and adenoidectomy for tonsillar hypertrophy with sleep disturbance. The patient's medical history included tonsillar hypertrophy and chronic rhinitis with no previous surgical history.

In the pre-operative bay, vital signs were blood pressure 116/88 mm Hg, heart rate 114/min, SpO_2 98% without supplemental O_2, and axillary temperature of 36.7°C. Preoperatively, the patient was given oral midazolam 7.5 mg. Once in the operating room, standard non-invasive monitors were applied, and the patient was positioned supine. An inhalation mask induction was performed with 8% inspired sevoflurane in O_2 10 L/min. Once an expired sevoflurane concentration of 3.2% was obtained, a 24-gauge peripheral intravenous (IV) catheter was inserted in the patient's left foot. A supplemental dose of propofol 30 mg IV was administered to facilitate endotracheal intubation. An atraumatic intubation was performed with video laryngoscopy, miller 1 blade and placement of a 4.5 mm microlaryngoscopy (cuffed) tube

secured. Auscultation revealed equal, bilateral breath sounds with a positive detection of $EtCO_2$. General anesthesia was maintained with inhaled sevoflurane.

Approximately 8 minutes after initiation of sevoflurane, the patient's $EtCO_2$ level had risen to 49 mm Hg. Five minutes later, levels had reached 65 mm Hg with a peak reading of 73 mm Hg. The patient's heart rate simultaneously trended upward with an average rate of 150/min and a peak rate of 157/min, sinus tachycardia. A skin temperature reading peaked at 36.9°C.

The surgeon was made aware and verbalized understanding of the need to urgently address a possible episode of MH. The triggering agent (sevoflurane) was discontinued, and a continuous propofol infusion was initiated at 100 mcg/kg/min. The hospital's emergency MH kit was obtained, and additional anesthesia providers paged to the operating room. Charcoal filters were placed on both expiratory and inspiratory limbs of the anesthesia machine, and oxygen flows were increased to 10 L/min. The national MHAUS hotline was activated, and dantrolene sodium (Ryanodex preparation, Eagle Pharmaceuticals) initiated. Lastly, a comprehensive collection of blood tests were completed to include an arterial blood gas sample, general chemistry panel, and lactic acid.

As expired concentrations of sevoflurane trended downward to marginal levels (0.1%), the patient's heart rate and $EtCO_2$ levels normalized in conjunction with sevoflurane removal. Rescue medications were withheld following symptom improvement. The surgical procedure was completed. The patient was extubated on O_2, 10 L/min via simple facemask, transported to the post anesthesia care unit (PACU), and recovered without complication. The patient did not require additional time or special monitoring in PACU.

Discussion

Malignant hyperthermia (MH) is a disorder that anesthesia practitioners rarely encounter in practice, but it is imperative for providers to recognize its symptoms and be able to respond appropriately. Malignant hyperthermia is an inherited autosomal dominant disorder that took many years to confirm, with the development of genetic testing only recently becoming available in 2005.[4] Mutations in the ryanodine receptor type 1 (RyR1) gene predisposes a patient to MH. Triggering anesthesia agents include all volatile gases used in general anesthesia (e.g., desflurane, sevoflurane, isoflurane, and halothane), and the depolarizing skeletal muscle relaxant, succinylcholine.[5] When a susceptible person is exposed to a triggering agent, it potentially sets off a chain of events. First, excessive calcium is released from the sarcoplasmic reticulum in skeletal muscle, resulting in a two- to threefold hypermetabolic state, followed by the attempt of energy-dependent body mechanisms to remove excess calcium from the myoplasm.[5-6] When these cellular processes are overworked, there is a drastic increase in oxygen consumption, CO_2 and heat production, and a depletion of ATP stores that leads to the generation of lactic acid (metabolic acidosis).[6] These stressors ultimately lead to sarcolemma destruction and a marked release of potassium, myoglobin and creatine kinase.[6]

In the presence of active malignant hyperthermia, studies have shown the earliest symptoms are an increase in $EtCO_2$, tachycardia, and muscle rigidity, which tends to manifest as masseter spasm after succinylcholine administration.[3] A retrospective chart review of pediatric MH cases

in the North American Hyperthermia Registry (NAMHR) revealed that sinus tachycardia and hypercarbia were among the most common presenting symptoms in children (observed in 73.1% and 68.6%, respectively).[7]

In this case, the first concerning symptoms noted were an abrupt increase in $EtCO_2$ and simultaneous tachycardia. At first, ventilator setting changes were instituted to increase minute ventilation, followed by a ventilator mode change to assess if the cause was related to patient-ventilatory compliance. Despite these interventions, $EtCO_2$ levels continued to rise to levels greater than 70 mm Hg. Manual ventilation was then initiated with an average respiratory rate of 26/min and tidal volume of 114 mL (~8mL/kg), however this failed to improve $EtCO_2$ levels. Meanwhile, the patient's heart rate was sustained at rates as high as 157/min. The presence of a masseter spasm was unable to be assessed due to the patient currently being positioned in a Crowe-Davis mouth gag for the surgical procedure. With the combination of concerning symptoms, which were refractory to standard interventions, the patient was treated as a possible MH crisis.

If MH is suspected, immediate interventions should include discontinuation of the triggering agent, calling for help, alerting the surgeon to conclude promptly, preparation and administration of dantrolene sodium IV bolus of 2.5 mg/kg, hyperventilation with 100% O_2 at flows of at least 10 L/min, and if fever is present, implementation of cooling.[4,6] The first intervention in this case was the discontinuation of sevoflurane and subsequent initiation of a continuous propofol infusion. Simultaneously the surgeon was notified. This was followed by the placement of activated charcoal filters on both limbs of the anesthesia machine with O_2 flows at 10 L/min. Additional anesthesia providers arrived at the bedside to activate the MHAUS hotline and obtained the emergency MH kit and prepared Ryanodex preparation of dantrolene sodium. While alternative diagnoses were ruled out, the patient's vital signs (heart rate and $EtCO_2$ levels) began to normalize with the decreasing levels of expired sevoflurane. With patient status improvement, the current MHAUS hotline recommendations were to withhold dantrolene sodium administration but continue to monitor and obtain laboratory studies per guidance from the live phone conversation with the MHAUS representative.

Other acute crisis recommendations from MHAUS include, obtaining a blood gas (arterial or venous) sample, comprehensive metabolic studies, core temperature and urine output monitoring as warranted by clinical severity of the patient.[2] Primary monitoring concerns are metabolic acidosis and/or electrolyte abnormalities, more specifically, hyperkalemia.[2] In this case, blood samples were not able to be drawn during the acute event and were obtained approximately 10 minutes after vital signs had normalized. Laboratory analysis did not show signs of metabolic acidosis or electrolyte abnormalities.

Following a potential MH crisis, it is important to ensure patient stability prior to transferring to the PACU. Indicators of stability include, declining or normal $EtCO_2$ levels, decreasing or stabilization of heart rate with no ominous dysrhythmias, hyperthermia is improving, and any muscle rigidity has resolved.[2] Prior to extubation of the trachea and transfer to the PACU, the patient was deemed stable, the surgery was successfully completed, and laboratory values were negative for any further MH related concerns. Successful transfer and recovery of the patient was completed, with the family being updated and counseled on potential concerns for future

anesthetic encounters. The patient was monitored in the PACU for an additional hour beyond standard protocol, but with vital signs remaining stable, she was able to discharge home with family. Due to the lack of a definitive MH diagnosis, this patient was not referred to MHAUS, but documentation was made in the patient's chart to avoid triggering agents with subsequent anesthetics. The attending anesthesiologist discussed the issues encountered during the case with the parents, and a printed document with the same information in the electronic health record was given to them.

One consideration for the elevated $EtCO_2$ levels was the possibility of the mouth gag device shifting and occluding the endotracheal tube. This theory is questionable since the patient was able to be ventilated with adequate tidal volumes throughout the entire procedure but is something that would have been valuable to assess during the case. Although the uncertainty of an actual MH crisis remains, the swift response and implementation of interventions helped to reduce the chances of any further patient decline. Real-time intraoperative patient management, MHAUS activation, and utilizing institution MH protocols to optimize the patient's outcome was a valuable learning experience to take away from this case.

References

1. Ellinas H, Albrecht MA. Malignant hyperthermia update. *Anesthesiol Clin*. 2020;38(1):165-181. doi:10.1016/j.anclin.2019.10.010
2. Healthcare Professionals. MHAUS. https://www.mhaus.org/healthcare-professionals/. Accessed September 20, 2021.
3. Mullins MF. Malignant hyperthermia: A review. *J Perianesth Nurs*. 2018;33(5):582-589. doi:10.1016/j.jopan.2017.04.008
4. Gulabani M, Gurha P, Ahmad S, Dass P. Intra-operative post-induction hyperthermia, possibly malignant hyperthermia: Anesthetic implications, challenges and management. *J Anaesthesiol Clin Pharmacol*. 2014;30(4):555-557. doi:10.4103/0970-9185.142860
5. Rüffert H, Bastian B, Bendixen D, et al. Consensus guidelines on perioperative management of malignant hyperthermia suspected or susceptible patients from the European malignant hyperthermia group. *Br J Anaesth*. 2021;126(1):120-130. doi:10.1016/j.bja.2020.09.029
6. Karlet M. Musculoskeletal system anatomy, physiology, pathophysiology, and anesthesia management. In: Nagelhout JJ, Elisha S, eds. *Nurse Anesthesia*. 7th ed. St. Louis: Elsevier; 2022: 841-845.
7. Nelson P, Litman RS. Malignant hyperthermia in children. *Anesth Analg*. 2014;118(2):369-374. doi:10.1213/ane.0b013e3182a8fad0

Mentor: Donna Nyght, DNP, MS, CRNA

Anesthetic Management of an Adult Patient with Fontan Physiology

Gabrielle Robarge, BSN
Yale New Haven Hospital School of Nurse Anesthesia

Keywords: congenital heart disease, adult, hypoplastic left heart syndrome, Fontan

Congenital heart disease (CHD) remains one of the most common genetic birth defects, affecting approximately 8:1,000 births.[1] Adequate medical and surgical interventions are necessary to prevent severe systemic and pulmonary changes that, if left uncorrected, would likely result in death. The adult with hypoplastic left heart syndrome (HLHS) will have undergone several staged corrective procedures.[2] Decreasing the amount of shunting in these patients is essential in maintaining adequate pulmonary and systemic blood flow.[2] Long-term sequelae of CHD may include congestive heart failure, conduction defects, residual intracardiac shunts, valvular dysfunction, and endocarditis.[3] This further highlights this patient population's unique physiology that must be considered when developing a safe anesthetic plan.

Case Report

A 28-year-old female presented to the operating room (OR) for a dilation and curettage (D&C) for spontaneous abortion. The patient's height and weight were 152.4 cm and 100.2 kg. Past medical history included morbid obesity with a body mass index of 43 kg/m^2, complex congenital heart disease with single ventricle physiology, status post-Fontan palliation at 3.5 years of age, and stent placement for left pulmonary artery stenosis. The patient reported the ability to climb a flight of stairs without stopping and a baseline metabolic equivalent (METS) of 4 was assessed. The patient had spontaneous closure of the Fontan fenestration. Daily medications included aspirin 81 mg, cholecalciferol 25 mcg, and a prenatal vitamin. Preoperative cardiac testing included an electrocardiogram (ECG) and transthoracic echocardiogram (TTE). The ECG revealed sinus arrhythmia with a tall R wave in V2, indicating right ventricular hypertrophy. A TTE showed laminar flow in the superior vena cava (SVC) and left pulmonary arterial pathway; mitral regurgitation was ruled out. Early mitral inflow velocity (E/A) and mitral annular early diastolic velocity (E/E') were evaluated to assess diastolic function. The E/A was less than 1, and E/E' was significantly elevated at 19; however, the lateral annulus was normal. There were limited views of the inferior vena cava (IVC) and the inferior Fontan pathway.

Assessment of the airway revealed a Mallampati III classification with thyromental and inter-incisor distances of 4 cm each. A cardiac evaluation revealed a regular heart rate and rhythm with normal S1 and S2 heart sounds, no appreciable murmur, rubs, gallops, or clicks. Pulses were $^{+}2$ bilaterally in the upper and lower extremities. The skin was acyanotic, without clubbing or edema. Lung sounds were clear bilaterally, with diminished sounds at the bases. Intravenous (IV) access was difficult and eventually established with a 22-gauge catheter. A 500 mL fluid bolus of lactated Ringers (LR) was administered in the preoperative area before transport to the OR.

The patient arrived in the OR and was placed supine with the head of bed (HOB) slightly elevated. Standard noninvasive monitors were applied. Initial vital signs were stable with a heart

rate of 64/min, SpO_2 95% on room air, and blood pressure 117/80 mm Hg. The ECG displayed normal sinus rhythm. General anesthesia was induced with midazolam 2 mg, fentanyl 75 mcg, lidocaine 60 mg, propofol 200 mg, and rocuronium 50 mg IV. The patient was intubated using a Macintosh 3 blade with a grade I view and a 7.0 mm endotracheal tube (ETT). Mechanical ventilation was established with synchronized intermittent mandatory ventilation; pressure controlled-volume guaranteed (SIMV PCV-VG) with a tidal volume (VT) of 400 mL and no positive end-expiratory pressure (PEEP). Anesthesia was maintained with sevoflurane 2% inspired concentration in a mixture of O_2 0.5 L/min and air 0.5 L/min. A propofol infusion at 25 mcg/kg/min was administered to reduce the risk of postoperative nausea and vomiting (PONV). The patient was placed in lithotomy position, and the attending surgeon performed the D&C.

Approximately 25 minutes after induction of anesthesia, the blood pressure was recorded as 190/110 mm Hg. Fentanyl 25 mcg and labetalol 5 mg were administered IV with good effect. Repeat blood pressures began trending towards the patient's baseline. At the end of the procedure, the patient was returned to supine position, and neuromuscular blockade was antagonized using sugammadex 4 mg/kg. The patient was breathing spontaneously with 4/4 twitches on train of four (TOF) monitor with sustained tetanus. After following commands, she was extubated to oxygen 2 L/min via a simple face mask. Vital signs remained stable within baseline values. The patient was transferred to the post-anesthesia care unit (PACU) in stable condition and discharged home on the same day after fulfilling discharge criteria.

Discussion

Advancements in medical and surgical management have improved survival for those diagnosed with complex CHD, with approximately 85% living into adulthood.[1] However, the morbidity and mortality for these patients increases when undergoing anesthesia.[3] Coupled with a multifaceted defect, adult congenital heart disease (ACHD) patients may also suffer from long-term complications from previous surgical procedures.[3] These may include heart failure, conduction defects, hypertension, residual shunts, valvular dysfunction, and endocarditis.[3] In this case, the patient required stent placement for left pulmonary artery stenosis. This was critical as maintenance of pulmonary blood flow is essential to maintain cardiac output (CO).

Hypoplastic left heart syndrome (HLHS) is a complex CHD affecting approximately 1:5,000 newborns.[2] Key pathology of HLHS includes hypoplasia of the left ventricle, mitral valve, aortic arch and aortic valve.[2] Before surgical intervention, medical management must be initiated at birth. The ratio of blood supplied to the pulmonary and systemic circulations depends on the resistance between these two vascular beds and the patency of the ductus arteriosus.[2] HLHS is a ductal-dependent lesion, and IV prostaglandin therapy is required to maintain ductal patency to ensure coronary artery and systemic perfusion from the right ventricle to the aorta.[2] Surgical management includes a series of staged procedures: Norwood, Glenn, and Fontan.

In 1971, Francis Fontan described the procedure after two patients with tricuspid atresia survived the operation.[4] Today, the Fontan has become the most widely applied surgical procedure for several CHDs.[4] The procedure includes disconnecting the IVC from the heart and attaching the IVC to the pulmonary artery with a conduit.[4] This results in deoxygenated blood from the systemic circulation traveling to the lungs without passing through the heart.

The Fontan circulation is unique because CO is dependent on pulmonary blood flow (PBF).[4] Typically, a fenestration is made between the conduit and right atrium.[4] Fenestration assists in lowering systemic venous pressures and acts as a "fail-safe" in the event of acute increases in pulmonary vascular resistance (PVR).[4] Spontaneous closure of the fenestration does occur and may not drastically change patient status. It was noted that this patient did have spontaneous closure of the Fontan fenestration, and considerations to avoid increases in PVR remain essential. Increases in PVR significantly reduce CO due to inadequate pressure gradients between the systemic and pulmonary vasculature (Figure 1).

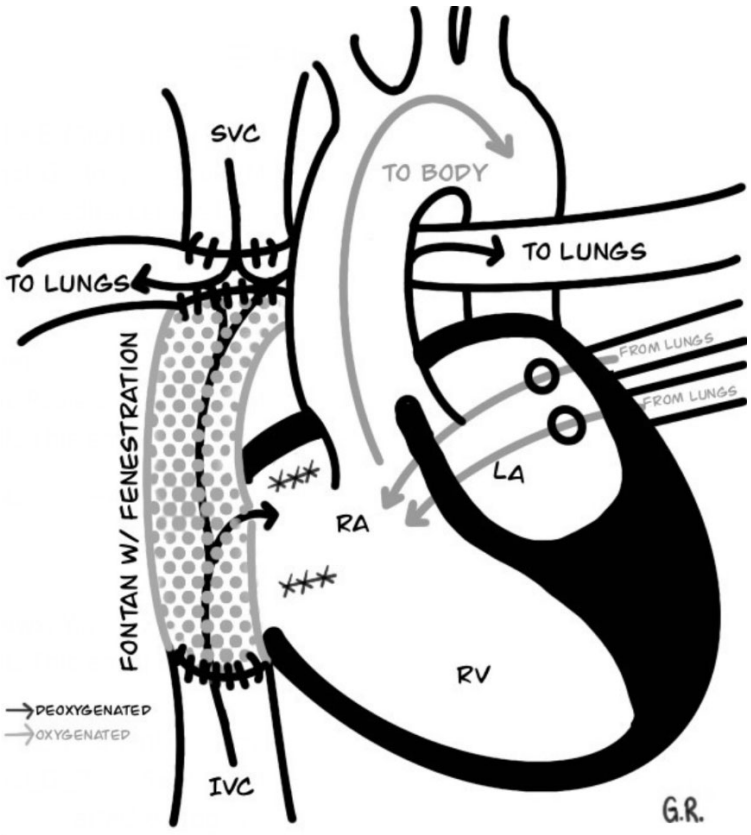

Figure 1. Fontan Physiology. Rendered by author.

The transpulmonary gradient is the driving force for blood flow through the pulmonary circulation.[5] This gradient occurs as a result of the central venous pressure minus the pulmonary venous atrial pressure.[5] Systemic venous pressures range from 10-15 mm Hg and pulmonary venous atrial pressures from 5-10 mm Hg, creating a transpulmonary gradient of 5-10 mm Hg.[5] In summary, maintenance of CO relies on an adequate pressure gradient ensuring sufficient flow to the pulmonary circulation.

Patients with Fontan circulation have unique anesthetic considerations. Of importance, general anesthesia can cause dramatic vasodilation, resulting in a decrease in pulmonary blood flow and CO.[3] Adequate preload is essential in maintaining venous capacitance, and prolonged periods of nothing by mouth (NPO) status should be avoided.[3] Administration of a bolus of isotonic IV fluids before induction of general anesthesia may assist in balancing hypotension and CO.[3] A

500 mL fluid bolus of lactated Ringers (LR) was administered preoperatively to this patient. Coupled with fluid management, avoiding acute increases in PVR is essential in maintaining CO. Hypercarbia, acidosis, and light anesthesia all contribute to increases in PVR.[3] Sudden desaturation may suggest excessive flow across the fenestration and may be the first sign of an acute increase in PVR. Mechanical ventilation poses another challenge for the Fontan patient. Significant increases in intrathoracic pressure alter the transpulmonary gradient, decreasing PBF and CO.[3,6] Options for mechanical ventilation include low-peak airway pressures, normocarbia, low PEEP, and VT of 6-8 mL/kg ideal body weight (IBW).[3,6] This patient was maintained on mechanical ventilation with SIMV PCV-VG and tidal volumes (VT) of 400 mL; PEEP was avoided to decrease risk of significant increases in intrathoracic pressures.

Anesthetic care should include the following considerations: avoidance or careful administration of drugs that have negative inotropic effects and those that increase PVR to preserve PBF and CO.[6] Induction agents that depress myocardial contractility may be detrimental in the Fontan patient. The transient systemic vasodilation caused by propofol is tolerated well if normovolemia is maintained.[6] A combination of volatile agent and propofol infusion was selected for this patient. The addition of a propofol drip at 25 mcg/kg/min was chosen to reduce the risk of PONV. Maintaining normal sinus rhythm, atrial emptying, and ventricular filling is critical. High concentrations of volatile anesthetics can increase the risk of arrhythmia and should be avoided.[6] Sinus rhythm was achieved throughout this case, with no evidence of decreased CO. A cardiostable anesthetic can be achieved by utilizing a combination of a short-acting opioid infusion such as remifentanil with a low inspired concentration inhaled agent.[6] In this case, the patient experienced transient hypertension which was successfully managed with pharmacologic agents. Factors that may have influenced this change in blood pressure may be explained by presence of increased preload from the lithotomy position and surgical stimulation.

Care of the adult patient with CHD undergoing anesthesia for noncardiac surgery is a growing population. It is essential for anesthesia providers to be familiar with perioperative concerns in order to optimize patient outcomes. These patients have complex cardiac physiology, and knowledge of the underlying defect, effects of palliative and corrective surgeries, and impact of anesthetic agents and procedures is crucial to the safe and comprehensive care of these patients.

References

1. Mutluer FO, Celiker A. General concepts in adult congenital heart disease. *Balkan Med J.* 2018;35(1):18-29. doi:10.4274/balkanmedj.2017.0910
2. Gobergs R, Salputra E, Lubaua I. Hypoplastic left heart syndrome: a review. *Acta Med Lit.* 2016;23(2):86-98. doi:10.6001/actamedica.v23i2.3325
3. King M, Belani K. Managing the adult patient with congenital heart disease. *Anesthesiology Clin.* 2020;38(3):643-662. Accessed June 1, 2022. https://doi.org/10.1016/j.anclin.2020.05.008
4. McNamara JR, McMahon A, Griffin M. Perioperative management of the Fontan patient for cardiac and noncardiac surgery. *J Cardiothorac Vasc Anesth*. 2021;36(1):275-285. https://doi.org/10.1053/j.jvca.2021.04.021
5. Yuki K, Casta A, Uezono S. Anesthetic management of noncardiac surgery for patients with single ventricle physiology. *J Anesth*. 2011;25(2):247-256. doi:10.1007/s00540-010-1081-4

6. Nayak S, Booker PD. The Fontan circulation. *BJA Educ.* 2008;8(1):26-30. https://doi.org/10.1093/bjaceaccp/mkm047

Mentor: Marianne S. Cosgrove, PhD, DNAP, CRNA, APRN, FAANA

Neurolytic Celiac Plexus Block and Opioid Needs in Pancreatic Cancer

Erin Grotts, DNAP, BSN
Bryan College of Health Sciences

Keywords: celiac plexus block, neurolytic celiac plexus block, pancreatic cancer treatment, pancreatic cancer, opioid use in pancreatic cancer.

Introduction

According to the American Cancer Society, about 60,430 people in the U.S. will be diagnosed with pancreatic cancer in 2021, and about 48,220 people will die from the condition.[1] Pancreatic cancer can be painful and is often diagnosed in late-stage form, leading to a low survival rate and low quality of life. These patients are often referred to palliative care for further treatment. The American Academy of Hospice and Palliative Medicine strives to ensure patients have the best care possible to maintain a high quality of life.[2] They stress that this standard of care is not possible without more providers who are willing and able to provide end-of-life care.

The pain and side effects from prescribed opioid use can hinder the small amount of time patients may have before death. This pain, which originates in the pancreas and is transmitted by splanchnic nerves though the celiac plexus, can be resistant to opioid analgesia.[3]

In adequate pain relief for cancer patients and those undergoing palliative care, the World Health Organization released a three-step process to manage pain. During the onset, nonsteroidal anti-inflammatory drugs (NSAIDs) are the first-line treatment.[4] If NSAIDs do not provide relief, mild opioids are the second line. If pain persists, a stronger opioid is then used as the third line treatment. It comes as no surprise that the literature identifies “pain” as the number one concern for these patients. Patients have overwhelmingly expressed that their pain is uncontrolled, despite multimodal treatments and opioid use. As the disease progresses and pain intensifies, doses of opioids are increased, leaving patients with many adverse effects like constipation, somnolence, confusion and nausea.[5,6]

One intervention, the neurolytic celiac plexus block, has demonstrated potential to reduce pain in patients. Reduction in pain can lead to less opioid use, thus decreasing drug side effects like constipation and nausea, and improving overall quality of life. [3,6-8] Reduction of opioid use has been reported in about 70-90% of patients who receive the block, and complications occur in less than 2% of patients.[9]

Methods

A population, intervention, comparison and outcome (PICO) question was developed to provide a framework for the research and literature review process: "Within the pancreatic cancer population, does a neurolytic celiac plexus block, compared to no block, decrease opioid use and improve pain scores within a 6-month period?"

This literature review utilized the following online databases: CINAHL Complete, Cochrane Collection, Medline Complete, and MedlinePlus. Keywords for the search included: *celiac plexus block, neurolytic celiac plexus block, pancreatic cancer treatment, pancreatic cancer, opioid use in pancreatic cancer.* The search yielded one meta-analysis, one randomized control trial, two retrospective studies, one prospective non-randomized study and one prospective cohort study. Studies included in this analysis were from years 2005 - 2019. This search was narrowed to 6 articles with a total sample size of 1,142 subjects. All studies evaluated the effectiveness of a neurolytic celiac plexus block on pain scores and opioid use in pancreatic cancer patients.

Literature Analysis

The literature was reviewed to determine the efficacy of the neurolytic celiac plexus block and its impact on pain scores and opioid use. In this analysis, "treatment group" will refer to the subjects receiving a neurolytic celiac plexus block. The term "block" will refer to the neurolytic celiac plexus block. The term "control group" will refer to the subjects receiving solely pharmacological treatment with non-steroidal anti-inflammatory drugs (NSAIDs) and opioids. The Visual Analog Scale (VAS) is the outcome measurement used and will be referred to as "pain score." All studies used a 0-10 scale, except Amr and Makharita who leveraged a 0-100 scale.[1]

Pain Scores

All six studies in this analysis demonstrated that a neurolytic celiac plexus block reduced pain scores significantly for the time periods under investigation. Results of a meta-analysis of 8 randomized control trials (RCTs), as well a prospective cohort study, demonstrated that patients who receive a neurolytic celiac plexus block reported lower pain scores compared to subjects in control groups. Both the meta-analysis and the prospective cohort study showed statistically significant reductions in scores at 30 days post block administration.[3,6] Zhong et al concluded that although there was a significant decrease in the mean pain scores within each RCT, the combined treatment group score compared to the combined control group was not statistically significant at 8 weeks and beyond.[6]

Two retrospective studies included in this literature analysis also revealed statistically significant favorable outcomes regarding subjects who were treated with neurolytic celiac plexus blocks for unmanageable pancreatic cancer pain. In a study by Molnár et al, the average pain rating in subjects prior to the block was an 8 on the visual analogue scale (VAS).[10] Thirty-five days post block, the average rating was reduced to 3. Rahman demonstrated similar findings, noting that the average baseline rating prior to the block was 8.48.[5] The rating was reduced to 5.73 after one

week, then increased slightly to 6.34 by month three. The researchers concluded that the return to higher pain scores in months four to six were due to disease progression and anticipated neurolytic agent duration. Despite this increase, however, pain scores and opioid usage never increased to, or surpassed, pre-intervention levels. A second long-term study by Amr and Makharita also supported the conclusion that pain and opioid use initially decreased, but increased around six months.[11] There was a decrease in pain scores at 2, 3, 4, 5, 6, 9, and 12 months after the procedure when compared to baseline. Although pain scores did slightly elevate after 6 months, they did not return to baseline levels.

Opioid Use

All six studies in this analysis concluded that a neurolytic celiac plexus block reduced opioid usage. It should be noted, however, that despite the administration of a block, no subjects were able to completely cease opioid or non-opioid pharmacologic use post block. Zhong et al[6] and Jain et al[3] both noted a significant decrease in opioid use in their treatment groups at 30 days, compared to the control groups. Molnár et al demonstrated a significant decrease in the number of subjects using oral Oxycodone and Fentanyl patches within 35 days. Initially, all subjects were using a Fentanyl patch for pain control, and at 35 days, zero subjects required use of a patch.[10]

Neurolytic Block Methodology Impact on Pain and Opioid Usage

The literature review demonstrated variability in the method of block placement and solutions administered across studies. Table 1 outlines the various injection solutions. Generally, there did not appear to be significant differences in outcomes across studies. However, some key differences in administration techniques did impact study outcomes.

Two studies focused on how block administration method impacted pain scores. Amr and Makharita[11] evaluated whether timing of the block made a difference in subject outcomes. Group I received the block early, followed by pharmacology treatment according to the WHO ladder as needed. Group II were first given analgesics according to the WHO ladder, and then received a block when pain scores reached a defined threshold. Although both groups saw a significant decrease in pain scores and opioid use, the method for Group II was more effective. Therefore, utilization of neurolytic celiac plexus block appears to be more effective later in the disease progression.

Yang et al utilized a multi-step administration process. Subjects received an initial block of 20 mL of 100% alcohol.[8] A pain catheter was also placed to allow for patient-controlled analgesia (PCA) for one week. The PCA bag contained 60 mL 2% lidocaine, 10 mg methylprednisolone and 190 mL of normal saline. The subjects were asked to remain prone for the first six hours after this initial encounter. They returned one week later for a block that was identical to the initial one, and the pain catheter was then removed. The study found a significant decrease in both pain scores and opioid use over a period of 6 months. When comparing the pain scores of this study to others where the baseline pain score was similar, it was noted that the scores were significantly lower.[8] For example, at three months, the average pain score was 3.2 ± 1.0, whereas the scores in Rahman[5] averaged 6.34.[8]

Table: Findings related to neurolytic celiac plexus block and impact on pain scores and opioid use.

Level of Evidence*	Block Group	Control Group	Pain Scores/Timing	Opioid Dosages/Timing	Significant Data
Zhong et al., 2013 N = 499 Meta-Analysis *Level 1.a	Ethanol in concentrations of 30%, 75%, 80%, 100%.	Opioid/ NSAID treatment	4 weeks: Mean difference between groups = -0.382 (P = 0.005) 8 weeks: Mean difference between groups = -0.265 (P = 0.223)	*Morphine mg/day mean difference between groups:* 4 Weeks: -49.77 (P = 0.005) Day prior to death: -48.29 (P< 0.001p	Combined block groups vs control groups had: ↓ pain score at 4 weeks. But did not maintain significance at 8 weeks. ↓ medication use.
Jain et al., 2005 N = 100 Prospective cohort study *Level 3.c	2% Lidocaine (unspecified amount) 50% alcohol 20 mL	Opioid/ NSAID treatment	Block Group: Pre-intervention 6.5 7 days: 2.1 (P = 0.030) 30 days: 2.2 (P = 0.005) Opioid/NSAID Group: Pre-intervention 6.2 7 days: 2.5 (P = 0.030) 30 days: 3.0 (P= 0.005)	*Morphine consumption:* Block Group Baseline: 58 1 month: 30 (P = 0.00) Opioids/NSAIDs Group Before block: 90 1 month post: 109 (P = 0.00)	↓ Pain scores for both groups at 7 days and 30 days. ↓ Morphine consumption in Block group at 30 days. ↑ Morphine consumption in the Opioids/NSAIDs groups at 30 days.
Molnár et al., 2019 N = 16 Prospective nonrandomized study *Level 3.e	1% Lidocaine 5 mL 70% ethyl 20 mL	NA	Pre-Intervention Avg: 8 35 days: 3 (P=0.002)	*Number of patients using each:* Pre-Intervention: Tramadol (20–80 mg) 0 Oxycodone (180–360 mg) 4 Fentanyl patch (720–1,000 mg) 12 35 Days: Tramadol 13 Oxycodone 3 Fentanyl patch 0	↓ Pain score, opioid use during 35 days.
Amr & Makharita, 2013 N = 60 Randomized control trial *Level 1.c	1% lidocaine 5 mL 70% alcohol 40 mL *(Block first then opioids as needed)*	1% lidocaine 5 mL 70% alcohol 40 mL *(Block administered once pain score reached a certain threshold.)*	Pre-intervention 84.4 ± 7.30 2 mo P<0.0001 Group 1: 34.54 ± 4.08 Group 2: 25.95 ± 4.18 3mo P<0.0001 Group 1: 47.52 ± 6.14 Group 2: 26.65 ± 4.0	*Opioid consumption:* 2mo P<0.0001 Group 1: 135.0 ± 39.86 Group 2: 97.5 ± 49.93 3mo P<0.0001 Group 1: 165.62 ± 39.94 Group 2: 100.0 ± 50.0 4mo P<0.0001	↓ Morphine consumption and pain scores in group II vs group I in month two and beyond.

			4mo P<0.0001 Group 1: 48.1 ± 5.9 Group 2: 26.8 ± 3.3	Group 1: 350.3 ± 70.71 Group 2: 140.62 ± 49.05	
Rahman, 2018 N = 455 Retrospective observational study *Level 3.e	0.5% bupivacaine with 1: 200,000 epinephrine test dose 1 mL 6% aqueous phenol 3-5 mL	NA	Pre-intervention: 8.48 P< 0.001 1 wk: 5.73 1 month: 6.52 2 months: 6.63 3 months: 6.34 4 months: 5.62 5 months: 4.81	*Morphine equivalents per 24 hours :* Baseline Mean: 125.27 mg/24hrs (P< 0.001) 1 month: 70 mg 2 months: 62 mg 3 months: 63 mg 4 months: 64 mg 5 months: 78 mg	Post block = ↓ pain scores and daily opioid consumption. A return to higher pain scores occurred in months 4-6 due to disease progression and anticipated neurolytic agent duration. Despite this increase, neither pain score nor opioid use returned to pre-procedure levels.
Yang et al., 2012 N = 12 Retrospective clinical data analysis *Level 3.e	20 mL of 100% alcohol (block) PCA catheter placed. Bag = 60 mL 2% lidocaine; 10mg methylprednisolone; 190 mL of normal saline. 5 mL loading dose 5 mL/h basal dose 5 mL bolus (30 min lockout) Prone position for 6 hours post block. 1 wk later: Identical block repeated. Catheter removed.	NA	Pre-intervention: 8.7 ± 1.0 1 day: 1.8 ± 1.1 P<0.001 1 wk: 1.9 ± 1.0 P <0.001 1 mo: 2.3 ± 1.2 P <0.001 3 mo: 3.2 ± 1.0 P<0.05 6 mo: 3.3 ± 1.5 P<0.05	*Morphine mg/day:* Pre-intervention: 155 ± 56 1 day: 0 P<0.001 1 wk: 0 P<0.00 1 mo: 30 ± 39 P <0.05 3 mo: 42 ± 44 P <0.05 6 mo: 40 ± 35 P<0.05	↓ Pain scores during 6 month duration. ↓ Morphine use during 6 month duration.
*Joanna Briggs Institute Levels of Evidence					

Conclusions

Pain in the pancreatic cancer patient is complex and difficult to treat. The literature analyzed in this review revealed that a neurolytic celiac plexus block is effective at decreasing both pain scores and opioid use in these patients. The literature demonstrated that although the block is effective, its ability to alleviate pain may wane after four months, which may be attributable to a combination of expected block duration and disease progression. However, in studies where data was collected for at least five months, it was noted that pain scores did not increase to, or surpass, pre-block baseline levels in that timeframe.[5,11] Opioid use in these longer-term studies also slightly increased over time, but also never returned to baseline dose requirements. As

discussed, despite the administration of a block, all subjects still required pharmacological treatment with opioids.

Although there was variability in the method of block administration across studies reviewed, the results universally indicate that the celiac plexus block is effective in decreasing pain scores and opioid use. Two studies that specifically focused on the administration process demonstrated that timing and method can also impact block effectiveness.[5,11] Future research should explore the implications of administration method, timing and neurolytic injection solution.

In clinical application, this analysis also illustrates the need for focused efforts to maintain pain control for the palliative care population. The American Academy of Hospice and Palliative Medicine[2] recently developed legislation to create more opportunities for interdisciplinary education and training in palliative care. There is a lack of literature exploring the role of anesthesia practitioners in management and administration of palliative care pain interventions. The lack of anesthesia provider involvement within end-of-life care communities and facilities should also be explored. Lukowski et al concluded that current barriers include: patient distance to pain providers and clinics, care coordinator lack of understanding of these services, and cost.[12]

When questioning the role of anesthesia practitioners in providing palliative care, it is key to look at the similarities between surgical patients and those in palliative care. Although the sources may be different, both populations experience pain, nausea, anxiety, and opioid side effects which certified registered nurse anesthetists are well versed in managing and treating.

References

1. Key statistics for pancreatic cancer. https://www.cancer.org/cancer/pancreatic-cancer.html Published January 21, 2021. Accessed December 5, 2021.
2. Expanding the hospice and palliative care workforce. http://aahpm.org/issues/workforce. Accessed December 5, 2021.
3. Jain PN, Shrikhande SV, Myatra SN, Sareen R. Neurolytic celiac plexus block: a better alternative to opioid treatment in upper abdominal malignancies: an indian experience. *J Pain Palliat Care Pharmacother*. 2005;19(3):15-20. doi:10.1080/J354v19n03_04
4. NCDs | WHO Guidelines for the pharmacological and radiotherapeutic management of cancer pain in adults and adolescents. WHO. https://www.who.int/publications/i/item/9789241550390. Accessed December 5, 2021.
5. Rahman A. Low volume neurolytic retrocrural celiac plexus block for visceral cancer pain: retrospective review of 507 patients with severe malignancy related pain due to primary abdominal cancer or metastatic disease. *Pain Phys*. 2018;1(21;1):497-504. doi:10.36076/ppj.2018.5.497
6. Zhong W, Yu Z, Zeng J-X, et al. Celiac plexus block for treatment of pain associated with pancreatic cancer: a meta-analysis. *Pain Pract*. 2014;14(1):43-51. doi:10.1111/papr.12083
7. Staats PS, Hekmat H, Sauter P, Lillemoe K. The effects of alcohol celiac plexus block, pain, and mood on longevity in patients with unresectable pancreatic cancer: a double-blind, randomized, placebo-controlled study. *Pain Med*. 2001;2(1):28-34. doi:10.1046/j.1526-4637.2001.002001028.x

8. Yang F-R, Wu B-S, Lai G-H, et al. Assessment of consecutive neurolytic celiac plexus block (Ncpb) technique outcomes in the management of refractory visceral cancer pain. *Pain Med*. 2012;13(4):518-521. doi:10.1111/j.1526-4637.2012.01332.x
9. Nitschke A, Ray C. Percutaneous neurolytic celiac plexus block. *Semin Intervent Radiol*. 2013;30(03):318-321. doi:10.1055/s-0033-1353485
10. Molnár I, Hegyi G, Zsom L, et al. Celiac plexus block increases quality of life in patients with pancreatic cancer. *J Pain Res*. 2019;(12):307-315. doi: 10.2147/JPR.S186659
11. Amr YM, Makharita MY. Comparative study between 2 protocols for management of severe pain in patients with unresectable pancreatic cancer: one-year follow-up. *Clin J Pain.* 2013;29(9):807-813. doi:10.1097/AJP.0b013e3182757673
12. Lukowski LI, Murry T, Hall J, Algozzine RF. Anesthesia practitioner involvement, invasive treatments, and need in hospice pain management: A survey of patient care coordinators. *Am J Hosp Palliat Care*. 2001;18(2):113-123. doi:10.1177/104990910101800210

Mentor: Holly A. Chandler, EdD, CRNA

Sphenopalatine Ganglion Block versus Autologous Epidural Blood Patch

Shreja Tahiliani, BSN, Kathleen Peters, BSN
Midwestern University

Keywords: postdural puncture headache, autologous epidural blood patch, sphenopalatine ganglion block.

Introduction

One of the most common methods of providing analgesia in a parturient is the insertion of an epidural catheter. Epidural analgesia is a popular request from many obstetric patients due to its effectiveness and safety.[1] The risks accompanying neuraxial techniques in the parturient are minimal but can significantly affect new mothers. Postdural puncture headache (PDPH) is the most serious reported complication following epidural catheter placement, occurring in approximately 0.7% to 1.5% of the obstetric population.[2] PDPH symptoms include a postural frontal headache relieved by supine position, nausea, nuchal rigidity, tinnitus, and photophobia.[1] Originally thought to be an acute problem, PDPH has also been associated with developing a chronic headache syndrome in approximately one-third of patients following accident dural puncture.[8] Postdural puncture headaches have associated side effects consisting of an increase in hospital length of stay following delivery, subsequent emergency room visits, impaired maternal healing, inability to care for the newborn, and decreased activities of daily living.[4]

Historically, the administration of an autologous epidural blood patch (AEBP) has widely been regarded as the gold standard for PDPH requiring treatment. However, this technique is invasive, requiring an additional epidural placement and venipuncture, and carries the added risk of infection. Additionally, AEBP only demonstrates a 75% success rate.[3] Whereas, the sphenopalatine ganglion block (SPGB) is a newer, less invasive technique where local anesthetic is applied in a transnasal fashion to treat a variety of headache syndromes and has a quicker

onset relief for PDPH than AEBP.[4] The few existing risks of SPGB are similar to those of a nasal swab and include bleeding due to the highly vascular nature of the nasal passages. This risk is theoretically increased with the newly postpartum population due to increased blood volume and venous engorgement.[1]

Methodology

Utilizing the PICO (population, intervention, comparison, outcome) guidelines for a review of evidence-based practice guidelines, the proposed question is: In postpartum patients diagnosed with a postdural puncture headache, does transnasal sphenopalatine ganglion block compared to autologous epidural blood patch improve symptoms during the postpartum period?
A university library database was utilized to access multiple EBSCOhost sponsored databases, including Cumulative Index to Nursing and Allied Health Literature Plus, Academic Search Complete, MEDLINE Complete, and Cochrane Central Register of Controlled Trials. This search yielded 170 results. Initially, terminology such as "anesthesia" and "epidural blood patch" was paired with "sphenopalatine ganglion block." This search yielded literature that supported the use of SPGB over AEBP due to its effectiveness and safety. Subsequently, the search terms "post-dural puncture headache" and "parturients" were paired with "epidural blood patch." This search identified the efficacy and complications associated with AEBP to address PDPH in postpartum patients. Inclusion criteria were isolated to literature focusing on parturients' who developed PDPH from neuraxial anesthesia. Results were narrowed to include works from scholarly journals within the last five years.

Literature Analysis

This literature analysis determined current published research focusing on the effectiveness of sphenopalatine ganglion block (SPGB) in addressing postdural puncture headaches (PDPH). Performing an autologous epidural blood patch (AEBP) was historically regarded as the gold standard; however, the risks may exceed those presented by SPGB, especially in postpartum patients.[3] Seemingly, the risks of SPGB are minimal, including mucosal irritation and nasal bleeding.[4]

Implications for autologous epidural blood patch. Delgado et al.[6] identified that 71% of parturients utilized neuraxial labor analgesia for vaginal delivery and studied the frequency of AEBP placement revealing that 68% of PDPH cases received a blood patch. AEBP was the most commonly pursued treatment for patients reporting a PDPH who had a history of epidural placement and vaginal delivery.[6] AEBP placement was repeated up to three times, with a median of 1 day between delivery and the first AEBP. Urits et al.[7] suggested an association between dural puncture, epidural blood patch, and consequent lower back pain. Barad et al.[8] highlights that PDPH precipitates new or worsening chronic headaches after childbirth in up to 33% of patients who have experienced unintended dural punctures.

Implications for Transnasal Spenopalentine Ganglion Block. Youssef et al.[3] identified pregnant and postpartum women as the highest risk population for developing PDPH due to gender, age, and greater exposure to neuraxial anesthesia. Both Greater Occipital Nerve Block

(GONB) and SPGB were compared to evaluate treatment for PDPH. A loss of CSF stimulates the sphenopalatine ganglion, resulting in cerebral vasodilation due to the release of acetylcholine, nitric oxide, and vasoactive intestinal peptide within the dural vasculature. Direct block of the sphenopalatine ganglion decreases the signaling, therefore relieving the PDPH.[3] Both GONB and SPGB were found to be equally effective for relieving symptoms of PDPH, and both techniques are safe, simple, and pose fewer risks to the patient than AEBP. Abelhadeem [9] concluded that transnasal SPGB should be considered a first treatment modality for PDPH due to its rapid onset and effectiveness. Analysis of the collected data demonstrated that the pain relief provided by SPGB was maintained for at least 24 hours immediately after the block was administered. Pre-procedural pulsatility index and mean flow velocity of cerebral perfusion were monitored via transcranial doppler (TCD) to assess the quality of the SPGB. TCD has been previously utilized to diagnose PDPH and identify cerebral vasodilation. Return of physiologic cerebral vasoconstriction was assessed via TCD on the SPGB group and correlated with the relief of symptoms.

Albaqami et al.[5] demonstrated that transnasal SPGB provided significant relief of headaches that developed after obstetric neuraxial block. Several local anesthetics were evaluated, and administration of 2% lidocaine proved to be the most beneficial. The SPGB is also identified as a cost-effective, portable treatment solution that can be done as an outpatient procedure. The use of a second SPGB was also observed to be safe and effective should the patient's first SPGB yield inadequate results. Takmaz et al.[2] evaluated the effectiveness of the SPGB at predetermined intervals utilizing the Visual Analog Scale (VAS). All of the patients had a post-procedure VAS score of <3, indicating adequate analgesia. Transnasal SPGB should be considered before AEBP due to the straightforward nature of the procedure and a low complication rate.[2]

Implications comparing autologous epidural blood patch and transnasal sphenopalatine ganglion block. According to Urtis et al.[7], patients who have been treated for PDPH with AEBP experience a higher incidence of chronic lower back pain. Barad et al.[8] found that 30% of patients experienced chronic debilitating migraines for up to a year post procedure. Cohen et al. [4] demonstrated that patients treated with SPGB following PDPH did not return to the emergency department to be evaluated for persistent headache, as opposed to 23.1% of patients whose PDPH was treated with AEBP, which is considered statistically significant as evidenced by a *p*-value of 0.03.

Limitations. Limitations include the historical use of AEBP, which is widely regarded as the gold standard treatment for PDPH, practitioners' subsequent hesitance to adopt a novel treatment modality, providers' unawareness of the SPGB procedure, and the lack of research surrounding PDPH interventions.

Practice Recommendations. The transnasal approach to the sphenopalatine ganglion block (SPGB) is a newer, less invasive technique that has provided quicker onset relief than epidural blood patch.[4] Research has demonstrated that patients in the SGPB group also reported no posttreatment complications, compared to 10% of the patients in the AEBP group who reported backache, vasovagal reactions, and temporary hearing loss.[4] The evidence demonstrates that SPGB is safer, less expensive, and provides quicker onset of headache relief in a vulnerable population, which may decrease the length of hospital stay and prevent recurring Emergency

Room visits. The low-risk nature of the SPGB procedure warrants consideration as the initial solution in the PDPH treatment algorithm. The SPGB is an inexpensive intervention that obstetric anesthesia practitioners can implement without requiring specific equipment and likely has less side effects as well as increased tolerance by patients.

Table. Summary of Literature comparing Autologous Epidural Blood Patch (AEBP) and Sphenopalatine Ganglion Block (SPGB)

Author, Year	**Level of Evidence**	**Population**	**Purpose**	**Findings**	**Limitation**
Abdelahleem, 2021[9]	Level II Prospective triple blinded controlled clinical trial	Patients who received a spinal or epidural and developed a PDPH within 5 days of a dural puncture, ages 18-60 of both genders	To prove the ability of SPGB in the management of PDPH and the use of transcranial doppler as an objective tool to monitor SPGB success	SPGB pain relief proved effective immediately after the block and was maintained for 24 hours. The transcranial doppler was a successful objective tool in monitoring SPGB.	SPGB performed without standardized equipment
Albaqami et al., 2022[5]	Level I systematic review	Obstetric patients with PDPH who were treated with SPGB	A systematic review was conducted to assess the efficacy of SPGB as a noninvasive treatment of PDPH.	Results concluded that 41 of 68 patients had effective management with significant relief of headache with no further interventions needed. In addition, a total of 27 of 68 patients had initially effective management with the SPGB but needed further interventions thereafter.	Small sample size, case reports with bias results
Barad et al., 2021[8]	Level I Literature review	Women who had unintended dural puncture with a large bore needle used for epidural catheter placement at delivery.	Aimed to bridge the knowledge gap for the neurologist as to the mounting body of obstetric anesthesia literature on the development of chronic headache after PDPH.	Obstetric patients who had unintended dural puncture with a large bore needle used for epidural catheter placement, 30% had chronic debilitating headache in the months following procedure and may persist for up to a year or longer.	Identifies problems with neuraxial anesthesia but does not offer alternatives
Cohen et al., 2019[4]	Level II Retrospective chart review	Obstetric patients who received UDP during labor epidural patient and experienced a PDPH	This study aimed to compare the effectiveness of treatment of PDPH with an AEBP versus with a SPGB.	A greater number of patients experienced a quicker onset of headache relief, without any new complications, from treatment with SPGB versus EBP.	Small sample size, no info on anesthetic practitioner who placed the epidural/ spinal
Delgado et al., 2019[6]	Level II Comparative/	Postpartum patients that	The incidence of PDPH and the	Patients undergoing cesarean delivery without neuraxial	No follow up data on

	observational study	developed PDPH as per labor and delivery insurance claims from Truven MarketScan Commercial Claims and Encounters	frequency of EBP utilization is heavily based on reports from academic medical centers. This study focused on private insurance databases to provide estimates of neuraxial labor epidural use and PDPH and AEBP incidence in the United States.	labor epidural had a higher incidence of PDPH than women with neuraxial labor epidural and vaginal delivery. This is presumably due to intentional dural puncture. AEBP was a commonly used remedy for the treatment of PDPH and was more commonly pursued in patients with a history of neuraxial labor epidural.	postpartum headaches, ER visits, back pain, etc.
Takmaz et al., 2021[2]	Level II Retrospective study	26 non-obstetric patients who were diagnosed with PDPH and unresponsive to conservative therapy or unable to continue it because of side effects.	Aimed to investigate the efficacy and safety of transnasal SPGB for treatment of PDPH in non- obstetric patients.	SPGB was successfully performed in all patients. Analgesia was achieved in all patients within 48 hours and no patient required treatment with AEBP.	These were not obstetric patients
Urits et al., 2020[7]	Level III Case control pilot study	Postpartum patients who experience PDPH following labor analgesia and were treated with an AEBP	Examine the association of chronic lower back pain and AEBP	PDPH treated with AEBP is associated with higher incidence of subsequent lower back pain in parturients	Small sample sizes, recall bias, statistical significance no achieved
Youssef et al., 2021[3]	Level II Randomized comparative single-blind trial	Patients who received spinal anesthesia for elective cesarean section, and then developed PDPH during hospitalization or within 5 days after dural puncture	To investigate the efficacy of SPGB and greater occipital nerve block to relieve PDPH and its associated symptoms	The supine and sitting headaches significantly decreased at 30 minutes after blocks were administered.	A limitation to this study was the small sample size.

Abbreviations: PDPH, postdural puncture headache; SPBG, sphenopalatine ganglion block; AEBP, autologous epidural blood patch; UDP, unintentional dural puncture

Conclusion

PDPH and associated symptoms increase hospital length of stay following delivery, are associated with subsequent emergency room visits, and interfere with maternal healing, care of the newborn, and activities of daily life in the postpartum period. PDPH may also lead to the

development of chronic migraines.[8] The evidence demonstrates that SPGB carries less risk, is less expensive, and provides quicker onset of headache relief in a vulnerable population, which may decrease the length of hospital stay and prevent recurring emergency room visits. It is crucial that the obstetric anesthesia practitioner is familiar with current practice guidelines and the empirical evidence which supports the comfort and safety of postpartum patients.

References

1. Kasson, BJ. Obstetric Anesthesia. In: Nagelhout JJ, Elisha S, Plaus K. *Nurse Anesthesia.* 5th ed. Saunders; 2013.
2. Takmaz SA, Karaoğlan M, Baltacı B, Bektaş M, Başar H. Transnasal sphenopalatine ganglion block for management of postdural puncture headache in Non-Obstetric Patients. *J Nippon Med Sch*. 2021;88(4):291-295. doi:10.1272/jnms.JNMS.2021_88-406
3. Youssef HA, Abdel-Ghaffar HS, Mostafa MF, Abbas YH, Mahmoud AO, Herdan RA. Sphenopalatine ganglion versus greater occipital nerve blocks in treating post-dural puncture headache after spinal anesthesia for cesarean section: A randomized clinical trial. *Pain Physican*. 2021;24(4):E443-E451. PMID: 34213869
4. Cohen S, Levin D, Mellender S, et al. Topical sphenopalatine ganglion block compared with epidural blood patch for postdural puncture headache management in postpartum patients: A retrospective review. *Reg Anesth Pain Med*. 2018;43(8):880-884. doi:10.1097/AAP.0000000000000840
5. Albaqami MS, Alwarhi FI, Alqarni AA. The efficacy of sphenopalatine ganglion block for the treatment of postdural puncture headache among obstetric population. *Saudi J Anaesth*. 2022;16(1):45-51. doi:10.4103/sja.sja_651_21
6. Delgado C, Bollag L, Van Cleve W. Neuraxial labor analgesia utilization, incidence of postdural puncture headache, and epidural blood patch Placement for privately insured parturients in the United States (2008-2015). *Anesth Analg*. 2020;131(3):850-856. doi:10.1213/ANE.0000000000004561
7. Urits I, Cai V, Aner M, et al. Post dural puncture headache, managed with epidural blood patch is associated with subsequent chronic low back pain in patients: a pilot study. *Curr Pain Headache Rep*. 2020;24(1):1. Published 2020 Jan 8. doi:10.1007/s11916-020-0834-5
8. Barad M, Carroll I, Reina MA, Ansari J, Flood P. Did she have an epidural? The long-term consequences of postdural puncture headache and the role of unintended dural puncture. *Headache*. 2021;61(9):1314-1323. doi:10.1111/head.14221
9. Abdelhaleem Abdelhaleem NF. Verification of sphenopalatine ganglion block success ssing transcranial doppler in management of patients with postdural. *Pain Physician*. 2021;24(5):E661-E668. PMID: 34323454

Mentor: Morgan Morrow, DNAP, CRNA

Educational Intervention on the Management of Postoperative Nausea and Vomiting

Christine Johnson, BSN
Goldfarb School of Nursing, Barnes-Jewish College

Keywords: Postoperative nausea and vomiting, PONV, Fourth Consensus Guidelines for the Management of Postoperative Nausea and Vomiting, Apfel score, antiemetics

Introduction

Postoperative nausea and vomiting (PONV) is a common adverse event related to general anesthesia; it has an incidence of roughly 30% in the general public and an occurrence of up to 80% in those who are considered high-risk.[1] Recently, new evidence-based guidelines have been released on the methods to treat, reduce, and prevent PONV.[2-5] This quality improvement project aimed to provide education for anesthesia professionals on the Fourth Consensus Guidelines for the Management of PONV[3] and improve provider adherence to these guidelines in managing PONV.

Design and Methods

Institutional Review Board (IRB) approval was obtained prior to the initiation of this project. A recruitment email was sent to certified registered nurse anesthetists (CRNAs) practicing at a large teaching hospital. A 6-minute educational video, a handout detailing the Fourth Consensus Guidelines for the Management of PONV, and a 5-question educational assessment quiz were emailed to CRNAs who consented to participate in the study. A pre-education chart audit and 3 post-education chart audits were completed to determine if compliance to the guidelines improved after the educational intervention. The following measurement instruments were used: the electronic medical record (EMR), a post-education quiz, a provider demographic questionnaire, and the Apfel simplified risk score.

Outcome

The mean age for participating CRNAs was 41.3 years (range 29 to 64) and were predominately female (67%) and Caucasian (83%). The mean provider years for practicing anesthesia was 8.8 years (range 1.5 to 25). The EMR review consisted of 30 pre-education and 62 post-education charts. There were 24 post-education chart reviews completed at the 2-week data point, 23 at the 4-week data point, and 15 at the 6-week data point. The average patient age was 57.3 years (range 20 to 90) and 59% were female. Apfel risk scores were primarily 2 (35%) or 3 (36%). The majority of patients received two or fewer antiemetics. Out of the 20 patients who had a history of PONV/motion sickness, 55% received the proper number of antiemetics recommended by the consensus guidelines. The total percent of correct antiemetics prescribed for all patients was 38%. The correct number of antiemetics given prior to education was 50%. At the 2, 4, and 6-week marks post-education compliance was 21%, 30% , and 53% respectively. There was no significant association between the pre-education and post-education groups ($\chi^2(1, N = 92) = 2.7$; $P = .05$, $\varphi = .17$) and whether the patient received the correct number of antiemetics. Additionally, there was no significant association between the correct number of antiemetics

given and if a student nurse anesthetist was involved in the case ($\chi^2(1, N = 92) = .02$, $P = .26$, $\varphi = .01$).

Conclusion

While compliance to the guidelines was initially low, it progressively increased during each data point, with the final 6-week mark showing improved compliance when compared to the retrospective cohort. Limitations include a small sample size and limited time for conducting the study. Recommendations include providing more options for antiemetics since some of the medications in the guidelines are not formulary. Focus should be placed on administering antiemetics in the preoperative area as an order set, much like oral pain medications. This would provide consistency in administration since some antiemetics are only available in oral form and must be taken prior to general anesthesia. In conclusion, CRNAs followed the new Fourth Consensus Guidelines for the Management of PONV more often after the educational intervention at the 6-week data point, supporting the importance and utility of instructional activities in the workplace.

References

1. Krieser KA, Riley JB, Baus JE, Hoffman JT, Sullivan JN, Lobato RL. PONV prophylaxis failure disproportionately affects female patients, despite intraoperative computerized decision support guidance. *Grad Med Educ Res J.* 2020;2(1):1-14. doi:10.32873/unmc.dc.gmerj.2.1.091
2. Chiu C, Aleshi P, Esserman LJ, et al. Improved analgesia and reduced post-operative nausea and vomiting after implementation of an enhanced recovery after surgery (ERAS) pathway for total mastectomy. *BMC Anesthesiol.* 2018;18:41. doi: 10.1186/s12871-018-0505-9
3. Gan TJ, Belani KG, Bergese S, et al. Fourth consensus guidelines for the management of postoperative nausea and vomiting. *Anesth Analg*. 2020;131(2):411-448. doi:10.1213/ANE.0000000000004833
4. Gupta R, Soto R. Prophylaxis and management of postoperative nausea and vomiting in enhanced recovery protocols: expert opinion statement from the American Society for Enhanced Recovery (ASER). *Perioper Med (Lond)*. 2016;5:4. doi: 10.1186/s13741-016-0029-0
5. Kranke P. General multimodal or scheduled risk-adopted postoperative nausea and vomiting prevention: just splitting hairs? *Br J Anaesth.* 2015;114:190–193. doi: 10.1093/bja/aeu344

Mentor: Bernadette Henrichs, PhD, CRNA, CCRN, CHSE, FAANA

Editorial

Happy New Year! This issue provides a nice variety of submissions – our traditional case reports, but also some evidence-based practice analysis reports and an evidence-based practice project abstract (our newest submission item). I would also like to point out an original illustration of Fontan physiology rendered by one of our student authors. This is a first for the student journal and can be found on page 24.

The International Student Journal of Nurse Anesthesia has evolved over time to offer additional options for authors to disseminate their work. With the transition to the practice doctorate, I am grateful and honored to provide this venue for nurse anesthesia student publication. This, of course, would not be possible without the numerous CRNA volunteers that have generously donated their time and talent as editors, reviewers, and mentors. On that note, I would like to thank all CRNAs for their contributions to our profession and continuing to keep patient safety at the forefront. I hope everyone had a wonderful National CRNA Week.

Sincerely,

Vicki Callan, PhD, CRNA, CHSE, FAANA
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 °C, pressure in mm Hg or cm H_2O. Report heart and respiratory rate as X/min (e.g. the patient's heart rate increased to 145/min). 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 bibliography software
___ All material is properly cited and referenced using AMA Style format
___ Generic names for drugs and products are used throughout and spelled correctly in lower-case
___ Units of measure are provided for all dosages, physical findings, and laboratory results
___ Endnotes, footnotes, direct quotes 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 National Library of Medicine (NLM) 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_5.19.19)
___ Item is submitted by the mentor
___ Subject heading format - ISJNA Submission_submission type_author last name_mentor last name