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TOPICS IN THIS ISSUE

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Front Cover:
On the front cover, Jenny Choe, BSN, RN, a doctoral student enrolled in the Northeastern University Nurse Anesthesia Program, performs a subarachnoid block on a patient during a mission trip to Kigali, Rwanda.

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Evidence-Based Practice Analysis Reports

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Keywords: neuromuscular disease, spinal muscular atrophy, SMA type 1, respiratory management, anesthetic management

Spinal muscular atrophy (SMA) type-1 is a type of autosomal recessive disorder that is characterized by cell degeneration in the anterior horn of the spinal cord.[1] The onset is between birth and 6 months of life.[2] SMA type-1 represents 45% of all cases and is marked by physical disability and early mortality.[2] Without respiratory support, it has a 50% mortality by 7 months and 90% by one year.[3] Anesthetic management challenges in patients with SMA type 1 are attributed to hypoventilation, recurrent lower airway infection, ineffective cough, anatomical airway problems, dysphagia, airway obstructions, and restrictive respiratory pathology.[1]

Case Report

A 24-year-old motorized wheelchair and respiratory-dependent male (53 kg, 160 cm) with SMA type 1 (Werdnig-Hoffman disease) presented for cystoscopy, ureteroscopy, cystolitholapaxy, laser lithotripsy, and ureteral stent replacement. His past medical history included severe restrictive respiratory physiology with 0 mL of vital capacity and dependence on continuous nasal ventilatory support (CNVS) since 4 months of age. He had no autonomous skeletal muscle function, no bulbar muscle function, and facial movement limited to several mm of eye movement. He had difficulty voiding and required catheterization. He has a feeding tube, and according to his parents, he communicates with his “eyes”. His computerized tomography (CT) scan was significant for an atrophic left kidney with diffuse cortical thinning and bilateral hydronephrosis secondary to obstructive calculi in the bilateral proximal ureters measuring up to 1.1 cm in the left ureter. His medical history was also significant for hypothyroidism and diabetes mellitus type 2.

After thorough consultation with the patient’s family and members of the surgical team, the anesthesia team designed a spinal anesthetic plan of care for this procedure. The patient arrived in the operating room suite accompanied by his mother, who has been his primary caretaker and knows all the parameters and expectations of his medical condition. Standard monitors were applied, and the patient was placed in a left lateral decubitus position. He remained on his CNVS with the following settings: pressure control 20 mm Hg and tidal volume 650 - 700 mL. After multiple failed attempts at spinal anesthesia, an epidural approach using the loss of resistance technique was attempted, but also unsuccessful. Eventually, using the midline approach at L3-4, the patient’s subarachnoid space was successfully accessed. When cerebrospinal fluid was confirmed, 1.2 mL of 0.75% of isobaric bupivacaine plain was injected into the subarachnoid space between. Pressure was held at the site for 3 minutes, vital signs remained stable throughout the procedure and no complications were noted.

The remainder of the intra-operative course was unremarkable for any significant respiratory complications. For prophylactic antibiotic, the patient received cefepime hydrochloride 2 grams

intravenously (IV). Hemodynamic stability was maintained using ephedrine 5 mg IV, glycopyrrolate 0.1 mg IV, phenylephrine 30 mcg/min IV, and lactated ringers 600 mL IV. Throughout the procedure, the patient's EKG remained in a sinus rhythm, and his vital signs ranged as follows: SpO_2 90 - 100%, heart rate 60 - 100/min, and systolic blood pressure 80 - 130 mm Hg. His CNVS parameters remained the same throughout the procedure. The patient was at his baseline level of consciousness in post anesthesia care unit and was discharged to home 45 minutes afterwards.

Discussion

In 1995, the homozygous deletion or mutation of the survival motor neuron 1 (SMN1) gene and the SMN gene-deficient disruption of other cellular processes were identified as the primary and contributory causes of SMA, respectively.[2] Early signs are usually seen in infants with weakness or hypotonia and diagnosis is made by standard molecular genetic testing, which confirms the disease in 95% of patients irrespective of disease severity.[2] Due to the severe respiratory disability in patients with SMA type 1, physical and medical respiratory aids are almost always required to preserve lung health, through normal alveolar ventilation maintenance and cough flow optimization. Some of these aids are in the form of lung volume recruitment, noninvasive positive pressure ventilatory support, daytime support, nocturnal support, assisted coughing, and oximetry feedback protocol.[4]

The patient in this case review had difficulties with expectoration rather than excessive secretion production. His weak inspiratory muscles and the resultant reduced inspiratory capacity limited his pre-cough inspired volume. This limitation resulted in regions of poor lung ventilation which contributed to his stiffened chest wall and lung tissue. In SMA type 1 patients, frequent oropharyngeal suctioning, nebulized normal saline, botulinum toxin injections into the salivary glands, and grape juice intake are some interventions that help break down thick secretions.[5] Medications such as glycopyrrolate have proven to be effective as well in controlling excessive secretions.[5]

A study by Pinto et al[6] suggested that nasal interface to CNVS is associated with decreased social interaction, impaired eating, impaired drinking, and air leaks when higher inspiratory pressures (> 15 cm H_2O) are used. The authors suggested that as it relates to daytime support, the use of an angled mouthpiece supported by a metal flexible arm can be an ideal alternative to CNVS for daytime ventilation in patients with functioning muscle and some preserved neck movement.[6] With the patient in this case review, however, a mouthpiece interface to NVS was strongly discouraged by his pulmonologist because of excessive secretions and management.

General anesthesia is preferably avoided in patients with SMA type 1, especially in surgical procedures where regional anesthesia is equally effective. A case report by Brown et al[1] presented a successful combination of neuraxial and regional anesthesia in a child with advanced SMA type 1 scheduled for Nusinersen maintenance therapy. In this case report, notes from the patient's pulmonologist stated that if intubation should become necessary, the patient should be on full mechanical ventilation (volume control), and his SpO_2 should be maintained on room air. Pulmonary consultation also indicated that if the patient's SpO_2 should decrease below 95% during intubation, then a mechanical in-exsufflation (MIE) via the endotracheal tube must be

applied until the SpO_2 returns to normal. These are some of the contributory considerations that led the anesthesia team to opt for a regional anesthetic.

Considering the severe bulbar-innervated muscle dysfunction seen in patients with SMA type 1, Vianello et al[7] suggest that deglutition ability should be assessed when deliberating extubation attempts. The authors recommend that the Gilardeau score, which provides a functional classification of deglutition impairment in patients with neuromuscular disease, be calculated before intubation and included as extubation criteria. The caveat would be that individuals with higher scores might have to be excluded from immediate extubation post-operatively.[7] With the patient in this case report, had the patient required general anesthesia with endotracheal intubation, conditions for his emergence from general anesthesia and removal of the endotracheal tube would have been that he was fully alert, his SpO_2 was normal on room air, and his mother was present to use the MIE as needed.

In conclusion, anesthetic management in patients with SMA type 1 is very challenging. Because of muscle atrophy and lower motor neuron dysfunction, succinylcholine can cause lethal hyperkalemia due to proliferation of postjunctional nicotinic receptors. Non-depolarizing neuromuscular blockers should be administered with caution as well, due to increased sensitivity. Chest weakness reduces vital capacity and minute ventilation and is worsened under general anesthesia. Bulbar muscle dysfunction results in severe dysphagia and increases the risk of pulmonary aspiration in patients with SMA type 1. Frequent suctioning was a considerable part of the anesthetic plan for the care of this patient. These concerns led the anesthesia team to avoid general anesthesia and utilize a spinal anesthetic for this case, a decision supported by the literature.

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Pediatric Pierre Robin Syndrome: Airway Management Case Study

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Keywords: Pierre Robin Syndrome, pediatric, airway obstruction, otolaryngology

Pierre Robin Syndrome (PRS) is characterized by a series of airway abnormalities including micrognathia, glossoptosis, and airway obstruction with or without cleft palate.[1,2] The presentation of PRS is isolated or associated with an additional genetic syndrome and there is no specific associated genetic abnormality.[1,2] The incidence is between 1 in 8,000-14,000 children with approximately 85% presenting with a cleft palate.[1] Pediatric patients are at a higher risk for complications during airway management and craniofacial abnormalities further increases the risk.[1–4] This case study reviews a pediatric patient with Pierre Robin Syndrome, undergoing general anesthesia in an otolaryngology case.

Case Report

A 2-year-old, 9.7 kg patient with isolated PRS presented for tonsillectomy with adenoidectomy to treat severe obstructive sleep apnea (OSA). The patient had a previous cleft palate repair and bilateral mandibular osteotomies with distraction device placement. Despite the full repair, the patient continued to have severe airway obstruction dependent on bilevel positive airway pressure.

On preoperative physical exam the patient had severe retrognathia, micrognathia, and clear breath sounds. Further airway assessment was unable to be obtained due to the child's age. Past medical records revealed the patient had tolerated four previous general anesthetics. The airway was previously managed with both an endotracheal tube using video laryngoscopy with a Storz C-MAC Miller 1 blade and laryngeal mask airway (LMA).

A pulse oximeter and 3–lead electrocardiogram (EKG) was placed on the patient for induction. An inhalational induction with sevoflurane 8% inspired concentration in O_2 10 L/m was used to achieve general anesthesia. A Guedel 70 mm oral airway was placed to relieve upper airway obstruction during mask induction and the patient remained spontaneously breathing throughout.

After induction the sevoflurane was decreased to 4% in O_2 2 L/m while intravenous access was obtained. Neuromuscular blockade was achieved with rocuronium, and the patient was easy to mask with the oral airway in place. Prior to airway manipulation a shoulder roll was placed. Using a Storz C-MAC Miller 1 blade, a grade four direct laryngoscopy view and grade 2b indirect laryngoscopy view was obtained. The airway was secured with a 4.0 endotracheal tube. Dexamethasone 4mg was administered, and five 90 mcg puffs of albuterol were administered. General anesthesia was maintained with a remifentanil infusion at 0.3mcg/kg/min and sevoflurane 1-1.5% expired in air 2 L/min.

The maintenance of anesthesia for the 32-minute tonsillectomy with adenoidectomy was uneventful. No long-acting pain management or opioid medications were administered. Upon

completion of the procedure, anesthetic vapors were discontinued, the patient was placed on O_2 10 L/min, neuromuscular blockade was antagonized with sugammadex, the endotracheal tube was suctioned, and five 90 mcg puffs of albuterol were administered through the endotracheal tube. When the patient was fully awake and making purposeful movements, they were extubated to face mask O_2 at 10 L/min. The patient quickly became agitated after extubation and received 8 mcg dexmedetomidine and 0.5 mg morphine. Continuous positive airway pressure (CPAP) at 10 cm H_2O was applied with face mask when the patient experienced respiratory distress with retractions. A Yankauer was used to suction copious oropharyngeal secretions and 0.1mg glycopyrrolate was administered.

The patient had severe retractions and was unable to ventilate spontaneously and was placed in reverse Trendelenburg. A complete upper airway obstruction occurred, and ventilation was not achieved with two hand masking and placement of a 70 mm Guedel oral airway. The patient desaturated to S_pO_2 80%. Immediate reintubation was achieved with 20 mg propofol and 10 mg rocuronium and the S_pO_2 returned to 99%. A Storz C-MAC Miller 1 blade and 4.0 endotracheal tube was used for successful reintubation. The patient was transported to the intensive care unit (ICU) with standard noninvasive monitors, sedated with a dexmedetomidine infusion 0.5 mcg/kg/min, and manually ventilated with a Jackson-Reese circuit. Transport was uneventful and the patient remained hemodynamically stable. The patient was extubated in the ICU two days later without negative sequalae.

Discussion

Pierre Robin Syndrome is characterized by micrognathia, glossoptosis, and airway obstruction. The presence of PRS can be isolated or associated with an additional genetic syndromes such as DiGeorge and Treacher Collins syndrome.[1,2] The retrognathic mandible causes displacement of the tongue towards the back of the mouth increasing the risk for upper airway obstruction.[1] PRS is associated with cleft palate in 85% of cases due to displacement of the tongue physically preventing the formation and fusion of palatal shelves.[1]

There are several treatment options to relieve airway obstruction in PRS patients. Non-surgical alternatives include prone positioning, nasopharyngeal airway placement, and CPAP.[2] Mandibular distraction osteogenesis is considered the definitive surgical treatment for airway obstruction.[1,2] The mandible is gradually lengthened to allow anterior movement of the tongue base and relieve airway obstruction.[1,2] After bilateral mandibular osteotomies, the patient continued to have severe OSA and presented for a tonsillectomy and adenoidectomy.

Tonsillectomy and adenoidectomy are common procedures to relieve airway obstruction in pediatric patients.[5] Patients with severe OSA may have an abnormal response during the intraoperative or postoperative period and should be managed cautiously. Increased opioid sensitivity is observed in this patient population due to alterations in the mu receptor from repeated episodes of oxygen desaturation.[5] An opioid sparing approach was utilized in this case to avoid increased respiratory depressant effect and prolonged emergence. Anesthetic vapors and sedatives also have an increased respiratory depressant effect on this patient population.[5] A remifentanil infusion was used to decrease the anesthetic vapor requirement and dexmedetomidine was administered after extubation to treat agitation. There is controversy over

whether an awake or deep extubation is recommended after this procedure and there is currently no evidence supporting either technique.[5] An awake extubation was selected due to the patient's severe OSA and requirement for bilevel positive airway pressure. Shared airway cases require exceptional communication between the anesthesia and surgery teams to prevent complications such as airway fires. Primary postoperative hemorrhage is a major shared complication that can further complicate the known difficult airway.[5] When the patient was initially in respiratory distress post-extubation, the decision not to place a nasopharyngeal or oral airway was made to reduce the likelihood of primary hemorrhage. An oral airway was placed as a last-line intervention when the patient was unable to be ventilated.

The most common perioperative complication in the pediatric population is respiratory compromise due to their unique anatomy and physiology.[3-5] Pediatric patients have a more cephalad larynx, a large occiput, and are high risk for hypoxia during airway management due to high metabolic demand and decreased reserve.[4] The highest risk age group is less than three years old.[3] PRS is associated with difficult airway management due to the features of micrognathia and glossoptosis.[3] Management strategies include appropriate patient positioning, oral or nasal airway placement, two-person masking, LMA placement to bypass the obstruction, and first-attempt videolaryngoscopy.[4] Neuromuscular blockade is recommended in pediatric patients with anticipated difficult airways to prevent complications.[3] When approaching a known difficult airway, it is crucial to have multiple plans in place, maintain ventilation between attempts, call for help after two unsuccessful attempts, and have difficult airway equipment in the room such as video-laryngoscope, fiberoptic, and LMA.[3]

In this case the first-line airway management technique was video laryngoscopy, a shoulder roll was used for positioning to assist with aligning axes, an oral airway was placed, rocuronium was administered to achieve neuromuscular blockade, and an appropriately sized LMA was opened and prepared prior to airway management.[3,4] When the patient was unable to be ventilated after extubation, help was called for immediately and the patient was prepared for reintubation with propofol and rocuronium. Neuromuscular blockade antagonism was achieved 20 minutes prior with sugammadex. When rocuronium is administered within 24 hours of sugammadex, there is risk of prolonged onset and short duration of action.[6] It is recommended to administer a benzylisoquinolinium neuromuscular blocking agent for a standard induction or a depolarizing neuromuscular blocking agent for a rapid sequence induction.[6]

Pierre Robin Syndrome patients have many airway considerations due to their altered anatomy. Shared airway cases such as tonsillectomy and adenoidectomy can further complicate airway management making communication between surgery and anesthesia imperative. It is crucial to have multiple plans in place, difficult airway equipment in the room, and call for help early. The use of succinylcholine would have been a better choice for this patient for reintubation in the setting of recent sugammadex administration and the inability to ventilate due to a more predictable onset and duration of action. The choice to administer rocuronium did not negatively impact the patient's outcome. The patient was successfully exubated in the ICU two days later and discharged home.

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Anesthetic Management for Pectus Excavatum Repair

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Keywords: Pectus excavatum, Nuss procedure, cryoablation, double lumen tube, MIRPE

Pectus excavatum (PE) is the most common congenital chest wall anomaly, accounting for 90% of cases.[1,2] Pectus excavatum can involve the third to seventh ribs or costocartilages, can be symmetrical or asymmetrical, and may include profound deformity of the xiphisternum. The anomaly affects males more than females at a 5:1 ratio and occurs in 1/300 to 1/1000 births. Pectus can first be recognized in infancy but is more commonly diagnosed during childhood.[1] The majority of PE cases are idiopathic; however, it can be associated with other congenital syndromes such as Marfan, Ehler-Danlos, Noonan and others.[1,2]

Case Report

A 46.6 kg, 165.7 cm, 14-year-old male presented for PE repair with Nuss procedure via thorascopic approach. Past medical history included mild intermittent asthma, since resolved, snoring, and underweight BMI. He had no known drug allergies. His preoperative studies included an electrocardiogram showing normal sinus rhythm, spirometry without evidence of bronchial obstruction before or after exercise, and no evidence of asthma. His preoperative computed tomography scan revealed severe PE with Haller index of 6.3. His past surgical history include trigger finger release of the left thumb. Physical exam revealed a thin adolescent with PE but was otherwise unremarkable.

A 20-gauge peripheral venous catheter (PIV) was inserted in the left hand in the preoperative area. Premedication of midazolam 2 mg was given intravenously (IV) prior to entering the operating room. Once in the operating room, standard noninvasive monitors were applied. A processed electroencephalogram monitor, was placed on the forehead with a baseline awake patient state index (PSI) of 96. The patient was preoxygenated with O_2 10L/min for 2 minutes. For induction of general anesthesia, fentanyl 100 mcg, lidocaine 20 mg, and propofol 200 mg were given IV. The ability to bag mask ventilate was confirmed and rocuronium 50 mg was given. Protective oval eye tape was placed. A videoscope Mac 3 blade was used to intubate the trachea with a 39 French double lumen endotracheal tube (DLT). The DLT was secured at 29 cm at the lip and pressure control ventilation with an inspiratory pressure of 20 cm H_2O, respiratory rate of 12/min, and positive-end-expiratory pressure of 5 cm commenced. Fiberoptic bronchoscopy was used to confirm DLT placement in the left main bronchus. General anesthesia was maintained with propofol infusion of 300 mcg/kg/minute and a remifentanil infusion at 0.2 mcg/kg/minute.

An additional 18-gauge PIV was placed in the right forearm. A nasopharyngeal temperature probe and forced air-warming device were applied. Cefazolin 1g and methadone 5 mg were given prior to surgical incision. Bilateral thorascopy incisions were made. The patient was placed on O_2 10L/min and one lung ventilation (OLV) was initiated with the left lung deflated in supine positon. Cryoablation was first completed by the surgical team to the left intercostal thoracic nerves T3-7 followed by intercostal nerve blocks at ribs 3-7 with 2 mL of bupivicaine 0.25% with epinephrine. Two lung ventilation was resumed for 2 minutes, and one lung ventilation was initiated with the right lung deflated. Cryoablation was completed by the surgical team to the right intercostal thoracic nerves T3-7 and intercostal nerve blocks at ribs 3-7. Ventilation continued with the right lung deflated for the placement of the Nuss bar and a mixture of O_2 1L/min and air 1 L/min.

During surgical closure, ketorolac 15 mg, acetaminophen 500 mg, and ondansetron 4 mg were given. When surgical closure was completed, two lung ventilation with recruitment maneuvers was resumed with O_2 10 L/min. The propofol and remifentanil infusions were stopped and sugammadex 100 mg was given. Once the patient was spontaneously breathing without pressure support, opening eyes to commands, and able to lift his head off the pillow, the DLT was removed and O_2 6 L/min was administered via face mask. Intraoperative fluids totaled 1.5 L of lactated ringers with an estimated blood loss of 10 mL. The patient was transported to the post-anesthesia care unit while remaining monitored. Total operative time was 4 hours.

Discussion

The majority of patients with PE are asymptomatic; however, depending on cardiopulmonary compression, patients may develop progressive symptoms. Common associated diagnoisis include restrictive lung disease, pain, mitral valve prolapse, and scoliosis.[1] Patients present for PE repair depending upon the severity of their disease as evidenced by their pulmonary function tests and Haller index. The Haller index quantifies the PE deformity by calculating the transverse and anteroposterior diameter and has a normal value of 2.5.[1] Patients who present for PE repair

typically have a Haller index above 3.2 or may present for cosmetic and or psychological reasons.[1,2,] This patient's Haller index was 6.3.

The use of cryoablation for minimally invasive pectus excavatum repair (MIRPE) with the Nuss bar is a newer surgical technique first performed in 2016.[3] This method disrupts the structure of nerves causing a Wallerian degeneration eliminating afferent conduction and pain perception, while leaving the myelin sheath and endoneurium of the nerve intact.[4,5] The nerve gradually heals and regains efferent function and sensation within 2–12 months.[4] The use of cryoablation eliminates the need for thoracic epidural catheters for pain control after PE repair.[2]

Anesthetic management goals for MIRPE included maintaining oxygenation during one lung ventilation, hemodynamic stability, immobility throughout surgery, and following the pain protocol for minimally invasive pectus excavatum repair that this children's center developed. The standard of care management for this MIRPE case including monitoring, OLV and analgesia are described above.

A total intravenous anesthesia (TIVA) technique was used for this case for maintenance of general anesthesia. TIVA as the primary anesthetic for PE repair has been used successfully in several studies.[2,5] Propofol and remifentanil infusions were titrated based upon the patient's heart rate, blood pressure, and SedLine (Masimo) reading, targeting a goal of 25 – 50 patient state index (PSI), a quantitative EEG index that has been demonstrated to provide hypnosis for surgery under general anesthesia.[6] The remifentanil infusion was decreased to 0.1 mcg/kg/min and was stopped at the completion of the surgery. Other centers have used a bispectral index (BIS) for monitoring depth of anesthesia with a target range of 40 – 60.[6] The SedLine (Masimo) was used due to availability of equipment at our center. Studies have found that both BIS and PSI values monitor hypnosis under propofol anesthesia but the PSI values tend to run 10-15 lower than the BIS values.[6]

Published Enhanced Recovery After Surgery (ERAS) protocols for PE repair have included acetaminophen 15 mg/kg, ketorolac 0.5 mg/kg, a long-acting opioid, bilateral cryoablation T4-8, postoperative nausea and vomiting (PONV) prophylaxis, and bilateral serratus anterior plane blocks.[3] For our patient, a similar protocol was followed that included a single dose of methadone 5mg IV as the long-acting opioid, acetaminophen 500 mg IV, ketorolac 15 mg IV, bilateral cryoablation T3-7, and ondansetron 4 mg IV for PONV. Dexamethasone was not given as part of PONV prophylaxis due to a prior unclear reported adverse reaction to steroids per the patient's father. Bilateral serratus anterior plane blocks were not administered by anesthesia professionals because the surgical team provided the cryoablation and local anesthesia was injected directly into the intercostal spaces.

Literature for MIRPE with cryoablation is limited. Cryoablation for PE repair has been shown to decrease hospital length of stay,[2,4] from 6 to 2.4 days.[4] The patient in this case was discharged on postoperative day two with oral pain medications. Cryoablation with PE repair has also demonstrated improved postoperative analgesia with decreased opioid requirements when using a multi-modal analgesic approach.[4,5] One disadvantage of cryoablation for PE repair is longer operative times.[4] Further research is required to study the recovery, short- and long-term benefits of cryoablation for PE repair.

Intraoperative and postoperative complications associated with PE repair can be serious. Complications associated with PE repair include pneumothorax, cardiac dissection, cardiac tamponade, perforation of heart or other great vessels, pericarditis, and pericardial effusion.[1] The focus of care for the patient was communication with the surgical team, age developmental communication with the patient, oxygenation while maintaining OLV, hemodynamic stability, and meticulous cardiovascular and brain function monitoring. Postoperative monitoring is imperative with a team cognizant of PE repair complications and pain control needs. This case was conducted without any complications; however, the addition of invasive arterial monitoring would have allowed for continual arterial pressures indicating any early detection of complications. Advances in surgical techniques require adjustments to the anesthetic management of patients presenting for PE repair. Communication, planning, pain management, and careful monitoring during the intraoperative and postoperative periods are crucial.

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Anesthesia Implications for Children with Bronchopulmonary Dysplasia

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Keywords: bronchopulmonary dysplasia, BPD, pediatric anesthesia, bronchospasm, perioperative respiratory complications

In the pediatric population, perioperative respiratory adverse events (PRAEs) are the most frequent complication of general anesthesia.[1] The risk of PRAEs is heightened in the presence of respiratory illness. Bronchopulmonary dysplasia (BPD) is the most common chronic lung disease of prematurity.[2] It is typically caused by prolonged mechanical ventilation at birth, high flow rates of oxygen, bacterial infections, and placental insufficiencies.[2] Children with BPD present with impaired alveolar gas exchange, increased airway reactivity, and decreased oxygen reserves.[2] Advances in neonatal medicine have resulted in greater numbers of these patients presenting for surgical procedures with general anesthesia. Implementing strategies to minimize the perioperative risks associated with BPD could reduce PRAEs.

Case Report

A 16-month, 9.6 kg male presented for a direct laryngoscopy and adenoidectomy. His medical history included premature birth at 27 weeks gestation, greater than 3-month neonatal intensive care stay, mechanical ventilation at birth, BPD, asthma, laryngomalacia, obstructive sleep apnea (OSA), nocturnal oxygen therapy, and global developmental delays. Home medications included nebulized albuterol as needed, budesonide/formoterol 80mg/4.5mg orally once daily and cetirizine 2.5 mg orally once daily.

During the preoperative assessment, the mother stated the child had no cough, rhinitis, or other symptoms of upper respiratory infection. Prescribed daily medications had not yet been given. Approximately 2 ½ weeks earlier, an episode of wheezing and dyspnea had been treated with nebulized albuterol with no recurrence of symptoms. She did, however, report noncompliance with administration of the prescribed nocturnal oxygen. Clear, bilateral breath sounds wereauscultated with a room air SpO_2 98%.

On arrival to the operating room, standard noninvasive monitoring was applied and an inhalation induction with sevoflurane 8% inspired concentration in O_2 4 L/min and N_2O 4 L/min was initiated. Spontaneous respirations were maintained until peripheral intravenous (IV) access was obtained and a lactated Ringer's infusion begun. Propofol 50 mg IV, fentanyl 5 mcg IV, glycopyrrolate 0.1 mg IV, dexmedetomidine 3 mcg IV, and dexamethasone 4 mg IV were administered.

Sevoflurane was decreased to 3% inspired concentration in O_2 4 L/min to maintain general anesthesia. The otorhinolaryngologist performed an uneventful rigid bronchoscopy, intubated the trachea with a 3.5 mm cuffed endotracheal tube (ETT), inserted to 11 cm at the lip, and flows were then adjusted to O_2 0.5 L/min and air 0.5 L/min. An uneventful adenoidectomy was completed and stable vital signs maintained. Sevoflurane and air were discontinued and O_2

increased to 8 L/min. The patient was suctioned and extubated when he began grimacing and grasping the ETT and O_2 6 L/min was administered by mask insufflation. He was transported to PACU with a SpO_2 100%, stable vital signs, no dyspnea, and with O_2 3 L/min via blow-by. After arrival to PACU, the patient became tachypneic with suprasternal and intercostal retractions. On arrival, auscultation of breath sounds revealed decreased air movement with an expiratory wheeze indicative of an acute bronchospasm. The patient's SpO_2 decreased to 74% and heart rate decreased to 45/min. Chest compressions and mask ventilation were initiated. Epinephrine 0.09 mg IV, glycopyrrolate 0.1 mg IV, and propofol 30 mg IV were administered. A 3.5 mm cuffed ETT was placed by direct laryngoscopy (DL) by the anesthesia practitioner. With no improvement in the clinical picture and no $ETCO_2$ measurement available, placement was questioned and the ETT was removed. An attempted reintubation with videolaryngoscopy by a second anesthesia practitioner was unsuccessful due to laryngeal edema.

As the attending otorhinolaryngologist arrived, propofol 20 mg IV and succinylcholine 20 mg IV were administered. The otorhinolaryngologist successfully intubated with a 3.5 mm micro-cuffed ETT by DL. Placement was confirmed by the presence of bilateral breath sounds, adequate chest rise, and steady SpO_2 increase to 88%. The heart rate increased to 120/min and chest compressions were discontinued. A nebulized combination of albuterol 2.5 mg, dexamethasone 4 mg, and racemic epinephrine 11.25 mg was administered via the ETT in O_2 4 L/min with manual positive pressure ventilation.

The patient was manually ventilated to maintain SpO_2 greater than 92% and transported to the pediatric intensive care unit. Postoperative care included nebulized albuterol and IV steroids. He was extubated and weaned to room air within 12 hours and discharged home the following day.

Discussion

With improved treatment modalities, more infants with BPD are thriving and may be presenting for surgical procedures.[2] These patients are at an increased risk of experiencing PRAEs including desaturation, bronchospasms, and laryngospasms.[2] Male gender, age less than 2 years, the presence of OSA and/or asthma, and surgical procedures on the airway increase that risk even more.[1,3]

Practitioners should ensure the continuation of home medications preoperatively.[3,4] In a recent randomized clinical trial with preoperative nebulized albuterol versus placebo, researchers found that 27% of children in the albuterol group experienced an adverse event compared to 47% in the placebo group.[4] With a safe, well-known profile and common availability, pretreating children with BPD with albuterol is recommended in all cases to decrease bronchial reactivity before airway manipulation.[4]

Reducing the stress response before surgery could be especially beneficial in this patient population. However, preoperative anxiolytics must be carefully considered. In a randomized controlled trial comparing intranasal midazolam, dexmedetomidine, and placebo on rates of PRAEs, researchers found that midazolam led to increased adverse events.[5] This increased incidence could be due to midazolam's effect of disinhibiting behavior.[5] In comparison,

dexmedetomidine suppresses airway reflexes and decreases agitation and bronchial reactivity. Therefore, dexmedetomidine may be superior to midazolam preoperatively.[3,5,6]

The mode of induction has been found to play a significant role in respiratory related outcomes. In a randomized controlled trial comparing inhalation induction with sevoflurane and IV induction with propofol, IV induction with propofol decreased PRAEs by almost half.[7] Propofol more effectively blunts reflex bronchoconstriction and better suppresses laryngeal reflexes when compared to sevoflurane.[6,7] When appropriate, an IV induction with propofol is recommended.[7] Inserting a laryngeal mask airway (LMA) is less stimulating to the patient with a reactive airway than placement of an ETT.[3,6,8] Regarding the case discussed, use of an LMA was not a viable option. Neuromuscular blockade agents that stimulate the release of histamine, such as atracurium and cisatracurium, should be avoided.[6] However, there is some evidence that rocuronium can cause bronchoconstriction due to its M2 and M3 receptor blocking effects.[8] When used with the high risk patient, rocuronium induced bronchospasm may be more likely. Vecuronium could be a safer choice.[6,8]

Other medications useful for attenuating PRAEs have been suggested. Administering glycopyrrolate after induction can provide protection by decreasing cholinergic-mediated broncoconstriction.[3,6] Lidocaine should also be considered for its ability to relax the smooth muscles of the airway.[3,6,8] A single dose of dexamethasone given after induction may decrease the risk of postoperative bronchospasm due to its anti-inflammatory effects.[6]

Maintaining general anesthesia with propofol as a total intravenous anesthesia (TIVA) technique may translate to a decreased incidence of PRAEs.[6] The suggested advantages of TIVA include decreased coughing, preservation of hypoxic pulmonary vasoconstriction, decreased incidence of bronchospasm and laryngospasm, improved ciliary function, and decreased airway reactivity.[6,8] TIVA could be the technique of choice in this high-risk population group.[6]

Ventilation strategies have not been adequately studied in BPD patients presenting for general anesthesia, but protective lung ventilation strategies are recommended. Avoiding high fractions of inspired oxygen decreases atelectasis formation and better maintains gas exchange surfaces.[3,8] Choosing between awake and deep extubation relies heavily on provider confidence, ease of airway manipulation, and patient risk factors. Dexmedetomidine has been associated with decreased agitation and airway reactivity during emergence and should be considered.[5] When comparing these evidence-based recommendations to the decisions made during the described case, it is recognized that some actions could have been taken to decrease the risk of PRAEs. The patient presented with multiple risk factors for developing PRAEs and could have benefited from preoperative albuterol administration.

After the patient is anesthetized and IV access obtained, a dose of propofol before airway manipulation and/or TIVA with propofol for maintenance could have further decreased the risk of bronchial reactivity. No lidocaine was administered intraoperatively or during the post-extubation cardiopulmonary event. Lidocaine 2 mg/kg could have been given to decrease airway reflexes.[8] Additional dexmedetomidine during emergence could have decreased reactivity and provided more protection in the early postoperative phase.

Utilization of a perioperative protocol for optimizing respiratory function could have prevented this patient's bronchospasm induced critical event. After a thorough review of the literature, it was determined that no protocols currently exist. The opportunity to standardize care with a protocol that provides a reference for anesthesia practitioners caring for high-risk patients could decrease PRAEs when compared to the current standard of care.

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Apneic Oxygenation for Excision of Human Papillomavirus on the Vocal Cords

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Keywords: Human papillomavirus (HPV), direct laryngoscopy

Laryngeal papillomatosis, also known as recurrent respiratory papillomatosis, is a benign neoplasm caused by human papillomavirus (HPV).[1] This condition causes masses in the larynx, trachea, or lungs and is a common cause of hoarseness and airway obstruction.[1] Laryngeal papillomatosis affects patients of all ages and is categorized as adult or juvenile, with the juvenile form noted to be more aggressive with more frequent recurrences.[2] The current standard of care treatment is surgical debulking by CO_2 laser ablation, microdebridement, or cryotherapy.[2] Airway surgeries present unique challenges for anesthetic management.

Case Report

A 21-year-old female with recurrent laryngeal papillomatosis presented for direct laryngoscopy, bronchoscopy, and excision of vocal cord papillomas with a CO_2 laser. On the day of surgery, the patient weighed 68kg and was 62cm tall with a BMI of 27.42 kg/m^2. The patient reported allergies to metoclopramide, ibuprofen, and bee venom. Past medical history was notable for recurrent respiratory papillomatosis, asthma, and gastroesophageal reflux disease (GERD), which was well controlled. Home medications included omeprazole and albuterol. Past surgical history included six previous airway surgeries of this nature with no noted anesthetic complications. Previous intubations via direct laryngoscopy indicated a grade 1 view per the Cormack-Lehane scale. The patient reported to be in good health on the day of surgery but noted an asthma exacerbation requiring a visit to the emergency room and cessation of smoking tobacco two weeks prior. Airway examination revealed a Mallampati score of 1, thyromental distance greater than 6cm, good oral aperture, and no limitations to neck extension. The patient was noted to have dysphonia but denied difficulty swallowing or any symptoms of airway obstruction. On auscultation, the lungs were clear bilaterally.

The patient was brought to the operating room, and standard noninvasive monitors were applied. Midazolam 2mg intravenously (IV) was administered. The patient was preoxygenated with O_2 14 L/min via hand-held facemask. The surgeon requested that the patient continue spontaneous ventilation initially for the direct laryngoscopy. An IV induction was done with fentanyl 100 mcg, lidocaine 30 mg, and propofol 100 mg IV bolus. A propofol infusion was initiated at 200 mcg/kg/min and subsequently titrated and maintained at 350 mcg/kg/min throughout the procedure. The patient's eyes were carefully taped and covered with appropriate laser-safe goggles, and a nasal cannula was applied with 4 L/min ancillary O_2. The patient was positioned supine with arms tucked bilaterally. The head of the bed was turned 90 degrees, and the surgeon positioned the patient in Rose position which utilizes a pillow placed under the shoulders to extend the head and neck.

General anesthesia with a natural airway was maintained with additional IV medications: propofol boluses totaling 450 mg, fentanyl 100 mcg, and dexmedetomidine 20 mcg to achieve

apnea for the laser ablation. Direct laryngoscopy was performed with a Miller blade size 3; a rigid bronchoscope was then utilized to visualize the trachea and proximal bronchi. The patient was apneic for the surgical excision of vocal cord papillomas using a CO_2 laser. She maintained SpO2 > 90% except for two episodes when it decreased to ~ 86%, and the procedure was paused. The trachea was then intubated by the surgeon, and mechanical ventilation was initiated via the anesthesia machine circuit until the SpO2 was 100%. The patient was then extubated, and the procedure resumed. To prevent an airway fire, a laser-safe endotracheal tube (ETT) was utilized, and inspired oxygen concentration (FiO_2) was maintained at less than 30% one minute before and while the laser was in use. In addition, the ancillary O_2 supplying the nasal cannula was turned off one minute before and while the laser was in use. A bottle of saline was kept within reach on the anesthesia machine. All staff in the room wore laser-appropriate goggles for eye protection and N95 masks due to the airborne precautions associated with the procedure. Additional prophylactic medication administered IV were dexamethasone 12 mg for airway edema, ondansetron 4 mg for antiemesis, and acetaminophen 1000 mg for postoperative pain. The patient tolerated the procedure well.

Upon completion of the procedure, an appropriate-sized oral airway was placed, and the patient was noted to be spontaneously ventilating. The patient was transferred to PACU on 6 L/min O_2 via the Jackson Reese circuit. She was observed for 90 minutes and discharged home after a full recovery. During telephone follow-up on post-op day one, the patient reported no complications and was "doing well."

Discussion

Airway surgery, especially in the upper airway, presents unique challenges for anesthetic management. It is, by nature, a "shared" airway that requires careful planning and collaboration between the surgical and anesthesia teams and provides the opportunity for various airway management techniques.[3,4] These techniques traditionally include general anesthesia with a secured airway via a smaller diameter ETT or intubating laryngeal mask airway, use of high-frequency jet ventilation, or apneic oxygenation, which involves the passive flow of oxygen into the lungs during apnea, often with high flow nasal oxygenation (HFNO).[3-5]

There are many benefits associated with HFNO. It avoids potential airway injury associated with intubation, higher airway pressures associated with a smaller diameter ETT, and enables better exposure and visualization of the surgical field.[3,4] However, apneic oxygenation is not appropriate for all patients and procedures, as CO_2 builds up due to lack of active ventilation and is not routinely monitored. Hypercarbia may ultimately result from HFNO, leading to respiratory acidosis.[3-5] Patient safety is a central consideration of airway management for any surgery, and the use of apneic oxygenation requires careful preoperative assessment of the patient, the surgical and anesthesia team’s comfort level and abilities, and the nature and timing of the procedure.[3,4]

General anesthesia with a natural airway enabled the surgeon to better visualize and intervene in the surgical field. This required maintaining an anesthetic depth at which the patient could tolerate this stimulating procedure while remaining hemodynamically stable. This was achieved with total intravenous anesthesia (TIVA) consisting of a high-dose propofol infusion with

intermittent boluses of propofol, fentanyl, and dexmedetomidine. Through ongoing discussions with the surgeon, the patient was kept spontaneously ventilating with a nasal cannula for the initial direct laryngoscopy so the movement of the papilloma could be visualized. Although HFNO could have been utilized, the patient tolerated the procedure well with a nasal cannula at 4 L/min flow, with end-tidal CO_2 ($ETCO_2$) monitoring.

A thorough preoperative assessment was done with a focus on the airway. Apneic oxygenation was appropriate for this patient as, although she had asthma with a recent exacerbation, she was optimized for surgery and in good health overall. According to prior anesthetic records, she was not considered a difficult airway; and was an easy intubation with a laser safe ETT. Intubation was confirmed with continuous $ETCO_2$ monitoring and breath sounds auscultated bilaterally. In addition, this was anticipated to be a short procedure lasting ~ 20 minutes. The patient tolerated it well, with SpO_2 remaining > 90%, except for two episodes where it decreased briefly to 86%.

An additional consideration is the increased risk of fire during airway surgery. All three elements of the fire triangle, including an ignition source, oxidizer, and fuel source, must be present for an airway fire to occur.[6] The following precautions remove various components of the fire triangle and decrease the risk of airway fire:

- ETT with metallic coating,
- packing the airway with saline-soaked sponges,
- coating facial hair with water-soluble lubricant,
- using flame retardant drapes,
- placing suction below drapes to limit the buildup of O_2,
- avoiding the pooling of prep solutions,
- a bottle or syringes of saline within reach to extinguish fires,
- and staff education for fire safety and prevention.[5]

In addition, FiO_2 must be maintained at less than 30% one minute before and during the use of laser or cautery. As always, constant communication between all team members throughout the procedure is vital to maintain patient safety.[6] The primary response to an airway fire includes removing the ETT immediately, simultaneously stopping the flow of oxygen, pouring water or saline into the airway to extinguish the residual flame, and ventilating with the lowest FiO_2 to reduce the incidence of smoldering and rekindling.[6] In the immediate aftermath, a careful airway exam with bronchoscopy should be performed to assess the need for reintubation.[6]

This case study demonstrates how apneic oxygenation with a nasal cannula can deliver an optimal operative field for airway surgery. It requires careful preoperative assessment of the patient, communication and collaboration of the surgical and anesthesia teams, and additional considerations for safety to ensure success.

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Fentanyl Requirements of the Neonate

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Keywords: fentanyl, neonate, pain

Premature infants are exposed to painful procedures during a vulnerable time of nervous system development. Extremely preterm infants, particularly those less than 28 weeks, spend approximately 81 days in neonatal intensive care units (NICU).[1] On average, NICU patients are exposed to fourteen painful procedures a day, including heel punctures, dressing changes, and injections, with 58% of painful procedures receiving no analgesic intervention.[1-3] This, in turn, exposes them to hundreds of painful procedures before discharge. Early exposure to painful procedures can alter cognition, behavior, health, and pain responses later in life.[1,2]

Case Report

A 5-month-old 3.365 kg patient had been in the NICU since delivery at 24 weeks and two days. This patient would receive half of their feeds, then become distended and stop tolerating them, leading to emesis. Due to this patient's unstable condition, a Ladd operation was scheduled in the NICU procedure room. Anesthesia professionals arrived with an anesthesia travel bag, medications, and an emergency cart. On arrival to the NICU procedure room, the patient was wide awake with an audible leak around the 4.0 mm uncuffed endotracheal tube on the NICU ventilator with a fraction of inspired oxygen (FiO_2) of 0.50 and SpO_2 94%. The patient had a single midline catheter in the left scalp infusing fentanyl 1mcg/kg/hr, dexmedetomidine 2 mcg/kg/hr, total parenteral nutrition, and dextrose 10% solution.

Once the standard noninvasive monitors were applied and all supplies were available, fentanyl ten mcg was given through the midline catheter and flushed with albumin 5%. The patient's alertness was unaffected, and another bolus of fentanyl five mcg was administered. Due to the small catheter size, the infusion pumps would alarm with each medication administration through the single midline. The decision to add another intravenous line was made. A 24 g catheter was inserted in the right saphenous vein, and at this time, the patient seemed unaffected by the total dose of fentanyl 15 mcg. A third dose of fentanyl five mcg was given through the newly placed peripheral intravenous line, again with no effect. Another dose of fentanyl five mcg was given, and at this point, propofol ten mg was given. The patient became less alert, and rocuronium ten mg was given to start the procedure. Shortly after the beginning of the procedure, the patient's SpO_2 decreased from 94% to 77%. Ventilator settings were adjusted to increase the FiO_2 from 0.50 to 0.70. The patient returned to a SpO_2 of 94%.

The patient had malrotation with a dilated terminal ileum and obstruction into the cecum. The surgeon noted many peritoneal bands from the colon over the duodenum. The bands were divided, the appendix was removed, and the mesentery secured. While the bands were divided, and within 15 minutes of giving the rocuronium, the patient's respiratory rate increased from 40/min to 72/min, and the heart rate increased from 150/min to 175/min. Fentanyl ten mcg was given, followed by rocuronium 5 mg, and the fentanyl infusion was increased from 1 mcg/kg/hr to 5 mcg/kg/hr. Despite another dose of fentanyl 15 mcg at the end of the procedure, the patient's respiratory rate remained at 72/min, heart rate 170/min, FiO_2 0.70, and SpO_2 98%. The patient was recovered by NICU nurses in the procedure room and was extubated that evening.

Discussion

Premature patients are at increased risk for underestimated pain levels, leading to inadequate analgesia management, which can progress to adverse outcomes related to health, behavior, pain processing, and cognition throughout life.[1,2] As the brain develops, the processing of somatosensory information can be altered, especially when an infant is exposed to multiple painful procedures. While mechanical ventilation is described as a discomfort that is often alleviated with sedation, painful stimuli in neonates can be categorized into mild, moderate, and acute. Mild stimulus includes artery puncture, venipuncture, and injections. Moderate stimulus includes heel puncture, lumbar puncture, and bronchial toilet. Acute stimulus includes surgical interventions and application of drainage devices.[3]

During the postnatal period, pain sensory fibers are still developing, and the experienced painful stimuli can affect the number of fiber types, leading to hypersensitivity and allodynia later in life.[1] Extremely preterm infants are at particular risk since lower gestational age children have a lower threshold and higher sensitivity to pain.[3] Early exposure to prolonged pain can alter pain processing, leading to a reorganized structure and function of the nervous system. Health conditions such as diabetes and hypertension, behavior disorders such as depression and anxiety, and cognition issues such as reduced brain volume and memory deficits are associated with neonatal pain.[1]

The difficulty of assessing premature infants' pain, coupled with a lack of information regarding neonates and narcotics, results in the complexity of neonatal analgesia. Neonates frequently

exposed to noxious stimuli can process this at the somatosensory cortex level with unnoticeable behavioral changes.[1] Additionally, pain assessment in the NICU is highly variable and occurs infrequently. An association between an earlier gestational age and less pharmacologic and non-pharmacologic interventions for painful procedures puts neonates at an increased risk for inadequate analgesia management.[2]

Not only is assessment of premature infants' pain lacking, but, often, the pharmacodynamics and pharmacokinetics of medications are not widely understood in neonatal populations, causing information to be extrapolated from children and adult responses to medications.[4] Due to a lack of evidence from the literature, it is challenging to provide dosage advice for medications in neonates. The infant in the case study was on a fentanyl drip of 1mcg/kg/hr, and studies show that plasma concentrations become stable at day 15 of postnatal age. From postnatal day ten onwards, the fentanyl loading dose is twice the maintenance dose, which leads to a quicker increase in plasma concentration and can assist in an abrupt effect of fentanyl.[5] Since there was a preexisting fentanyl 1mcg/kg/hr infusion, an appropriate loading dose would have been fentanyl 6.73 mcg for this patient. Translating this amount to an appropriate bolus dose before the procedure started would result in this patient receiving over five times the appropriate loading dose, with what appeared to be no effect. Over time, this tolerance could have contributed to the substantial increase of opioids required.[5] Additionally, the increased volume of distribution in neonates has been attributed to a reduced degree of respiratory depression after fentanyl administration, which has been seen in fentanyl doses as high as ten mcg/kg.[6]

The potential consequences of fentanyl exposure must be compared to the significant comorbidities from untreated pain. Fentanyl exposure as a neonate is associated with motor delays at five years old in a dose-dependent manner.[7] Although pain can cause behavior disorders such as depression, neonates exposed to less fentanyl experienced more depressive symptoms at five years old compared to same-age children who received more fentanyl in the NICU.[1,7]

Non-pharmacological interventions have been studied to decrease the potential consequences of narcotic exposure, while providing pain relief during mild and some moderate intensity painful procedures. During these procedures, breast feeding and the use of 25% sucrose to calm the neonate was found statistically significant when compared to distilled water, non-nutritive suckling, rocking, and no intervention.[8]

The difficulty of assessing pain in neonates, combined with the poorly understood effect of narcotic medication, makes neonatal pain treatment challenging. It is essential to anticipate and recognize neonatal pain and promptly treat it to decrease the likelihood of adverse consequences later in life. Utilizing non-pharmacological interventions could mitigate mild to moderate pain during procedures, helping those with lower pain thresholds, while narcotic interventions could remain as the primary response used for acute pain procedures, addressing the concerns of untreated pain.

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A Case Study of Uvula Necrosis

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Keywords: Uvula necrosis, intubation, ETT, globus sensation, sore throat

Airway management with endotracheal intubation is routinely used in general anesthesia. Commonly discussed patient risks of airway device placement include injuries to the lips and teeth, but rarely are soft tissue and airway trauma discussed. Uvula injury and necrosis are rare complications that arise from tissue compression caused by airway devices, suctioning, or medical instruments.[1] A 2019 literature review found 53 postoperative uvula necrosis case reports with various patient demographics and associated devices spanning a forty-year period.[4]

Case Report

A 40-year-old 74 kg, 162 cm, female presented for a bilateral mammaplasty reduction and abdominoplasty under general anesthesia. Pertinent past medical history included anxiety, hyperlipidemia, macromastia, and diastasis recti with allergies to latex with a moderate urticaria reaction. Prior surgical history was two cesarian sections and an umbilical hernia repair; the latter was associated with postoperative nausea and vomiting. The pre-operative physical exam had no abnormal findings. Airway assessment showed Mallampati II with a long uvula, mouth opening of approximately 6 cm, normal dentition, and normal neck range of motion. The patient was premedicated with scopolamine 1 mg transdermal patch, aprepitant 40 mg capsule by mouth, and 2 mg midazolam intravenously (IV) for anxiety before transport to the operating room.

Standard noninvasive monitors were applied in the operating room, and the patient was pre-oxygenated at 10 L/min via facemask for three minutes prior to induction. General anesthesia was induced with IV fentanyl, lidocaine, propofol, and rocuronium. Once the patient became unconscious, mask ventilation was attempted, and a 90 mm oral airway was required for adequate ventilation. Mask ventilation with an oral airway was performed for two minutes. Direct laryngoscopy with a Macintosh size 4 blade showed a Cormack-Lehane grade 2a view. The trachea was then easily intubated with an un-styleted 7.0 mm cuffed endotracheal tube in one attempt. Placement of the endotracheal tube was confirmed by bilateral breath sounds and capnography, with a depth of 21 cm at the teeth. The endotracheal tube was secured midline in the oral cavity with tape across the maxilla. Mechanical ventilation was initiated via volume control and synchronized intermittent mandatory ventilation modes. General anesthesia was maintained with sevoflurane at 1.6% exhaled concentration in O_2 at 0.35 L/min and air at 1.65 L/min. The teeth and soft tissues were visually inspected for trauma or injury; none were noted, and atraumatic intubation was documented in the airway note. An esophageal temperature probe was placed through the oral cavity into the esophagus. The patient then underwent bilateral mammaplasty reduction and abdominoplasty under general anesthesia. A phenylephrine infusion was initiated and titrated to meet the surgical team's mean arterial pressure goal of greater than 100 mm Hg.

After the bilateral mammaplasty reduction and abdominoplasty were completed, the patient's neuromuscular blockade was antagonized with sugammadex, and inhaled anesthetics were discontinued. A 90 mm oral airway was placed, and the oropharynx was blindly suctioned with a bulb tip yankauer suction at 200 mm Hg wall suction until no further secretions were removed. The patient followed commands, met extubation metrics, and was extubated.

After arriving in the post-operative recovery unit (PACU), the patient denied any pain or nausea. Before discharge from the PACU, the patient complained of a "loogie" (a build-up of phlegm or saliva in the throat) sensation in her throat that she could not swallow. The patient denied any pain but reiterated the feeling of the need to try to swallow something large. Examination of the oropharynx revealed an erythematous and congested uvula tip. There was a discussion with the surgeon and the patient about the possibility of uvula edema, which could progress to uvula necrosis. The care team worked with the patient to devise the best course of action; the plan was to use over-the-counter analgesics for discomfort, observe symptoms, and notify the surgical team of worsening symptoms.

On postoperative day three, the patient was phoned for follow-up on their symptoms. The only postoperative complaint was a globus sensation in the back of the throat that caused gagging and throat discomfort, and the uvula tip had a very dark appearance. An otorhinolaryngologist was consulted and recommended a methylprednisolone 4 mg oral steroid pack, which was then prescribed to the patient. On postoperative day seven, the patient reported via phone call that the tip of the uvula had necrotized and fallen off, resulting in complete relief of globus sensation and throat discomfort.

Discussion

Uvula necrosis is a rare airway complication primarily discussed in case studies. A possible etiology for uvula necrosis includes mechanical compression of the vasculature to the uvula, resulting in ischemia.[2] Mechanical compression can come from endotracheal tubes, laryngeal mask airways, endoscopes, vigorous suctioning causing mechanical damage, fiber optic scopes, and bronchoscopes.[5] Uvula necrosis risk factors include male gender, relaxation of muscles and soft tissues during anesthesia, airway devices residing midline after placement, multiple devices present in the oropharynx, and a long uvula.[4]

In this case study, modifiable risk factors included taping the endotracheal tube to the midline and upon emergence, placing an oral airway next to the endotracheal tube, and blind suctioning performed with a high degree of suction power. One patient-specific risk factor in this case study for uvula necrosis was having a long uvula. Proposed techniques to avoid uvula necrosis include positioning endotracheal tubes or oral instrumentations like an endoscope to either side of the mouth, decreasing suction intensity, and avoiding blind suctioning in the oropharynx.[4] Some symptoms of uvula necrosis are sore throat, dysphagia, globus sensation, odynophagia, dyspnea, and gagging.[1] The patient, after fully recovering from anesthesia and discharging from the PACU, exhibited a sore throat, globus sensation, odynophagia, and gagging. Uvula necrosis is diagnosed through clinical examination of the oropharynx and symptom correlation.[1] Physical examination of the patient revealed an erythematous and congested uvula tip.

For most patients, uvula necrosis resolves within fourteen days.[4] In those cases, the necrotic tissue sloughs off.[4] There is no standard treatment plan; antibiotics are not warranted, and surgery to remove the necrotic tip does not accelerate relief.[4] Supportive treatments for uvula necrosis include acetaminophen, oral steroids, antihistamines, and topical epinephrine to the uvula.[3] Our patient was encouraged to take acetaminophen for pain, but their chief complaint was a globus sensation and gagging from the elongated uvula. With consultation of an otorhinolaryngologist, a methylprednisolone 4 mg oral steroid pack was added and provided temporary relief until the necrotic portion was sloughed off.

Lessons learned from this patient include increased awareness of the location of airway devices, suctioning intensity, and vigor. Personal practice changes in the informed consent process include elaborating on airway risks beyond injury to the lips or teeth. This elaboration could serve as a disclaimer that any part of the airway is at risk for unintended injury, from the lips to the vocal cords. Personal practice changes during airway management include increased vigilance regarding tube positioning and ensuring the tube is entirely on either side of the tongue

and not just moving diagonally from the mouth to the posterior oropharynx. A final personal practice change includes increased attentiveness to the vacuum intensity set on the suction device. The airway suctioning vacuum intensity should be below 200 mm Hg for adults and 80 to 120 for neonates.[6] This case outcome was benign with no lasting patient harm but highlighted how small, seemingly insignificant details like tube securement location or suction intensity can have real consequences.

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Moderate Sedation in a Patient with Systemic Sclerosis

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Keywords: systemic sclerosis, scleroderma, pulmonary, mouth opening, anesthesia management, moderate sedation

Systemic sclerosis or scleroderma (SSc) is a rare autoimmune disorder of connective tissues with an incidence of 10-50 new cases per million people per year.[1] SSc causes systemic inflammation, severe fibrosis, and vascular injury. The classic presentation of symptoms involves thickening or hardening of the skin and Raynaud's syndrome in the extremities. Disease progression can evolve to include severe pulmonary disease, as well as alterations in the gastrointestinal, renal, cardiovascular, and musculoskeletal systems.[2] This multisystem involvement creates many challenges for anesthesia professionals who must manage these patients in the perioperative environment.

Case Report

A 51-year-old female weighing 53 kg presented to the endoscopy lab to undergo a colonoscopy for colorectal cancer screening. Anesthesia was consulted to perform moderate sedation due to the patient's complex past medical history (PMH). Her PMH included SSc, Raynaud's, and acro-osteolysis controlled with mycophenolic acid, diltiazem and vitamin D3. Her PMH also included pulmonary hypertension, controlled with sildenafil, gastroesophageal reflux disease (GERD), controlled with esomeprazole, and interstitial lung disease (ILD) uncontrolled with medications. Lastly, the patient also reported taking tramadol as needed for pain.

A comprehensive chart review was performed prior to the procedure. The patient's pulmonary function test (PFT) showed a restrictive pattern with a forced vital capacity (FVC) of 46% of the predicted value, forced expiratory volume in 1 second (FEV1) of 45% of the predicted value, with an FEV1/FVC ratio of 0.77. The PFT also showed a severely limited total lung capacity of 2.37 L, which is 47% of the predicted value. The six-minute walk test portion of her PFT had to be aborted due to extreme shortness of breath after approximately 4 minutes of walking, with desaturations to 90% on room air. Her most recent echocardiogram showed a normal ejection fraction of 60-65% with mild left ventricular hypertrophy and an estimated pulmonary artery systolic pressure of 33 mm Hg, indicative of mild pulmonary hypertension. Computerized tomography (CT) of the chest showed basilar predominant honeycombing and traction bronchiectasis compatible with scleroderma-related interstitial lung disease in the usual interstitial pneumonia pattern. The CT chest also showed a dilated distal esophagus containing frothy material, suggesting esophageal dysmotility secondary to scleroderma. A maxillary CT scan showed a pathologic fracture of the left body and ramus of the mandible with near complete fragmentation extending to the left condyle with loss of cortical continuity for which the patient was being evaluated by Oral and Maxillofacial Surgery for surgical repair.

Upon arrival to the preoperative area, her baseline vital signs showed a heart rate (HR) of 75/min, blood pressure (BP) of 116/57 mm Hg, SpO_2 of 97% on room air, and a respiratory rate (RR) of 21/min. The patient's airway exam revealed a mallampati score of 4. She had a limited mouth opening with a maximal incisor opening (MIO) of 2.5 cm and exhibited pain due to her jaw fracture, and limited neck mobility. While the patient did have a history of both GERD and esophageal dysmotility, she did not have any symptoms on the morning of the procedure.

In the endoscopy suite, the patient was placed on standard noninvasive monitors, high-flow nasal cannula (HFNC) was applied with O_2 delivered at 35 L/min. Anesthesia was induced by administering 2 mg of midazolam and 10 mcg of dexmedetomidine as intravenous boluses. Additional doses of 5 mcg of dexmedetomidine were administered as needed to maintain patient comfort, totaling 25 mcg. The procedure duration was 17 minutes, with the patient remaining hemodynamically stable and ventilating spontaneously throughout. After the procedure, the patient was transitioned from HFNC to a nasal cannula with O_2 at 2 L/min. In the recovery area the patient had an extended observation period of one hour to monitor her ventilation status. Upon discharge from the post-anesthesia care unit, the patient's vital signs were HR 68/min, BP 94/69 mm Hg, RR 19/min, and SpO_2 of 96% on room air.

Discussion

The systemic effects of SSc impact anesthetic care throughout the perioperative phase of care. Due to Raynaud's and a decrease in vascularity caused by fibrosis, SpO2 and noninvasive BP monitoring are often inaccurate.[3] As a result, an arterial line may be necessary for accurate BP measurements during procedures. Finger pulse oximetry was challenging due to the patient's history of Raynaud's and acro-osteolysis which resulted in amputation of all fingertips. To combat this problem, an ear probe was placed, and accurate pulse oximetry was obtained throughout the case.

Airway management is a common problem in patients with SSc due to facial deformities, difficult mask ventilation, small mouth opening, and decreased cervical spine mobility.[3] Maximal incisor opening has been directly correlated with disease severity in patients with SSc, with an MIO of less than 3 cm indicative of severe systemic disease.[4] The patient had an MIO of 2.5 cm due to a combination of tight skin and temporomandibular joint dysfunction resulting from facial bone resorption due to decreased vascularity, ischemia, and pressure necrosis.[5] Therefore, the decision was made to avoid endotracheal intubation altogether and proceed utilizing a HFNC delivering 100% FIO_2. The benefits of a HFNC included not only increasing the patient's PaO_2, but also providing an extended safe apneic time in the event the patient stopped spontaneously ventilating and required intervention. In the event of an airway emergency, medications for a rapid sequence induction, a video laryngoscope, and a fiberoptic scope were prepared and placed in the room.

Although the most visually apparent complications of SSc involve the skin and musculoskeletal systems, the most common life-threatening manifestations tend to be within the cardiopulmonary system. Fibrosis of the vasculature begins in the arterioles and microvascular system but then, with disease progression, affects larger vessels.[2] This leads to autonomic instability, progressive congestive heart failure, decreased cardiac reserve, and arrhythmias.[6] The release of fibroblasts also leads to fibrosis of the lungs, causing ILD, which is the leading cause of mortality in patients diagnosed with SSc.[6] Patients with SSc are often found to have restrictive lung disease, decreased lung capacities on PFTs, and non-specific interstitial pneumonia on chest radiography.[7] In addition to restrictive lung disease, this patient was also diagnosed with mild pulmonary hypertension, further increasing her risk of intraoperative complications.

All attempts should be made to avoid endotracheal intubation in patients suffering from severe SSc with ILD due to the correlation between mechanical ventilation and increased mortality in this patient population.[6] Regional, neuraxial, and sedation have been found to be the safest methods to administer anesthesia.[6] If intubation is necessary for the procedure, the anesthetist can expect increased airway pressures, decreased lung compliance, and difficulty maintaining oxygenation.[3] In addition, it is estimated that up to 90% of patients with SSc suffer from GERD and are at an increased risk of gastric aspiration, meaning that a rapid sequence induction should be considered.[6] This presents a unique challenge due to the high likelihood of difficult airway placement. As a precaution, it is recommended to pretreat all patients with a histamine-2 (H2) blocker preoperatively to decrease aspiration risks.[6] An H2 blocker was not administered preoperatively to this patient and, in retrospect, should have been given, especially with her history of GERD and esophageal dysmotility. Another consideration should be surgical

positioning. Due to skin thinning, fibrosis, and a decrease in subcutaneous fat, these patients are at risk of pressure-induced skin breakdown.[6] While this was a short endoscopic procedure in the lateral position, careful positioning and padding should be utilized for all surgical procedures.

The decision was made to perform the case under moderate sedation to keep the patient spontaneously ventilating to avoid the risks associated with intubation during general anesthesia. It was determined that the risks of possible patient discomfort during the procedure far outweighed the risks associated with general anesthesia. While this case was performed safely and effectively with sedation, great caution should be taken while anesthetizing patients with SSc to minimize complications in this patient population.

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The Pediatric Patient with Severe Obstructive Sleep Apnea

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Keywords: pediatric anesthesia, obstructive sleep apnea, obesity, adenotonsillectomy

The incidence of obstructive sleep apnea (OSA) among pediatric patients is approximately 1-5.8% but is growing with the increase in rates of childhood obesity (5.6% girls and 7.8% boys).[1] Though the risk factors for pediatric OSA can be multifactorial, such as craniofacial abnormalities and obesity, the main culprit is hypertrophy of adenoids and tonsils.[1-2] The first

line treatment for pediatric OSA includes adenotonsillectomy (AT),[2] which warrants an understanding of pediatric OSA classifications and anesthetic considerations for this patient population.

Case Report

An 11-year-old female (92.4 kg, 150 cm, body mass index 36.5 kg/m^2) presented to the pediatric intensive care unit (PICU) for symptoms of severe OSA. Results of the patient's sleep study showed an apnea-hypopnea-index (AHI) of 136.5 events per hour, with a SpO_2 of 89% for 165 minutes and a nadir SpO_2 of 61%. Upon her clinic visit, the patient was directly admitted to the PICU, due to the results of her sleep study. The patient endorsed sleeping at home in a recumbent position with three pillows and waking up several times during the night. The patient had no known allergies and no prior surgical history. Past medical history included OSA and obesity. During her admission to the PICU, a bi-level positive airway pressure was used for sleeping with a setting of 10 cm H_20 inspiratory positive airway pressure and 5 cm H_20 expiratory positive airway pressure, to maintain SpO_2 above 90%. A venous blood gas showed: pH 7.38, PCO_2 46 mm Hg, PO_2 105 mm Hg, and HCO_3 27 mm Hg. Laboratory results showed elevated triglycerides and LDL, with a normal thyroid panel.

On hospital day two, the patient was scheduled for an AT. Pre-operative airway examination showed a Mallampati score of one, hypertrophied tonsils, full range of motion in neck and jaw, and normal thyromental distance. Once in the operative room, the patient was given supplemental O_2 8 L/min with a high-flow nasal cannula. The patient was placed on standard noninvasive monitors and given intravenous (IV) midazolam 2 mg through a pre-existing intravenous catheter. The patient was positioned in reverse Trendelenburg and a ramp was created with blankets to align the oral, pharyngeal, and laryngeal axis. The patient was switched to a standard anesthesia facemask with a flow of O_2 at 8 L/min until a fraction of end-expiratory O_2 above 80% was obtained. The patient was induced with IV lidocaine 100 mg and IV propofol 350 mg. Video laryngoscope was used for a grade one view and intubation was successful on the first attempt.

Pressure control mechanical ventilation was initiated with settings of 18 respirations/min, 12 cm H_2O inspiratory pressure, 5 cm H_2O positive end-expiratory pressure, and FiO_2 of 50%. The patient was maintained on sevoflurane for the duration of the case and received IV dexamethasone 10 mg, phenylephrine 100 mcg, acetaminophen 650 mg, dexmedetomidine 24 mcg, and ondansetron 4 mg. Before extubation the patient was positioned upright, had return of gag reflex and was able to follow simple commands.

The patient was transported to the PICU with a facemask on O_2 6 L/min. She did not require supplemental oxygen while awake, but she was placed on O_2 2 L/min via nasal cannula while sleeping and maintained a SpO_2 above 90%. Post-operative pain was controlled with oral acetaminophen 650 mg and ibuprofen 400 mg. The patient maintained a SpO_2 above 96% without O_2 during the day and while sleeping at night on postoperative day 2 and was discharged home on postoperative day 3. The 6-week follow-up clinic visit noted a resolution of nocturnal symptoms and no requirement for supplemental oxygen during sleep.

Discussion

The 2012 American Academy of Pediatrics (AAP) guidelines define childhood OSA as a "disorder of breathing during sleep characterized by prolonged partial upper airway obstruction and/or intermittent complete obstruction that disrupts normal ventilation during sleep and normal sleep patterns." Preoperative screening for OSA should include a history of signs and symptoms such as frequent snoring (> 3 nights a week), labored breathing during sleep, sleeping in a seated position, daytime sleepiness, attention deficit, and learning problems.[2] Also, a physical exam must note the patient's weight, size of tonsils/adenoids, and any anatomical disfigurements.[4-5] The gold standard for diagnosis of OSA is by an overnight in-laboratory polysomnography (PSG).[2] Clinical practice guidelines recommend PSG for children with obesity, Down syndrome, craniofacial abnormalities, sickle cell disease, mucopolysaccharidoses, or children under the age of two.[6] The results from the PSG are then used to calculate an AHI, which categorizes OSA as mild (1-5), moderate (5-10) or severe (>10).[2]

The first line of treatment for treating OSA in children with AHI >10 or children with hypertrophy adenoids and tonsils is an AT.[1] Research has shown that children with severe OSA have an increased risk of complication during induction, and postoperatively.[3] Therefore, it is imperative to follow current guidelines to ensure patient safety and prevent adverse events. The patient in discussion is at an increased risk for desaturation during induction due to her obesity and the severity of OSA. Research shows that patients with severe OSA were more likely to need positive airway pressure, oral or nasal airway adjuncts, or emergent intubations during induction.[3] To mitigate risk, preoxygenation of patients with a continuous positive airway pressure or non-invasive positive pressure ventilation to tolerate periods of apnea has shown benefit.[5]

Intraoperative considerations should include the administration of dexamethasone, and the use of non-opioid anesthetic.[5-7]A one-time dose of IV dexamethasone 0.25-0.5 mg/kg (maximum dose of 10mg) has shown to help with prevention of nausea and vomiting and improve pain scores in the first 24 hours.[6] Intraoperative administration of ketamine, dexmedetomidine, nonsteroidal anti-inflammatory drugs (NSAIDs), and/or acetaminophen has shown to be suitable alternatives to opioids and effective in pain control for AT.[7] For children with severe OSA, a non-opioid anesthetic has decreased demand for postoperative opioids without an increase in the occurrence of severe pain.[7] Research shows that repeated episodes of desaturations alter the mu receptors and decrease the analgesic demand.[8] Therefore, if opioids are indicated, the standard dose should be reduced by one-third to one-half.[8] The patient's actual weight may grossly overdose the patient, therefore, ideal body weight was utilized to determine the appropriate dose for all medication administered.[6] Furthermore, extubating awake and in the upright position was deemed most appropriate for the patient due to her body habitus and increased risk of airway obstruction.[1,5]

Obstructive sleep apnea persists in about 20-75% of children after an adenotonsillectomy, especially in patients who are young, obese or have chronic asthma.[1] Risk for postoperative complications for OSA patients include respiratory depression which can result in readmission to higher level care or death. Therefore, current guidance recommends overnight postoperative

monitoring for patients under 3 years old or those who have severe obstructive sleep apnea.[4] The patient must be monitored on pulse oximetry and demonstrate the ability to maintain adequate oxygenation on room air while awake and asleep before discharge to an unmonitored setting.[5] In children, benzodiazepines and other sedative medications should be avoided due to the increased risk of respiratory depression and airway obstruction.[4] Instead, the guidance recommends the use of ibuprofen and acetaminophen for pain control.[5]

The rising rate of obesity among pediatric patients has brought new challenges to the anesthetic management of patients undergoing adenotonsillectomy for obstructive sleep apnea. To ensure the best outcome for this patient, anesthesia practitioners are recommended to avoid deep extubation, use ideal body weight for medication dosing, and administer non-opioid analgesia, steroids, and antinausea prophylaxis.

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The Use of Angiotensin II for Post-Cardiopulmonary Bypass Hypotension

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Keywords: angiotensin II, cardiac surgery, vasoplegia, cardiopulmonary bypass, vasodilatory shock

Vasoplegia is a common outcome following cardiopulmonary bypass and is seen in upwards of 37% of patients following cardiac surgery.[1] While traditional treatments using catecholamines and arginine vasopressin can be useful short-term, they can often lead to undesired side effects. Angiotensin II (AT-II), initially approved by the Food and Drug Administration (FDA) in 2017, is a synthetic human peptide used as a tertiary treatment for vasodilatory shock.[2] The aim of this case report is to examine the effects of Angiotensin II as a treatment for post-cardiopulmonary bypass hypotension.

Case Report

A 78-year-old male (76.9 kg) with severe mitral valve regurgitation and multivessel coronary artery disease (CAD), underwent a coronary artery bypass graft (CABG) times two with mitral valve replacement, tricuspid valve repair, and left atrial appendage exclusion. Significant medical history included nonrheumatic mitral valve regurgitation, acute diastolic congestive heart failure, hypertension, multivessel CAD, moderate pulmonary hypertension, and dyspnea on exertion. Pre-induction arterial line with non-invasive monitors were applied, in addition to a pulmonary artery catheter placed for hemodynamic monitoring throughout the case. An initial cardiac output of 3.24 L/min, cardiac index of 1.67 L/min/m^2, and systemic vascular resistance (SVR) of 1,456.79 dynes/sec/cm^{-5} were obtained before surgical intervention.

Before cardiopulmonary bypass (CPB), a phenylephrine infusion was initiated at 40 mcg/min to maintain adequate cerebral oximetry as well as mean arterial pressure (MAP) within 20% of baseline. With initiation of CPB, the phenylephrine infusion was stopped, and norepinephrine was started at 2 mcg/min. After separation from the CPB circuit, epinephrine was initiated at 4 mcg/min, norepinephrine was increased to 6 mcg/min, and milrinone was started with a bolus of 2,000 mcg and then an infusion at 0.25 mcg/kg/min. Inhaled nitric oxide was also started preemptively for elevated pulmonary artery pressures and moderate pulmonary hypertension. Cardiac output and index were obtained after CPB separation and calculated to be 5.6 L/min and 2.88 L/min/m^2, respectively. Hypotension continued with a blood pressure (BP) of 45/33 mm Hg, MAP 39 mm Hg, heart rate of 86/min, and SVR 471.43 dynes/sec/cm^{-5}. Reinitiating CPB was discussed with the surgeon but determined unnecessary at the current time. After 20 minutes of continuous infusions, with increasing norepinephrine rates, a vasopressin infusion was also started at 0.04 units/min. After an additional 30 minutes, the BP remained around 91/59 (65) mm Hg. The decision was made to initiate angiotensin II at 5 ng/kg/min, eventually titrating upwards of 20 ng/kg/min over several minutes. At this time, the norepinephrine infusion was decreased to 4 mcg/min. The chest was closed, vital signs stable, and the patient was taken to the intensive

care unit on the following vasopressor requirements: epinephrine 4 mcg/min, norepinephrine 4 mcg/min, milrinone 0.25 mcg/kg/min, vasopressin 0.04 units/min, and angiotensin II 20 ng/kg/min.

The patient reportedly self-extubated later that evening, with no respiratory complications and was placed on O_2 4 L/min via nasal cannula. Invasive lines and catheters were removed in the following manner: arterial line post-operative day (POD) 1, mediastinal chest tubes POD 1, foley catheter POD 6, pulmonary artery catheter POD 6, and subclavian central venous line POD 7. Several hours after open heart surgery on the same day, the angiotensin II and vasopressin infusions were able to be titrated off with the patient's vital signs remaining hemodynamically stable. The remaining vasopressor infusions were discontinued in the following order: epinephrine POD 2, milrinone POD 3, and norepinephrine POD 4. A complete 2D echocardiogram was performed on POD 6, showing an ejection fraction similar to preoperative studies with no regurgitation or stenosis of the mitral and tricuspid valves. On POD 7, the patient's heart rhythm converted to atrial fibrillation remaining hemodynamically stable and was later discharged that day. A foley catheter remained in place for urinary retention. Follow up visits were scheduled with cardiothoracic surgery, cardiology, and urology in the upcoming weeks.

Discussion

Vasoplegic shock is a well-documented occurrence in which a patient experiences hypotension and decreased systemic vascular resistance (SVR) despite increasing vasopressor requirements after separation from CPB, in the absence of other shock states. While the underlying physiology behind vasoplegic shock is not fully understood, several risk factors include beta-blockers, amiodarone, heparin, calcium channel blockers, angiotensin-converting-enzyme inhibitors, and CHF.[3] Additional factors include systemic inflammatory response, duration of CPB, contact with foreign componenets of the CPB circuit, and long cross clamp times. Conventional treatment modalities relied heavily on catecholamine infusions (i.e., norepinephrine and epinephrine) which progressed to hydroxocobalamin and methylene blue. Methylene blue has a very limited use in cardiac surgery due to its increased morbidity found in several studies.[3] Vasoactive medications are not without the increased risks of further organ damage, tachyarrhythmias, ischemia, and necrosis. Despite the current medical interventions mortality rates remain greater than 40%.[3]

In 2017, the FDA approved Angiotensin II, the first synthetic human peptide and potent vasoconstrictor, for the use in refractory hypotension due to shock states.[4] The renin-angiotensin-aldosterone system (RAAS) regulates blood pressure, electrolyte balance, fluid homeostasis, and glomerular filtration rate throughout the body.[5] When a decrease in blood pressure is identified, renin is released activating the RAAS cascade. Renin converts angiotensinogen into angiotensin I, which is then converted to angiotensin II by the angiotensin-converting enzyme (ACE) in the lungs.[6] Angiotensin II for the Treatment of High-Output Shock 3 (ATHOS-3) is the landmark study which determined the clinical efficacy of angiotensin II. To date, it is the largest randomized, double-blind, placebo-controlled clinical trial to determine the utilization of

angiotensin II in shock states.[6] In the ATHOS-3 study, patients receiving angiotensin II achieved faster rates of MAP target ranges and lower catecholamine requirements within the first several hours of therapy.[3] It was also determined that the overall 28 day mortality rate was decreased in the angiotensin II group, as compared to the placebo group.[3] In addition, patients with acute kidney injury requiring renal replacement therapy (RRT) had an increased survival rate with a higher rate of RRT discontinuation by day 7, while on angiotensin II.[3]

Initiation of angiotensin II has a recommended starting dose of 20 ng/kg/min, with a maximum dose of 80 ng/kg/min. AT-II is metabolized by ACE and aminopeptidase A primarily in the plasma and erythrocytes. The plasma half-life of angiotensin II is predicted to be less than 1 minute and is not affected by either hepatic or renal function.[4] Overall, the effects of AT-II are to increase BP by potent vasoconstriction of the peripheral vessels and to increase aldosterone synthesis. AT-II and its catecholamine-sparing effects may lessen the side effects seen with high dose vasopressors and can allow for significant decreases in catecholamine usage. While AT-II has helped in the treatment of vasoplegic shock, thromboembolic events have been seen in as high as 13% of patients undergoing cardiac surgery.[4] Since cardiothoracic surgery patients are already at an increased risk of thromboembolic events, this major side effect may be enough reason to avoid AT-II if possible.

Looking back, several factors throughout this case persuaded the use of angiotensin II. The main factor being the propensity of the surgeon and his willingness to use this medication early on, often when only two vasopressors have been initiated. When discussing the initiation of AT-II with the anesthesia and surgical teams, angiotensin II was started before any of the other concurrent catecholamine infusions were maxed out. In addition, the time frame from starting the first catecholamine infusion to AT-II infusion was less than hour, and more time could have been given to see if an effect occurred. By following the patient to the intensive care unit, it was seen that the AT-II infusion was stopped less than five hours after surgery was completed. Needing to ask whether the infusion was needed at all. Additional studies are merited to identify optimal dosing, timing, and implications of angiotensin II in post-cardiopulmonary bypass vasoplegic shock.

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Effect of Magnesium Sulfate on Pediatric Emergence Agitation and Delirium

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Keywords: pediatric, emergence agitation, emergence delirium, magnesium sulfate

Approximately 450,000 children in the United States received surgery each year, and 10 – 80% experienced emergence agitation.[1,2] Volatile anesthetics, such as sevoflurane and desflurane have been linked to increased incidences of emergence delirium.[2] Due to its low blood/gas partition coefficients, non-irritating quality, low cost, rapid onset and rapid offset, Sevoflurane is used in pediatric anesthesia as the volatile of choice in both inhalation induction and maintenance.[1,3,4] However, sevoflurane possessed neuro-excitatory effects and was believed to be strongly associated with emergence agitation and delirium.[4,5] Although emergence agitation/delirium occurrs at any age, it is most prevalent from ages 3 to 9 years old.[1] The pediatric population is defined by the United States Department of Food and Drug Administration (FDA) as encompassing the age ranges between birth and 21 years of age.[6]

Pediatric emergence agitation is the "self-limited state of psychomotor excitement during awakening from general anesthesia".[7] Pediatric emergence agitation is found to be distressing and deleterious for the child, parents, and staff involved.[3] Patients may experience injury ranging from unintentional removal of clinical devices, for example, intravenous catheter (IV), to surgical dehiscence or evisceration. Parents and clinical staff may also suffer from injuries related to this phenomenon. The incidence of emergence agitation places strain on the healthcare system's resources and requires additional intervention and medical personnel to oversee the patient.

Magnesium sulfate is a compound that gained popularity in clinical anesthesia as an adjuvant agent. By blocking N-methyl-D-aspartate (NMDA) receptors from glutamate, magnesium sulfate causes sedation, analgesia, and muscle relaxation.[5] Magnesium sulfate has been shown to

decrease the amount of sevoflurane required to maintain general anesthesia by up to 50%. Studies of magnesium sulfate infusions in adult populations demonstrated a reduced incidence of emergence agitation/delirium.[7,8] Publicized for its relative safety and low cost, magnesium sulfate is a possible solution to attenuate the incidence of emergence agitation and delirium in the pediatric population.

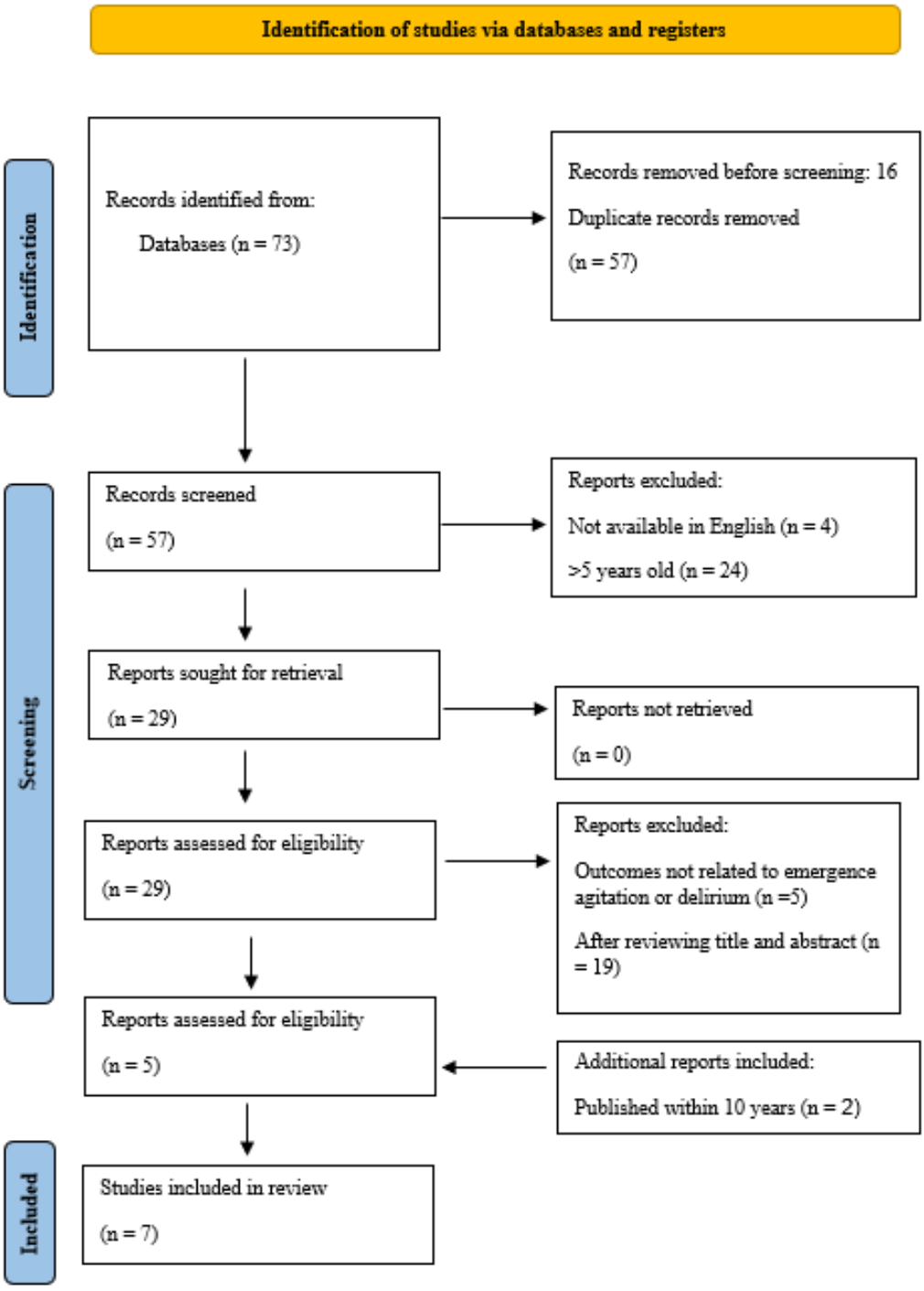
Despite its use in the adult population, a gap in the literature exists about the efficacy of magnesium sulfate infusion in pediatrics. An ambiguity about the delineation between emergence delirium and emergence agitation is also present in current literature. Some experts noted a stark contrast between emergence agitation and emergence delirium.[7] In a study on emergence delirium on veterans, Tolly et al.[1] asserted that emergence agitation occurs when one emerges from anesthesia and progresses to consciousness. On the other hand, emergence delirium is an acute change in consciousness after emergence agitation. However, other researchers found the two terms to be synonymous.[2,9] This inconsistency in definition contributes to the wide variation in pediatric emergence agitation and delirium incidences. This manuscript aims to evaluate the use of magnesium sulfate administration in pediatric patients undergoing general anesthesia and its effect on pediatric emergence agitation and delirium.

Methods

After reviewing the current literature and supporting studies, researchers developed an encompassing PICO question: In pediatric patients who received general anesthesia, does the intraoperative administration of magnesium sulfate infusion, compared to no magnesium sulfate infusion, decrease the incidence of emergence agitation and delirium? Researchers reviewed four databases to identify 73 records pertinent to the PICO question: *PubMed, Cumulative Index to Nursing and Allied Health Literature (CINAHL), Google Scholar, and Medline Complete*. When the researchers conducted the search, the terms emergence, emergence delirium, delirium, emergence agitation, agitation, pediatric, child, children, magnesium and magnesium sulphate [sic], magnesium sulfate, and general anesthesia were used to identify scholarly articles.

After a preliminary search yielded 5 articles published within the last five years, researchers expanded the time frame to 10 years and re-examined 24 publications that were previously excluded. After reviewing the abstract, title and contents of the articles, 2 articles were added for a total of 7 articles used in this review. Inclusion criteria consisted of evidence that utilized magnesium sulfate IV infusions and sevoflurane for general anesthesia, defined the age limit of children that participated in the study, and articles that examined unaccompanied magnesium sulfate infusion as one of the experimental variables in the study. Exclusion criteria pertained to articles that did not use magnesium sulfate infusions, evidence that used non-IV infusion methods of magnesium sulfate administration, articles utilizing mixtures of magnesium sulfate with other medications, studies conducted on procedures not requiring general anesthesia and studies that examined the analgesic use of magnesium sulfate. The following PRISMA flow diagram (see Figure 1) demonstrates the screening process.

Figure 1. PRISMA 2020 Flow Diagram



Literature Analysis

A summary of all studies presented using the Summary of Individual Evidence Table[10] is presented in Table 1. Elsharnouby et al. found statistically significant results in Pediatric Anesthesia Emergence Delirium (PAED) scores of pediatric patients when comparing a control and intervention group. PAED scores at the time of admission, 5 minutes, and 15 minutes recorded a *P<.001*.[12] At 30 and 45 minutes from admission, a *P*-value of 0.04 was documented.[12] Additionally, at 60 minutes, *P*-value = 0.03.[12] However, at 75 minutes, there was no statistically significant decrease in PAED scores with a *P*-value of 0.1.[12] These findings support using magnesium sulfate to decrease emergence delirium.[12]

Abulatif et al.[13] conducted a randomized, controlled, double-blinded study and supported intraoperative magnesium sulfate reduces emergence agitation in the pediatric population. The PAED scores for the intervention group demonstrated a P-value of 0.03 at admission and P<.001 after 60 minutes.[13] These findings support the use of magnesium sulfate for the reduction of emergence delirium.[13]

Bondok and Ali's study tested the effects of intraoperative magnesium sulfate on reducing emergence agitation in pediatric patients. Patients in the intervention group were administered a 20 mg/kg loading dose of 10% magnesium sulfate over 15 minutes and subsequent infusion at a rate of 10 mg/kg/hour. The patient's agitation was assessed on a scale from 1 to 4.[14] The study recorded the number of patients who were agitated along with the duration of the agitation.[14] In the PACU, no emergence agitation was detected in the intervention group (N=0). However, emergence agitation was detected in 11 patients in the control group, *P*<.001.[14] Additionally, the intervention group's duration of agitation was zero, compared to the control group, which had a mean duration of agitation of 8.2 minutes, *P*<.001.[14]

Benzon et al[15] studied the effects of intraoperative magnesium sulfate infusions in children undergoing tonsillectomy. The study showed no statistically significant difference in emergence agitation between the magnesium and control group.[15] Average PAED scores at wake-up between the magnesium and control groups were 9 and 7, *P*=.65, while the control group recorded a lower agitation score versus the magnesium group.[15] PAED scores 5 minutes after waking up, 5 in the magnesium group versus 6.5 in the control group, *P*=.8.[15] And lastly, PAED scores at 30 minutes after wake-up were zero for both groups.[15]

A randomized, placebo-controlled study by Khatiwada et al[16] examined the effects of magnesium sulfate infused intraoperatively on pediatric patients undergoing general anesthesia for hernia and hydrocoele surgery. Agitation was scored utilizing the pain discomfort scale (PDS), and a child was considered agitated if the score exceeded three.[16] The study observed two patients experiencing agitation in the intervention group versus four in the control group.[16] However, this was not statistically significant, *P*=.68.[16]

Table 1: Summary of all studies presented using the Summary of Individual Evidence Table[10]

Citation **Level of Evidence[a]** **Quality Rating[b]**	**N** **Intervention** **Control**	**Findings**	**Comments**
Elsharnouby et al., 2015 Level of Evidence: 1 Quality Rating: A	Overall N: 47 patients Intervention N: 24 patients Control N: 23 patients	Children between ages 3-10 with ASA[c] score of 1 or 2. Reduction in time of discharge (mean) Control Group: 68 minutes Intervention Group: 53 minutes Reduction in PAED[d] score Control N: 9 Patients Intervention N: 3 patients	Randomized, double-blinded, controlled study. The magnesium group received 10% magnesium sulfate 40 mg/kg over 10 minutes, followed by 15 mg/kg/h. The control group received an equal volume of 0.9% sodium chloride. PAED was used to assess agitation. Limited by the lack of measurement of serum magnesium levels.
Abdulatif et al., 2013 Level of Evidence: 1 Quality Rating: A	Overall N: 70 patients (65 completed the study) Intervention N: 35 patients (2 did not receive allocated intervention) Control N: 35 patients (3 did not receive allocated intervention)	Patients between ages 4-7 with ASA score of 1 or 2 Emergence agitation occurred more frequently in the control group versus the intervention group: Control: 23 patients Intervention: 12 patients Children who required rescue fentanyl for post-op pain were similar in both groups: Control: 23 patients Intervention:15 patients	Children in magnesium group receive an intravenous loading dose of 30 mg/kg of a 10% solution over 10 minutes Followed by a continuous dosage of 10 mg/kg/hr, it was turned off at the end of the surgery after the discontinuation of sevoflurane. Children were assessed using the PAED score; a score greater than 10 was documented as agitation. Limited by not maintaining the magnesium sulfate infusion when the patient arrived in the PACU[e], since maintaining a magnesium infusion until complete sevoflurane washout could have yielded a lower agitation score.

Bondok et al., 2014 Level of Evidence: 1 Quality Rating: A	Overall N: 50 patients (42 completed the study) Intervention N: 20 patients Control N: 22 patients	Male children ages 3-6 years old with ASA status 1 or 2. Incidence of Agitation: Intervention Group = 0 Control Group = 11	Randomized controlled trial. Patients underwent inguinal herniorrhaphy. Airway used for all patients was an LMA[f] versus ETT[g] for other studies. Intervention group received a magnesium loading dose 10% magnesium sulfate 20 mg/kg over 15 minutes; infusion: 10 mg/kg/hour. Control group received 0.9% sodium chloride. Pain was evaluated using the Wong-Baker FACES pain rating scale[h]. Children reporting a pain score of more than 2 during recovery were excluded.
Benzon et al., 2015 Level of Evidence: 1 Quality Rating: A	Overall N: 60 patients Intervention N: 30 patients Control N: 30 patients	Patients ages 4-10 years old, with ASA classification I – III undergoing tonsillectomy by a single surgeon. Emergence agitation measured using the PAED score every 5 minutes for the first 30 minutes, then every 15 minutes until 1.5 hours after surgery. Median PAED scores at wake up: Intervention: 9 Control: 7 Median PAED scores 5 minutes after wakeup Intervention: 5 Control: 6.5	Magnesium protocol: loading dose of 30 mg/kg given over 15 minutes; continuous magnesium infusion of 10 mg/kg/hr. Control group received normal saline. Oral midazolam was given as a premedication or parents were present for induction. All patients were tracheally intubated. Maintenance of anesthesia: nitrous oxide with oxygen 1:1 with sevoflurane. Emergence delirium was measured utilizing the PAED scale. Study limited by the patients undergoing one type of procedure such that the results cannot be generalized to a different surgery.

		Median PAED score 30 minutes after wake up Intervention: 0 Control: 0 No significant difference in PAED scores.	
Khatiwada et al., 2020 Level of Evidence: 1 Quality Rating: A	Overall N: 131 patients Intervention N: 66 patients Control N: 66 patients	Children aged 3-12 years old underwent hernia and hydrocele surgery. Agitation was defined as a PDS[i] score greater than 3. Intervention: 2 patients were defined as agitated. Control: 4 patients were defined as agitated. P=0.68	Patients were premedicated with promethazine hydrochloride 0.5 mg/kg. Patients underwent general anesthesia with a LMA. Induction was used using halothane and oxygen. Magnesium protocol: 15 mg/kg was infused at 1 ml/min. Anesthesia was maintained with 1.5 to 2% halothane in oxygen. The control group received normal saline of equal volume. Agitation was assessed when the child opened their eyes spontaneously or started crying or moving. Agitation score was evaluated utilizing the pain discomfort scale (PDS). Limited by the omission of a plasma magnesium level concentration.

Lee YJ et al., 2020 Level of Evidence: I Quality Rating: A	Overall N: 86 patients Intervention N: 42 patients Control N: 44 patients	57.1% of participants who received the magnesium sulfate infusion experienced emergence agitation in comparison to the 77.3% (N=34) in the control group that experienced emergence agitation.	Conducted at Seoul National University Budang Hospital. Patients underwent strabismus or epiblepharon repair surgery and received general anesthesia performed using a sevoflurane inhalation induction, LMA placement, and sevoflurane maintenance. Intervention group received a 10% magnesium sulfate loading dose of 30mg/kg for 10 min then maintenance dose of 10 mg/kg. Control group received 0.9% saline intraoperative infusion. Serum magnesium levels taken at the end of surgery. Emergence agitation evaluated using the PAED scale. Limited by the researcher's failure to describe how the evaluation was conducted and who it was conducted by.
Lee JH et al., 2020 Level of Evidence: I Quality Rating: A	Overall N: 65 patients Intervention N: 32 patients Control N: 33 patients Participating children were randomly administered either a 10% magnesium sulfate or 0.9% saline intraoperative infusion	No significant difference between the control (78.8%) and experimental group (84.4%) in the presence of emergence delirium.	All patients underwent strabismus surgery. Patients received general anesthesia through sevoflurane inhalation induction, LMA placement, and sevoflurane maintenance anesthetic. Intervention group received a 10% magnesium sulfate loading dose of 30mg/kg for 10 min then maintenance dose of 10 mg/kg.

			Control group received 0.9% saline intraoperative infusion. Serum magnesium levels taken at the end of surgery. Patients assessed for preoperative anxiety and then again in PACU using the Yale Preoperative Anxiety Scale and PAED. This study was limited by the omission of detailing who the evaluator was and their credentials before evaluating the participants for emergence delirium. Additional limitations include the inability to generalize findings to other surgeries.

[a] Johns Hopkins Nursing Evidence-Based Practice (JHNEBP) Model Evidence Levels. Level I includes randomized controlled trials (RCTs), systematic reviews of RCTS, and experimental studies. Level II includes quasi-experimental studies, systematic reviews of quasi-experimental studies and RCTS or quasi-experimental studies alone. Level III includes non-experimental studies and qualitative studies; Level IV: Includes clinical practice guidelines and consensus, and authority opinion. Level V includes non-research evidence, experience, case reports, and expert opinion.[11]

[b] JHNEBP Model Quality Ratings. A is high quality. B is good quality; C is low quality or has major flaws.

[c] American Society of Anesthesiologists (ASA) status. 1: healthy patient, 2: mild systemic disease, 3: severe systemic disease, 4: severe systemic disease that is a constant threat to life, 5: a patient who is not expected to survive, 6: organ donor.

[d] Pediatric Anesthesia Emergence Delirium (PAED) Scale. 1: The child makes eye contact. 2: The child makes purposeful movements. 3: The child is aware of their surroundings. 4: The child exhibits restlessness. 5: The child is inconsolable.

[e] Post-anesthesia care unit

[f] Laryngeal mask airway

[g] Endotracheal tube

[h] Wong-Baker FACES pain rating is a pain scale used for children consisting of 6 different drawings of faces that incrementally increase in the amount of pain portrayed. A score of 1 indicates no pain and 10 indicates the worst pain.

[i] Modified Pain Discomfort Scale. Crying, Moving, Agitation evaluated from a zero to two scale. 0: not crying, none, asleep/calm. 1: Crying that is responded to pain, restless moving, mild agitation. 2: Crying that is not responding to comforting, thrashing movement, severe agitation or hysterical.

The randomized control trial performed by Yea-Ji Lee et al[17] indicated that an intraoperative magnesium sulfate infusion correlated, *P*=.046, with a decreased incidence of emergence agitation in pediatric patients, such that only 57.1% of the intervention group experienced emergence agitation in comparison to 77.3% in the control group. This study also identified a discrepancy in the severity of emergence agitation in the first evaluation between the two groups that were statistically significant, *P*=.019. The control had a median PAED score of 15.0 in comparison to the intervention group's median PAED score of 11.5.[17] After 30 minutes from the first evaluation, no participants in either group demonstrated symptoms of emergence agitation.[17]

In contrast, the evidence of the randomized control trial on pediatric patients by Ji-Hyun Lee et al.[18] indicated no difference in emergence delirium between control, 78.8%, and intervention, 84.4%, groups. Researchers of this study also examined the incidence and severity of postoperative pain between the two groups.[18] However, there was no statistically significant difference between the 2 groups, and neither group required rescue medication for pain.[18] Finally, the researchers identified no correlation between preoperative anxiety and the PAED score of the participants, nor did any of the participants experience adverse side effects or prolonged PACU stays.[18]

Conclusion

Evidence from the evaluated articles indicated that the effect of intraoperative magnesium sulfate infusion on pediatric emergence agitation and delirium remains unsettled. Four studies indicated a reduction in the incidence of emergence agitation and delirium,[12-14,17] while three studies found evidence that proved to be statistically insignificant.[15,16,18] Evidence indicated a possible correlation between magnesium sulfate administration and the incidence of emergence agitation and delirium.

Limitations encountered when analyzing each article included the background of the assessor, clinical experience and amount of people assessing the patient. Some studies focused on a single type of surgery while others examined a variety of cases.[12-18]Another limitation cited was not maintaining the magnesium sulfate infusion when the patient arrived in the PACU; maintaining a magnesium infusion until complete sevoflurane washout could have yielded a lower agitation score.[12] Furthermore, studies examining patients undergoing a specific surgery cannot generalize about children undergoing a different type of surgery.

The inquiry into the efficacy of magnesium sulfate infusions in treating pediatric emergence delirium and emergence agitation remains unanswered. It is recommended that more research should be done on the use of magnesium sulfate infusion on pediatric patients. The implication of these studies can help guide anesthesia practitioners to implement protocols at their facilities to help lower emergence agitation. Magnesium sulfate has the potential to improve peri-anesthetic care for the pediatric patient.

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Editorial

I do not publish by theme, but there is definitely a pediatric flavor to this issue! Over half of the articles in this publication involve pediatric cases, ranging from neonates to teenagers, and cover an interesting variety of conditions and management strategies. On that note, I would like to thank all of our pediatric CRNAs for providing such exquisite care to this special group of patients and their families, bridging the gap between clinical anesthesia and emotional support.

Sincerely,

Vicki Callan, PhD, CRNA, CHSE, FAANA
Editor

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GUIDE FOR AUTHORS

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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
___ All matters that are not common knowledge to the author are referenced appropriately
___ Generic names for drugs and products are used throughout and spelled correctly in lower-case
___ Units are designated for all dosages, physical findings, and laboratory results
___ Endnotes, footnotes not used
___ Jargon/slang is absent

Heading
___ Concise title less than 70 characters long (including spaces)
___ Author name, credentials, nurse anesthesia program, graduation date and email are included
___ Three to five **Keywords** are provided

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

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

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

References
___ Adheres to AMA Style format
___ Reference numbers are sequenced beginning with 1 and superscripted
___ References are from anesthesia and other current (within past 8 years) primary source literature
___ Journal titles are abbreviated as they appear in the PubMed Journals Database
___ Number of references adheres to specific item guidelines (1 textbook allowed for case reports only)
___ Internet sources are currently accessible, reputable, and peer reviewed

Transmission
___ The article is sent as a Word document attachment to **INTSJNA@AOL.COM**
___ The file name is correctly formatted (e.g. PedsPain_Smyth_GU_Pearson_5.19.09)
___ Item is submitted by the mentor
___ Subject heading format - ISJNA Submission_submission type_author last name_mentor last name