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Bone Cement Implantation Syndrome
Opsoclonus-Myoclonus Syndrome
Thyroid Hormone Resistance
Hereditary Spherocytosis
Hereditary Angioedema
RSI for Pyloromyotomy
Submental Intubation
Inhalation Burn Injury
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Remote Anesthesia
Cerebral Oximetry
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Nasal Intubation
Encephalocele
Cerebral Palsy
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Front Cover:
Lauren Runnels, RN, BSN, a graduate student enrolled in the Nurse Anesthesia Program at Louisiana State University Health Sciences Center, provides anesthesia care for an infant undergoing surgery for myelomeningoceole repair. The photo was taken by Andrew Pitt, CRNA, MSN.

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Patient Safety and Remote Anesthesia

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Keywords: remote, anesthesia, safety

Anesthesia practitioners are becoming increasingly involved in procedures taking place in locations remote from the operating room. Reports state that 12.4% of all anesthetic care is performed outside of the operating room. Providing care outside of the operating room can pose significant risk to patients. Anesthesia practitioners must be vigilant in order to prevent oversedation with subsequent respiratory distress and hemodynamic instability. Practicing anesthesia outside of the operating room leaves the practitioner with less access to equipment and trained staff to intervene during a crisis. Planning for needed equipment, medication administration and ventilation strategies reduce potential anesthetic complications.

Case Report

An 87-year-old, 165 cm, 64 kg male with a history of non-small cell lung cancer presented for a computed tomography (CT) guided biopsy of a right upper lobe lung tumor. Additional history included hypertension, grade II tricuspid murmur, an enlarged thyroid cartilage with left sided tracheal deviation, and a hiatal hernia repair. His medication regimen included: cyanocobalamin, lorazepam, sertraline, and terazosin. Lab values were within normal limits. A pre-anesthetic evaluation was completed and no significant findings were reported. The anesthetic plan was discussed with the patient, and consent was obtained prior to initiation of the procedure.

In anticipating needs for this 87 year old gentleman, a general anesthetic was planned. As the procedure required prone positioning, the risks of using regional or monitored sedation methods of anesthesia were high. If intubation became necessary, the time needed to secure this airway due to field avoidance could result in harm to the patient.

In preparation for this anesthetic, multiple endotracheal tube sizes, laryngeal mask airways, and a fastrach laryngeal mask airway (LMA North American, San Diego, CA) were in the room, in case difficult ventilation or difficult intubation should arise. All standard monitors were implemented during this case. Continuous observation of capnography and pulse oximetry is vital to ensure a patent airway and adequate ventilation. In addition, emergency medications including a phenylephrine infusion, primed, and programmed for the infusion pump, was prepared.

An 18 gauge peripheral intravenous (IV) access was established, and midazolam 1 mg was administered in the outpatient holding room. The patient was then transported to the procedural area. While the patient remained supine on the stretcher, pre-oxygenation was accomplished and an intravenous induction was achieved with the administration of lidocaine 60 mg, propofol 120 mg, and rocuronium 40 mg. Successful intubation with a 7.5 mm endotracheal tube (ETT) was verified by direct visualization, positive end tidal carbon dioxide, and
bilateral chest rise; the endotracheal tube was secured.

The patient was then positioned prone in the CT scanner in order for the surgical team to gain access to the biopsy site. At this time, working in conjunction with the radiology technician, the patient was slowly moved in and out of the CT scanner to ensure all equipment, intravenous lines, and monitors would extend the needed distance. The ETT and IV line were taped to the moving part of the scanner to allow extra support during movement of the CT table. Maintenance anesthesia of one MAC of isoflurane was sufficient during draping and prepping of the patient; vital signs were stable.

Maintenance anesthesia included one percent isoflurane by means of a ventilator, with intermittent administration of fentanyl for analgesia, and paralysis was maintained with vecuronium. Standard monitors including blood pressure, ECG, ETCO₂, temperature, and pulse oximetry were utilized throughout the case. Due to field avoidance related to positioning and scanning, maintaining paralysis and sedation was a challenge as anesthesia professionals had to exit the room often during imaging.

Due to constant movement of the scanner, it was difficult to monitor the patient’s level of neuromuscular blockade with the peripheral nerve stimulator throughout the procedure. The patient began coughing once the initial puncture of the skin was made. Maintenance anesthesia was increased to 1.5 MAC of isoflurane for this purpose. The train of four was placed on the posterior tibial nerve as the lower extremities were accessible when access to the patient was acceptable. The patient’s blood pressure was labile throughout the case, with a systolic as low as 70, and intermittent administration of phenylephrine was necessary to maintain hemodynamics within 20 percent of the patient’s baseline. A 500 milliliter fluid bolus of lactate ringer’s solution was administered, but the patient remained hypotensive at times. A phenylephrine infusion was initiated during the case to maintain hemodynamic stability. The maximum infusion rate during the case was 80 mcg/min.

At the end of the procedure the isoflurane was discontinued and neuromuscular blockade was antagonized with neostigmine and glycopyrrolate. Due to the general risk of postoperative nausea and vomiting after general anesthesia, ondansetron 4 mg was administered prophylactically. The patient demonstrated adequate respiration and purposeful movement, and the endotracheal tube was removed without complications. The phenylephrine infusion was slowly titrated down throughout emergence, and successfully discontinued upon extubation. Oxygen 6 L/min was administered by face mask, and the blood oxygen saturation was 100%. The patient was transported to outpatient recovery on a stretcher, with pulse oximetry and oxygen support by face mask. A post-operative CT scan was negative for pneumothorax development, and the gentleman was discharged home later that day. No anesthesia or surgical complications were reported.

Discussion

The demand for anesthesia care in a multitude of settings outside of the operating room has surged in recent years. Vigilance in anesthesia is of utmost importance in any setting; however, when anesthesia administered outside of the operating room, extra caution and planning is warranted. In a 2009 study, researchers concluded that severity of injuries for remote locations claims were greater than those associated
with operating room claims. In addition, the ratio of death was almost double in remote anesthesia claims.\textsuperscript{5} Adverse respiratory events are the most common injury in and out of the operating room. However, adverse respiratory events associated with remote locations are double that of occurrences in the operating room. Inadequate oxygenation/ventilation is the most common respiratory event, occurring seven times more frequently in remote locations.\textsuperscript{5} There are a multitude of factors which render delivery of safe anesthesia care in the remote setting difficult. Some of these include inadequate staff/support, unfamiliar environment, deficient resources, and cramped small working spaces.\textsuperscript{5} When complications arise, trained staff and needed equipment may be several minutes away. Planning ahead for potential and unforeseen complications will allow the anesthesia practitioner to be prepared to intervene and maintain patient safety should a crisis surface. Vigilance is required when checking anesthesia equipment, ventilatory monitors, airway equipment and ensuring back up oxygen and suction equipment are working and available.\textsuperscript{6}

The American Society of Anesthesiologists (ASA) updated their official guidelines with regards to caring for patients in non-operating room locations in 2008. These guidelines include eleven essential components to ensure safe anesthesia care:

- A reliable source of oxygen as well as backup supply must be available.
- The anesthesia location must have a reliable source of suction.
- If inhalation anesthetics are utilized, a scavenging system must be adequate and operational.
- Three items must be available in all remote anesthesia locations; a self-inflating hand resuscitator bag which can deliver positive pressure and administer at least 90 percent oxygen, anesthesia drugs, equipment and supplies, as well as monitoring equipment coinciding with ASA standards. In addition, the ASA guidelines elaborate that if inhalation anesthesia is to be utilized, an anesthesia machine which functions to standards employed in the operating room must be at hand.
- Electrical outlets sufficient to supply an anesthesia machine and monitoring equipment are required. Any location designated as a “wet location,” should have isolated electric power or electric circuits with ground fault circuit interrupters available.
- Adequate lighting must be available and a battery-powered light source should be present.
- Ensure sufficient space for necessary equipment and personnel.
- An emergency cart including a defibrillator, emergency drugs and other equipment to facilitate cardiopulmonary resuscitation must be accessible.
- Ensuring appropriately trained support staff is available, and locating a
A reliable means of two-way communication is essential.

- Observing building and safety codes which comply with facility standards should be carried out.
- Lastly, post-anesthesia management including trained staff with appropriate equipment must be on hand for transport and post-operative care.7

Ensuring the offsite anesthesia location was adequately equipped with emergency supplies was the most crucial aspect in anticipation for this case. Unforeseen complications can arise, and proper preparation allows for immediate resolution. Adhering to ASA guidelines will safeguard anesthesia practitioners and ensure adequate provisions are assembled for the case. Anesthetists must be flexible in remote settings and efficient preparation will assist in managing a difficult situation with confidence.2

References


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Multi-faceted Anesthetic Implications of Cerebral Palsy

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Keywords: Cerebral Palsy, pediatric, mask induction, seizures, non-verbal

Cerebral palsy is a broad term used to describe a constellation of symptoms, including varying degrees of motor, sensory, and intellectual impairment.1 Although a single causative factor has not been identified, premature birth seems to be a major risk factor.2 The incidence of cerebral palsy is 2 in 1000 live births and it accounts for 60% of motor impairment in childhood.1,2 Due to the numerous physiologic impairments that can be associated with cerebral palsy, many
children will require surgical intervention and anesthesia during their lifetime. Fortunately, with knowledge of the anesthetic implications related to cerebral palsy, successful management of these patients can be accomplished.

**Case Report**

A 17 year old, 40 kg female patient presented for bilateral hip resection arthroplasty to improve her range of motion and ease of care. As a neonate, she was diagnosed with cerebral palsy after exhibiting cognitive and physical impairment. Her medical history included seizures, contractures, and gastroesophageal reflux. The patient’s surgical history included an abductor tendon release and gastrostomy tube placement. Daily medications included levetiracetam and baclofen, which she received the morning of surgery. Her hematocrit level was 42%. The patient was severely contracted in all four extremities. Breath sounds were clear bilaterally. The nonverbal patient displayed signs of severe anxiety, including screaming and turning away from medical personnel. Her caretaker was able to calm her. Preoperative intravenous (IV) access could not be obtained secondary to the patient’s contractures and severe anxiety. Assessment of her Mallampati classification could not be obtained due to her anxiety and cognitive impairment.

In the operating room, noninvasive monitors were applied and oxygen was administered via facemask at 10 L/min. After baseline vital signs were obtained, sevoflurane 8% was administered via facemask. After loss of consciousness, an 18 gauge IV catheter was inserted in the right external jugular vein, flushed with 0.9% normal saline, and maintained with hydroxyethyl starch 500 ml infusing. Fentanyl 50 mcg and propofol 160 mg were administered IV. Direct laryngoscopy was performed using a Macintosh 3 blade and a 7.0 mm endotracheal tube was inserted into the trachea. Correct placement was confirmed and mechanical ventilation was initiated. Oxygen flow was decreased to 2 L/min, and sevoflurane end-tidal concentration was maintained between 1.7 and 2.9%. Glycopyrrolate 0.2 mg was administered IV.

The patient was initially positioned in right lateral decubitus position and was turned later to left lateral decubitus position. Pressure points were padded with foam. An upper body forced-air warming blanket was applied and her body temperature was maintained between 36.1 and 36.6°C. Lactated ringers 1000 mL was initiated once the hydroxyethyl starch was completed. Intra-operatively, her systolic blood pressure ranged from 97-149 mmHg and diastolic blood pressure ranged from 52-78 mmHg. Her heart rate ranged from 90-130 bpm. A maximum heart rate of 130 beats/min was noted after initial incision; she received fentanyl 50 mcg. Fentanyl was administered incrementally throughout the anesthetic for a total of 200 mcg. SpO2 was maintained at 99-100%, and end-tidal CO2 was maintained between 29 and 39 mmHg. Breath sounds remained clear throughout the anesthetic.

At the conclusion of the anesthetic, ondansetron 4 mg was administered IV and sevoflurane was discontinued. When the patient met extubation criteria, including breathing spontaneously with adequate tidal volumes of at least 300 mL, maintaining an end tidal CO2 of 35-39 mmHg, and demonstrating a positive gag reflex, the endotracheal tube was removed. She was taken to the post anesthesia care unit where her vital signs remained stable. She appeared moderately anxious upon arrival to the post anesthesia care unit, and her
A caretaker was brought in to sit with her at the bedside. She received 1300 mL of IV fluid replacement and had a total blood loss of 250 mL.

**Discussion**

Cerebral palsy presents in varying forms, and can therefore, be difficult to manage. In mild cases, cognitive symptoms are non-existent. In more severe forms, cognition and communication can be affected greatly. It is very important for the anesthesia practitioner to assess this preoperatively. The patient’s parents or caregivers can be useful sources of health history information and anxiety relief. Another issue arising from having a cognitively impaired patient is lack of understanding and cooperation on the patient’s part. For instance, assessing the patient’s Mallampati classification was very difficult. This can also make the assessment of postoperative pain difficult. Postoperative analgesia should be administered on a continuous basis for this reason. However, opioids should be administered with caution in these patients because they can accumulate and cause over-sedation, suppression of the cough reflex, and respiratory depression.

Gastroesophageal reflux is common in patients with cerebral palsy and may be severe enough to require a Nissen fundoplication. For this reason, tracheal intubation is strongly recommended. In one study by Wass et al., looking at cerebral palsy patients undergoing elective or emergent surgery at the Mayo Clinic between 1992 and 2008, the frequency of aspiration was only 0.5%. This is most likely attributed to taking nothing by mouth for six hours, use of antacids, and application of cricoid pressure during intubation. Some anesthesia practitioners believe that a laryngeal mask airway can be used depending on the surgical requirements and patient’s medical condition. Some believe there is no evidence that a rapid sequence induction is safer than an inhalation induction in patients with cerebral palsy.

Respiratory problems are frequently encountered. Reactive airway disease and recurrent pneumonia, resulting from repeated insults such as aspiration, can occur. The patient should be assessed for wheezing preoperatively. This patient had no wheezing preoperatively nor during the anesthetic. These patients have increased oral secretions with a decreased ability to cough and clear them. This can pose problems for the anesthesia practitioner including impaired visualization of the glottis during intubation, and risk of laryngospasm. This is often remedied using glycopyrrolate. It is also recommended to suction the patient before and after intubation. Postoperatively, these patients are at risk for respiratory failure due to a poor cough reflex and reduced respiratory drive, making them prone to secretion retention, basal atelectasis, and pulmonary collapse. These patients benefit from being in a high dependency or intensive care unit after surgery.

Due to thin skin, a small amount of subcutaneous tissue, and muscle atrophy, hypothermia is a major concern. In one study conducted by Wass et al., hypothermia was one of the most common perioperative complications associated with cerebral palsy. Wass and associates stated that they can be equated to large neonates because they have large surface area to body weight ratios and they conserve heat poorly. Prolonged neuromuscular blockade and delayed emergence are two consequences of hypothermia. The use of forced air warming devices in the preoperative holding
area may minimize heat loss during the early stages of the anesthetic, along with preheating the operating room. Intravenous fluid warming devices and humidifying circuits may be used during the procedure as well. Although this patient was not warmed in the preoperative area, an upper body warming blanket was used intraoperatively and maintained body temperature between 36.1 and 36.6 °C. No other warming measures were utilized during this anesthetic.

Patients with cerebral palsy often develop muscle contractures caused by differential growth rates between long bones and spastic muscle groups. This can make surgical positioning difficult. The anesthesia practitioner must ensure that all pressure points are padded and protected from nerve injury. This patient was turned laterally to each side during the procedure. Foam pads were used to pad pressure points. As in this patient, contractures can cause difficulties in obtaining vascular access. Vascular access was obtained in the external jugular vein.

Approximately one-third of cerebral palsy patients have epilepsy. Many patients are prescribed anticonvulsant and antispasticity medications. Many of these medicines can influence the pharmacokinetic profile of commonly administered medications. Some anticonvulsants induce liver enzymes and can lead to increased requirements of nondepolarizing neuromuscular blockers. The surgeon had requested that the patient not be given neuromuscular blockers, therefore this problem was not witnessed during this case. The research regarding succinylcholine use on these patients is equivocal. It has been shown that the succinylcholine-induced potassium release in CP patients is not significantly different from healthy patients. One theory is that their muscles were never fully developed and functioning, therefore extra-junctional receptors did not proliferate as they would in a normal patient. Another study demonstrated extra-junctional receptors in up to 30% of patients with cerebral palsy. Due to conflicting evidence, succinylcholine was not used to facilitate intubation in this case.

Numerous factors should be considered when caring for a patient with cerebral palsy. With proper planning, anesthetic management can be without incident, as in this case. If neuromuscular blockers are essential for the surgery, the anesthesia practitioner must be aware of the increased requirements often seen with patients taking anticonvulsants. Temperature regulation can be difficult and aggressive efforts must be taken to conserve heat and maintain normothermia. Many factors can complicate the anesthetic management of a cerebral palsy patient. However, successful management can be accomplished through understanding of the disease process and proper planning.

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Hypertrophic pyloric stenosis (HPS) is a commonly diagnosed gastrointestinal anomaly in the pediatric population. With HPS, hypertrophy of the pyloric region of the stomach results in a gastric outlet obstruction leading to the inability of stomach contents to pass into the intestines and progressive projectile vomiting. HPS in the pediatric population often presents as a medical, rather than a surgical emergency. Following medical management with fluid and electrolyte therapy, a surgical pyloromyotomy is generally performed, leading to a set of unique and challenging anesthetic implications. Techniques and thoughts on stomach decompression, intubation, and muscle relaxation for pyloromyotomy varies widely, although all patients must be considered at high risk for aspiration.

Case Report

A 1-month-old, 4.2 kg, full-term male presented for pyloromyotomy. He was admitted to the hospital with a history of nonbilious and nonbloody emesis for previous 1 week. The patient was a first-born child resulting from an uncomplicated pregnancy, birth, and postnatal period. His medical and surgical history was otherwise negative. Upon admission, the child was thin with decreased skin turgor, sunken fontanelles, and a pyloric “olive” noted with palpitation of the abdomen.

Following medical management that included correction of fluid and electrolyte status the patient was brought to the operating room for open pyloromyotomy. Upon arrival to the operating room (OR), a non-invasive blood pressure cuff, 5-lead electrocardiogram, pulse oximeter, nasal temperature probe, and precordial stethoscope were applied to the child. A 22 gauge intravenous (IV) line was already in place on the child’s right hand and a normal saline infusion at 30ml/hr was started. A 10 French orogastric (OG) tube was placed, suctioned with <5 ml gastric contents, and removed prior to IV induction. The child was administered 100% oxygen with 10 liters/minute (L/min) by face mask and given fentanyl 8 mcg, propofol 10 mg, and rocuronium bromide 5 mg IV. Cricoid pressure was applied at the start of induction.
and maintained until the trachea was intubated via direct laryngoscopy with a miller 1 blade and a 2.5mm cuffed endotracheal tube (ETT). Following confirmation of proper ETT placement cricoid pressure was released and the patients’ respirations were controlled with a mechanical ventilator. Anesthesia was maintained with sevoflurane 3% inspired concentration in a mixture of oxygen 1.5 L/min and air 1.5 L/min.

After the completion of a 30 minute successful open pyloromyotomy and uneventful anesthetic management, peripheral nerve stimulation used to assess neuromuscular function revealed no muscular twitches following administration of train-of-four (TOF) stimulation. Post tetanic stimulation also resulted in no muscular twitches requiring continued mechanical ventilation and support. Following another 30 minute time frame, the decision was made to transfer the patient to the post anesthesia care unit (PACU), and continue to mechanically ventilate him there until neuromuscular blockade could be safely antagonized. Sevoflurane was discontinued and the patient was transferred to PACU on 100% O₂ at 15 L/min via ambu-bag. Further sedation was not required and fifteen minutes after arriving in PACU with continued mechanical ventilation, TOF stimulation revealed 2 twitches. The child’s neuromuscular blockade was subsequently antagonized with glycopyrrolate 0.04 mg and neostigmine 0.2 mg. Following complete reversal of neuromuscular blockade with tetany >5 seconds and the return of spontaneous respirations and protective reflexes, the oropharynx was suctioned and the trachea was extubated without incident. The rest of the child’s PACU and hospitalization stay was uneventful.

Discussion

HPS is commonly diagnosed between 2 and 12 weeks of age with an incidence of 2-5 per 1,000 live births, and is approximately four times more common in first born males than females.¹,² Although the cause of HPS remains poorly understood, the pathophysiology along with its presentation and diagnosis are fairly straightforward. The pylorus which is located at the distal end of the stomach is connected to the duodenum by the pyloric sphincter. Pyloric hypertrophy inhibits the pyloric sphincter from opening and allowing stomach contents to pass into the intestines resulting in emesis.³ Persistent and progressive vomiting usually occurs 30-60 minutes after a feeding. Electrolyte abnormalities ensue over time. With the loss of sodium, potassium, chloride, and hydrogen via gastric fluid; hypokalemic, hypochloremic primary metabolic alkalosis with a secondary respiratory acidosis can occur.¹ Palpation of the hypertrophied pylorus, as an olive-shaped mass in the epigastrium, can successfully be performed by skilled practitioners’ with accurate diagnosis 99% of the time.² The confirmatory and diagnostic study of choice for HPS is Ultrasonography.¹

Pyloromyotomy remains the definitive treatment for pyloric stenosis with a high success rate and a mortality rate of 0.4%.¹,² However, as mentioned earlier, it is performed non urgently only after fluid and electrolyte abnormalities have been corrected.² A serum chloride greater than 100 mEq/dl and serum bicarbonate less than 28 mEq/dl are generally indicative of adequate fluid and electrolyte correction and readiness for surgical repair.¹ Our patient met these criteria after in hospital medical management.
The anesthetic plan for these patients must address two key factors. First, patients must be viewed as having full stomachs, and second, they must be considered at high risk for postoperative apnea. Stomach decompression, awake intubation (AI) or rapid sequence induction (RSI), muscle relaxation, and opioids all deserve discussion and consideration. There is little controversy in the literature regarding the need for stomach decompression or the use of opioids. A large bore naso-gastric or oro-gastric catheter should be inserted and suctioning ensued prior to induction of anesthesia. It has been indicated that multiple patient positions should be considered to ensure complete stomach decompression. Furthermore, long acting sedatives and opioids are generally not required and have the potential to delay emergence as well as increase the risk of postoperative apnea.

Awake intubation is generally discussed as the induction method of choice for an anticipated difficult airway, and RSI with either succinylcholine or rocuronium for all others. Although succinylcholine has been noted to have detrimental side effects in the pediatric population it is still frequently indicated and utilized for RSI.

RSI in pediatrics is a controversial issue with a great deal of variability noted. Classic RSI (pre-oxygenation, administration of hypnotic and neuromuscular blocker, application of cricoid pressure, and apnea until intubated) in pediatrics is often a time critical procedure that results in high levels of stress for the anesthesia professional and possible oxygen desaturation in the patient. A low tolerance for apnea generally occurs often resulting in the potential for forced mask ventilation and unsuccessful intubation attempts. Therefore, a controlled RSI techniques designed to reduce time pressure, hypoxia, and anesthesia professional stress are becoming increasingly accepted. A controlled RSI technique consists of pre-oxygenation, administration of hypnotic and non-depolarizing muscle relaxant, gentle mask ventilation (<12cmH2O), and intubation after deep anesthesia and complete neuromuscular blockade.

Additionally, current literature is suggesting that the use of cricoid pressure (CP) is an ineffective intervention that is used without scientific evidence or randomized trials to support its use. Radiological and magnetic resonance imaging studies indicate that the cricoid ring and esophagus are usually not in alignment. CP results in lateral displacement of the esophagus up to 90% of the time rendering it ineffective. Furthermore pediatric patients have soft and easily damageable laryngeal structures that are likely to sustain damage with the application of 20-40 Newtons of pressure. Additionally, visualization of the glottis can also be impaired, especially in infants, with CP. The speed of induction, and insuring complete paralysis prior to intubation, has been suggested to be more important in preventing regurgitation and aspiration than CP. Clinical data shows aspiration complications occur almost exclusively with inadequate depth of anesthesia and neuromuscular blockade. Rushed intubation attempts from stressed anesthesia professionals during a classic RSI technique can result in bucking, straining, coughing, difficult laryngoscopy, and regurgitation with aspiration.

In conclusion, a pyloromyotomy performed for the correction of pyloric stenosis has several unique anesthetic management considerations. Although some anesthetic practices are more controversial than others, it is clear that this population is at high risk.
for aspiration. Therefore, a thorough assessment, pre operative optimization of fluid and electrolyte status and individualized plan weighing the risk/benefit ratio of the anesthetic technique is essential.

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Nasal Intubation for Outpatient Dental Procedures

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Keywords: nasal intubation, anesthesia, nasotracheal, dental procedures, outpatient

Young client populations as well as those with mental handicaps often require anesthesia for dental procedures. Medical and surgical interventions necessitating an oropharyngeal approach as required in oral and maxillofacial surgeries provide obvious obstacles for the anesthesia practitioner. Nasotracheal intubation may provide the most advantageous airway management for outpatient dental procedures.¹

Case Report

The patient was a 23-year-old, 70 kg, 157 cm male with a past medical history of cerebral palsy, cognitive impairment, seizures, recurrent aspiration pneumonia, gastro-esophageal reflux disease, and scoliosis. He presented to the outpatient operating room for dental cleaning under anesthetic management. The patient’s past
surgical history consisted of a spinal fusion, intrathecal baclofen pump placement, surgical hip and knee releases, prior dental treatments, a gastric tube placement, and multiple irrigations and debridements for spinal and abdominal wounds. There was no personal or family history of anesthesia complications such as post-operative nausea and vomiting.

Pre-operative assessment revealed that the patient had no food or drug allergies, had nothing to eat or drink past midnight, had clear lungs sounds bilaterally with S1, S2 heart sounds audible. A 20 gauge peripheral IV had been placed in the right hand by nursing. The procedure and general anesthetic plan were confirmed through the patient’s mother with verification the patient had no history of epistaxis. Airway examination was limited due to lack of patient cooperation. The patient received midazolam 1 mg IV and was transported to the operating room where standard monitors were applied. The patient was pre-oxygenated with 100% FiO2 for five minutes. A smooth IV induction was accomplished with fentanyl 50 mcg, lidocaine 100 mg, propofol 145 mg, and rocuronium 50 mg IV. The patient was easy to mask ventilate. Oxymetazoline spray was applied via both nares in preparation for nasal intubation. The left naris was serially dilated using nasal trumpets lubricated with lidocaine jelly. A 6.5 cuffed nasal RAE tube was softened in warm water and subsequently inserted into the left naris and advanced into the oropharynx. Direct laryngoscopy was performed with a grade 1 view of the glottis using a MAC 3 laryngoscope blade. The nasal RAE tube was advanced through vocal cords without requiring Magill forceps. The intubation was atraumatic. End tidal carbon dioxide and bilateral breath sounds were confirmed. The procedure proceeded uneventfully and the patient was extubated after emergence.

Discussion

Patients unable to cooperate with traditional dental examination and treatment frequently undergo general anesthesia in order to receive care.1 The oropharyngeal access required to address dentition competes with space reserved for airway management.2 Nasal intubation provides an alternative airway management technique for surgeries in which oral intubation is not plausible in addition to providing several other notable advantages.

Specific nasotracheal tubes assist in angling breathing circuits away from the surgical field thereby allowing the dental team an even greater unobstructed workspace. The nasal RAE (Ring, Adair, Elwyn) tube, for instance, is designed with a bend at the naris permitting the tube to be sloped over patients’ foreheads. The stability of the manufactured bend permits the angled formation without significant risk of kinking.3

Nasotracheal tubes can prevent tube occlusion resulting in negative pressure pulmonary edema (NPPE).6 Negative pressure pulmonary edema arises when an obstruction of the upper airway allows negative intrathoracic pressure to pull fluid from the pulmonary capillaries into the alveoli. One cause of NPPE is the obstruction of airflow through an endotracheal tube due to patient biting.6 Nasotracheal tubes do not pass through the mouth and therefore obstruction of air related to patient biting is prevented.

Another favorable aspect of nasotracheal intubation is that the intubation procedure is
similar to nasogastric tube placement as well as traditional oral intubation techniques.\textsuperscript{2,4} The nasotracheal tube is lubricated to prevent friction and is placed into a naris at a perpendicular angle with tube movement along the floor of the nose.\textsuperscript{2} As the nasotracheal tube becomes visible in the oropharynx, it can be directed through the vocal cords using direct laryngoscopy.\textsuperscript{4} The similarities among the procedures can help to facilitate quick mastery of the nasotracheal intubation technique.

Despite the advantages to nasotracheal intubation, significant disadvantages exist. The beneficial bend of the nasal RAE tube, for example, can prove to be unfavorable. The bend can be too distal or proximal to a specific patient’s naris inhibiting proper positioning of the tube.\textsuperscript{3} Nasal tubes must also be of smaller diameter to accommodate the size of the naris and to reduce nasal injury.\textsuperscript{2} This causes greater resistance to flow and inhibits tracheal suctioning.\textsuperscript{1,3}

Although nasotracheal intubation can resemble oral intubation or nasogastric tube placement, there are additional measures required. Nares are prepared with a topical anesthetic such as lidocaine 3–4% with phenylephrine 0.25–1%.\textsuperscript{2} Nasal trumpets of increasing size prepared with water-soluble jelly should also be used to determine patency of the naris prior to nasotracheal tube insertion.\textsuperscript{3} Advancement of the nasotracheal tube through the vocal may also require manipulation with Magill forceps.\textsuperscript{4}

Notable complications have been associated with nasotracheal intubation. Epistaxis is the most common and generally results from mucosal damage. While intranasal topical vasoconstrictors are often utilized to address this concern, they present risks of their own.\textsuperscript{2,7} One case review found four pediatric and five adult patients to have suffered hypertensive crisis or pulmonary edema associated with topical phenylephrine use. Three of the cases resulted in death from cardiac arrest.\textsuperscript{7} Other complications have included local infection, maxillary sinusitis, abscess formation and bacteremia related to mucosal wall damage.\textsuperscript{2} Valdes et al concluded that nasotracheal intubation over oral intubation led to greater numbers of micro-organisms in the bloodstream and suggested preference of orotracheal intubation for patients at risk for endocarditis.\textsuperscript{5}

Absolute contraindications to nasotracheal intubation include placement in individuals with known head or facial trauma due to the risk of brain penetration. Anticoagulation is also considered a contraindication due to increased risks of epistaxis.\textsuperscript{3,4}

The patient presenting for outpatient dental treatment was a candidate for nasotracheal intubation. A nasal RAE tube was chosen for his care and did provide a means for removing the ventilator circuit from the surgical field. The patient’s nares were prepared with vasoconstrictive spray and the tube lubricated with lidocaine jelly. The tube was easily passed through the nares with no evidence of epistaxis. Placement of the tube through the vocal cords was easily accomplished with use of a Mac 3 laryngoscope blade. The patient did well with no anesthetic complications or adverse events.

Nasal intubation has clear value. Cognitive impairment prevented this patient from tolerating routine dental treatment. Nasotracheal intubation afforded him an opportunity to have his teeth cleaned under more tolerable circumstances while allowing the dental team access to his oral aperture. Keeping the potential complications in mind
and remaining vigilant, anesthesia practitioners utilize nasotracheal tubes to help facilitate the care of patient populations in need.

References


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5-HT3 Receptor Antagonism following Suspected Bone Cement Implantation Syndrome

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Keywords: bone cement implantation syndrome (BCIS), Bezold-Jarisch reflex, methyl methacrylate, total hip arthroplasty, serotonin, thromboxane, pulmonary embolism, fat embolism, air embolism.

Embolism during total hip arthroplasty (THA) has been described in the literature since the 1970’s.1,2 The consequences following a pulmonary embolism of any kind focuses on the changes in normal cardiopulmonary circulation, the inflammatory responses of the injured pulmonary endothelium and cardiopulmonary reflexes.3 Bone Cement Implantation Syndrome (BCIS) has been defined by some authors as hypoxia and/or hypotension, with or without loss of consciousness during the cementation process of an orthopedic prosthesis insertion.4 It is widely accepted that the primary pathophysiologic insult leading to the signs and symptoms of BCIS is pulmonary embolism. In addition to hip procedures, Bone Cement Implantation Syndrome (BCIS) has also been associated with vertebral and shoulder procedures.4 The following case report describes suspected BCIS with pulmonary embolism and the subsequent patient management.
Case Report

A 64-year-old female (175 cm, 121 kg) presented for surgical revision of a prior right total hip arthroplasty due to ongoing pain. Preoperative differential diagnoses were metal debris versus infection. Her medical history included arthritis, hypertension, type II diabetes mellitus, hyperlipidemia, hypothyroidism, and gastroesophageal reflux disease. She indicated no prior complications associated with surgery or anesthesia or any known drug allergies. Her preoperative evaluation included hematologic and biochemical laboratory tests which were within normal ranges. Initial hemoglobin was 12 g/dl. An 18g intravenous catheter was placed and a lactated Ringers infusion was initiated. Midazolam 1 mg was administered upon transport to the operating room (OR) suite. A combined spinal-epidural (CSE) was performed while the patient was in the sitting position while inhaling oxygen 6 L/min by simple face mask. A subarachnoid dose of 1% tetracaine 1 ml was administered followed by the placement of a 20 gauge epidural catheter. Once the epidural catheter was secured the patient was assisted into the supine position. A propofol infusion was initiated at 50 mcg/kg/min and fentanyl 50 mcg was administered. A second 18 g intravenous catheter was placed and the patient was repositioned into the left lateral decubitus position with monitoring as follows: electrocardiogram, non-invasive blood pressure (NIBP), respiratory rate, end-tidal carbon dioxide, and pulse oximetry (SpO2). Vital signs were within acceptable ranges and a T9-T10 level of sensory anesthesia was achieved. Six minutes after repositioning surgical incision was made.

Approximately 125 min following surgical incision the patient demonstrated a decrease in heart rate from 63 beats per min (bpm) to 47 bpm. NIBP measurements demonstrated a decrease from 100/65 mmHg to 76/28 mmHg. At this time, a distinct odor was detected in the OR suite, which was identified as methyl methacrylate (a component of bone cement). At this time the anesthetist suspected the occurrence of BCIS and the rate of propofol infusion was reduced. Our patient was minimally responsive to auditory stimulation at this time. Up to this point the estimated blood loss (EBL) was 900 ml and the patient had received 3 L of crystalloid intravenous fluids and 2 units of packed red blood cells. Additionally the patient’s urine output remained greater than 1ml/kg/hr and SpO2 values remained stable at 99-100%; nonetheless, the rate of oxygen flow was increased from 6 to 10 L/min. Ondansetron 8 mg was immediately administered along with glycopyrrolate 0.8 mg in two doses, approximately 2 min apart. Once the patient’s heart rate had increased above 70 bpm, phenylephrine 100 mcg was administered. The patient’s vital signs returned to normal and did not require further intervention. The changes in vital signs and the above interventions took place over approximately fifteen min and coincided with the implantation of the femur prosthesis.

The intraoperative phase continued without any further episodes of bradycardia or hypotension. Due to a total EBL of 1200 ml the patient received three units of packed red blood cells. Hydromorphone 1.5 mg IV was administered throughout the case. At the end of the procedure the propofol infusion was discontinued, and the patient was able to respond to all commands and denied any pain. She was transported to the post anesthesia recovery unit for continued monitoring.
Described in the above case report was a suspected episode of BCIS and an associated pulmonary embolus. Bone cement has been a mainstay of orthopedic surgery since the 1960’s. It has been shown to improve joint stability and prosthesis implantation. The implantation of bone cement provides a mechanism by which emboli may reach the pulmonary vasculature. Embolized substances may include air, fat (marrow), platelet/fibrin aggregates, bone particles, and methyl methacrylate particles. Animal reports indicate a tenfold increase in emboli seen with cemented versus uncemented arthroplasty. Patients with hip fractures, in particular inter-trochanteric fractures, are at increased risk for BCIS. Rationale for this increased risk centers on the demonstration that femoral neck fractures, bone cement implantation, and prosthesis insertion (common with inter-trochanteric fractures) can cause significant increases in pulmonary vascular resistance (PVR) by way of pulmonary embolism which leads to the signs and symptoms of BCIS. This elevation in PVR impairs right ventricular systolic function leading to a decrease in cardiac index and stroke volume index. Complete cardiovascular collapse has been described in case reports following the implantation of methyl methacrylate. The rapid assessment and integration of sensory information presented in the OR suite permitted rapid treatment of suspected BCIS and the associated pulmonary embolism.

The pathophysiologic consequences of BCIS are associated with both mechanical and biochemically mediated changes to the cardiopulmonary system. The cardiovascular consequences following pulmonary embolism can be understood in part with the explication of the cardiopulmonary responses to serotonin, thromboxane, interleukin, and endothelin. These mediators are released by activated platelets in the lungs following embolization and have potent effects on the pulmonary vasculature which in turn have significant effects on the myocardium. Serotonin, the most prominent mediator released is the most powerful pulmonary vasoconstrictor known. While a potent vasoconstrictor of pulmonary vasculature, serotonin has been shown to cause dilation of the peripheral arterioles. The combined effects of increased pulmonary vascular resistance (PVR) in the setting of decreased right ventricle preload (systemic vasodilatation) can have devastating effects on cardiac output (CO). As CO is impaired, hypotension can be further exacerbated. Ultimately, hypoxia, hypopnea, hypoventilation, bradycardia, and hypotension are resultant. Combined, these effects can precipitate cardiovascular arrest and death if not recognized and treated promptly. To attenuate the potent pulmonary vasoconstrictor properties of serotonin we chose to administer the 5-hydroxytryptamine3 (5-HT3) receptor antagonist, ondansetron.

In addition to these chemical mediators, the effects of the Bezold-Jarisch reflex (BJR) contributes to our understanding, integration, and appropriate intervention of suspected BCIS. The Bezold-Jarisch reflex, described as bradycardia, vasodilatation, hypotension, and apnea resulting from stimulation of cardiac mechanoreceptors, may have a role in the pathophysiology of PE. This reflex, thought to act independently of baroreceptor and vagal reflexes, may contribute to the potentiation of vasomotor inhibition independent of heart rate modulation. Thus what may be observed is hypotension in the setting of sustained bradycardia, an anti-baroreceptor effect. Glycopyrrolate was administered to...
counter the cholinergic response triggered by what may be a vaso-vagal reaction, Bezold-Jarisch reflex, or combination of the two. The BJR is also partially mediated by intracardiac chemoreceptors. These chemoreceptors are sensitive to serotonin, and are primarily 5-HT$_3$ type receptors. Along with decreases in myocardial preload animal studies suggest that serotonin may be contribute to the induction of the BJR. The use of ondansetron to counter the effects of serotonin induced increases in PVR may have also attenuated the BJR by modulating efferent vagal activity. The use of ephedrine may have been indicated for its positive chronotropic and vasoconstrictive properties in the setting of bradycardia and hypotension. However, caution must be taken if it is accepted that pulmonary vasoconstriction plays a pivotal role in this pathologic process, as the use of a potent vasoconstrictor in the absence of serotonin antagonism may have deleterious effects that further complicate patient resuscitation as a rapid increase in right ventricle preload in the setting of increased PVR may lead to acute right ventricular failure. For this reason the use of glycopyrrolate and ondansetron was chosen as initial therapy.

Recognition of patients at risk for BCIS plays a key role in the preparation of an anesthetic plan. Risk factors include; cemented procedures, pre-existing pulmonary hypertension, significant cardiac disease (New York Heart Association class 3 or 4), pathological inter-trochanteric fracture, and long stem prosthesis placement. Anesthesia professionals are responsible for initial supportive treatment and should include the administration of 100% oxygen, IV fluids, and support of heart rate and blood pressure with chronotropic and vasoactive medications. In this case, the early recognition of suspected BCIS, along with an in-depth understanding of the chemical and physiologic mediators responsible for the symptoms observed, contributed to the prompt and sustained resolution of symptoms.

References


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**Anesthetic Management of Hereditary Spherocytosis**

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**Keywords:** spherocytosis, hemolytic crisis, anesthetic management

Hereditary spherocytosis (HS) is the most common inherited hemolytic anemia in the United States, affecting 1 out of 5000 individuals.¹ The disorder is characterized by red blood cells that have weakened surface membrane skeletal proteins. This structural anomaly results in spherically shaped erythrocytes that are osmotically fragile with a shortened circulation half-life. The anesthetic risk is relative to the severity of anemia and commonly includes increased risk of infection, splenomegaly, gallstone formation, and hemolytic anemia.² The key to successful management of such a patient is the avoidance of triggering factors and vigilant perioperative monitoring.²

**Case Report**

The case reviewed involved a 37-year-old, 83.5 kg male with a medical history of mild HS, mild intermittent asthma, and hepatitis B. He presented to the outpatient surgery setting for anal fistulotomy. Surgical history included a cholecystectomy and appendectomy at the age of 31 years. The patient reported becoming jaundiced for several days without further complication following each prior surgery. He reported a total plasma bilirubin concentration of 0.7 mg/dL and a hemoglobin concentration of 12.8 g/dL that were drawn within the year. Labs were not repeated in the immediate preoperative period.

Physical and airway assessments were completed; both were unremarkable with no icterus or jaundice noted. The patient denied a past medical history of hemochromatosis or aplastic crisis. The patient’s allergies included codeine and current home medications included acidophilus, adefovir, albuterol, fluticasone-salmeterol, folic acid, glucosamine-chondroitin, ibuprofen, and a multivitamin.

The patient was transferred to the operating room and standard monitors were applied, with alarms on and parameters checked. Midazolam 2 mg and cefazolin 2 g were administered to the patient. Pre-oxygenation was performed. General anesthesia was induced by administering fentanyl 100 mcg, lidocaine 80 mg, propofol 200 mg, and rocuronium 25 mg. Mask ventilation was performed without difficulty. Direct laryngoscopy was performed with a miller 2 blade, yielding a grade 1 view. A standard 7.0 cuffed endotracheal tube was passed under direct vision and the trachea was
intubated. Intubation was performed without complication and end-tidal carbon dioxide and bilateral breath sounds were confirmed. Respirations were controlled by mechanical ventilation.

Anesthesia was maintained using fentanyl and sevoflurane. The patient was placed in prone jack-knife position, with proper joint alignment confirmed and pressure points padded. The anesthetic course was uncomplicated. The patient received 1400 mL of lactated ringer’s solution throughout the case. At the conclusion of surgery, the patient was given ondansetron 4 mg. Neuromuscular blockade was antagonized with neostigmine 3 mg and glycopyrrolate 0.4 mg. The patient was returned supine and placed in Fowler’s position. He resumed spontaneous respiration and appropriately followed commands; the trachea was extubated. Oxygen 6 L/m was administered via a Mapleson C circuit. The patient was monitored for signs of apnea and hypoxemia and transferred to the post-operative care unit in stable condition.

Discussion

Hereditary Spherocytosis is characterized by spherically-shaped erythrocytes, or spherocytes, and is diagnosed by the presence of spherocytes on a peripheral blood smear. The spherical shape of affected erythrocytes is caused by weakened surface membrane skeletal proteins. Spherocytes have decreased membrane surface area, decreased elasticity, increased osmotic fragility, and reduced circulation half-life compared to normal erythrocytes.2 Spherocytes are trapped and destroyed in the spleen, resulting in hemolysis, anemia, jaundice, and splenomegaly. Common complications include cholelithiasis and hemolytic episodes. Anemic patients also have enhanced iron absorption, which can lead to severe iron overload in the form of hemochromatosis.3 In the majority of cases, the disorder is inherited in an autosomal dominant pattern, however sporadic mutations and recessive cases have been described.1

Medical management of HS includes folate supplementation and routine blood testing, both of which the current patient was undergoing. Though there is no strong evidence to support universal folic acid supplementation, it is often utilized due to the increased need for folic acid during periods of increased hematopoiesis.4,5 Laboratory data including a complete blood count, basic metabolic panel, and transferrin saturation may be useful for alerting clinicians to sub-clinical hemolysis in the perioperative period. However, for patients with mild HS, such as the current patient, the value of routine or frequent blood testing is debatable and unnecessarily disruptive as long as the patient remains asymptomatic.3 For those with moderate to severe HS, or asplenic patients, the need for blood testing is relative to the severity of their symptoms.3,5 In addition, practitioners should be aware that pseudohyperkalemia is a laboratory artifact common among patients with HS, due to a high rate of potassium leakage out of the red cells as spherocytes cool and hemolyze after venipuncture.4

Surgical management of HS may include symptomatic cholecystectomy and/or splenectomy. Gallstone formation is common among HS patients, and a cholecystectomy may be performed for symptomatic management of recurrent cholelithiasis, as was the case for the current patient. Additionally, approximately 30% of HS patients suffer from severe or recurrent hemolytic episodes and undergo splenectomy.6 Splenectomy is curative
because it eliminates anemia secondary to sequestration and hyperbilirubinemia and reduces the reticulocyte count to normal levels.\(^7\) Though splenectomy does not alter the osmotic fragility of the spherocytes, a reduction in presenting symptoms is generally observed.\(^8\) Unfortunately, this symptomatic relief is accompanied by an increased risk for thromboembolic events, up to 5 times greater than those who had not undergone splenectomy.\(^9\) Asplenic patients are also at an increased risk of sepsis.\(^3\) The current patient did not require splenectomy because his hemolytic episodes were self-limited and directly related to periods of acute stress or infection. His hemolytic episodes had never developed into aplastic crisis.

The anesthetic risk of patients with HS is believed to be relative to the severity of their anemia. Patients are considered lowest risk when in a steady state of hemolysis and highest risk during exacerbation due to concurrent infection.\(^1\) The stress, trauma and inflammation related to surgery can place HS patients at an increased risk for perioperative thromboembolic events due to extracellular fluid sequestration.\(^6,10\) Aggressive hydration and preemptive erythrocyte transfusion are commonly recommended to minimize risks of surgery.\(^10,11\) The current patient was liberally hydrated with lactated ringer’s solution 1400 mL during the twenty-eight minute procedure. Lower extremity sequential compression devices were utilized in the operating room and in the immediate post-operative period to reduce the risk of thrombosis. No preemptive erythrocyte transfusion was deemed necessary, as he presented asymptomatic with stable hemoglobin and bilirubin levels.

Additional recommended perioperative management of patients with HS includes the avoidance of hypoxia, hypothermia and intraoperative acidosis. Anesthesia practitioners should replace blood loss when necessary and maintain normal body temperature to minimize vasoconstriction and circulatory stasis.\(^11\) The patient was given 95% FiO2 throughout the procedure, and his pulse oximeter oxygen saturation was maintained at or greater than 98%. A pharyngeal temperature probe was used to monitor for hypothermia. A forced air warmer was utilized to maintain normothermia. Common signs and symptoms of acute hemolytic anemia include pallor, shortness of breath, and fatigue.\(^3\) In an anesthetized patient, dark or decreased urine output, fever, or unexplained tachycardia may indicate the onset of acute hemolysis.\(^11\) None of these symptoms were noted in the current patient throughout the perioperative period. Follow up care included monitoring for jaundice and post-operative bilirubin analysis.

The patient with HS is at increased risk for developing various perioperative complications, such as aplastic crisis and hemolytic episodes. Prevention and management may include preemptive erythrocyte transfusion, aggressive hydration, and avoidance of hypoxia, hypothermia, and acidosis. Clinicians should replace blood loss when necessary and maintain normal body temperature. Post-operative care includes monitoring for jaundice and bilirubin analysis. This case report has presented an example of anesthetic management of a patient with HS in the outpatient surgical setting.

References

Anesthesia for a Child with Opsoclonus-Myoclonus Syndrome

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Keywords: opsoclonus-myoclonus syndrome, Kinsbourne syndrome, Dancing Eyes-Dancing Feet syndrome, opsoclonus-myoclonus ataxia, neuroblastoma

Opsoclonus-myoclonus syndrome (OMS) is a rare neurologic disorder characterized by a sudden onset of jerking movements of the eyes (opsoclonus), myoclonus, and ataxia.\(^1\,^2\)

OMS, also known as Kinsbourne syndrome or Dancing Eyes-Dancing Feet syndrome, affects 1 in 10 million people. Children are most often affected and usually present between the ages of 6 and 36 months with severe irritability and an inability to walk.\(^2\)

OMS is considered an autoimmune disease, and is associated with a neuroblastoma in up to 50% of cases.\(^1\,^2\)

Case Report

A 20-month-old, 84 cm, 12 kg female presented for insertion of an implanted central venous access device for the administration of intravenous immunoglobulin (IVIg) and cyclophosphamide. A diagnosis of OMS was made at 14 months of age when the patient became unable to sit or stand and had accompanying abnormal eye movements that were consistent with opsoclonus. Symptoms improved after initiation of synthetic adrenocorticotropic hormone
The patient was born at 37-weeks gestation as a healthy twin. Developmental milestones were appropriately met. There was no history of significant infections or illnesses. Computed tomography (CT) scan of the head, neck, chest, abdomen, and pelvis and iodine-123 metaiodobenzylguanidine scintigraphy (MIBG) were negative for tumors. The patient had no known allergies. Prescribed medications included synthetic ACTH, trazadone, ranitidine, calcium carbonate, and trimethoprim/sulfamethoxazole. Recent lab values showed white blood cells 9 K/μl, hemoglobin 11.4 g/dl, hematocrit 33.7%, and platelets 273 K/μl. Vital signs were 36.3°C oral temperature, blood pressure 133/59 mmHg, heart rate 123 bpm, respiratory rate 26, and SpO2 96% on room air. The patient was irritable on physical examination and showed generalized weakness, fine motor tremors, and delayed speech.

The patient was brought into the operating room for insertion of an implanted central venous access device. Standard monitors were applied and inhalation induction was initiated with nitrous oxide (N2O) 7 L/min, O2 3 L/min, and sevoflurane 8% inspired concentration. A 22 gauge peripheral intravenous (IV) catheter was inserted into the left hand. A laryngeal mask airway (LMA; LMA North America, San Diego, CA) size 2 was inserted without difficulty. Cefazolin 200 mg and hydrocortisone sodium succinate 12 mg were administered IV prior to incision. Anesthesia was maintained with sevoflurane 2.5%-3.0% expired and a combination of O2 and N2O at 1 L/min each. Spontaneous respirations were maintained throughout the procedure and vital signs remained stable. Ondansetron 2 mg and ketorolac 6 mg were administered IV approximately 20 minutes and 5 minutes before the completion of the procedure, respectively. At the end of the procedure the LMA was removed without complications. Upon arrival to the recovery room the patient was awake with stable vital signs.

Discussion

Opsoclonus-myoclonus syndrome is usually triggered by an acute infection or a neuroblastoma, in which case it is referred to as a paraneoplastic syndrome.1,2,4-6 The characteristic symptoms are opsoclonus (multidirectional, chaotic eye movements), non-epileptic myoclonus of any muscle, ataxia preventing the ability to sit or stand, and behavioral changes such as irritability and sleep disturbances.2 The exact cause is not entirely clear but an autoimmune theory is supported by increased B cells in cerebrospinal fluid and an improvement of symptoms with immunosuppression via drug therapy.2 After a diagnosis of OMS is made, it is important to rule out the presence of an associated neuroblastoma. A CT scan, urine catecholamine levels, and MIBG should be obtained.2

Treatment of OMS mainly focuses on immunosuppression and antineoplastic therapy. Patients are most commonly prescribed steroids and/or synthetic ACTH, which has been shown to provide disease remission.1,2 Prophylactic therapy with ranitidine and calcium carbonate is usually employed to decrease the occurrence of peptic ulcer disease and osteoporosis, respectively, that can occur with ACTH therapy. Trazadone is useful to decrease sleep disturbances that can be a result of drug therapy or the disease itself.1 Hypothalamic-pituitary-adrenal axis suppression from chronic use of ACTH should be anticipated and a steroid stress
A dose was administered at the beginning of the case for this reason. The immunosuppression that accompanies the therapies used to treat OMS prompted the administration of cefazolin prior to incision.

It is important to note that this is a non-epileptic form of myoclonus and anticonvulsants such as phenytoin, carbamazepine, and lamotrigine are not useful and may make the myoclonus worse.\(^1\)\(^3\) Haden, McShane, and Hold\(^5\) reported several instances in which OMS was misdiagnosed as status epilepticus. These children were placed on the mechanical ventilator after the administration of phenytoin did not help.\(^5\) Once the diagnosis of OMS was made, therapy with ACTH was initiated and the patients were eventually able to be discharged home.\(^5\)

Additional medications that should be avoided in patients with OMS include all narcotic analgesics, ketamine hydrochloride, pentobarbital, diphenhydramine, haloperidol, and chlorpromazine.\(^1\)\(^3\) These drugs can be ineffective, cause excitation, or worsen symptoms.\(^1\) Midazolam may cause excitation as well and should be avoided.\(^3\) Etomidate should be avoided since it can cause acute adrenocortical insufficiency and lead to an adrenal crisis.\(^7\) Propofol is considered to be a safe option.\(^1\)

Antihistamines should not be administered to these children because they can cause hyperactivity or relapse of OMS symptoms.\(^3\) Additionally, anything containing pseudoephedrine or antihistamines can cause a hypertensive crisis, especially if the patient is on ACTH or steroid therapy.\(^3\)

The decision to use an LMA to secure the airway was based on several factors. According to Jamil et al\(^9\) hemodynamics remain more stable with the use of an LMA as compared to endotracheal intubation. Less postoperative airway complications were also observed with the use of the LMA, including laryngospasm, bronchospasm, and soft tissue damage.\(^9\) The patient was not considered to have a full stomach, the length of the procedure was anticipated to be less than one hour, and complete muscle relaxation was not necessary so an LMA was considered an appropriate choice. Nitrous oxide was added to sevoflurane for its ability to provide analgesia intraoperatively as a means to avoid administering opiates.\(^8\) Ketorolac was administered prior to emergence to assist with postoperative pain control. The procedure progressed without complications and the patient did not suffer any adverse events.

Anesthesia professionals need to be familiar with rare diseases, but more importantly, be aware that there are rare diseases out there with serious anesthesia implications. When a patient with an unfamiliar diagnosis is encountered, take a few minutes and do a little research. We sat down with the parents of our OMS patient who provided us with a list of medications to avoid in these children. In the patient’s chart, we found documentation from the child’s neurologist outlining the course of her illness and explaining the disease process. This signifies the importance of being attentive to anesthetic implications associated with unfamiliar disease and drawing on multiple resources in each unique case.

References

Clinical Management of Hereditary Angioedema

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Keywords: angioedema, facial swelling, emergency airway, C1 inhibitor, tracheal intubation

Hereditary angioedema is an autosomal dominant disease caused by a deficiency in functional C1 inhibitor resulting in unconstrained tissue bradykinin formation. Angioedema is characterized by recurrent episodes of non-pruritic, non-pitting, subcutaneous, or submucosal edema caused by increases in vascular permeability involving one or multiple areas of the body. Attacks may involve arms, legs, hands, feet, bowels, genitalia, trunk, face, tongue, and/or larynx. Hereditary angioedema affects 1 in 50,000 persons and accounts for 2% of angioedema cases.

Case Report

A 57-year-old, 175 cm, 119 kg, Caucasian male presented for an outpatient colonoscopy and drainage of rectal abscess. Past medical history included hyperlipidemia, esophageal reflux, dyspnea, sleep apnea, hypertension, obesity, former smoker, rectal abscess, and angioneurotic edema. His medications included hydrocodone, omeprazole, rosuvastatin, amlodipine, hydrochlorothiazide, multivitamin, valsartan, and danazol 200 mg.
Anesthesia assessment was conducted with special focus on the patient’s history of hereditary angioedema. The patient was diagnosed with hereditary angioedema at age 20, joining his mother and brother who also suffered from the disorder. The patient stated that angioedema attack triggering events for him include routine dental visits and wood working. He had been admitted to the emergency room numerous times because he felt the symptoms of an angioedema attack which included the sensation of neck fullness and warm tingling feelings in surrounding tissues. These attacks were associated with oropharyngeal and facial swelling. The patient stated that he recognized symptoms of an impending attack and would seek medical treatment immediately.

Guided by the history and airway examination, it was determined that a minimally invasive anesthetic to avoid airway manipulation, such as light sedation with local anesthetic, would be safest for the patient. The airway assessment revealed a mallampati class II with a thyromental distance > 6 centimeters, and mouth opening < 6 centimeters. The patient stated he had taken his daily dose of danazol (androgen) prior to hospital arrival. Administration of C1 esterase inhibitor is the standard of care in an acute attack of angioedema. To prepare for reactions during the procedure, the anesthesia team attempted to locate C1 inhibitor concentrate within the hospital. Unable to locate the C1 inhibitor concentrate, we obtained 4 units of fresh frozen plasma for the operating room in case of an acute attack of hereditary angioedema. Emergency airway equipment and a tracheostomy kit were also immediately available.

Standard monitors were applied, and oxygen was delivered via non-rebreather face mask.

The anesthetic included midazolam 2 mg, fentanyl 100 mcg, and infusions of ketamine 50 mg and propofol 200 mg, all administered intravenously. Spontaneous ventilation was maintained, and vital signs remained stable throughout the procedure. The procedure was completed without airway manipulation. The airway was reassessed for mucosal or lingual swelling. Post-operative airway patency was unchanged from that found during the preoperative assessment. Specific instructions were given to the post-operative care nurses related to the patient’s history of hereditary angioedema and the potential airway complications that may occur. The instructions included emergency airway interventions and the possible necessary administration of fresh frozen plasma. Arrangements were made for extended observation postoperatively for prolonged airway patency monitoring.

**Discussion**

Angioedema is an abrupt episodic swelling of the skin and mucous membranes without lesions. This is due to an increase in submucosal or subcutaneous capillary permeability leading to plasma extravasation and non-pitting edema. Laryngeal edema demonstrates as feeling of a lump, tightness of the throat and dysphagia. This subsequent swelling can encroach upon airway patency causing life-threatening airway emergencies leading to hospitalization and, in some cases, death. Hereditary angioedema is a rare form of angioedema that results from an autosomal dominant deficiency of C1 esterase inhibitor. C1 esterase inhibitor is a serine protease inhibitor that regulates the complement system, affecting the antibody-independent and fibrinolytic pathways. Normally, serpin inhibits bradykinin, factor XII, and enzymes of the contact activation
During hereditary angioedema attacks, bradykinin, a vasoactive nonapeptide, is activated and causes increased capillary permeability.1 There are two major forms of hereditary angioedema, each characterized by mutations of the C1 inhibitor gene. C4 levels measured during acute attacks may be used as a screening tool to rule out hereditary angioedema. Biochemical markers, plasma C4 levels and functional C1 inhibitor are low during acute attacks recognizing tissue bradykinin formation.4 Surprisingly, these levels are normal between attacks. A person with hereditary angioedema experiences an average of 20 attacks per year.5 Patients usually become symptomatic in the second decade of life.1 These intermittent and unpredictable attacks are associated with insults such as dental procedures, infection, snoring, daily activities (typing), and stress.5 The swelling progresses over 24-72 hours and terminates 1-3 days later.5

During attacks, patients feel sensations of stretching, tingling, and numbness. Angioedema can exhibit in the extremities, abdomen, face, and neck. Overall, the most dangerous problem of hereditary angioedema is upper airway obstruction. Upper airway swelling occurs in one-half to two-thirds of people diagnosed with hereditary angioedema. Unresponsive to antihistamines and steroids, acute attacks involving the airway can be lethal. Prophylactic treatment with androgens (danazol) is beneficial both long term and prior to procedures that may “trigger” an angioedema attack.1 Androgens (anabolic steroids) increase hepatic synthesis of C1 esterase inhibitors.1 These have been shown to be effective in decreasing attacks by >80% in 97% of patients.5

When faced with an acute attack, the first form of treatment should be the use of C1 inhibitor concentrate (25 U/kg) or fresh frozen plasma (two to four units).1 Attacks that have been treated with C1 inhibitor tend to resolve within 30-60 minutes after intravenous injection. The fresh frozen plasma replaces the deficient enzymes but can potentially worsen an attack because there are high concentrations of complement components of human plasma substrate resulting in the generation of bradykinins. Subcutaneous epinephrine (0.3mg every 10 minutes) has also shown to be transiently effective.2 Mild cases of angioedema may require only oxygen and monitoring with pulse oximetry. Severe attacks may need emergent treatment because they usually involve the airway. Managing attacks of the oropharynx focus on emergency interventions including the establishment of a patent airway and/or tracheal intubation. However, in extreme edematous attacks along the full course of the airway, even a tracheostomy may be ineffective in establishing a lifesaving airway.

A priority for anesthetic management of the hereditary angioedema patient is careful preparation and an understanding of the significant risks that face these patients. Prior to elective surgery in which airway manipulation is anticipated, patients with hereditary angioedema should be pretreated. Prophylactic treatment includes androgens (danazol), intravenous plasmin inhibitors (e.g., e-aminocaproic acid), plasma, or all three.6 It is imperative that C1 inhibitor concentrates (25U/kg) be readily available should an acute attack occur.1 In most cases C1 inhibitor concentrates are not available because of its high cost. Regional anesthesia is a safe alternative and tolerated well.1 Instrumentation of the oropharynx should be avoided if at all possible. If endotracheal intubation cannot be avoided then prophylactic techniques should be initiated. Tracheal jet ventilation has been used.
successfully but not without risk of pneumothorax. A tracheostomy kit and trained personnel can be vital to securing a patent airway. Even with proper emergency airway management preparation, swelling may extend into the lower airway and ultimately cause death.¹

Preoperative preparation of the patient in this case followed evidenced based practice guidelines. Thorough assessment of patients with hereditary angioedema, as well as understanding the pathology and associated risks are very important for preventing an acute, airway crisis during anesthesia management. The lesson learned in this case was how a detailed patient interview, assessment, and anesthetic emergency preparation is crucial to avoiding potentially life threatening episodes.

References


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Ventilator Management of the Inhalation Burn Injured Patient

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**Keywords:** Inhalation burns, intraoperative ventilator management, burn anesthesia management, barotrauma

An estimated 450,000 individuals suffer burn injuries in the United States annually, with roughly 45,000 of them facing hospitalization, and approximately 3,500 resulting in death.¹ Airway burn injury often presents a daunting task to the anesthetist; how it is managed can be the difference between life and death.¹ Managing acute lung injury involves balancing the competing objectives of insuring appropriate ventilation and oxygenation while minimizing adverse effects.² Care must be exercised to prevent further morbidity from oxygen toxicity, hemodynamic compromise, barotrauma, or alveolar over-distension.³
Case Report

A 51-year-old, 82 kg, 172 cm, male with estimated 90% total body surface area burns to the face, torso, and bilateral upper and lower extremities, presented to the operating room (OR) for debridement and escharotomy within 24-hours of admission to the hospital emergency department. The patient suffered extensive full-thickness burns to the aforementioned areas after an explosion occurred while he was working under an automobile with a lighted cigarette. No medical history was available. However, the patient was reported to be a “long-time smoker.”

Upon arrival to the hospital, a size 8.0 endotracheal tube (ETT) was placed into the trachea, as well as a right radial artery catheter and a right subclavian triple-lumen catheter. Assessment findings included a Mallampati IV airway classification, singed facial hair, and massive lip and facial burn involvement. Upper and lower airway involvement was confirmed by fiberoptic bronchoscopy. Vital signs upon arrival to the OR were as follows: heart rate 120 beat/min, blood pressure 90/49 mm Hg, and temperature 35.5°C. Positive pressure ventilation was delivered via bag-valve mask at approximately 20 breaths/min, resulting in an O2 saturation (SpO2) of 86%, as measured by ear probe pulse oximetry.

Volume controlled mechanical ventilation was initiated with a tidal volume (Vt) of 550 ml, rate of 24 breaths/min, peak-inspiratory pressure 38 mm H2O, fraction of inspired O2 (FiO2) 1.0, and positive end-expiratory pressure (PEEP) 10 cm H2O. Pertinent laboratory findings included hemoglobin of 8.1 g/dL and hematocrit of 23.3%. Arterial blood gas results revealed a pH of 7.28, PCO2 of 56 mm Hg, bicarbonate of 19 mEq/L, and PaO2 of 59 mm Hg. The carbon monoxide level was unavailable. Balanced anesthesia was initiated by administration of isoflurane 0.15%, O2 at 3 L/min, with fentanyl 25 mcg (total 325 mcg) and hydromorphone 0.2 mg (total 2 mg) intravenous (IV) boluses as hemodynamically tolerated. Neuromuscular blockade was maintained with pancuronium 10 mg IV boluses.

Warmed crystalloids (7 L) were administered using a rapid fluid infusion device, along with albumin 5% (500 ml) infusions on two occasions, packed-red blood cells 8 units, one unit of cryoprecipitate 1 unit, fresh frozen plasma 2 units, and a 10-pack of platelets. Calcium chloride 1 G IV was provided, and an epinephrine infusion was initiated at 0.02 mcg/kg/min but later discontinued. Phenylephrine 10 mcg/ml boluses were administered supportively as needed. Electrocardiogram leads were placed with surgical staples and alligator clamps.

Discussion

Oxygen transport is compromised when smoke inhalation is encountered due to hemoglobin’s increased affinity for CO, which competes with O2 for binding sites.4 Carbon monoxide combines chemically with hemoglobin in erythrocytes, having an affinity for hemoglobin approximately 210 times more than O2.4 Due to the patient’s inhalation injuries and significant cigarette smoking history, a decreased O2 carrying capacity was suspected. The need to maximize hemoglobin’s ability to transport oxygen is paramount when lung tissues have been compromised. Normal diffusion of alveolar O2 and CO2 assumes normal lung parenchyma and capillary structures. The resulting lung damage that accompanies inhalation burns reduces alveolar and capillary function exponentially. Hypoxia
and hypoxemia may lead to decreased surfactant production, and even destruction of surfactant, which can cause atelectasis.\textsuperscript{3,5}

Resorption atelectasis arises slowly and can be directly linked to small $V_t$ in combination with a $FiO_2$ of 1.0. Lung regions with low ventilation to perfusion ($V/Q$) ratios, those containing alveoli that are only partially expanded by inspired gases, appears to be the areas most affected.\textsuperscript{6} Oxygen gets “sucked-out” of the alveolus to the point of collapse. Efforts to expand atelectatic alveoli, known as “recruitment maneuvers,” were attempted many times intra-operatively by increasing $V_t$ and PEEP. Once opened, alveoli are more easily reopened with increasingly less pressure.\textsuperscript{6} Therefore, increasing PEEP after recruitment maneuvers is an accepted practice.

Alveolar edema compromises gas exchange, while edematous airways impede airflow and secretion clearance.\textsuperscript{2} During mechanical ventilation, flooding of alveoli with secretions arising from compromised gas exchange and edematous airways often produce competing effects.\textsuperscript{2} Positive end-expiratory pressure can increase arterial oxygenation by increasing functional residual capacity; however, too much PEEP may cause barotrauma to alveoli that are normally functioning and increase physiologic dead space and worsen gas exchange.\textsuperscript{5} Decreased cardiac output, increased intracranial pressure, renal dysfunction, and decreased splanchnic perfusion have been seen with increased ventilation pressures.\textsuperscript{5}

Patient positioning can have profound effects on $V/Q$. For example, reverse trendelenburg positioning decreases the alveolar-arterial $O_2$ tension gradient resulting in the same beneficial effects on oxygenation that PEEP provides.\textsuperscript{6}

Administration of inhaled nitric oxide can provide selective vasodilation of ventilated areas of the lung reducing shunting.\textsuperscript{6} Accepted rescue ventilation modalities which have positive $V/Q$ effects include high frequency percussive ventilation and high frequency oscillatory ventilation. High frequency percussive ventilation machines deliver high frequency, sub-tidal volume breaths, followed by passive exhalation to a preset baseline continuous positive airway pressure that enhances clearance of airway secretions.\textsuperscript{6} High frequency oscillatory ventilation oscillates the lung around a constant mean airway pressure higher than used with conventional ventilation. Both of these modalities afford lung protection against barotrauma. Extracorporeal membrane oxygenation (ECMO) circulates the patient’s blood through a semipermeable membrane that exchanges oxygen and carbon dioxide and can be used when other methods are ineffective.\textsuperscript{6}

The patient’s extensive anterior torso burns made mechanical ventilation efforts almost impossible. Inflexible eschar and underlying tissue edema can prevent chest wall motion and limit ventilation.\textsuperscript{1} Research findings support using low $V_t$ with acute lung-injured patients. Maintaining plateau pressures below 30 cm $H_2O$ is also suggested to reduce the risk of further airway injury.\textsuperscript{6-8} An initial $V_t$ of 800 ml was implemented in efforts to improve oxygenation. Peak inspiratory pressure reached 48 cm $H_2O$ and a PEEP of 18 cm $H_2O$ presented concern for barotrauma. Ultimately, the $V_t$ was reduced to 450 ml and PEEP was maintained at 18 cm $H_2O$, with a frequency of 28 breaths/min.

Severe burns often result in systemic inflammatory responses that can exacerbate problems related to ventilation. These responses tend to peak in severity within 48
to 72 hours of injury. Edema of the upper airways begins within hours and persist for 2 to 4 days. Micro-vascular, toxin, and broad physiologic changes are also associated with direct burn injury to the lungs. Advanced respiratory complications like acute respiratory distress syndrome, respiratory failure, V/Q mismatch, and pulmonary edema present between 1-5 days post injury, and diffusion abnormalities are increased with burns of the upper and lower airways. Additionally, atelectasis and pulmonary edema can be directly linked to copious fluid administration, and to increased physiologic dead space caused by pulmonary capillary permeability. Liberal fluid replacement, which is crucial to hemodynamic stability and renal preservation, can lead to V/Q mismatching complications.

Research supports the use of low V_{\text{t}} and application of high PEEP when providing ventilation for the acute lung-injured patient. In this instance, many of the supported interventions were used in an effort to maintain oxygenation, with exception of high frequency ventilation modalities, and ECMO, which were unavailable during the intraoperative period. In retrospect, the use of higher PEEP and a higher respiratory rate may have better facilitated alveolar ventilation. Decreasing the FiO_{2} might have decreased atelectasis during mechanical ventilation, as compression atelectasis can be linked directly with small V_{\text{t}} and FiO_{2} of 1.0. These measures, theoretically, could have reduced the risk of iatrogenic injury while improving oxygenation.

**References**


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Anesthetic Management of a Pediatric Patient with Thyroid Hormone Resistance

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Keywords: resistance to thyroid hormone (RTH), thyroid storm, thyrotoxicosis, hyperthyroid crisis, pediatric

Resistance to thyroid hormone (RTH) is a rare human genetic disease involving impaired tissue responsiveness to thyroid hormones associated with non-suppressible thyroid stimulating hormone (TSH).\textsuperscript{1} The incidence of RTH is approximately 1:40,000-50,000 live births, and inheritance is autosomal dominant.\textsuperscript{2,3} This syndrome is characterized by elevated serum thyroid hormones (thryoxine (T\textsubscript{4}) and triiodothyronine (T\textsubscript{3})) and a normal or increased thyrotropin (thyroid stimulating hormone (TSH)). There exists very little literature regarding the anesthetic management of patients with RTH. It is important to understand the implications RTH presents in the perioperative period in order to safely care for the affected patient and prevent complications.

Case Report

An 8-year-old, 27 kg, 124 cm (BMI 17.4) female presented for laparoscopic appendectomy after a diagnosis of appendicitis. Past medical history included RTH, asthma, growth hormone deficiency, and developmental delay. No past surgical history existed for the patient and no family history of complications with general anesthesia was reported. The patient’s medications included daily inhaled budesonide, daily oral montelukast sodium, and oral atenolol used to manage episodes of tachycardia as needed. No known drug allergies existed. Preoperative vital signs were all within normal limits for her age. Thyrotropin was within normal limits, while T3 and T4 were elevated. The patient had no recent hospitalizations with the exception of the current admission. Pre-operative airway assessment was benign, revealing no abnormal masses indicative of airway obstruction. A general anesthetic with inhalational induction and endotracheal intubation was planned.

Upon arrival to the operating room, an inhalational induction was performed with a mixture of oxygen 3 L/min, nitrous oxide 7 L/min and sevoflurane incrementally titrated to 8%. The patient remained cooperative and non-combative during induction. Intravenous (IV) access was established and nitrous oxide was discontinued. The patient was oxygenated with O\textsubscript{2} 10 L/min while fentanyl 50 mcg IV and vecuronium 1 mg IV were administered to assist with laryngoscopy and oral endotracheal intubation. Direct laryngoscopy was performed and a 6.0 cuffed endotracheal tube was advanced past the vocal cords into the trachea. Correct placement of the endotracheal tube was confirmed with the presence of end-tidal CO\textsubscript{2} and bilateral breath sounds. Anesthesia was maintained with sevoflurane 2.0-3.0% inspired concentration in a mixture of oxygen 1 L/min and air 1 L/min. A continuous remifentanil infusion 0.5 mcg/kg/min was initiated after induction. An esmolol infusion was prepared, clamped, and connected to the patient’s IV line, immediately available for administration and titration should tachycardia become evident. A laparoscopic appendectomy was performed.
performed in approximately 30 minutes without any unusual incidents. The patient’s vital signs remained stable and within normal limits.

Upon the surgery’s conclusion, neuromuscular blockade was antagonized with the administration of neostigmine 2 mg IV and glycopyrrolate 0.2 mg IV. Sevoflurane and the remifentanil infusion were discontinued, and the trachea was extubated upon meeting all criteria for awake extubation. Her vital signs remained stable and within normal limits during emergence and removal of the endotracheal tube. The patient was then taken to the post anesthesia care area for recovery; she exhibited no unusual signs or symptoms and vital signs remained within normal limits.

Discussion

Resistance to thyroid hormone is a rare genetic disorder characterized by reduced peripheral tissue sensitivity to serum thyroid hormones, T3 and T4. The syndrome is distinguished by elevated levels of T3 and T4 co-existing with inappropriately normal or elevated levels of TSH.1 The presence of these laboratory parameters with the exclusion of other conditions that may mimic these results compel the diagnosis of RTH versus other disease-states exhibiting hyperthyroid conditions.1,3,4 Normally, an increased release of thyroid hormone creates negative feedback within the thyrotropic cells in the pituitary, reducing TSH secretion.5 The majority of RTH patients are asymptomatic, eumetabolic and clinically euthyroid, which exists at the expense of increased levels of thyroid hormones with a non-suppressed TSH.3,4 Many patients require no specific treatment.3,6 Clinical manifestations of hyperthyroidism, such as tachycardia, can be effectively treated with cardioselective beta-blockers.6 Other possible clinical signs of RTH include goiter (which can result in upper airway compression), delayed skeletal maturation, decreased weight, hearing loss, attention-deficit hyperactivity disorder, decreased intelligence quotient, learning disabilities, developmental delay, and tachycardia.1,2 Resistance to thyroid hormone is relatively rare, with just over 1000 registered cases.7

Presented here is a case of the successful anesthetic management of a pediatric patient afflicted with RTH undergoing a laparoscopic appendectomy. As there is a dearth of literature describing patients with RTH undergoing anesthesia, the optimal method for anesthetic management of patients with RTH has not been established. The various clinical manifestations of RTH, as well as the potential for adverse events, behoove the anesthesia practitioner to perform a thorough preoperative assessment and remain vigilant during the perioperative period while caring for these patients. The successful outcome of this case was attributed to meticulous preparation in anticipation of potential adverse events associated with RTH. A careful preoperative airway assessment was performed, difficult airway equipment was immediately available to manage undiagnosed airway compression, a continuous remifentanil infusion was maintained intraoperatively to blunt sympathetic stimulation, and an esmolol infusion was immediately available to treat tachyarrhythmias.

Patients with RTH are at increased risk for undiagnosed airway compression due to masses, goiters, and thyroid enlargements.2,4 Furthermore, since patients with RTH can have occasional manifestations of hyperthyroidism, they are at risk for thyrotoxicosis, otherwise known as thyroid storm, which is a life-threatening state of hyperthyroid crisis.6 In a review of the
literature, one particular case report presents twins with RTH showing increased peripheral tissue resistance to the effects of thyroid hormone except for their hearts, which retained sensitivity to thyroid hormones and were subject to the hyperthyroid effects of increased thyroid hormone. Thus, the real potential for undiagnosed airway compression, thyrotoxicosis, and the development of harmful tachyarrhythmias exists.

In the present case, one of the main goals of anesthetic management was the avoidance of intraoperative stress factors which could stimulate the sympathetic nervous system (SNS) and induce thyrotoxicosis. Mindful of this, careful decisions were made to choose anesthetic options which would mitigate development of this potentially lethal condition. For induction, vecuronium was selected for neuromuscular blockade instead of rocuronium, due to the latter’s minor yet existent vagolytic tendencies and risk of precipitating an increase in heart rate. The next challenge involved establishing an adequate anesthetic depth, which was imperative to avoid exaggerated SNS response and thyrotoxicosis. An optimal level of anesthesia was sustained with the use of a remifentanil infusion in combination with inhaled sevoflurane of 1.0-1.5 MAC. Fluids and phenylephrine were immediately available for the management of hypotension. Drugs possessing the potential for stimulating the SNS, including ephedrine, desflurane, pancuronium, and ketamine, were avoided. An esmolol infusion was prepared and immediately available for titration in the event that sustained intraoperative tachycardia occurred due to thyrotoxicosis. Reversal of neuromuscular blockade was achieved by administering neostigmine 0.07 mg/kg IV and glycopyrrolate 0.005 mg/kg IV. In order to reduce the risk of tachycardia due to muscarinic blockade, the dose of glycopyrrolate was selected using the lower range for pediatric patients, i.e. 0.005-0.01 mg/kg.

Another important consideration presented by the literature involved managing the perioperative threat of thyrotoxicosis with the presence of a cooling blanket or ice packs. Both would have been helpful in the management of hyperthermia in the event of thyrotoxicosis. It may have been tempting to premedicate the patient with thiamazole, an antithyroid medication, to prevent possible hyperthyroid crisis. However, the high levels of circulating thyroid hormone in patients with RTH are considered to be an adaptive response to tissue resistance, and antithyroid medications like those given to patients with primary thyrotoxicosis are contraindicated. In fact, failure to differentiate RTH from primary thyrotoxicosis has resulted in the inappropriate treatment of nearly one-third of patients. An extremely important caveat involves recognizing that the symptoms of thyrotoxicosis (hyperpyrexia, tachycardia, and hypermetabolism) often mimic malignant hyperthermia (MH). The anesthesia practitioner should be wary of the potential of MH as a differential diagnosis in this patient population. Distinction between the two intraoperatively and postoperatively can prove to be very difficult, especially in pediatric patients who are hyperthyroid.

In conclusion, the perioperative management of a patient with RTH can be challenging and there is no precedent which establishes optimal technique. Presented here is the successful anesthetic management of a pediatric patient with RTH consistent with the existing current literature and the best available evidence. Further studies should be performed regarding the perioperative management of RTH patients.
due to the relative dearth of information available on this topic.

References


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**Anesthetic Management of a Patient with DiGeorge Syndrome**

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**Keywords:** DiGeorge, 22q11.2 Deletion Syndrome, Ventral Septal Defect, Nissen Fundoplication, Pediatric

DiGeorge, conotruncal face, and velocardiofacial syndromes are most commonly caused by chromosome band 22q11.2 deletions. The incidence for the collective syndromes is 1 in 4000 and identified as chromosome 22q11.2 Deletion Syndrome (22q11.2 DS). Cardiac defects, abnormal facies, thymic hypoplasia, cleft palate, and hypocalcemia (CATCH 22 syndrome) are all commonly present. Patients with 22q11.2 DS require surgery for multiple anomalies associated with the syndrome; heart repair and gastrointestinal modification are common. Thymic hypoplasia presents an immunodeficiency concern. In 88% of patients with DiGeorge Syndrome, the 22q11.2 DS is present.

**Case Report**

A 28-day-old, 3.2 kg, full-term male neonate (with no delivery complications) diagnosed with DiGeorge Syndrome and 22q11.2 DS was scheduled for a Nissen Fundoplication and insertion of gastrostomy tube. His diagnosis was dysphagia. The patient failed
a barium swallow study test indicating aspiration. Two weeks prior, the patient had a ventricular septal defect (VSD) patch repair with no complications. Status post VSD repair, a 2D echo showed the patch in place with an ejection fraction (EF) of 65%. He also had thymic hypoplasia. The patient presented, from the PICU, to surgery on O₂ at 3 L/min via nasal cannula, SpO₂ of 92% was noted. Ranitidine 4 mg IV was given preoperatively. A left foot saphenous 24 gauge intravenous line was in place with D5 0.45% NS infusing at 12 ml/hr. A nasogastric tube (NGT) was present. The patient’s ASA Physical Status was a III.

In the operating room, standard monitors were applied. A difficult airway cart was deemed unnecessary due to review of previous anesthetic records and physical assessment. The NGT was suctioned with no return. Pre-oxygenation by mask with O₂ at 8 L/min was delivered until an SpO₂ of 100% was achieved. An intravenous induction was started with fentanyl 5 mcg, propofol 15 mg, and rocuronium 2 mg. Direct visual laryngoscopy was performed with a 0 Miller blade yielding a Grade I laryngeal view. A 3.0 mm internal diameter uncuffed endotracheal tube was inserted with a leak at 15-20 cm H₂O and secured at 9 cm at the lips. Adequate chest expansion, bilateral breath sounds, condensation in the tube, appropriate tidal volumes, and positive end-tidal carbon dioxide were present. Cefazolin 75 mg was administered. Next, a double lumen right femoral central venous catheter was placed, by surgeon, with NS to be infused.

General anesthesia was maintained with O₂ at 2 L/min and sevoflurane 3.3%. A second dose of fentanyl 5 mcg was given as well as a second and third dose of propofol of 5 mg and 3 mg respectively. Neuromuscular blockade was antagonized at the end of the case with neostigmine 0.2 mg and glycopyrrolate 0.04 mg. Total fluids for the case included 12 ml of D5 0.45% NS and 35 ml of NS (both were delivered through fluid warmers). After the neonate demonstrated 5-second tetanus without fade, 5-second head lift, and regular respirations the airway and stomach were suctioned and the trachea was extubated with positive pressure. The patient was transported to the PICU in right lateral decubitus position while maintaining adequate unassisted spontaneous ventilation on monitors and blow-by O₂.

**Discussion**

In 1968, DiGeorge Syndrome was first described. Subsequent research has shown that 88% of patients with DiGeorge Syndrome have a deletion in chromosome 22. Dysfunction of the neural crest cell and anterior heart field along with other anomalies are traced to the early embryonic development stage. Velocardiofacial and conotruncal anomaly face (CTAF) patients also share clinical features with DiGeorge Syndrome, also resulting from a micro-deletion in chromosome 22. The patient with 22q11.2 DS may present with multiple clinical manifestations with high variability among related and unrelated patients. There are 180 signs and symptoms which have been described with 22q11.2 DS. The major clinical features associated with 22q11.2DS include: congenital heart disease (CHD), immunodeficiency, hypocalcemia, palate anomalies, speech disorders, failure to thrive, otorhinolaryngologic issues, dysmorphic facies, renal anomalies, skeletal anomalies, cognitive disabilities, and psychiatric disorders.

Neonates with 22q11.2 DS have an 80% occurrence of cardiovascular anomalies and 13-18% have a VSD. Tetralogy of Fallot, interrupted aortic arch, and truncus
arteriosus communis are the most common CHD cardiac lesions associated with 22q11.2 DS. Kyburz et al. concluded that children with 22q11.2 DS and congenital heart defect are at high risk for mortality and morbidity, dependent on the severity of the cardiac lesions and other noncardiac anomalies. According to Kyburz et al.’s original research, patients with 22q11.2 DS and a VSD had a 36% mortality rate at a 10-year follow up. Furthermore, Kyburz et al. found the neonatal period had higher mortality rates and of the patients studied 6% died perioperatively. Patients with a small VSD may experience minimal complications and the VSD may close spontaneously within the first five years. However, a larger VSD will complicate the patient’s status, usually by developing congestive heart failure around one year of age and, in many times, if corrective surgery is not performed in these advanced large VSD patients, it becomes too late for surgery. Thus, patients with large VSDs typically have surgery within the first year.

Potential complications post-operatively for a VSD patch are: pulmonary hypertension and RV dysfunction, residual left-to-right shunt, LV dysfunction, heart block, respiratory insufficiency, and aortic insufficiency. This patient had a large VSD and required surgery two weeks prior. The patient’s 2-D echo showed an appropriately placed VSD patch with no complications and an EF of 65%. The patient also showed no VSD patch complications perioperatively with his current surgery.

Patients with DiGeorge Syndrome are at risk for aspiration perioperatively. Patients with DiGeorge Syndrome may develop a failure to thrive due to lack of nutrition. This patient was not able to receive nourishment through his gastrointestinal tract because of his high risk of aspiration and nutrition was a concern. Nissen fundoplication and a gastrostomy tube would provide nourishment for this patient. The major concerns for this patient regarding anesthesia were VSD complications and the risk of aspiration. Although the patient had dysmorphic facial features, intubation of the airway was not difficult. Due to his hypoplastic thymus and possible immunological deficiencies, meticulous implementation of aseptic and sterile techniques was stressed. Labs were normal, no hypocalcemia. The neonate with DiGeorge Syndrome may present with a 49% incidence of hypocalcemia, which can lead to muscle cramps and even seizures. No renal or skeletal anomalies were noted. His palate was unremarkable. Cognitive, psychiatric, and speech disorders would have been premature to evaluate. This patient successfully underwent surgery without complication.
References


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**Anesthesia Management in a Newborn with an Encephalocele**

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**Keywords:** encephalocele, microencephaly, neural tube defect, craniotomy, Chiari type 3

Encephalocele is a rare neural tube defect resulting from failure of the neural tube to close completely during fetal development. This disorder is characterized by a sac-like protrusion containing cerebral spinal fluid and membranous layers that cover the spinal cord and skull. Furthermore, this sac may include herniated brain tissue. Encephaloceles are frequently associated with other craniofacial abnormalities including microcephaly and brain malformations. This is a case report of the anesthetic management of a newborn with an occipital encephalocele and a Chiari type 3 malformation.

**Case Report**

The patient was a 6-day-old male born at 37 weeks via elective cesarean section (weight 2.4 kg, height 0.42 m) to a healthy 28-year-old G2P1001. The patient’s medical history was significant for microcephaly, encephalocele, intrauterine growth restriction, and 46 XX chromosome.

The patient was subsequently scheduled for a craniotomy and skull autograft cranioplasty repair of an occipital encephalocele. Due to witnessed apnea at birth and the encephalocele, the patient was monitored in the neonatal intensive care unit (NICU). The encephalocele measured approximately 9.4 cm x 7.2 cm x 8.6 cm as
reported by CT scan. The patient was not fed for 4 hours prior to surgery.

Upon arrival to the perioperative holding area the infant was quiet in the lateral position with his head hyperextended and mouth open. While in the pre-operative suite, audible rhonchi could be heard during normal tidal volume breathing.

Intraoperatively, three blankets were stacked under the patient’s body in order to lay the patient supine without pressure on the fluid filled sac. Following IV induction with propofol, intubation was attempted by direct laryngoscopy. The first direct laryngoscopy attempt resulted in an esophageal intubation as a result of difficulty visualizing the cords. To aid in better vocal cord visualization, the patient’s shoulders were elevated off of the blanket rolls and extra support was given to the sac while extending the patient’s head. This technique allowed for better visualization and a successful tracheal intubation with a 3.0 mm cuffed endotracheal tube. Two 24 gauge intravenous catheters were in place, and a left femoral 22 gauge arterial line was inserted. Multiple attempts were made to establish a central line under ultrasound guided technique, without success. A foley catheter was placed by the circulating nurse. During the procedure the patient was positioned prone with his head secured in a DORO® headrest. His chest was supported with pediatric gel rolls laterally, and a precordial Doppler was secured on the left anterior chest wall. Anesthesia was maintained for the procedure with 0.6% sevoflurane in oxygen 1 L/min and air 1 L/min. Once the surgeon began operating on the exposed brain tissue and cerebellum, frequent 20-30 bpm decreases in heart rate were observed. Atropine 0.1 mg was administered for bradycardia with a rate of 92 beats/min and the surgeon was notified. Transient drops in heart rate improved with repositioning of the surgical instruments. Pressure-controlled mechanical ventilation was utilized maintaining tidal volumes of 34-41mL and a respiratory rate of 18 breaths/min.

The patient received a total of packed red blood cells 225 mL, plasmalyte150 mL, 5% albumin 60 mL, D10W 69 mL, calcium gluconate 200 mg, sodium bicarbonate 4.2% 8 mEq, atropine 0.1mg, cisatracurium 3.5 mg, cefazolin 124 mg and fentanyl 5 mcg. The fluid filled encephalocele drained approximately 150 mLs of cerebral spinal fluid, estimated blood loss was 100 mL, and his urinary output was 55 mLs via foley catheter. The patient was transferred to the NICU with the ETT in place with manual ventilation of oxygen 10 L/min. He remained hemodynamically stable during the transfer.

Discussion

Nervous system malformations are common in pediatric patients and frequently result in early mortality. During normal growth and development, closure of the neural tube occurs in a 4-6 day period with completion occurring around the 29th day of postconception. Encephalocele is a rare neural tube defect occurring in 1 to 3 in 10,000 live births. In addition, more than 70% of encephaloceles are found posteriorly and herniate through the defect in the occipital portion of the skull. This defect consists of neural tissue and meninges that herniate through deficient skin and bony structures either anteriorly and posteriorly. This patient’s posterior herniating tissue caused vagal response symptoms when stimulated. Initially these 30 beat decreases in heart rate were managed with atropine 0.1 mg. Subsequent episodes required open
communication with the surgeon and discontinuation of that particular surgical stimulation to minimize the decrease in heart rate.

Anterior encephalocele may be associated with anomalies of underlying brain or orbital structures, or involve the pituitary gland. Nasofrontal encephaloceles may be obvious as nasal obstructions or a cerebrospinal fluid leak. These nasofrontal abnormalities may manifest late in life. Posterior encephaloceles carry a high mortality risk. Survivors have severe neurodevelopmental disabilities. Furthermore, hydrocephalus occurs in 50% of patients with occipital encephalocele. Agenesis of the corpus callosum, Chiari III malformation, subependymal nodular heterotopia and blindness are other common findings. This patient’s encephalocele drained approximately 150 milliliters of CSF upon incision. The patient’s vital signs greatly fluctuated, requiring a large volume of crystalloid and colloid infusion. Unfortunately, these large amounts of fluid resulted in dilutional thrombocytopenia with a platelet level of 18,000 µL. Removing CSF can result in serum electrolyte imbalances which may occur during aspiration of CSF, hypothermia, and blood loss. Rapid opening and drainage of the sac may trigger hemodynamic changes such as bradycardia and even cardiac arrest. Recent literature suggests continuous arterial monitoring and slow controlled efflux of CSF.

Posterior encephaloceles can be a significant airway and positioning challenge. Depending on the size and location of the encephalocele, the patient may not be able to lie supine without putting pressure on the sac. Restriction in neck movement may present a challenge with direct laryngoscopy. Numerous positioning aids are available to allow for easier laryngoscopy and sac protection. Silicone and foam supports may be on top of each other until the height matches that of the encephalocele sac. These allow the neonate to be placed supine on the devices, while one individual supports the head. Alternatively, blankets may be stacked under the patient’s body, preventing pressure on the occipital sac in the supine position. Placing the neonate in the lateral position allows direct laryngoscopy without the use of commercial aids and avoids pressure on the posterior sac. Our attempt at this method was unsuccessful since the patient’s sac extended so far posteriorly that a majority of the upper torso was without support on the blankets. Another option requires one practitioner to stabilize the patient’s head, while another supports the pelvis, allowing the third anesthesia practitioner to intubate the trachea. This technique provided us the ability to adjust the patient easily, allowing for direct visualization of the vocal cords and successful intubation.

Different airway devices may be required to establish a secure airway. Furthermore, various commercial devices are available for purchase to assist with patient positioning and securing of the encephalocele sac. These devices prevent direct pressure and possible rupture of the sac. Airway preparation was the most crucial step in the anesthesia care plan. Although the initial visualization was challenging, extra anesthesia practitioners proved to be the key to obtaining adequate sniffing position. Diligent care must be taken to protect the posterior sac from pressure and possible rupture. The choice of support or products used to accomplish this must be unique for the individual patient. Lastly, the importance of planning for a crisis must not be underestimated. An emergency airway protocol should be
employed as part of preparing for a difficult airway related to positioning challenges.

Reference


Mentor: Brenda Wands, CRNA, MBA, PhD

Submental Intubation in Oral Maxillofacial Trauma Surgery

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Keywords: airway management, submental endotracheal intubation, submandibular intubation, maxillofacial trauma injury, mylohyoid intubation.

Airway management in maxillofacial trauma surgery often requires special consideration and alternate methods for surgical access instead of conventional endotracheal intubation. Additionally, prolonged intubation time may not be required thus submental endotracheal intubation is an appropriate alternative. The submental endotracheal intubation technique first described by Hernandez Altemir (1986)\(^1\) provides a practical and secure way of airway management rather than traditional tracheostomy. With this technique, the endotracheal tube is passed through the submental triangle through the floor of the mouth. While tracheostomy remains the mainstay for maxillofacial trauma surgery its use is not without significant risks. Risks associated with tracheostomy include tracheal stenosis, tracheoesophageal fistula, tissue scarring, infection, and even death.\(^2\)

Case Report

A 20-year-old, 70 kg, 190.5 cm Caucasian male presented to the emergency room status post motor vehicle accident versus wall. The patient was unrestrained, ejected from the vehicle, and sustained multiple fractures of the face and left radius and ulna. At the scene he was immediately intubated to maintain airway patency due to hypoxia, airway bleeding, and loss of consciousness. A computed tomography scan revealed bilateral pneumothoraces and a small subdural hematoma to the right side which
did not require neurosurgical management. He was then transferred to the operating room for immediate open reduction internal fixation (ORIF) of the left forearm. His initial surgery proceeded without incident or anesthesia complication, and he was transferred intubated to intensive care unit.

Two days post-ORIF the patient required further surgery to repair the facial and orbital fractures sustained in the accident. The patient had a significant past surgical history for tonsillectomy/adenoidectomy, right hip ORIF, right foot/ankle repair, and ORIF of left forearm. Past medical history included well-controlled asthma with occasional use of fluticasone/salmeterol inhaler and smoking. The patient’s physical examination revealed marked facial edema and lacerations over the entire face and scalp. Due to limited mouth movement and inability to prognath the patient’s airway examination included a Mallampati score of 4, with a thyromental distance greater than 3 fingerbreadths (6.5cm). The limited mobility of the patient's mouth was attributed to bilateral zygomatic, pterygoid, mandibular, maxillary, and LeForte I-III fractures.

Prior to arrival in the operating room midazolam 2 mg was given for anxiolysis. Before the start of induction, an ear-nose-throat (ENT) surgeon and emergency airway equipment (video laryngoscopy, tracheostomy kit, bronchoscopes, LMA, and airway introducers) were present in case the need for a surgical airway was deemed necessary. A standard induction was performed with the patient denitrogenated with oxygen 10 L/min via facemask for 10 min. Induction medications were administered as follows: lidocaine 2% 60 mg, fentanyl 100 mcg, propofol 150 mg, glycopyrrolate 0.2 mg and succinylcholine 100 mg. The trachea was intubated via direct laryngoscopy with a 7.5 cuffed Oral Rae endotracheal tube (ETT) with a Grade I Cormack-Lehane view of the vocal cords. Positive end-tidal CO₂ and bilateral breath sounds were present. Rocuronium 50 mg was administered after 4/4 post-tetanic twitches were noted and throughout the case for maintenance of neuromuscular blockade.

After the endotracheal tube was secured the ENT surgeon made a 2cm incision at the inferior border of the submental triangle and pulled the endotracheal tube through the incision. During this process the patient was disconnected from the ventilator for approximately two minutes while pulling the ETT through the submental aperture. Hemodynamic parameters remained stable throughout the formation of the submental incision. Correct endotracheal tube placement was verified by video laryngoscopy, adequate ETCO₂, bilateral breath sounds, and easy ventilation. The ETT was sutured to the skin with 3-0 nylon suture and taped for added security.

Anesthetic depth was maintained with Sevoflurane at 2% and ventilator settings were as follows: synchronized intermittent mandatory ventilation (SIMV) rate 8 breaths/min, tidal volume 650 ml, FiO₂ 50%, I:E ratio of 1:2, air 1 L/min and oxygen 1 L/min, with SpO₂ 100%. For maximum hemodynamic control, a right radial arterial line and additional intravenous line were placed. Fluid maintenance was achieved with Plasmalyte infusing at 150ml/hour and dilaudid was utilized throughout for adequate pain control. There was minimal blood loss with approximately 500 ml documented for the duration of the surgery. Upon completion of surgery the endotracheal tube was disconnected and passed in reverse order through the submental incision and normal endotracheal intubation was resumed. The Oral Rae ETT was replaced with an ETT exchanger and a
standard 7.5 oral ETT was employed; correct placement was verified via fiberoptic and video laryngoscopy. Lastly, the external submental incision was sutured and the intraoral incision healed secondarily. The duration of the surgical case was 12 hours with no perioperative anesthesia complications noted. The postoperative course was uneventful; the patient was escorted to the intensive care unit where his postoperative care was maintained. He was successfully discharged five days postoperatively in stable condition.

Discussion

Although tracheostomy is the mainstay for airway management in maxillofacial trauma, literature suggests new alternatives to its use. Airway management can be complex in patients with maxillofacial trauma. The need for an unobstructed surgical field without any interference and simultaneous assessment of soft tissues creates the need for alternative airway management in this patient population. Nasotracheal intubations are not ideal in maxillofacial trauma due to increased risk of passing the endotracheal tube through the cranium causing cerebrospinal fluid leaks, meningitis, sepsis, or severe neurological trauma. Conversely, oral intubation may interfere with proper maxillomandibular reduction. These methods also do not provide adequate surgical access if fixation of oral, nasal, and LeFort I-III fractures of the face is required. Submental orotracheal intubation offers such an alternative while reducing surgical complications and the need for tracheostomy. Submental intubation is especially suitable if postoperative intubation is only short-term or mechanical ventilation is not required. Thus, unnecessary and unwarranted complications associated with tracheostomy can be avoided using this method of intubation. Though submental intubation offers a practical and secure way to manage the airway it is not without risks. Potential complications associated with submental orotracheal intubation include superficial infection of the submental incision, trauma to Wharton’s ducts, bleeding, and sublingual gland injury. Shenoi, et al., in their experience review concluded that submental intubation is associated with low morbidity and can replace tracheostomy in selected cases of maxillofacial trauma. The submental intubation technique is contraindicated in patients who require a prolonged period of assisted ventilation. Caubi, et al. recommended the use of a 7.0 or 7.5 diameter tube to reduce the risk of deviation or compression of the tube. Submental intubation is contraindicated in patients with severe neurological damage or major thoracic trauma who need repeated surgical interventions.

The decision to proceed with submental intubation for this patient was based on the short-term intubation requirements needed. The patient did not present as a difficult airway and was previously intubated and extubated without incident. Although the computed tomography scan showed a small right subdural hematoma, he did not elicit any neurological deficits and no further neurological intervention was compulsory. Additionally, the panfacial fractures sustained did not allow for standard or nasotracheal endotracheal intubation due to the need for unobstructed surgical access to facial and oral structures. Performing a traditional tracheostomy was deemed inappropriate and a modified submental intubation with a 7.5 Oral Rae endotracheal tube was performed. The surgery proceeded without any anesthesia complications and the patient was successfully discharged five days postoperatively. Initially the airway was secured via traditional endotracheal
intubation and secured in place with tape while the submental incision was made per the ENT surgeon. This technique demonstrated more secure control of the airway with no impedance in the surgical field. There were no problems passing the endotracheal tube through the submental incision. There was minimal bleeding noted from the submental incision site. Submental oro-tracheal intubation proved to be the optimal method of airway management for this patient. It allowed uninterrupted surgical access while avoiding tracheostomy. If presented with this type of case in the future utilizing an armored reinforced endotracheal tube might be helpful to avoid any tube kinking due to acute angles of the Oral Rae tube. Although an armored tube was not used there was no damage to the endotracheal tube and no anesthesia complications noted.

References


Mentor: Brenda A. Wands, CRNA, MBA, PhD

Knowledge of Cricoid Pressure by Certified Registered Nurse Anesthetists

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Keywords: cricoid pressure, Sellick’s maneuver, Certified Registered Nurse Anesthetists

Introduction

Pulmonary aspiration is a risk of anesthesia with potential negative outcomes. Cricoid pressure, or “Sellick’s maneuver,” is used by anesthesia professionals to prevent aspiration of gastric contents during laryngoscopy. There is variability in application of this maneuver that may be related to a deficiency in proper training and education on competent application. The purpose of this research study was to determine education methods of cricoid pressure application in correlation with integration into practice. The hypothesis was that there is an inconsistency of training and education received by nurse anesthetists that may decrease the efficacy of cricoid pressure application.

Methods

A non-experimental, anonymous survey containing 10 questions was sent out to 1,000 randomly-selected nurse anesthetists throughout the United States. The survey consisted of demographic questions, years in anesthesia practice, the presence or absence
of formal training, education of cricoid pressure application, regular incorporation into daily practice, finger technique, pressure applied, and related negative outcomes. SPSS software was utilized in the analysis of data collected.

**Results**

Of the 1,000 surveys sent, there were 241 completed responses (24.1%). There were 204 respondents (84.7%) formally trained in the application of cricoid pressure, while 37 (15.3%) were never formally trained. When respondents were asked if they had experienced negative outcomes from not utilizing cricoid pressure, 19.8% said “Yes” (47/237), while 80.2% said “No”. The most frequent negative outcome reported was vomiting during induction. When asked if the respondents had experienced negative outcomes from utilizing cricoid pressure, 18.6% said “Yes” (44/237), while 81.4% said “No”. The most frequent negative outcomes reported with using cricoid pressure were: 1) trouble passing the endotracheal tube, 2) distortion of the airway, 3) difficult laryngoscopy, and 4) impaired view for intubation. The majority of respondents (87.8%) believe that it would be beneficial to patient care to incorporate training on cricoid pressure in advanced airway clinics.

**Discussion**

Demographics (age, gender, and years of practice) had no correlation to the incidence of formal training in the use of cricoid pressure. The research questions of this study included: is the education currently and previously offered regarding application of cricoid pressure insufficient and ineffective for practitioners to consistently know how to perform the maneuver correctly in the clinical setting? The research also questioned whether the degree of detail used in teaching Sellick's maneuver through formal training correlates with the knowledgeable application of the technique in the clinical setting. A higher percentage of knowledge of the appropriate amount of pressure to be applied was shown by those indicating previous formal training (33.8% compared to 18.9%). There was no correlation with a history of formal training for questions involving the correct number and choice of particular fingers. A limitation to this study was that a high percentage of CRNAs reported having been formally trained, preventing further statistical analysis. A follow up study with a manikin and hidden pressure gauge to observe how practitioners are actually performing cricoid pressure may be an appropriate next study.

**Mentor:** Lynn L. Lebeck, CRNA, PhD
Does cerebral oximetry monitoring decrease the incidence of cerebral ischemia?

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Keywords: cerebral oximetry monitoring, sitting shoulder surgery, cerebral ischemia, beach chair position, cerebral perfusion pressure, near infrared spectroscopy

Introduction

The sitting position, also known as beach chair position (BCP), has commonly been used in shoulder surgery since the 1980s. The standard BCP is described as sitting the patient upright at 45-90 degrees.¹ The BCP provides several advantages for the surgeon, including avoidance of brachial plexus strain, reduced risk of direct neurovascular trauma, excellent intra-articular visualization, and ease of conversion to an open procedure if necessary.¹ However, BCP may result in decreased cerebral blood flow, especially when combined with hypotension and low end tidal carbon dioxide (ETCO₂).² The decrease in cerebral blood flow can cause cerebral ischemia, spinal cord injury, visual loss, and ophthalmoplegia. Events have recently been reported, including 4 cases of brain and spinal cord ischemia and 8 cerebrovascular events.¹ There were also cases of reported visual loss and ophthalmoplegia.¹ Adverse outcomes have alerted the anesthesia and surgical communities to further assess the situation. Cerebral ischemia can contribute to undesirable outcomes, indicating that the standard monitoring modalities currently utilized by anesthetists may no longer be sufficient.

Near infrared spectroscopy (NIRS), also known as cerebral oximetry, provides a non-invasive way to measure regional cerebral oxygen saturation (rScO₂).² Some studies have indicated that cerebral oximetry monitoring can play an important role in detecting early cerebral hypoperfusion in procedures at risk for adverse neurologic outcomes, such as shoulder surgery in the sitting position.² NIRS developed to assess cerebral oxygenation is easy to use, less expensive than other invasive techniques, and can be quickly and easily applied.³ Near infrared light readily penetrates the skull, making it possible to perform real-time assessment of regional (frontal) cortical oxygenation (rSO₂). This is done by placing an adhesive strip of electrodes on the forehead.⁴ The electrodes may need to be covered with a towel to prevent light from interfering with the reading. The output from the monitor reflects the balance between oxygen delivery and utilization by that region. The typical range of rSO₂ is 55-80%. Absolute values less than 50% or a 20% drop from baseline often indicate that an intervention is needed, such as correcting the patient’s MAP or ensuring the head and neck are in alignment.⁴ Current literature from 2009 to 2012 was reviewed to determine if cerebral oximetry monitoring decreased the risk of cerebral ischemia in patients undergoing shoulder surgery in the sitting position.

Methodology

Evidenced Based Practice Model
The Iowa Model of Evidenced Based Practice was utilized to identify the clinical problem. The PICO format was used to formulate the research question. The PICO guidelines include patient population, current intervention, contrasting
intervention, and the outcome of interest. Patients undergoing shoulder surgery in the sitting position was the population of interest. Cerebral oximetry use was the current intervention. The contrasting intervention was not using cerebral oximetry, and the outcome of interest was the incidence of cerebral ischemia.

**Purpose**
The purpose of this review was to determine if the use of cerebral oximetry monitoring in patients undergoing shoulder surgery in the sitting position decreased the incidence of cerebral ischemia. The posed clinical question was, “Does intra-operative cerebral oximetry monitoring decrease the incidence of cerebral ischemia in patients undergoing shoulder surgery in the sitting position?”

**Search Terms**
Sitting shoulder surgery, beach chair position, cerebral oximetry, cerebral oximetry in shoulder surgery, cerebral ischemia in shoulder surgery, adverse neurologic outcomes in beach chair position, cerebral perfusion, cerebral oxygen saturation, near infrared spectroscopy.

**Search Models**
A systematic keyword search was utilized using the electronic databases of CINAHL, PubMed, Medline, Cochrane Library, and OVID from the years 2007-2012. Articles were limited to those written in English in the past 8 years, the position (BCP), the surgery (shoulder), cerebral oximetry use, and focus was placed on neurological outcomes.

**Levels of Evidence**
There was one randomized control trial that provided level I evidence, 2 case report articles that provided level III evidence, one article that provided level IV evidence, and the remaining 4 articles provided level II evidence. To determine the level of evidence, Stetler’s evidenced rating scale was used.

**Literature Review**
Scheeren, Schober, and Schwarte discussed the background and current applications of monitoring tissue oxygenation by near infrared spectroscopy (NIRS). In their review, they discuss the frequency of tissue hypoxia in the surgical setting and surgeries in which NIRS may be useful. They state that measuring and obtaining adequate tissue oxygenation may prevent postoperative complications; therefore, may be cost effective. The authors state that one application of cerebral NIRS monitoring is during shoulder surgery in the BCP. In shoulder surgery cerebral desaturation may be caused from postural hypotension, head and neck manipulation, or by thromboembolic events. When comparing the lateral decubitus position with the BCP, the patients in the BCP had significantly more cerebral desaturations (80% versus 0%). Although there was more cerebral desaturation in the BCP, there were no adverse neurological events reported. The authors suggested that perhaps this was due to the short surgery times in these patients.

Jeong et al. examined the effects of different anesthetic agents on cerebral oxygenation in patients having shoulder arthroscopy in the BCP. They examined whether jugular venous bulb oxygenation (SjvO2) and cerebral tissue oxygen saturation (SctO2) were interchangeable. Patients were assigned randomly to a sevoflurane-nitrous group or to a propofol-remifentanil group. Mean arterial pressure (MAP), heart rate, SjvO2, and SctO2 were measured before and after placing the patient in the BCP. The MAP remained significantly higher in the sevoflurane-nitrous group and the SjvO2 in the sevoflurane-nitrous group was higher.
The SctO2 and the incidence of cerebral desaturation did not significantly change between groups. The authors concluded that “the margin of safety against impaired cerebral oxygenation is greater and SjvO2 is more preserved with sevoflurane-nitrous than with propofol-remifentanil anesthesia. SctO2 may not be reliable in detecting a low SjvO2 during the surgery in BCP.”

Despite the frequency of low SjvO2 in the propofol-remifentanil group, there were no new neurological deficits observed in this study. None of the patients in the study had cerebral pathology and it was unclear how the results would differ with cerebrovascular disease.

Lee et al. conducted a prospective study on the effects of the BCP and induced hypotension on regional cerebral oxygen saturation (rSO2) in patients having arthroscopic shoulder surgery by using NIRS. MAP and rSO2 were recorded before induction, right after induction, after BCP, immediately after induced hypotension, 1 hour after induced hypotension, and after positioning back into the supine position after surgery. The MAP and rSO2 both decreased after BCP. The MAP was significantly lower after BCP than right after induction. The MAP values immediately after induced hypotension and 1 hour after induced hypotension were significantly lower than right after induction and BCP. Although there was a further decrease in MAP after induced hypotension, there was no additional decrease in rSO2 right after induced hypotension or 1 hour after induced hypotension. There were 2 episodes of reported cerebral desaturation; however, a decline in cognitive function or other neurologic complications was not observed.

Murphy et al. conducted a prospective, cohort study to examine the incidence of cerebral desaturation events in patients undergoing shoulder surgery in the BCP vs. lateral position. Baseline heart rate, MAP, arterial oxygen saturation, and regional cerebral tissue oxygenation (SctO2) were recorded before patient positioning and every three minutes for the duration of the surgical procedure. The incidence of cerebral desaturation was greater in the BCP group. SctO2 values were also lower in the BCP group throughout the intraoperative period. Eight patients (all in the BCP group) had 12 events of SctO2 less than 55%. Nine of the events resolved within 1 minute of an intervention. Interventions included increasing the MAP with vasopressors or a fluid bolus, increasing ETCO2 by decreasing ventilation, or increasing FiO2 concentrations. The episodes ranged from 30 seconds to nine minutes. Despite this occurrence, no neurological deficits were observed in the patients of this cohort study.

Fischer et al. reported a case on a 63 year old female undergoing shoulder surgery in the BCP after 4 cases of ischemic brain injury were reported in patients undergoing orthopedic surgery in the upright position. They report the use of cerebral oximetry monitoring to guide interventions. During positioning there was a decrease in blood pressure accompanied by a decrease in SctO2. Throughout the surgery her SctO2 ranged from 49-86%. The initial supine SctO2 value was 65%. After intubation this rose to 78%. After raising the patient into the BCP the MAP decreased to <70 mmHg. This was accompanied by a decrease in SctO2 to 49%. This was treated with phenylephrine and MAP and SctO2 were normalized. Throughout the surgery 11 additional boluses of phenylephrine were given for either decreased MAP or SctO2. When the patient resumed spontaneous ventilation, there was an increase in end-tidal carbon dioxide (ETCO2) accompanied by an increase in SctO2 to >80%. This
resulted in stable hemodynamics throughout the rest of the case. After completion of the surgery the patient awoke and was extubated. She reported no neurological deficits on the first post operative day.

Moerman et al. evaluated the occurrence of regional cerebral oxygen desaturation in patients undergoing shoulder surgery in the BCP in a prospective, observational, blinded study. The anesthesiologist was blinded to the regional cerebral oxygen saturation level and was not informed about the purpose of the study. A relative decrease of more than 20% occurred in 80% of patients when placed into the BCP. Decreases were directly related to blood pressure and ETCO2. The authors concluded that NIRS may be valuable in this group of patients to detect cerebral hypoperfusion. None of the patients in this study developed gross neurological or cognitive dysfunction postoperatively. They concluded that NIRS could have the potential to improve patient care in these situations.

Jeong et al. determined the incidence and risk factors of SjvO2 desaturation in the sitting position. It was also assessed whether SctO2 measured by NIRS and SjvO2 can be used interchangeably. The patients either underwent anesthesia with propofol and remifentanil or with sevoflurane and nitrous oxide. The type of anesthesia was not randomized and depended on practitioner choice. One group of practitioners always uses inhalational agents and the other group always uses IV anesthesia. All were placed in the BCP and measurements were recorded of the patients MAP, HR, SjvO2, and SctO2 before BCP and after BCP. All measurements decreased significantly after patients were placed in the BCP. Anesthesia practitioners were blinded to the SjvO2 and SctO2 levels. SjvO2 desaturation occurred in 56% of the patients receiving propofol-remifentanil anesthesia and in 21% of the patients receiving sevoflurane-nitrous anesthesia. MAP < 50 mmHg and propofol-remifentanil anesthesia were identified as risk factors. They determined that SctO2 may not replace SjvO2 for determining cerebral oxygenation. None of the patients suffered gross neurological or cognitive dysfunction postoperatively.

Dippman, Winge, and Nielsen reported a case series in which two cases were examined. Case 1 was a 46 year old male with no past medical history. He had good exercise tolerance and did not smoke or drink. He suffered from shoulder pain due to direct trauma that caused a rotator cuff tear. Case 2 was a 58 year old male who suffered from low back pain, smoked 30 cigarettes per day, and consumed 60 ml of alcohol per day. He had no other medical history. Both patients underwent shoulder surgery in the BCP and anesthesia was maintained with propofol and remifentanil. NIRS was used to assess changes in frontal lobe oxygenation. In case 1, frontal lobe oxygenation increased from 76% to 80% after preoxygenation. The heart rate and MAP did not change after preoxygenation, but decreased after anesthesia was administered. After anesthesia, the MAP decreased from 93 mmHg to 47 mmHg and the frontal lobe oxygenation decreased to 46%. With administration of 5 mg of intravenous ephedrine, the MAP rose to 71 mmHg and frontal lobe oxygenation was restored. The frontal lobe oxygenation remained increased throughout the surgery. In case 2, preoxygenation increased the frontal lobe oxygenation from 60% to 66%. The initial MAP was 97 mmHg and after anesthesia was administered it decreased to 51 mmHg.
<table>
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<tr>
<th>Study</th>
<th>Design</th>
<th>Sample Size</th>
<th>Position</th>
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<td>Murphy et al., 2010¹</td>
<td>Prospective Cohort Study</td>
<td>124</td>
<td>BCP- n=61</td>
<td>Standardized GA</td>
<td>During desaturation event MAP was increased</td>
<td>• More cerebral desaturation events in the BCP group (80.3% vs. 0%)</td>
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<td>20</td>
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<td>• No neurological deficits noted</td>
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<tr>
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<td>56</td>
<td>BCP</td>
<td>GA with P/R or</td>
<td>MAP &lt; 50 mmHg treated</td>
<td>• SjvO² and ScvO² decreased significantly</td>
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<td>• No neurological deficits</td>
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<td>Scheeren, Schober, Schwarte,</td>
<td>Review</td>
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<td>• Discussed wide range of scenarios for rSO² use</td>
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<td>• No neurological deficits</td>
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<td>36</td>
<td>BCP</td>
<td>GA with P/R or</td>
<td>Decreased MAP treated</td>
<td>• MAP decreased more in P/R group</td>
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<td>S/N per random</td>
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<td>• SjvO² lower in S/N group (11 +/- 10% vs. 23 +/- 9%)</td>
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<td></td>
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<td>• ScvO² desaturation not significantly different between groups</td>
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<td>• S/N anesthesia provided wider margin of safety against impaired cerebral</td>
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<td>Decreased MAP treated</td>
<td>• MAP &lt;70 mmHg and patient treated 11 separate times</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>• No neurological deficits</td>
</tr>
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</table>

Table 1. Recent literature regarding cerebral oximetry monitoring
and frontal lobe oxygenation decreased to 56%. The administration of 5 mg of ephedrine increased the MAP and the frontal lobe oxygenation returned to 60%. Frontal lobe oxygenation may decrease by 40% in patients undergoing anesthesia in the BCP. This may be rapidly restored with the use of ephedrine. A decreased MAP must be corrected in the BCP because the patient is at risk for cerebral desaturation. In order to monitor and correct these decreases, the authors recommend the use of cerebral oximetry monitoring. In the supine position, frontal lobe oxygenation is not reduced after anesthesia, but even in the healthy patient the BCP may cause severe cerebral desaturation.

Conclusions

Overall, the incidence of cerebral ischemia during shoulder surgery is very low. None of the studies examined in this review cited any new neurological deficits in their outcomes after shoulder surgery in the BCP; however, one of the case studies was reported due to the fact that 4 cases of ischemic brain injury were previously reported in patients undergoing orthopedic surgery in the upright position. These incidences prompted several of the studies. In a survey among orthopedic surgeons the overall incidence of cerebrovascular events occurring during shoulder surgery was 0.00291% (8 out of 274,225) and all events were associated with the BCP. This low incidence could be attributed to the short duration of the surgeries. There were several events of cerebral desaturation in the studies reviewed, but they were quickly corrected by administering vasopressors and increasing the MAP.

Based on the current evidence, more randomized control trials are needed to investigate whether or not cerebral oximetry monitoring decreases the incidence of cerebral ischemia. Data are limited in the use of cerebral oximetry monitoring to prevent cerebral ischemia in shoulder surgery in the BCP. In the studies reviewed, there were no post operative neurological deficits. However, it is unclear whether the use of cerebral oximetry prevented the deficits. In conclusion, future studies should focus specifically on the efficacy of NIRS monitoring in preventing ischemic neurologic events.

References

5. Jeong H, Jeong S, Lim HJ, Lee J, & Yoo KY. Cerebral oxygen saturation measured by near infrared spectroscopy and jugular venous bulb oxygen saturation during arthroscopic shoulder surgery in beach chair position under sevoflurane-nitrous oxide or propofol-


**Mentor:** Laura S. Bonanno, CRNA, PhD

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**Editorial**

It was a pleasure to attend the 2013 AANA Annual Meeting (now renamed the Annual Congress) and connect with so many of the volunteer editors, reviewers, and mentors that make this journal so great. Some of you I met for the first time – it is so good to put a face with the name as we communicate primarily via email. Thank you all for everything you do to sustain the ISJNA. Associate Editor Julie Pearson and I have new plans to increase visibility of the journal so stay tuned!

Best,

Vicki C. Coopmans, CRNA, PhD
Editor

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

To access prior issues of the ISJNA visit the following link:

[www.aana.com/studentjournal](http://www.aana.com/studentjournal)
MISSION STATEMENT
The International Student Journal of Nurse Anesthesia is produced exclusively for publishing the work of nurse anesthesia students. It is intended to be basic and introductory in its content. Its goal is to introduce the student to the world of writing for publication; to improve the practice of nurse anesthesia and the safety of the patients entrusted to our care.

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

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

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

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

General guidelines
1. Items for publication must adhere to the American Medical Association Manual of Style (AMA, the same guide utilized by the AANA Journal and such prominent textbooks as Nurse Anesthesia by Nagelhout and Plaus). The review process will not be initiated on reports submitted with incorrect formatting and will be returned to the mentor for revision. Please note the following:
   a. Use of abbreviations is detailed in Section 14. Spell out acronyms/initialisms when first used. If you are using the phrase once, do not list the acronym/initilialism at all.
   b. Instructions regarding units of measure can be found in Section 18. In most cases The International System of Units (SI) is used. Abbreviations for units of measure do not need to be spelled out with first use. Some examples: height/length should be reported in cm, weight in kg, temperature in °C, pressure in mm Hg or cm H2O.
   c. In general, first use of pulmonary/respiratory abbreviations should be expanded, with the following exceptions: O2, CO2, PCO2, PaCO2, PO2, PaO2. Please use SpO2 for oxygen saturation as measured by pulse oximetry.
   d. Use the nonproprietary (generic) name of drugs - avoid proprietary (brand) names. Type generic names in lowercase. When discussing dosages state the name of the drug, then the dosage (midazolam 2 mg).
   e. Use of descriptive terms for equipment and devices is preferred. If the use of a proprietary name is necessary (for clarity, or if more than one type is being discussed), give the name followed by the manufacturer and location in parenthesis:
      “A GlideScope (Verathon Inc., Bothell, WA) was used to . . . .”
      Please note, TM and ® symbols are not used per the AMA manual.
   f. Examples of referencing are included later in this guide.
2. Report appropriate infusion rates and gas flow rates:
   a. When reporting infusion rates report them as mcg/kg/min or mg/kg/min. In some cases it may be
      appropriate to report dose or quantity/hr (i.e. insulin, hyperalimentation). If a mixture of drugs is being
      infused give the concentration of each drug and report the infusion rate in ml/min.
   b. Keep the gas laws in mind when reporting flow rates. Report the liter flows of oxygen and nitrous oxide
      and the percent of the volatile agent added to the gas mixture. Statements such as “40% oxygen, 60% nitrous
      oxide and 3% sevoflurane” do not = 100% and are thus incorrect. For example, “General anesthesia was
      maintained with sevoflurane 3% inspired concentration in a mixture of oxygen 1 L/min and air 1 L/min”.
3. Only Microsoft Word file formats will be accepted with the following criteria:
   a. Font - 12 point, Times New Roman
   b. Single-spacing (except where indicated), paragraphs separated with a double space (do not indent)
   c. One-inch margins
   d. Place one space after the last punctuation of sentences. End the sentence with the period before placing the
      superscript number for the reference.
   e. Do not use columns, bolds (except where indicated), or unconventional lettering styles or fonts.
   f. Do not use endnote/footnote formats.
4. Do not use Endnotes or similar referencing software. Please remove all hyperlinks within the text.
5. Avoid jargon.
   a. 'The 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 was administered by face mask."
   c. The patient was intubated and put on a ventilator. “The trachea was intubated and respiration was
      controlled by a mechanical ventilator.
   d. The patient had been on Motrin for three days. “The patient had taken ibuprofen for three days.”
   e. Avoid the term “MAC” when referring to a sedation technique - the term sedation (light, moderate, heavy,
      unconscious) sedation may be used. Since all anesthesia administration is monitored, the editors prefer to
      use specific pharmacology terminology rather than reimbursement terminology.
6. Use the words “anesthesia professionals” or “anesthesia practitioners” when discussing all persons who
   administer anesthesia (avoid the reimbursement term “anesthesia providers”)
7. References
   a. Again, the AMA Manual of Style must be adhered to for reference formatting.
   b. All should be within the past 8 years, except for seminal works essential to the topic being presented.
   c. Primary sources are preferred.
   d. All items cited must be from peer-reviewed sources – use of internet sources must be carefully considered
      in this regard.
   e. Numbering should be positioned at the one-inch margin – text should begin at 1.25”.
8. See each item for additional information.
9. Heading for each item (Case Report, Abstract, EBPA Report) must adhere to the following format:

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

**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 do not count against the word count. Case reports with greater than 1400
words will be returned to the mentor for revision prior to initiation of the review process. The following template demonstrates the required format for case report submission.

**Heading** (see #9 above in General Guidelines)

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

**Case Report** (bold, 400-500 words)

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

- Patient description: height, weight, age, gender.
- History of present illness
- Statement of co-existing conditions/diseases
- Mention the current medications, generic names only. (Give dosage and schedule only if that information is pertinent to the consequences of the case.)

**Significant** laboratory values, x-rays or other diagnostic testing pertinent to the case. Give the units after the values (eg. Mmol/L or mg/dL).

- Physical examination/Pre-anesthesia evaluation - significant findings only. Include the ASA Physical Status and Mallampati Classification only if pertinent to the case.
- Anesthetic management (patient preparation, induction, maintenance, emergence, post-operative recovery).

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

**Discussion** (bold, 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** (bold)

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

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

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

**Research Abstracts**

Research abstracts are limited to 500 words. References are not desired but may be included if considered essential. Note that this abstract is different from a research proposal. This abstract reports the outcome of your study. Use the same format described for the case report with the exception of the section headings:
**Heading** (see #9 above in General Guidelines)

**Introduction** (bold)

A brief introductory paragraph including purpose and hypotheses.

**Methods** (bold)

Include research design and statistical analyses used

**Results** (bold)

Present results – do not justify or discuss here.

**Discussion** (bold)

Discuss results

**References** (bold)

Not required, but a maximum of 5 references is allowed.

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

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

**EBP Analysis Reports**

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

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

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

**Heading** (see #9 above in General Guidelines)

**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.

**Methodology** (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)
Review and critique the pertinent and current literature, determining scientific credibility and limitations of studies reviewed. Your synthesis table would be included in this section. Your review and discussion of the literature should logically lead to support a practice recommendation. Subheadings may be used if desired.

**Conclusions**

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.

**References**

A minimum of 8 references is recommended, with a maximum of 12 allowed.

**Letters to the Editor**

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

**AMA MANUAL OF STYLE**

The following is brief introduction to the *AMA Manual of Style* reference format along with some links to basic, helpful guides on the internet. The website for the text is [http://www.amamanualofstyle.com/oso/public/index.html](http://www.amamanualofstyle.com/oso/public/index.html). It is likely your institution’s library has a copy on reserve.

- [http://www.docstyles.com/amastat.htm#Top](http://www.docstyles.com/amastat.htm#Top)
- [http://healthlinks.washington.edu/hsl/styleguides/ama.html](http://healthlinks.washington.edu/hsl/styleguides/ama.html)

Journal names should be in *italics* and abbreviated according to the listing in the PubMed Journals Database. The first URL below provides a tutorial on looking up correct abbreviations for journal titles; the second is a link to the PubMed where you can perform a search.


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

### Journals

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

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

**Journal, 6 or fewer authors:**


**Journal, more than 6 authors:**

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

Text:

Chapter from a text:

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

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

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

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

Examples:


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

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

http://grad.georgetown.edu/pages/reg_7.cfm

HOW TO SUBMIT AN ITEM
Manuscripts must be submitted by the mentor of the student author via e-mail to INTSJNA@aol.com as an attachment. The subject line of the e-mail should be “Submission to Student Journal”. The item should be saved in the following format – two-three word descriptor of the article_author’s last name_school abbreviation_mentor’s last name_date (e.g. PedsPain_Smyth_GU_Pearson_5.19.09)
REVIEW AND PUBLICATION
If the editor does not acknowledge receipt of the item within one week, assume that it was not received and please inquire. Upon receipt, the Editor will review the submission for compliance with the Guide to Authors. If proper format has not been following the item will be returned to the mentor for correction. This is very important as all reviewers serve on a volunteer basis. Their time should be spent ensuring appropriate content, not making format corrections. It is the mentor’s responsibility to ensure formatting guidelines have been followed prior to submission.

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

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

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

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

Vicki C. Coopmans, CRNA, PhD
Goldfarb School of Nursing at Barnes-Jewish College
4483 Duncan Ave., Mailstop 90-36-697
St. Louis, MO  63110
### SUBMISSION CHECK LIST

<table>
<thead>
<tr>
<th>AMA Manual of Style and other format instructions are adhered to.</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Total word count not exceeded (1400 for case report, 500 for abstract, 3000 for EBPA).</em></td>
</tr>
<tr>
<td><em>The item is one continuous Word document without artificially created page breaks.</em></td>
</tr>
<tr>
<td><em>Verbatim phrases and sentences are quoted and referenced.</em></td>
</tr>
<tr>
<td><em>All matters that are not common knowledge to the author are referenced.</em></td>
</tr>
<tr>
<td><em>Generic names for drugs and products are used throughout and spelled correctly in lower-case.</em></td>
</tr>
<tr>
<td><em>Units are designated for all dosages, physical findings, and laboratory results.</em></td>
</tr>
<tr>
<td><em>Endnotes, footnotes not used.</em></td>
</tr>
<tr>
<td><em>Jargon is absent.</em></td>
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</tbody>
</table>

### Heading
- Concise title less than 70 characters long
- Author name, credentials, nurse anesthesia program, graduation date and email are included.
- Five **Keywords** are provided

### Case Report
- Introduction is less than 100 words.
- Case Report section states only those facts vital to the account (no opinions or rationale)
- Case report section is 400-500 words and not longer than the discussion.
- Discussion section is 600-800 words.
- Discussion of the case management is based on a review of current literature
- Discussion concludes with lessons learned and how the case might be better managed in the future.

### Abstract
- The 500 word count maximum is not exceeded.
- Abstract reports the outcome of your study.
- Includes Introduction, Methods, Results, and Conclusion sections.

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

### References
- AMA Style for referencing is used correctly.
- Reference numbers are sequenced beginning with one and superscripted.
- References are from anesthesia and other current primary source literature.
- All inclusive pages are cited, texts as well as journals.
- Journal titles are abbreviated as they appear in the PubMed Journals Database.
- Number of references adheres to specific item guidelines.
- Internet sources are currently accessible, reputable, and peer reviewed.

### Transmission
- The article is sent as a attachment to **INTSJNA@AOL.COM**
- The file name is correctly formatted (e.g. PedsPain_Smyth_GU_Pearson_5.19.09)
- It is submitted by the mentor with cc to the student author
- The words “Submission to Student Journal” are in the subject heading.