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TOPICS IN THIS ISSUE

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Front Cover: Winners of the 2010 Annual AANA Anesthesia College Bowl, listed from left to right with the Nurse Anesthesia Program they were enrolled in at the time: Kenneth Doyon, Trover Health System/Murray State University; Brady Banares, Medical College of Georgia; Howard Drews, Oakland University Beaumont; Jessica Burton-Vigil, Albany Medical College; Andrea Atkins, Goldfarb School of Nursing at Barnes-Jewish College; Laura Niday, Wake Forest University Baptist Medical Center, University of North Carolina at Greensboro.

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Positive End Expiratory Pressure in Laparoscopic Surgery

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Keywords: Positive End Expiratory Pressure, oxygenation, ventilation, hernia, laparoscopic

Positive end expiratory pressure is frequently used to aid in mechanical ventilation during the intra-operative period. Maintaining arterial oxygenation is paramount to positive patient outcomes and positive end expiratory pressure (PEEP) can enhance oxygenation. While PEEP is rarely mandatory, the increased alveolar recruitment and compliance seen with early use of PEEP overcomes complications seen with laparoscopic surgery and hypoxia post-operatively. The following case report describes how PEEP is used to maintain adequate arterial oxygenation during a basic laparoscopic procedure.

Case Report

A 64-year-old, 104 kg, 167 cm Hispanic male presented for a laparoscopic ventral hernia repair. The patient was initially diagnosed with a ventral hernia a year prior. His past medical history included hypertension, gastroesophageal reflux disease, hiatal hernia, non-insulin dependent diabetes mellitus, and an enlarged prostate. The patient also experienced periods of obstructive sleep apnea requiring the use of a Continuous Positive Airway Pressure machine when he sleeps. His surgical history included laparoscopic choleycystectomy, laparoscopic appendectomy, colon resection, nissen fundiplication, and cardiac catheterization. His current medications consisted of hydrochlorothiazide, simvastatin, pantoprazole, terazosin, loperamide, fluoxetine, cholestyramine, amlodipine and tamsulosin. His preoperative vital signs were within normal limits

The patient was pre-medicated with midazolam 2 mg intravenously (IV) via an 18 gauge peripheral angio-catheter. In the operating room, standard monitors were placed on the patient and fentanyl 100 mcg IV was administered. General anesthesia was induced utilizing lidocaine 60 mg, propofol 200 mg, and rocuronium 50 mg IV. Successful mask ventilation was followed by direct laryngoscopy with a Miller 2 blade. A 7.5 mm oral endotracheal tube (ETT) was easily inserted between the vocal cords and secured at the lip at 22 cm. Positive bilateral breath sounds were auscultated and positive end tidal carbon dioxide was observed. Mechanical ventilation was initiated. The stomach was decompressed with an oral gastric tube and an esophageal stethoscope was placed into the patient's esophagus. Volume control ventilation was utilized to maintain adequate ventilation and $ETCO_2$ was maintained at 32-35 mmHg. Positive end expiratory pressure (PEEP) measuring 10cm H_2O was required to maintain adequate arterial oxygen saturation above 98 %. It was assumed that the patient's obesity lead to less than 100 percent oxygen saturation. General anesthesia was maintained with Sevoflurane at an end tidal concentration of 2.2 % and O_2 at 2 L/min. Optimal surgical conditions were maintained by administering an additional dose of rocuronium 30 mg IV 22 min after incision.

The ventral hernia repair was completed in 76 minutes. Train of four (TOF) peripheral

nerve stimulation was monitored during the case. At the conclusion of surgery, a TOF stimulation showed 4/4 twitches was documented and the neuromuscular blockade was antagonized with neostigmine 4 mg and glycopyrrolate 0.6 mg IV. Ondansetron 4 mg IV was administered. The ETT was removed following 5 sec of sustained tetany and a 5 sec head lift. The patient's tidal volume at extubation was observed at 5 ml/kg. Oxygen was delivered via nasal cannula at 3L/min as the patient was transported to the post anesthesia care unit (PACU).
Upon arrival to the post-anesthesia care unit (PACU) the patient was admitted to the medical/surgical unit for further monitoring. No nausea, vomiting, or excessive pain was reported by the PACU RN two hours after the end of surgery. Minimal throat pain was reported by the patient.

Discussion

Laparoscopic ventral hernia repairs are a common surgical procedure that anesthesia practitioners should be familiar with due to their frequent occurrence. Ventral hernias result from weakened abdominal musculature that allows intestinal structures to pass through the muscle. While often benign, this herniation can restrict blood flow to the displaced portion of the bowel (strangulation) leading to a cell death and necrosis.[1]

Laparoscopic surgery continues to gain favor within the surgical arena as it has lead to greater patient satisfaction, decreased hospital length-of-stay, less post-operative pain and shorter surgical time.[2] In order to create a work space within the abdomen during the repair of a ventral hernia, a pneumoperitoneum is created by insufflations of the abdomen, most often with carbon dioxide. This insufflation leads to increased intra-abdominal pressures thereby displacing the diaphragm superiorly. This rostral diaphragmatic displacement, along with the generally preferred Trendelenburg position, greatly decreases functional residual capacity (FRC) and "may reduce the FRC below the closing volume, which predisposes to airway closure and atelectasis."[3]

The application of positive end expiratory pressure (PEEP) during mechanical ventilation gives the anesthesia practitioner an opportunity to prevent airway closure, atelectasis and subsequent decreases in arterial oxygen saturation.[4] The obese population is especially at risk for these effects secondary to body habitus, general anesthesia and pneumoperitoneum. Obesity leads to an increase in airway soft tissue formation, and relaxation of that tissue can occlude the upper airways leading to airway closure. General anesthesia leads to soft tissue relaxation. Central obesity requires larger volumes of abdominal insufflation leading to a greater level of pneumoperitoneum. The higher pressure required to visualize abdominal contents creates a necessity to maintain higher airway pressures to maintain adequate oxygenation. PEEP creates a continuous positive pressure throughout the ventilation cycle that counteracts both the anatomical and physiological changes that lead to decreased arterial oxygen saturation during laparoscopic surgery. Providing PEEP enables alveoli to maintain adequate tension and avoid collapse when intra-abdominal pressures increase thereby enhancing oxygenation. Research has shown that the physiological respiratory effects of pneumoperitoneum can be reversed with the addition of 10 cmH_2O of PEEP intra-operatively.[4]

An additional component to most anesthetics for laparoscopic surgeries including laparoscopic ventral hernia repair involves the administration of a muscle relaxant. The use of muscle relaxant benefits the anesthesia practitioner by enhancing orotracheal intubation and provides the surgical team with less abdominal wall tension. This lack of tension allows for a greater degree of insufflation thereby increasing the work space and visibility within the abdomen itself. With muscle relaxation the abdomen can be insufflated with less pressure to obtain adequate work space than if muscle relaxation medication was not utilized. Avoiding relaxation would require a greater insufflation pressure to obtain an adequate space to remove diseased organs. The increased intra-abdominal pressure increases airway pressures and can exacerbate atelectasis and collapse the airway.[5] PEEP can serve to counteract the pressurized abdomen by applying continual force in an opposing manner to help maintain respiratory mechanics through alveolar recruitment.

PEEP was initiated during this surgical procedure as a method to improve arterial oxygen saturation; this was reflected in a sustained increase in oxygen saturation. Decreases in arterial oxygen saturation secondary to decreased respiratory mechanics during laparoscopic surgery perpetuate hypoxemia. Hypoxic changes within the body initiate multiple compensatory cascades that require even more oxygen, a vital element that is lacking due to decreased gas exchanging ability secondary to alveolar collapse. The loss of alveolar tension inhibits the ability to ventilate which leads to retained CO_2. Retaining CO_2 can constrict pulmonary vasculature, increase intracranial pressure and decrease arterial oxygen levels. All of these effects increase the likelihood that extubation will be delayed stemming from alveolar collapse. It is the responsibility of the prudent anesthesia professional to gain an understanding of, and implement PEEP when necessary to maintain adequate oxygenation.

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Free Gracilis Muscle Flap Grafting

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Keywords: Gracilis flap, microvascular reconstructive surgery, free flap, dopamine, phenylephrine, dobutamine

The gracilis or musculocutaneous flap is a favored donor muscle for reconstructive microsurgery. It can be used as a local flap or free flap. The use of free flaps in repairing lower extremity defects has enabled reconstructive surgeons to preserve limbs that not too long ago would have been amputated.[1] Intraoperative events such as hypoperfusion and vasoconstriction may affect the outcome of the flap viability. Therefore, a thorough understanding of the procedure and good communication with the surgical team is vital.

Case Report

A 25 year-old, 69 in, 111 kg male was scheduled for a debridement of his left fibula with a free muscle flap and a split thickness skin graft. He suffered severe thermal and blast injuries at work when a 50 gallon drum exploded. The injuries were significant and involved the trunk, lower extremities from the knees down and the lower third of his arms. Following this incident, he required bilateral lower extremity fasciotomies, burn excisions and grafting of tissue on his upper and lower extremities. His course was complicated by left lower extremity infection with enterococcus and pseudomonas. Antibiotics failed to resolve the infection, and surgical intervention for osteomyelitis became necessary. He had a 10 pack per year smoking history but he had ceased smoking two weeks prior to surgery. His surgical history was significant for several skin graft surgeries and two toe amputations on his left foot. The only medication he was taking at that time was bupropion. Physical assessment on the day of surgery was positive for limited neck extension due to scaring, a Mallampati class II airway, and a healed tracheotomy site resulting from his initial injuries.

Preoperatively the patient was medicated with midazolam 2 mg intravenously (IV) and given heparin 5000 units subcutaneously Once in the operating room the patient was placed on the surgical table with a full length body warmer underneath. A bladder temperature probe from the foley catheter was utilized for assessment of core body temperature.

The patient was preoxygenated with 100% oxygen and standard noninvasive monitors were applied. General anesthesia was induced with lidocaine 100 mg, an additional dose of midazolam 2 mg, fentanyl 200 mcg, proprofol 180 mg and rocuronium 40 mg IV. A bougie was utilized to facilitate placement of the endotracheal tube due to an inadequate view of the glottis. General anesthesia was maintained with desflurane, 1 L/min O_2 and 1 L/min air. A fentanyl infusion was initiated and titrated between 1-3mcg/kg/hr. Fluid was administered through fluid warmers and an upper body warmer was applied in addition to the full body warmer.

The procedure lasted 15 hours and the patient was hemodynamically stable throughout, with the exception of a few episodes of hypotension. His blood pressure was monitored closely and hypotension was

treated with fluid boluses, hetastarch and low dose dopamine. His urine output was also monitored to ensure at least 30ml/hr. At the conclusion of the case the endotracheal tube was removed and he was brought to the post anesthesia care unit in stable condition. Shortly after transfer, doppler signals of blood flow were negative at the flap site. The patient was returned to the operating room emergently secondary to presumed graft anastomosis thrombosis.

Discussion

Flap viability is the most important consideration for microvascular reconstructive surgery. If a reexploration is needed this may result in complete loss of tissue. A major goal during anesthesia and in the early postoperative period is maintenance of optimal hemodynamics to maximize flap perfusion and flow across the anastomosis.[2] Anastomotic patency is diminished by vasospasms or vascular constriction. The most common causes of vasoconstriction include dehydration, hypothermia, pain, and administration of vasoconstrictors.[3] Several interventions were carried out intraoperatively in this case to combat these clinical problems.

Low blood flow may cause vasoconstriction and create a challenge for the surgeon when the anastomosis is needed. Appropriate cardiac output (CO) is needed for adequate blood flow to the flap. Cardiac output is dependent on preload and can be affected by hypovolemia.[2] Preoperatively, the patient's baseline blood pressure was 130/60 mmHg with a mean arterial pressure (MAP) of 80 mmHg. During the procedure the goal was to keep the MAP greater than 65 mmHg. Intraoperatively, the patient was hydrated with lactated ringers at a rate of 400 ml/hr. During hypotensive episodes with a MAP of 58 mmHg, the first intervention was a 500 ml fluid bolus. The type and amount of fluids are important. Free flaps are at an increased risk for developing edema due to poor lymphatic drainage and an inability to absorb excessive interstitial fluid.[4] Crystalloids should be used for replacing maintenance fluid requirements only and colloids for plasma losses.[4] This will maintain the volume of the intravascular space by increasing oncotic pressure. After the initial fluid bolus, the patient's blood pressure (BP) remained low at 85/42 mmHg with a MAP of 50 mmHg, and the decision was made to administer one liter of hetastarch. After this intervention, his BP increased to 100/60 mmHg with a MAP of 65 mmHg.

The use of vasoconstrictors for hypotension is controversial. Because phenylephrine is believed to be deleterious to CO and flap flow, low dose dopamine was selected for further treatment of hypotension.[5] Dopamine was initiated at 3mcg/kg/min when the BP was 98/45 mmHg with a MAP of 62 mmHg, despite one liter of hetastarch. In doses of 3-10 mcg/kg/min, dopamine exerts a positive inotropic effect increasing CO in a dose-dependent manner, primarily as a result of beta one receptor stimulation.[2] Dopamine was used intermittently throughout the case for short periods of time with a dosage range of 2-3 mcg/kg/min. This assisted with manipulation of CO and blood flow to the surgical area. After the initiation of dopamine, the BP increased to 120/45 mmHg with a MAP of 70 mmHg.

In a study published by Suominen et al., the effect of intravenous dopamine and dobutamine on circulation during a microvascular procedure was investigated.[2] The result of this effort suggested that dobutamine may offer advantages over dopamine when administered at a fixed level of 8 mcg/kg/min. The use of dobutamine

resulted in a hemodynamic effect that was similar in all patients and increased CO while decreasing SVR. Additionally, a simultaneous increase in donor and recipient artery flow was recorded.[2] Dopamine responses were inconsistent and unpredictable. In some patients, the infusion of dopamine exerted a pronounced alpha adrenergic effect, increasing SVR.[2] In addition, one patient had to be excluded from the study and dopamine infusion stopped because of a marked increase in both MAP and SVR. Dopamine did in fact increase CO, though less than dobutamine, and caused a higher increase in BP. Neither of these effects enhanced blood flow in the arteries measured during this study.

Phenylephrine use has been deemed a risk factor for reducing blood flow and contributing to flap failure. However, in a porcine model study debating the impact of phenylephrine use investigators demonstrated that phenylephrine caused a 30% increase in MAP, whereas heart rate, CO, and flap blood flow did not change.[6] Therefore, use of this agent caused an increase in blood pressure without compromising skin blood flow. Based on this porcine model, it could be inferred that phenylephrine is not as deleterious to microcirculation as past research has suggested. Phenylephrine is an alpha 1 agonist and a potent vasoconstrictor, and is known to constrict mainly larger arterioles. It has virtually no effects on terminal arterioles that respond to alpha 2 agonists such as norepinephrine.[6] This may be why phenyleprhine did not affect blood flow to the flap in the porcine model. It is important to note here that this research was carried out under normovolemic conditions. When patients are hypovolemic, results may vary.[6]

Unfortunately, we can not be certain what contributed to the cause of flap failure in this case. One could argue the operative time played a significant role in the outcome. Intraoperatively, the anesthesia professional maintained normovolemia, normothermia, and pain control, and used very little dopamine. Alternative treatment could have included the use of dobutamine instead of dopamine. In the reviewed literature, dobutamine appears to have the most positive effect on flap flow. Anesthetic management for patients undergoing reconstructive surgery for free flaps requires vigilant monitoring. Hypoperfusion and necrosis remain important clinical problems. In a patient who is hemodynamically compromised, CO and systemic perfusion are key components to the viability of the flap. Consideration of the appropriate vasopressors to support microcirculation is crucial. More research is needed to clear up the controversies regarding the use of phenylephrine in the anesthetic management of free flaps. Until then, dobutamine and dopamine are the preferred agents, with some evidence favoring dobuatmine.

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Intraoperative Median Nerve Injury

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Keywords: peripheral nerve injury, paresthesia, perioperative weakness, anesthesia, complications, positioning.

General anesthesia relaxes the muscles of the body and eliminates protective reflexes that enable patients to respond to pain or ischemic injury. [1] The anesthetist must recognize body contours of not only the old and obese, but also the young, lean and healthy in order to adequately pad and protect patients against intraoperative nerve injury. Body builders may be more susceptible to certain nerve injuries, such as median nerve injury, due to anesthesia practitioners not recognizing risks associated with this population.[2] Vigilance is the best defense in preventing intraoperative nerve injuries.

Case Report

A 24-year-old male underwent anterior urethroplasty with buccal graft under general endotracheal anesthesia. Past medical history was unremarkable for systemic illness; the patient had a lean muscular build: height=180 cm, weight=92 kg BMI= 28 kg/m^2. The patient was positioned supine and legs placed in low lithotomy. Arms were secured on arm boards with less than 90 degree abduction. The hands were supinated and padded. Standard monitors were placed, including a noninvasive blood pressure cuff cycling every 3 minutes on the upper left arm. The arm was inspected for color and warmth following cuff placement and determined not to be compromising peripheral blood flow to the distal arm when deflated. Intravenous access was obtained in the right hand preoperatively. The surgery progressed for 3.5 hours without apparent intraoperative complications. The buccal graft was harvested by an oromaxillofacial surgery team while the urethra was repaired by urologic surgeons. Full-body draping made intraoperative assessment of the patient's position challenging.

The patient was visited postoperatively the following morning and complained of bilateral sensory deficits to the index, middle, and ring fingers. This was described as tingling sensations accompanied by a dull sensation to touch. He denied having a preexisting neuropathy of any kind. The patient also noted motor weakness upon attempting to approximate his left index finger and thumb. His right and left hands

were compared for strength and mechanism of movement. Although he was able to approximate his thumb and index finger, the approximation felt mechanically different to the patient. He was using different muscles of his left hand to achieve a weak grasp and was unable to hold a cup. No motor weakness to the right hand was observed. The patient was a deep sea diver by trade but denied any history of cervical neck injury. There was no edema, erythema or visual evidence of traumatic injury in either arm. The patient was informed of the iatrogenic median nerve injury and reassured that most nerve injuries resolve spontaneously. He was discharged on postoperative day 1 and contacted by telephone on postoperative days 3 and 4. The numbness and weakness were partially resolved by postoperative day 3 and completely resolved on postoperative day 4. He was instructed to contact the Anesthesia Department if symptoms were to reoccur. Neurology consultation was planned if symptoms had persisted beyond 1 week.

Discussion

Peripheral nerve injury is a common, often avoidable complication following surgery, and is also a common cause for litigation. Although most legal cases involving nerve injuries in the operating room do not have a definitive mechanism of injury,[3] it is important to explore suspected causes in order to prevent future nerve injury. Differential diagnoses to consider in this patient included median nerve palsy, traumatic neuroma, and cervical radiculopathy. [4-7] Nerve palsy may be classified as neuropraxia, axonotmesis or neurotmesis due to stretch, compression, ischemia, or trauma. The acute nature and short duration of symptoms suggested neuropraxia, the mildest grade of nerve injury that has the best prognosis for complete recovery. Neuropraxia involves a reduction in ion conduction that may include myelin damage, but without damage to the axon.[4]

Since trauma and ischemia were not apparent, stretch and compression remained as possible mechanisms of injury. Traumatic neuroma would be plausible after a cutting injury such as traumatic venipuncture,[8] but there was no evidence of venipuncture to the left antecubital fossa. As previously stated, both arms were outstretched flat, supinated, and secured to arm boards. After surgery, numbness was evident in the same distribution of the median nerve bilaterally. Carpal tunnel syndrome can cause of this type of paresthesia;[2] however, it is unlikely in this scenario because the wrists were positioned neutrally, atraumatically, and no previous symptoms were noted. The sensory disruption in this patient is consistent with an injury of the median nerve near the antecubital space,[6] and the mechanism is likely due to nerve stretching.

Weakness was only present in the left arm, which was connected to the non-invasive blood pressure cuff. The blood pressure cuff cycling every three minutes for four hours could have caused a compression injury to the nerve's larger motor fibers. Inflatable cuffs can compress the nerve and cause intraneural edema.[9] These cuffs can also physically displace the Schwann cells along a nerve, but the extent would be related to the amount and duration of pressure exerted.[10] This mechanism is consistent with the patient's motor deficit in approximating the thumb and forefinger of the left hand, a function of the median nerve. The patient's continued ability to achieve a weak grasp with the left hand is consistent with a compensatory use of the adductor pollicus and first dorsal interosseous muscle which is supplied by the ulnar nerve.[6] Nerve injury

proximal to the cuff is not likely due to the upper arms and shoulder being in their neutral position, less than 90 degrees abducted. Nerve injury distal to the cuff is possible, due to inadvertent compression from surgical staff at the level of the anterior interosseous nerve.[7] This form of compression was not observed during the procedure.

Stretch, compression and ischemia may work synergistically to cause nerve injury. All three mechanisms could impair ionic transport and nerve function.[4] Each of these mechanisms could individually cause neuropraxia, but may also be a consequence of the primary injury. For example, a patient with an acute injury due to nerve stretch may develop edema within the surrounding structures that cause compression of blood vessels supplying the nerve, inducing ischemia.[11] For the patient discussed here, fully extended arms could have made the nerve more vulnerable to compression injury, since the nerve was maximally elongated before the blood pressure cuff would cycle and stretch the nerve further.

Hyperextension of the elbow during surgery is a recognized cause of nerve injury.[3] The phenomena of median nerve injury has been observed to happen more often in young men.[2] Moreover, a person with a large posterior protrusion of the triceps muscles may be at a greater risk for hyperextension of the elbow when there is no reciprocating posterior support of the forearm (opposing hyperextension). The operating room team must ensure that each arm is appropriately padded to prevent hyperextension and reduce the risk of median nerve injury. If nerve injuries do occur despite the practitioner's best effort, they should be referred to a neurologist for EMG testing and/or MRI.

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Laryngospasm after Cleft Lip and Palate Repair

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Keywords: airway obstruction, laryngospasm, pediatric airway management, cleft lip and palate repair

Post-extubation laryngospasm is a common upper airway obstruction occurring after tracheal intubation in children. Upper airway surgical procedures increase the incidence of laryngospasm due to airway blood and secretions. The overall incidence of laryngospasm during cleft lip and palate repair is approximately 6%.[1] Abnormal excitation of laryngeal reflexes causes laryngeal muscle contraction resulting in laryngospasm.[2] Unrecognized laryngospasm can lead to severe respiratory compromise and death. It is imperative that anesthesia professionals are proficient in not only recognizing and treating this respiratory complication, but also taking steps to prevent it. This case report will detail a post-extubation laryngospasm after cleft lip and palate repair.

Case Report

A 6 month old, 8 kg, 66 cm, male infant presented for elective cleft lip and palate surgical repair. His past medical history included diabetes insipidus, micrognathia, hydronephrosis, gastroesophageal reflux disease and atrial septal defect. The infant had no surgical history. No food or drug allergies were noted and no laboratory tests were ordered. Physical examination revealed clear breath sounds and regular cardiac rhythm and rate. The infant was classified as American Society of Anesthesiologists III physical status. The infant's last intake was at 0300 the morning of surgery consisting of pedialyte.

Upon arrival to the operating room, standard noninvasive monitors were placed. An inhalation induction with sevoflurane and nitrous oxide was achieved. Intravenous (IV) access was obtained via the right saphenous vein with a 25 gauge IV catheter. Fentanyl 10 mcg and rocuronium 5 mg IV were administered. Laryngoscopy was performed utilizing a Miller 1 blade and the infant's trachea was intubated with a 3.5 mm ID cuffed endotracheal tube (ETT) without complication. General anesthesia was maintained with sevoflurane.

The procedure was uneventful. Neuromuscular blockade was antagonized with neostigmine 0.4 mg and glycopyrrolate 0.08 mg IV after the train of four revealed 4:4 muscle twitches. Bilateral nasal packing was placed and the stomach and oropharynx were gently suctioned by the surgeon. After spontaneous ventilation resumed, the endotracheal tube was removed without complication. The infant was positioned in

left lateral recovery position and a pediatric face mask with oxygen at 4 L/min was used for transport to the post anesthesia recovery unit (PACU). Upon arrival to the PACU, the infant was reassessed and was determined to be exchanging air well with stable vital signs and 100% SpO_2. Care was transferred to the PACU nurse. After several minutes in the PACU, the infant suffered an acute airway event causing oxygen desaturation and bradycardia. It appeared that blood or secretions traveled to the vocal cords resulting in laryngospasm. After resuscitation with positive pressure ventilation and insertion of a 3.5 mm ID ETT by anesthesia personnel, the infant was subsequently transferred to the pediatric intensive care unit (PICU) for monitoring and suffered no adverse sequelae.

Discussion

Laryngospasm is a partial or complete closure of the glottis occurring commonly in children. The most common causes of a laryngospasm include extubation of the trachea during stage 2 of anesthesia and stimulation of the vocal cords by blood, oral secretions, suction catheters, artificial airways and laryngoscope blades. The overall incidence of laryngospasm is 0.87%, while the incidence for the first 9 years of life is 1.74%. [2] Infants with craniofacial abnormalities undergoing cleft lip and palate repair should alert the anesthesia professional of possible difficult airway management. Current literature supports the use of continuous positive airway pressure (CPAP), jaw thrust, pharmacological interventions and tracheal intubation for laryngospasm management.[2] Jaw thrust, CPAP, and tracheal intubation for airway protection were implemented for laryngospasm management in this infant.

In this case, the infant's medical history of cleft lip and palate, micrognathia and age placed him at greater risk for laryngospasm. The reported incidence of laryngospasm following general anesthesia correlates inversely with age. [3] Infants with cleft lip and palate often have high arched palates and a prominent maxilla while characteristics of micrognathia include a smaller hypomental space with the tongue positioned more posteriorly making laryngoscopy challenging. Surgical induced airway trauma, narrowed pharyngeal space and bleeding increase laryngospasm probability. Features of a laryngospasm include paradoxical chest movement, intercostal recession, tracheal tug, and lastly a crowing sound is heard during a partial laryngospasm while no sound is heard during complete laryngospasm. [4] If left untreated, a laryngospasm can lead to oxygen desaturation, bradycardia, severe respiratory distress, and death. Laryngospasm should be considered in the differential diagnosis of a patient exhibiting inspiratory stridor and rapid oxygen desaturation. Bronchospasm and supraglottic obstruction exhibit similar signs and symptoms. Jaw thrust or head tilt maneuver with or without insertion of oral or nasal airway while applying gentle positive pressure would relieve both supraglottic obstruction and partial laryngospasm. [2]

Anesthesia professionals caring for pediatric patients should be knowledgeable of the multitude of anatomic and physiologic differences in comparison to the adult patient. A less experienced anesthesia professional increases the risk of laryngospasm. [5] Pediatric patients with congenital facial anomalies often have challenging airways. Another risk factor for laryngospasm in cleft lip and palate repair is the presence of an upper respiratory tract infection (URI). [5] General anesthesia in the

presence of a URI increases the risk of laryngospasm 2-3fold .[6] During cleft lip and palate repair, children with common cold symptoms have a 23% incidence of perioperative respiratory complications. [1]

Laryngospasm prevention should include a multimodal approach, incorporating pharmacological and non-pharmacological interventions. The infant should be optimized prior to undergoing general anesthesia. During this case, non-pharmacological interventions implemented included: ensuring the infant had optimized homeostasis, airway suctioning prior to tracheal extubation, extubation in a very light plane of anesthesia, and lastly, placing the infant in left lateral recovery position.

In reviewing current literature, several pharmacological modalities have been evaluated for effectiveness in preventing a laryngospasm. A subhypnotic dose of propofol 0.5mg/kg IV has been shown to decrease the incidence of post-extubation laryngospasm after tonsillectomy with or without adenoidectomy by attenuating laryngeal reflexes. [3] Common side effects of propofol use include hypotension, apnea and bradycardia and were not observed with this dosage. In addition, airway support may be necessary due to transient ventilatory depression caused by propofol. [3] The benefits of laryngospasm prevention far outweigh the possible side effects of propofol administration. Another study found that IV lidocaine is effective in preventing post extubation laryngospasm in children undergoing cleft palate surgery.[7] A dose of lidocaine 1.5mg/kg IV was used to blunt the laryngeal reflex and decrease post-extubation laryngospasm of children undergoing cleft lip and palate surgery. [7] Lidocaine is a relatively safe drug used commonly in anesthesia practice for its ability to attenuate airway reflexes.

Endotracheal tube cuff lidocaine has also been studied and compared to IV lidocaine to determine its effectiveness in preventing laryngospasm in short pediatric surgeries.[8] Intra-cuff lidocaine did not alter the incidence of laryngospasm and is not superior to intravenous lidocaine in short pediatric surgeries. [8]

Retrospectively, a modification of the anesthesia plan used would include incorporating both pharmacological and non-pharmacological modalities in decreasing the incidence of a laryngospasm. The addition of lidocaine 1.5 mg/kg IV or a subhypnotic dose of propofol 0.5 mg/kg IV immediately prior to extubation to attenuate the laryngeal response has shown to be effective in decreasing laryngospasm following upper airway surgery.

Anesthesia professionals must be vigilant in not only identifying and treating laryngospasm, but more importantly, taking steps to prevent laryngospasm in the pediatric population undergoing upper airway procedures. Utilizing all available means of preventing laryngospasm is necessary to improve outcomes for pediatric patients undergoing cleft lip and palate repair by reducing the incidence of post-extubation laryngospasm, thereby decreasing mortality and morbidity in this population.

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Importance of Endotracheal Tube Cuff Assessment

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Keywords: Endotracheal tube, cuff, pressure, assessment, manometer

The importance of endotracheal tube cuff (ETTc) pressure assessment is well documented. Studies have revealed adequate cuff pressures to fall within the range of 20 cmH_20 and 30 cmH20.[1] Endotracheal tube cuff pressures below 20 cmH_2O may allow inspired anesthetic gases to escape from patients' lungs, polluting the operating room environment. An ineffective cuff-tissue seal may also lead to inadequate ventilation and an increased risk of aspiration.[2,3] Conversely, cases of overinflated ETTc pressures resulting in tracheal tissue damage at the site of the inflatable cuff following tracheal intubation appear throughout the literature as well.[4]

Case Report

A 73 year-old male diagnosed with prostate cancer was scheduled to undergo a robotic radical cystectomy, prostatectomy, and urethrotomy with ileal conduit diversion procedure. He stood 175 cm tall and weighed 82.6 kg. Allergies to sulfa and steroid medications, symptoms of which included the formation of a rash and gastrointestinal bleeding respectively, were reported. The patient's past medical history included hypertension, diabetes, and chronic lumbar pain with radiculopathy to his right lower extremity. His medication regimen included tamsulosin hydrochloride, lisinopril, glipizide, and hydrochlorothiazide and his preoperative laboratory results were within normal limits with the exception of a blood urea nitrogen level of 36 mg/dl and creatinine level of 2.1 mg/dl.

The patient's airway exam provided the following information: Malampatti II, thyromental distance of 6 cm, and mouth opening distance of 5 cm. Following the administration of midazolam 2 mg intravenous (IV), the patient was transported to the operating room (OR). Once in the OR, noninvasive heart rate, blood pressure, and oxygen saturation monitors were applied. He was pre-oxygenated with O_2 10 L/min via facemask and general anesthesia was induced with fentanyl 150 mcg, lidocaine 100 mg, propofol 100 mg, and rocuronium 5 mg IV. One attempt at direct laryngoscopy with a miller 2 blade was used to atraumatically place a size 7.5 ETT. Once secured, the ETTc pressure was adjusted to 20 cmH_20 using the pop-off valve and manual ventilation bag to audibly assess for the presence of an air leak around the ETTc. During the course of the 97 min procedure the patient required ephedrine 20 mg IV and phenylephrine infused at a rate of 0.33 mcg/kg/min IV to maintain his baseline blood pressure.

Thirty-one minutes after incision the institution's biomedical engineering team was summoned to assess a recurring "apnea pressure" alarm on the Apollo anesthesia machine in use. Multiple measures were taken to assess and resolve the issue, such as changing the breathing circuit, adjusting the $ETCO_2$ monitoring line seal, and reassessing the ETTc pressure, which remained at 20 cmH_20. Once the problem was resolved the case proceeded without complications and no further ETTc pressure assessments were undertaken. Upon the conclusion of the procedure, neostigmine 4 mg and glycopyrrolate 0.8 mg IV were administered. With the return of spontaneous ventilation, sustained head lift lasting 5 seconds in duration, and no notable fade using train of four nerve stimulation the ETT was removed and the patient was transported to the post-anesthesia care unit. The following day, the patient denied experiencing any residual complications from the anesthetic including throat discomfort due to the presence of the ETT.

Discussion

While a number of complications resulting from under inflation of an ETTc have been known to occur over the course of a procedure, over inflation of an ETTc is also a concern.[2,3] Endotracheal tube cuff over inflation damages tissues within the airway by compressing the mucosa against tracheal cartilage and overcoming the mean capillary pressure of 30 mmHg.[5] Ischemic effects can result in as little as 10 hours and may eventually lead to the formation of tracheal stenosis, trachea-esophageal fistulas, recurrent laryngeal nerve palsy, and vocal fatigue.[6-8]

Five ETTc pressure management techniques are routinely observed in practice and include the predetermined volume technique, the palpation technique, the syringe technique, the minimal occlusive volume technique, and manometer utilization.[9] The predetermined volume technique requires the practitioner to simply inject a set amount of air from a syringe into the inflatable ETTc. This is often followed by the palpation technique, in which the anesthesia professional palpates the associated pilot balloon and adjusts the contents of the inflatable cuff until an appropriate tension is achieved.

Another maneuver, termed the syringe technique, uses a syringe as a pressure relief valve by attaching it to the pilot balloon for the duration of the procedure. This method is inexact, however, given that most syringes require varying amounts of pressure to change the plunger depth. The minimal

occlusive volume technique takes a more individualized approach by decreasing the volume of the cuff until an audible leak from the seal is detected, at which point air is once again added to the cuff in increments of 0.2 ml until an airtight seal is attained.

Though the techniques used to estimate ETTc pressure described above are used widely in practice, the only reliable and measurable means of measuring ETTc pressures requires the use of a manometer. This device, when attached to the pilot balloon, provides the practitioner with an unbiased and specific pressure value. The contents of the inflatable cuff can then be adjusted accordingly to achieve the desired ETTc pressure. Unfortunately, manometers are not always easily accessible in the operating room environment. For this reason, the minimal occlusive volume technique was employed to assess the ETTc pressures during the case detailed above.

Though widely known and utilized in practice, research has shown ETTc pressure estimation techniques to be unreliable indicators of cuff pressures. A study conducted by Rose et al. found the average ETTc pressure of patients under the care of anesthesia practitioners to be 41 cmH_20, far exceeding the pressure needed to cause tissue ischemia.[10] Even experienced anesthesia practitioners are unable to correctly and reliably estimate the pressures within ETT cuffs. No relationship between the cuff pressure measurements and the credentials or tenure of the intubating practitioner has been established.[11] Other studies focusing on patients treated in the emergency department, intensive care unit, and in-flight transport settings revealed similar results.[5,7] Thus, practitioners must use a manometer to assess the ETTc pressures of all intubated individuals in their care, including those intubated elsewhere.

Although the patient featured in the case discussion was unaffected by the placement or management of the endotracheal tube, a retrospective analysis of the ETTc pressure assessment method utilized revealed multiple shortcomings. First, and foremost, a manometer would have provided an unbiased, definitive ETTc pressure measurement. Furthermore, patient movement has been shown to affect the pressures within endotracheal tube cuffs.[12] While the patient was not repositioned during the duration of the case, movement often occurs following tracheal intubation and at specific points in many procedures. Therefore, periodic ETTc pressure assessments throughout the procedure would provide an added measure of patient safety. In addition, although nitrous oxide was not employed in this particular case, infusing the ETT cuff with saline or lidocaine rather than air on a routine basis would prevent ETTc variation in the instances in which the gas is used.[13] By employing these assessment tools and techniques, anesthesia practitioners can potentially improve the short- and long-term outcomes faced by individuals undergoing both acute and chronic tracheal intubation.

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Intraperitoneal Hyperthermic Chemotherapy

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Keywords: fluid management, temperature regulation, intraperitoneal hyperthermic chemotherapy

Cytoreduction with intraperitoneal hyperthermic chemotherapy (IPHC) is a technique being used in the treatment, curative or palliative,[1, 2] of cancers such as peritoneal mesothelioma or pseudomyxoma peritonei.[3] Due to the large number of patients with a diagnosis of peritoneal carcinomatosis,[1] the likelihood that anesthesia professionals will be faced with the challenge of managing such cases may be increasing.[2] Familiarity with the surgical procedure and an understanding of its potential dynamic impact on the patient's overall stability are imperative.

Case Report

A 48 year old male presented to his primary care physician with abdominal bloating. After computed tomography scan revealed the presence of an abdominal mass and associated findings suggested disseminated peritoneal adenomucinosis (DPAM), or

pseudomyoma peritonei, the patient was scheduled for cytoreduction and IPHC.

The patient's height and weight were 178 cm and 109 kg, respectively. Furosemide was prescribed to treat lower extremity edema. Preoperative laboratory values were within normal limits with the exception of hemoglobin 12.6 g/L, hematocrit 38.9%, glucose 410 g/dL, and albumin 2.9 g/dL.

Once the patient's identification, NPO status, allergies, and correct procedure were confirmed, he was premedicated with midazolam 2 mg through an existing 18 gauge peripheral intravenous catheter (IV). Upon arrival to the operating room (OR), standard monitors were applied and preoxygenation begun. A modified rapid sequence induction was accomplished with lidocaine 100 mg, propofol 160 mg, and rocuronium 80 mg. Cricoid pressure was held until equal breath sounds were heard bilaterally and positive end tidal CO_2 confirmed via the endotracheal tube. Central venous access was obtained and central venous pressure (CVP) was monitored continuously thereafter. CVP values ranged from 8-20 mmHg during the case. The right radial artery was cannulated and invasive blood pressure monitoring was initiated. A forced air warmer and an in-line fluid warmer were utilized. The temperature ranged from 33.9˚ to 38.0˚C. Eleven liters of ascitic fluid were evacuated from the abdomen by the surgical team prior to debulking. Anesthesia was maintained with isoflurane and an infusion of sufentanil (385 mcg total). Neuromuscular blockade was maintained with vecuronium. Output included estimated blood loss of 1000 ml and urine output of 1800 ml. Intravenous fluid totals were as follows: lactated ringer's 1L, 0.9% sodium chloride 2L, hetastarch 1L, albumin 5% 4L, 750 ml, plasma-lyte 8L, and packed red blood cells 1,125 ml.

At procedure completion, muscle relaxant was pharmacologically reversed, the oropharynx was suctioned, and spontaneous respirations with regularity, adequate tidal volumes and S_{PO2} were noted. No stridor was present after extubation of the trachea. The patient was transported with supplemental oxygen to the intensive care unit (ICU) where he remained for 24 hours. He was then transferred to a regular hospital room and discharged 12 days later.

Discussion

One of two techniques, "closed" or "coliseum", may be used for IPHC. The closed technique involves the traditional large incision for abdominal exploration and debulking followed by incomplete closure of wound, leaving an opening for instillation and later drainage of the chemotherapeutic medication. This small opening is then closed upon termination of the procedure. One disadvantage of the closed technique is that as the abdomen expands with the introduction of fluid, there is an increase in intraabdominal pressure which could reduce lung compliance and impair oxygenation.[1] The coliseum technique entails leaving the abdomen suspended open as the chemotherapeutic agent is delivered to the open abdomen. A hyperdynamic state in patients undergoing IPHC has been attributed to the heated fluids placed into the abdominal cavity. The temperature, cardiac index, heart rate, and oxygen consumption are all found to increase, while pH decreases.[1, 4] With either technique, maintenance of normothermia and normovolemia, along with pain management, will be primary concerns.

Cytoreductive surgery with IPHC may be considered a biphasic procedure with each phase accompanied by its own set of

concerns. The first phase includes cytoreduction, or debulking of the tumor. A large abdominal incision is created followed by excision of abnormal tissue and drainage of ascitic fluid, if present. Fluid losses may be extensive [4] during this phase. Large blood loss is also possible, particularly if the tumor is quite invasive or if coagulopathy is present.[1] These changes result in large fluid shifts which may result in significant hemodynamic instability. Another concern throughout this phase is of hypothermia, therefore it is necessary to closely monitor core temperature and be prepared to apply a forced warm air blanket and IV fluid warmer if necessary. However, in anticipation of the impending hyperthermic therapy, it is desirable to allow the body temperature to fall moderately.

The second phase consists of the introduction of warmed (42-45°)[1, 2, 3] chemotherapy agent to the abdominal cavity. The patients position is then altered from tilt, left and right, to slight trendelenburg and reverse trendelenburg which allows maximal contact between tissue surfaces and chemotherapeutic agent preventing the higher systemic levels found with the traditional intravenous route.[2] During this phase anesthesia professionals may need to address hyperthermia and untoward effects related to the specific chemotherapy agent used. Increases in temperature beyond normal range are expected and may be treated by cooling ambient temperature, discontinuing fluid warmer use, and starting the cooling blanket. Fluid balance remains a concern during this phase as insensible fluid losses, especially through perspiration, may be extensive.

Most scholars agree on maintaining preload and adequate circulating volume; other issues of fluid therapy, however, remain debatable, including monitoring techniques, as well as amount and type of fluid to be delivered.[1, 2, 5-7] In the presented case, CVP values guided preload assessment. However, the use of CVP and pulmonary capillary wedge pressure monitoring as tools to guide fluid management has been questioned.[5,7] Some scholars agree that esophageal doppler monitoring is a superior tool for guiding fluid administration.[7,8] Others propose ventilation-induced plethysmographic variations as a superior guide to optimizing patient fluid status.[7] Despite what monitoring technique is chosen, anesthesia professionals must plan for judicious use of a combination of fluids for patients undergoing cytoreduction with IPHC.

Anesthesia professionals should be aware of the patients risk for both hypo- and hyperthermia. While core temperature is normally closely regulated and maintained within tight parameters by the hypothalamus, the same cannot be said for the operative patient's temperature control.[9] In the OR, not only may the patient's temperature be affected by such variables as exposure to a cold environment, cool IV fluids, extended duration of surgery, and evaporation, but temperature regulation is also compromised in several important ways by anesthetics.[9,10] First, behavioral alterations, such as seeking more clothing when exposed to cold ambient temperatures, are no longer possible.[9,10] Autonomic control of temperature is impaired so that triggering temperatures lie outside normal thresholds,[9] allowing fluctuations far beyond normal ranges. Furthermore, anesthetics may cause vasodilation with subsequent, unintended heat loss.[10] Lastly, neuraxial anesthesia, if employed, may impair thermoregulation. The mechanisms by which normal responses become altered are not fully understood but may lead to ineffective compensation for changing

temperatures, contributing to either hypo- or hyperthermia.[9]

The long procedural duration and large incision require thoughtful implementation of pain control interventions. In 2008 Schmidt reported better control of pain, increased likelihood of tracheal extubation, and shorter hospital stays were likely to occur with use of continuous thoracic epidurals, both intraoperatively and postoperatively. The procedure in this case lasted nearly thirteen hours and included right hemicolectomy, bilateral stripping of diaphragms which led to pneumothorax and chest tube placement, cholecystectomy, splenectomy, distal pancreatectomy, omentectomy, partial resection of right hemidiaphragm, pelvic stripping, and peritoneal wall stripping followed by IPHC. Postoperatively, the patient was placed on intravenous patient controlled analgesia with hydromorphone. In restrospect, placement of an epidural catheter may have increased the patient's level of comfort, decreased narcotic requirements throughout the case, and shortened hospital stay.

Fluid management, temperature regulation, and pain management are top priorities in the care of patients having cytoreductive surgery with IPHC. Being familiar with the procedure and associated requirements of care are essential to a positive outcome.

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Malignant Hyperthermia: An Anesthetic Plan

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Keywords: malignant hyperthermia, succinylcholine, total intravenous anesthesia, in vitro contracture test, caffeine-halothane contracture test

Malignant hyperthermia (MH) is a rare, inherited disorder of skeletal muscle.[1] MH signs and symptoms are caused by an abnormal, diffuse release of calcium from the sarcoplasmic reticulum. Signs and symptoms include tachycardia, muscle rigidity, hyperthermia, increases in end-tidal carbon dioxide (CO_2) and oxygen consumption, and metabolic and respiratory acidosis. Specific anesthetic triggers for MH currently used in the United States include the volatile agents desflurane, isoflurane and sevoflurane and the depolarizing muscle relaxant succinylcholine.[2] According to a review by Rosenberg, et al, MH occurrence during anesthesia ranges from 1:5,000 to 1:50,000-100,000 anesthesias.[3]

Case Report

A 52 year old, 97 cm, 166 kg female presented for a total abdominal hysterectomy and panniculectomy. Past medical history was significant for morbid obesity, hypertension, Type II diabetes, obstructive sleep apnea, endometrial cancer and a maternal family history of malignant hyperthermia. The patient's aunt died from MH and her cousin had a positive *in vitro* contracture test (IVCT). The patient was not biopsied for MH and had never undergone general anesthesia. The patient had no known drug allergies and current medications included lisinopril-hydrocholorthiazide, metoprolol, glipizide, metformin, and simvastatin.

Preoperative evaluation revealed American Society of Anesthesiologist's (ASA) physical status III, Mallampati class III, thyromental distance of 5 cm, finger breadth mouth opening of 5 cm, limited neck range of motion and a large neck circumference. Breath sounds were clear to auscultation bilaterally and heart rate and rhythm were regular. Preoperative vital signs were blood pressure of 129/50 mmHg, heart rate of 73, and Sp_{O2} of 91% on room air. Complete blood count, complete metabolic panel and coagulation studies were within normal limits.

The patient was medicated with fentanyl 50 micrograms (mcg), midazolam 1 milligram (mg), celecoxib 400 mg, and pregabalin 150 mg in the regional anesthesia procedure room. Multiple attempts at an epidural cathether were unsuccessful and the patient was transported to the operating room. Prior to patient arrival the anesthesia gas machine had a continuous flow of oxygen at a rate greater than 10 liters per minute for 30 minutes, the CO_2 absorbent had been changed and the vaporizers made inaccessible. ASA standard monitors were applied and the patient was pre-oxygenated with 10 liters oxygen via face mask. A dexmedetomidine infusion was started at 0.7mcg/kg/hr, 100 mcg of fentanyl was given intravenously (IV) and an awake fiber optic tracheal intubation was accomplished by the anesthesiologist. Endotracheal intubation was confirmed by positive end-tidal carbon dioxide on the monitor and bilateral breath sounds were auscultated. General anesthesia (GA) was induced with a 200mg bolus of propofol and 50mg of

rocuronium IV. A bispectral index monitor (BIS) was applied to the patient's forehead. GA was maintained with continuous infusions of dexmedetomidine, propofol and remifentanil. Infusions were titrated to maintain the BIS monitor at 40-60.

The remainder of the case proceeded without incident. At the end of the case the patient was noted to have scleral edema from being in trendelenburg position for more than six hours. A leak test around the endotracheal tube was negative and the patient remained intubated to the post anesthesia care unit with her head elevated to decrease airway edema.

Discussion

MH can lead to significant morbidity and mortality following general anesthesia. In order to understand more about MH and provide information for clinicians, the European Malignant Hyperthermia Group (EMHG) and the North American Malignant Hyperthermia Group (NAMHG)[4] have been formed. The diagnosis of MH is currently carried out by *in vitro* testing. It is considered the "gold-standard" for diagnosis of MH. The NAMHG administers the caffeine-halothane contracture test (CHCT) and the EMHG administers the in vitro contracture test (IVCT). Testing can be painful and many patients are unable to be tested due to the expense and the limited number of testing centers. There are only six NAMHG approved biopsy centers in North America.[4]

CHCT and IVCT are used interchangeably. Their differences include the number of muscle fibers, the amount of agents being used and the values used for positive and negative results.[4] The IVCT takes the skeletal muscle biopsy, exposes it to increasing amounts of caffeine and halothane, then stimulates it and measures the muscle contraction.[5] The patient's cousin underwent IVCT testing at the Chicago testing center and was found to be positive for MH. While the facility where this patient had her surgery was designated as a CHCT testing center, the patient was unable to obtain a CHCT due to the test's cost.

Patients who have been diagnosed as MH susceptible through IVCT or have a known history of MH have two options for anesthesia: a) GA with non-triggering agents or, b) regional anesthesia, if appropriate. GA with non-triggering agents includes the use of IV or non-volatile anesthetics, analgesics and non-depolarizing muscle relaxants. Inhalational, non-triggering anesthetics include nitrous oxide and xenon. IV anesthetics include propofol, benzodiazepines, dexmedetomidine, barbiturates, etomidate and ketamine hydrochloride. Opioid based analgesics include fentanyl, sufentanil and remifentanil. Non-depolarizing muscle relaxants used for GA in MH patients include rocuronium, pancuronium, vecuronium and cisatricurium. Most local anesthetics, either amide or ester based, can be used along with IV agents, to achieve the desired amount of analgesia and amnesia during regional anesthesia.[1] An attempt was made to administer regional anesthesia to the patient, but was unsuccessful. Total IV anesthesia was administered successfully.

The anesthetic gas machine must be prepared before anesthesia can be administered to MH susceptible patients. Past recommendations required flushing the circuit with a fresh gas flow of 10 liters per minute for at least 10 minutes. These recommendations were based on older styles of anesthetic machines with no rubber components like the Ohmeda Modulus 1 and 2 machines.[6] Newer anesthetic machines

like the Drager Fabius GS, require significantly longer fresh gas flows to completely wash out inhalational anesthetics to an acceptable level of less than five parts per million.[1,7] Unable to measure the amount of inhalational anesthetics left in the circuit, we made the decision to flush the circuit with flow rates greater than recommended amounts for longer than suggested times. At no point during the procedure did the patient display any signs or symptoms of MH.

Regional anesthesia was chosen as the best alternative for this patient due to the patient's size and the fact that the patient's airway exam suggested the possibility of a difficult airway. Every effort was made to administer regional anesthesia through an epidural catheter, but the regional anesthesia team was unable to insert an epidural catheter after multiple attempts. Discussions with the anesthesia care team led to altering the plan by conducting an orotracheal intubation via flexible fiber optic scope. The endotracheal tube was passed through the glottic opening without incident.

While MH can be a lethal result of GA, there are options available to anesthesia professionals to deliver anesthesia safely. The safest option for this patient would have been an epidural catheter with local anesthetics infusing continuously during surgery and moderate sedation with an IV anesthetic. However, it was not possible to administer regional anesthesia, and the next safest choice was intubation of the trachea using a flexible fiber optic scope while the patient was sedated, but still awake.

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Intraoperative Blood Transfusion Reaction

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Key words: transfusion, reaction, acute lung injury, general anesthesia, intraoperative

With surgery comes the risk of blood loss. When blood loss is in excess of ten to twenty percent of a patient's blood volume, a blood transfusion may be required.[1] Although this generally occurs without complication, there are risks. The risks include transfusion related acute lung injury (TRALI), hemolytic reaction, febrile reaction, anaphylactic reaction and complications such as hypocalcemia, hyperkalemia, coagulopathy, and hypothermia. There are signs and symptoms to monitor for when a transfusion reaction occurs, however these become difficult to assess under general anesthesia. It is important to discern the signs early, as early recognition results in improved patient outcomes.

Case Report

A 65 year old female presented for a right cadaveric kidney transplant. Medical history included diabetes, hypertension, gout, osteoarthritis, reported poorly controlled gastroesophageal reflux disease, and end stage renal disease for which she was receiving peritoneal dialysis. Surgical history was significant for a cervical spine fusion with no anesthetic complications. The patient had no known allergies. Home medications included atenolol, bisacodyl, amlodopine, furosemide, omeprazole, fluoxetine, temazepam, and calcium carbonate. The morning of surgery the patient took her atenolol and omeprazole. Preoperative labs were as follows; blood urea nitrogen 64 mg/dl, creatinine 9.3 mg/dl, blood glucose 131 mg/dl, hemoglobin 9.9 g/dl, hematocrit 28.5%, platelets 187,000, white blood cell count 7.8, international normalized ratio 0.96, prothrombin time 10 s, and partial thromboplastin time 24.8 s. Airway assessment revealed Mallampati 2, oral aperture 3 cm, and thyromental distance 3 cm.

Midazolam 2 mg IV and sodium citrate 30 mls by mouth were administered preoperatively and the patient was transported to the operating room where standard monitoring was applied. After preoxygenation, a modified rapid sequence induction was performed with cricoid pressure held. General anesthesia was induced with etomidate 28 mg, fentanyl 100 mcg, lidocaine 30 mg, and rocuronium 70 mg. Direct laryngoscopy was successful with placement of a 7.0 endotracheal tube. A triple lumen central line was placed in the right internal jugular vein and an arterial line was placed in the left radial artery. Dexamethasone 100 mg was given prior to incision consistent with the renal transplant protocol being used.

Several hours into the case, the patient received two units of packed red blood cells (PRBC's) for a hematocrit of 24% and decreasing blood pressure (80/40 mmHg) and SpO_2 saturation (96% on O_2 100%). After completion of the second unit, the patient's heart rate (HR) increased from 60 beats per minute (bpm) to over 100 bpm, the systolic blood pressure (SBP) decreased from 100 mmHg to approximately 70 mmHg, and the central venous pressure increased from 18 mmHg to 30 mmHg. The patient was unresponsive to phenylephrine,

vasopressin, and dopamine boluses and infusions, which all were initiated after completion of the blood transfusion in an attempt to correct the refractory hypotension. An epinephrine infusion was initiated and diphenhydramine and calcium chloride were administered. It was at this point that a transfusion reaction was suspected. A Transesophageal echocardiogram (TEE) was performed, showing a preserved ejection fraction, moderate mitral regurgitation, moderate to severe tricuspid regurgitation, and the left ventricluler underfilling. After initiation of the epinephrine infusion the patient's vital signs stabilized, and TEE revealed improved forward blood flow.

By the end of the procedure all vasopressors had been weaned off. Mannitol and furosemide were administered per renal transplant protocol during the middle of the procedure. At the end of the case there was 30 ml of urine output in the foley catheter bag. The patient was transferred to the intensive care unit with the endotracheal tube in place and stable vital signs. A blood transfusion reaction was suspected to have occurred, but never confirmed as blood samples were never sent to the lab for analysis.

The patient was discharged home nine days postoperatively after experiencing delayed graft function, but with creatinine levels continuing to decrease and requiring only intermittent hemodialysis. A one month postoperative biopsy of the transplanted kidney revealed residual acute tubular necrosis with no acute rejection.

Discussion

Transfusion reactions are classified as either hemolytic or nonhemolytic. Nonhemolytic transfusion reactions consist of febrile reactions, anaphylactic reactions and transfusion related acute lung injury (TRALI). Hemolytic transfusion reactions occur when the patient is transfused with PRBC's that are ABO incompatible with their own blood. This results in intravascular hemolysis that occurs through activation of the complement system. A hemolytic reaction is generally severe. Signs include fever, chills, skin flushing, hypotension, tachycardia, nausea, lumbar and substernal pain, hemoglobinuria, dyspnea, and oozing in the surgical field.[1,2] Treatment includes immediate cessation of the transfusion, liberal administration of isotonic fluids, sodium bicarbonate to increase urine pH > 8, osmotic diuretics, and if rapid blood loss is occurring, fresh frozen plasma and platelet transfusions.[1-3] Repeat compatibility testing should also occur.

Febrile reactions are fairly common and frequently indicate an interaction between antibodies of the recipient and antigens on the leukocytes of the donor. It is manifested by an increase in temperature > 1^{o} C while transfusing the blood product or shortly thereafter. Treatment involves slowing of the transfusion, administration of antipyretics, and subsequent transfusions with leukocyte poor PRBC's.[1,2,4]

Anaphylactic reactions occur in patients who have an immunoglobulin A (IgA) deficiency. They develop autoantibodies against IgA and a severe anaphylactic reaction can occur when transfused with IgA containing blood products. Signs include hypotension, bronchospasm, dyspnea, loss of consciousness, respiratory arrest and shock. Treatment includes immediate cessation of the transfusion, IV fluids, and epinephrine. An anaphylactic reaction is severe, occurs immediately after initiation, and takes only several milliliters of blood to cause a reaction.[1]

An interaction between transfused blood products and the patient's white blood cells (WBC's), TRALI was the leading cause of transfusion related fatalities in years 2005-2009.[1,3,5,6] The WBC's aggregate in the pulmonary microvasculature and congestion results, leading to noncardiogenic pulmonary edema. It presents as a respiratory distress syndrome that is manifested by pulmonary edema, dyspnea, marked hypoxemia, fever and hypotension. This reaction can be severe and when suspected the transfusion should be stopped immediately. Support of vital signs and the respiratory system may be necessary and IV fluids should be administered. Diuretics are not indicated, as the respiratory distress is due to microvascular injury rather than fluid overload. TRALI

Transfusion of PRBC's has other commonly occurring complications that can manifest as changes in hemodynamic status. Stored PRBC's contain an anticoagulant preservative comprised of citrate-phosphate-dextrose-adenine (CPDA). The citrate in CPDA binds calcium. Therefore, administration of multiple units of PRBC's can theoretically lead to hypocalcemia and resultant decreased cardiac function. Hypocalcemia should be treated with IV calcium chloride or calcium gluconate. Citrate is primarily metabolized by the liver and thus, hypocalcemia may be more pronounced in patients with hepatic disease.[1,3]

The extracellular potassium concentration in stored PRBC's steadily increases with time. In one study, PRBC's that had been stored for ten days, had a mean potassium concentration of 27.3 mEq/L. This level of concentration given at a rapid rate could potentially be enough to induce cardiac dysrhythmias. Treatment of hyperkalemia may include administration of IV calcium, 50% dextrose, insulin, sodium bicarbonate, and/or furosemide.[1-3,7]

Many signs of a transfusion reaction are masked by general anesthesia. Several of the signs mimic the side effects of general anesthesia such as hypotension and tachycardia. With an adverse transfusion reaction, the hypotension and tachycardia is often unresponsive to treatment, as occurred in this case. The patient's hypotension was unresponsive to pheynylephrine, dopamine, and vasopressin. It was never confirmed if the refractory hypotension was due to a transfusion reaction or not, but the patient's hemodynamics did seem to improve after administration of epinephrine and diphenhydramine which are standard treatments for an anaphylactic reaction. It was a quandary as to whether a transfusion reaction occurred; why did it not manifest until after the second unit, and why with such severity? With all the activity of the situation a sample of the patient's blood and the transfusion bag of PRBC's was never sent to the blood bank for analysis to confirm if and what type of reaction had occurred. If a transfusion reaction is suspected, the patient's blood and the transfused blood should always be sent for repeat compatibility testing.

In conclusion, it is imperative that anesthetist's are vigilant during transfusion of blood products, and consider any significant changes in hemodynamics or respiratory parameters as a potential adverse reaction. This report is intended to help learning anesthesia practitioners recognize the signs of an adverse reaction quickly and manage them appropriately to allow for the safest possible outcome for the patient.

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Alpha-2 Agonists and Postoperative Delirium

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Keywords: Postoperative delirium, emergence delirium, alpha$_2$ -agonists, clonidine, dexmedetomidine

Postoperative delirium (PD) is a transient disturbance of consciousness occurring shortly after surgery.[1] Although more common in pediatric and elderly populations, PD in any patient is associated with increased morbidity, delayed functional recovery, prolonged hospital stay, and mortality.[1] Identifying patients at risk for PD and developing an anesthetic plan to prevent or attenuate PD is a challenge faced by anesthesia professionals daily.

Case Report

The patient was a 182.9 cm, 136.9 kg, 47 year old, obese male with a past medical history of hypertension, dyslipidemia, and a transient ischemic attack two years ago. He presented to the operating room for a split thickness skin graft revision and Wound V.A.C (KCI, Inc., San Antonio, TX) placement to his perineum due to Fournier's gangrene infection. Past surgical history included a herniated disk repair 15 years ago, initial perineal debridement two weeks ago at an outside hospital, and two other perineal revisions. No anesthetic

complications were noted in the medical record; however during the preoperative interview, the patient stated he "went crazy" in the recovery unit with the first two debridement surgeries. A dexmedetomidine bolus and infusion were used during the last revision. The infusion was discontinued after emergence and according to the patient he did not experience delirium. The airway assessment was unremarkable except for the presence of facial hair and a short thick neck. Medications included Benazepril and simvastatin daily.

The patient was premedicated in the holding room with intravenous (IV) midazolam 2 mg and clonidine 50 mcg. Standard monitors were applied in the operating room (OR) and the patient was pre-oxygenated. A smooth IV induction was accomplished using fentanyl 250 mcg, thiopental 400 mg, and rocuronium 70 mg. The patient was easy to mask ventilate with an oral airway in place. Direct laryngoscopy was performed using a Miller 2 blade and intubation of the trachea with a 7.5 mm endotracheal tube was successful. Anesthesia was maintained with isoflurane and additional boluses of IV fentanyl 100 mcg as needed. Midway through the case, the patient was given an additional 50 mcg of clonidine IV to achieve a therapeutic dose (0.5-5 mcg/kg), and the two hour surgery progressed uneventfully. Neuromuscular blockade was antagonized with neostigmine 5 mg and glycopyrrolate 1 mg. The patient was extubated without complication and transported to the post anesthesia care unit (PACU) with supplemental oxygen. During the post-operative visit, the patient stated that he "woke-up comfortably" in the recovery unit.

Discussion

Identifying patients at risk for PD is an important part of the preoperative assessment and interview. In this case, the patient had a history of PD and was able to communicate this information to the anesthesia professional. The patient stated during the interview that he "went crazy" and remembered waking up in restraints after his first two surgeries. The patient communicated that he was given "some medicine" in his IV during his last surgery, and he did "much better". A review of the anesthetic case record from his most recent surgery revealed that the patient was intraoperatively placed on a dexmedetomidine infusion to prevent postoperative delirium.

Risk factors for PD include: age 70 or older, history of delirium, history of alcohol abuse, established cognitive impairment, underlying cerebral disease, prolonged cardiac bypass time, preoperative use of narcotics and preoperative depression.[1,2] Other factors that may contribute to development of PD include: physical illness, infection, hormone disorders, nutritional deficiencies, metabolic disturbances and electrolyte imbalance.[1]This condition is usually transient, lasting from hours to days; However, if it is not identified and treated early, PD can lead to irreversible dementia or death.[1] PD increases the risk of complications such as pulmonary edema, myocardial infarction, respiratory failure, pneumonia, and death.[1] PD also presents challenges for staff who care for these patients in the PACU and intensive care unit (ICU). Safety concerns for patients experiencing PD include self-extubation, pulling out catheters, respiratory failure after exubation, prolonged hospital stay, increased bleeding from operative site, and disturbance of other patients.[3,4] Treatment for patients experiencing PD often includes the use of opiates, benzodiazepines and antipsychotics such as haloperidol. The use of these medications is

not without risks and often contributes to or exacerbates the delirium. Alpha$_2$-agonists such as dexmedetomidine and clonidine have been established as an effective therapy to prevent or attenuate PD without significant respiratory depression. Dexmedetomidine and clonidine provide sedation and maintain stable blood pressure and a slower heart rate.[3] Use of alpha$_2$-agonists as a pre-medication allows for a significant reduction in the amount of anesthetic required.[5] Reductions in minimum alveolar concentration (MAC) of inhaled anesthetics has also been demonstrated with the use of alpha$_2$-agonists preoperatively.[5] Decreasing the amount of inhalation agent and opioids administered may help decrease the occurrence or severity of emergence or postoperative delirium. Alpha$_2$-agonists provide dose-dependent sedation, analgesia, sympatholysis and anxiolysis without respiratory depression.[1]

This patient expressed concern regarding his anesthesia. Waking up restrained caused him great emotional distress. Aside from emotional injury, anesthesia professionals must consider potential threats to the physical safety of patients as well as the PACU staff. The patient was a strong, large male who if became agitated and confused was capable of harming himself and the staff. The use of chemical restraints, such as narcotics or benzodiazepines, would put the patient at increased risk of respiratory complications due to his obesity. Protection of the surgical site, the skin graft and Wound V.A.C. was another consideration. Disruption of the graft would possibly require more surgery or lead to increased scarring. The application of physical restraints, used only as a last resort, comes with a list of legal and safety concerns also. The patient's history and past successful use of a dexmedetomidine infusion was discussed with the anesthesia care team. Intravenous clonidine was chosen as an alternative to a dexmedetomidine infusion because it was less expensive. Clondine was initially given as a premedication in the holding room, and then repeated halfway through the case to achieve a smooth emergence.

Both clonidine and dexmedetomidine have been used successfully for this patient in preventing PD. Dexmedetomidine is a more potent alpha$_2$-agonist and has a faster offset than clonidine which may be desirable in same day or outpatient procedures. Clonidine is a less expensive alternative to dexmedetomidine, lasts longer, and can be continued in the postoperative period as an oral or transdermal medication. Anesthesia professionals should consider the use of alpha$_2$-agonists in the care of patients identified as at risk for PD. Decreasing the occurrence of PD will improve postoperative outcomes and increase patient satisfaction.

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Phenylephrine and TRAM Graft Survival

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Keywords: Phenylephrine, TRAM flap, graft survival, vasopressor

A positive outcome of breast reconstruction with a pedicled transverse rectus abdominus myocutaneous (TRAM) flap is dependent on the ability of the flap to be perfused during and after surgery. Factors such as temperature, volume status, hematocrit, and blood pressure can affect the perfusion of the flap. Successful anesthetic management of TRAM flap surgeries are often accomplished with the goal of avoiding vasopressors, such as phenylephrine, because of the risk of vasoconstriction and hypoperfusion of the flap. The following case report illustrates an unsuccessful TRAM flap procedure where no phenylephrine was used to maintain blood pressure.

Case Report

The patient was an 81 kg, 65 year old female, with no history of vascular disease, admitted for a left breast reconstruction with transverse rectus abdominus myocutaneous (TRAM) flap. She had a history of breast cancer with bilateral mastectomy and consumed approximately seven alcoholic drinks per week. She had multiple previous surgeries with no anesthesia complications. Pre-operative labs were unremarkable.

The patient was met in holding room and one 22 gauge intravenous (IV) catheter was placed in the right wrist. The patient was taken to the operating room and assisted to the operating room table. She was then placed in the supine position, routine monitors were applied and was pre-oxygenated with $O_2$100% via facemask. The patient's preinduction blood pressure was 118/73. Medications given for induction were lidocaine 50 mg, propofol 200 mg, and rocuronium 50 mg. Intubation with a 7.0 endotracheal tube was accomplished via direct laryngoscopy with a Miller 2 blade. The endotracheal tube was secured at 22 cm at the teeth and both eyes were taped closed. An esophageal stethoscope/temperature probe was inserted and a lower body air warmer was placed over the lower extremities. An 18 gauge IV was inserted in the right arm and an inline fluid warmer was then attached.

Anesthesia was maintained with approximately 0.5 MAC of isoflurane, 1 liter N_2O, 1 liter O_2, and a total of 3 mg of dilaudid. Neuromuscular blockade was maintained with a total of 3.5 mg of pancuronium. The patient received lactated ringers 3500 ml, normal saline 500 ml, and 6% hetastarch 500 ml. Estimated blood loss was 150 ml and urine output was 825 ml via Foley catheter. Prior to the end of the case, dexamethasone 10 mg, droperidol 0.625 mg,

and odansetron 4 mg were given. Systolic blood pressure was maintained between 96 and 125 mmHg and temperature ranged from 36.6 to 37.2 degrees centigrade throughout the case. The patient's vital signs were remained stable throughout the case.

At the end of the case neuromuscular blockade was antagonized with neostigmine 4 mg and glycopyrrolate 0.8 mg. When the patient was responsive and spontaneously breathing she was orally suctioned, the endotracheal tube was removed, and she was placed on O_2 100% via facemask. The case lasted approximately six hours. During the fifth hour, the surgeon placed a pain pump in the abdomen which infused 0.25% bupivicaine at 2 ml/hr. The left breast was unable to be completely closed due to what the surgeon termed "poor perfusion due to venous congestion". The end of case hematocrit and hemoglobin were 34 and 11.4 g/dl respectively. The patient required two additional surgeries which eventually resulted in graft survival.

Discussion

During a pedicled TRAM flap surgery, the harvested tissue is usually only perfused through one artery and one vein. The flap is harvested from the abdominal area and left connected to a blood supply through the pedicle. The flap is then pulled under the fascia superiorly and pulled up to form the new breast. Among other considerations, it is critical for the anesthesia practitioner to maintain a stable and sufficient blood pressure throughout the surgery to adequately perfuse the flap. The induction of general anesthesia and the maintenance of general anesthesia can often lead to hypotension. Generally vasopressors are avoided because of the risk of constricting the only vessel perfusing the tissue.[1] The concern of the negative effects of vasopressor use, specifically phenylephrine, might have on the flap is shared by the anesthesia practitioner as well as the plastic surgeon.

In the case presented, the flap failed to be perfused adequately, and for that reason could not be closed at the end of the case. No vasopressors were used during the case. The patient's mean arterial pressure was maintained above 65 mmHg throughout the case with boluses of colloids and crystalloids. The question, however, must be asked, would the use of phenylephrine have hurt or helped in this case.

Phenylephrine is a synthetic noncatecholamine that is highly specific to alpha-1 adrenergic receptors. Phenylephrine is a strict alpha-1 agonist. It has little, to no, inotropic or chronotropic effects, although reflex bradycardia can occur. In usual IV doses, phenylephrine is a stronger venoconstrictor than arterial constrictor.[2]

In a 2010 retrospective study by Chen et al., of 187 patients undergoing different flap surgeries showed no difference in the outcome between the patients who received ephedrine and/or phenylephrine and those that did not. In fact, the authors found that, phenylephrine did not increase the rates of reoperation or flap loss. [3] An animal study in 1999 compared the use of phenylephrine with the use of sodium nitroprusside in porcine free flaps. Ultrasound flowmetry was used to measure total blood flow in the flaps and laser Doppler flowmetry was used to measure microcirculation. The results showed decreased blood flow when sodium nitroprusside was used, but no negative effect to free flap blood flow when phenylephrine was used. The phenylephrine caused a 30% rise in systemic vascular resistance and arterial pressure, but no change in the free flap blood flow.[4]

A similar study in 1997 assigned pigs to have either general anesthesia or "extradural" anesthesia. The pigs' microcirculation was then tested at a normovolemic state and at a hypovolemic state (10% blood loss). The results of the study indicated that an infusion of phenylephrine caused a slight increase in microcirculatory flow in pigs who were normovolemic but the microcirculatory flow in hypovolemic pigs remained unchanged. [5]

With the lack of human studies on this subject, it is difficult to come to a conclusion concerning the use of phenylephrine during TRAM flap procedures. Summarizing most of the studies, it appears that the IV administration of phenylephrine does not decrease the flow in the microcirculation to flaps in normovolemic patients. The studies reviewed suggest that there is no rise in negative outcomes when phenylephrine is administered. Therefore, anesthesia practitioners should fully resuscitate the patient with fluids and may use phenylephrine to increase mean arterial pressure if needed. The anesthesia practitioner should also understand that the increase in blood pressure may not necessarily equate to increased perfusion of the flap.

In the case provided, no vasopressors were used. The patient was sufficiently rehydrated with fluids. However, the TRAM flap was not well perfused at the end of the case. However, two additional surgeries were required to assure the grafts survival. From the research found, one could conclude that administering phenylephrine to the patient probably would not have decreased perfusion to the flap, but most likely would not have increased perfusion to the flap either. It is apparent that there is currently an insufficient number of human studies and recent research on the subject. To come to a definitive conclusion more research is needed on the subject of phenylephrine use and TRAM flap survival.

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Awake Craniotomy Airway Management

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Keywords: Awake craniotomy, laryngeal mask airway, LMA, airway management, awake insertion

The asleep-awake-asleep craniotomy poses significant challenges, since the anesthetist must ensure rapid emergence after the first asleep segment, patient comfort and cooperation during the awake segment, and satisfactory surgical conditions throughout the procedure. Of special importance is management of the patient's airway, particularly at the beginning of the second asleep segment when the airway must be secured while the patient is positioned in the right lateral decubitus position (RLD) in Mayfield skull clamps. This case study will discuss the novel approach of inserting the laryngeal mask airway (LMA; Laryngeal Mask Company, Henley-on-Thames, UK) when the patient is awake, immediately prior to the second asleep segment.

Case Report

The patient was a 100 kg, 188 cm, 65 year-old male who presented for surgical resection of a left temporal glioblastoma using an awake speech mapping technique. Three months before surgery, he had symptoms of word-finding difficulty, garbled speech, and fatigue, and underwent radiation and temozolomide chemotherapy.

The preoperative assessment revealed a history of hypercholesterolemia. The patient's medications included levetiracetam 750 mg and dexamethasone 0.5 mg twice daily and atorvastatin 40 mg and esomeprazole 40 mg once daily. Preoperatively, an 18 gauge catheter was inserted and propofol 30 mg was administered as a small sedative dose. In the operating room, standard monitors were applied and general anesthesia was induced with propofol 220 mg. A size four LMA was inserted into the oropharynx and placement was confirmed with the end tidal carbon dioxide monitor and auscultation of breath sounds. Anesthesia was maintained using desflurane 2% in two liters oxygen and a propofol infusion at 140 mcg/kg/min. An arterial catheter was inserted into the patient's right radial artery. Somatosensory evoked potential monitoring was initiated. Mayfield skull clamp fixation was placed after subcutaneous infiltration of bupivacaine 1% and the patient was positioned into RLD position. Cefazolin 2 g, dexamethasone 10 mg, and an infusion of mannitol 100 g were administered.

Prior to incision, the surgeon infiltrated bupivicaine 1% around the frontal branches of the trigeminal nerve. A craniotomy was performed and the dura mater was opened. The desflurane vaporizer and propofol infusion were turned off. The LMA was deflated to reduce noxious stimuli, and it was removed after the patient showed signs of sufficient arousal. Five liters of oxygen via facemask were provided during the speech mapping segment of surgery. A middle temporal corticectomy was performed using stereo-tactic guidance to locate the tumor.

After 70% of the tumor was resected the surgeons decided to end the resection because the patient began experiencing naming errors. The surgeons then requested general anesthesia for surgical closure.

Lidocaine 5% jelly was applied to the posterior facing side of the deflated LMA and it was placed into the oral cavity. As the lidocaine anesthetized the oropharynx, the LMA was slowly advanced into position. Insertion was well tolerated, and after taking note of end tidal carbon dioxide measurements the LMA was inflated. Propofol 150 mg was then administered to re-induce general anesthesia. The desflurane vaporizer and propofol infusion were restarted, and the surgical site was closed. The post-operative course was uneventful. The patient's neurological assessment met the preoperative baseline, and he was discharged on post-operative day four.

Discussion

An asleep-awake-asleep craniotomy is necessary when tumor resections are in the vicinity of eloquent cortex, as in this case, where the tumor affected the Broca's speech area in the dominant hemisphere. Utilization of awake speech mapping during radical tumor resection facilitates maximal removal of the tumor while decreasing the chance of postoperative neurologic deficits.[1,2] Intraoperative complications such as convulsions, increased intracranial pressure, anesthesia induced respiratory depression, and nausea are all airway management concerns that significantly increase patients' morbidity and mortality.[3] Therefore, relative contraindications for the asleep-awake-asleep anesthetic technique include obesity, obstructive sleep apnea, known difficult airway, and symptomatic gastroesophageal reflux disease.[1,2] This patient was an ideal candidate because he did not have any of these contraindications.

Stimulating procedures were performed during the asleep segments of the surgery, including arterial and foley catheter insertion, scalp blockade, Mayfield skull clamp fixation, craniotomy, opening the dura mater, and surgical closure. To secure the airway during the asleep segments, several different airway management methods may be used, including conscious sedation and nasal or oral intubation. In this case, the LMA was selected because it is easily placed, allows the patient to spontaneously breathe, prevents airway obstruction, and is associated with a lower incidence of coughing during the surgery.[4,5] Also, ventilation may be supplemented should apnea or hypercarbia occur.[4]

Of particular importance in this case was safely re-inducing and securing the airway for the second asleep phase. Case reports addressing reinsertion of an LMA for the second asleep segment recommend inducing the patient and then inserting the LMA while the patient is asleep.[1,5] However, these case reports described only craniotomies performed on patients in the supine position. In this case, if the LMA could not be inserted or properly seated after the patient was asleep, emergency management of the airway would be difficult, since the patient was fixed by a Mayfield skull clamp in the RLD position with an open surgical field. After considering the possibilities, it was determined that a safer technique would be awake insertion of the LMA by topically anesthetizing the oropharynx.

The awake LMA insertion technique, though not published in awake craniotomy case studies, has been evaluated in other clinical settings. These studies have proven that awake insertion of the LMA with a variety of different topical local anesthetics is well tolerated in nearly 100% of study patients (Table 1).[6-10] This approach facilitates airway insertion and management while reducing the risk of coughing and patient discomfort. Clinical research further suggests that the awake LMA insertion

technique may be used as an alternative to awake fiberoptic endotracheal intubation for management of the difficult airway.[9,10]

With the patient in the RLD position, the airway was considered difficult, and therefore the awake LMA insertion technique was determined to be a safe method of securing the patient's airway for the second asleep segment. If the LMA could not be inserted or properly seated into the oropharynx while the patient was in the RLD position, it would have been possible to convert to an awake fiberoptic endotracheal intubation technique to secure the airway for re-induction.

The clinical research recommends a low dose remifentanil infusion for pain not otherwise controlled, because it may be quickly titrated to the desired level of analgesia.[1] Furthermore, concomitant propofol and remifentanil infusions may provide greater hemodynamic stability during the asleep segments than propofol and desflurane.[1,3,11] As table 1 illustrates, remifentanil also facilitates awake insertion of the LMA.[8,10] However, remifentanil was not chosen for the patient, since the local anesthetic provided excellent analgesia, no hemodynamic instability occurred throughout the procedure, and unnecessarily adding remifentanil has the disadvantage of causing respiratory depression and post-operative hyperalgesia.[3,4]

It is imperative for the anesthesia practitioner to individualize approaches for patient care during an asleep-awake-asleep craniotomy. Prior to surgery, the anesthetist must counsel the patient about what will occur during the awake segment. Although the patient in this case tolerated the awake segment, the anesthesia practitioner should consider several pharmacologic interventions to avert patient restlessness and nausea and to facilitate patient compliance. Positional and surgical discomfort may be reduced with diclofenac sodium or an opioid infusion.[5] Restlessness may be lessened with administration of haloperidol to decrease anxiety, though it was not needed for this patient, because he was extremely cooperative and mentally prepared for his surgery.[5] Dexamethasone was administered to the patient in order to reduce cerebral swelling and to prevent nausea.[12] A prophylactic dose of ondansetron prior to the awake segment of this case would have further reduced the risk of nausea associated with anesthesia, so it should have been considered for the patient.[5]

Anesthetists should strongly consider use of an LMA for asleep-awake-asleep craniotomies because it decreases the chance of intraoperative complications by preventing airway obstruction, minimizing the chance of coughing, and facilitating ventilatory support if the patient experiences respiratory arrest. Furthermore, it is easily inserted and well-tolerated by patients. For these reasons, the LMA has grown in popularity for awake craniotomy procedures, and more frequent use of the awake insertion technique described in this case study may improve anesthetic management of patients undergoing an asleep-awake-asleep craniotomy.

Table 1. LMA Placement in Awake Patient Populations

Article	Succesful 1st Attempts/ Total Patients	Patient Population	Oropharyngeal Local Anesthetic	Adjunct Intravenous Anesthetic	LMA Device
Keller, et al. 2001[6]	10/11	ASA 1	10 puffs of lidocaine spray 1%	None	LMA Ctrach
Bilgin, et al. 2006[7]	3/3	Unstable cervical spine	Lidocaine spray 10%, lidocaine jelly 2% on LMA	None	LMA CTrach
Lee, et al. 2006[8]	10/10	ASA 1	4 mL nebulised lidocaine 4%, 12 sprays of lidocaine 10%	Remfentanil 2 ng/mL effect-site concentration	Standard LMA
Wender, et al. 2007[9]	3/3	Morbidly obese	8-10 mL Topical lidocaine 4%	Midazolam 2 mg	LMA Ctrach
Lopez, et al. 2009[10]	20/21	Difficult airway	20 mL lidocaine 1% gargle, lidocaine spray 10%	Midazolam 1-2 mg, remifentanil 0.1 mcg/kg/min	Standard LMA

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Perioperative Care for a Patient with Mitochondrial Disease

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Keywords: mitochondrial disease, anesthesia, perioperative care.

Mitochondrial disease is defined as a heterogeneous multisystem group of metabolic disorders that result from a defect in the mitochondria.[1] These diseases frequently target the electron transport system of the mitochondrial respiratory chain which provides cellular energy through the oxidative phosphorylation process.[2] The abnormal mitochondria are caused by mutations in the mitochondrial deoxyribonucleic acid (DNA), nuclear DNA, or mitochondrial ribonucleic acid.[2,3] Mitochondrial disease may also be due to deficiencies in substrate transport or usage, such as abnormalities in the pyruvate dehydrogenase complex, fatty acid transport and utilization, or the Krebs cycle.[3]

Case Report

A 16-year-old girl who weighed 31 kg, and stood 56 inches tall, presented for dental restoration and extraction. Her medical history review showed mitochondrial disease, cerebral palsy, gastroparesis, and developmental delay. The patient had no known drug, food, or latex allergies. Current medications included daily multivitamins, nutritional supplements, and metoclopramide as needed. She had no anesthetic complications from previous surgeries. No significant abnormal laboratory values were found. Preoperative evaluation revealed a Mallampati III classification, thyromental distance of 5 cm, and poor neck muscle control.

In the preoperative holding area, the patient was premedicated with 15 mg of midazolam via gastrostomy tube. Lidocaine 4% topical anesthetic cream was applied to the skin prior to intravenous (IV) catheter placement. A 22-gauge peripheral IV catheter was placed and a continuous maintenance infusion of 10% dextrose in 0.45% normal saline (D_{10} 0.45%NS) was started at 70 ml/hr. Blood glucose by finger stick was 76 mg/dL. The patient was then transported to the operating room and standard monitors applied.

Induction of general anesthesia was accomplished by mask with 6% sevoflurane inhalation in a mixture of 6 liters nitrous oxide and 4 liters oxygen. Following the induction, fentanyl 15 mcg, vecuronium 3 mg, and dexamethasone 8 mg were administered IV followed by three minutes of mask ventilation. To prepare for nasal intubation, two sprays of topical phenylephrine 0.25% were administered into each nostril. During mask ventilation, the patient's left nostril was dilated using #24 through #28 French soft nasal trumpets. A 6 mm cuffed nasotracheal tube was used for tracheal intubation. Correct placement of the endotracheal tube was verified with positive end-tidal carbon dioxide and auscultation of bilaterally equal breath sounds. Anesthesia was maintained with sevoflurane in a mixture of air and oxygen.

Total surgical time was approximately 90 minutes with an estimated blood loss of 15 ml. The patient received a total of 200 ml of D_{10} 0.45% NS and 250 ml of normal saline. Ondansetron 4 mg IV was administered 30 minutes prior to the end of surgery.

Neuromuscular blockade was antagonized with neostigmine 2 mg IV, and glycopyrrolate 0.4 mg IV was administered to decrease the potential side effects of neostigmine. Once the patient was awake and demonstrated adequate spontaneous ventilation, the nasotracheal tube was removed.

In the post-anesthesia care unit the patient's blood glucose was 116 mg/dL, and the dextrose infusion was continued for one hour. She was given 120 ml of her tube feeding through her gastrostomy tube without any evidence of nausea or vomiting. After three hours in the recovery room the patient was discharged home.

Discussion

Mitochondria are rod-shape organelles that provide energy needed for cellular function. The most important roles for mitochondria are the production of adenosine-triphosphate (ATP) through the oxidative phosphorylation process, and the regulation of cellular metabolism.[3] Disorders of mitochondrial function are thought to be a result of deficiencies in ATP synthesis and electron transport thereby causing a variety of defects in mitochondrial energy metabolism. Symptoms typically begin early in life and appear in those organs with the highest energy demand (e.g. central nervous system, retina, liver, and cardiac and skeletal muscles). It is also important to note that there are variations in some forms of the mitochondrial disorders where overt clinical presentation can be delayed until late childhood or into young and middle adulthood. [3]

Common signs and symptoms of decreased mitochondrial energy, that are a concern for the anesthesia practitioner, include seizures, altered level of consciousness, central apnea, reactive airway disease, cardiac conduction disturbances, hypertrophic or dilated cardiomyopathy, dysphagia, reduced lower esophageal sphincter tone, skeletal muscle weakness, liver function impairment, and hypoglycemia.[1,3] Individuals with mitochondrial abnormalities are extremely susceptible to tissue ischemia and further mitochondrial damage during times of stress, infection, surgery, and anesthesia.[3]

Patients with mitochondrial disease present with unique anesthetic challenges. These patients are at a greater risk for developing metabolic acidosis and cardiac and pulmonary complications.[3] Metabolism during the fasting period shifts toward fat utilization as an energy source. The ability to metabolize fat for a patient with mitochondrial disease may be limited by the impairment of beta oxidation of fats.[4] The administration of lactate free IV fluids for fluid maintenance is highly recommended and should be commenced during the preoperative fasting period to prevent hypoglycemia and increased plasma lactate levels.[5] Thus, to avoid a metabolic crisis, fasting should be kept at a minimum, surgery should be scheduled first thing in the morning, and providing a dextrose infusion is imperative for this patient population. This patient's surgery was scheduled at 0730 in the morning, she was NPO from her tube feedings for four hours and clear liquids for two hours, and a dextrose infusion started immediately upon arrival to the preoperative holding area.

Particular attention should be given to avoid administering drugs that are known to interfere with mitochondrial function. Propofol impairs mitochondrial function by uncoupling oxidative phosphorylation, inhibiting the electron flow along the electron transport chain, antagonizing beta receptor activity, and directly acting on

calcium channel proteins diminishing contractility.[5] The development of metabolic acidosis and prolonged anesthesia recovery time has been associated with the use of propofol.[6] Additionally, the lipid formulation may increase the risk of toxicity due to impaired fatty acid oxidation.[6] Other induction agents such as ketamine, etomidate and thiopental have not shown any adverse events.[4] It was decided that, for this case, an inhalation induction would be more beneficial for this patient over the use of an IV agent. Overall, the use of propofol should be limited to airway emergencies or other life threatening emergency situations in this patient population.

Inhalation anesthetic agents (isoflurane, sevoflurane, halothane) affect mitochondrial function by inhibiting complex I activity of the electron transport chain, thereby altering oxidative phosphorylation.[4,7] Exposure to isoflurane and halothane have shown to reduce mitochondrial function, such as oxygen consumption, by 20% and sevoflurane by 10% at concentrations equal to twice minimum alveolar concentration (MAC).[7] In addition, halothane further depresses mitochondria with the addition of nitrous oxide.[7] Sevoflurane was chosen for this case because it has the least decrease in mitochondrial function. During the anesthetic induction sevoflurane was increased to more than twice the MAC for approximately two minutes then reduced down for a maintenance rate of 1.5% to 1.8% end tidal concentration with no adverse effects. In the literature reviewed desflurane was not mentioned.

Patients who display signs of myopathy may have an exaggerated sensitivity to neuromuscular blockade. The residual effects of nondepolarizing muscle relaxants could further exacerbate their intrinsic muscle weakness and increase the risk of postoperative ventilatory failure.[7] Therefore, it is advised to reduce the induction dose of muscle relaxants and use careful titration of subsequent doses. The preoperative evaluation of this patient revealed poor neck muscle control; no adverse events occurred with the full dose of vecuronium, but future consideration of a reduction in the dose of vecuronium would be prudent. Susceptibility to malignant hyperthermia is also a consideration as with any patients with more familiar muscular dystrophies.[7] The use of succinylcholine should be reserved for airway emergencies.

Anesthetic agents such as benzodiazepines hinder adenosine nucleotide translocase, a component of mitochondrial permeability, and barbiturates will inhibit the electron transport chain and uncouple oxidative phosphorylation.[4] Both classes of pharmacologic drugs have not shown any adverse events in this patient population. Additionally, there have been no reports on the contraindications of opioid use.[4] The patient received midazolam and fentanyl with no anesthetic complications.

Mitochondrial disorders lead to the impaired production and conversion of ATP and regulation of cellular metabolism thereby resulting in the progression of multiple organ dysfunction. Patients with mitochondrial disorders are at an increased risk of metabolic decompensation. Preoperative fasting can be hazardous and lead to elevated lactate levels which is further exacerbated by stress, therefore vigilance to fluid management is essential. During the perioperative period particular attention should focus on avoiding anesthetic agents that are known to alter mitochondrial function. The literature review has demonstrated that anesthesia can

be carried out safely with an informed anesthesia professional and a proper preoperative assessment and evaluation.

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Anesthetic management of a patient with Progeria

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Keywords: Progeria, Hutchinson-Gilford Progeria syndrome, fiberoptic intubation, difficult airway, TMJ

Hutchinson-Gilford Progeria Syndrome (progeria) is a genetic disorder leading to premature aging; it occurs in 1:4,000,000 live births and there are only 78 known people living with this disorder in the world.[1,2] The average life expectancy of children with progeria is 13 years, and they usually succumb to normal difficulties associated with aging such as cardiac or neurologic events.[2] The syndrome leads to a variety of physical changes including cardiac disease, cerebrovascular disease, skeletal degeneration, and craniofacial changes, all of which can present difficulties for anesthesia professionals.[2,3]

Case Report

A 22 year-old female presented for bilateral temporomandibular joint prosthetic implants. She was 147 cm tall and weighed 38 kg. Her surgical history included bilateral buccal pad placement with sliding genioplasty 3 weeks prior. Her medical history included progeria, smoking socially for 5 years, asymptomatic heart murmur, gastroesophageal reflux, osteolytic disease, and arthritis. She was taking acetaminophen as needed and had allergies to penicillin and tape.

Physical exam of the patient showed small stature, small nose, mandibular and maxillary hypoplasia, alopecia, and prominent stiff joints. The patient had a heart rate of 76 with regular rate and rhythm and 1/6 systolic ejection murmur noted. Electrocardiogram showed left atrial enlargement, anterior ischemia, and T-wave abnormality. Echocardiogram was not done. The patient denied any history of myocardial infarction, chest pain, or hypertension, and the patient reported good exercise tolerance. Bilateral breath sounds were clear to auscultation and the patient denied respiratory difficulties. Chest x-ray results were unavailable. Airway exam showed thyromental distance < 6 cm, neck full range of motion, and Mallampatti could not be assessed due to a wired jaw.

In preparation for surgery, the patient received dexamethasone, oxymetazoline nasal spray to nares, and topical 2% lidocaine jelly. A nasal airway was inserted into left nare. The patient was given midazolam 0.5 mg and ketamine 30 mg intravenously. Monitors were placed and oxygen 10 L/min was administered via face mask for 7 min. The surgeon and tracheostomy tray were immediately available prior to induction since the patient's jaw was to remain wired throughout induction. A laryngotracheal topical anesthesia kit was used to administer 4% lidocaine 3mL to supraglottis and glottis prior to intubation. Spontaneous respirations were maintained due to known difficult airway, previous awake fiberoptic intubation, and wired jaw. A cuffed #6 nasal ETT was placed using a pediatric fiberoptic bronchoscope. The ETT was successfully placed on the second attempt while the patient maintained spontaneous respirations. The carina was visualized with the fiberoptic bronchoscope, chest rise and fall noted, bilateral breath sounds were auscultated, and end tidal CO_2 was verified upon placement of endotracheal tube and after it was sutured by surgeon.

Anesthesia was maintained with isoflurane 1%, oxygen 2 L/min, fentanyl 25 mcg IV and ketamine 20 mg IV as needed. No neuromuscular blockade was used. Upon completion of surgery, isoflurane was discontinued, and the patient was transferred intubated and sedated to the intensive care unit. Respirations were controlled and oxygen was administered using an ambu bag via the ETT during transfer. Surgery was uncomplicated.

Discussion

A thorough airway examination is an essential part of any preoperative assessment, especially for a patient with a history of awake fiberoptic intubation. Patients with progeria are known to have difficult airways due to the craniofacial manifestations of the syndrome including mandibular hypoplasia, micrognathia, and small mouth openings with decreased TMJ mobility.[2,4,6] In this case, direct laryngoscopy was not an option due to a wired jaw, so an awake nasal fiberoptic intubation was performed allowing for spontaneous respirations throughout induction. A surgeon was present for induction in the event that an emergency tracheostomy became necessary. Direct laryngoscopy has been successful in some patients with Progeria, however, most cases are done utilizing awake or asleep fiberoptic intubation depending on the patient's airway and situation.[2,4-6] According to the literature, the asleep fiberoptic intubations are done due to younger age with less severe deformities or lack of patient cooperation for awake intubation.[4,6] Small nares and an anterior glottis can lead to difficulty in nasal

intubation, although in this case it was done with minimal difficulty. [2]

There are many ways to anesthetize the airway for awake intubations, including topical application, atomization, and nebulization of local anesthetic or by specific nerve blocks, however, no literature was found with the use of regional anesthetic techniques and progeria.[7,8] In this case, 2% lidocaine jelly was applied to anesthetize nasal mucosa and 4% lidocaine was directly applied to supraglottis and glottis prior to intubation. Nebulized lidocaine was not used, but may have been effective to additionally anesthetize the airway and having the patient breathe through her nose. Topical techniques were chosen for this case because of the ease of administration and patient safety with minimal risks. Adequate respiratory effort must be demonstrated prior to extubation. The literature shows patients with Progeria can be extubated without incident, however this patient remained intubated post-operatively because of concern for edema from the surgical procedure.[4,6]

Cardiac disease is a major cause of death in progeria and can be manifested as atherosclerotic plaque, myocardial ischemia, hypertension, and myocardial infarction.[1,6,9] Aspirin prophylaxis is a common therapy in these patients; this patient discontinued aspirin therapy 7 days before surgery due to the risk of bleeding.[2] In this case, the patient denied any significant cardiac history, was asymptomatic, and had good exercise tolerance, thus a cardiac echo and stress test were not performed. A full cardiac workup prior to surgery is reported in some cases, and in others it is not.[4,5,6] Despite the patient being asymptomatic, a cardiac echo and stress test prior to surgery would have been beneficial to have a better evaluation of the patient's cardiac function and extent of cardiovascular disease. Adverse cardiac events are precipitated by hypovolemia and hypotensive episodes.[2] The patient remained normotensive throughout surgery and there were no arrhythmias or ischemic changes on the EKG perioperatively or postoperatively. The atherosclerotic disease can also affect the cerebral vasculature leading to transient ischemic attacks or strokes, which are another leading cause of death with Progeria.[2,4,6,9] In this case, the patient did not report having any neurologic symptoms. Recent literature does not report adverse neurologic outcomes postoperatively, but it remains an important risk factor and prevention of such an event is essential in anesthetic management.[4,6]

Some of the musculoskeletal changes seen with progeria include osteolytic disease, arthritis, and contractures.[2,3,5,6] Arthritis and osteolytic disease lead to degeneration of joints and bones, including the temporomandibular joint.[2] With this patient, prosthetic joint implants were placed to facilitate joint mobility. Arthritis leads to prominent and stiff joints in patients with Progeria, therefore positioning is an important anesthetic consideration.[2,5,6] In this case, extra padded support was placed under contracted extremities to avoid damage to joints.

Research on progeria and anesthesia is limited due to its infrequency. Anesthesia professionals must focus on the physical status of each individual patient and known complications of progeria. There are many important anesthetic considerations with this rare genetic condition that must be taken into account in order to appropriately care for the patient.

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Emergence Delirium in the Post-Operative Adult

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Keywords: delirium**,** disorientation, combative, emergence, postoperative

Patients who wake up after anesthesia agitated, uncooperative, and combative pose a risk of harm to themselves and caregivers. The goal of this article is to raise awareness of emergence delirium by describing what it is, who it affects, how it affects patients, potential causes, and possibilities for its prevention.

Case Report

A 57 year old, 120 kg, African American male presented for a cadaveric kidney transplant. He had a past medical history significant for end stage renal disease requiring hemodialysis for the past 7 years, insulin dependent diabetes mellitus for 15

years, hypertension, nonobstructive coronary artery disease, latent hepatitis C, peripheral vascular disease, secondary hyperparathyroidism, anemia, and prostate cancer. He had a remote history (15 years prior) of consuming 1 gallon of liquor and 6 cans of beer per week, and smoking 1.5 packs per day for 15 years. He had multiple past surgeries with no anesthesia complications noted.

In the holding room, the patient's vital signs were within normal limits except for his blood pressure which was elevated at 150/80. His oxygen saturation was 97% on room air and his blood glucose was 138mg/dl. Midazolam 1 mg IV was administered prior to moving the patient to the operating room. In the OR, a smooth IV induction was performed with lidocaine 80mg, propofol 300mg, and succinylcholine 140mg.

A radial arterial line was placed and a central line was inserted with the patient in Trendelenburg position. A bispectral index monitor was placed on the patient and the value was maintained below 60 throughout the case. Anesthesia was maintained with isoflurane with intermittent boluses of fentanyl and hydromorphone throughout the procedure as needed, totaling 500mcg of fentanyl and 1.5mg of hydromorphone. Relaxation was maintained with cisatracurium throughout the case. Neuromuscular blockade was reversed when the train of four was 4/4 with 5mg of neostigmine and 0.6mg of glycopyrrolate. Following reversal, the patient could sustain a head lift for 5 seconds and a nasal trumpet was inserted before extubation to prevent airway obstruction. The endotracheal tube was removed, and oxygen was administered at 100% FiO_2.

The patient was reassured of his surroundings, and an explanation was given of the time, place, and events. The patient was combative, agitated, yelling, and was unable to follow commands. The patient's oxygen saturation was 100%. Three people were needed to hold the patient's arms to prevent harm or discontinuation of his central and arterial lines. The patient remained combative, would not follow commands, and it became impossible to safely move him from OR table to the gurney. Ketamine 40mg IV was administered and he was assisted to the bed by the OR staff. An additional dose of ketamine 20 mg was administered for transport to the intensive care unit (ICU). The patient maintained a patent airway, was not agitated or combative, and had an oxygen saturation of 100%. Upon arrival to the ICU, the patient was cooperative and his vital signs were stable. On follow-up, the patient was alert and oriented and denied any anesthetic complications.

Discussion

Emergence delirium is an occurrence of disorientation, combativeness, and/or uncooperativeness after undergoing anesthesia. Emergence delirium, also known as emergence agitation, is difficult to quantify because no common tool exists to rate this phenomenon. Research indicates that approximately three to five percent of adults undergoing general anesthesia experience emergence delirium.[1] The incidence occurs more frequently with pediatric patients, ranging from ten to eighty percent.[2,3]

The possible dangers associated with emergence delirium include patient injury such as self-extubation, removal of catheters, and/or hemorrhaging. Another possible risk is harm to caregivers.[1] Patients experiencing emergence delirium may

require physical and/or chemical restraints. Physical restraint may require multiple personnel to control the patient. Such complications could also lead to disruption of sutures or unintended extubation, leading to airway compromise.[1] Emergence delirium can also prolong a patient's stay in the post anesthesia care unit and lead to unplanned admissions.

Several studies have been conducted in an attempt to discover the etiology of emergence delirium. Lepousé's study of 1,359 patients found a greater frequency of agitation among patients following abdominal and breast surgery (42% and 7%, respectively). The duration of surgery was longer in agitated patients, 108 minutes versus 72 minutes in non-agitated patients. Patients who received preoperative benzodiazepine administration showed a two-fold increase of emergence delirium. Other possible etiologies found in this study included the existence of a tracheal tube and pain. No difference was found with different inhalational anesthetics; however, there was found to be less emergence delirium in patients anesthetized with propofol than with inhalational agents.[1]

Yasui et. al. tested the effects of volatile anesthetics on rat brains to search for a possible etiology of emergence delirium. It was proposed that the inhalational agents, especially sevoflurane, caused direct excitation of neurons in the locus coeruleus.[4] The locus coeruleus is a source nucleus that sends noradrenergic signals in the central nervous system and is also a main target of alpha-2 agonists.[4] The locus coeruleus plays an important role in controlling the overall activity level of the brain. The study of the rat brains showed that dexmedetomidine, an alpha-2 agonist, had sedating effects in the locus coeruleus which prevented emergence delirium by hyperpolarizing neurons and preventing their response to sevoflurane.[4]

Other studies indicate that alpha-2 agonist administration after induction of anesthesia decreased the occurrence of emergence delirium. Administering dexmedetomidine after induction reduced the occurrence from 47.6% to 4.8% and clonidine, another alpha-2 agonist, resulted in a decline from 39% to 0%.[5]

The etiology of emergence delirium is still unclear but pain appears to be a promoting factor since administration of analgesic agents reduces its incidence. A variety of medications have been used to prevent or treat emergence delirium, including propofol, opioids, alpha 2–agonists, ketamine and other sedatives. One study showed that the IV administration of ketamine or nalbuphine just before discontinuing anesthesia did not delay awakening or postanesthesia care discharge times but did decrease episodes of emergence delirium.[3]

Propofol has been studied for the prevention of emergence delirium in pediatric patients. The use of a 2mg/kg bolus at the beginning of a procedure did not reduce the occurrence of emergence delirium, probably due to propofol's short duration of action.[2] However, a dose of 1mg/kg at the end of an anesthetic was shown to significantly decrease the occurrence of emergence delirium.[2]

There are multiple treatment options and an array of pharmacologic agents to prevent emergence delirium in patients undergoing anesthesia. The research finding that sevoflurane does cause direct excitation of the locus coeruleus and that the alpha-2 agonists prevent depolarization at the same site. This finding makes the argument that drugs working as agonists at the alpha-2

receptors are the proposed answer to successful prevention of delirium.

Lepousé's study was examined to further research why abdominal and breast surgeries had an increase in incidence of emergence agitation. Some possible explanations from a pathophysiological viewpoint is that upper abdominal and thoracic surgeries decrease a patient's functional residual capacity (FRC).[6] Patients are usually lying supine for breast and abdominal surgeries which further decreases their FRC.[6] This makes it difficult for patients to take a full vital capacity breath and if breathing seems difficult to a patient, it could lead them to become anxious and potentially cause agitation, confusion, and delirium.

Considering the possible causes of emergence delirium, some risk factors are evident in this case. This patient was having a kidney transplant which may lead to increased stress and anxiety beyond that associated with more routine surgery. Preoperative anxiety is a known risk factor in that 12% of anxious patients became agitated postoperatively.[1] The patient was undergoing abdominal surgery and he was placed supine, both factors decreasing the patient's FRC. He also had a prolonged duration of general anesthesia with an inhalational agent greater than 360 minutes, which was much greater than the earlier mentioned risk factor of 108 minutes. Because of his large abdominal incision, the patient could have required a larger dose of opioids than was received or could have benefited from an alpha 2-agonist. His postoperative agitation could have been caused by pain. All of these factors can be taken into account when planning on how to prevent emergence delirium and in discerning an individual patient's risk for developing emergence delirium.

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Anesthetic Considerations for Malignant Hyperthermia

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Keywords: malignant hyperthermia, end-tidal CO2, laparoscopic surgery

Malignant hyperthermia (MH) is a serious autosomally dominant inherited disorder of skeletal muscles in humans.[1] An inherited mutation in the gene for the ryanodine receptor 1 (skeletal) account for the vast majority of MH cases.[1,2] This complex syndrome is triggered by volatile inhalational anesthetics, and the depolarizing muscle relaxant succinylcholine. These agents cause an increased release of intracellular calcium from the sacroplasmic reticulum in muscles of susceptible individuals.[1,2] A MH crisis is characterized as a hypermetabolic state, resulting in muscle rigidity, hypercarbia, hyperthermia, tachycardia and eventually rhabdomyolysis. The early clinical manifestations associated with an MH attack, increased CO_2 production and tachycardia, are often observed in patients undergoing routine laparoscopic surgery.[3] Consequently, early detection with prompt management is crucial to preventing this potentially life-threatening syndrome.

Case Report

A 55 year-old, 183 cm, 93 kg male presented for a laparoscopic cholecystectomy, secondary to cholecystitis. The patient reported no past medical history, other than cholecystitis. The patient was not on any medications. The patient reported no previous surgeries. The patient noted that his father had a history of malignant hyperthermia, and had tested positive for MH susceptibility.

Measures were taken to remove all MH-triggering medications. The succinylcholine was removed from the anesthesia chart. The anesthesia machine was prepared with placement of a new breathing circuit, and the soda lime canisters were changed. The vaporizers were removed and the Datex-Ohmeda Aestiva anesthesia machine (GE Healthcare, Madison, WI) was flushed with oxygen at 10 L/min for one hour. The malignant hyperthermia cart was also placed in the room and opened.

Preoperative anxiolysis was achieved with midazolam 2 mg IV. Standard monitors were applied. Oxygen was administered by a facemask. General anesthesia was induced with fentanyl 100 mcg IV and propofol 100 mg IV. Adequate ventilation was verified by the Puritan-Bennet end-tidal CO_2 monitoring system. Once the ability to ventilate was established, rocuronium 50 mg IV and propofol 100 mg IV was administered. Direct laryngoscopy with a Macintosh 4 blade revealed visualization of the vocal cords. The trachea was intubated with an 8.0 mm endotracheal. Placement was confirmed with bilateral breath sounds, and positive end tidal CO_2. The ventilator was set to pressure control to maintain a respiratory rate of 10 breaths/min and a tidal volume of 600 ml/min.

Maintenance of anesthesia was established with a continuous infusion of propofol 150 mcg/kg/min. After the abdomen was insufflated with CO_2, the end tidal CO_2 began to increase from the baseline number of 33 mmHg to 43-47 mmHg. The patient's heart rate increased from 88 bpm to 103 bpm. The blood pressure remained relatively

constant with the first reading of 128/63 mmHg. Fentanyl 50 mcg IV was administered and the respiratory rate was increased to 12 breaths/min. The heart rate remained in the low 100's with no further elevation of the end tidal CO2 after a repeated dose of fentanyl 50 mcg IV, and the change in the ventilator setting. The propofol infusion was weaned in increments of 50 mcg/kg/min to off during the emergence period. The patient was extubated without difficulty and transported to PACU.

Discussion

Malignant hyperthermia (MH) is an inherited skeletal muscle disorder that is characterized as a hypermetabolic state that manifests in susceptible patients when exposed to triggering agents. Triggering agents are known to include all of the volatile anesthetic gases, as well as the depolarizing muscle relaxant, succinylcholine.[1,2] In this case, the patient was aware of the risk of MH susceptibility. The patient's father suffered from an episode of MH, and later tested positive for the in vitro contracture test. This is a test in which a muscle fiber contracts when it is exposed to halothane or caffeine. [3] This test possesses a high degree of sensitivity, however; it can be very expensive and due to the varying genetic mutations of MH, this test raises questions related to reliability.[2] Less invasive ways of testing for MH are being trialed, such as *in vivo* micodialysis of skeletal muscle and the testing of blood lymphocytes.[5,6]

Many patients do not have prior knowledge of their propensity for this genetic abnormality. Typically, cases of malignant hyperthermia arise after the induction of general anesthesia, with one or more of the triggering agents. Increased intracellular calcium release causes augmented metabolic stimulation. This results in a rapid increase in the production of CO_2 leading to increased end-tidal CO_2, tachycardia, and lactic acidosis. There are several other differential diagnoses that may result in these symptoms, resulting in a late diagnosis of MH. The anesthesia practitioner may consider ventilation problems, equipment malfunction, drug toxicity or abuse, infection, sepsis, thyroid storm, or iatrogenic causes such as carbon dioxide insufflation during laparoscopy.[6] Later signs of a MH crisis include muscle rigidity, hyperkalemia, and hyperthermia, and may be a lethal combination in a patient with undiscovered MH. Dantrolene blocks the continuous release of calcium from the sarcoplasmic reticulum ablating the ongoing hypermetabolism.[7] Early recognition of an MH crisis is paramount, so that the triggering agents can be discontinued, and Dantrolene quickly initiated. When given in a timely manner dantrolene can be life saving, but it does not come without disadvantages. Dantrolene can cause difficulty in weaning from a ventilator, as it can cause serious respiratory muscle weakness. Moreover, the traditional form of dantrolene is cumbersome and time consuming to draw up and administer, therefore the newer forms that offer rapid reconstitution are advantageous.[5]

When considering the differential diagnoses, detection of MH may be even further delayed during laparoscopic surgery. Common physiological changes that may be associated with laparoscopic surgery are similar to those that are seen with MH. Laparoscopic surgeries have become routine procedures that are thought to be more beneficial to patients, as they allow for less invasive interventional methods. These minimally invasive surgeries produce significantly less trauma while allowing for

a quicker recovery time. However, in order to create adequate surgical visualization, the abdominal cavity is insufflated with CO_2. This produces a pneumoperitoneum, and may lead to the absorption of CO_2 systemically across the peritoneum.[8]

This systemic absorption of CO_2 can quickly lead to an increased end-tidal CO_2, tachycardia, and acidemia. As these are also the hallmark signs seen with the early onset of MH, it becomes clear how early detection of MH can be masked. Another compounding factor that could lead to misdiagnosis is associated around the use of high insufflation pressures. This leads to an increase in intra- abdominal pressure, which further exacerbates the tachycardia, and increased systemic vascular resistance.[9] When the pneumoperitoneum is established with room temperature carbon dioxide, there can be a marked decrease in core body temperature, as well as, urine output.[10] Hyperthermia is a late but critical sign of MH and can be overshadowed by this phenomenon.

Given this patient's familial history of MH susceptibility, a total intravenous anesthetic was used, avoiding any triggering agents. After the creation of a pneumoperitoneum, despite all the preventative efforts, the patient experienced tachycardia and an increase in end-tidal CO_2. It is imperative at this point for the anesthesia professional to rule out a MH diagnosis. In order to do this the astute clinician must: increase minute ventilation, check core body temperature, assess fluid status, ensure proper hydration, and ascertain adequate anesthetic levels. In this case, the administration of fentanyl IV for tachycardia and an increase in the respiratory rate resulted in maintaining hemodynamic stability with no further rise in the end-tidal CO_2. Interventions in one or more of these modalities should correct the physiological presentation. Failure to return to homeostasis indicates to the practitioner that MH should be highly suspected and the algorithm for MH needs to be employed.

This patient had an uneventful surgical experience without any anesthetic sequela. Anesthesia practitioners always need to be aware of the potential for MH in any patient, but particularly for those with a familial history undergoing laparoscopic surgery. It is clear through the data presented here that the physiological changes, which accompany the surgical procedure, can mask the signs and symptoms of malignant hyperthermia. The establishment of a differential diagnosis is of paramount importance as malignant hyperthermia has a mortality rate of 70% when dantrolene is not promptly administered.[1]

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Management of Medulloblastoma in a Remote Location

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Keywords: primitive neuroectodermal tumor, medulloblastoma, propofol, deep sedation, prone position.

Medulloblastoma is a primitive neuroectodermal tumor (PNET) of the central nervous system (CNS) that occurs mainly in the cerebellum.[1] Medulloblastoma accounts for 14-20% of all pediatric CNS tumors.[2] Medulloblastoma occurs mostly in males, less than 10 years old, with a peak incidence at 5 years of age.[3] In the United States, there are nearly 400 new diagnosed cases of medulloblastoma each year, and only 1% to 2% of medulloblastoma cases have metastatic spread at initial presentation.[3] This case report examines a pediatric patient with PNET of the spine probably resulting from a medulloblastoma undergoing two procedures requiring anesthesia outside of the operating room.

Case Report

A 6 year-old, 111cm, 19kg male presented for insertion of a double-lumen, indwelling central line catheter and computer topography (CT), laser guided, face mask creation. Three weeks prior, the patient had acute back pain, and became unable to walk. His MRI revealed a large thoracic spinal mass. The patient then underwent a thoracic 6-12 laminectomy with a 90% resection of the thoracic spinal tumor. Pathology diagnosed the tumor as a primitive neuroectodermal tumor (PNET).

The patient was born full term without complications. He had no significant past medical history. His medications included ciprofloxacin, acetaminophen, dexamethasone, oxycodone, docusate, polyethylene glycol, and senna. Current lab values revealed hemoglobin 11.8g/dl,

hematocrit 34.8%, WBC 8.7K/µl, and platelets 664K/µl. The physical exam revealed the presence of a left hemiparesis.

On the morning of surgery, the patient's vital signs were oral temperature 36.8□ C, blood pressure 108/69, heart rate 90bpm, respiratory rate 18, and SpO_2 100% on room air. A 22g peripheral intravenous (IV) catheter was inserted in the left hand. Midazolam 1 mg, glycopyrolate 0.1 mg, and cefazolin 560 mg were administered. The patient was brought into the interventional radiology suite for placement of a double-lumen, Hickman catheter (Bard Access Systems, Inc., Salt Lake City, UT) under fluoroscopy, and standard monitors were applied. The patient was pre-oxygenated, and IV induction was initiated with fentanyl 20 mcg, lidocaine 20 mg, and propofol 100 mg. Upon loss of eyelid reflex, a laryngeal mask airway (LMA; LMA North America, San Diego, CA) size 2 was inserted without difficulty. Anesthesia was maintained with sevoflurane 2.6% expired and O_2 2 L/min. At the end of the procedure, the patient's vital signs were stable, and he was breathing spontaneously. The LMA was removed "deep" without complications, and the patient's oropharynx was suctioned. An oral airway was inserted, and the patient continued to breathe spontaneously with a 200 ml tidal volume. He was placed on oxygen 8 L/min via simple face mask. A propofol intravenous infusion was initiated at 150 mcg/kg/min via a syringe infusion pump, and the patient was transported to the CT simulation room for face mask creation with a portable monitor and oxygen.

Upon arrival in the CT simulation room, the oral airway and simple face mask were removed. A portable monitor with blood pressure, SpO_2, heart rate, and EKG remained on the patient throughout the case. The patient was placed in the supine and prone positions, and he remained immobilized with spontaneous breathing of room air throughout the procedure. Upon completion of the procedure, the propofol infusion was discontinued, and the patient was transported to the post anesthesia care unit (PACU). Upon arrival in the PACU, his vital signs were stable, and he did not have any complications.

Discussion

Medulloblastoma typically arises from the cerebellar vermis, and usually grows into the fourth ventricle obstructing the cerebral spinal fluid (CSF) flow.[3] Obstruction in the CSF results in signs of increased intracranial pressure (ICP) and hydrocephalus.[3] Signs of increased ICP include irritability, headache, nausea, early morning vomiting, lethargy, seizures and papilledema.[3,4] Ataxia and difficulty handling objects may also be presenting symptoms of medulloblastoma because of cerebellar involvement.[3] Medulloblastoma is known to spread through the CSF resulting in a primitive neuroectodermal tumor (PNET) of the spine, which can lead to spinal cord compression, vascular compromise, and paralysis of the limbs, bladder, and bowel functioning.[1-3,5] This metastatic spread of the medulloblastoma tumor to the spine is referred to as leptomeningeal dissemination, and occurs in 10% to 35% of all medulloblastoma cases.[3] Other presenting signs and symptoms of spinal tumors include severe weakness, acute pain, ataxia, and increased ICP from tumor compression of the spinal cord or nerve roots.[1-4]

Treatment of medulloblastoma, including PNET of the spine, is multimodal and involves surgical excision followed by radiation therapy and chemotherapy.[1,3,4] Following surgical resection of the tumor, insertion of an indwelling central line catheter is required to administer

chemotherapy agents, and a custom-made, plastic-matrix face mask is necessary to deliver precise CT laser guided radiation.[5,6] Insertion of an indwelling central line catheter, such as a double-lumen, Hickman is usually done under general anesthesia in the operating room setting or in the interventional radiology suite. The face mask is created by immobilizing the pediatric oncology patient with deep sedation, and molding the patient's face with a plastic-matrix to match the contour of the face. While making the face mold, the facial structures are scanned by CT in the prone and supine position to map the head and face for future craniospinal radiotherapy.

The anesthesia practitioner's role is to provide both general anesthesia and deep sedation during these procedures. The use of general anesthesia and deep sedation for diagnostic and therapeutic procedures on pediatric patients performed outside the operating room has increased substantially in recent years.[7] Procedures performed outside the operating room require the anesthesia professional to provide the same level of safety, anxiolysis, sedation, and analgesia, as procedures performed inside the operating room.[7]

At the end of the first procedure, the decision to remove the LMA with the patient still deeply anesthetized was based on the patient's condition. He had stable vital signs, regular spontaneous respirations, a tidal volume of 200 ml, and was easy to mask ventilate. The decision of whether to remove an LMA "deep" or "awake" in children is controversial.[8] The benefits of removing the LMA deep are decreased episodes of airway reactivity and laryngospasm on LMA removal, but there is a increased risk of aspiration and airway obstruction as the child emerges from anesthesia in the PACU.[8-10] Awake removal of the LMA benefits are that it allows for return of the patient's protective reflexes, but the risks of removing the LMA awake are increased episodes of vomiting, biting, excessive salivation, and airway reactivity.[8,10] According to Burgoyne *et al*., awake removal of the LMA resulted in an increased incidence of laryngospasm compared to removing it deep with an oral airway and face mask in place.[9]

During the face mask creation procedure it was required that the patient not have anything (face mask, nasal cannula, oral airway) altering his normal facial structure. A patent airway can be safely accomplished in a deeply, sedated pediatric patient without using an ETT or LMA in the prone or supine position.[6,11,12] During the face mask creation in the CT simulation room there are periods of time when the anesthesia professional cannot stay in the room with the patient. During these times, observation of the deeply sedated patient's, heart rate, blood pressure, and SpO_2 values was performed using a video camera. The emergency airway crash cart was readily available throughout the procedure.

Schreiber, Ribeiro Karpienski, and Strehl reported one brief episode of airway obstruction out of 155 cases performed on 11 pediatric oncology patients that received multiple radiation treatments under deep sedation in the prone position.[6] Airway obstruction during anesthesia is due to decreased muscle tone in pharynx and larynx causing upper airway narrowing, especially at the level of the epiglotitis.[8] If airway obstruction does occur, airway patency can be corrected by informing the radiology team, rotating the patient back to supine or lateral position, applying a chin lift, performing a jaw thrust maneuver, providing continuous positive airway

pressure (CPAP), and by suctioning the oropharynx secretions.[6,8]

Although this patient did not have any adverse events during the above procedures under anesthesia, the risk for complications such as, apnea, hypoxia, regurgitation, pulmonary aspiration, and laryngospasm was present. Anesthesia professionals must be able to recognize these potential complications to ensure prompt treatment of the pediatric medulloblastoma patient undergoing procedures outside the operating room.

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Total Intravenous Anesthesia for Pediatric MRI

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Keywords: Propofol anesthesia, propofol-based sedation, total intravenous anesthesia, magnetic resonance imaging anesthesia

Magnetic resonance imaging (MRI) is a noninvasive, painless and radiation-free diagnostic procedure. The use of MRI scans has increased tremendously due to improved diagnostic evaluations when compared to conventional x-rays or computerized tomography. Depending on the diagnostic need, an MRI scan lasts 30-60 minutes or more and requires a cooperative patient who can remain motionless for the entire procedure. This can be problematic in the pediatric population. Many pediatric procedures can be carried out under sedation; however, deeper anesthesia is required in most cases of failed sedation. While the number of MRI procedures continues to grow, there is and will be a greater demand for MRI anesthesia in this population.[1]

Case Summary

A 5-year-old ASA 2 female was scheduled for a follow up MRI of the abdomen and pelvis with intravenous (IV) contrast. The patient was 5 years post orthotopic liver transplant for fulminate hepatic failure. The patient experienced organ rejection a year after her transplant that had resolved. Medications included mycophenolate mofetil, nystatin, prednisone, trimethoprim/sulfamethoxazole, tacrolimus and valganciclovir. The patient's allergies included piperacillin/tazobactam. She was born full-term via cesarean section. She had no recent upper respiratory infection and last food and water intake was 8 hours prior to surgery. Airway assessment showed a Mallampati class I, a mouth opening of >3 cm and normal neck range of motion. The patient's lungs were clear to auscultation and preoperative vital signs (VS) were blood pressure (BP) of 100/66, heart rate (HR) 88 bpm, respiratory rate (RR) 24 breaths/min and SpO_2 100% on room air (RA).

In the preoperative holding area, a 22-gauge IV access was started in the left hand and was saline locked. The patient, accompanied by her parent, was escorted to the MRI suite. The patient was seated on the scanner while the parent remained with her. The pulse oximeter was applied and after confirming a RA SpO_2 reading and HR, induction ensued with lidocaine 10 mg and a titrated dose of propofol 70 mg IV. The patient was positioned in the MRI scanner, O_2 2 L/min was administered via nasal cannula, and standard monitors were applied. A propofol infusion was started at 250 mcg/kg/min. Continuous monitoring of the patient's status and VS were observed via the monitor outside the MRI suite. The patient maintained unassisted spontaneous respirations throughout the procedure. Twenty-five minutes prior to the end of the scan, gadopentetate 3.8 ml was given IV. The procedure lasted 104 min and the patient remained stable. The propofol infusion was discontinued. The patient was transferred to a stretcher, placed in a semi-lateral position and taken to the postoperative unit. The patient's VS were BP 88/64, HR 80, RR 19 and SpO_2 of 100% on O_2 2 L/min. She remained stable in the postoperative unit and was discharged home one hour later.

Discussion

The MRI suite is a noisy, dark, claustrophobic environment that can create fear in patients, especially children.[2] A pediatric MRI is often carried out utilizing sedation.[1] However, if the child is old enough to follow commands and cooperate, sedation may not be needed. The presence of a parent in the MRI suite, or distraction techniques like movie watching, can sometimes allay fears and negate the need for sedation. Sedation is administered if these strategies do not work.

Unfortunately, there is no guarantee that sedation will work effectively. A study by Dalal et al showed a 22.5% inadequate sedation rate among infants who received oral chloral hydrate compared to the 1.4% who received a propofol infusion for MRI scanning. In Dalal's study, sedation was deemed inadequate if the MRI procedures could not be performed after the administration of a maximum dose of chloral hydrate.[3] While midazolam is one of the common medications used for sedation, it may not be effective as the sole sedative agent for a lengthy MRI because of its short duration of action which requires it to be re-dosed or administered in combination with another agent such as fentanyl. Administering a combination of sedatives is a practice not suitable for children, especially those with neurological conditions, due to the increased risk of respiratory complications.[1]

Utilizing anesthesia during an MRI may be expensive but it will facilitate a predictable process and quality scan.[1] It will also eliminate problems like "sedation failure, prolonged induction time, repeated MRI sequences, rescheduled scans, cost with respect to increased personnel time and downtime of the MRI scanner."[2] Eliminating these factors results in efficacy, efficiency and safety. The elimination of sedation failure guarantees the completion of the procedure in a shorter time. This is because there is full control of unintentional movements or behavior that sedation, alone, cannot guarantee. This eliminates the potential harmful outcome of prolonged sedation such as respiratory depression.[3] It also shortens the time of remotely monitoring the patient from outside the MRI suite. Additionally, it helps minimize psychological trauma and maximize the potential for amnesia.[2] Thus, utilizing anesthesia for an MRI scan offers an effective and safe anesthetic experience compared to sedation. This helps meet the expectations of the parent and the child for a pain-free and anxiety-free experience.[4]

Total intravenous anesthesia (TIVA) can be utilized for an MRI scan. The decision to use positive pressure ventilation (PPV) or not is dependent on the patient's present state of health, existing comorbidities and airway anatomy. Studies done by Lutterbey et al have shown an increased incidence of atelectasis in intubated children as compared to spontaneously ventilating children maintained on a propofol infusion without an artificial airway or use of PPV. In fact, atelectasis may be seen at the early stage of MRI scanning in intubated children and even those who are on intermittent PPV.[5] This makes TIVA without PPV a more appealing approach, which also eliminates the need for a ventilator in the MRI suite.

Ketamine, dexmedetomidine and propofol are commonly used medications for TIVA. However, unfavorable side effects of ketamine and dexmedetomidine such as emergence delirium and hemodynamic instability discourage their use in children.[1] Propofol is a rapid acting, sedative-hypnotic agent which is not classified as a barbiturate

or benzodiazepine and has an arm-brain circulation time of 90 to 100 seconds.[6] Its lipophilic property allows for a quick induction of sedation and anesthesia. However, propofol requires repeated boluses or continuous infusion for maintenance.[6] The recommended pediatric induction dose is 2.5-3mg/kg with a maintenance dosing of 125–350 mcg/kg/min.[6] Rapid titratability, short pharmacologic effect and preservation of spontaneous ventilation make it an ideal agent for MRI anesthesia.[6] Nevertheless, respiratory depression is a considerable risk.[3] Vigilance, early recognition of airway compromise and immediate airway interventions are required.

According to Sury and Smith, propofol is recognized as the best of all IV medications for pediatric sedation.[7] They claim the short induction and recovery time are its advantages. A study by Koroglu et al involving 60 children between 1 and 7 years of age showed equally successful imaging between dexmedetomidine and propofol.[8] However, propofol showed faster induction, recovery and discharge times than dexmedetomidine.[1,7] Another prospective trial done by Pershad et al revealed considerably shorter recovery, sedation induction and total scan time compared with a pentobarbital/midazolam/fentanyl regimen.[6]

Providing offsite anesthesia in the MRI suite is always a challenge not only to the patient and radiologist, but to the anesthetist as well. Issues to consider are the lack of direct control over patient's airway and contact with the patient once the procedure starts. It is vital for the anesthetist to be adequately trained in airway and emergency management especially in this setting.[1] Pediatric MRI examination use has grown exponentially and propofol-based TIVA is an efficient regimen to meet this rising demand.[2,4]

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Mentor: Terrie Norris, CRNA, EdD

It is with great pleasure that I present the updated Guide for Authors, included below in this Summer issue. While there are no major changes to the types of items accepted for publication, clarification in has been added in several areas such as evidence-based practice analysis report guidelines, AMA Manual of Style formatting, and the submission checklist. In an effort to honor the original intent of the journal of publishing reports while the author is still a student, the editorial board is making a renewed effort to reduce the review interval, which has increased over the past few years. Submission guidelines have also been adjusted such that items must be submitted at least three months prior to the author's graduation date. You may appreciate that the Guide for Authors is seven pages long – as a journal geared toward student authors it is our intention to emphasize the importance of attention to detail (much as we do in clinical practice) in item preparation. The guide is not meant to be overwhelming but helpful in the sense that it provide appropriate direction to the novice author. We are committed to providing a positive experience for student authors, and inspiring a career commitment to scholarly activity. I'd like to close this editorial by restating the mission:

> "***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.**"

Vicki C. Coopmans, CRNA, PhD
Editor

INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA
GUIDE FOR AUTHORS

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/initialism 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 ^{o}C, pressure in mm Hg or mm H_20.
   c. In general, first use of pulmonary/respiratory abbreviations should be expanded, with the following exceptions: O_2, CO_2, PCO_2, $PaCO_2$, PO_2, PaO_2. Please use SpO_2 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 <u>one</u> 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 <u>patient</u> 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 <u>patient</u> 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.)

<u>Case Reports</u>
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)
[space]
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.
[space]
Case Report (bold, 400-500 words)
[space]
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.
[space]
Discussion (bold, 600-800 words)
[space]
Describe the ***anesthesia*** implications of the focus of the case report citing current literature. Describe the rationale for your actions and risk/benefits of any options you may have had. This section is not merely a pathophysiology review that can be found in textbooks. *Relate the anesthesia literature with the conduct of your case noting how and why your case was the same or different from what is known in the literature.* Photographs are discouraged unless they are essential to the article. Photos with identifiable persons must have a signed consent by the person photographed forwarded to the editor via first class mail. Diagrams must have permission from original author. This is the most important part of the article. In terms of space and word count this should be longer than the case presentation. End the discussion with a summary lesson you learned from the case, perhaps what you would do differently if you had it to do over again.
[space]
References (bold)
[space]
A minimum of 5 references is recommended, with a maximum of 8 allowed. 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.
[space]
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)
[space]

Introduction (bold)
[space]
A brief introductory paragraph including purpose and hypotheses.
[space]
Methods (bold)
[space]
Include research design and statistical analyses used
[space]
Results (bold)
[space]
Present results – do not justify or discuss here.
[space]
Discussion (bold)
[space]
Discuss results
[space]
References (bold)
[space]
Not required, but a maximum of 5 references is allowed.
[space]
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)
[space]
Introduction (bold)
[space]
Briefly introduce the reader to the practice issue or controversy, describe the scope or significance or problem, and identify the purpose of your analysis. Describe the theoretical, conceptual, or scientific framework that supports your inquiry.
[space]
Methodology (bold)
[space]
Include the format used for formulating the specific question you seek to answer, search terms and methods used, and levels of evidence.
[space]
Literature Analysis (bold)
[space]

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.
[space]
Conclusions (bold)
[space]
Summarize the salient points that support the practice recommendation and make research-supported recommendations that should improve the practice issue, while also acknowledging any limitations or weaknesses
[space]
References [bold]
[space]
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. It is likely your institution's library has a copy on reserve.
http://www.docstyles.com/amastat.htm#Top
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.
http://www.nlm.nih.gov/bsd/viewlet/search/journal/journal.html
http://www.ncbi.nlm.nih.gov/pubmed

The International Student Journal of Nurse Anesthesia (ISJNA) is not listed in the PubMed Database. For the purpose of citing the ISJNA *in this Journal* use "**Int Student J Nurse Anesth**" as the abbreviation. 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:
Hamdan A, Sibai A, Rameh C, Kanazeh G. Short-term effects of endotracheal intubation on voice. *J Voice.* 2007;21(6):762-768.

Journal, more than 6 authors:
Chen C, Nguyen MD, Bar-Meir E, et al. Effects of vasopressor administration on the outcomes of microsurgical breast reconstruction. Ann Plast Surg. *2010;65(1):28-31. PMID: 20548236.*

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:
Stoelting R, Dierdorf S. *Anesthesia and Co-Existing Disease*. 3rd ed. Philadelphia: Churchill Livingstone; 1993:351-354.

Chapter from a text:
Burkard J, Olson RL, Vacchiano CA. Regional anesthesia. In Nagelhout JJ, Plaus KL, eds. Nurse Anesthesia*. 4th ed. St. Louis:Elsevier; 2010:977-1030*

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

Gupta A, Aggarwal N, Sharma D. Ultrasound guided ilioinguinal block. *The Internet Journal of Anesthesiology.* 2011;29(1).
http://www.ispub.com/journal/the_internet_journal_of_anesthesiology/volume_29_number_1/article/ultrasound-guided-ilioinguinal-block.html. Accessed August 1, 2011.

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

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

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.