

EDITORIAL	3
CASE REPORTS	
Subcutaneous Emphysema During Laparoscopic Prostatectomy	3
<i>Dia Tite</i>	
Managing Intraoperative Hemorrhage for Placenta Previa	5
<i>Melissa L. Black</i>	
Probable Ephedrine Induced Serotonin Syndrome Symptoms	7
<i>Mark A. Moser</i>	
Jet Ventilation for Pediatric Laryngoscopy	9
<i>Debra A. Heffner</i>	
Hemodynamic Control during Emergence from Intracranial surgery	10
<i>Lisa Killam</i>	
Safety Measures for Prone Positioned Lumbar Laminectomy	12
<i>Nicholas B. Girdner</i>	
Magnesium Sulfate Use in Pheochromocytoma Resection	14
<i>Shana M. Saeger</i>	
Regional Anesthesia for Carotid Endarterectomy	16
<i>Amanda W. Lassiter</i>	
Endovascular Repair of Abdominal Aortic Aneurysm	18
<i>Clifford Gonzales</i>	
Hemoglobin Replacement in Ischemic Heart Disease	19
<i>Kären K. Embrey</i>	
Hyperkalemia in the Patient with Alport's Syndrome	21
<i>Jeanine Riddle</i>	
Prolonged Apnea Following Succinylcholine	23
<i>Matthew L. Toomey</i>	
Identifying Aspiration Risk in Children with Cerebral Palsy	25
<i>Bryan K. Dehner</i>	
Latex Allergy Management During Cesarean Delivery	26
<i>Theresa M. McSherry</i>	
Circuit Obstruction Due to Absorber Wrapping	28
<i>Christina M Pinkerman</i>	
Topics for Discussion	30
Guide for Authors	30

Editorial

Welcome New and Continuing Students

For many, this is your first issue of the Student Journal of Nurse Anesthesia. Thanks to a grant from Baxter Healthcare Corporation, this Journal is being sent free to all nurse anesthesia students who are associate members of the American Association of Nurse Anesthetists (AANA). Mailing labels are purchased for each issue from the AANA.

Journal Goals:

- participation by all Nurse Anesthesia programs.
- publication of a journal unique to nurse anesthesia students.
- publication of academic work in a short turn-around time, facilitating publication while the author is still a student.
- publication of topics that may not be considered by other professional journals.
- publication in a manner that is user friendly to the student author.
- publication in a format that is concise and understandable to student readers.

The Journal is a collection of case reports that address clinical anesthesia topics pertinent to students of nurse anesthesia. A case report is a description of an anesthetic administered by you, the student author. The synopsis of the case emphasizes details pertinent to the topic and demonstrates how the case relates with current literature. It concludes with lessons learned.

Subcutaneous Emphysema During Laparoscopic Prostatectomy **Dia Tite, M.S.N.**

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Over the last few decades, laparoscopy has become a useful tool in the diagnosis and treatment of a number of illnesses.¹ Advantages of laparoscopic surgery include reduced post-operative pain, shorter hospital stay, and rapid return to normal activity.^{2,3} Among the rare complications of laparoscopy are those related to the spread of insufflating gas from the peritoneum to the thoracic space, which may create a pneumomediastinum, pneumopericardium, pneumothorax and subcutaneous emphysema.¹ Anesthesia professionals must be aware of the adverse effects of laparoscopy using carbon dioxide insufflation on cardiovascular and respiratory function.² This case report demonstrates anesthetic management of subcutaneous emphysema with hypercarbia during laparoscopic radical prostatectomy.

We also accept **abstracts** of graduate school research papers.

We would like **photos** to use on the front cover of your journal.

Before submitting anything to us, always consult the Guide to Authors in the most recent issue. Work with a faculty mentor to prepare your writing for submission.

The success of this Journal is dependent upon your participation in collaboration with your faculty. Demonstrate your personal and professional commitment for the safety of all patients by sharing your experiences formally in a journal, such as this one.

A Milestone

Your Journal is now *The International Student Journal of Nurse Anesthesia*. It is the only journal in the world written by and for student nurse anesthetists. We are happy to accept articles from students anywhere in the world. The editorial staff of this journal acknowledges the challenges you face as you prepare to enter the profession of nurse anesthesia. Publication is an important step for you and the profession. Take the step and be recognized. **With the new name there is a new email address so make a note: INTSJNA@aol.com**

Good luck as you continue your journey to becoming a nurse anesthetist!

Ronald L. Van Nest, CRNA, MA

Julie A. Pearson, CRNA, MSN

Case Report

A 52 year-old, 94-kilogram male, classified ASA 2 presented for a laparoscopic radical prostatectomy for prostate cancer. His medical history was significant for hypertension and anxiety. Surgical history included an appendectomy and left knee surgery. Current medications were valsartan and escitalopram. His physical examination, vital signs and laboratory values were within normal limits. Airway evaluation revealed no limitation on cervical range of motion, a Mallampati class II view, and a thyro-mental distance of three finger breadths.

The patient received midazolam 2 mg and cefazolin 1 gm preoperatively. In the operating room, the patient was positioned supine and standard monitors were applied. Anesthesia was induced with lidocaine 100 mg, propofol 200 mg, fentanyl 100 mcg and atracurium 50 mg. Endotracheal tube placement was confirmed and mechanical ventilation was instituted. Anesthesia was maintained with oxygen and desflurane. Neuromuscular blockade was maintained with subsequent doses of atracurium.

The abdomen was insufflated with carbon dioxide (CO₂) at 15 mmHg and the patient was placed in the Trendelenburg position. After approximately 60 minutes, the patient's end tidal carbon dioxide (ETCO₂) gradually began to rise despite adjustments in tidal volume (TV). Within the next 20 minutes, the ETCO₂ rose to 72 mmHg despite further adjustments in TV and respiratory rate. The peak inspiratory pressure increased to 45 cm H₂O and oxygen saturation decreased to 93%. Upon physical examination, there was extensive subcutaneous emphysema in the chest, neck and face. Breath sounds were equal bilaterally. The abdominal CO₂ insufflation pressure was decreased resulting in increased tidal volume, decreased ETCO₂ and improvement of oxygen saturation. The subcutaneous emphysema gradually diminished.

At the conclusion of surgery, neuromuscular blockade was antagonized with neostigmine 4 mg and glycopyrrolate 0.5 mg. After meeting extubation criteria, the endotracheal tube was removed. Supplemental oxygen was provided via nasal cannula and the patient was transferred to the recovery room. The patient displayed adequate ventilation and oxygenation post-operatively. A chest radiograph was obtained in the recovery room which revealed low lung volumes and subcutaneous air in the soft tissues of the neck and chest wall bilaterally. After full anesthetic recovery, the patient was discharged to home later that day.

Discussion

Laparoscopic complications can occur secondary to pneumoperitoneum depending on intra-abdominal pressure, amount of absorbed CO₂, circulatory volume of the patient, ventilation technique used, underlying pathologic conditions and type of anesthesia.⁴ Carbon dioxide is highly soluble and can easily diffuse from the peritoneal cavity into the circulation and subcutaneous tissues.^{4,5,6} Absorbed CO₂ can only be eliminated through the lungs and hypercarbia can only be avoided by a compensatory hyperventilation achieved by increasing the minute ventilation in anesthetized patients.

Subcutaneous emphysema occurs in 0.3-3% of laparoscopic procedures.⁴ Although subcutaneous emphysema alone may not have serious consequences, it can be an indicator of capnothorax and therefore should be excluded in the presence of subcutaneous

emphysema. Chest radiograph and physical examination excluded capnothorax in this case. Independent risk factors for the development of subcutaneous emphysema are maximum ETCO₂ of 50 mmHg or more, the use of six or more operative ports and operative time greater than 200 minutes.⁶ This patient had a maximum ETCO₂ of 72 mmHg and an operative time of approximately 240 minutes.

Maintaining the intra-abdominal pressure below 12 mmHg reduces the incidence of ventilatory changes.⁴ Although the intra-abdominal insufflation pressure was reduced to 12 mmHg after the development of hypercarbia, it was initially set at 15 mmHg. Compensatory ventilation can be impeded by the Trendelenburg position and severe hypercapnia can develop even with aggressive ventilation. This patient was placed in the Trendelenburg position intermittently to better visualize the operative field. Pneumoperitoneum may also cause changes in the lung shape and distortion of the lungs which can decrease lung compliance.⁷

An increase in ETCO₂ by 10 mmHg develops without controlled ventilation, and large tidal volumes (12-15 ml/kg) should be instituted during laparoscopy to prevent progressive alveolar atelectasis and hypoxemia and allow CO₂ elimination.⁴ The "alveolar recruitment strategy" is also recommended, which consists of manual ventilation to an airway pressure of 40 cm H₂O for 10 breaths over one minute followed by usual mechanical ventilation with mild positive end-expiratory pressure (5 cm H₂O).⁴ This improves arterial oxygenation without cardiovascular compromise or respiratory complications.

Post-operatively, a patient with subcutaneous emphysema may require prolonged mechanical ventilation. Up to 120 liters of CO₂ can be stored in the human body during pneumoperitoneum which may delay CO₂ elimination. When subcutaneous emphysema develops in the neck, the patient is at risk for upper airway obstruction⁴ and therefore requires close monitoring of the airway. Although this patient exhibited significant subcutaneous emphysema in the neck, he demonstrated no adverse respiratory effects post-operatively. Also, hypercapnia is compounded by respiratory depression, which can occur following general anesthesia. When alveolar ventilation is kept constant, the partial pressure of CO₂ in the arterial blood will return to 95% of prepneumoperitoneum values approximately 45 minutes after desufflation.³

It is imperative that the nurse anesthetist be cognizant of the potential respiratory complications associated with laparoscopy. The surgeon needs to be aware that lower insufflation pressures attenuate pathophysiologic responses and reduce the incidence of complications.⁴ The surgeon should be informed immediately if the intra-abdominal pressure exceeds 12 mmHg and when hypercarbia or subcutaneous emphysema develops. Awareness of the potential complications and symptoms

that preclude these complications can help prevent serious adverse outcomes associated with laparoscopic surgery.

References

1. Perko G, Fernandes A. Subcutaneous emphysema and pneumothorax during laparoscopy for ectopic pregnancy removal. *Acta Anaesthesiol Scand.* 1997;41:792-794.
2. Perrakis E, Vezakis A, Velimezis G, et al. Randomized comparison between different insufflation pressures for laparoscopic cholecystectomy. *Surg Laparosc Endosc Percutan Tech.* 2003;13:245-249.
3. Massimo G, Da Broi U, Antonutto G, Pasetto A. The effect of laparoscopic cholecystectomy on cardiovascular function and pulmonary gas exchange. *Anesth Analg.* 1996;83:134-140.
4. Gutt T, Oniu T, Mehrabi A, et al. Circulatory and respiratory complications of carbon dioxide insufflation. *Dig Surg.* 2004;21:95-105.
5. Liem MSL, Kallewaard JW, de Smet AM, van Vroonhoven TJ. Does hypercarbia develop faster during laparoscopic herniorrhaphy than during laparoscopic cholecystectomy? *Anesth Analg.* 1995;81:1243-1249.
6. Murdock C, Wolff A, Van Geem T. Risk factors for hypercarbia, subcutaneous emphysema, pneumothorax, and pneumomediastinum during laparoscopy. *Obstet Gynecol.* 2000;95:704-709.
7. Pelosi P, Foti G, Cereda M, Vicardi P, Gattinoni L. Effects of carbon dioxide insufflation for laparoscopic cholecystectomy on the respiratory system. *Anaesthesia.* 1996;51:744-749.

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Managing Intraoperative Hemorrhage for Placenta Previa

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Placenta previa manifests as a partial or complete obstruction of the internal cervical os and is a complication of approximately 0.4 % of all pregnancies.¹ The incidence of morbidity and mortality from placenta previa is high during pregnancy as well as the time of delivery, primarily due to hemorrhage. A cesarean delivery is recommended to minimize blood loss in these cases. Due to the potential of significant blood loss, the intraoperative management of placenta previa presents a significant challenge to the anesthesia professional who must ensure normovolemia. This case report discusses the anesthetic management of intraoperative hemorrhage from placenta previa.

Case Report

A 50 year old gravida 4, para 3 female presented at 27 weeks gestation with twins. A complete placenta previa was noted upon examination and confirmed by ultrasound. This patient was transferred to the labor and delivery unit from an outside hospital. No significant medical history other than iron deficiency anemia and gastroesophageal reflux disease (GERD) was reported on transfer. Prior surgical history revealed three previous cesarean deliveries without complications that all resulted in live births. On arrival the patient complained of shortness of breath, productive cough and lower extremity edema. The diagnoses of non-cardiogenic pulmonary edema and pneumonia were made. She was admitted for observation until an increase in vaginal bleeding was noted that was exacerbated by her continuous coughing. Due to the risk for both fetal and maternal compromise, a decision was made to perform an urgent cesarean delivery.

The patient weighed 68 kg at a height of 5'2". Her vital signs preoperatively were blood pressure 149/81mmHg, pulse 93 bpm, respiratory rate 22 bpm and temperature of 36.5°C. Laboratory values included hemoglobin 8.3 gm/dl, hematocrit 28.9%, platelet count 120,000 and PT/PTT 12.4 sec and 32 sec respectively. Late decelerations were noted on fetal heart rate monitoring. The anesthesia plan was a combined spinal and epidural anesthesia (CSE).

Prior to induction of anesthesia two 14 gauge intravenous catheters were placed and a 20 gauge radial arterial catheter was placed to provide continuous beat-to-beat monitoring of blood pressure. The patient was then given a 500 ml bolus of crystalloid solution and oxygen via nasal cannula. She was immediately transferred to the operative suite and positioned for placement of the CSE. Physiologic monitoring included blood pressure, heart rate, oxygen saturation, urine output via Foley catheter and fetal heart tones. A CSE was successfully placed. A dose 10 mg bupivacaine, 10 mcg fentanyl and 200 mcg morphine was injected intrathecally followed by placement of an epidural catheter into the epidural space. The epidural catheter was tested with 3 ml of local anesthetic to confirm it was not in the subarachnoid or intravascular space and secured. The subarachnoid anesthesia sensory level was determined to be adequate and the cesarean delivery proceeded without complications. Near the end of the procedure the spinal anesthetic was augmented with 2% lidocaine with 1:200,000 epinephrine 5cc, and 100 mcg fentanyl via the epidural catheter.

Intraoperative systolic blood pressures ranged from the 70s to 110s. Intraoperative diastolic blood pressures ranged from the 50s to 70s. Tachycardia was noted after marked decreases in blood pressure. Blood loss totaled three liters. Six units of packed red blood cells, one unit of platelets, one unit of fresh frozen plasma, and three and a half liters of crystalloid solution were administered to maintain adequate organ perfusion and hemostasis. Brief periods of hypotension intraoperatively were managed with supplemental doses of ephedrine. Oxytocin and cefazolin were administered intravenously following cross clamp of the umbilical cord. Hemabate was administered intramuscularly.

A hysterectomy was immediately performed. Afterwards, the incision was closed and the patient was transferred to the surgical intensive care unit for close observation. Angiographic embolization of the uterine artery was not considered due to the emergent nature of this case; however, it is a potential treatment in lieu of a hysterectomy. A week post-partum, the babies remained in stable condition in the neonatal intensive care unit. The mother was transferred from the ICU to a step-down unit and then discharged to home.

Discussion

The incidence of prematurity and fetal/neonatal death is much higher with placenta previa when compared to a normal pregnancy. While the exact etiology of this condition is unknown, clinical and epidemiological studies have correlated placenta previa with advanced maternal age, a history of previous pregnancies, male fetuses, previous cesarean delivery and prior spontaneous or induced abortions.² There is also a higher incidence of placenta previa in twins who were implanted using in vitro fertilization (IVF).³ Mothers who smoke, use drugs or have had placenta previa in a previous pregnancy are at increased risk.

Preparation for the management of intraoperative hemorrhage is essential in order to deliver safe and effective anesthesia care to the patient with placenta previa. Average blood loss during a cesarean delivery for placenta previa varies depending on the type of anesthesia administered. Under regional anesthesia, the average blood loss is approximately 600 ml. Under general anesthesia, average blood loss may exceed this volume by a factor of three.⁴ Despite this data, the use of regional versus general anesthesia remains a matter of personal preference among anesthesia practitioners. Advocates of general anesthesia believe that the sympathectomy caused by regional anesthesia will create a significant challenge in controlling arterial blood pressure in conjunction with severe hemorrhage. Those who prefer to utilize regional anesthesia believe that sympathetic compensation during general anesthesia masks - blood loss and could result in sudden onset circulatory collapse. The advantage of regional anesthesia is that it allows for the close coupling of blood pressure and intravascular fluid volume as

vasoconstriction does not occur with this method allowing for quick recognition of decreased intravascular volume.⁴ Regardless of the chosen method of administering anesthesia, it is important for the anesthesia practitioner to be prepared for significant blood loss during the delivery of a patient with placenta previa.

Interventions in the management of placenta previa may begin with weekly phlebotomy as early as the start of the third trimester in preparation for autologous blood transfusion. The recommended phlebotomy draw is 400 milliliters (ml) a week up to a suggested autologous donation of 1200-1500 ml.¹ Due to the hypercoagulable state that occurs in many women during pregnancy, storing the blood in a citrate-phosphate-dextrose adenosine solution is essential in preventing coagulation of stored blood. Autologous blood transfusion will eliminate the risk of transfusion reaction, transmission of infection and development of antibodies that may be associated with homologous blood transfusion. This patient had not donated autologous blood prior to arrival as she was only 27 weeks gestation and the emergent nature of the delivery was not conducive to donation during her hospital stay. Homologous blood, platelets and fresh frozen plasma were therefore used for resuscitative purposes.

Prior to taking the patient to the operating room, preparations should be made for the management of intraoperative hemorrhage. Ensuring that the patient has two large bore intravenous (IV) catheters to allow for faster flow rates of IV solutions and placing an arterial catheter for continuous monitoring of blood pressure and blood gases are both recommended interventions which were performed in this case.⁵ Additional monitoring, aside from standard monitors, may include five lead EKG, urinary catheter, and invasive monitors to measure fluid status. Due to the emergent nature of this case, a central line was not inserted and invasive monitoring of fluid status was not instituted. As circulatory volume is believed to be significantly compromised in these patients during delivery, insertion of these lines may prove beneficial during the intraoperative period. Breath sounds must be auscultated throughout the procedure to detect fluid volume overload.⁵

The outcome of this case was favorable for both mother and babies. Autologous blood was not available; however, a type and crossmatch was performed prior to transporting the patient to the operating room. Preoperative preparation is key to the management of placenta previa and to the avoidance of adverse maternal and fetal outcomes.

References

1. Yamada, T, Mori, H, Ueki, M. Autologous blood transfusion in patients with placenta previa. *Acta Obstet Gynecol Scand.* 2005;84:255-259.

2. Faiz, AS, Ananth, CV. Etiology and risk factors for placenta previa: an overview and meta-analysis of observational studies. *J Matern Fetal Neonatal Med.* 2003;13:175-190.
3. Smithers, PR, Halliday, J. High frequency of cesarean section, antepartum hemorrhage, placenta previa, and preterm delivery in in-vitro fertilization twin pregnancies. *Fertil Steril.* 2003;80:666-668.
4. Parekh, N, Husaini, SWU, Russell, IF. Caesarean section for placenta praevia: a retrospective study of anaesthetic management. *Br J Anaesth.* 2000; 84:725-730.
5. Macmullen, NJ, Dulski, LA, Meagher, B. Red alert: perinatal hemorrhage. *The Am J Matern Child Nurs.* 2005;46-51.

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Probable Ephedrine Induced Serotonin Syndrome Symptoms

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Serotonin syndrome is an iatrogenic pharmacologic syndrome induced by concomitant use of serotonergic medications.^{1,2} Excessive serotonergic activity in the nervous system results in a triad of symptoms including altered levels of consciousness, altered autonomic nervous system function and abnormal neuromuscular activity.¹ Serotonin syndrome symptoms range from mild to fatal without predilection to sex, race, or age.^{1,2,3} In 2002, approximately 28,000 patients experienced serotonin syndrome symptoms, nearly 8,000 involved potentially lethal effects and close to 100 died.³ Anesthesia professionals use several serotonin syndrome triggering agents daily throughout the perioperative period (meperidine and other opioids, cocaine, and ephedrine). The following case report discusses the implications and management of a patient with probable serotonin syndrome symptoms during a laparoscopic cholecystectomy.

Case Report

A 63-inch tall 52 year-old female weighing 76 kilograms presented for laparoscopic cholecystectomy. She had a history significant for anxiety, major depressive disorder, hypertension, and gastroesophageal reflux disease. She also recently experienced a severe episode of serotonin syndrome related to intravenous (IV) meperidine administration during a gall bladder attack. She required intensive care admission without mechanical ventilation as a result of this iatrogenic induced occurrence. Her medications included bisoprolol fumarate/hydrochlorothiazide, clonazepam and ranitidine. She was also taking phenelzine which decreases serotonin metabolism. On physical examination her heart rate was 73 bpm with a regular rate and rhythm. She had a systolic blood pressure between 118-129 mmHg. Her lungs were clear to auscultation. Her oxygen saturation was 92-96% on room air. She was anxious, but alert and oriented. She stated no allergies to foods or medications. She had experienced no anesthetic complications during previous surgeries. The anesthetic

evaluation demonstrated a Mallampati class III airway. She had no range of motion limitations, thyromental distance was 3 fingerbreadths, and mouth opening of 2 finger breadths. She was considered an ASA class II.

She was given midazolam 2 mg IV and taken to the operating room where she received fentanyl 50 mcg IV and was preoxygenated via face mask. Standard monitors were applied. Then fentanyl 50 mcg IV and droperidol 0.625 mg IV was administered. General endotracheal anesthesia was induced slowly with lidocaine 50 mg IV and propofol 200 mg IV. Easy mask ventilation was confirmed and rocuronium 50 mg IV was administered for muscle relaxation. A 7.0 mmID endotracheal tube was placed without difficulty. Dexamethasone 5 mg IV was administered.

Anesthesia was maintained with 1 L/m of oxygen, 1 L/m of air, and 1.3% sevoflurane. She maintained a SaO₂ of 98-100 % throughout the procedure. Bispectral Index Monitoring was also utilized and remained in the mid 20's throughout the procedure.

After induction, the patient's systolic blood pressure (SBC) decreased into the low 80s with a heart rate in the low 50s. Ephedrine 5 mg IV was administered without effect. An additional ephedrine 10 mg IV was given producing a systolic blood pressure of 130-140 mmHg. Within 15 minutes of the patient's blood pressure return to normal, her temperature changed from 34.6 to 37.5°C and continued to increase. Her systolic blood pressure increased to 202 mmHg with a heart rate of 90-100 bpm and she became diaphoretic. Her ETCO₂ remained normo-carbic. Her pupils were dilated. She was given an additional 100 mcg fentanyl IV without effect on SBP or heart rate. A total of labetalol 25 mg IV and hydralazine 20 mg IV was administered. The systolic blood pressure returned to 105-123 mmHg with a heart rate of 80-90 bpm. Cold IV fluids were administered along with a cooling blanket utilizing cool forced air to maintain an esophageal temperature of 35.6-36.7°C.

At the end of the procedure the neuromuscular blockade was antagonized with neostigmine and glycopyrrolate, the trachea was extubated, and the patient was taken to recovery room. Postoperatively her oral temperature remained < 37.1°C. She was later admitted to day hospital and discharged to home after 23 hours without complications.

Discussion

Serotonin syndrome usually presents as a triad of signs and symptoms involving a change in the level of consciousness, autonomic instability, and increased neuromuscular excitability.² This triad of signs and symptoms is usually the result of traditional monoamine oxidase inhibitors concomitantly used with other serotonergic medications. Serotonergic medications include selective serotonin reuptake inhibitors, tricyclic antidepressants, meperidine, cocaine, and ephedrine. The primary detectable signs and symptoms of a patient under general endotracheal anesthesia can involve hypo/hypertension, hyperthermia, hyperhidrosis, rigidity (without paralytics), mydriasis, and tachycardia.^{1,4,5} All of these signs and symptoms are vague and descriptive of not only light anesthesia, but of serotonin syndrome's primary differential diagnoses: Neuroleptic Malignant Syndrome and Malignant Hyperthermia.^{1,6} The patient in this case study developed an increase in core temperature, became hypertensive, and tachycardic. She also became diaphoretic with dilated pupils. All of these signs appeared within 15 minutes of IV administration of ephedrine. Ephedrine, an indirect acting sympathomimetic, which increases norepinephrine availability, can lead to a hypertensive crisis and provoke a serotonergic syndrome.^{6,7} This patient responded well to increased fluids, cooling, labetalol and hydralazine. The patient was believed to have experienced a serotonin syndrome reaction.

Opioids have also been implicated in provoking serotonin syndrome causing increased rigidity (especially of the lower extremities), hypertension, and hyperthermia. Meperidine is the most common opioid contraindicated for concomitant use with MAOIs and other serotonergic medications.^{6,7} The patient in this case study had a severe serotonin syndrome episode when given meperidine for a gallbladder attack in the emergency department. That episode required an admission to the intensive care unit without a need for respiratory support. There she received cyproheptadine (serotonergic antagonist), cold intravenous fluids, and cooling blanket treatments.⁸

In most cases of serotonin syndrome, with early recognition and institution of supportive therapy outcomes are usually good, as in both instances experienced by the patient in this case report. However, serotonin syndrome can be fatal. Serotonin syndrome can lead to acute renal failure, disseminated intravascular coagulation, multiple organ dysfunction syndrome, and acute respiratory distress syndrome.^{4,9} Serotonin

syndrome can be diagnosed using Sternbach's Criteria. However, the anesthesia professional may be better served using a revision of this system which may allow a more prompt and accurate diagnosis under general anesthesia.⁹ This revised system includes the more common symptoms which could be detected under general anesthesia. Treatment of serotonin syndrome consists chiefly of immediate cessation of the offending medications and supportive care as necessary. Cyproheptadine, a serotonin antagonist, remains an option which may shorten the duration of signs and symptoms.⁴

It continues to be more critical than ever for anesthesia professionals to perform a thorough preoperative history and physical examination in this era of polypharmacy. The preparation of anesthetic delivery must be individualized. Professionals delivering anesthesia must be able to adapt their anesthetic delivery to any circumstance arising in the perioperative period. If this patient undergoes another surgical procedure, her serotonergic medications should be stopped at least 2 weeks prior to the procedure.⁶ She should be adequately hydrated preinduction and the most hemodynamically stable method of induction and delivery of anesthesia should be used throughout the perioperative period. Her known sensitivity to serotonin syndrome triggering agents must be noted on all anesthesia forms.

References

1. Mills KC: Serotonin syndrome: A clinical update. *Crit Care Clin.* 1997;13:764-783.
2. Jones D, Story DA. Serotonin syndrome and the anaesthetist. *Anaesth Intensive Care.* 2005 Apr;33(2):181-187.
3. Boyer EW, Shannon M. Current concepts: The serotonin syndrome. *N Engl J Med.* 2005;352(11):1112-1120.
4. Mason PJ, Morris VA, Balcezak TJ. Serotonin syndrome. Presentation of 2 cases and review of the literature. *Medicine.* 2000;79:201-209.
5. Marcucci C, Sandson NB, Dunlap JA. Linezolid-bupropion interaction as a possible etiology of severe intermittent intraoperative hypertension. *Anesthesiology.* 2004;101(6):1487-1488.
6. Yao FF. Yao & Artusio's *Anesthesiology: Problem-oriented Patient Management.* 5th ed. New York: Lippincott, Williams, & Wilkins;2003:1138-1141, 632.
7. Stoelting RK, Dierdorf SF. *Anesthesia and Coexisting Diseases.* 4th ed. New York: Churchill Livingstone; 2002:632-633.
8. Arnold DH: The central serotonin syndrome: Paradigm for psychotherapeutic misadventure. *Pediatr Rev.* 2002;23:427-432.

9. Radomski JW, Dursun SM, Revely MA, Kutcher SP. An exploratory approach to the serotonin syndrome; an update of clinical phenomenology and revised diagnostic criteria. *Med Hypotheses*. 2000;55:218-224.

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Jet Ventilation for Pediatric Laryngoscopy

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Manual jet ventilation is used in pediatric and adult surgeries where wide, unobstructed access to the entire endolarynx is needed. Studies have shown its effectiveness in laser laryngeal surgery, suspension microlaryngoscopy, aortic coarctation repair, and tracheal stenosis.^{1,2,3,4} In this approach a manual jet ventilator is connected to a side port of the laryngoscope. Inspiration is achieved by placing the end of the device supraglottically and depressing the handle on the device for 1 to 2 seconds. High pressure oxygen (30-50 psig) is directed through the glottic opening and into the lungs using the Venturi effect which also transports surrounding air into the opening. Expiration is passive and should last 4 to 6 seconds to avoid air trapping and barotrauma.⁵ The use of manual jet ventilation is an important addition to pediatric laryngoscopy as shown in this case study.

Case Report

A four month old female weighing 5.23 kilograms presented to the operating room for a suspension laryngoscopy and bronchoscopy. The patient was born with a type IV laryngotracheal cleft, which is a full laryngotracheoesophageal cleft and has a mortality rate of 93%.⁶ A gastric tube was placed the second day of life and the cleft was repaired within the first two months of life. Complications remained after the initial procedures and the patient's trachea remained intubated and feedings continued through the gastric tube. Attempts at extubation were unsuccessful as the patient had emergent respiratory distress and was unable to exchange air. Two attempts at tracheostomy were unsuccessful, both resulting in profound desaturation and bradycardia with subsequent cardiopulmonary resuscitation being required.

The surgical plan for this case was to perform a suspension laryngoscopy and bronchoscopy to determine what was causing the respiratory failure with extubation and impeding tracheostomy placement.

Vital signs upon entering the operating room were 100/50 mmHg, 140 bpm, and SaO₂100%. The circle system was connected to the number 3.5 mmID uncuffed endotracheal tube and oxygen with 3% sevoflurane was administered. Induction was supplemented with propofol 10 mg via the central line. Neuromuscular blockade was achieved with rocuronium 3mg and fentanyl 5mcg was given for analgesia. A leak around the endotracheal tube was

recorded at 15 cm of H₂O pressure. The patient was easily ventilated through the endotracheal tube.

When it was determined that the vocal cords needed to be viewed without the impedance of the endotracheal tube it was removed under direct laryngoscopy. Jet ventilation was connected through a side port of the laryngoscope and oxygen was administered supraglottically. The inhalation agent was discontinued. A 10 mg bolus of propofol was given and an infusion was started at 100 mcg/kg/minute. Additional doses of rocuronium and fentanyl were given to supplement neuromuscular blockade and analgesia. Jet ventilation was continued for 20 minutes with no resultant desaturation.

Once the surgical team concluded their inspection with no definitive diagnosis, a number 3.5 uncuffed endotracheal tube was placed back through the vocal cords under direct laryngoscopy. The propofol infusion was discontinued and nitrous oxide was administered at 50 %. The neuromuscular blockade was antagonized and once spontaneous ventilation resumed the endotracheal tube was removed and air movement was visualized via direct laryngoscopy with no additional conclusions as to the cause of respiratory distress.

The laryngoscope was removed to observe respiratory function without the support of the endotracheal tube. It was immediately determined that the patient was not exchanging air. Attempts to ventilate with two person positive pressure mask ventilation were unsuccessful. Oxygen saturation began to drop and reached 80%. Direct laryngoscopy was performed, a number 3.5 uncuffed endotracheal tube was placed, and 100% oxygen was administered. Positive end tidal CO₂ was recorded and bilateral breath sounds were auscultated. The oxygen saturation quickly returned to 100%. The decision was made to allow the patient to awaken completely and leave the endotracheal tube in place. Dexamethasone 2.5 mg was given to prevent swelling. The patient was transported to the NICU while breathing 100% O₂ and fully monitored. Since this procedure the patient had a tracheostomy and as of this writing remains in the NICU.

Discussion

Jet ventilation is an excellent choice for surgeries that require a wide unobstructed view of the entire endolarynx.¹ This is especially important in patients that

may not tolerate the intermittent-apnea technique, in which periods of ventilation by face mask are alternated with periods of apnea, or insufflation of high flows of oxygen through a small catheter in the trachea.⁵ Infants are one such group of patients that have decreased reserve and can quickly become hypoxic. This was evident by the previous cases performed on this patient during which cardiopulmonary resuscitation was initiated in response to hypoxemic and bradycardic episodes.

Jet ventilation is an important adjunct for airway management. Transtracheal tracheal jet ventilation is a part of the ASA Difficult Airway Algorithm.⁷ However, there are concerns in the administration of jet ventilation. Major complications include the potential for tracheal injury, barotrauma and pneumothorax due to repeated exposure to high-pressure gas flow. Studies have reported death in neonates that received 8 hours or more of high frequency jet ventilation in an intensive care unit.⁸ These incidents are rare and are reportedly improved with humidification of delivered gas. Other disadvantages are the inability to administer inhalational agents and to measure end-tidal CO₂.³

The advantages of a controlled operative field need to be weighed against the disadvantages listed above. In certain circumstances the use of jet ventilation provides the advantage of continuous, controlled ventilation when unobstructed view of the larynx is required by the surgical team.

References

1. Borland LM, Reilly JS. Jet ventilation for laser laryngeal surgery in children. Modification of the Saunders jet ventilation technique. *Int J Pediatr Otorhinolaryngol.* 1987;14:65-71.

2. Rubin JS, Patel A, Lennox P. Subglottic jet ventilation for suspension microlaryngoscopy. *J Voice.* 2005;19:146-150.
3. Cotter T, Russo P, Tobias JD. Intraoperative jet ventilation during aortic coarctation repair in an infant. *J Cardiothorac Vasc Anesth.* 2004;18:207-209.
4. Biro P, Hegi TR, Weder W, Spahn DR. Laryngeal mask airway and high-frequency jet ventilation for the resection of a high-grade upper tracheal stenosis. *J Clin Anesth.* 2001;13:141-143
5. Morgan GE, Mikhail MS, Murray MJ. *Clinical Anesthesiology.* 3rd ed. New York: Lange Medical Books; 2002.
6. Briggs RD, Deskin RW. Congenital laryngeal anomalies. Grand round presentation, UTMB, Dept. of Otolaryngology. 2002.
7. American Society of Anesthesiologists Task Force on Difficult Airway Management. Difficult airway algorithm. 1992.
8. Boros SJ, Mammel MC, Coleman JM, et al. Neonatal high-frequency jet ventilation: Four years' experience. *Pediatrics.* 1985;75:657-663.

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Hemodynamic Control during Emergence from Intracranial surgery

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Strict control of blood pressure is of significant importance during neurosurgical procedures. Periods of hypertension during neurosurgery or on emergence from anesthesia can result in cerebral edema and/or postoperative bleeding.¹ Blood pressure variations in neurosurgical patients often arise from factors related to their pathology.

Early intracranial hypertension after elective craniotomy occurs in approximately 20% of patients. Intracranial hemorrhage (ICH) triggers a significant portion of this incidence. It is possible to control blood pressure during emergence from craniotomy without affecting cerebral blood flow (CBF). There is an association between intraoperative or immediate (12 hours) postoperative hypertension and the development of subsequent ICH.²

ICH after craniotomy can substantially increase length of stay, morbidity and mortality, and cost of care. This case describes the maneuvers utilized to maintain tight blood pressure parameters intraoperatively, upon emergence, and postoperatively, in a patient undergoing intracranial surgery.

Case Report

A 58 year old, 111 kilogram, 62 inch tall, female presented for a craniotomy and excision of a right sphenoid wing meningioma. She complained of a two week history of worsening headache and had been emergently admitted to the hospital, following a grand mal seizure four days before surgery. Work up revealed a right sphenoid wing mass.

Medical history included hypothyroidism, hypertension, and remote endometrial cancer. The patient's medications included synthroid, multivitamin, and atenolol. She did take the atenolol on the day of surgery.

Preoperative vital signs were within normal limits with an oxygen saturation of 94% while breathing room air. Lung sounds were clear bilaterally and heart sounds were normal. Airway assessment revealed a Mallampati II classification. She was classified as an ASA II.

Midazolam 2 mg was administered intravenously (IV) for anxiolysis. Standard monitors were placed and 100% oxygen by mask was administered. Induction drugs included propofol 100 mg, fentanyl 50 mcg, vecuronium 10 mg, and lidocaine 100 mg. During mask ventilation with 10 L/min of oxygen, isoflurane was added at 1.2%. Tracheal lidocaine 160 mg was given prior to the trachea being successfully intubated with a #7 endotracheal tube (ETT) using a curved size 3 laryngoscope blade. A 20 gauge radial arterial catheter was inserted after induction. Isoflurane 1.1% was used for maintenance throughout the case with air and oxygen delivered at 1 L/min each.

Intraoperative hemodynamics were stable and the surgery was uneventful. Paralysis was maintained with vecuronium throughout the case. The patient received a total of 600 mcg fentanyl, 20 mg vecuronium, 1 gm phenytoin, and 4 mg ondansetron. Neuromuscular blockade was antagonized with glycopyrrolate 0.6 mg and neostigmine 4 mg. Sevoflurane at 1.7% was substituted for the isoflurane during closure.

At emergence, the patient's blood pressure increased from 100/60 mmHg to 150/90 mmHg. Labetalol, in 10-20 mg boluses, was administered in titrated doses to control the blood pressure. A total of 100 mg labetalol was given over a 45 minute time period. The blood pressure decreased slightly to 140/82 mmHg. Although the desired goal of systolic below 120 mmHg was not successfully achieved, the patient awoke calmly without any coughing when the ETT was removed. She had a patent airway and followed commands. The patient's blood pressure remained elevated post extubation for 10 minutes until a nitroprusside infusion was initiated at 0.5 mcg/kg/min. It was increased to 1 mcg/kg/min within 5 minutes. The blood pressure responded immediately and decreased to 120/64 mmHg. The patient was transferred to the post anesthesia care unit where her neurological status was satisfactory, vital signs were within normal limits, and her blood pressure was stable at 118/50 mmHg. The patient required the nitroprusside infusion for 1 hour postoperatively. She recovered uneventfully, and was discharged to home on postoperative day five with no neurologic deficits.

Discussion

Hypertension is a common emergence phenomenon that can have serious consequences and often has a neurologic

etiology in the post craniotomy patient. In a two-part prospective clinical trial of elective craniotomy patients, Bruder et al. reported transcranial doppler (TCD) data that confirmed cerebral hyperemia on anesthetic emergence. This occurred in patients anesthetized with propofol or isoflurane. There was a 60-80 % increase in CBF velocity on extubation from preinduction baseline.²

Beta blockers, calcium channel blockers or ACE inhibitors have been used successfully to induce normotension/hypotension during isoflurane anesthesia. All have been shown to be effective in controlling emergence hypertension. There are many studies that have supported the use of short acting beta blockers during emergence. Grillo compared the effects of esmolol to placebo, and found that esmolol blunted the increase in cerebral blood flow velocity during recovery from neurosurgical anesthesia.⁵ This finding supported the hypothesis that sympathetic stimulation contributes to cerebral hyperemia during emergence from craniotomy.⁴

In another study, a calcium channel blocker (nicardipine) and a combined alpha-beta blocker (labetalol) for control of emergence hypertension during craniotomy for tumor surgery proved very effective. Nicardipine was combined with enalaprilat for control of post craniotomy emergence hypertension. Enalaprilat IV, half-life 11 hours, was given to patients at closure of the dura mater to provide long-term antihypertensive protection. Enalaprilat (maximum effect 28.1 minutes) was supplemented with labetalol (onset 10-20 seconds) which was 98% effective in controlling the plasma rennin levels and catecholamine effect. Labetalol is ideal for patients undergoing craniotomy because it does not influence CBF or CBF auto regulation.¹

In the presented case, labetalol was the only agent used until it was decided that another agent, nitroprusside, was required. From the time that the blood pressure was elevated it took a total of one hour to reduce the blood pressure to a desired and accepted range for this patient. In addition to labetalol, a calcium channel blocker could have been added for more rapid control of the blood pressure. This patient's pre existing hypertension may have contributed to her post op hypertension, even though she had received her beta blocker on the morning of surgery.

In addition to managing the hemodynamic changes which accompany emergence, it is important to consider the choice of inhalation agent for intracranial surgery. This is paramount during neurosurgery because of the varying effects of inhalation agents on CBF, ICP and auto regulation of CBF. In the case presented, isoflurane was used for 4 hours and sevoflurane was substituted for the last hour of the case.

Gauthier et al. compared inhalation agents and time to recovery. The report also evaluated postoperative neurological assessment in long duration intracranial

cases. Sevoflurane has many properties that support its use in neuroanesthesia. It preserves dynamic autoregulation better than isoflurane. This reflex is completely abolished by 0.5 MAC of desflurane. In contrast to desflurane, sevoflurane does not increase intracranial pressure up to a concentration of 1.5 MAC in normocapnia. Up to the dose of 1.5 MAC sevoflurane is not as potent a cerebral vasodilator as isoflurane or desflurane and it also better preserves carbon dioxide reactivity and metabolic coupling.³

Factors related to emergence hypertension after craniotomies are most likely different from those common to other surgical procedures. Surgical pain is not likely the dominant trigger in the development of postoperative hypertension. It is important to be aware of the potential for neurological sequelae caused by emergence hypertension associated with neurosurgery. Anesthesia professionals should be prepared with several agents to treat the hemodynamic variability while preserving the ability to assess neurologic status. Interference with central auto-regulation, intracranial events and central reflexes in addition to airway stimulation, can provide an explanation for the high incidence of emergence hypertension after craniotomy.

References

1. Kross R, Ferri E, Leung D, et al. A comparative study between a calcium channel blocker (nicardipine) and a combined alpha-beta-blocker (labetalol) for the control of emergence hypertension during craniotomy for tumor surgery. *Anesth Analg.* 2000;91:904-909.
2. Armin S. Cerebral hyperemia, systemic hypertension, and perioperative intracranial morbidity: is there a smoking gun? *Anesth Analg.* 2002;93:485-487.
3. Gauthier A, Girard F, Bordreault D, Ruel M, Todorov A. Sevoflurane provides faster recovery and postoperative neurological assessment than isoflurane in long-duration neurosurgical cases. *Anesth Analg.* 2002;95:1384-1388.
4. Grillo P, Bruder N, Auquier P, Pellissier, Gouin, F. Esmolol blunts the cerebral blood flow velocity increase during emergence from anesthesia in neurosurgical patients. *Anesth Analg.* 2003;96:1145-1149.
5. Yao, F. *Anesthesiology: problem orientated patient management.* 5th Ed. Philadelphia: Lippincott Williams and Wilkins; 2003:524.

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Safety Measures for Prone Positioned Lumbar Laminectomy

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A lumbar laminectomy is a procedure involving the removal of the lamina from the affected lumbar vertebrae. Lumbar laminectomies are performed for treatment of radiculopathies, spinal stenosis, cysts/tumor, and degenerative spondylolisthesis.¹ The prone position, also known as the ventral decubitus position, is the most common position used for posterior spinal surgeries. Reported complications of the prone position include eye, ear, neck, breast, and brachial plexus injury as well as abdominal and thoracic compression resulting in respiratory and circulatory compromise.^{2,3,4} The eyes are of particular concern with an estimated incidence of 0.01%-1% visual loss after surgery involving the prone position.⁵ The following case report describes the positioning and anesthetic management of a patient undergoing a lumbar laminectomy.

Case Report

A 79-year-old, 5'1", 56.9 kg, ASA 3 female presented for an elective lumbar laminectomy. The patient was scheduled for this procedure after experiencing acute onset pain involving her right lower extremity. The pain originated in her hip and extended down the

posterolateral aspect of her right leg into her foot. The pain in her foot was limited primarily to the plantar surface. She reported decreasing ability to walk over the past few years and seeks assistive devices, such as a motorized cart. The patient stated when she is able to lean on an object she is able to walk longer distances.

Previous surgical history included partial thyroidectomy, bilateral hip replacements, hysterectomy, and bilateral cataract extraction. The patient's medical history was significant for lumbar stenosis, right S₁ radiculopathy, spondylolisthesis of L₄ on L₅, hypothyroidism, hypertension, COPD, and arthritis. Current medications included quinapril, levothyroxine, and ditropan.

Laboratory studies were all within normal limits. A chest x-ray displayed bibasilar atelectasis. An EKG revealed sinus rhythm with left ventricular hypertrophy. A cardiac perfusion scan from December of 2000 demonstrated no ischemia. However, an echocardiography report from the same time period listed an ejection fraction of 28%, a dilated left ventricle, and global hypokinesis. Subsequent stress tests were negative.

A physical examination prior to anesthesia revealed a Mallampati class 4 airway with a thyromental distance of less than 5cm. She was hypertensive in the pre-operative area, blood pressure 194/90 mmHg, and had crackles in her lung bases bilaterally with no improvement after coughing and deep respiration. No other abnormalities were noted on physical examination.

Pre-operative medication included midazolam, 1 mg IV. Pre-oxygenation was instituted for approximately five minutes. Induction was completed using lidocaine, propofol, rocuronium, and fentanyl. Intubation of the trachea was accomplished by direct laryngoscopy. Maintenance of anesthesia was achieved with desflurane, oxygen and N₂O. One-hundred percent oxygen was administered prior to repositioning. The patient was transferred from the gurney to an operating table with Wilson frame into the prone position. Immediately after repositioning endotracheal tube placement was reconfirmed. Proper head and upper body positioning was achieved utilizing the "prone view" head cushion, arm boards, and foam padding. Once positioning was secure and safety ensured the table was placed in approximately five to ten degrees of reverse Trendelenburg position. Padding and positioning were assessed repeatedly throughout the case. Phenylephrine and ephedrine were administered intermittently throughout the case to maintain a blood pressure of approximately 140/90 mmHg. A predetermined ideal mean arterial pressure (MAP) for this patient was set at no less than 70 mmHg.

Upon completion of the lumbar laminectomy the patient was returned to the supine position for extubation. Extubation criteria were met and the endotracheal tube was removed uneventfully. Prior to leaving the operating room the patient was awake, alert, and following verbal commands. A check for visual loss was conducted. The patient denied any visual disturbances and correctly identified a number of objects placed within her visual field. The patient had an uneventful post-operative course and was discharged to home without any apparent deficits related to surgical or anesthetic care.

Discussion

The ventral decubitus or prone position can put patients at risk for a multitude of long-term injuries to areas including the eyes, ears, breasts, neck, and brachial plexus. In addition, intra-operative complications can include cardiac and pulmonary compromise in certain patient populations. Visual loss is one of the greatest feared post operative complications. Factors that may increase risk include elevated intraocular pressure and decreased blood pressure. The prone position has been repeatedly shown to cause increased intraocular pressure (IOP).^{2,5,6} The cause of this increased pressure may be due to direct pressure on the globe and the additional pressure that is often present when a slight (5-10 degree) reverse Trendelenburg is not utilized.⁵ During this case the operating table was placed in approximately five to ten

degrees of reverse Trendelenburg to reduce the risk of increased IOP.

Hypotension may also play a role in visual loss when in combination with increased intraocular pressure.⁶ During the procedure special attention was taken to avoid hypotension. The patient did experience several fluctuations in blood pressure that were subsequently treated with either phenylephrine or ephedrine. The lowest recorded blood pressure throughout the case was 100/62 mmHg with an approximate average blood pressure over the six hour period of 138/70 mmHg.

The ears and breasts of patients can suffer injuries due to compression. Injuries to the breast may include severe stretching of the sternal borders or in extreme cases rupture of a prosthetic breast. A more favorable technique is maintaining the breasts cephalad and medial. Additionally, ensuring adequate padding under the breasts will prevent tissue necrosis in lengthy procedures. Similarly the ears should be positioned without folds or significant pressure. Brachial plexus injuries can occur with improper abduction and flexion of the arms during initial positioning or due to the patient sliding on the operating table in a cephalad or caudal direction. Avoidance of these injuries can be accomplished through adequate padding at the cubital tunnel, properly securing the patient to the operating table, and limiting the abduction as well as stretching at the glenohumeral joint which can lead to compression of the neurovascular bundle.⁶

Two other altered systems affected by the prone position include the cardiovascular and pulmonary systems. The shifting and compression of the abdominal contents in semi-restrictive frames, such as the Wilson frame, can cause increased inferior vena cava pressures as well as a decreased FRC and lung compliance.^{3,4} However, when the abdomen is allowed to hang freely such as with the Jackson table there is virtually no difference in FRC and lung compliance as compared with the supine position.⁴ The patient in this case was positioned with all of these factors in mind except for use of the Jackson table. The goal was optimal positioning without compromising safety or functional residual capacity (FRC). In this situation the patient was on a Wilson frame but did not exhibit any respiratory complications. Pulse oximetry averaged 99% over a six hour period with peak pressures ranging from 25-30 cmH₂O.

This case was a good learning opportunity for proper positioning and management of a patient in the prone position. It is important for the anesthesia practitioner to be aware of recommended guidelines for prone positioning and prepare accordingly. Proper technique along with vigilance in assessment of positioning and monitoring of oxygenation and blood pressure reduces the risk of patient injury and promotes a quicker recovery. In this case, visual loss and other injuries were avoided. Future research is needed to better understand which

individuals are at greatest risk for visual loss and the best approach to managing these patients in the prone position.

References

1. Epstein NE. Lumbar laminectomy for the resection of synovial cysts and coexisting lumbar spinal stenosis or degenerative spondylolisthesis: an outcome study. *J Neurosurg Spine* 2004;29:1049-1055.
2. Warner MA, Martin JT. Patient positioning. In: Barash P, Cullen B, Stoelting R. eds. *Clinical Anesthesia*. 4th ed. Philadelphia: Lippincott Williams & Wilkins;2001:655-658.
3. Pelosi P, Croci M, Calappi E, et al. Prone positioning improves pulmonary function in obese patients during general anesthesia. *Anesth Analg* 1996;83:578-583.
4. Palmon SC, Kirsch JR, Depper JA, Toung TJK. The effect of the prone position on pulmonary mechanics is frame-dependent. *Anesth Analg* 1998;87:1175-1180.
5. Ozcan MS, Praetel C, Bhatti MT, Gravenstein N, Mahla ME, Seubert CN. The effect of body inclination during prone positioning on intraocular pressure in awake volunteers: a comparison of two operating tables. *Anesth Analg* 2004;99:1152-1158.
6. Cheng, MA, Todorov A, Tempelhoff R, McHugh T, Crowder CM, Laurysen C. The effect of prone positioning on intraocular pressure in anesthetized patients. *Anesthesiology* 2001;95:1351-1355.

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Magnesium Sulfate Use in Pheochromocytoma Resection

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Pheochromocytomas are rare tumors that arise from chromaffin cells and are usually found in the adrenal medulla. Pheochromocytoma accounts for 0.1% of all cases of hypertension.¹ The cardinal symptoms include paroxysmal headache, sweating, and palpitations associated with severe hypertension. These predominately norepinephrine-secreting tumors, can act as potentially lethal catecholamine reservoirs. Resection has the potential for severe intraoperative and postoperative complications.²

Case Report

A 15 year-old, 47.7 kg female presented to the Emergency Department with tachycardia, severe hypertension, and a pounding headache. The patient was admitted and diagnosed with a right suprarenal mass. Phenoxybenzamine was ordered and later labetalol was added. Upon admission, the patient was also diagnosed with Von Hippel-Landau disorder. The patient's past medical history was significant for reactive airway disease. Family history revealed that her mother had a hemangioblastoma and two cousins had pheochromocytomas. The patient had no known drug allergies.

She underwent a renal ultrasound, MRI of the abdomen with contrast, and tumor imaging of the whole body. All were suggestive of pheochromocytoma. Urine laboratory tests revealed an elevated normetanephrine, total metanephrines, and vanillylmandelic acid levels.

The patient was admitted to the Pediatric Intensive Care Unit (PICU) for hydration to stabilize hemodynamics; a magnesium sulfate (MgSO₄) bolus

(1gm) and infusion (1 gm/hr) was initiated for 2 days prior to her scheduled surgery.

The preoperative airway assessment showed a Mallampati¹ airway and intact dentition. A general anesthetic with intravenous (IV) induction was planned along with a thoracic epidural for postoperative pain management. The teenager refused the epidural.

In the operating room after pre-oxygenation general endotracheal anesthesia was administered with propofol 150 mg, rocuronium 40 mg, and fentanyl 100 mcg. Her blood pressure remained stable between 100-130 mmHg systolic throughout induction. An additional peripheral IV, arterial line, and central venous line were placed.

Anesthesia was maintained with 1.2 percent isoflurane, 1gm/hr MgSO₄ and a sodium nitroprusside (SNP) infusion titrated between 0.5 to 2 mcg/kg/min. The patient's vital signs remained stable throughout the procedure (systolic 90-110's). However when the tumor was manipulated the blood pressure increased to 225/110 mmHg. Several boluses of 0.5 mg phentolamine and 5 mg of labetalol were given. The SNP infusion was increased to 2.5 mcg/kg/min and the magnesium sulfate infusion was increased to 1.5 gm/hr. The blood pressure returned to baseline after about 5 minutes and the tumor was successfully resected. The SNP and MgSO₄ infusions were titrated off and a 300 cc bolus of lactated Ringers was given.

The neuromuscular blockade was antagonized and the trachea was extubated in the operating room without incident. The patient's pain was controlled post-operatively with morphine patient controlled

analgesia(PCA) and subcutaneous lidocaine On-Q pump. The patient was discharged to home on postoperative day three.

Discussion

Von Hippel-Lindau disease (VHL) is a rare, genetic multi-system disorder characterized by the abnormal growth of tumors throughout parts of the body.³ The tumors form in the central nervous system (CNS) and are often benign. They may be comprised of highly vascularized beds of blood vessels called hemangioblastomas. Hemangioblastomas may develop in the brain, retina, or in other locations in the CNS. Endocrine tumors may develop in the adrenal glands, kidneys, or the pancreas. Patients with VHL disease often experience symptoms of dizziness, vision problems, headaches, problems with balance and walking, weakness of the limbs, and high blood pressure. These patients are at high risk for acquiring cancer, especially of the kidneys.⁴

Surgical and pharmacological treatment has evolved in the last decade for the treatment of pheochromocytomas.⁵ Adrenalectomies are increasingly being done utilizing laparoscopic techniques which may have adverse consequences associated with the creation of a pneumoperitoneum. This case was done as an open procedure due to surgeon preference for better exposure.

Patients who present with signs and symptoms of pheochromocytoma can develop a hypertensive crisis without rapid, aggressive treatment. The points of hemodynamic instability occur during induction, tumor manipulation, tumor ligation and resection, and postoperatively. When conventional therapy has failed, case reports show the use of MgSO₄ for hemodynamic control has been effective.⁶ MgSO₄ is predominately an arteriolar dilator which decreases peripheral vascular resistance.³ Magnesium ions inhibit the release of catecholamine from the normal adrenal medulla and adrenergic nerve terminals.⁷ Beta blocker use can generally be reserved for arrhythmias. Recommended dosing is to administer a loading bolus of 40-60 mg/kg followed by an infusion of 1-2 grams/hr.^{3,7} Blood levels should be monitored and maintained between 2-4

mmol/L, correlated with clinical symptoms. Magnesium is inexpensive, safe, and simple to use.

This patient was stable throughout the case with the exception of one episode of hypertension as the tumor was manipulated requiring multiple vasodilating agents. During this episode, phentolamine was not initially effective. It has been documented that tachyphylaxis often develops with phentolamine use.¹ Other literature reveals that many practitioners are now using nicardipine infusions or boluses as an adjunct to the MgSO₄ infusion.³ In this case MgSO₄ infusion, preoperative hydration and alpha adrenergic blockade with phenoxybenzamine, and direct acting vasodilators were titrated effectively to maintain stability.

References

1. Morgan G, Mikhail M, Murray M. *Clinical Anesthesiology*. 3rd ed. New York, NY: Lange; 2002:222-223, 747-748.
2. Kizer JR, Koniaris LS, Edelman JD, Sutton MG. Pheochromocytoma crisis, cardiomyopathy, and hemodynamic collapse. *Chest*. 2000;118:1221-1223.
3. Minami T, Adachi T, Fukuda K. An effective use of Magnesium Sulfate for intraoperative management of laparoscopic adrenalectomy for pheochromocytoma in a pediatric patient. *Anesth Analg*. 2002;95:1243-1244.
4. Van Poppel H, Nilsson S, Algaba F, et al: Precancerous lesions in the kidney. *Scand J Urol Nephrol Suppl* 2000; (205): 136-165
5. Joris J, Hamoir E, Hartstein, G, et al. Hemodynamic changes and catecholamine release during laparoscopic adrenalectomy for pheochromocytoma. *Anesth Analg*. 1999;88:16-21.
6. James MF, Cronje L. Pheochromocytoma crisis: the use of Magnesium Sulfate. *Anesth Analg*. 2004;99:680-686.
7. Prys-Roberts C. Pheochromocytoma—recent progress in its management. *Br J Anaesth*. 2000;85:44-57.

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Regional Anesthesia for Carotid Endarterectomy

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Carotid artery stenosis is an important cause of cerebrovascular accidents. Strokes are the third leading cause of death in the United States and cost \$14 billion yearly in medical expenses and lost productivity.^{1,2} The majority of strokes result from carotid atherosclerotic disease forming fibrous plaques which grow to compromise the vascular lumen.¹ Carotid endarterectomy is the 2nd most common vascular operation in the U.S.¹ and may be performed under general, regional, or local anesthesia. The following case report discusses anesthetic management of carotid endarterectomy under regional anesthesia.

Case Report

A 64 year-old male weighing 79 kilograms presented to the emergency department with right sided arm weakness for 3 days and intermittent mental confusion. His gait and ability to write had also deteriorated. Computed tomography revealed no masses or bleeding. A cerebral arteriogram confirmed critical stenosis of the left internal carotid artery with patent anterior and posterior communicating arteries. Heparin therapy was started and the patient was scheduled for left carotid endarterectomy.

The patient's past medical history included hypertension, hyperlipidemia, and tobacco use (although he had stopped smoking 30 years prior). Medications included simvastatin, lisinopril, and the current heparin therapy. Physical examination revealed a Mallampati class I airway, a thyromental distance of 3 fingerbreadths and full neck range of motion. His heart rate was 50-60 beats per minute with a systolic blood pressure between 160-190 mmHg. His lungs were clear to auscultation. Oxygen saturation was 98% while breathing 2 liters of oxygen via nasal cannula. Lab values were within normal limits preoperatively and his heparin therapy was within the established guidelines. An echocardiogram revealed normal left ventricular systolic function with an ejection fraction of 60%. A neurologic exam revealed cranial nerves grossly intact, symmetrical facial muscles and expressions, and right grip strength 4/5 and left 5/5. The patient exhibited intact sensation of all 4 extremities, denied visual changes, headache, or slurred speech. The patient denied having anesthetic complications during a previous hemorrhoidectomy.

The patient was taken to the operating room and oxygen was administered at 2 liters per minute via nasal cannula while monitors were applied. Fentanyl, 25 mcg intravenous (IV), was given while a radial arterial line was placed with 1% lidocaine for local anesthesia. An additional 25 mcg of Fentanyl was given IV for placement

of a left superficial cervical plexus block with 30 milliliters of 0.5% ropivacaine. This patient tolerated the procedure well. Cefazolin 1gram IV was given prior to incision at the request of the surgeon. During the case the oxygen saturation was maintained at 99-100% on 2 liters oxygen via nasal cannula. Phenylephrine and nitroglycerine drips were connected to the IV line, readily available to maintain hemodynamic stability. The patient's systolic blood pressure ranged from 110-220 mmHg intraoperatively. Five thousand units of heparin were given IV prior to occlusion of the carotid artery. The patient was then asked a series of questions, and his responses were monitored. The patient was also asked to perform motor tests, such as a hand grasp, to assess motor deficits. As the cross-clamp was applied the patient was repeatedly asked the series of questions to monitor for any changes in response or confusion. Grip strength was also monitored during this time. At one point the patient began to feel nauseated and hot. A precipitous decrease in his blood pressure from 180 to 110 systolic and decrease in heart rate from 70-50 beats per minute was noted and the phenylephrine drip was initiated, along with administration of glycopyrrolate 0.2 mg. He quickly responded to the medications and immediately began to feel better without further incident. Responses to questioning and grip strength remained unchanged throughout the procedure. Once the cross clamp was removed, the nitroglycerin drip was initiated to maintain systolic blood pressure 110-150 mmHg at the surgeon's request.

At the conclusion of the case, ondansetron 4 mg was given IV. The patient denied pain and the nitroglycerine drip was turned off. The patient was monitored in the neuro-intensive care unit for one night without incident. A tertiary exam revealed stable vital signs, intact cranial nerves, movement of all extremities, 5/5 grip strength on the left and right side and no other deficits. The patient was discharged to home on postoperative day 2 and continued with an uneventful postoperative course.

Discussion

Carotid endarterectomy is a surgical intervention to relieve the neurologic symptoms of carotid atherosclerosis and to prevent cerebral infarction with resulting permanent deficits.² There are multiple methods of anesthesia delivery which may be employed to facilitate a carotid endarterectomy. These methods include general, regional, or local anesthesia. Controversy does exist over which method, regional or general anesthesia, provides the better outcome. Regardless of the anesthetic chosen,

there are four goals to anesthetic management for carotid endarterectomy which include: 1) protect the brain and heart from ischemic injury; 2) maintain hemodynamic stability within the patient's autoregulatory range; 3) minimize the stress response to surgery; and 4) have an awake cooperative patient that is able to perform a satisfactory neurologic examination at the end of surgery.² There is some evidence from non-randomized studies that regional anesthetic techniques are associated with improved hemodynamic stability, possibly a lower rate of myocardial infarction, reduced intensive care unit utilization, and reduced cost.³

The primary advantage of regional techniques is an awake patient who can be neurologically evaluated intraoperatively for neurologic deficits. In the event neurologic symptoms occur, other interventions, such as temporary shunt placement may be indicated.⁴ Other advantages include the avoidance of expensive neurologic monitors, greater blood pressure stability, improved neurologic tolerance to carotid artery cross-clamping, decreased vasopressor requirements, and a decreased need for shunting.² Regional anesthesia is most often achieved by deep and/or superficial nerve block of cervical vertebrae 2-4. The advantages of a superficial nerve block are the ease of placement and avoidance of the phrenic nerve paralysis with possible respiratory compromise, that may complicate the deep block.² The superficial block may require local anesthetic supplementation by the surgeon. Small amounts of fentanyl, midazolam, or propofol may be given for block placement. Superficial cervical block can provide effective anesthesia and adequate surgical conditions comparable to a combined deep/superficial cervical block.⁵

There are multiple disadvantages to regional anesthesia for carotid endarterectomy. The primary limiting factor is patient cooperation. Anxiety and fear related to being awake during surgery compound the problem. Sedation is limited due to the amount of patient cooperation required for neurologic testing intraoperatively. Fear and apprehension can initiate sympathetic responses and result in altered hemodynamics.¹ Other disadvantages include the inability to use pharmacologic cerebral protection, the possibility of seizures or loss of consciousness with carotid cross-clamping, and poor access to the airway if

conversion to general anesthesia is required.² Complications due to block placement, including local anesthetic toxicity, phrenic nerve block, and superior laryngeal nerve block, are possible especially with administration of a deep cervical plexus block. Regional anesthesia for carotid endarterectomy may not be the best choice for the difficult airway patient due to possible conversion to general anesthesia during the procedure; however, the historical incidence of conversion from regional to general anesthesia is relatively low at 2-3%.²

There is no conclusive evidence at this time that regional anesthesia is safer than general anesthesia, however a large randomized study is currently underway.³ In the case reported, regional anesthesia was successful primarily due to administration of an adequate block and a cooperative patient, resulting in an acceptable surgical field. A patient with claustrophobia would not be a good candidate for carotid endarterectomy under regional anesthesia, and general anesthesia would most likely be the better choice. It is essential that patients are properly selected for regional anesthesia. Close communication between the nurse anesthetist and surgeon will provide for a safe and successful operative experience.

References

1. Nagelhout J, Zaglaniczny K. *Nurse Anesthesia*. 2nd ed. Philadelphia: WB Saunders Company; 2001:495-500.
2. Allain R, Marone LK, Meltzer J, Jeyabalan G. Carotid endarterectomy. *International Anesthesiology Clinics*. 2005; 43:15-38.
3. Baringer C, William JM, McCrirrick A, Earnshaw JJ. Regional anesthesia and propofol sedation for carotid endarterectomy. *ANZ J Surgery*. 2005; 75:546-549.
4. Morgan GE, Mikhail MS, Murray MJ. *Clinical Anesthesiology*. 3rd ed. NY:Lange Medical Books; 2002:470-472.
5. de Sousa AA, Filho MAD, Faglone Jr W, Carvalho GTC. Superficial vs. combined cervical plexus block for carotid endarterectomy: a prospective, randomized study. *Surgical Neurology*. 2005; 63:22-25.

Mentor: Michael Rieker CRNA, DNP

Endovascular Repair of Abdominal Aortic Aneurysm
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Approximately 40,000 patients have open repairs of abdominal aortic aneurysms (AAA) each year in the United States.¹ Perioperative mortality from elective open repair of AAA has declined over the past decades to 2%-4% at present.¹ Endovascular aneurysm repair involves the introduction of a prosthetic graft through the femoral artery.² There are numerous advantages of the endovascular technique, such as a decreased mortality rate as compared with open repair of AAA. This case study identifies the advantages of endovascular repair of an AAA on anesthetic management.

Case Report

An 83 year old, 79-kilogram female patient presented for an elective repair of a 6.3 cm infrarenal abdominal aortic aneurysm diagnosed through CT scan. The patient's medical history was significant for hypertension, Parkinson's disease, noninsulin dependent diabetes mellitus, chronic deep vein thrombosis and gastroesophageal reflux disease. The patient's surgical history included; drainage of a left kidney abscess at age 14. Her only medication was warfarin, which was held 2 days prior to surgery. Her significant laboratory findings were: glucose 122 mg/dl, BUN 35mg/dl and creatinine 1.9 mg/dl. Her preoperative blood pressure was 150/100 mmHg.

In the operating room she was preoxygenated by mask, after standard monitors were applied. General endotracheal anesthesia was induced with lidocaine 100 mg, fentanyl 150 mcg, etomidate 10 mg, rocuronium 50 mg. A 7.0-mmID oral endotracheal tube was inserted and desflurane was titrated in a 50% oxygen and air mixture. Muscle relaxation was maintained with cisatracurium. A total of 350 mcg of fentanyl was used for the entire procedure supplementing analgesia provided by the inhalation anesthetic.

A radial arterial catheter was placed which revealed a blood pressure of 164/72 mmHg. Intravenous metoprolol was incrementally administered for a total of 3 mg, which maintained the blood pressure between 98/50 mm/Hg and 122/64 mmHg. Cefazolin 1gm intravenous was administered prior to the start of the procedure.

The groin area was prepped bilaterally for surgery. A cutdown of both femoral arteries was performed. An 8-French sheath, guide wire, and pig-tailed catheter were then inserted. Arteriography was used to evaluate the renal, superior mesenteric and iliac arteries. Intravenous heparin 6,000 units were administered at the surgeon's request. The ACT was taken every 30 minutes, and results

maintained at 301-500 seconds. The blood pressure was maintained between 98/50 to 104/60 mmHg prior to insertion of the stent-graft at the left hypogastric artery. An angioplastic balloon was placed proximal to the graft to correct a type I endoleak. Prior to removing the sheath, intravenous protamine sulfate 50 mg was administered. The surgeon assessed the femoral circulation by doppler. Total volume of intravenous normal saline infused was 2000 ml; the estimated blood loss was 80 ml. At the end of the procedure intravenous ondansetron 4mg, neostigmine 4mg and glycopyrrolate 0.6mg were administered. The trachea was extubated when extubation criteria were met, 15 minutes after the completion of the procedure. The patient was taken to the Post Anesthesia Care Unit where she had an uneventful recovery, and was discharged to home in seven days.

Discussion

An aneurysm is a localized dilation of an arterial wall.³ Aortic aneurysms are classified as either abdominal or thoracic. The common sites for abdominal aneurysms are infrarenal and near the iliac arteries. The cause of aneurysms are multifactorial including atherosclerosis, genetic (e.g. hyperhomocysteinemia), inflammatory (e.g. polyarteritis), infectious (e.g. syphilis), vascular (e.g. Marfan's) mechanical (e.g. trauma) or hemodynamic (e.g. hypertension) and elderly males have a higher risk of development of abdominal aortic aneurysms (AAA).¹ Smoking exacerbates these other predisposing factors.¹

Diagnosis of AAA is made by physical examination, intravenous contrast CT scan, ultrasonography, MRI or aortogram. Treatment modalities include medical (e.g. control of blood pressure) or surgical interventions. Surgical treatments consists of open or endovascular repair. Elective repairs of AAA are done when the aneurysm diameter is > 6 cm, or expands > 0.5 cm in a six month period, or if the patient becomes symptomatic of aneurysm rupture.¹

Endovascular aneurysm repair involves the introduction of a prosthetic graft through the femoral arteries. The graft is fixed in place to the non-aneurysmal infrarenal neck and iliac arteries with a self-expanding or balloon-expandable stent rather than sutures.² The operating room should have the capability of angiography and portable radiological devices. Norris reports, "This approach can be undertaken without the large incisions, extensive dissections, prolonged aortic cross-clamp times and significant blood loss and fluid shifts associated with open aortic repair. Endovascular aortic repair is associated with greater hemodynamic stability, a reduced stress response, shorter

postoperative length of stay and improved analgesic control compared with open aortic repair.”¹

This approach can be done using local, regional or general anesthesia.⁴ An advantage of the procedure is an assumed reduced need for intraoperative fluid administration due to minimal blood loss and small incisions. Although minimal fluid administration is anticipated, two large gauge intravenous catheters should be placed to anticipate any bleeding complication.

Arterial blood pressure monitoring is imperative for accurate measurement and to facilitate frequent laboratory analysis such as arterial blood gas, ACT's, hemoglobin and potassium. Hypotensive technique (systolic blood pressure less than 100 mmHg) is recommended during insertion of the stent-graft to avoid migration.¹ Drugs commonly used in this technique are nitroglycerin or nitroprusside. Other drugs administered to maintain hemodynamic stability include phenylephrine and esmolol.

Due to the small incision and the use of local anesthesia by the surgeon at the operative site, the patient's opioid requirements are decreased. Prevention of hypothermia is a frequent concern. Warm IV fluids, upper body warming blanket and lower fresh gas flows can help to avoid this complication.

An endoleak is a complication specific to endovascular aortic repair. It is the inability to obtain or maintain complete exclusion of the aneurysm sac from arterial blood flow.¹ Endoleaks can be classified into four types, depending on the location of leak:

I - there is inadequacy of seal between endograft and aortic wall at the proximal or distal attachment of sites;

II - retrograde filling of aneurysm sac from patent lumbar, intercostals or inferior mesenteric arteries;

III - structural failure of endograft that allows blood to the aneurysm sac;

IV - related to the porosity of graft used.¹

When the endoleak occurs after the deployment of the stent-graft, then it is called primary. Secondary endoleaks refer to when the leak occurs after the initial seal.¹

Endovascular approach to repair AAA has several advantages from an anesthetic management perspective compared to open repair. Since patients with AAA are mostly elderly and with co-morbidities, minimal hemodynamic changes may occur as compared to an open repair contributing to a reduction in the use of vasopressors, fluids and opioid analgesics.⁵

References

1. Norris EJ. Anesthesia for vascular surgery. In: Miller RD, ed. Anesthesia. 6th ed. Philadelphia: Elsevier; 2005:2070-2092.
2. Townsend CM. *Sabiston Textbook of Surgery: The Biological Basis of Modern Practice*, 17th ed. Philadelphia: Elsevier Saunders; 2004: 1973-1978.
3. Bullock LB, Henze RL. *Focus on Pathophysiology*. Philadelphia: Lippincott; 2000: 440-441.
4. Parra JR, Crabtree T, McLafferty RB, et al. Anesthesia technique and outcomes of endovascular aneurysm repair. *Ann Vasc Surg*. 2005;19:123-129.
5. Ghansah JN, Murphy JT. Complications of major aortic and lower extremity vascular surgery. *Semin Cardiothoracic Vasc Anesth*. 2004;8:335-361.

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Hemoglobin Replacement in Ischemic Heart Disease

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Although guidelines for perioperative transfusion have been developed, no controlled studies have documented hemoglobin (HGB) concentrations at which transfusions improve postoperative outcomes or prevent myocardial ischemia or infarct.¹ In patients with ischemic heart disease (IHD) increased perioperative mortality is associated with HGB levels below 10 g/dL.² In vitro data suggest that peak oxygen-carrying capacity occurs at a hematocrit (HCT) of 30%, the value associated with HGB of 10g/dL in a normovolemic patient.¹ Higher HCT levels may be associated with increased blood viscosity and sluggish blood flow while levels acutely below 30%

may produce hypoxemia.¹ The decision to transfuse blood products in the patient with IHD combines balancing the risks of decreased oxygen carrying capacity against the risks of transfusion.

Case Report

A 75 year-old, 105 kg male patient with extensive left parotid squamous cell carcinoma was scheduled for left temporal bone resection, radical parotidectomy/neck dissection and cervical rotation versus free flap closure. The patient's past medical history included tobacco use

for more than 60 years, IHD, angina, congestive heart failure, hypertension, cerebral vascular accident, renal insufficiency, osteoarthritis and type II diabetes. He was maintained preoperatively on metformin, glyburide, felodipine, isosorbide, furosemide, KCl, nitroglycerin, naproxen, simvastatin, and metoprolol. The patient had a cardiac ejection fraction of 60% and an initial HCT of 37.6% (HGB 12.7). He was evaluated as an ASA IV.

After standard monitor placement, preoxygenation and sedation, induction of anesthesia was achieved via rapid sequence with fentanyl 500 mcg, etomidate 12 mg and succinylcholine 120 mg. Following induction, a central venous pressure (CVP) catheter and an arterial line were placed. Maintenance of anesthesia was achieved with isoflurane 0.6 to 1.3 MAC and hydromorphone titration. Intraoperative neuromuscular blockade was not utilized. The fraction of inspired oxygen (FiO₂) was maintained at 50% throughout the case and the patient's oxygen saturation remained between 97% and 100%. Intraoperative electrolyte levels remained within normal limits after 20 mEq potassium chloride was infused intravenously over one hour. Blood glucose levels did not require intraoperative pharmacologic intervention. One gram of IV calcium chloride was administered as an inotropic agent and to correct an ionized calcium level of 0.97 mmol/L. Cardiac leads II and V5 were monitored throughout the case for indications of myocardial ischemia. The patient's initial and final intraoperative hematocrits were 37.6% and 32.6% respectively with a nadir of 27.9% (HGB 9.0). The procedure lasted 10 hours and 15 minutes. Estimated blood loss for the procedure was 2100cc and urine output via indwelling catheter was 2500cc. Crystalloid infusion (plasmalyte) totaled 8000cc and colloid infusion (hespan) totaled 1000cc. Four units of packed red blood cells (PRBC) were also transfused.

At the end of the case the patient met extubation criteria and the endotracheal tube was removed. On the first postoperative day the patient's hematocrit was 32%. He remained under observation in the surgical intensive care unit though he was neurologically and hemodynamically stable and without ischemic EKG changes.

Discussion

The standard management for acute, significant intraoperative blood loss includes prompt cessation of bleeding and rapid restoration of intravascular volume with red blood cells as well as colloid and crystalloid solutions.¹ Despite this strategy to avoid perioperative anemia, it remains unclear which patients and under which conditions this approach will be of benefit.³ The relationship between degree of anemia and risks for myocardial ischemia in the surgical patient remains perplexing.³ This is due in part to the lack of a clear definition in the literature of anemia and also to a lack of information on the impact of perioperative anemia on clinical and functional outcomes.³

Some studies find no difference in mortality between patients with varying degrees of anemia while others suggest an increased chance of survival in patients with anemia.³ Confounding the situation further is that evidence from the Multicenter Study of Perioperative Ischemia Research Group indicates that the postoperative risk of myocardial infarction in patients undergoing coronary artery bypass is increased if the patient's HCT level is elevated.³ Additionally, Bombardini et al. retrospectively compared patients treated with intraoperative transfusion to those subjected to normovolemic hemodilution and found that the second group had lower mortality rates, primarily due to lower incidence of pulmonary thromboembolism.³ Conversely, a study of patients whose immediate postoperative HCT was <28% after radical prostatectomy, reported increased incidence of intraoperative and postoperative myocardial ischemia on electrocardiography.³ Despite these apparent contradictions, the preponderance of evidence suggests mortality in surgical patients is inversely related to HGB concentrations.³ The patients with the most severe anemia suffer the highest mortality rates.

The estimated preoperative blood volume of the patient in this case was 7,875 cc (75 cc/kg). The patient's initial HGB level was 12.7 g/dL (HCT of 37.6%). Allowable blood loss had been calculated at 1.6 L which would have maintained the patient's HCT at or above 30%. This value is associated with a HGB of 10 g/dL and the level below which mortality rates for patients with IHD are increased.² In light of the patient's history of IHD and the predicted procedural blood loss, aggressive fluid management and the need for intraoperative transfusion were anticipated preoperatively.^{1,4,5} CVP trends throughout the case guided fluid maintenance and volume replacement included that of actual blood loss which totaled 2100 cc. Four units of PRBC were transfused to maintain adequate oxygen carrying capacity once the patient's HCT dropped below 30%.

In healthy patients and those not on beta blocker therapy, the initial response to acute blood loss is a compensatory increase in cardiac output.³ The patient in this case was maintained preoperatively on beta blockade therapy which attenuates sympathetic activity and prevents compensatory hemodynamic responses to the effects of acute anemia.^{1,3} Additionally, when volume losses alone are restored with crystalloid, young healthy patients can tolerate very low HCT levels, however, the ability to compensate for blood loss may be hampered in patients under anesthesia, especially those with cardiovascular disease.³ Further, a distinction must also be drawn between the chronically anemic patient who may counteract low HGB levels with compensatory elevation in 2,3 diphosphoglycerate levels, which facilitate a rightward shift in oxy-hemoglobin dissociation, and the patient with severe acute blood loss.¹ While adequate oxygen carrying capacity may be corrected or maintained with intraoperative PRBC administration, transfusion may

also be associated with increased risks including those of perioperative thrombosis.

Guidelines for the intraoperative management of acute anemia in patients with IHD are not currently well delineated. This is due in part to the lack of a formal definition of anemia in the literature and also to a lack of current research on the impact of perioperative anemia on clinical, functional and quality of life outcomes.³ Therefore, the decision to transfuse blood products in the patient with IHD should be made on an individual, case by case basis. As the relationship between degree of anemia and risk for myocardial ischemia in the surgical patient remains unclear, the decision to administer blood products must cautiously weigh the risks associated with decreased oxygen carrying capacity against those of transfusion, including perioperative thrombosis.

References

1. Stoelting RK, Dierdorf SF. *Anesthesia and Coexisting Diseases*. 4th ed. New York: Churchill Livingstone; 2002:471–476.
2. Armas-Loughran B et al: Evaluation and management of anemia and bleeding disorders in surgical patients. In *Current Medical Diagnosis & Treatment*. 4th ed. New York: Lang Medical Books/McGraw-Hill; 2005:42.
3. Shander A, Knight K, Thurer R, Adamson J, Spence R. Prevalence and outcomes of anemia in surgery: a systematic review of the literature. *Am J Med*. 2004;116:58–69.
4. Prough DS, Mathru M. Acid-base, fluids and electrolytes. In Barash PG, *Clinical Anesthesia*. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2001:179.
5. Jaffe RA, Samuels SI. *Anesthesiologist's Manual of Surgical Procedures*. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2004:170.

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Hyperkalemia in the Patient with Alport's Syndrome **Jeanine Riddle, B.S.N.** Barnes Hospital College at Washington University Medical Center

Alport's syndrome (AS) is an inherited disorder of basement membranes arising from mutations in collagen, whose primary manifestation is progressive glomerulonephritis.¹ When fully manifested, chronic renal failure (CRF) ensues.² It is estimated to affect 1:5000 to 1:10000 people and accounts for 1% to 2% of end stage renal failure in the western world.³ There are currently no specific medical treatments for AS but genetic and pharmacologic therapy studies are being engineered and tested. Renal transplantation is usually successful, with a small percentage of patients developing anti-glomerular basement membrane nephritis of the allograft resulting in loss of the graft.¹

Case Report

A 37-year-old female, weighing 110.8 kg, height of 73 inches, ASA class III presented to the operating room for a creation of left arm AV fistula secondary to CRF. Past medical history included hypertension (HTN), hyperparathyroidism, end stage renal disease (ESRD), AS, and rectal cancer which was treated with radiation and chemotherapy. Past surgical history included Cesarean-delivery, tubal ligation, total abdominal hysterectomy, and excision of rectal cancer. Past social history included tobacco use of 1 ppd x 21 years. The patient denied alcohol and illicit drug use and reported no known drug allergies. Home medications included lisinopril, sodium bicarbonate, calcitrol and vitamin D. The preoperative physical exam was unremarkable

with normal vital signs. The airway was a Mallampati classification II, with a thyromental distance of 3 cm. The preoperative EKG and chest x-ray were normal. Laboratory values were all unremarkable with the exception of an elevated potassium (K⁺) level of 5.8 mEq/L.

An 18 g. peripheral IV was started in the holding area and D₅NS was started. At 0730, the elevated K⁺ level was treated with 50 mEq sodium bicarbonate, 25 gm dextrose and 6u regular insulin. Prophylactic antibiotic therapy was administered and the patient was premedicated with 2 mg midazolam prior to transport to the operating room. Unconscious sedation was the anesthetic plan of choice due to short duration and minimal invasiveness of the procedure. The patient received oxygen via nasal cannula and maintained stable vital signs throughout the procedure. The patient received a total of 700 mg propofol, 100 mg lidocaine, 150 mcg fentanyl, and 5 mg midazolam. At 0810, a blood glucose level was checked and shown to be 87 mg/dL. At 0830, a K⁺ level was checked and revealed a value of 5.9 mEq/L. Another specimen was sent to verify and confirm the results, which again showed a K⁺ level of 5.9 mEq/L. The elevated K⁺ was treated with another 50 mEq sodium bicarbonate, 25 gm dextrose and 6 u of insulin. A blood glucose level was checked at 0900 and was 88 mg/dL. At 0911, a repeat K⁺ level was sent and came back at 6.2 mEq/L. The surgeon was notified that the K⁺ level was not responding to treatment and the decision was made to admit the

patient for continued monitoring.

The patient was transported to the post anesthesia care unit in stable condition where she received intravenous fluids of .45NS at 100ml/hour, a single dose of 100mEq/L sodium bicarbonate for treatment of acidosis and 40mg furosemide for a K⁺ level of 6.2 mEq/L. She was then transferred to the floor for continued monitoring. All preoperative medications were resumed except for lisinopril. On post-operative day 1, her K⁺ level was 5.8 mEq/L and EKG was normal. By evening, the labs displayed a K⁺ level of 5.2 mEq/L. After continued monitoring with no adverse effects, she was discharged to home the following day with a K⁺ level of 5.2 mEq/L and instructions to follow-up with her primary physician.

Discussion

The kidneys are primary excretory organs and are instrumental in regulating fluids and electrolytes.⁴ When they cease to function normally, the body cannot excrete excess potassium, thus disrupting homeostasis. Hyperkalemia is defined as a serum potassium level greater than 5.0 mEq/L and can be lethal if left untreated. The most common cause of hyperkalemia is acute or chronic renal failure. The kidneys will excrete approximately 90% of the body's excess potassium, while the other 10% is eliminated through stool and perspiration. Signs and symptoms of hyperkalemia may include areflexia, muscle weakness and/or pain, hyperactive bowel sounds and diarrhea. Respiratory decompensation or failure may also occur, but unfortunately these signs and symptoms often go undetected until cardiac symptoms arise.⁵

Patients with AS are typically prescribed angiotensin-converting enzyme (ACE) inhibitors (lisinopril, enalapril) to lower intraglomerular pressure.⁶ However, these medications interfere with renal potassium excretion and may lead to hyperkalemia. The incidence of hyperkalemia with the use of ACE inhibitors appears to be relatively low, 0%-6% in patients with normal renal function; however, in patients with renal insufficiency it can be as high as 5%-50%.⁸ Unfortunately, there is little information to guide monitoring and treatment of patients taking ACE inhibitors who have renal insufficiency. It is suggested that patients with renal insufficiency are at increased risk for hyperkalemia, and should either have their potassium levels judiciously monitored on a regular basis or reconsider using the ACE inhibitor.⁷

It is recommended that hyperkalemia be treated if serum levels are greater than 5.5mEq/L prior to elective surgery. The standard treatment regimen for hyperkalemia consists of 10-20 ml of 10% calcium gluconate IV, which antagonizes the effects of potassium on the heart, 50-100 mEq of sodium bicarbonate IV, and 25-50g of glucose with insulin, 10-20 units IV, to facilitate the movement of potassium from plasma into the cells. These are only temporary measures for treating

hyperkalemia because they only redistribute, not eliminate the potassium from the body. If the patient does not respond appropriately and displays life threatening clinical symptoms such as prolonged PR intervals progressing to loss of P waves, widening QRS complexes and peaked T-waves, dialysis should be considered. Prolonged progressive hyperkalemia can lead to ventricular tachycardia and fibrillation, with the most likely cardiac event being asystole.⁶

Managing the renal impaired patient intraoperatively can be challenging. Therefore, it is important to understand that they have the ability to adapt to changes by increasing their secretion of K⁺, thus allowing a better tolerated and more gradual development of hyperkalemia.⁶ Therefore, the K⁺ level should not be the only clinical determining factor for initiation of treatment. Treatment of hyperkalemia should be initiated in the presence of EKG changes such as peaked narrow T-waves, a prolonged PR interval, disappearance of P waves and a widened QRS interval.⁵ Tachycardia may also be seen early, while bradycardia may be seen later.⁵ In this case, the patient did not exhibit any EKG changes, but due to her initial K⁺ level being greater than 5.5 meq/ml, the decision was made to administer treatment.

It is also important to know what medications can impair renal elimination of K⁺ or can cause transcellular shifts of K⁺. Medications used during the perioperative period that may cause a change in the K⁺ level include heparin, nonsteroidal anti-inflammatory drugs, ACE inhibitors, potassium sparing diuretics and cyclosporine. Beta-blockers, depolarizing muscle relaxants, barbiturates, opioids, digitalis preparations, and reductase inhibitors can also contribute to elevated K⁺ levels, due to their ability to shift K⁺ out of cells thus increasing circulating K⁺.⁵

In summary, there are many factors that play a role and may contribute to hyperkalemia in the renal impaired patient. Hyperkalemia can be life threatening and it is therefore imperative that the anesthesia professional is aware of the causes of hyperkalemia and initiate meticulous monitoring modalities during the perioperative period to prevent associated complications.

References

1. Kashtan CE. Alport syndrome: an inherited disorder of renal, ocular, and cochlear basement membranes. *Medicine*. 1999;78:338-360.
2. Robbins SL, Cotran RS. *Pathologic basis of disease*. 7th ed. Philadelphia: Elsevier Saunders; 2005:988.
3. Pirson Y. Making the diagnosis of alport's syndrome. *Kidney Int*. 1999;56:760-775.
4. Susan-Simmons H. Evaluating chronic kidney disease risk. *Nurse Pract*. 2005;30:12-25.

5. Burger CM. Hyperkalemia: when serum K⁺ is not okay. *Emergency*. 2004;104:66-70.
6. Stoelting RK, Dierdorf SF. *Anesthesia and Co-existing Disease*. 4th ed. Philadelphia: Churchill Livingstone; 2004:346, 361-362, 383-384.

7. Reardon LC. Hyperkalemia in outpatients using angiotensin-converting enzyme inhibitors: how much should we worry. *Arch Intern Med*. 1998;158:26-32.

Mentor: Vicki C. Coopmans, CRNA, PhD

Prolonged Apnea Following Succinylcholine **Matthew L. Toomey, B.S.N.** Virginia Commonwealth University

Succinylcholine is the standard muscle relaxant used with rapid sequence induction of anesthesia. It remains the drug of choice for this type of induction due to its rapid onset of action and short duration of muscle paralysis. However, prolonged apnea is a potential complication in patients with decreased or abnormal plasma cholinesterase. The following topics are discussed in this article: physiology of plasma cholinesterase, identification of co-morbidities that can disrupt the enzyme, interventions if clinical issues manifest, and follow-up care.

Case Report

A healthy 62 year old (72 kg), ASA 2 female was scheduled for a hysteroscopic myomectomy for an endometrial myoma. The patient had a medical history of anemia, borderline hypertension, and gastro-esophageal reflux. The patient was on multiple-drug therapy for control of her reflux. The patient also required several pillows at night to elevate her head for reflux prevention. The patient's past surgical history included a repair of a hiatal hernia with general anesthesia. The patient's current medications included lansoprazole for esophageal reflux, calcium carbonate, and a multiple vitamin tablet. The patient had no known drug allergies. Metabolic panel and complete blood count were normal except for a hemoglobin count of 10.2 g/dL. The patient had nothing by mouth for nine hours and had not taken any medications the morning of surgery.

During the pre-anesthetic workup, the patient stated that she "took a long time recovering from anesthesia," following her previous surgery, but denied any knowledge of requiring ventilator support post-operatively. The patient denied any known family complications with anesthesia. The decision was to proceed with a general anesthetic using a rapid sequence induction.

An 18 gauge peripheral intravenous catheter was placed in the pre-operative holding area, and the patient was premedicated with metoclopramide 10 mg, ranitidine 50 mg, and midazolam 2 mg prior to entering the operating room. Upon arrival to the operating room, standard monitors were applied. All vital signs were stable, and the patient was pre-oxygenated for five minutes. Anesthesia was induced using a rapid sequence technique with lidocaine 80 mg, fentanyl 150 mcg, propofol 150 mg,

succinylcholine 80 mg, and cricoid pressure was initiated. Direct visual laryngoscopy was performed and a 7.5 mmID endotracheal tube was placed without difficulty. Anesthesia was maintained with a mixture of oxygen, air, and sevoflurane.

Ten minutes after induction, muscle relaxation was assessed using a peripheral nerve stimulator on the facial nerve and no response was noted. At 20 minutes post-induction, neuromuscular function was reassessed and remained absent, and the possibility of abnormal plasma cholinesterase was discussed. The peripheral nerve stimulator was then checked for proper functioning and found to be adequate. At 35 minutes into the case, the surgeon announced conclusion of the procedure and the patient remained paralyzed and ventilator dependent. The patient remained on ventilator support and volatile anesthetic agent to ensure amnesia. Neuromuscular function was evaluated every 10 minutes for response to peripheral nerve stimulation. Fifty minutes after induction of anesthesia the patient regained spontaneous ventilation and demonstrated full recovery of the train-of-four. Sevoflurane was discontinued and the patient was given 100% oxygen in preparation for extubation. The patient's respiratory rate remained at 12 breaths per minute with tidal volumes of 400 ml. Sustained head lift on command was observed, SpO₂ remained at 100% and the patient denied any shortness of breath. The trachea was extubated, and the patient was given 100% oxygen via facemask. The patient was subsequently transferred to the post-anesthesia care unit without incident. Labs were sent which revealed a pseudocholinesterase value of 852 units/liter (normal range 1000-3500 units/liter), and a dibucaine number of 45% (normal 73-80%). Education was provided regarding pseudocholinesterase abnormality and the need to inform future anesthesia professionals of this diagnosis. The patient was admitted to the hospital for 24-hour observation and subsequently discharged to home.

Discussion

Standard rapid sequence induction for intubation of the trachea includes a muscle relaxant with rapid onset and short duration of action. Succinylcholine displays these characteristics under normal circumstances. Prolonged relaxation following succinylcholine administration may

be a result of either a decreased amount or decreased activity of plasma cholinesterase.

There are two main types of enzymes responsible for the metabolism of choline esters. The first, true cholinesterase, or acetylcholinesterase, is found in red blood cells, nerves and muscles and is highly specific for hydrolyzing acetylcholine.¹ The other, plasma cholinesterase, also known as nonspecific cholinesterase or pseudocholinesterase, as its name implies, is predominantly found in the plasma. This type of cholinesterase is capable of metabolizing both aliphatic esters and choline, such as that found in succinylcholine.²

Plasma cholinesterase abnormalities can result in delayed metabolism of several compounds used in anesthesia including succinylcholine, mivacurium, procaine, and cocaine.² In the case of succinylcholine and mivacurium, this can lead to prolonged skeletal muscle relaxation and apnea.

There are several causes of variant pseudocholinesterase enzymes. Inherited variations include mutations of the expression of normal plasma cholinesterase alleles causing a lack in the natural ability to hydrolyze ester bonds.^{2,3} Homozygous atypical plasma cholinesterase involves mutations of both alleles. Heterozygous atypical plasma cholinesterase only involves mutation of one allele.³

In patients carrying the homozygous form of atypical pseudocholinesterase, there is a profound decrease in enzymatic function leading to prolongation of muscle relaxation lasting greater than one hour following succinylcholine administration. The frequency of this form ranges from 1:3,000 to 1:10,000 individuals.³ With the heterozygous atypical enzyme, there is still enough hydrolyzing capability that only patients with concurrent illnesses or a decrease in total quantity of enzyme will generally show sensitivity to succinylcholine. In this case, prolongation of paralysis will last greater than 5 minutes, but less than one hour. The frequency of this form is more common, ranging from 1:25 to 1:480.³

A number of conditions and predisposing factors may alter the activity of pseudocholinesterase including liver disease, malignancy, malnutrition, heart disease, renal disease, burns, oral contraceptives, estrogen therapy, and pregnancy.² There is a 20% decrease in plasma cholinesterase activity in the first trimester of pregnancy, and a 33% decrease shortly preceding delivery with normal levels returning by the 4-6th postpartum week.⁴

Management of patients with succinylcholine sensitivity and prolongation of muscle paralysis consists of sedation and ventilatory support until neuromuscular function naturally returns. Frequent assessment using peripheral nerve stimulation is essential and can be used to rule out other causes of apnea such as opioid overdose.^{2,3} Practitioners should also confirm recovery of neuromuscular function before administering a

non-depolarizing muscle relaxant following succinylcholine to confirm normal recovery. Some measures to accelerate recovery consist of transfusing blood or fresh frozen plasma. Plasma cholinesterase remains active in these products and is not destroyed in the process of blood bank preparations.⁵ However, this method is discouraged due to the risk of blood borne diseases and/or complications related to transfusion.

The most frequently used diagnostic indication of the atypical form of pseudocholinesterase is by the use of the local anesthetic dibucaine.^{2,3} Dibucaine inhibits the enzymatic activity of normal pseudocholinesterase by 80%. The “dibucaine number” (DN) measures the quality of normal pseudocholinesterase activity. Thus, a DN of 80% represents normal pseudocholinesterase activity, a DN of 40%-70% represents the heterozygous variant, and a DN <30% represents the atypical homozygous variant.³ The patient in this case report demonstrated a dibucaine number of 45%. This value, along with an apnea time of 50 minutes, confirms the diagnosis of heterozygous atypical pseudocholinesterase.

It remains essential for anesthesia professionals to perform a thorough preoperative evaluation to optimize the anesthetic plan for each patient. For some patients, it is important to weigh the risks and benefits of anesthetic techniques with individual patient co-morbidities. This case demonstrated the decision to pursue a plan based upon a certain co-morbidity (reflux disease), and the consequence that inadvertently followed. Early recognition of the problem, initiating the necessary treatment interventions, and proper follow-up care for the patient is essential. Further investigation of this patient's prior anesthetic (recall she mentioned she “took a long time recovering from anesthesia”) may have identified the plasma cholinesterase abnormality and a different anesthetic plan could have been instituted.

References:

1. Augustinon DB. Cholinesterase: a study in comparative enzymology. *Acta Physiol Scand.* 1948;15:1-182.
2. Davis L, Britten JJ, Morgan M. Cholinesterase. *Anaesthesia.* 1997;52:244-260.
3. Kalow W, Straron N. On distribution and inheritance of atypical forms of human serum cholinesterase as indicated by dibucaine numbers. *Canadian Journal of Biochemistry and Physiology.* 1957;35:1305-1317.
4. Blitt CD, Petty SC, Alberternst EE, Wright BJ. Correlation of plasma cholinesterase activity and duration of succinylcholine during pregnancy. *Anesth Analg.* 1977;56:78-81.
5. Lanks KW, Sklar GS. Stability of pseudocholinesterase in stored blood. *Anesthesiology.* 1976;44: 428-430.

Mentor: Chuck Biddle CRNA, PhD

Identifying Aspiration Risk in Children with Cerebral Palsy

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Cerebral palsy is a disorder of control of movement and posture, appearing early in life secondary to a central nervous system (CNS) lesion or dysfunction that is not the result of a recognized progressive or degenerative brain disease.¹ Lack of oxygen to the brain or trauma to the head during labor and delivery are known causes of cerebral palsy.² Cerebral palsy is classified according to the extremity involved and the characteristics of the neurologic dysfunction. Even though the exact cause of cerebral palsy is unknown, it is assumed that problems during birth are major contributors.³ Midforceps delivery and low Apgar scores are factors that are associated with the development of cerebral palsy. Common manifestations of cerebral palsy include skeletal muscle spasticity, choreoathetosis, dystonia, cerebral ataxia, mental retardation, and speech defects. Patients often develop contractures and fixed deformities, which may require orthopedic surgery to help lessen the deformity.³ Patients with cerebral palsy frequently have dysphagia, gastroesophageal reflux, delayed gastric emptying and reduced bowel motility.⁴ In addition, many patients have associated conditions such as, recurrent upper respiratory tract infections, and anemia that may affect their anesthetic management.⁵ Due to aspiration risks, these children are managed with tracheal intubation and extubation should be delayed until these children are fully awake.

The following is a case report of a child presenting with suspected cerebral palsy who underwent orthopedic surgery of his left wrist. The intended surgery was to release a severe contracture and fuse the wrist in a natural resting position. This surgery was intended for both cosmetic and functional reasons.

Case Report

A 15-year-old, 55 kg, ASA II, male with a suspected history of cerebral palsy resulting from a brain hemorrhage at birth, displayed severe contractures and paralysis of the left wrist. He presented for a radial carpal fusion of the left upper extremity. There were no labs, no medications, nor previous surgical history for this patient. The patient and his parents denied history of gastroesophageal reflux, pulmonary complications, or diminished airway reflexes. The patient did not display signs of mental retardation nor speech defects. However, the airway examination identified a Mallampati class III and a narrow mouth opening of only two fingerbreadths. The thyroid-mental distance was three fingerbreadths. He had full range of motion, but difficulty sticking out his tongue during the oral examination.

The parents consented for general anesthesia. Premedication included midazolam 1 mg intravenously

(IV). Induction included fentanyl 100 mcg IV followed by lidocaine 100 mg, propofol 100 mg, and rocuronium 40 mg. The patient was easily ventilated while waiting for the neuromuscular blocker to take effect. A 7.5 mmID, cuffed endotracheal tube (ETT) was appropriately placed under direct laryngoscopy, which revealed a Grade I view. The cuff was inflated and tracheal placement of the ETT was confirmed. During surgery, the patient's anesthetic was maintained with isoflurane and a 60:40 mixture of nitrous oxide and oxygen.

Approximately one and a half hours prior to the end of the surgery, the patient's train-of-four displayed 4 strong twitches and sustained tetany. After the procedure ended, all anesthetic gases were discontinued and oxygen was administered at 10 liters per minute. The patient displayed signs of stage II anesthesia (disconjugate gaze, rapid and irregular breathing) followed by attempts to open his eyes and purposeful arm movements. Preparation to extubate the patient was initiated by removing the tape that secured the ETT. Immediately the patient vomited about 50 milliliters of greenish bile appearing fluid. He was turned to his side and his oral pharynx was suctioned. After the airway was cleared, the ETT was deflated and the trachea was extubated without signs of respiratory distress. The patient was immediately taken to the post anesthesia care unit for recovery and was released to home the same day.

Discussion

Many children with cerebral palsy have coexisting conditions that place them at increased risk for aspiration. Gastroesophageal reflux, delayed gastric emptying, recurrent upper respiratory tract infections, and anemia are a few conditions identified that will affect the anesthetic management of these patients.⁵

Patients with cerebral palsy should be identified as having higher risk for aspiration and precautions to prevent an occurrence should be initiated. The administration of an antiemetic acting on one receptor type typically reduces the incidence of postoperative nausea and vomiting (PONV) by about 30%, whereas the use of a combination of antiemetics acting on different receptors can further reduce this incidence.⁶ In this case, the patient was suspected to have a mild form of cerebral palsy that occurred at birth. He did not display any other signs for a higher risk of aspiration. Premedication to prevent aspiration was not initiated and the patient was taken to surgery. A successful, controlled intubation was performed without rapid sequence. During emergence, the patient began to show signs that he could be extubated when he suddenly vomited. Fortunately, the ETT was in place and the cuff was inflated.

Even though the child did not aspirate in this situation, the propensity for aspiration was likely. Patients with cerebral palsy should be considered an aspiration risk because of the likelihood of gastroesophageal reflux and delayed gastric emptying. The use of combination antiemetic therapy includes choosing medications that work at different receptor sites known to cause nausea or vomiting such as a dopamine antagonist, serotonin antagonist, and a corticosteroid.⁶

Precautions that reduce the occurrence or severity of aspiration include: the use of medications to lower gastric pH, implementing a rapid sequence induction with cricoid pressure, and ensuring that the patient is fully awake with protective airway reflexes prior to extubating them after general anesthesia.

References

1. Ashwal S, Russman BS, Iasco PA, et al. Diagnostic assessment of the child with Cerebral Palsy: Report of the quality standards subcommittee of the American Academy of Neurology and the practice committee of the Child Neurology Society. *Neurology*. 2004; 62: 851.
2. Cerebral Palsy help. Cerebral Palsy: causes & symptoms. Available at: <http://www.cerebralpalsyhelp.com/cp.html>. Accessed October 15, 2005.
3. Stoelting RK, Dierdorf SF. *Anesthesia and Co-Existing Disease*. 4th ed. Philadelphia: Churchill Livingstone; 2002:702-703.
4. Register BC, Hansel DE, Hutchins GM, Levey EB, Sponseller PD, Leet AI. Postoperative gastric rupture in children with Cerebral Palsy. *J Pediatr Orthop*. 2005; 25:280.
5. Spiroglou K, Xinias I, Karatzas N, Karatzas E, Arsos G, Panteliadis C. Gastric emptying in children with Cerebral Palsy and gastroesophageal reflux. *Pediatr Neurol*. 2004; 31:177-182.
6. Gan TJ. Postoperative nausea and vomiting- can it be eliminated? *JAMA*. 2002;287: 1233-1236.

Mentors: Ramona Domen-Herbert, LCDR, NC, USN, CRNA, MSN

Joseph F. Burkard, CDR, NC, USN, CRNA, DNSc

Latex Allergy Management During Cesarean Delivery

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Anaphylactic reactions in patients with known history of latex allergy can be severe, but particularly alarming are the reports of this reaction in patients with no known history of the allergy.¹ The frequency of latex allergy is < 1% in the general population but is 5-17% among health care workers and as high as 30-60% among patients with central nervous system malformations.¹ Symptoms of latex allergy may progress rapidly and unpredictably to anaphylaxis.² It is essential to know the signs of anaphylaxis and be prepared for the unexpected arrival of these life-threatening symptoms. The rate of sensitization has been increasing since its first recognition in 1979, and the first indication of this allergic reaction is frequently being seen by anesthesia professionals.

Case Report

A 37-year-old female, 5'6", 75 kilogram, G₃P₃, presented for elective repeat cesarean delivery at 39-weeks gestation. The patient's past medical history included sickle cell trait and uterine fibroids. Her past surgical history included three previous cesarean sections. She denied any allergies, but mentioned during her last cesarean delivery, 4-years ago, she experienced perioperative respiratory arrest with intubation and admission to the ICU postoperatively. She attributed this to an allergic reaction to an antibiotic given to her, but she admitted taking this medication since then without any allergic symptoms.

Upon arrival in the operating room standard monitors were applied and oxygen was administered via nasal cannula. Spinal anesthesia was induced in the sitting position at L₂-L₃ level with 13 mg 0.75% bupivacaine with fentanyl 20 mcg, and morphine 0.15 mg. She was then assisted to a supine position with left uterine displacement. Her blood pressure was monitored every 1-3 minutes and it was maintained with ephedrine in 5-10 mg increments, totaling 20 mg during the procedure.

A T₆ level of spinal anesthesia was achieved prior to skin incision. Immediately after the delivery of the baby, the surgeon requested 20 units of oxytocin in the intravenous fluid and to forego any antibiotics. During the uterine repair, with the uterus out on the abdomen, the patient began to experience chest heaviness, dyspnea, and restlessness, with decreasing depth and rate of respirations. She had facial flushing and redness on her neck, hands and arms. The EKG showed sinus tachycardia at 140 bpm, her BP 89/52 mmHg and her oxygen saturation decreased to 88%.

The pulse oximetry continued to rapidly decrease to 82%. Supplemental oxygen at 100% was provided by facemask with assisted ventilation. Oxygen saturation immediately improved to 91%. Bilateral breath sounds were decreased with wheezing heard throughout.

Diphenhydramine 25 mg IV, epinephrine 0.2 mg SQ, and a repeat dose of diphenhydramine 25 mg IV was given for suspected latex allergy. The Foley catheter was removed and was replaced with a silicon catheter; the surgeon's gloves were replaced with latex free gloves and all latex containing products were removed from the immediate surgical field.

Within 15 minutes from the onset of symptoms her oxygen saturation improved to 96%, vital signs returned to baseline and on auscultation of her lungs minimal wheezing was heard. The flushing of her face and redness of the chest and upper extremities began to fade. With hemodynamic status stabilized, surgery was completed without further incident.

Prior to transfer to the recovery room her oxygen saturation remained 97% on room air. Upon admission to the recovery room, she was given 3L/min of nasal oxygen, resulting in an oxygen saturation of 100%. The patient and her husband were notified that she had a latex allergy and medical follow-up would be necessary.

Discussion

When latex touches mucosa, as opposed to intact skin, the chance of developing an allergy increases significantly. Mucosal tissues are only one-twelfth as thick as the skin and lack the skin's ability to repel the latex. The risk increases with mucosal contact with latex gloves during surgery and direct contact with urinary catheters used routinely in surgery.³ In this case, the patient began to show symptoms of a latex-induced hypersensitivity reaction once the uterus was externalized on the abdomen and repair had begun.

High-exposure areas, such as operating rooms and labor and delivery suites where powdered latex gloves are used, contain sufficiently elevated concentrations of aerosolized latex to produce significant symptoms in sensitized persons.²

Latex may cause an immediate hypersensitivity reaction varying from contact urticaria to systemic anaphylaxis with laryngeal edema that requires lifesaving intervention.⁴ The amount of exposure needed to cause sensitization or symptoms is not known. This condition was identified in the 1930s, it is not well understood, difficult to definitively diagnose and most importantly, difficult to eliminate from the numerous products which are the sources of the allergens.⁵

The most effective strategy in the treatment of latex allergy is avoidance; however, there is a large group of sensitized people who have not been identified and who do not recognize that their symptoms are caused by latex allergy.² A complete and thorough medical history remains the most reliable screening test to predict the likelihood of an anaphylactic reaction. Once the hypersensitivity reaction begins, immediate treatment must be initiated to prevent further progression.⁵

Initial therapy is to stop administration and reduce absorption of the antigen: remove all latex from the surgical field, change gloves, discontinue all antibiotic and blood administration, maintain the airway and administer 100% oxygen, intubate the trachea if indicated, continue fluid administration with crystalloid or colloid, administer epinephrine (IV, SQ, endotracheal), and discontinue all anesthetic agents.⁶

Secondary therapy includes: diphenhydramine 50 mg maximum dose, ranitidine 50 mg maximum dose, glucocorticoids (hydrocortisone or methylprednisolone), administration of aminophylline for bronchospasm (loading dose, followed by infusion), administer inhaled β_2 -agonist for bronchospasm, and administer continuous catecholamine infusion for blood pressure support.⁶

History provided by this patient was our initial clue of a perioperative allergic reaction. The initial thought was that it was an antibiotic reaction, but this proved untrue when the patient stated she had taken this antibiotic since that time, on several occasions. During this elective cesarean delivery, the symptoms reappeared without any antibiotic use and the onset of respiratory distress with bronchospasm progressed quickly before the latex protocol was enacted. The use of the protocol provided a steady improvement in the patient's respiratory distress and other allergic symptoms. The early identification and treatment of this life-threatening hypersensitivity provided the patient with the best possible outcome.

References

1. Santos R, Hernandez-Ayup S, Galache P, et al. Severe latex allergy after a vaginal examination during labor: a case report. *Am J Obstet Gynecol* 1997;177:1543-1544.
2. Reddy S. Latex Allergy. *American Academy of Family Physicians*. 1998;57. Available at: <http://www.aafp.org/afp/980101ap/reddy.html>. Accessed October 30, 2005.
3. Lenehan GP. Latex allergy: separating fact from fiction. *Travel Nursing 2004 Supplement* 2004;34:12-18.
4. AANA Latex Protocol. Available at http://www.latexallergyresources.org/resourcemanual/section4/aana_latex_protocol.cfm. Accessed October 30, 2005.
5. Thompson R. Chemical Allergy "The other Latex Allergy. Available at http://www.latexallergyresources.org/resourcemanual/section8/source_to_surgery-1298.cfm. Accessed October 30, 2005.
6. Holzman RS. What is the optimal perioperative management for latex allergy? In: Fleisher LA, ed. *Evidence-based practice of anesthesiology*. Philadelphia, Saunders; 2003:111-116.

Mentor: Art Zwerling, CRNA, MS, MSN, FAAPM

Circuit Obstruction Due to Absorber Wrapping
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Up to 33% of critical incidents during anesthetic administration may be attributed to a failure to properly check the equipment.¹ A meticulous anesthesia machine check should be performed, as outlined by the Food and Drug Administration (FDA), at the beginning of each clinical day.² Any changes causing a potential loss of integrity to the circle system, such as replacing the carbon dioxide absorbent, changing the breathing circuit, or adjustments to the inspiratory and/or expiratory flow valves, also warrant a machine assessment.

Case Report

An 89 year-old male (66kg, 157.5cm), ASA status II, presented for a reverse right total shoulder secondary to end-stage degenerative changes of the glenohumeral joint. The patient's medical history included osteoarthritis resulting in various surgical interventions and prostate cancer. He reported a remote smoking history of 20 pack years, but he stated he quit 60 years ago. He denied any cardiac or pulmonary history. His medication regimen at the time included acetaminophen, donepezil hydrochloride, and propoxyphene napsylate. He reported an allergy to codeine.

Preoperatively, the patient's vital signs were within normal parameters. Laboratory studies included serum metabolic panel, complete blood count, and coagulation evaluation. A urinalysis was also performed. All values were within normal limits with the exception of the red blood cell count (3.99 million/microliter), hemoglobin (12.9 grams/deciliter), and hematocrit (38.3%). An electrocardiogram revealed a normal sinus rhythm (73 beats per minute) with a right bundle branch block. Chest x-ray results showed bilateral lung calcifications but no infiltrates or effusions.

The patient was interviewed and past medical documents were reviewed as well as current studies. Airway assessment revealed an edentulous patient with a Mallampati class I airway, thyromental distance of three fingerbreadths, and full range of motion of his neck. The anesthetic plan was discussed with the patient and was agreed upon. The plan was for a general endotracheal anesthetic after the placement of an interscalene block (for postoperative analgesia). The patient was given midazolam 1mg for anxiolysis. A 22 gauge stimulator needle with a peripheral nerve stimulator was utilized for identification of the brachial plexus. Thirty milliliters of ropivacaine (0.5%) was injected in divided doses of 5 milliliters each. The patient tolerated the procedure well, and his vital signs remained stable throughout.

A Drager® Narkomed® 4 anesthesia machine was checked per FDA guidelines for use during the surgery.

The patient was transported to the operating room. Standard monitors were applied. Preoxygenation was initiated with 100% oxygen via face mask. Anesthesia was induced utilizing 50 mcg of fentanyl and a total of 120 mg of propofol in divided doses. The patient was then given 40 mg of rocuronium for intubation. The patient was found to be difficult to mask ventilate, but adequate tidal volumes were maintained and end-tidal carbon dioxide was present. After 90 seconds of manual ventilation, laryngoscopy was performed. A laryngoscopic grade I view of the cords was obtained using a Macintosh 3 blade. An 8.0 mmID endotracheal tube was inserted orally under direct visualization. Bilateral breath sounds were verified and end tidal carbon dioxide was noted on the monitor. Inspiratory sevoflurane was administered at 1.25%. The vital signs remained within normal limits throughout induction and intubation. The oxygen saturation was maintained at 100% despite difficulty with mask ventilation.

Mechanical ventilation of the patient's lungs was initiated. Initial settings were 96% oxygen concentration, respiratory rate of 12 breaths per minute, tidal volume of 0.51 liters, and an inspiratory to expiratory ratio of 1:2 seconds. Inspiratory flow levels were set to moderate. Peak inspiratory pressures measured 39 to 41cm H₂O. Four puffs of albuterol were given via the endotracheal tube as it was thought the patient may be experiencing a bronchospasm despite a lack of wheezing. Surgical preparation of the patient proceeded with taping of the patient's eyes, placement of an eye protector, and insertion of an esophageal stethoscope/temperature probe. After four minutes of preparation, a decision was made to obtain a chest film. Peak inspiratory pressures remained at 41cm H₂O. Oxygen saturation continued to read 100%. Placement of the endotracheal tube was again verified by auscultation of the lungs. Bilateral breath sounds were present, however were found to be diminished in both bases. The chest x-ray was found to be unchanged from the preoperative results. Due to the elective nature of the case, the decision was made to cancel the case for further workup of the patient's pulmonary status. A pulmonary consult was ordered by the surgeon.

The patient was transported to the post anesthesia care unit intubated & ventilated via manual ventilation with 100% oxygen. Upon arrival, machine ventilation was initiated with ventilator settings of: intermittent mandatory ventilation at 12 breaths per minute, tidal volume of 0.6 liters, 100% oxygen concentration and +5cm H₂O of positive end expiratory pressure. Peak inspiratory pressures were within normal limits as were the patient's vital signs. Lung sounds were clear to auscultation bilaterally. A complete report was given to the nurse

receiving the patient and care of the patient was assumed by the assigned nurse.

Careful inspection of the anesthesia machine and circuit revealed the source of the problem. When the carbon dioxide absorber canister was changed prior to surgery, the wrapper had not been removed.

Discussion

Absorbents are supplied in various types of packaging which can be divided into two types: bulk, resealable containers or disposable, prefilled containers. With the resealable type of receptacle, it is necessary to close the packaging as soon as possible after filling the canister to avoid any interaction with environmental carbon dioxide, loss of moisture or deactivation of the various indicators used to signal exhaustion of the absorbent. Disposable, pre-filled containers are either wrapped entirely in plastic or have labels on the top and bottom of the receptacle which must be removed before insertion. The newly filled canister is then placed into the bottom position as the remaining canister is moved into the top position of the housing. The base of the housing is raised; tightening the canisters together causing a seal in the housing of the absorber system.²

Packing of pre-filled, disposable carbon dioxide absorbent containers has been modified over the past several years in response to reports of user error by failing to remove the wrapper or labels prior to use. Norman, et al proposed switching the wrapping to one that was opaque or tinted or ensuring that it is physically impossible to place a wrapped container into the absorber canister.³ Ransom and Norfleet suggested a change to a bulkier red wrapping after a similar incident at a University of North Carolina Hospital in 1997.⁴ In the current case, the wrapper was clear with instructions in large red lettering to, "Stop! Please remove shrink-wrap before use". The container did fit into the canister without hindrance upon insertion.

Coinciding with other reports, the anesthesia machine in this case appeared to pass the complete required FDA machine check prior to use for administration of anesthesia.^{3,4} Further investigations cited exposed varying degrees of obstruction produced by each plastic, shrink-wrapped absorbent container. Small holes formed, and aligned coincidentally, as a result of the shrink-wrapping process will allow for some gas flow through the canister.⁴ Upon reenacting a machine check in which a wrapped absorber container was placed into the

same machine used in the reported case, the following results were found. All checks of the machine were within normal parameters except when the ventilation system was tested. Fresh gas flows were set at 5 liters per minute with a tidal volume of 1000cc. When the ventilator was turned on, peak inspiratory pressures immediately rose to 42cm H₂O and the maximum tidal volume given was 750cc. When attempting to spontaneously inspire through the circuit, inspiration was difficult. A sedated patient may not be aware of the difficulty or a poor mask fit may allow for inspiration of environmental air. Difficulty in mask ventilation after induction should be a red flag of an obstruction in the closed circle system.

The FDA recommendations for machine checkout were revamped in 1993 after it was determined that the 1986 guidelines were too complicated and did not facilitate detection of apparatus faults.² Interestingly, the previous guidelines included the recommendation that the anesthetic professional breathe through the anesthetic circuit before use. Potential contamination of the circuit would be a major concern to many anesthesia providers, however this maneuver would clearly identify an obstruction.

Despite modifications to the wrapping of carbon dioxide absorber containers, there continue to be instances of improper insertion causing an obstruction of the anesthetic breathing circuit. A final solution to the problem may be found if the absorber packaging was modified making it too bulky for accidental placement into the canister or by wrapping the absorber in an opaque material such as foil.

References

1. Dorsch JA, Dorsch SE. *Understanding Anesthesia Equipment*. 4th edition. Philadelphia: Lippincott; 1999.
2. Anesthesia apparatus checkout recommendations, 1993. *APSF Newsletter*: Fall 1994:35.
3. Norman PH, Daley MD, Walker JR, Fussetti S. Obstruction due to retained carbon dioxide absorber canister wrapping. *Anesth Analg*. 1996;83:425-426.
4. Ransom ES, Norfleet EA. Obstruction due to retained carbon dioxide absorber canister wrapping [Letter]. *Anesth Analg*. 1997;84:703.

Mentor: Anna Hofacker CRNA, MS

POINTS FOR DISCUSSION

These are questions based on articles in this issue

1. What are some complications of an abdominal laparoscopy?
2. What are the anesthesia implications of a placenta previa?
3. What are the signs and symptoms of a serotonin syndrome?
4. What are complicating factors in jet ventilation?
5. What factors affect intracranial pressure on emergence from a craniotomy?
6. What factors may affect visual loss in a prone procedure?
7. What are the optimum drugs to use for pheochromocytoma and why?
8. What is your opinion of the best anesthetic for a carotid endarterectomy? Why?
9. Describe the classifications of endoleaks.
10. What is the basis for our current criteria for giving a blood transfusion?
11. What are the anesthesia implications for a patient with glomerulonephritis?
12. What does a dibucaine number tell you?
13. What are the anesthesia implications for a child with cerebral palsy?
14. How would you recognize a latex allergy during a cesarean delivery?
15. When doing a machine check, how would you discover the wrapper still on the soda absorbent?
16. **What is the new email address for the Student Journal?**

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

Student authors may submit case reports, research abstracts and letters to the editor. Articles 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*.

CASE REPORT PREPARATION

Students prepare the case report with the guidance of a mentor: faculty, CRNA, or physician. The author must have had a significant role in the conduct of the case/anesthetic. Only students may be authors. One or more students may participate in writing the article, at the discretion of the mentor. Mentors are identified at the end of the article.

Format

Case reports must be submitted in the format described in *American Medical Association Manual of Style*. This is the same format found in the *AANA Journal* and such prominent textbooks as *Nurse Anesthesia* by Nagelhout and Zaglaniczy. Reference numbers should be printed in consecutive order as superscripts above the base line and printed after the ending period. Do not format the paper with page numbers. Keep the word count between 1000 – 1200. The references do not count against the word count.

Type

Use Microsoft Word, 12 font, single-spaced, one-inch margins. Do not use columns, bolds, unconventional lettering, styles, or endnote/footnote formats. Place one space after the last punctuation of sentences. Do not indent; separate paragraphs with a double space. End the sentence with the period before placing the superscript number for the reference.

STRUCTURAL GUIDELINES

Title

Make the title brief and specific to the subject. Use no more than 56 letters and spaces.

Author

First name, middle initial and last name, current academic degree(s) and the name of the nurse anesthesia program, *anticipated date of graduation and e-mail address*. (Items in italics will be removed prior to publication.)

Introductory Paragraph

This should be brief and focus the reader's attention to the reason for writing the paper. 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. *Limit word count to less than 100.*

Case Report

This portion discusses the case you did, so it is written in the *past tense*. Don't give any reasons for your actions or behaviors in this section; just 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 the present illness

Statement of co-existing medical problems

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 pre-op work ups. If because of the nature of the illness one of these might be expected to be abnormal, but is not, then that should be mentioned

Physical examination / Pre Anesthesia evaluation - significant findings only. Include the ASA Physical Status and Mallampati Classification if significant to the case.

The conduct of the pre-induction events – monitors, lines.

If the case report is about intra-operative cardiac or respiratory events then there should be considerable detail given to the pre-op findings, even if they were negative.

Induction and maintenance drugs.

Intra-op and surgical events of significance.

Emergence and extubation drugs and events of significance. If the case report is about post operative

airway problems then there should be extensive description of the criteria for and process used for extubation or removal of an LMA.

Discuss the post-op follow-up including ICU or discharge to home.

Despite the detail presented here it is only to help the author organize the structure of the presentation. Under most circumstances if the findings are negative or not contributory to the case then don't mention it at all.

The purpose of the case presentation is to set the stage, or "hook" the reader to the real point of your paper which is the discussion and teaching/learning derived from the case. The case presentation should be *approximately 400 words*.

Discussion

Describe the *anesthesia* implications of the problem 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, *approximately 600 words*.

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. *Approximately 100 words*.

References

Reference format for the Student Journal follows the *AMA Manual of Style*.

<http://healthlinks.washington.edu/hsl/styleguides/ama.html>

<http://www.liu.edu/cwis/cwp/library/workshop/citama.htm>,

<http://www.samford.edu/schools/pharmacy/dic/amaquickref.pdf> ,

Type the references like the regular text. Reference numbers should be in consecutive order from the beginning of the article. (Do not use the "endnote" or "footnote" keys).

References cannot come exclusively from textbooks. This is also an exercise in evaluating and using current literature.

Mentor

Enter your full name, academic credentials and e-mail address. The e-mail address will be deleted prior to publication.

Reference Examples

Journals. Use the correct abbreviations for the journals. See “*List of Journals Indexed in Index Medicus*.” It is also the way they are abbreviated in Medline. The Student Journal of Nurse Anesthesia is not cited in Index Medicus. For the purpose of citing this Journal *in this Journal* use “**Int Stu J Nurse Anesth**” as the abbreviation. The names of journals and text books are printed in *italics*.

Note where commas and periods are and are not present. A journal may have a volume number and consecutive pages throughout the volume. Note that the month is not used unless the journal starts its page numbers at one each month. Cite only the year, volume [not number] and pages. The pages are inclusive and **do not omit digits**.

Journal, more than one author:

Gold RS, Pollard Z, Buchwald IP. Asystole due to the oculocardiac reflex during strabismus surgery: a report of two cases. *Ann Ophthalmol* 1988;20:473-477.

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** and add “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 and the pages. Place a semicolon after the year, a colon after the volume number. The pages are the inclusive pages. Use the journal’s abbreviations as found in *Index Medicus* (Medline).

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, one or more authors

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

Each chapter was written by those authors. Note the inclusive page numbers **are** cited.

Text, one or more editors

Donlon JV. Anesthesia for eye, ear, nose, and throat surgery. In: Miller RD, ed. *Anesthesia*. 5th ed. Philadelphia: Churchill Livingstone; 2000:2173-2198.

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.

WEB sites. Only peer reviewed web sites may be used and cited. Authors are cautioned to not copy and paste from these without full credit and quotation marks where appropriate. If the contents of the web site are also available in paper print form (Journals) then that mode should be located and cited. *Cite the author(s) if known. Title of the article.* Journal or name of the site. Year;volume: pages. *Available at – identify the full site description.* Accessed Month day, year in four digits.

Pulmonary embolism. Available at <http://www.emedicine.com/med/topic1958.htm>. Accessed April 8, 2003.

Thatcher VS. *The History of Anesthesia with Emphasis on the Nurse Specialist*. Philadelphia: J.B. Lippincott;1953. Available at <http://www.aana.com/archives/pdf/0001INTR.pdf>. Accessed September 25, 2004.

ACADEMIC INTEGRITY

Issues of academic integrity are the primary responsibility of the author and mentor. Accurate and appropriate acknowledgement of sources is expected.

“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

Any violation will be cause for rejection of the article for further consideration for publication.

VOCABULARY USAGE/SPELLING

Avoid jargon.

The patient was reversed.

Did you physically turn the patient around and point him in the opposite direction? “The neuromuscular blockade was antagonized.”

The patient was put on oxygen.

“Oxygen was administered by face mask.”

The patient was intubated and put on a ventilator.

“The trachea was intubated and respiration was controlled by a mechanical ventilator.

The patient had been on motrin for three days.

“The patient had taken ibuprofen for three days.”

Use only accepted abbreviations.

Define all abbreviations when first used. Avoid abbreviations that are not common to anesthesia professionals.

Avoid the term “MAC” when referring to a sedation technique.

The terms heavy sedation, or unconscious sedation may be used along with the names of the drugs administered, or planned to be used for that sedation. Since all anesthesia administration is monitored, the editors prefer to use specific pharmacology terminology rather than reimbursement terminology.

Use the words “anesthesia professionals” or “anesthesia practitioners.”

When discussing all persons who administer anesthesia avoid the reimbursement term, “anesthesia providers.”

Use the generic name of drugs and medical devices

Avoid trade names. Type generic names in lowercase. When discussing dosages state the name of the drug, then the dosage and route.

Reporting infusion rates and gas flow rates

When reporting infusion rates report them as mcg/kg/min or mg/kg/min. If more than one drug is infused in the same device then give the quantity and concentration of each drug and report the infusion rate in cc/min.

Keep the gas laws in mind when reporting flow rates. Avoid statements such as: 40% oxygen, 60% nitrous oxide and 4% sevoflurane. (It doesn't = 100%). Report the liter flows of oxygen and nitrous oxide and the percent of the volatile agent added to the gases.

HOW TO SUBMIT THE ARTICLE

Manuscripts should be submitted by the CRNA mentor of the student author. Send them via e-mail to **INTSJNA@aol.com** as an attachment. The subject of the e-mail must be clearly identified as a submission to the Student Journal.

Prior to attaching the article save the article in “Save As” in this form - [A two word descriptor of the article (space) author's last name (space) school abbreviation (space) mentor's last name]. Example: PedsPain-Smyth-GU-Pearson

If the editor does not acknowledge receipt of your article within five days, assume that it was not received and please inquire.

REVIEW AND PUBLICATION

Upon receipt, the Editor reviews the article for compliance with the Guide to Authors and reference format, making minor corrections as needed. The Editor sends that copy to two reviewers simultaneously who are not affiliated with the originating program.

The reviewers recommend publication to the Editor or call his attention to flaws in the manuscript that can only be corrected by the author. The reviewers may make changes to the articles that improve their clarity and brevity, without making substantive changes to the clinical points.

Mentors of the papers will be asked to serve as peer reviewers of other students' case reports and they will be listed as contributing editors in each remaining issue of that volume.

The Editor may accept or reject the reviewers' changes or recommendations. 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.

If there is time, prior to the editor's deadline to the publisher, the edited revised version is returned to the mentor for final approval. Only the most minor changes may be made at that time.

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 the article was submitted late, or required several edits it may not be returned to the mentor for a final review.

The decision of the Editor is final.

RESEARCH ABSTRACTS

Research Abstracts are limited to 400 words. References are not desired. Note that this abstract is different from a research proposal. This abstract reports the outcome of your study. Use the following format:

Title

Author(s)

Full Name and degree (s)

Anesthesia Program

Anticipated date of graduation

E-mail address

Introduction

Purpose

Hypotheses

Methodology (include statistical analyses used)

Results

Conclusion

Mentor

Full name

Academic degrees

E-mail address

LETTERS TO THE EDITOR

Students may write letters to the editor for publication on 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.

Letters should be submitted as an attachment using Microsoft Word. Follow the format style as required of other articles in this journal.

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. Please also identify the photographer. Digital photos are preferred, but they must be of high quality, and e-mailed to us as you submit articles. 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:

The International Student Journal of Nurse Anesthesia
c/o Ronald Van Nest
1306 Anglesey Drive
Davidsonville, MD 21035

CHECK LIST

Footnote numbers start at number one and continue sequentially.

Total word count is reasonably close to requirements.

The article is in one continuous document without artificially created page breaks.

Program and academic verbiage is removed such as keywords, running heads, “partial fulfillment . . .”

Document is in 12 font and single spaced.

Paragraphs are separated by double space with no indents.

Heading

The title accurately and concisely describes the issue of the discussion and is not just a general description of the type of case or co-morbidity.

The author and mentor are fully identified with academic credentials of each.

Emails of the author and mentor(s) are provided.

The author’s graduation date is provided.

Introductory Paragraph

Historical background, demographics and/or epidemiology of the problem is presented and referenced.

Reference numbers are sequenced beginning with one and superscripted.

“Endnote and footnote keys” are not used.

Introduction is less than 100 words.

Case Report

The case presentation section states only facts and only those facts vital to the account (no opinions, rationale,...).

The case report section is less than 400 words. (It should not be longer than the discussion)

Generic names for drugs and products are used throughout, spelled correctly and in lower case letters.

Units are designated for all dosages, physical findings and laboratory results.

Jargon is absent.

Abbreviations are defined when first used.

Discussion section

All matters that are not common knowledge to the author are referenced.

Verbatim phrases and sentences are quoted and referenced.

The discussion section is approximately 1.5 times longer than the case presentation section.

The discussion focuses on the anesthesia management based on review of the literature and is not a synopsis of a pathophysiology text.

References are from anesthesia and other current primary source literature.

The discussion concludes with lessons learned and how the case might be better managed in the future.

References

AMA Style for referencing is used, with italics where appropriate

All inclusive pages are cited, texts as well as journals.

The journal titles are abbreviated as they appear in *Index Medicus*.

Textbook references are avoided when the information is available in current journals.

Web sites are used only when currently accessible, reputable, and peer reviewed, and the information is not available in print media.

Transmission

The article is being sent to **INTSJNA@AOL.COM** as an attachment.

It is being sent by the mentor, preferably; or by the student author with CC to mentor, stating that it is being sent with the mentor’s approval.

The Words “Student Journal” are in the subject heading.