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Chronic Obstructive Pulmonary Disease

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Refractory Hypotension with ACEIs

Adult Aortic Coarctation Repair

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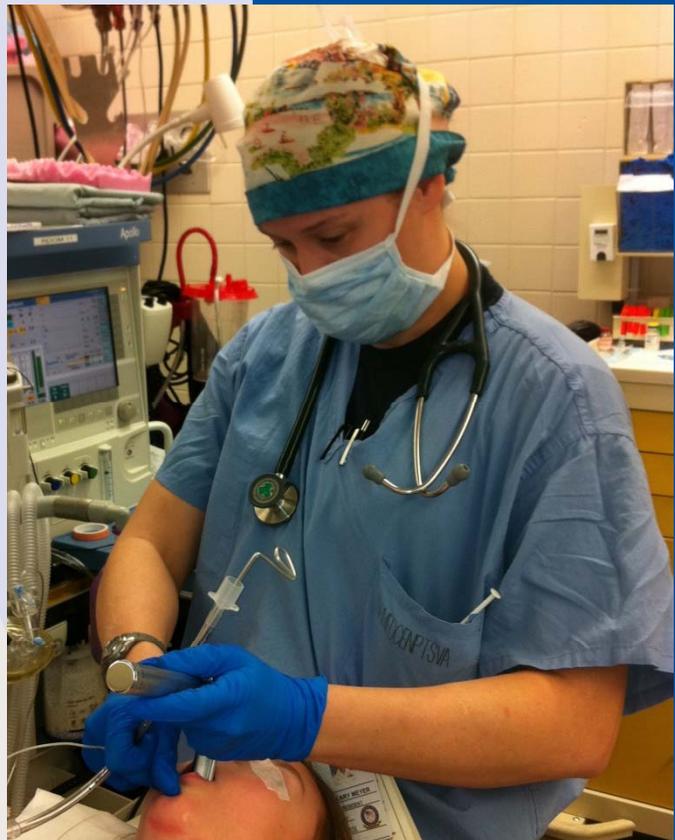
Vagal Nerve Stimulators

Awake Tracheostomy

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Front Cover:

LT Hilary Meyer BSN, NC, USN, a senior graduate student in the Navy/Uniformed Services University of the Health Sciences Graduate Nurse Anesthesia Program, performs laryngoscopy on a pediatric patient.

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Perioperative Anaphylaxis

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Keywords: anaphylaxis, anesthesia, cefazolin, cross-sensitivity, latex, serum tryptase.

Perioperative anaphylaxis is a serious complication that anesthesia professionals may face unexpectedly during an operation. It is estimated that generalized anaphylactic reactions resulting from medications given during procedures occur from 1 in 5,000 to 1 in 25,000 cases and can be fatal up to 6% of the time.¹ Due to the fact that multiple agents are administered over a short period of time during anesthesia, it is difficult to identify the responsible agent unless there is an intradermal test post-anaphylaxis. Immediate intervention is necessary to treat severe generalized anaphylactic reactions. This case study focuses on recognition and management of perioperative anaphylaxis.

Case Report

A 50 year-old, 170 centimeter 70 kg male presented for a right inguinal hernia repair. The patient was alert and oriented to person, time and event. His past medical history was significant for chronic back pain due to fractured vertebrae after a motorcycle accident in 2006. The patient had subsequent spinal fusion between thoracic vertebrae 5 and 7 (T5-T7). The patient denied allergies to medications or any complications during previous anesthetics. However, he did acknowledge methamphetamine and tobacco abuse for over 20 years. His current medications included nystatin, vardenafil, baclofen, famotidine, tolterodine. The patient, surgeon, and anesthesia team discussed the perioperative plan of general anesthesia with

use of a laryngeal mask airway (LMA). The patient was given intravenous (IV) doses of midazolam 2 mg, cimetidine 300 mg and cefazolin 1000 mg.

Basic monitors for anesthesia care meeting standards set by the American Society of Anesthesiologists (ASA) were initiated upon arrival to the operating room and the patient was pre-oxygenated with 10 L/min via face mask. A second dose of midazolam 2 mg was administered and general anesthesia induced with IV fentanyl 50 mcg, lidocaine 100 mg, and propofol 200 mg. An LMA size 4 was inserted and placement confirmed by positive end-tidal carbon dioxide (EtCO₂). General anesthesia was maintained with sevoflurane 2.0% in a mixture of oxygen 1.5 L/min and nitrous oxide 1.5 L/min. Dexamethasone 20 mg was administered for post-operative nausea and vomiting prophylaxis. The anesthetist assisted the patient's respiration for the first five minutes post induction until spontaneous respiration resumed.

The operation was uneventful with stable vital signs and spontaneous respirations maintaining within 20% of his baseline systolic blood pressure of 130 mmHg and heart rate of 70 beats per minute (bpm). Approximately 60 min into the case, shortly after the surgeon began to close the incision, the patient's heart rate suddenly increased into the 110's. The patient's systolic blood pressure decreased to 70 mmHg. The patient's blood pressure was unresponsive to the administration of phenylephrine and ephedrine. The anesthesia team noticed increasing peak airway pressures and decreasing EtCO₂ with diminished breath

sounds. Sevoflurane was turned off and oxygen flow was increased to 15 liter/min. The attending anesthesiologist was called to the room to help as the surgeon continued to close the incision. The LMA was replaced with an endotracheal tube. Right radial arterial line was placed. Epinephrine 20 mcg was administered IV and the blood pressure improved briefly to greater than 80 mmHg systolic. Arterial blood pressure began to drop less than thirty seconds to a systolic of 70 mmHg. Epinephrine was repeated at a higher dose of 100 mcg and systolic blood pressure was restored to 120 mmHg following a larger dose of epinephrine. In addition, methylprednisolone 125 mg, diphenhydramine 50 mg, and a fluid bolus of 2000 ml given. An epinephrine infusion was initiated at 10 mcg/min.

The surgeons closed the incision and the patient was transported to the intensive care unit with an epinephrine infusion at 5 mcg/min and a propofol infusion. The patient's serum tryptase level, an indicator of mast cell degranulation seen after allergic reactions, was 131 nanograms (ng)/milliliter two hours after the operating room event. Normal serum tryptase levels are 0.8-1.5 ng/ml.² The patient was weaned from the ventilator the next morning and discharged home later that day. The discharge diagnosis was an intraoperative anaphylactic reaction.

Discussion

There are two important points to consider during cases of perioperative generalized anaphylaxis. The first is the ability to treat and promptly stabilize the patient's condition when anaphylaxis occurs, and the second is skin testing to determine the responsible agent.

The incidence of a generalized adverse medication reaction during anesthesia is

reported to be 0.02-0.004% .^{3,4}

Neuromuscular blocking agents (NMBAs) are the most common causes of generalized reactions with rocuronium the most prevalent, accounting for 58%, followed by latex (16.7%), and antibiotics (15%).^{3,5,6} The beta-lactam antibiotics which include penicillin and cephalosporin account for 80% of all generalized reactions to antibiotics.¹ This patient did not receive NMBAs, but was exposed to both latex and a beta-lactam antibiotic. However, to determine the exact agent responsible for anaphylaxis, complete skin testing with suspected agents should be completed with the patient. It is recommended that skin testing be performed 4-6 weeks post anaphylaxis to prevent a false-positive result.^{6,7} At the time this report was written, the patient was 4 weeks post anaphylactic event and scheduled to visit the allergy clinic within 2 weeks. The following review of the patient's record showed that the appointment was rescheduled and the patient cancelled the appointment.

The predominant manifestation of intraoperative anaphylaxis is hypotension, presented with or without bronchospasm.⁸ When there is an abrupt decrease in blood pressure, allergic reaction to latex should be considered as well as drugs administered. The high-risk patients are those with spina bifida, fruit allergies, history of atopy, and chronic exposure with latex such as health care workers and multiple previous operations.^{8,9}

Neuromuscular blocking agents are accounted for more than half of perioperative anaphylaxis incidents. Neuromuscular blocking agents structurally fall into two general categories, either aminosteroidal or benzyliisoquinolinium compounds, and due to these structural similarities, incident of cross-sensitivity is

high. Pre-operative skin testing is necessary for a patient who is allergic to any muscle relaxants to determine sensitivity of the likely used drugs for the planned anesthetic.⁸

The medical literature provides multiple opinions regarding the cross-sensitivity between the common classes of two beta-lactam antibiotics, particularly penicillin and cephalosporin. One source indicated that among the penicillin allergic patients, possible cephalosporin allergic reactions were reported to be 2.5% compared to 1.3% among non-penicillin allergic patients.⁸ The American Academy of Pediatrics evidence-based guidelines endorse the use of cephalosporin antibiotics for patients with reported allergies to penicillin for the treatment of acute bacterial sinusitis and acute otitis media.⁸ However, anesthesia sources argue that there are structural similarities between penicillin and cephalosporin, thus cross sensitivity between the two may occur at 2-7%.¹⁰ In addition, there is 50 % increase to the cross sensitivity rate if a patient has a history of an anaphylactic reaction to penicillin; therefore cephalosporins should not be administered.⁹

If the perioperative team had knowledge of the patient's prior reaction to penicillin, an alternative other than cefazolin could have been chosen. However, the patient was interviewed on different occasions, including at the preoperative clinic visit and on the day of surgery by different personnel. The patient appeared to be a competent historian.

Vigilance, attentiveness, and the ability to promptly intervene throughout the operative procedure are important priorities for the anesthesia practitioner faced with a patient experiencing perioperative anaphylaxis. Treatment must be immediate and aggressive, tailored to the severity of the

patient's reaction. Interventions include discontinuing all drug administration which may have caused the reaction, administering 100% oxygen, and intravenous fluids. Pharmacological treatment of anaphylactic reactions may include the administration of epinephrine, ranitidine, diphenhydramine and methylprednisone. The literature suggests an initial epinephrine dose of 100-500mcg, and depending on severity of reaction, an epinephrine infusion starting at 1mcg/min.⁹

In most cases, anaphylaxis occurs rapidly after the administration of the responsible agent, although delayed reactions may also occur.^{4,6,9} For example, the onset of symptoms related to exposure to latex may be delayed for greater than one hour.⁹ In this case, the patient's hypotension and bronchospasm did not occur until one hour after the administration of cefazolin, induction agents, and probable exposure to latex. It is possible that the patient had anaphylactic reaction to latex which caused the delay in immediate response with massive mast cell degranulation and subsequent histamine release. Nevertheless, early recognition of a change in the patient's status, prompt administration of appropriate drugs such as epinephrine, immediate airway protection, and the ability to restore hemodynamic homeostasis is an essential skill for anesthesia practitioners.

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Anesthesia Care for Chronic Obstructive Pulmonary Disease

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Keywords: chronic obstructive pulmonary disease (COPD), prone position, anesthesia

Chronic obstructive pulmonary disease (COPD) is manifested by partially reversible progression of limited airflow. The diagnosis consists of chronic bronchitis involving small airway obstruction and emphysema with alveolar enlargement, lung parenchyma destruction, loss of elasticity, and closure of the small airways.¹ Additionally, advanced age and COPD are both risk factors for perioperative complications.² It is important to understand the multitude of physiologic changes associated with prone positioning, aging, and COPD. This case report will focus on the anesthetic considerations for the elderly, COPD patient.

Case Report

A 77 year old, 93kg, 72 in, caucasian male presented for double balloon enteroscopy after initial diagnosis of benign large intestinal neoplasm. His medical history was significant for obstructive sleep apnea, bronchiectasis, coronary artery disease, COPD, hypertension, congestive heart failure, gout, and smoking tobacco, 3 packs daily for 50 years. His surgical history included bilateral carotid endarterectomy, coronary artery bypass post myocardial infarction, pacemaker, and orthopedic spine. The patient denied history of anesthesia complications. Routine medications included carvedilol, furosemide, omeprazole, and lisinopril. Preoperative physical exam revealed a Mallampati class III airway and breaths sounds revealed mild expiratory wheezes throughout. Vital signs

were blood pressure 145/60, heart rate 89, respiratory rate 18, and temperature 98.0 F.

The patient was preoxygenated with 100% oxygen for 5 minutes and standard monitors were applied. Intravenous (IV) induction was performed with lidocaine and propofol. The patient was difficult to mask ventilate with an oral airway in place, and the assistance of a second anesthesia practitioner was needed to compress the breathing bag. After the ability to mask ventilate was verified, rocuronium 50 mg IV was given. Direct laryngoscopy was performed; after three attempts to secure the airway, a bougie was needed to assist with placement of the endotracheal tube. Sevoflurane was used for anesthesia maintenance with end tidal concentrations maintained at 2-2.2%. Controlled ventilation was maintained with a tidal volume of 800ml and respiratory rate of 10. End tidal CO₂ was maintained between 31-36 mm Hg. The patient was later turned to the prone position.

Thirty minutes before the end of the procedure, the patient was turned supine and gradually weaned off the ventilator. End tidal CO₂ was 40-45 mmHg during spontaneous ventilation. At the conclusion of the procedure, the inhalation agent was discontinued, neostigmine and glycopyrrolate were administered for full reversal, and 100% oxygen was delivered via the endotracheal tube. When tidal volumes were adequate and the patient was fully awake, the endotracheal tube was removed and 100% oxygen was delivered via face mask. During emergence, the patient became hypercapnic with an end tidal CO₂ of 60 mmHg, a dusky facial appearance, and his blood pressure increased to 170/90mmHg. The patient's breathing was assisted by mask ventilation, but the end tidal CO₂ remained elevated. The patient

was then changed to controlled ventilation. The patient's dusky facial appearance resolved and end tidal CO₂ levels decreased to 40 mmHg. Oxygen was administered via nonrebreather mask at 15 L/min and vital signs stabilized.

Discussion

COPD is a chronic disease that manifests as emphysema or bronchitis. The pathology of the emphysema component is characterized by irreversible destruction and enlargement of the airways distal to the terminal bronchioles while the clinical diagnosis of chronic bronchitis includes productive cough with sputum for at least 3 months for two consecutive years.² Mortality rate is increased 10-fold in COPD patients compared to patients without lung pathology.³ Anesthetics and analgesics administered may alter bronchial tone, central regulation of breathing, and neural drive to respiratory muscles leading to complications. Additionally, patient positioning and mechanical ventilation may alter ventilation/perfusion matching and lead to atelectasis in dependent lung regions. Surgical trauma will alter respiratory function when disruption of respiratory musculature occurs.³

There are a multitude of physiologic changes associated with aging and prone positioning. Age-related changes of the pulmonary system include alterations in lung parenchyma and alveoli which lead to reduced lung recoil, dilation of alveolar ducts, development of enlarged airspaces, and reduction of alveolar surface area.⁴ Physiologic changes of prone positioning include uniform pulmonary blood flow, increased functional residual capacity, improved ventilation-perfusion distribution, and decreased cardiac index and output.⁵

Preoperative evaluation of the elderly patient with COPD is determined on an individualized basis and may include a chest x-ray to determine presence of hyperinflation or pulmonary infection, spirometry for measurement of lung function, and arterial blood gas analysis to provide measurement of gas exchange.³ Factors associated with increased risk of severe postoperative pulmonary complications include increased mucus secretion, hyperinflation, and abnormalities of both preoperative forced expiratory volume in one second (FEV1) and diffusing capacity.³ Premedication may create risk for hypoventilation. However, it may be beneficial for the patient with anxiety, which can exacerbate dyspnea.³

Some anesthetic agents may be more beneficial than others for the elderly patient with COPD. All volatile inhalation agents are potent bronchodilators. Sevoflurane reduces airflow resistance and suppresses responses to airway stimuli better than desflurane.³ Propofol is superior to thiopental or etomidate in blunting airway reflexes and reducing bronchospasm during intubation.³ In COPD patients, propofol induces bronchodilation and improves respiratory mechanics secondary to a decrease in peak inspiratory pressures and respiratory system resistance resulting in improved dynamic compliance of the airways.³ Histamine releasing medications, such as thiopental, atracurium, and morphine may lead to bronchospasm in the patient with COPD.³ Mechanical ventilation parameters should also be carefully chosen for optimal management of the COPD patient.

The goals of mechanical ventilation in the COPD patient should include sufficient arterial oxygenation and strict avoidance of gas trapping.³ If the patient is normocapnic

or mildly hypercapnic, arterial carbon dioxide partial pressure (PaCO_2) is the major stimulus for breathing regulation. However, in severe COPD, ($\text{PaCO}_2 > 50 \text{ mm Hg}$) and arterial oxygen partial pressure is $< 50 \text{ mm Hg}$, hypoxia becomes the respiratory drive.³ Therefore, it would be prudent to avoid high arterial oxygen partial pressures which can lead to hypoventilation.³ During mechanical ventilation, plateau pressure should be maintained less than $30 \text{ cm H}_2\text{O}$ to decrease risk of pulmonary hyperinflation and barotrauma.³

Controlled mechanical ventilation may be needed in the patient with COPD, especially in patients with FEV1/FVC ratio < 0.5 or preoperative $\text{PaCO}_2 > 50 \text{ mm Hg}$.¹ Controlled mechanical ventilation optimizes oxygenation. Slow inspiratory rates (6-10 breaths per minute) and adequate expiration times are needed to minimize turbulent airflow and to maintain optimal ventilation-perfusion matching.¹

For the patient reported here, a chest x-ray, arterial blood gas analysis, and spirometry should have been obtained preoperatively. This baseline information would have provided a clearer depiction of the severity of this patient's COPD. Intraoperatively, the anesthetic medications used for this patient included sevoflurane, propofol, and rocuronium. No opioids were administered, in order to reduce the risk of respiratory depression and hypoxemia postoperatively. For postoperative prophylaxis of hypercarbia, controlled mechanical ventilation should have been continued until the end of the procedure after the patient was turned supine and muscle relaxant antagonist was administered, while closely monitoring ETCO_2 and pulse oximetry. This would have provided clinical data on ventilation and oxygenation in the supine position resulting in the ability to determine

if mechanical ventilation should be continued postoperatively and if the patient met criteria for removal of the endotracheal tube.

In conclusion, all patients should have a thorough preoperative evaluation including assessment of past medical history, review of systems, physical exam, arterial blood gas analysis, and spirometry to determine severity of the disease. Additionally, intraoperative anesthetic medications for optimal management of the COPD patient would include propofol, sevoflurane, and non-histamine releasing muscle relaxants. However, existence of cardiac depression and coexisting diseases would also guide decision-making for the anesthesia medications that would provide optimal outcomes. The decision to continue mechanical ventilation and remove the endotracheal tube should also be determined on an individualized basis according to the patient's condition.

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Awake Tracheostomy for Trismus and Oropharyngeal Tumor

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Keywords: awake tracheostomy, trismus, oropharyngeal tumor, difficult airway

Difficult airways can be categorized into two groups; emergent situations and non-emergent situations with known compromised airway anatomy.¹ Pre-anesthetic evaluation and diagnostic tests can be utilized to determine the safest and least invasive airway management technique for patients with oropharyngeal tumors.² Awake airway management is part of the American Society of Anesthesiologists

difficult airway algorithm.³ When oral and nasal fiberoptic laryngoscopy is not feasible, awake tracheostomy may be considered the safest approach to secure an airway especially in those with known difficult anatomy.⁴ The importance of recognizing difficult situations and planning ahead is a priority as failure to ventilate is associated with mortality and damage to the central nervous system that could be permanent.⁵

Case Report

A 57 year old, 38 kg, 157.5 cm female presented for full dental extraction with alveoplasty, sinonasal endoscopy with biopsy, and microlaryngoscopy with biopsy. She was recently diagnosed with inoperable squamous cell carcinoma involving the left tonsil, soft palate, nasopharynx, vallecula, and base tongue. A previously performed fiberoptic bronchoscopy revealed a patent glottis opening. Past medical history was significant for esophageal squamous cell carcinoma, metastasis to head and neck, status post esophagectomy and gastrectomy with esophageal pull up and chemoradiation therapy, hypothyroidism, chronic obstructive pulmonary disease, right-sided hemorrhagic cerebrovascular accident, and iron deficiency anemia.

Anesthetic evaluation and assessment revealed limited neck extension, a non-tender left mandibular mass, dysphagia, and odynophagia. An airway assessment was unattainable due to trismus, although tongue deviation and worsening shortness of breath over the last three days were noted. The patient also stated she was unable to breath through her nose. Auscultation of her lungs revealed decreased left base breath sounds. Preoperative hemoglobin and hematocrit was 10.5 g/dl and 34.2% respectively. She also had an activated partial thromboplastin time (aPTT) of 49.3 seconds and plasma potassium of 3.2 mmol/L. The patient was receiving morphine, heparin, levothyroxine, and zolpidem prior to the procedure. After the assessment was performed the anesthesia team consulted with the surgical staff to discuss airway management. The anesthesia team recommended securing a surgical tracheostomy prior to the induction of anesthesia. Local anesthetic infiltration by the surgical team and minimal sedation by the anesthesia team would be provided for

the tracheostomy procedure with the goal of maintaining the patient in an awake state with spontaneous respirations. The surgical team agreed with this plan.

Potassium 40 meq in normal saline 250 ml was initiated in the preoperative holding area. No other premedication was administered. The patient was transported to the operating room receiving oxygen (O₂) 4 L/min via nasal cannula. Once in the operating room the patient was transferred to the operating room table and noninvasive monitors were placed on the patient. The surgical team provided local anesthetic to the area that consisted of 1% lidocaine 14 ml with epinephrine 1:100,000. The patient was given a total of midazolam 1 mg and ketamine 10 mg, both in divided doses. Supplemental O₂ delivery was maintained at 4-6 L/min via nasal cannula throughout the tracheostomy procedure which lasted 50 min. The patient remained responsive throughout the procedure and generally comfortable, although stating she was uncomfortable at times. Reassurance was provided to the patient without additional local anesthetic being given. After correct placement of the tracheotomy tube was confirmed and a general anesthetic was administered for the remainder of the procedure.

Discussion

Oropharyngeal tumors and distorted neck anatomy can be challenging for anesthesia professionals to maintain an adequate airway. In this case awake tracheostomy was decided to be the safest option after collaboration between the surgical and anesthesia teams. Awake fiberoptic intubation was contemplated but decided against because of the difficulty of anesthetizing the oropharynx, nasopharynx, and trachea. When performing an awake

fiberoptic intubation adequate anesthesia to the oropharynx and/or the nasopharynx for comfort and cooperation on insertion of the fiberoptic bronchoscope is performed.⁶ The trismus the patient presented with prevented local anesthetic administration and access to pass the bronchoscope. A nasal fiberoptic intubation could have been a possibility although it too was invaded by the tumor, appearing to be the probable reason for the patient having restricted airflow.

Anesthesia to the vocal cords must also occur when planning an awake fiberoptic intubation, which can be accomplished by a transtracheal block.⁶ A transtracheal block was performed in our patient for passage of the tracheostomy by the surgical team, although in an awake fiberoptic without proper anesthesia to the upper airway, the fiberoptic bronchoscope may not have reached the level of the vocal cords because of discomfort to the patient. The previous bronchoscopy performed in the emergency room by the emergency room physicians proved that the degree of invasion of the oropharyngeal tumor from the left tonsil involving the soft palate, nasopharynx, vallecula, and base of tongue would have not allotted easy passage of the endotracheal tube. The use of bronchoscopy to evaluate the airway in patients with oropharyngeal tumors and patients with worsening respiratory failure scheduled for surgery proves to be an important tool to determine airway management during the surgical procedure.²

Another concern with fiberoptic intubation for this patient was the potential to cause trauma to the tumor, which would likely cause bleeding that could result in difficulty with further visualization, and excessive blood loss considering her prolonged aPTT. This is often a major concern of airway

tumors that warrants consideration when choosing an airway method.²

When oral or nasal intubation is not feasible, awake tracheostomy is performed.¹ While the patient was scheduled for an elective tracheostomy, it was decided that performing the tracheostomy as the airway management technique was appropriate in this situation. An awake tracheostomy was selected by the anesthesia staff over an asleep technique due to the concern of sedation resulting in a collapsed airway along with the inability to obtain a secure airway prior to the procedure as mentioned above. When sedation is given the soft tissue relaxes potentially obstructing the airway with a decreased diameter and patency of the pharynx, which can be even more severe in a patient with a tumor present in the airway.³ There are several other advantages of having an awake patient which include having the ability to maintain spontaneous ventilation and decreased risk of aspiration because of the ability to maintain esophageal sphincter tone.

A disadvantage of maintaining the patient in an awake state includes possible discomfort, the patient becoming uncooperative, and the patient remembering the procedure. Midazolam 1 mg and ketamine 10 mg were administered to this patient for comfort and sedation. Ketamine was selected for its pharmacological properties that allow the patient to maintain spontaneous ventilation when given in small doses and the bronchodilatory effects it exhibits, although administration of larger doses has the potential for producing respiratory depression.³ Midazolam was administered for its anxiolytic properties. An alternative would have been dexmedetomidine, an α_2 agonist, to maintain spontaneous ventilation without causing respiratory depression.⁶

The surgical team provided local anesthetic, including a transtracheal airway block, and infiltration of the skin prior to incision but the patient still complained of mild to moderate discomfort throughout the procedure. If we would have chosen to use dexmedetomidine, its analgesic properties could have aided in relieving the discomfort experienced by the patient. Although ketamine has greater analgesic properties than dexmedetomidine, the dose was restricted by the respiratory depressive effects it exhibits.

This case reflects a collaborative effort by the surgical and anesthesia staff to safely manage a patient with a very tenuous airway. Often the surgical team relies heavily on the expertise of the anesthesia team for the safest method to secure a patient's airway prior to induction of anesthesia. Teamwork between both the anesthesia and surgical staff and maintaining open communication between all parties involved in the care of the patient can assure the most appropriate method is used. Discussing potential complications preoperatively can prevent devastating airway situations intraoperatively.

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Management of Adrenalectomy for Pheochromocytoma

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Keywords: adrenalectomy, pheochromocytoma, hypertension, norepinephrine, endocrine

Pheochromocytoma is a rare tumor that arises from chromaffin cells in the adrenal

medulla or in other paraganglia of the sympathetic nervous system.¹ Patients present with a variety of symptoms reflecting excessive secretion of norepinephrine, epinephrine or dopamine into the circulation.^{2,3} Pheochromocytoma

accounts for less than 0.3% of all cases of hypertension and surgical intervention is recommended for survival.^{4,5} This case report reviews pertinent literature and discusses anesthetic management for adrenalectomy in patients with a pheochromocytoma.

Case Report

A 40 year old, 57 kg, 168 cm female presented for an open adrenalectomy of a left adrenal mass. Past medical history was significant for acute controlled hypertension, with a baseline blood pressure (BP) of 128/68 mmHg, and palpitations resulting from a diagnosed norepinephrine-secreting adrenal tumor. Pertinent medications included terazosin 2 mg daily with the last dose 9 hours prior to anesthetic induction. A baseline electrocardiogram evidenced normal sinus rhythm at 73 beats per minute (BPM). Physical examination, vital signs and laboratory tests were unremarkable.

Preoperatively, a thoracic epidural was placed for postoperative analgesia. Induction of general anesthesia was accomplished with fentanyl 50 mcg, lidocaine 100 mg, propofol 150 mg, and rocuronium 50 mg, followed by endotracheal intubation. General anesthesia was maintained with 1.2% end-tidal concentration of isoflurane in a 50:50 air to oxygen mixture at a total of 1 liter/min. An arterial line and double lumen central line were placed.

During the first 2 hours, the patient's BP averaged 115/72 mmHg and heart rate (HR) averaged 83 BPM. Fentanyl, 250 mcg, had been titrated to maintain +/- 20% of the patient's baseline HR and BP. Rocuronium, in 10 mg increments, was administered once per hour to maintain 1 of 4 twitches. As adrenal gland dissection occurred, the patient's BP and HR increased to 178/80

mmHg and 90 BPM, respectively. Nitroglycerin boluses of 200 mcg were given IV every minute. After 20 doses (200 mcg/dose), a nitroglycerin infusion was initiated at 500 mcg/min, which effectively brought the BP to 155/80 mmHg and HR to 82 BPM. Over the next 15 minutes the patient's BP and HR gradually rose to a maximum of 182/88 mmHg and 90 BPM. During the same 15 minutes the nitroglycerin infusion was titrated to 999 mcg/min with no resolution of the hypertension. Since nitroglycerin was no longer effective in controlling the SBP, evidenced by nearly 15,000 mcg total of nitroglycerin being administered over 35 minutes, the surgeon was alerted and the surgical procedure was temporarily halted while a nitroprusside infusion was initiated at 1.5 mcg/kg/min. The nitroglycerin infusion was discontinued and the BP returned to 118/78 mmHg with a HR of 82 BPM.

Surgery resumed, and 5 minutes after tumor resection, the BP decreased to 90/58 mmHg. Nitroprusside was discontinued and norepinephrine was initiated at 2 mcg/min. For the remainder of the case and into the postoperative phase, norepinephrine was titrated, with 6 mcg/min as the highest dose, to maintain SBP greater than 110 mmHg. Lactated ringers solution, 3500 mL, was administered prior to and 1500 mL administered after the tumor resection. Blood loss was estimated at 150 mL. Extubation in the operating room and recovery in the post-anesthesia care unit (PACU) were uneventful. In order to maintain +/- 20% of the patient's baseline BP, norepinephrine was maintained at 2 mcg/min in the PACU and titrated off over the next 48 hours in the intensive care unit. No adverse complications were reported in the post-operative period.

Discussion

Many methods exist regarding pheochromocytoma management. Most agree that preoperative antagonism is necessary to prevent unpredictable responses to α -adrenergic agonists.⁶⁻¹⁰ Many agents have been successful, including selective α -1-antagonists, calcium channel-antagonists, and combined α - and β -antagonists.^{6,7,9,11} A 2 week treatment of phenoxybenzamine, with the last dose 10 hours before surgery, has been shown to be effective at antagonizing adrenergic receptors.^{2,3,6,7} However, greater use of perioperative vasopressors has been required in patients pretreated with nonselective α -antagonists versus α -1-selective antagonist.¹⁰ The patient in the presented case was managed according to research recommendations since her last dose of terazosin, a selective α -1 adrenergic antagonist, was taken nine hours prior to surgery.¹⁰

General anesthesia, with central venous and arterial catheters, is the recommended technique for an adrenalectomy. Utilizing regional anesthesia for postoperative analgesia must be balanced against the risks of hypertension during placement.^{5,6} Hypertension did not occur during epidural placement in the presented case.

Thiopental, propofol, and etomidate have all been safely used for induction.^{2,6,7} Agents causing indirect increases in catecholamine levels, such as ketamine and ephedrine, should be avoided.⁶ Morphine and meperidine cause histamine release, a trigger of pheochromocytoma crisis.⁷ Additionally, meperidine causes sympathetic stimulation. Thus, both opioids should be avoided.⁷ Sevoflurane is the ideal inhalational anesthetic since it is rapidly titratable, lacks the sympathetic stimulation of desflurane and has less negative inotropic effects

compared with isoflurane, enflurane, and halothane.⁷ Muscle relaxation with vecuronium is best since minimal changes in arterial pressure and plasma catecholamine levels occur without the mild vagolytic effect of rocuronium.^{7,12}

Hypertension during pheochromocytoma resection results from noxious stimuli, direct tumor manipulation, or both.⁷

Norepinephrine secreting tumors, as the one in this case study, make systolic arterial blood pressure (SBP) rise from baseline BP 20 seconds after surgical manipulation.² In anticipation of this event, phentolamine, 2 mg IV, has shown to suppress high SBP increases.^{2,13} Labetalol, in 5 mg increments, has proven to control tachycardia accompanying surges in SBP.^{2,6,7} A patient in a hypertensive crisis related to pheochromocytoma may need direct acting vasodilators that reduce preload and afterload, such as nitroprusside.

Nitroglycerin mainly affects capacitance vessels, which only reduces preload.⁷ In this case nitroprusside was effective in controlling this patient's hypertension and should have been initiated earlier.

Nitroglycerine, even at greater than recommended dosages (5 mcg/min initially, with 10-20 mcg/min increases every 3-5 minutes until desired response; consider alternative if >200 mcg/min), was ineffective.¹⁴ There is no absolute dosing limit for nitroglycerin, but risks of toxicity increase with dosages >200 mcg/min; therefore, alternative therapy should be considered.¹⁴

Magnesium sulfate has shown promise as an anesthetic adjunct in pheochromocytoma resection and is the foremost supplemental therapeutic strategy for combating insufficient adrenergic antagonism.¹⁵ Magnesium inhibits release of catecholamines from the adrenal medulla

and from adrenergic nerve terminals.^{2,16} Combined with preoperative α -antagonism, a loading dose of magnesium sulfate (40-60 mg/kg) followed with an infusion of 1-2 grams/hour to achieve serum concentrations between 2-4 mmoles/liter, maintains an arterial pressure within 30 mmHg of the preoperative value.^{2,16} Magnesium sulfate infusions, not including a loading dose, that maintain serum concentrations between 2-4 mmoles/liter improve peripheral perfusion, reduce BP, improve pulmonary edema, and maintain intraoperative hemodynamic stability.¹⁶ A magnesium sulfate infusion may have enhanced hemodynamic stability in the presented case.

Most patients' arterial pressure lowers to below pre-handling arterial pressure 5 minutes after ligation of the last major emptying vein from the tumor.² Persistent hypotension may occur from inadequate intravascular volume, residual effects of preoperative α -adrenergic antagonism, sudden increases in venous capacitance, or hemorrhage.⁷ In addition to replacing blood loss, IV fluids should be administered, from 500 to 3000 mL before and approximately 1200 mL after tumor resection.⁶ In the case presented, IV fluid administration was adequate according to available studies. Norepinephrine, phenylephrine and dopamine have all been recommended to maintain SBP.⁷ In the presented case, norepinephrine proved effective in restoring baseline BP after tumor resection was completed.

Postoperatively nearly 50% of patients remain hypertensive for a few days resulting from elevated catecholamine stores in adrenergic nerve endings, fluid excess, return of autonomic reflexes, inadvertent ligation of renal artery, or presence of a residual tumor.⁷ Persistent hypotension may also occur from blood loss, vascular

compliance alteration, residual adrenergic antagonism, and suppression and/or down-regulation of the remaining contralateral adrenal gland.^{2,7} In the case presented, persistent postoperative hypotension occurred despite adequate fluid resuscitation accompanied by minimal blood loss. The presumptive cause was remaining adrenergic antagonism, suppression and down-regulation of the contralateral adrenal gland, or both.

Based on evidence presented, ideal management of the presented case would have included sevoflurane as the inhalational anesthetic and vecuronium as the muscle relaxant. A loading dose and infusion of magnesium sulfate could have been added to help maintain hemodynamic stability. Most importantly, initiation of nitroprusside should have occurred earlier as it is a more appropriate choice for managing hypertension in this context. This case report provides valuable lessons in the anesthetic care of patients with pheochromocytoma. Although the patient's outcome was satisfactory, this case highlights the importance of carefully considering pharmacologic properties and pathophysiologic context for optimal anesthetic management.

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Suspected Protamine Allergy

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Keywords: protamine allergy, NPH insulin, antiprotamine antibodies

morbidities and be able to implement a plan of care that minimizes risks.

Patient allergies can have a great impact on the anesthetic plan for a case. One such medication which is associated with a higher risk of allergic reactions is protamine. Allergic reactions to protamine can be very severe, ranging from systemic vasodilation to pulmonary vasoconstriction to anaphylaxis.¹ When faced with the potentially life-threatening consequences of a protamine allergy, an anesthesia practitioner must consider the patient's co-

Case Report

A 65 year old, 185.4 cm, 148 kg, ASA 4², white male presented to the electrophysiology lab for an atrial flutter ablation. Past medical history included morbid obesity, obstructive sleep apnea (OSA), coronary artery disease with a history of multiple myocardial infarctions (MI) from 1990-2007, chronic obstructive pulmonary disease (COPD), gastro-

esophageal reflux disease (GERD), and type 2 diabetes. Surgical history included two coronary artery bypass graft surgeries, several cardiac catheterizations, and a coronary stent placed after his most recent MI in 2007. Home medications included aspirin, warfarin, dipyridamole, furosemide, NPH insulin, clopidogrel, and asmanex. Allergies included ticlid, augmentin, ciprofloxacin, and doxycycline. The patient reported having an edematous reaction at the end of his most recent cardiac procedure, but he was unable to provide specific information. An airway exam revealed the patient to have a thick neck, full beard, Mallampati class II, with full range of motion in his neck.

In the holding room, an 18 g intravenous (IV) line was placed in the right hand, and a normal saline infusion was started.

General anesthesia was induced with fentanyl 150 mcg, lidocaine 100 mg, propofol 120 mg, and rocuronium 50 mg. Mask ventilation required a 10 cm oral airway, with one person holding the mask while another ventilated the patient. Direct laryngoscopy was performed using a Miller 3 blade; intubation of the trachea was successful and atraumatic. General anesthesia was maintained with isoflurane. A 20 g arterial line was started in the right radial artery. Femoral sheaths were inserted, heparin was administered, vital signs remained stable, and the procedure was without incident. Protamine was withheld; it was suspected that the patient had a protamine allergy based on his edematous reaction during his most recent cardiac procedure.

At the conclusion the patient was in sinus rhythm, and the femoral sheaths were removed. Due to a suspected protamine allergy, the patient was not administered protamine to reverse the anticoagulation

from heparin. Therefore, the patient was required to remain supine for at least six hours as a precaution to prevent bleeding at the sheath insertion site. Given the patient's significant co-morbidities, and the need to remain supine for six hours, the patient was transported intubated and sedated to the intensive care unit (ICU). The patient was extubated later that day once he no longer had to remain supine. Stable overnight, he was transferred out of the ICU the next morning.

Discussion

This patient reported “blowing up like a balloon” when given a medicine at the end of his most recent cardiac procedure. The patient did not know what medication caused this reaction, but it was speculated that protamine was the causative agent, particularly since the patient takes NPH insulin. There is an increased rate of reaction to protamine among individuals taking NPH insulin.¹ Protamine is a non-human protein that is added to NPH insulin, and individuals that take this type of insulin can produce antiprotamine antibodies (IgG), as well as IgE antibodies.³ The presence of these antiprotamine IgG and IgE antibodies predisposes an individual to an allergic reaction when exposed to protamine. Research has also shown that individuals using forms of insulin that do not contain protamine do not develop these antibodies.³

Allergic reactions to protamine can be very severe, including anaphylaxis, pulmonary vasoconstriction, and bronchospasm, but in a study of patients undergoing ablation of atrial fibrillation, the most common reaction was severe hypotension.⁴ However, individuals that have been previously exposed to protamine and have developed the IgG and IgE antibodies are the group most at risk for an anaphylactic reaction.³

The patient in this case had been exposed to protamine during a previous cardiac procedure, and he was using protamine-containing NPH insulin, therefore he was at an increased risk for a life-threatening allergic reaction.

With this information, the decision was made not to administer protamine to this patient. Therefore he needed to remain supine post-procedure for at least six hours as a precaution against hemorrhage after femoral sheath removal. The anesthesia plan was adjusted to accommodate for this extended period of supine positioning due to concerns about the patient's respiratory status. Concern over the patient's respiratory status stemmed from his body habitus and his medical co-morbidities. The patient was morbidly obese, with a body mass index (BMI) of 43. Such a body habitus leads to an increased incidence of post-procedure atelectasis, especially when a patient is in the supine position.⁵ Obesity also negatively impacts an individual's respiratory dynamics, causing a decrease in lung compliance, tidal volume, functional residual capacity, expiratory reserve volume, forced vital capacity, maximum voluntary ventilation, and respiratory muscle strength.⁵⁻⁹

The patient's co-morbidities of concern included OSA, which decreases a person's airway diameter and increases one's vulnerability to respiratory depressants.⁹ With OSA, the use of general anesthesia and sedatives decreases pharyngeal tone, lowers the body's threshold to respond to hypoxia, and increases the risk for an upper airway obstruction.^{6,9} In addition, the patient had COPD, the hallmark of which is airway obstruction and a subsequent decrease in airflow.¹⁰ Airway obstruction caused by COPD is worsened by obesity.¹¹ The patient remaining in the supine position post-

procedure was also a concern, as the supine position is associated with a decrease in total lung capacity, reserve volume, vital capacity, and overall gas exchange.¹² The patient was at an increased risk for aspiration if left supine after having received general anesthesia.¹³ The patient's obesity and GERD also increased his risk for aspiration.^{14, 15}

The concern for potential airway complications if the endotracheal tube (ETT) was removed in the supine position led to the decision to leave the ETT in place and to transfer the patient to the intensive care unit. The risk of airway compromise or more severe complications was too great, considering the difficulty of mask ventilation during the induction of anesthesia; therefore a more conservative plan was followed.

Allergic reactions can range from benign to life-threatening. When faced with the possibility of a life-threatening reaction, the anesthesia practitioner must be able to adapt the anesthetic plan to meet the specific needs of each patient. In this case, a patient's potential allergy to protamine and his medical co-morbidities led to a more cautious course of action, but one that was ultimately safe and provided a successful outcome.

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Refractory Hypotension with ACE Inhibitor Therapy

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Keywords: angiotensin-converting enzyme inhibitor; angiotensin receptor blocker; intra-operative hypotension, anesthesia, diuretics

Drugs affecting the renin-angiotensin system (RAS) have existed for approximately three decades. Millions of people in the United

States are using medications that affect the renin-angiotensin system to treat hypertension, congestive heart failure, and chronic renal failure.¹ Currently, two classes of drugs affect the RAS, angiotensin-converting enzyme inhibitors (ACEI) and angiotensin receptor blockers (ARB). These drugs are associated with intra-operative

hemodynamic instability. Although it is still a topic of debate whether to withhold ACEI and ARB agents prior to surgery, many anesthesia professionals have learned to expect intra-operative hypotension that is difficult to control by conventional means.²

Case Report

A 68 year old, 115 kg, 175 cm male presented for a circumcision to correct a phimosis. His past medical history included diabetes, COPD, hyperlipidemia, and obstructive sleep apnea. Additionally, the patient had a history of hypertension and had an internal cardiac pacemaker for his underlying atrial fibrillation. The patient was allergic to amoxicillin. Current medications included allopurinol, carvedilol, doxazosin, fexofenadine, fluticasone nasal spray, furosemide, glipizide, ibuprofen, lisinopril, simvastatin, and warfarin. The patient reported taking his lisinopril the morning of surgery as instructed. Laboratory results indicated anemia with a hemoglobin of 11.7 g/dl and a hematocrit of 36.6%. The patient's blood urea nitrogen and creatinine were slightly elevated at 28 mg/dL and 1.62 mg/dL respectively. Physical examination revealed clear breath sounds and a regular cardiac rate and rhythm. The patient's airway assessment was unremarkable for airway abnormalities.

In the holding room, a 20 gauge intravenous (IV) catheter was inserted and a lactated ringer's infusion was initiated. In the operating room, standard monitors were applied. Oxygen at 8 L/min was administered via facemask. Preoperative blood pressure was 156/96 mmHg with a heart rate paced at 65 beats per minute, throughout the case. The patient underwent an uneventful IV induction with lidocaine 80 mg, etomidate 20 mg, sufentanil 10 mcg and succinylcholine 140 mg. The trachea was

intubated without difficulty and positive bilateral breath sounds as well as end tidal CO₂ confirmed.

General anesthesia was maintained with desflurane and 0.6 L/min of air and 0.4 L/min of oxygen. Post induction blood pressure was initially 140/90 mmHg. Approximately, 20 minutes after induction the patient's blood pressure decreased to 120/80 mmHg and despite the reduction of desflurane from 6% to 4% and an increase of IV fluid rate it continued down to 80/50 mmHg. A phenylephrine infusion was initiated and titrated to the desired blood pressure. The phenylephrine infusion was increased with minimal effect, only increasing the patient's blood pressure to 100/60 mmHg.

The procedure was completed 50 minutes after induction and the patient's blood pressure returned to 122/68 mmHg. The patient's oropharynx was suctioned and he was breathing spontaneously. The patient was extubated with positive pressure. His airway was patent and vital signs stable. He was transported to the recovery room on facemask oxygen. In the recovery room the patient was comfortable, alert and oriented. His postoperative blood pressure was 139/82 mmHg and heart rate remained paced at 65 beats per minute.

Discussion

Hypotension is relatively common during anesthesia. Typically, decreasing the depth of anesthesia and administration of IV fluid boluses easily corrects hypotension. Occasionally, it is treated with sympathomimetic drugs such as phenylephrine, ephedrine, or epinephrine. The literature documents a high incidence of intra-operative refractory hypotension with

patients taking medications for hypertension that affect the RAS.^{3,4}

Current research on this topic indicates the combination of an ACEI or ARB used in conjunction with a diuretic is the best predictor of post-induction hypotension.² The patient in this case was taking lisinopril, an ACEI, and furosemide, a diuretic, making his risk of intra-operative hypotension high. The literature indicates that patients taking ARB and ACEI required high doses of phenylephrine to maintain normotension.⁴ The phenomenon is specific to ACEI and ARB used with diuretic therapy. Other combinations of anti-hypertensives do not display a noticeable frequency of hypotension, such as calcium channel blockers used with diuretic therapy.²

Anesthesia induction inhibits the sympathetic nervous system, which is a compensatory mechanism to control organ perfusion and blood pressure. Hypotension post-induction can be antagonized by the RAS.¹ Angiotensin II is a serum peptide that increases systemic vascular resistance by vasoconstriction. Its antecedent, angiotensinogen, is split by renin to form angiotensin I. Next, the angiotensin-converting enzyme further splits angiotensin I to form angiotensin II. Angiotensin II increases blood pressure by binding to the angiotensin type-1 receptor to produce vasoconstriction. Patients taking medication that affect this system are left with minimal compensatory mechanism to counter the hypotensive effects of general anesthesia.^{2,5,6} Due to the inhibition of the RAS from lisinopril, the patient in this case was left with minimal capacity to compensate for the reduction of sympathetic nervous system (SNS) tone related to general anesthesia which lead to hypotension after induction.

The RAS and the SNS are interrelated. The results of two animal studies describe the interaction between the RAS and the alpha-1 adrenergic receptor. Hu et al observed that angiotensin-II stimulates alpha-1 adrenergic receptor protein expression in vascular smooth muscle of rat aorta.⁷ The more angiotensin-II present the more alpha-1 adrenergic receptors present. This finding suggests that in the presence of angiotensin-II inhibition there is a decreased alpha 1 adrenergic receptor protein expression. An explanation for the reduced effectiveness of sympathomimetic drugs can be postulated. Angiotensin-II facilitates vasoconstriction indirectly through the expression and function of the vascular alpha-1 adrenergic receptor gene. Godinez-Hernandez et al. demonstrated low doses of an ACEI inhibit this phenomenon.⁸ The patient in this case study was given large doses of a direct alpha-1 agonist, phenylephrine, with a minimal vasoconstrictive response. The research of Godinez-Hernandez et al and Hu et al lend an explanation for the refractory hypotension experienced during the case.^{7,8}

These studies begin to predict why hypotension can be refractory to sympathomimetics but potentially reactive to other vasoconstrictors such as vasopressin. Vasopressin agonizes V1 vascular specific receptors to cause vasoconstriction. Vasopressin utilizes a separate mechanism to the SNS and angiotensin-II activation to increase systemic vascular resistance and therefore has been used effectively to counter refractory hypotension in the patient receiving ACEIs and ARBs.^{2,9} The use of vasopressin in this way is not a standard practice and although considered was not utilized for this patient. Directed research is needed on the use of vasopressin for general anesthesia induced hypotension in patients with RAS inhibition.

Prevention is an additional way to mitigate hypotension associated with RAS. Patients taking drugs that affect the RAS the morning of surgery often develop a post-induction sympathomimetic refractory hypotension.⁶ Discontinuation of ACEI and ARB has been proposed to minimize the hypotension associated with these therapies. The patient in this case was not instructed to discontinue his ACEI, as there is no established standard practice regarding if or when to discontinue these medications.

Research confirms that patients taking an ARB the morning of surgery developed more severe and frequent episodes of hypotension post-induction and required more vasoconstrictor treatment when compared to discontinuing these drugs 24 hours before surgery.⁶ The half-life of most of the medications affecting the RAS is approximately 10 hours. Patients instructed to discontinue their ACEI or ARB greater than 10 hours before surgery had a 20% points lower incidence of hypotension post-induction than patients that continued their ACEI or ARB therapy 10 hours prior to surgery.¹⁰

The patient in this case took the ACEI as instructed the morning of surgery and experienced hypotension during anesthesia. There is no consensus regarding the discontinuation of ACEI and ARB prior to surgery and the use of vasopressin to correct hypotension associated with these drugs. Standards based on evidence weighing the benefits and risks associated with these issues needs to be established.

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Anesthesia for Adult Aortic Coarctation Repair

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Keywords: Adult coarctation of aorta, anesthesia, lumbar drain, aortic cross clamp, congenital heart disease.

Most patients with unrepaired coarctation of the aorta die before the age of 50.¹ Early death is related to severe hypertension, left ventricular hypertrophy, congestive heart failure, myocardial infarction, aortic dissection, stroke or infective endocarditis.^{1, 4} Although surgical management of coarctation of the aorta was first reported 65 years ago, reported cases in patients older than 50 years of age are rare.^{1, 11} This case report describes the anesthetic considerations for managing hemodynamic changes and maintaining adequate distal perfusion during cross clamping and preventing complications during coarctation repair in a middle aged patient.

Case Report

A 58 year-old, 65in, 78kg female with a history of persistent hypertension, sinus bradycardia, and hyperlipidemia presented for surgical repair of coarctation of the aorta. Medications included hydrochlorothiazide,

clonidine, and losartan. Echocardiography demonstrated an ejection fraction of 60-65%, trace aortic insufficiency, and post ductal coarctation with a systolic gradient of 22 mmHg. Computed tomography imaging confirmed a coarctation with an estimated diameter of approximately 1cm immediately preceded by an area of aortic dilation. Pre-operative vital signs revealed a 20mmHg difference in blood pressure (right arm greater than the left), bounding upper extremity and very weak lower extremity pulses. Due to increased exercise intolerance, the decision was made to repair the coarctation.

The patient was premedicated with midazolam, standard monitors applied, supplemental oxygen administered via nasal cannula and a dexmedetomidine infusion initiated. A lumbar cerebrospinal fluid drain catheter, right radial and femoral arterial lines and right internal jugular Swann-Ganz catheters were inserted. The dampened femoral MAP was 20-30 mmHg less than the radial MAP.

Following pre-oxygenation, general anesthesia was induced with lidocaine, fentanyl, etomidate and succinylcholine. Direct laryngoscopy was performed and the trachea intubated with a 37Fr left sided double lumen tube (DLT). Tube placement and left lung isolation were confirmed by a combination of auscultation and fiberoptic bronchoscopy before and after positioning in the right lateral position. Anesthesia was maintained by a combination of air, oxygen, isoflurane, dexmedetomidine, and intermittent boluses of fentanyl, midazolam and pancuronium. The ICP was maintained at less than 50% of the CVP by intermittently draining CSF. A total of 250mL of CSF was drained over a 7 hour period.

Blood pressure was controlled with nitroglycerin and sodium nitroprusside. MAP averaged 60mmHg following induction, but decreased to 40mmHg during a test cross clamp. Therefore, heparin was administered and a partial left heart bypass initiated. Femoral MAP was maintained above 50mmHg during cross clamping. Mannitol was also administered at this time. At the end of the procedure, protamine was administered, two-lung ventilation resumed and the DLT exchanged for a 7.5 ETT. The patient was transferred intubated to the CVICU and extubated a few hours later. Pain control was achieved via a thoracic epidural catheter. Postoperative physical assessment was significant for mild facial edema, no neurologic or renal complications were noted. She had bounding lower extremity pulses with equal radial and femoral arterial blood pressures. The patient was transferred to the medical surgical floor and recovered with no complications.

Discussion

Coarctation of the aorta is defined as a congenital disorder whereby an aortic segment constricts with localized medial thickening, some infolding of the medial and superimposed neointimal tissue.¹⁰ Even though the coarctation can involve a long segment of the aorta, it typically consists of a discrete disc-like ridge extending into the aortic lumen.⁴ It usually presents during the first few weeks of life, however in a small percentage of patients this disease process goes unrecognized until adulthood.^{7, 10} Coarctation which occurs just distal to the left subclavian artery at the site of aortic ductal attachment is referred to as postductal coarctation and occurs most frequently in young adults. It is most commonly detected by murmur, hypertension or blood pressure discrepancies during routine examination.^{1, 4, 7} Treatment is usually considered when the systolic gradient is >20mmHg with patient sedated during catheterization.⁸

Surgical repair of the thoracic aorta presents a tremendous challenge.⁹ The nurse anesthetist must be knowledgeable of the physiologic perturbations of the lateral position and one-lung ventilation, hemodynamic changes associated with aortic cross clamping, partial cardiopulmonary bypass, and prevention of spinal cord ischemia and renal failure.^{4, 9} Spinal cord injury (paraplegia) is one of the most serious complications of thoracoabdominal aortic surgery, with morbidity and mortality as high as 40%.^{3, 7} Spinal cord ischemia results from interruption or decrease blood flow to the anterior spinal artery or the radicular branches of the intercostals arteries.⁴ During aortic cross clamping, hypotension and decreased distal perfusion to these arteries

decreases the spinal cord perfusion pressure (SCPP) based on the formula:

$$\text{SCPP} = \text{MAP} - (\text{CSFP or CVP} \text{ [whichever is higher]}).$$
³

Where CVP is the central venous pressure and CSFP is the cerebrospinal fluid pressure.

Several techniques can be implemented to protect the spinal cord against ischemia. These include hypothermia, avoiding glucose containing solutions, lowering the CSFP, minimizing cross clamp time, using partial left heart bypass and pharmacologic interventions (steroids, barbiturates, and mannitol).⁷ Of these methods, the most promising is CSF drainage since aortic cross clamping cause acute rises in intracranial and intraspinal pressures.⁹ During the case under consideration continuous monitoring of CSFP and intermittent drainage of CSF was instituted. This method has been shown to be superior to continuous CSF drainage with decreased incidence of intracranial hematoma.² Decreasing CSFP while maintaining MAP leads to an increase in SCPP.³

During the procedure, test aortic cross clamping caused the femoral MAP to decrease from 60 to 40mmHg. Since a femoral MAP >50mmHg is generally believed to be required for adequate renal and spinal cord perfusion⁴ a partial left-heart bypass was instituted. With a beating heart, oxygenated blood is shunted from the left atrium and delivered to the distal aorta. This setup creates two separate but independent circulations; a proximal circulation that delivers oxygenated blood from left ventricle to proximal aorta and a distal circulation which delivers blood drained from the left atrium to distal aorta through a centrifugal pump. The centrifugal pump can be adjusted to control distal perfusion.⁹ In

this case the centrifugal pump was set to delivery 50% of the cardiac output to the distal aorta maintaining a femoral MAP of 55-60mmHg during cross clamping. Proximal hypertension is controlled during cross clamping by adjusting centrifugal pump instead of titrating vasodilators. Vasodilators are reserved for instances of both proximal and distal hypertension. To avoid a coagulation catastrophe heparin must be administered prior to institution of bypass.⁹

Prophylactic administration of mannitol has been shown to decrease intracranial pressure (ICP) by increasing plasma osmolarity. Increased plasma osmolarity draws water from the brain by producing an osmotic gradient.⁷ Decreased ICP leads to decrease in CSFP, thus increasing the SCPP. Beside its neuroprotective properties, mannitol has been used for renal protection. However, to be effective mannitol must be administered prior to the onset of renal failure.⁶

Surgical correction of an aortic coarctation in an adult presents multiple challenges to the nurse anesthetist. Surgical exposure requires a left sided thoracotomy with one lung ventilation, aortic cross clamping causes acute proximal hypertension but distal hypotension. This places the patient at risk for postoperative paraplegia and renal failure. By carefully controlling the proximal and distal aortic and intraspinal pressures, these complications can be minimized. This requires close collaboration and communication with the surgeon, the perfusionist and the entire operating room team.

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Anesthesia for the Epileptic Patient Treated with Vagal Nerve Stimulation

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Keywords: seizures, epilepsy, vagus nerve stimulator, anesthesia, cerebral palsy

Epilepsy affects two million people in the United States, 3,000 of whom are below the age of 14.¹ Seizures refractory to medical management and not amenable to surgical correction amendable may be treated with an adjunctive therapy, vagal nerve stimulation

(VNS). These devices have been shown to improve the quality of life, minimize the number of anti-epileptic drugs (AEDs), and reduce the incidence of seizures in epileptic patients.^{2,3,4} Given the association between epilepsy and cognitive impairment, this population often requires anesthesia for standard office-based procedures.^{1,5} This case report will discuss the anesthetic

considerations for an epileptic patient receiving VNS therapy.

Case Report

An eight year old, 26 kg male patient presented for complete oral rehabilitation and botulinum toxin injections of the salivary glands. Past medical history was significant for spastic cerebral palsy (CP), including quadriplegia, severe cognitive delays, intractable generalized seizures (seven – ten daily seizures), asthma, and gastroesophageal reflux disease. Past surgical history included VNS device placement, nissen funduplication, and gastric tube placement. The patient's medications included lamictal, levetiracetam, zonisamide, clonazepam, glycopyrrolate, lansoprazole, fluticasone, and ipratroprium. Preoperative blood pressure was 101/67 mmHg, heart rate 125 beats/minute, respiratory rate 18 breaths/minute, and oxygen saturation (SaO₂) was 97% on room air. Preoperative Laboratory values were not ordered. His physical status was classified as an ASA IV given the severity of the seizures and cerebral palsy. A general anesthetic with nasotracheal intubation was planned. Because of the severity of the seizure history the decision was made to keep the VNS on during the procedure.

Once in the operating room, an inhalation induction was performed with O₂ 3 LPM and nitrous oxide 7 LPM and sevoflurane incrementally titrated to 8%. Approximately two minutes after mask induction, generalized seizure activity was noted. Nitrous oxide was discontinued, a 24 gauge peripheral IV was placed, and midazolam 2 mg IV was administered in an attempt to halt seizure activity. Once seizure activity ceased and the patient remained hemodynamically stable, the decision was made to proceed with the surgery.

The patient was oxygenated with 7 LPM O₂, cricoid pressure was applied, and laryngoscopy was performed after propofol 60 mg, rocuronium 15 mg, and fentanyl 10 mcg to facilitate nasotracheal intubation. A cuffed 5.5 endotracheal tube was passed easily through the right nare. Direct laryngoscopy with a Miller two blade revealed a grade I view, and the endotracheal tube was easily advanced into the trachea using Magill's forceps. End Tidal CO₂ and bilateral breath sounds were confirmed and anesthesia was maintained with 3.0% sevoflurane in O₂100%. Approximately 30 minutes into surgery, additional seizure activity was noted. Midazolam 1 mg and fentanyl 5 mcg were given IV without successful resolution of seizures. The anesthetic agent was switched from sevoflurane to isoflurane, and sodium thiopental 50 mg IV was administered incrementally with successful resolution of seizure activity. An arterial blood gas sample was drawn and results were within normal limits. Prior to starting the surgery grounding pads were placed far away from the VNS to minimize electrocautery interference.

Upon completion of surgery isoflurane was discontinued and the patient was extubated after meeting criteria. The patient was taken to the post anesthesia care unit for recovery. The patient demonstrated no additional seizure activity in the immediate postoperative period, and was discharged to home that day. No increase in seizure activity was noted upon discharge.

Discussion

CP is a disorder of varying symptoms due to neurologic lesions or abnormalities occurring in early development. It affects 0.7 per 1000 live births in the United States.⁶ Children with CP often suffer from mild to

severe cognitive impairment, and have a high incidence of seizure disorders.⁷ The severity of the seizure disorder is directly proportionate to the degree of disability.^{1,5}

Epilepsy is neurologic disorder of recurring seizure activity due to inherent or acquired causes.⁷ Pharmacological treatment with AEDs is the initial treatment option for epileptic seizures. Despite advances in safety and efficacy of newer AEDs, 25-30% of epileptics are resistant to medical management.¹ Non-pharmacologic treatment options for those with intractable epilepsy include epilepsy surgery, cerebellar stimulation, thalamic stimulation, ketogenic diet, and VNS.^{1,3}

Vagal nerve stimulator placement is a common surgical treatment option for epileptic patients who are resistant to medical management and/or are not candidates for epilepsy surgery.^{2,3,4} A retrospective study by Helmers et al. demonstrated a reduction in seizures on average by 36.1% at three months and 44.7% at six months after VNS implantation.² VNS involves the interruption of neuronal excitability through intermittent transmission of electrical impulses to the left vagus nerve, but the exact mechanism is not known. The unit is implanted subcutaneously in the left upper chest and connects to the left vagus nerve in the neck via a lead wire.⁸

Providing safe anesthesia for the epileptic patient treated with VNS presents a unique set of challenges for the anesthesia practitioner. Epileptic patients treated with VNS are often cognitively impaired, thus requiring anesthesia for routine office-based procedures (i.e., complete oral rehabilitation, etc).^{1,5} Knowledge of the VNS generator and its possible impact on the surgical equipment in the operative milieu is key.

Comparable to a pacemaker, electrocautery may impair and/or interfere with the VNS generator. Therefore, grounding pads should be placed far away from the VNS unit and its functional status should be assessed postoperatively.^{1,4} Unlike the magnet used for cardiac pacemakers, a specialized magnet is provided with the VNS generator to halt or prevent seizure activity or turn off the VNS unit and should be available. To prevent or shorten a seizure, the magnet is placed over the VNS for less than one second, thereby producing a surge of stimulation to the vagus nerve. Conversely, the VNS can be temporarily inhibited by positioning the magnet over the unit for greater than 65 seconds.⁴ Given the high seizure frequency of the patient presented in this case, the decision was made to leave the VNS unit on and maintain its current settings. A special VNS magnet was available.

Vagal nerve stimulation-related side effects are common and may increase the anesthetic risk in this patient population. Reports of respiratory issues during VNS include: decreased airway flow and respiratory effort, airway obstruction, and various types of laryngopharyngeal dysfunction such as vocal cord adduction and/or paresis, glottis obstruction, and aspiration.⁴ Vocal cord dysfunction may be due to direct surgical trauma or secondary to vagus nerve stimulation. Thus, patients treated with VNS should be considered at risk for and managed accordingly. Of additional consideration is the fact that one third of intractable epileptics are diagnosed with obstructive sleep apnea (OSA). The combination of VNS and OSA has been shown to increase airway obstruction during anesthesia. Strategies to minimize respiratory complications consist of: turning off the VNS unit or decreasing vagal nerve stimulation frequencies and intensity

perioperatively, prolonged monitoring in the postoperative care unit, and the use of short-acting anesthetic agents.⁴ In this case an inhalation induction with intravenous placement after induction was chosen given the patient's age and severity of cerebral palsy, cognitive dysfunction and seizures. However after intravenous line placement cricoid pressure was applied and direct laryngoscopy was performed; a cuffed endotracheal tube was placed to minimize the patient's aspiration risk.

The anesthetic plan for the epileptic patient must factor in common side effects of AEDs, the pharmacokinetic-altering effects AEDs have on anesthetics, and the pro and anticonvulsant effects of anesthetic agents. Side effects of AEDs are numerous and vary depending on the agent. Drowsiness and sedation are commonly reported side effects of AEDs. Carbamazepine, phenytoin, and phenobarbital enhance the liver's metabolism of propofol, muscle relaxants, and opioids, and other drugs so dosing adjustments may be needed to achieve the appropriate clinical effect.¹

Intravenous anesthetic agents, such as methohexital, fentanyl, etomidate, and ketamine have been shown to be both pro and anticonvulsant in nature. While conflicting, the majority of literature supports the safe administration of propofol to epileptic patients. Halogenated inhalational agents produce dose-dependent decreases in EEG amplitude and frequency. Though desflurane and isoflurane produce burst suppression at non-toxic concentrations, sevoflurane's effects on EEG activity and seizure-inducing potential are widely conflicting. Evidence of sevoflurane's pro and anticonvulsant properties exist,^{9,10} with two case studies reporting seizure-like activity with sevoflurane induction.^{8,11} Nitrous oxide's

effects on seizure potential remain controversial.^{1,12} In the case presented, the patient had a seizure upon induction with sevoflurane and nitrous oxide. Although the propensity for both agents to produce epileptiform activity remains controversial, an intravenous induction with midazolam and propofol may have been a safer alternative. However this would have necessitated placement of an intravenous line prior to induction, which may have been difficult in a child with severe CP.

A study conducted by Niesen et al found that perioperative seizure risk is high in epileptic patients with frequent baseline seizures and seizure activity prior to surgical admission.⁵ Although frequency of perioperative seizures is unrelated to anesthetic technique or surgical procedure, the anesthesia practitioner should be prepared to treat perioperative seizures with intravenous or rectal benzodiazepines or rapid onset barbiturates, and maintain airway patency and patient safety during seizure activity.⁵

Thorough preanesthetic evaluation should include seizure severity and frequency and an updated list of medications.¹ Anesthetic considerations for epileptic patients treated with VNS include a thorough understanding of the VNS generator and its physiologic implications, and the effects of anesthetics on seizure threshold and potential interactions with AEDs.⁴ Perioperative seizures occur often in epileptics with frequent baseline seizures and recent seizure activity, so the anesthesia practitioner must be prepared to treat seizures in the perioperative period.⁵

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Neonatal Progeroid Syndrome

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Keywords: progeria, anesthesia, Neonatal Progeria syndrome, NPS

Wiedemann-Rautenstrauch syndrome (WRS), or Neonatal Progeria syndrome (NPS), is a rare medical condition often referred to as “accelerated aging” in the pediatric population. WRS is a cluster of conditions causing an “old man” appearance and characterized by developmental delays, minimal subcutaneous adipose,

microcephaly, heart defects and natal teeth.¹⁻⁶ The life expectancy is a few months to a few years.^{2,5,6} As progeroids age they develop medical conditions associated with aging such as atherosclerosis, angina, stroke, coronary vascular disease, and arthritis.^{2,7,8} The manifestations of WRS present many anesthetic considerations. This case report describes general anesthesia in a 7-year-old with NPS.

Case Report:

A 7 year-old, 15 kg, 42 in (BMI 13) female with a history of NPS was admitted for computed tomography, x-rays, orthopedic and cardiology examinations and dental restoration under general anesthesia. The patient was microcephalic with global developmental delay, violent outbursts, self-hitting, and a partial complex seizure disorder for which she was taking valproate. Atrial septal and ventricular septal defects were present at birth, however review of the patient's medical records revealed they had closed and she had normal cardiac function. Her subcutaneous adipose was minimal to absent, and the patient had joint contractures of the upper extremities with shallow hip sockets and mild scoliosis. Her cardiac examination was unremarkable with normal heart sounds. She had a small oral opening and poor dentition. She did not tolerate an oral examination.

After obtaining informed consent from her mother, the patient was transferred to the operating room for an inhalation induction with parental presence. Anesthesia was induced with 8% sevoflurane and 4 L/min oxygen and 4 L/min nitrous oxide. At the same time a pulse oximeter, blood pressure cuff and ECG were applied. Once inhalation induction was complete a peripheral intravenous catheter was placed and a propofol infusion was instituted at 200 mcg/kg/min. After administration of 2 mg/kg bolus of propofol the patient was intubated with a 4.5 cuffed endotracheal tube. The endotracheal tube was selected for intubation based on the patient's #5 digit size. The patient had a grade II view with an anterior laryngeal inlet and small oral aperture; however the patient was easily intubated. The endotracheal tube was secured, oral airway placed, eyes taped

closed and a skin temperature probe applied to the patient's forehead.

The patient was transported to the radiology suite with propofol infusion maintained at 200 mcg/kg/min and was manually ventilated with oxygen using a pediatric Jackson-Reese open breathing circuit. Hip radiographs and a non-contrast head computed tomography were obtained. Manual ventilation was maintained throughout with momentary pauses during the CT scan. The patient was then returned to the OR. Propofol infusion was maintained during transit. Once back in the OR, anesthesia was maintained with sevoflurane at 1 MAC and the propofol infusion was titrated between 50 – 100 mcg/kg/min. Radiant heat lamps were applied. In the OR orthopedic surgeons carried out a hip examination. A 12-lead EKG was obtained and the cardiology team completed a transthoracic echocardiogram. The oral surgeon then performed full dental restorations. Upon completion of the dental procedure the propofol infusion and inhaled agents were discontinued. The patient was prepared for emergence and extubated uneventfully.

Discussion:

To date there are no case reports describing anesthetic management of a child with NPS. One letter to an editor exists describing propofol infusion syndrome in an NPS child after 6 hours of 300 mg/hr propofol infusion.⁹

NPS is a rare autosomal recessive condition that carries multiple co-morbidities that can imp[act anesthesia management.⁵ Although there are some reported cases of children with NPS surviving to teenage years, most die in infancy or toddlerhood secondary to respiratory illness and subsequent sepsis.

^{2,3,5,6} Microcephaly, developmental delays, brain lesions, and respiratory infections are common. Growth retardation, both intrauterine and post-natal, and natal teeth (teeth present at birth) are diagnostic features. ¹⁻⁶ NPS patients tend to have small, retrognathic mouths with arched or “V” shaped palates. ^{4,5} Laryngeomalacia and gastroesophageal reflux may be present. ² Adipose accumulation in the buttocks is a characteristic feature of NPS. However, similar to other progeroid conditions, minimal subcutaneous adipose elsewhere is common as is prominent superficial vasculature, especially on the scalp. ^{1-6, 8}

Unlike Hutchinson-Gilford Progeria (HGP) syndrome, a more common yet very rare condition where diagnosis is typically made between one and two years of life, NPS is often diagnosed at birth. Mental retardation tends to be absent in HGP and the typical lifespan is 13 years, whereas that of the NPS child is a few months to years. ¹⁻⁸ Although they are different syndromes, many progeroid features overlap. As the HGP patient ages atherosclerosis, cerebral vascular accidents, angina and myocardial infarctions become prominent, with MI being the leading cause of mortality. ^{7,8} Initial onset of angina in the progeroid occurs at approximately 6 years of age. ⁸ In literature, discussion of cardiovascular disease specifically related to the NPS child is nearly absent. This is perhaps due in part to the rare occurrence of NPS as well as early mortality in the NPS child. In this case cardiovascular concerns were paramount when considering anesthesia technique.

Developmental delay of varying degrees is common in the NPS child. In this case the patient showed global developmental delay with combative behavior and documented history of hitting herself. Although the primary cause of death reported in the NPS

patient is pulmonary in nature, they are also at increased risk for intracranial hemorrhage especially from trauma. ^{2,3} Prolonged combativeness for IV placement or slow induction were of concern, so a rapid inhalation induction with sevoflurane and nitrous oxide was planned to minimize unnecessary agitation, hypertension, and to decrease the risk of intracranial hemorrhage.

Considering the small oral opening, high palate, poor dentition and anterior larynx difficult airway equipment should be immediately available. Traditional pediatric airway formulas may not be helpful in preparing intubation equipment for the progeroid since these formulas are typically age-based. We prepared several laryngoscope blades, and had a glide scope and fiberoptic scope available. Endotracheal tube size was based on the patient’s #5 digit size. Oral endotracheal intubation in this patient was uneventful.

The prominent vessel visibility in the progeroid tends to facilitate peripheral IV placement. This visibility is, in part, secondary to the paucity of subcutaneous adipose which may further inhibit thermoregulation in the anesthetized child. In this instance a skin temperature probe was placed in lieu of esophageal temperature probe to facilitate oral access during the dental restoration. Active warming measures were instituted with radiant warming lights. Our patient remained normothermic.

In conclusion, preparing and carrying out a case for a NPS child requires consideration of patient size, current health status and common complications associated with progeroids. Patient size has implications on equipment choice such as intubation equipment and anesthesia circuit size, as well as thermoregulation, fluid and medication administration and ventilator

settings. These tend to be diminutive relative to patient age. The young NPS patient's vulnerability to sepsis and pulmonary morbidity must be considered. A thorough pulmonary evaluation should be performed preoperatively and extra attention to surgical site preparation and antibiotic administration should be made. In this case the patient age was approaching that of the common age for atherosclerosis and cardiovascular disease as seen in the HGP patient. With no literature speaking to the risk of cardiovascular disease in the aging NPS child, prudence should be employed in planning anesthesia care considering some important features are shared among progeroids.

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Intraoperative Hypothermia and Delayed Awakening

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Keywords: thermoregulation, hypothermia, delayed awakening, neuromuscular blockade, residual weakness

Anesthesia practitioners are well aware that anesthetized patients are at risk of hypothermia. Both neuraxial and general anesthetics impair the patient's ability to

recognize and respond to alterations in temperature, both behaviorally and autonomically.¹ Intraoperative hypothermia has many commonly known physiologic effects, the most severe of which are increased intraoperative blood loss, myocardial ischemia, and delayed wound healing or infection.² An additional, less

frequently discussed effect of hypothermia may be seen with drug metabolism: specifically, changes in temperature alter the effects of neuromuscular blocking agents.³ The anesthesia practitioner must account for these effects when dealing with cases of hypothermia.

Case Report

A 54 year old, 58 kg female was admitted for left total hip replacement. The patient's surgical history was unremarkable with anesthetics complicated only by nausea. The medical history was significant for osteoarthritis, hypertension, gastroesophageal reflux disease, hypothyroidism, hepatitis C virus (HCV), cirrhosis, smoking, and a gastrointestinal bleed. Medications included aldactone, avelox, B-complex caps, furosemide, propranolol, protonix, synthroid, temazepam, and ultram. A preoperative complete blood count revealed a hemoglobin of 10.4 g/dL, hematocrit of 30.6% and platelet count of 47,000. A comprehensive metabolic panel (CMP) from one month prior revealed an albumin of 2.7 g/dL, AST of 119 units/L, ALT of 111 units/L with all other values within normal limits. Preoperative vital signs were unremarkable including a temperature of 36.7 °C.

The patient received a left femoral nerve block of 30 mL of 0.5% bupivacaine with epinephrine and clonidine 50 mcg. Intravenous (IV) sedation included fentanyl 100 mcg and midazolam 4 mg. In the operating room, she was preoxygenated then induced intravenously with fentanyl 50 mcg, lidocaine 50 mg, propofol 120 mg, and rocuronium 50 mg. Direct laryngoscopy was performed with a Miller 2 laryngoscope. A 7.0 endotracheal tube was advanced through the vocal cords and secured at 21 cm at the

lip. Placement was confirmed via auscultation of equal bilateral breath sounds and detection of end tidal carbon dioxide. An esophageal stethoscope was placed. The patient was positioned for the operation. As part of standard monitoring, the patient's core temperature as measured through the esophageal stethoscope went from 35.0°C after positioning to 34.8°C at the end of the procedure. A fluid warmer and forced air warmer was utilized throughout case. Vecuronium 4 mg IV was given to maintain surgical relaxation during the three-hour procedure with the last 2 mg dose given 45 minutes prior to the procedure end. After induction, no additional narcotics were utilized. Isoflurane was utilized throughout case to maintain general anesthesia but end tidal concentration was noted to be 0% on emergence.

During emergence, the patient was noted to have one twitch with train of four (TOF) monitoring. Neuromuscular blockade was antagonized with neostigmine 3 mg and glycopyrrolate 0.6 mg IV. She had four twitches with TOF stimulation and equal response to double burst stimulus after administration of the reversal agent. The patient, however, was lethargic and not following commands. She appeared weak with decreased muscular tone and was not able to maintain adequate spontaneous ventilations (minute ventilation less than 2L). The patient was taken to the post anesthesia care unit (PACU) with assisted ventilations and placed on T-piece upon arrival. Vital signs were stable other than an oral temperature of 35° C. The patient was uneventfully extubated within 30 minutes of arrival to PACU at which time she was noted to have an oral temperature of 36° C.

Discussion

Patients receiving both regional and general anesthetics are at risk for hypothermia related to the loss of their ability to adapt to the cold operating room environments via methods such as vasoconstriction and shivering.⁴ Hypothermia has many adverse effects that are recognized by anesthesia practitioners including increased blood loss, decreased wound healing, decreased patient comfort, increased length of stay in the recovery room, and increased postoperative shivering with subsequent increases in oxygen consumption.^{2,5} Altered metabolism of anesthetic agents is a less commonly recognized side effect of hypothermia. Multiple studies have recognized the effects of hypothermia on neuromuscular blocking agents, and residual paralysis continues to occur after anesthesia despite advances in monitoring capabilities.^{3,6-8}

Studies have found that the effects of hypothermia on neuromuscular blockers (NMBs) are likely related to a decrease in clearance of the NMBs at lower core temperatures.⁷⁻⁸ This decrease in metabolism prolongs the action of the drug.³ A second hypothesized relationship between hypothermia and muscle relaxation involves neostigmine, the most commonly used reversal agent.³ Neostigmine has a prolonged time to maximum effect in hypothermia but displays no change in clearance, maximum effect or duration of action.⁹ Not only are the NMBs altered, but the strength of muscle tissue itself is also altered with decreased core temperatures. The adductor pollicis muscle, which causes the thumb to adduct and is one of the most frequently used sites for monitoring twitch response during the use of NMBs, has a 10% decrease in twitch response from baseline for every decrease in core temperature of 1°C.³ When muscle

relaxation is achieved with vecuronium, the decrease in twitch response increases to 20% with each one degree drop in temperature.³ Heier and Caldwell suggest that all patients needing full muscle function after surgery should be warmed to 36°C after anesthesia prior to discontinuing ventilatory assistance.³

In addition to noted hypothermia, the above patient had multiple comorbidities that would alter her metabolism of NMBs including HCV, cirrhosis, hypothyroidism, and home use of benzodiazepines and other agents metabolized by the liver. She was noted to be euthyroid at the time of surgery, but her liver function tests were elevated to twice the normal value and her serum albumin was low enough to cause altered protein binding. Though this article primarily addresses hypothermia, all of these comorbidities likely played a role in the patient's delayed recovery from the NMBs.

She was induced with rocuronium 50 mg (0.86 mg/kg), and subsequently regained one twitch 30 minutes after the dose. She recovered 2/4 twitches an hour after the initial dose at which point she was given 2 mg of vecuronium (0.034 mg/kg). Her final dose of vecuronium was almost an hour later when she regained one twitch, approximately 45 minutes before the procedure end. While switching between NMBs as in this case is common practice in some institutions, the practice is becoming less frequent with the development of newer NMBs with fewer side effects, and more research is necessary in this area to determine whether these combinations are additive, synergistic or antagonistic.¹⁰

The patient's temperature in the holding area was within normal limits at 36.7° C but quickly dropped to 35° C during the prolonged positioning for the procedure. She

was given no additional narcotics after induction. During the attempted awakening, her end tidal isoflurane decreased to 0%. Her twitch response to the NMBs was monitored throughout the case. It would have been prudent to decrease the dosing after noticing her prolonged reaction to the initial and subsequent doses. The NMB was antagonized at an appropriate time at a dose of 0.05 mg/kg. She regained 4/4 twitches with no fade and an equal response to the double burst stimulus yet remained lethargic and unable to follow commands with a spontaneous minute ventilation of less than 2L. Considering her neostigmine dose (0.05 mg/kg) was on the lower side of the accepted range (0.04-0.08 mg/kg) and her clinical presentation of neuromuscular weakness, she may have benefited from an additional dose of neostigmine. Other than allowing more time for the NMB to be eliminated and the cholinesterase inhibitor to effectively antagonize the NMB, the only other change for the patient in the PACU was that she was warmed to 36°C.

Methods of warming patients vary in different practices but often include forced air warming and fluid warming. Hasankhani and associates¹¹ found that core temperatures were 0.5 to 0.6°C warmer in patients whose IV fluids were warmed. A second article discussed the technology behind forced air warming. Bräuer and Quintel¹² found that forced air warming alone may be ineffective. Multiple studies conclude that patients should be pre-warmed for 30-60 minutes prior to the anesthetic to aid in preventing heat loss from redistribution.¹²⁻¹⁴

The Surgical Care Improvement Project (SCIP), a public report program aimed at grading healthcare quality, focuses on maintenance of normothermia as a quality indicator.¹⁵ SCIP's indicators of quality can

be met by keeping the patient's temperature above 36.0°C or by utilizing forced air warming devices on patients who remain hypothermic.¹⁵ The quality indicator applies to patients receiving both general and regional anesthetics. In the case of the above patient, both a forced air warming device and a fluid warmer were utilized at maximum temperatures.

Determining the exact cause of this patient's delayed awakening from the anesthetic is a complex task. In addition to her likely altered liver metabolism at baseline, one probable cause is an alteration in NMB metabolism due to marked hypothermia. Care should have been taken to utilize measures including a hot air warmer or fluid warmer sooner after positioning was complete. The patient would likely have benefited from a higher room temperature prior to induction and a subsequent readjustment of the room temperature after the patient was safely positioned and covered. Considering the patients' multiple comorbidities that could affect the anesthetic, more vigilance given to her core temperature and actions to treat it may possibly have eliminated the additional risks of hypothermia.

This case emphasizes the importance of considering complications associated with hypothermia such as delayed recovery from neuromuscular blockade.

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Blood Component Ratios in Massive Transfusions

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Keywords: Blood, component, ratio, massive, transfusion

Blood transfusion is a key element in the intraoperative management of trauma patients. With hemorrhagic shock being the second most common cause of death in trauma patients¹, it is important to be able to manage and replace traumatic blood loss. In recent research the ratio of red blood cell to platelet and plasma transfusions has been questioned on the battlefield. This is beginning to modify practice in the operating room. Mortality rates significantly decrease when the ratio is 1:1 versus replacing one unit of plasma and platelets for every four or more units of Packed Red Blood Cells (PRBC).³

Case Report

A 28 year old, 70 kg, ASA 5E male with multiple stab wounds to the abdomen, scapulas, and upper extremities was brought to the emergency room with an endotracheal tube (ETT) in place. The patient was unresponsive despite receiving etomidate and succinylcholine for placement of the ETT. The patient was hypotensive with diminished peripheral pulses. Initial systolic blood pressures were palpable at 60mmHg with improvement to 90mmHG after 2 units PRBC's and fluid resuscitation administered in the Emergency Department (ED). A femoral 6 French catheter was placed, along with 14 gauge intravenous (IV) catheters in bilateral upper extremities. A chest x-ray performed in the ED showed a left pneumothorax. On assessment, a 10 cm laceration was noted on the abdomen with protruding bowel. Due to the exposed

bowel, profound hypotension and penetrating wounds, the patient was taken emergently to the operating room.

Standard monitors were applied and bilateral breath sounds were auscultated, but severely diminished on the left side. Two rapid infusers were connected, one to the femoral catheter and the other to one of the peripheral IV catheters. All available anesthesia professionals were mobilized and the massive transfusion protocol initiated. Oozing was noted from all invasive lines and there was profound bleeding from the peripheral and scapular injuries. Volume resuscitation continued during the surgical procedure with multiple episodes of hypotension due to massive surgical blood loss. As the procedure progressed, the patient required frequent suctioning of copious amounts of frothy red sputum from the ETT, and continued with significant bleeding from the abdomen as well as the other stab wounds.

For fluid resuscitation the patient received a total of 38 units (11360 ml) PRBC's, 4 units platelets, 34 units (8400 ml) of plasma, 500 ml hetastarch, 1500 ml albumin, 8000 ml 0.9% normal saline, and 5,000 ml Plasmalyte. Medications given were atropine 0.8 mg, calcium chloride 3 gm, epinephrine 4900 mcg, phenylephrine 1200 mcg, and sodium bicarbonate 8 amps. Due to excessive blood loss in multiple hospital locations and during transport, it was difficult to calculate the total blood loss, which was conservatively estimated at greater than 5000 ml in the operating room alone. The surgical team was unable to successfully control the massive bleeding while the patient had progressed to pulseless electrical activity. The decision to terminate resuscitation was agreed upon by all practitioners present.

Discussion

Infusion of PRBCs, crystalloid and colloid products are the primary treatment to replace intra-operative fluid loss and massive bleeding from traumatic injury.⁴ The goal to maintain hemodynamic stability is to replace the volume as fast as it is being lost in most situations. Commonly, PRBCs and crystalloid are the first line replacement with other component therapy being utilized based on obtained laboratory values or after 6 to 10 units of PRBCs, depending specifically the institution's protocol. In massive bleeding resuscitation, replacement therapy often cannot wait on laboratory results to guide component replacement. For massive transfusions, defined as more than 10 units of PRBCs in 24 hours after traumatic injury, recent literature has suggested that PRBCs be replaced simultaneously in a 1:1 ratio with plasma and platelets.⁵

Historically, plasma was replaced when the coagulation studies, prothrombin time (PT) or partial thromboplastin time (PTT) values exceeded 1.5 times normal or after 6-10 units of PRBCs were administered. Coagulopathy in acute trauma is directly related to the severity of the injury and contributes to patient mortality.³ In 2008 researchers found there is a small window of opportunity to aggressively replace coagulation factors to prevent a dilutional coagulopathy which may lead to an unmanageable depletion of coagulation factors resulting in more profound hemorrhage.⁶ A complex coagulation disorder develops due to extensive hemodilution and loss of procoagulants and reduced thrombin activation. Fibrinogen is essential to coagulation; however, after extensive hemodilution; fibrinolysis then occurs, breaking down the fibrin clots due to the decrease in antifibrinolytic proteins.⁷

This profound hemorrhage due to coagulopathy can result in a near unstoppable bleeding cascade leading to eventual exsanguination, either in the operating room or the intensive care unit.

The current data from modern battlefield medicine suggests replacing PRBCs in a high ratio of 1:1 with both plasma and platelets.² This recommendation is based on prophylactically maintaining coagulation factors, therefore maintaining coagulation at near normal levels during the resuscitation. By replacing each unit of PRBCs with plasma and platelets, the goal is to maintain normal coagulation times and prevent a further decline in patient status in the hours following resuscitation. Conventional practice of anesthesia professionals has been to limit blood component therapy to minimize the effects of pulmonary complications in the trauma patient. This practice of limiting component therapy to avoid increased pulmonary damage was also studied, and the outcomes were unremarkable in individuals receiving high ratio 1:1 replacement versus lower ratios of 1:6 or greater.³

Managing acute and rapid blood loss is an important responsibility of the anesthesia practitioner. Strategies by which to replace these losses have been changing both on and off the battlefield. It is frequently impossible to guide replacement based on laboratory findings as it has been demonstrated that while fibrinogen drops below a critical threshold of 1 g/l after a loss of about 150% of circulating blood volume, critical values of coagulation factors and platelet count are not observed until after a loss of more than 200% of blood volume.⁸ Therefore the replacement must be guided by protocol and evidence based practice.

Trauma resuscitation data from the battlefields of present wars has translated into delivering higher ratios of plasma and platelets with each unit of PRBCs. This higher ratio promotes normal coagulation, replacement of fluid volume, no appreciable pulmonary risk and less mortality. When massive blood loss requires rapid replacement of blood components, each unit of PRBCs should be replaced on a 1:1 ratio with the other blood components of plasma and platelets to avoid acute traumatic coagulopathy and promote better survivability. There has been no compelling literature on component ratio for routine replacement of blood loss in the operating room.

In this case, the patient expired from uncontrollable traumatic blood loss related to massive stab wounds that could not be surgically controlled. Massive blood transfusion was employed in an attempt to maintain normal hemodynamic status. Copious amounts of blood products and fluids were administered in unequal ratios. Current literature states that these blood components should now be administered in equal ratios to provide better coagulation and long term survival.

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Anesthetic Management of a 17 Month Old with Wilms Tumor

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Keywords: Wilms tumor, pediatric, nephrectomy, anesthetic management, African-American

Wilms tumor is the most prevalent kidney cancer diagnosed in children. Wilms tumors compose 5.9% of cancers occurring in children less than 15 years of age and 0.2% of cancers in children greater than fifteen.¹ The incidence rate is slightly higher in the African American population and less frequent in males than in females.² Hypertension is seen in about 25-50% of cases and is usually attributed to an increase in renin activity.² The following case report describes the anesthetic implications for a patient undergoing a left nephrectomy for a Wilms tumor.

Case Report

A 17 month old 11 kg, 80 cm, ASA physical status III, African American female with a history of hypertension, sickle cell trait, tonsillitis, nausea and vomiting presented with a renal mass for exploratory laparotomy and nephrectomy. The patient had been treated with amoxicillin/clavulanate potassium 488 mg QD for her tonsillitis and amlodipine 2.5 mg QD for hypertension. Preoperative antibiotics included cefuroxime 570 mg and metroenidazole 119.25 mg. Laboratory values were all within normal limits. The patient's preoperative vital signs were; heart rate (HR) of 153 bpm, blood pressure (BP) of 117/74, respiratory rate (RR) of 28 breaths/min, oral temperature of 36.0 C, and 100% SpO₂ breathing room air. A left subclavian single lumen central line was in

place and intravenous induction commenced with fentanyl 20 mcg, propofol 50 mg, and rocuronium 20 mg. The trachea was intubated with a 4.5 millimeter internal diameter uncuffed endotracheal tube using a Macintosh 1 blade and the tube was placed 15 cm at the lip. Anesthesia was maintained with 2.7% sevoflurane in O₂ at 2 liters per minute flow. Pressure controlled ventilator settings included peak inspiratory pressures of 20 cm of H₂O and RR of 29. A right radial arterial catheter was placed for continuous blood pressure monitoring and a nasogastric tube was positioned in the left nare. A thoracic epidural catheter was placed with bupivacaine 0.125% initiated at 3ml/hr for the duration of the anesthetic. No lower extremity movement was noticed after the epidural was placed and after emergence. Fentanyl was administered IV intermittently throughout the procedure for pain control, with a total dose of 20 mcgs.

Ondansetron 1.65 mg was administered before emergence. When spontaneous ventilation was achieved, the oropharynx was thoroughly suctioned and the trachea was extubated after the patient was able to open her eyes. 100% O₂ via face mask was placed and the patient was transported to the post anesthesia care unit (PACU) with the epidural infusing. Total anesthesia time was approximately 4 ½ hours. Blood loss throughout the case was estimated to be 200 ml and total intravenous fluids were 650 ml. Total dose of epidural bupivacaine was 16.8 mg with the infusion continuing throughout emergence and decreased to 2 ml/hr during transport to PACU. The patient was hemodynamically stable during emergence and the post-operative recovery period was uneventful.

Discussion

Multiple considerations must be examined when conducting the anesthetic management of patients with Wilms tumors. Comorbidities, tumor size affecting tidal volumes, hypertension associated with renin release, blood loss related with the surgical procedure and associated coagulopathies must be taken into account. Beckwith-Wiedemann syndrome (BWS) should be taken into consideration when conducting an airway assessment. This congenital disorder has been linked with Wilms tumor and it is associated with macrosomia, omphalocele, macroglossia, and visceromegaly. The anesthesiologist may have to consider using airway equipment sized differently from that usually chosen based on the patient's age and weight in a patient with Wilms tumor who exhibits BWS.³ Nitrous oxide should be avoided because bowel distention may be a risk, along with an increased chance of nausea and vomiting.⁴

Intraoperative hemodynamic instability may be attributable to surgical manipulation of the IVC impeding venous return.³ Close observation of blood pressure and communication with surgical staff is essential to avert periods of hypotension. Wilms tumor is often associated with increased plasma concentrations of renin, but not all patients are hypertensive. Renin stimulates a chain reaction that produces angiotensin II, which is a potent arteriolar vasoconstrictor.³ It is this process by which hypertension exists in Wilms tumor patients. Beta-adrenergic blockade should be used with caution in patients with uncontrolled hypertension because it may exacerbate the hypertensive effects by inhibiting the beta-adrenergic vasodilatory mechanism.³ Angiotensin converting enzyme (ACE) inhibitors along with phenoxybenzamine, phentolamine, and sodium nitroprusside

have been used for intraoperative control of blood pressure. The incidences of perioperative hypertensive or hypotensive episodes related to fluctuating renin concentrations are rare with adequate preoperative control of blood pressure.³

The patient did not require any vasopressors or vasodilators during the case. Amlodipine was the only medication used for blood pressure control preoperatively. Possible reasons for stable blood pressure during the case include adequate pain control and vasodilatation from the sympathetic block of the epidural, and the effects of the amlodipine that was administered the previous night. It is common practice to use continuous infusions of local anesthetics in epidurals intraoperatively and for post operative pain control.⁵ Continuous infusions permit the block to be maintained at a constant level. The recommended dosing infusion rates should be a maximum of 0.4 mg/kg/hr of bupivacaine following the initial establishment of the block, with this dose reduced by 30% for infants less than 6 months of age.⁵ For this particular patient, the maximum calculated dose would be 4.4mg/hr (3.52ml/hr). The patient's actual rate was 3ml/hr, less than the maximum recommended dose. The level of block for this patient was unknown due to the young age. Sufficient pain control and vasodilatation intraoperatively could very well be the explanation of why no hypertensive episodes or dramatic blood loss were experienced throughout the procedure.

The patient had a history of sickle cell trait (SCT) so a complete blood count (CBC) was carefully evaluated for any signs of hemolytic anemia. Factors that favor a sickle cell crisis include hypoxemia, acidosis, intracellular hypertonicity or dehydration, increased 2,3-DPG levels, or increased or decreased temperatures.⁶ SCT is usually a

benign condition but can be considered a risk factor for venous thromboembolism.¹ Sickle cell trait carriers can be twice as likely to have a thromboembolism than non sickle cell trait carriers and Sickle cell disease (SCD) is much more of a concern than SCT.⁷ The CBC was within normal limits for this SCT patient who showed a pre operative hemoglobin of 12.6 g/dl, hematocrit of 39.6%, and a mean corpuscular volume of 90 fl. The patient did not exhibit any evidence of hematuria or sickle cell crisis, and the insufflating gas and intravenous fluids were prophylactically warmed and humidified.

Our patient's tumor was 15 cm in length and 12 cm in width. With such a large tumor, problems with ventilation including high peak pressures and reductions in tidal volume could be anticipated. During this procedure, peak airway pressures were never higher than 20cm/H₂O and there were no problems with maintaining tidal volumes. It is also important to check platelet function and bleeding time preoperatively since less than 10% of patients with Wilms tumors have been known to have Von Willebrand's disease and 30% of patients present with hematuria.³ Intraoperative blood loss can be a significant factor because of the tumor's location and possible involvement of the renal vein and vena cava.⁴ Bleeding time and platelets were unremarkable in this patient.

A Wilms tumor operation can be an extensive and multifaceted procedure. It requires that the anesthetist consider all possible complications and co-morbidities that may arise during the surgery.

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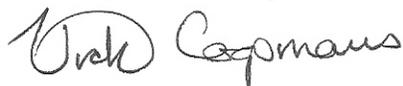
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- Mentor:** Christine Langer, CRNA, MS, MSED, MSN

Editorial

This spring issue offers a variety of case reports from over half a dozen Nurse Anesthesia Programs. We continue to add to our list of new reviewers:

Matthew Bishop, CRNA, MSN; Tripler Army Medical Center
 Kevin Buss, CRNA, MS; Uniformed Services University
 Stephanie Fan, CRNA, MSN; Washington University School of Medicine
 Claire Farren, CRNA, MS; Washington University School of Medicine
 Marjorie A. Geisz-Everson, CRNA, PhD; Louisiana State University Health Sciences Center
 Marco Gidaro, CRNA, MSN; Einstein Medical Center
 Michael Neill, CRNA, MSN; Tripler Army Medical Center
 Kelly Wiltse Nicely, CRNA, PhD; University of Pennsylvania
 Jason Penfold, CRNA, MSN; Naval Medical Center, San Diego
 Matt Seymour, CRNA, MS; Naval Medical Center, San Diego
 Lori Stone, CRNA, MSN; Decatur Memorial Hospital /Bradley University

This does not mean we don't have opportunities for those of you who still want to participate, so please don't hesitate to contact me if you are interested in getting involved in the student journal. Also stay tuned for updates to the Guide for Authors later this year!



Vicki C. Coopmans, CRNA, PhD
 Editor