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Subdural Catheter

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Perioperative MI

Gum Bougie

Eclampsia



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Front Cover:

Sarah Ranahan RN, BSN, a graduate student in the Northeastern University Nurse Anesthesia Program, places a subarachnoid block on a patient with significant kyphoscoliosis during a mission trip at Kibagabaga Hospital in Kigali, Rwanda.

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The Gum Elastic Bougie: An Airway Alternative

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Keywords: difficult intubation, gum elastic bougie, bougie, anesthesia, limited mouth opening

A difficult airway is defined as a situation in which the “conventionally trained anesthesiologist experiences difficulty with mask ventilation or intubation”.² Although direct laryngoscopy is the accepted practice for endotracheal intubation, there are several alternative techniques available to the anesthesia practitioner.² The gum elastic bougie or Eschman Introducer is a technique to consider. This device is simple to use, very portable and less costly than many alternative devices currently on the market. Direct visualization of the vocal cords is not necessary and in most instances this technique can be performed safely by one practitioner in a timely manner.

Case Study

A 68 year old, 74 inch, 109 kilogram Caucasian male, presented for elective laparoscopic cholecystectomy. His history included controlled hypertension, hypercholesteremia and well-controlled gastroesophageal reflux. There was indication of sleep apnea as per the patient’s large neck circumference and self-report of snoring loud enough to wake himself on occasion, but no confirmed diagnosis. Medications consisted of metoprolol, simvastatin and pantoprazole daily. Although the patient quit smoking 18 years ago, he had smoked for 35 years. The patient drank four alcohol beverages daily for the past 45 years. Previous surgeries consisted of right knee arthroscopy and appendectomy without anesthesia complications. Physical

exam revealed a Mallampati Class IV airway, thyromental distance of 7 cm, a 3cm mouth opening and normal neck extension. It was noted that the patient had prominent, large facial bones and was edentulous. General endotracheal anesthesia was planned for the patient. The patient was premedicated with midazolam 1mg intravenously and taken to the operating room. He was transferred to the operating table and standard monitors were applied. The patient was pre-oxygenated for approximately 4 minutes just prior to induction with intravenous fentanyl 50mcg, lidocaine 80mg and propofol 200mg. The patient was very difficult to mask ventilate; effective mask ventilation required two anesthesia practitioners. The patient was given rocuronium 40mg. Direct laryngoscopy was attempted with a Macintosh 4 blade, revealing a grade four laryngeal view (Cormack and Lehane classification). An intubation without direct view of the vocal cords was unsuccessful. The second anesthesia practitioner attempted direct laryngoscopy with the same equipment and also with a Miller 3 blade and was also unsuccessful.

The patient was oxygenated via facemask while the Eschman Introducer/gum elastic bougie was prepared. Intubation was attempted with the bougie and again was unsuccessful. However, after a second attempt with the bougie, the bougie was placed in the trachea, and the patient was successfully intubated with an 8.0 internal diameter endotracheal tube. Intubation was confirmed by the washboard/clicking sensation of the tracheal rings on the tip of the bougie, bilateral breath sounds with

positive pressure ventilation and a positive end-tidal carbon dioxide tracing.

The surgical procedure was uneventful. At the conclusion of the procedure, the patient's neuromuscular blockade was antagonized with intravenous neostigmine 5mg and glycopyrrolate 0.8mg. After return of spontaneous ventilation at a rate of 14 breaths per minute and a tidal volume of 600ml, the patient was able to follow directions and maintain a head lift of five seconds as well as bilateral hand grasps of moderate strength. The patient was successfully extubated in the operating room and taken to the recovery area with 3 liter of oxygen via nasal cannula and a 99% oxygen saturation. The patient was discharged home later the same day without complications.

Discussion

In 1993 the American Society of Anesthesiologists (ASA) developed a task force on the management of the difficult airway and established an algorithm that has become a staple of airway management. Revised in 2003, the algorithm walks the practitioner through the appropriate steps to manage the difficult airway. The algorithm is brief and simple. Although it encourages the use of alternative methods of intubation, none are specifically mentioned with the exception of the laryngeal mask airway (LMA). After this step, the algorithm simply states that emergency pathways are to be established.

It is recommended that after three unsuccessful attempts at intubation the anesthesia practitioner should move to the Difficult Airway Algorithm. The bougie is not specifically mentioned in the algorithm; however it is implied by the phrasing of alternative methods.¹ Another airway algorithm: The SLAM Emergency Airway Flowchart (SEAF) does mention the bougie

as an alternative airway technique along with several others and extends itself to use outside the hospital setting.⁴

The Eschman Introducer, also known as the gum elastic bougie is a solid or hollow, semi-malleable stylet that can be blindly inserted into the trachea. The Eschman (Eschman Healthcare, Kent, England), introduced in 1949, was 60cm long, 15Fr gauge and angled at 40 degrees at the distal end for blind tracheal placement. The current version is available in different sizes as well as disposable and reusable versions. The introducer can be placed under the epiglottis and directed anteriorly towards the trachea. The practitioner may feel a distinctive "washboard clicking" as the introducer enters the trachea. If the clicking is not encountered then the introducer may be in the esophagus and a second attempt might be necessary. Once the introducer is in the trachea, the endotracheal tube is threaded over it and pushed down past the vocal cords.³ The introducer is then removed and confirmation of placement of the endotracheal tube is obtained.

Having the best possible view of the larynx is very important for bougie insertion, however as previously stated it is not a necessary component. Proper head positioning will optimize the practitioner's view. Cricoid pressure applied by an assistant is also helpful, but may also make the view worse or passing of the introducer impossible. The gum elastic bougie is an inexpensive, portable and easy to use alternate airway technique that can assist in the tracheal intubation of the patient with a difficult airway in a timely manner. This is a device that can easily be used inside and outside the hospital setting. The bougie should be considered in difficult airway management, especially in areas that do not offer fiber optic equipment or where the

anesthesia practitioner has not been trained in the use of advanced airway equipment. There are several conditions that are associated with a difficult intubation scenario and should lead the practitioner to consider alternative methods. These consist of tumors, congenital abnormalities, infections, foreign bodies, trauma, obesity, inadequate neck extension and anatomic variations.¹ The airway assessment of this patient indicated that he may be a difficult intubation and it was imperative that the anesthesia practitioner prepare for alternative intubation techniques. The bougie was chosen based on ease of use and availability as well as cost. The bougie in this facility was disposable, however reusable ones are also available and can aid with cost containment. This facility offers multiple alternatives such as the McGrath (Aircraft Medical Ltd, Edinburgh, Germany), Claris (Claris Medical Systems, Minneapolis, MN) and other fiber-optic options. The McGrath was open and ready for this particular case if there was failure to intubate with the gum elastic bougie. While the bougie was selected for this learning opportunity for difficult airway management, alternative options were discussed prior to and during the case.

The anesthesia practitioner must also keep the difficulty of the intubation in mind prior to removal of the endotracheal tube. Patients who are difficult to intubate should be awake and meet several criteria for extubation such as adequate tidal volume, antagonism of neuro muscular blockade, ability to raise head off the pillow for five seconds, and equal hand grip strength prior to removing the endotracheal tube, as it may be more difficult to reintubate due to previous airway trauma and swelling from prior attempts. In this particular case, the patient met all of the above mentioned criteria prior to extubation.

This case highlights the importance of practitioner competence in the use of alternative endotracheal intubation techniques. Practicing alternative methods for difficult airways on patients that are not considered difficult airways is very important for anesthesia practitioners. The practitioner does not want the first time they have a difficult airway scenario to be the first time they have used an alternative airway technique. This aides the practitioner with experience to develop skills that will quickly and safely secure a patent airway with little or no harm to the patient. A gum elastic bougie offers this opportunity and should be considered.

References

1. Morgan GE, Mikhail M, Murray M, eds. (2006) *Clinical Anesthesiology*. 4th ed. McGraw-Hill Companies; 108, 112.
2. Barash P, Cullen B, Stoelting R, eds. (2006) *Clinical Anesthesia*. 5th ed. Philadelphia: Lippincott, Williams and Wilkins; 618, 619, 637.
3. Latta IP, Stacey M, Mecklenburgh J, Vaughan RS. (2002). Survey of the use of the gum elastic bougie in clinical practice. *Anaesthesia* 57(4): 379-384.
4. Rich JM, Mason AM, Ramsay MAE. (2004). The SLAM emergency airway flowchart: a new guide for advanced airway practitioners. *AANA Journal*. 72(6): 431-439.
5. Baba RA, Spray D, Al-Sahaf MH. (2006). Management of a difficult airway assisted by the use of a combination of fiber optic bronchoscope and gum elastic bougie. *Internet Journal of Anesthesiology*. 10(2): 9(17ref).

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Recognition and Treatment of Perioperative Myocardial Ischemia

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Keywords: myocardial ischemia, coronary artery disease, anesthesia, perioperative care, surgical procedures, complications

Each year approximately 30 million people have surgery in the United States.¹ Out of these 30 million people, one third has coronary artery disease (CAD), or is at risk for developing CAD.¹ Adverse cardiovascular events, including perioperative myocardial ischemia, complicates more than one million of these operations¹ and can lead to morbidity and mortality.² Considering the threat of these extreme negative outcomes, the anesthesia professional should be well informed of the patient's history, have a knowledge of myocardial ischemia signs and symptoms, and be ready to act accordingly.

Case Report

An 81-year-old, 152 centimeter, 50 kg female was admitted for an eye infection and non-healing corneal ulcer. While in the hospital, the patient fell and sustained a right hip fracture. She presented to the operating room for a right hip hemiarthroplasty. Her medical history included hypertension, coronary artery disease status post stent placement in 2004, transient ischemic attack, dementia, and anemia. The patient had no known drug allergies. Her medications included escitalopram, ezetimibe, metoprolol, pramipexole, memantine, hydrochlorothiazide, clopidogrel, erythromycin ophthalmic, and valacyclovir. Recent lab work included hemoglobin of 12 g/dl and hematocrit of 32%. Current electrocardiography (ECG) showed sinus rhythm at 78 beats per minute.

Upon assessment, the patient's heart was regular in rate and rhythm with a blood pressure of 105/52, and her lungs were clear and equal to auscultation.

The patient was brought to the operative suite where standard AANA monitors, a 20 gauge intravenous catheter (IV), and a 20 gauge arterial line were placed. The patient was pre-oxygenated and a smooth IV induction was performed with fentanyl 50 mcg, etomidate 40 mg, and rocuronium 40 mg. The airway was secured through atraumatic direct laryngoscopy and a 6.5 endotracheal tube. Bilateral breath sounds were equal to auscultation and end-tidal carbon dioxide (ET CO₂) was present. General anesthesia was maintained with isoflurane, fentanyl, vecuronium, and a combination of oxygen and air for a FiO₂ of .60. The patient's blood pressure was labile for the beginning of the procedure, but stabilized after hydralazine and nitroglycerin were given. However, ST depression was noted in leads II and V5 as the surgery progressed. The patient's vital signs were stable at that time with a heart rate in the 80's and a systolic blood pressure ranging from the 140's to 150's. The FiO₂ was increased to 1.0 and increments of 20 mg of esmolol were given until the patient's systolic blood pressure returned to her baseline in the 120's. The anesthesiologist and surgeon were notified of the ST depression. A complete blood count, cardiac enzymes, and an arterial blood gas were sent. Lab results were within normal limits with the exceptions of hemoglobin of 10.5g/dl and hematocrit of 31%. The case was completed without further complications and resolving ST depression.

The patient was given a total of esmolol 280 mg, fentanyl 200 mcg, hydralazine 20 mg, nitroglycerin 180 mg, ondansetron 4 mg intravenously. Neuromuscular blockade was antagonized with neostigmine 4 mg IV and glycopyrrolate 0.6 mg IV. The patient was extubated and remained stable during transport to PACU. A cardiology consult, Intensive Care Unit (ICU) bed, ECG, and additional lab work were ordered. The patient remained in the ICU for 3 days following the operation with visible ST depression, but normal cardiac enzymes.

Discussion

Stress from surgery can cause substantial myocardial ischemia on patients and may arise without warning.^{2,3} Anesthesia professionals can decrease the risk of intraoperative complications through preparation and vigilance for potential myocardial ischemia. Pre-operative patient assessment, awareness of myocardial ischemia signs and symptoms, and prompt treatment are all important factors for the anesthesia professional to reduce potential for patient morbidity and mortality.

Pre-operative patient assessments should always include a risk assessment. Risk assessment evaluates co-morbidities, exercise tolerance, and the proposed surgery, to determine the risk of perioperative complications, such as myocardial ischemia.¹ A history of angina, advanced age, diabetes mellitus, or prior cardiac problems such as coronary artery disease, hypertension, a history of myocardial infarction, arrhythmias, or congestive heart failure are all high risk factors for perioperative complications.¹ A patient's current medical status and ability to tolerate exercise, as defined by ability to climb 1-2 flights of stairs without dyspnea, or a METS of 4 or greater, are also

important in determining a patient's risk assessment.¹ Finally, the type of surgery can suggest a patient's risk for perioperative complications. Abdominal, thoracic, major orthopedic, vascular, and emergency procedures are all considered high risk surgeries.^{1,2} The anesthesia professional should attempt to optimize cardiac function.¹

Risk assessment is important in patient care, but early recognition and treatment of perioperative myocardial ischemia is the most important way to improve patient outcomes. Knowledge of a patient's health history, current cardiovascular status, and baseline vital signs is the first step in recognizing variations from a patient's normal status. Ischemia of myocardial cells occurs when the oxygen supply does not meet the oxygen demand.⁴ This mismatch between oxygen supply and demand can occur for many reasons including, but not limited to, decreased coronary blood flow, tachycardia, hypotension, hypertension, or anemia.⁴ Careful ECG monitoring during the perioperative period can help with early ischemia detection and prevention of ischemia from worsening to injury or infarct.⁴ Other monitors such as oximetry and blood pressure may also provide information to recognize conditions which lead to imbalances of myocardial oxygenation. The common symptoms associated with the development of myocardial ischemia can be masked by the administration of anesthesia.⁴ Classical signs and symptoms of ischemia include a sudden onset of shortness of breath, fatigue, chest pain, neck or jaw pain, arm pain, nausea or vomiting, or diaphoresis.⁴ Symptoms that may be seen perioperatively include hemodynamic instability (hypertension or hypotension), a decreased O₂ saturation, and ST segment changes on the ECG.⁴ The diagnosis of ischemia is often from ST segment depression or elevation of at least 1

mm on two or more leads, best observed in leads II and V₅.⁴ The severity of the depression or elevation is often determined by the severity of the ischemia.⁴ Other ECG changes such as T-wave inversion or R-wave changes may also be indicative of ischemia.⁴

Once myocardial ischemia is noted, prompt treatment is important to prevent further damage to the myocardium. Increasing the patient's FiO₂ helps reduce the oxygen supply and demand mismatch. Concerns have existed as to the risk of isoflurane and desflurane worsening ischemia due to coronary steal.^{5,6} However, Ebert et al states sevoflurane and isoflurane are comparable in the risk for cardiac complications, and Kossick states that sevoflurane, isoflurane, or desflurane may be used to maintain anesthesia.^{6,7} Coronary steal occurs when the coronary arteries are maximally dilated and blood flow is redirected from the ischemic areas of the heart to the nonischemic areas, thus worsening the ischemia.⁶ Hypotension or vasodilators such as nitroglycerin or sodium nitroprusside can worsen the coronary steal effects.⁶ This steal phenomenon is reversible if normotension is achieved.⁶ Beta blockade is recommended for risk reduction during ischemia. Beta blockade is effective in reducing ischemia by decreasing myocardial oxygen demand and inotropy by reducing the heart rate, the force of contraction, and the myocardial maximal oxygen consumption.⁵ Esmolol can be useful for its short half life while metoprolol will have longer acting effects.⁵ Maddox found that clonidine, an alpha-2 receptor agonist, can lower the incidence of perioperative ischemia and that nitrates have varying reductions on ischemia.¹ As hypothermia during the intraoperative period and post operative ischemia are related, it is important to maintain a patient's temperature.⁸ Backlund et al states that the

type of anesthesia, general versus regional, has no effect on myocardial ischemia.⁸ However, a study by Scheinin et al on elderly patients with hip fractures showed that starting a continuous epidural bupivacaine/fentanyl analgesic regimen preoperatively decreases the amount of myocardial ischemia.⁹

For the present case, a thorough health history was obtained prior to surgery. The information on the patient's cardiac history indicated a risk for myocardial ischemia. Increasing the patient's FiO₂ from .60 to 1.0 improved her myocardial oxygen supply. Continuing isoflurane as the volatile agent for anesthesia is disputed by current research. Ebert et al supports that isoflurane and sevoflurane are equal volatile agents for cardiac complications while Botero et al support sevoflurane use.^{5,7} Giving esmolol to reduce the patient's myocardial ischemia by reducing the heart rate, force of contraction, and oxygen consumption is also in agreement with current literature.⁵

Intraoperative myocardial ischemia can increase morbidity and mortality if not recognized and treated promptly. Anesthesia professionals must remain vigilant with our care and up-to-date with our current practice guidelines. We have the potential to improve our patient outcomes and reduce the risks associated with intraoperative myocardial ischemia.

References

1. Maddox TM. Preoperative cardiovascular evaluation for noncardiac surgery. *Mt Sinai J Med* 2005;72:185-191.
2. Leppo JA. Preoperative cardiac risk assessment for noncardiac surgery. *AM J Cardiol* 1995;75:42D-51D.

3. Marsch SCU, Schaefer HG, Skarvan K, Castelli I, Scheidegger D. Perioperative myocardial ischemia in patients undergoing elective hip arthroplasty during lumbar regional anesthesia. *Anesthesiology* 1992;76:518-527.
 4. Akhtar S. Ischemic heart disease. In Hines RL, Marschall KE, eds. *Stoelting's Anesthesia and Co-Existing Disease*. 5th ed. Philadelphia: Churchill Livingstone; 2008:16-22.
 5. Botero C, Smith CE, Morscher AH. Anemia and perioperative myocardial ischemia in a Jehovah's Witness patient. *J Clin Anesth* 1996;8:386-391.
 6. Kossick MA. Inhalation anesthetics. In Nagelhout JJ, Zaglaniczny KL, eds. *Nurse Anesthesia*. 3rd ed. St. Louis: Elsevier; 2005:81-101.
 7. Ebert TJ, Kharasch ED, Rooke GA, Shroff A, Muzi M, and the Sevoflurane Ischemia Study Group. Myocardial ischemia and adverse cardiac outcomes in cardiac patients undergoing noncardiac surgery with sevoflurane and isoflurane. *Anesth Analg* 1997;85:993-999.
 8. Backlund M, Lepantalo M, Toivonen L et al. Factors associated with post-operative myocardial ischaemia in elderly patients undergoing major non-cardiac surgery. *Eur J Anaesthesiol* 1999;16:826-833.
 9. Scheinin H, Virtanen T, Kentala E et al. Epidural infusion of bupivacaine and fentanyl reduces perioperative myocardial ischaemia in elderly patients with hip fracture – a randomized controlled trial. *Acta Anaesthesiol Scand* 2000;44:1061-1070.
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Hypertension after Clonidine Withdrawal

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Keywords: clonidine, hypertension, withdrawal, alpha-2 agonist, catecholamine

Clonidine is a centrally acting selective alpha 2 adrenergic agonist. Clonidine can reduce anesthetic requirements, improve perioperative hemodynamic stability, provide sedation, and modulate pain pathways resulting in analgesia. It has the ability to attenuate sympathetic nervous system output by a negative feedback mechanism at the alpha 2 receptor site. This drug has proven to be particularly effective in treating severe hypertension or renin-dependent hypertensive disease. Abrupt discontinuation of clonidine therapy can result in a phenomenon labeled rebound hypertension. This increase in systemic

blood pressure may be associated with an increase in circulating catecholamines and intense vasoconstriction.¹

Case Report

A 29-year-old African American female, weighing 82kg and 65 inches tall, presented for an elective total abdominal hysterectomy. This patient's medical history consisted of hypertension and smoking. Her home medication regimen included clonidine 0.1mg by mouth every day, which she had not taken for three days. Her preoperative baseline vital signs were: blood pressure (BP) 148/97mmHg, heart rate (HR) 78 beats per minute (bpm), and oxygen saturation (SpO2) 98% on room air. Her

preoperative laboratory values and echocardiogram were normal. The anesthetic plan was general anesthesia with endotracheal tube (ETT) placement.

The patient was medicated preoperatively with intravenous (IV) midazolam 2 mg and fentanyl 150 mcg upon entering into the operating room. She was positioned supine and standard ASA monitors were placed. Her blood pressure cuff was positioned on her right upper extremity for the entire case. The patient was preoxygenated and anesthesia induced. Preinduction vital signs were: BP 135/76 mmHg, HR 83 bpm, SpO₂ 100%.

General anesthesia was induced with 2% lidocaine 60 mg and propofol 200 mg IV. The patient was mask ventilated and rocuronium 50 mg IV was given. Mask ventilation was continued with 100% oxygen and 4% sevoflurane. After adequate neuromuscular blockade was verified, the patient's trachea was intubated with a 7.0-mm cuffed endotracheal tube without difficulty. After confirmation of end-tidal carbon dioxide (ETCO₂) and bilateral breath sounds, the ETT was secured and anesthesia was maintained with sevoflurane.

Approximately 90 minutes into the case, the patient's blood pressure increased to 160/110 mmHg, with HR 82, and SpO₂ 99%. Adequate depth of anesthesia was investigated. The patient lacked any spontaneous respiratory effort and her pupils were 2.0 mm (reactive) bilaterally. The end-tidal sevoflurane measurement read 3%. Neuromuscular stimulus via train of four revealed one out of four twitches. Other sources of hypertension were ruled out including hypercarbia, hypoxia, hyperthermia, and distended bladder. Analgesia was a concern and fentanyl 100

mcg IV and hydromorphone 1 mg IV were administered.

No changes were noted in subsequent blood pressure measurements. No ischemic changes on electrocardiography were noted. Incremental doses of labetalol 5mg intravenous were administered for a total dose of 20mg. After the labetalol was given, the patient's blood pressure transiently decreased to 140/80 mmHg and then increased to 160/115 mmHg. Hydralazine 20mg IV was titrated in with a reduction in BP to 150/80 mmHg. After 30 minutes, the patient's blood pressure increased to 160/100 mmHg. Nitroglycerin 40mcg intravenous was given which resulted in her blood pressure decreasing to 140/76 mmHg.

At the end of surgery, sevoflurane was discontinued, oxygen was delivered at 10L/min, and the neuromuscular blockade was fully reversed with neostigmine 4mg and glycopyrrolate 0.8mg IV. On emergence, a nitroglycerin IV infusion at 50 mcg/kg/min was initiated to maintain her BP between 130/80 mmHg and 140/78 mmHg.. The patient was admitted for 24 hour observation and clonidine was reinstated upon her admission to the nursing unit. The following day, the patient was discharged from the hospital without any other complications.

Discussion

Hypertension can be a secondary complication of other medical conditions such as renal disease, pregnancy, or endocrine abnormalities. More commonly, it is idiopathic (essential) which accounts for 80-95% of patients suffering from hypertension.² Treatment options for hypertension vary from single-drug therapy to a multiple-drug regimen targeting several receptor sites. Most patients with mild

essential hypertension require only a single-drug plan which may consist of a thiazide diuretic, ACE inhibitor, angiotensin-receptor blocker, beta-adrenergic blocker, or calcium channel blocker.²

Although the underlying cause of essential hypertension is not known, the central nervous system (CNS) is suspected to play a key role.³ Hypertension that is resistant to numerous antihypertensive agents may be controlled with a centrally acting alpha-2 agonist, which supports the theory of CNS involvement in essential hypertension. Clonidine acts on centrally located alpha-2 receptor sites by inhibiting release of the neurotransmitter norepinephrine. When this receptor site is activated, sympathetic stimulation is reduced on peripheral arterioles resulting in a decreased blood pressure.^{1,3} This reduction in circulating sympathetic catecholamines produces a profound modulation on blood pressure. Clonidine is not considered a first line drug for hypertension treatment. This drug is usually incorporated in patients' treatment plan when other anti-hypertensive medications have failed to produce results.

Studies do not have a definitive answer on whether the sudden increase in blood pressure after abrupt cessation of clonidine is a true rebound response or just a return to the patient's pretreatment blood pressure level.⁴ Although there does not seem to be a direct correlation between the incidence of rebound hypertension and the patient's dosage, the marked elevation in blood pressure has been reported to occur within 24 to 36 hours of cessation of clonidine.⁴ On the other hand, reports have also produced evidence of patients that did not experience any rebound hypertension upon clonidine withdrawal.⁴ Either way, the treatment plan is the same, and preparations should be made when a patient presents with

a history of recent termination or noncompliance with clonidine or other anti-hypertensive therapy.³

In summary, this case report presents a noncompliant hypertensive patient who failed to take her prescribed clonidine for three days prior to scheduled elective surgery. It demonstrates clonidine withdrawal displayed as a severe hypertensive crisis from adrenergic discharge. If the hypertensive patient has good myocardial reserve function, this may not be significant. For the severely hypertensive patient with poor myocardial reserve, a dangerous increase in afterload may result.⁵ The anesthesia practitioner must have a plan or algorithm for treatment of a severe intraoperative hypertensive crisis. If a patient is admitted for preoperative evaluation and recent history demonstrates noncompliance with anti-hypertensive medications, especially clonidine, the anesthetist should prepare for aggressive perioperative control of a sympathetic mediated discharge of norepinephrine. If surgery is elective, cancelling the surgery should be considered.

Prescribing and administering an alpha-2 agonist, such as clonidine or dexmedetomidine, in the preoperative environment for a noncompliant hypertensive patient can reduce sympathetic discharge before it occurs, resulting in a more controllable blood pressure. In addition, the administration of this drug in the preoperative period can provide an additional anesthetic benefit by providing an adjunct for anxiolysis and pain control. The main goal in the management of these patients is to prevent perioperative myocardial ischemia. Hypertension treatment can involve various different medications with different sites of action, onset, and duration. A thorough

understanding of the pharmacodynamics of each anti-hypertensive agent and the etiology of hypertensive crisis is essential before implementing a multimodal treatment plan.²

References

1. Stoelting R, Hillier S. *Pharmacology and Physiology in Anesthetic Practice*. Philadelphia, PA: Lippincott Williams & Wilkins, 2006:340-344.
2. Morgan G, Mikhail M, Murry M. Anesthesia for patients with cardiovascular disease. *Clinical*

Anesthesiology. 4th ed. New York, NY: Lange Medical Books/McGraw-Hill, 2006:444-452 .

3. Lowenstein J. Clonidine. *Ann Intern Med*. 1980;92:74-77.
4. Husserl F, deCarvalho J, Batson H, Frolich E. Hypertension after clonidine withdrawal. *South Med J*. 1978;71:496-7.
5. Bruce D, Croley T, Lee J. Preoperative Clonidine Withdrawal Syndrome. *Anesthesiology*. 1979;51:90-92.

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Transthoracic Approach to Epidural Abscess Decompression

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Keywords: epidural abscess, transthoracic approach, somatosensory evoked potentials, motor evoked potentials, diabetic neuropathy.

Epidural abscesses are a rare but serious condition. The abscess may occur related to regional anesthesia techniques or occur spontaneously from vertebral osteomyelitis.¹ Surgical decompression is often necessary to treat this condition, especially if it leads to spinal cord compression and neurological symptoms. A transthoracic approach is often necessary for anterior decompression of these abscesses with special neurological monitoring involving motor evoked potentials (MEPs) and somatosensory evoked potentials (SSEPs). This complex spinal procedure may be further complicated when the patient is morbidly obese or has multiple co-morbidities.

Case Report

A 51 year-old male weighing 146 kg with a history of insulin dependent diabetes mellitus, hypertension, hyperlipidemia, and osteoarthritis presented with methycillin resistant staphylococcus aureus bacteremia, osteomyelitis, and an epidural abscess at T9-T10 with spinal cord compression. The patient's history included recent changes in mental status, but he was awake and alert on presentation. He also demonstrated profound bilateral lower extremity weakness, and pitting edema in both feet. The 12-lead electrocardiogram demonstrated sinus tachycardia. His magnetic resonance image showed anterior paraspinal abscess with spinal cord impingement at T9, and a posterior paraspinal abscess at L3. Other findings of the physical exam included a peripherally inserted central catheter, and a persistent heart rate of 103 beats per minute, though treated with metoprolol. His blood pressure was 197/108 and the oxygen

saturation was measured at 93% on room air. Laboratory results were within normal limits except for hemoglobin of 7.4 g/dL, hematocrit of 22.6%, and a blood urea nitrogen of 5 mg/dL. The patient's medications consisted of oxycodone-acetaminophen, alprazolam, sitagliptin, and carisoprodol.

In the operating room, standard monitors were applied. Following pre-oxygenation, anesthesia was induced intravenously with Propofol 200 mg, Fentanyl 150 mcg and rocuronium 50 mg and the trachea was intubated with a right-sided 39 french double lumen tube using a Macintosh 4 laryngoscope. Placement of the tube was confirmed through the presence of end-tidal carbon dioxide and by auscultation. Volume control mechanical ventilation was initiated. An arterial catheter and central venous line were inserted. The patient was then positioned right lateral decubitus on the operating room table. Endotracheal tube placement was reconfirmed by visualization with a fiberoptic bronchoscope. Isoflurane was titrated from 0.6% to 0.9% and neuromuscular blockade was avoided to facilitate SSEP and MEP monitoring. Sufentanil and phenylephrine infusions were titrated throughout the case. SSEP and MEP monitoring were initiated; however, it was decided to discontinue those monitors due to difficulty obtaining a signal. After discontinuing the SSEP and MEP monitors, akinesia was maintained with vecuronium. Serous fluid was noted in the endotracheal tube (ETT) which persisted throughout the case even after multiple suctioning passes and gravity drainage of the tube. Due to high baseline peak airway pressures, the surgeons elected to avoid one lung ventilation, instead retracting the lung for surgical exposure. The patient maintained a high level of oxygen saturation and peak inspiratory pressures remained at baseline. The patient

required two units of blood due to decreased hematocrit and labile blood pressures. Arterial blood gases remained within normal limits. Postoperatively, the patient continued to be mechanically ventilated and sedated in the intensive care unit, where he was hemodynamically stable overnight. The patient returned to the operating room the next morning as scheduled for a posterior approach spinal fusion to stabilize his spine following the abscess repair.

Discussion

During spinal surgery, the surgeon may desire to attempt to monitor the status of the motor and sensory tracts of the spinal cord. Damage to these tracts can occur from low perfusion pressures, inappropriate positioning, or direct surgical trauma. Myogenic motor evoked potential (MEP) allows intraoperative assessment of the corticospinal tract integrity of descending motor pathways.² Somatosensory evoked potentials monitor the dorsal columns and are typically produced by stimulating peripheral nerves and monitoring for reception of the signal in the cerebrum.³ As practiced in this case, it is important to avoid neuromuscular blockade when employing MEP monitors. After administering rocuronium 50 mg intravenously for tracheal intubation, neuromuscular blockers were not administered again until the SSEP and MEP monitoring had been discontinued. Intravenous techniques are advantageous when monitoring SSEP because inhaled anesthetics cause a dose-dependent increase in latency and decrease in amplitude of cortical responses, leading to insufficient monitoring.³ In this particular case, due to the size of the patient and estimated length of the case, it was felt a purely intravenous technique would be impractical from a cost and resource management standpoint. The chosen volatile agent (isoflurane) was

maintained at low levels during the beginning of the case so as not to impede the neurological monitoring. Inhalation agents lead to a dose dependent increase in latency and a decreased amplitude.³ Therefore, if the SSEP and MEP baselines are established after the volatile agent has been initiated, maintaining a constant low level of volatile agent should still allow one to monitor trend changes in neurological status.

Patients who have been affected by diabetes mellitus for several years often develop peripheral diabetic neuropathy. Diabetic neuropathy results from axonal degeneration and primary demyelination from schwann cell dysfunction. Long neuronal fibers appear to be affected first as symptoms initially appear distally in the hands and feet then progress proximally. Small myelinated and unmyelinated somatosensory fibers also become involved.⁴ When diabetic neuropathy has become extensive, it will be difficult to obtain a reliable intraoperative recording of MEPs and SSEPs, regardless of the anesthetic technique used. It was decided to discontinue MEP and SSEP monitoring during the case based on the poor quality of monitoring.

An option in this scenario would have been to convert to a total intravenous anesthesia (TIVA) technique to remove any influence of volatile anesthetic on signal acquisition. However, it was decided TIVA would not be beneficial as the significance of the patient's diabetic neuropathy with the additional complications of spinal cord compression from the epidural abscess led to the difficulty obtaining data during this case.

Decompression of a spinal abscess may require an anterior approach, depending on the location of the lesion. The surgeon can gain excellent access to the to the anterior thoracic spine, the vertebral bodies, intervertebral disks, spinal canal, and nerve

roots by using a transthoracic approach.⁵ A posterior approach may be applied if spinal stabilization is necessary. The patient in the present case had significant osteomyelitis along with his advanced epidural abscess, such that an anterior approach was performed to drain the abscess and later a posterior approach was used in a procedure to stabilize the spine.

The transthoracic approach typically requires one lung ventilation (OLV) to optimize the surgical view. However, one lung ventilation can be difficult with the morbidly obese patient. According to Lohser, et al., "successful OLV in morbidly obese patients is technically possible in the lateral position if the panniculus falls away from the body and unloads the dependent diaphragm."⁶ Patient positioning can also affect the peak airway pressures. In a study by Brodsky, et al., it was demonstrated that higher peak pressures were required to deliver the same tidal volumes to single lung ventilated patients in the lateral position as compared to patients in the supine position.⁷ In the lateral position, the nondependent lung is easier to ventilate. The dependent lung is preferentially perfused. Together, these lead to ventilation/perfusion mismatch. Systemic hypoxia related to a large alveolar to arterial oxygen tension difference often occurs with OLV.⁷ Intermittent alveolar recruitment, continuous positive airway pressure to the collapsed lung or positive-end-expiratory pressure may be crucial to avoid hypoxia in the patient.⁷ The morbidly obese patient in the present case required increased peak airway pressures (28-40mmHg) for mechanical ventilation with a tidal volume of 8 ml/kg and maintaining an end-tidal CO₂ of less than 40 mmHg.

This patient had limited mobility due to his size, and he was assumed to suffer from obstructive sleep apnea. This patient was

likely at increased risk for systemic hypoxemia related to a probable ventilation/perfusion mismatch and subsequent postoperative pulmonary complications if OLV was utilized during this surgery. When planning for the case, it was felt that OLV would offer the most advantageous surgical visualization of the epidural abscess, however, during the case, based on the individual patient condition including morbid obesity, poor general condition, persistent fluid in the ETT, and high peak airway pressures, the decision was made to retract the lung for surgical exposure of the spine rather than attempt to optimize their view by requiring OLV.

Spinal surgeries are delicate procedures requiring vigilant monitoring and care by the anesthesia practitioner. Field exposure should be maximized and when possible, neurological monitors should be utilized. While much attention is often given to glucose management in diabetes, one must also realize the disease may play a significant role in neurological monitoring. It may be valuable to obtain pre-operative baseline readings using SSEP and MEP monitors to suggest the efficiency of intraoperative monitoring. Obesity also increases the difficulty of positioning, quality surgical exposure, and ventilation. An anesthesia practitioner must consider all aspects of an individual's health when determining techniques to use for administering a safe and effective anesthetic.

References

1. Bluman EM, Palumbo MA, Lucas PR. Spinal epidural abscess in adults. *J Am Acad Orthop Surg.* 2004;12(3):155-163.
2. Hayahsi H, Kawaguchi M, Abe R, et al. Evaluation of the applicability of sevoflurane during post-tetanic myogenic motor evoked potential monitoring in patients undergoing spinal surgery. *J Anesth.* 2009;23:175-181.
3. Cann DF. Acute hypotension in a patient undergoing posttraumatic cervical spine fusion with somatosensory and motor-evoked potential monitoring while under total intravenous anesthesia: A case report. *AANA J.* 2009;77(1):38-41.
4. Said G. Diabetic neuropathy-a review. *Nature.* 2007;3:331-340.
5. Komanapalli CB, Eller JL, Sukumar MS. *Thoracotomy for Exposure of the Spine.* CTSNet. 2007. Available at http://www.ctsnet.org/sections/clinicalresources/thoracic/expert_tech-37.html. Accessed June 17, 2009.
6. Lohser J, Kulkarni V, Brodsky JB. Anesthesia for thoracic surgery in morbidly obese patients. *Curr Opin Anaesthesiol* 2007;20:10-14.
7. Brodsky JB, Wyner J, Ehrenwerth J, Merrell RC, Cohn RB. One-lung anesthesia in morbidly obese patients. *Anesthesiology.* 1982;57:132-134.

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Anesthetic Management of the Parturient with Eclampsia

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Keywords: preeclampsia, eclampsia, obstetric anesthesia

Anesthesia practitioners are often required to manage obstetric patients diagnosed with preeclampsia. Practitioners must be acutely

aware of the hemodynamic and physical changes associated with this condition and prepared for the potential development of eclampsia. This case report examines a parturient presenting with severe preeclampsia that rapidly progressed to eclampsia. Seizure activity refractory to treatment resulted, requiring an emergent cesarean section due to maternal and fetal compromise.

Case Report

A 28 year-old female presented for induction of labor due to severe preeclampsia, with diagnostic criteria including hypertension post 20 weeks gestation (range of 140s-150s/76-99), intra-uterine growth retardation, and headache. Two large bore intravenous (IV) catheters were administered, labs drawn, and a magnesium sulfate infusion initiated at 2 grams/hour after a 4 gram magnesium sulfate bolus. Epidural placement was planned when the patient had dilated to 4 cm or experienced discomfort.

During an assessment of deep tendon reflexes, the patient exhibited right arm tremors which progressed to a grand mal seizure. Oxygen was delivered via non-rebreather facemask and an additional bolus of 8 grams magnesium sulfate administered without termination of seizure activity. Pharmacologic therapy progressed to thiopental 50 mg IV and seizure activity abruptly halted. The patient's vital signs were stable and fetal heart tones appropriate upon reassessment. Epidural catheter placement was planned in thirty minutes barring any additional seizure activity. Thirty-one minutes later, the patient demonstrated further seizure activity. Additional boluses of thiopental 50 mg were administered in conjunction with the continued magnesium sulfate infusion.

Despite therapy, seizure frequency increased to approximately 15 episodes over the next 45 minutes. After consultation with neurology, a phenytoin infusion was initiated. Shortly thereafter, the patient once again demonstrated seizure activity, with resultant loss of all IV access.

Simultaneously, the patient's oxygen saturation decreased and signs of fetal compromise observed. Large bore IV access was regained and midazolam 2 mg IV was administered, which terminated further seizure activity. However, fetal heart rate decreased to the 80's with loss of variability and anesthesia for emergent cesarean section was requested by the attending obstetric physician.

Upon arrival to the operating room, standard monitors were applied, and a rapid sequence induction with cricoid pressure conducted utilizing thiopental 300 mg and succinylcholine 140 mg IV. Direct visual laryngoscopy with a Macintosh 3 blade revealed a large tongue, copious secretions, and an anterior airway with a grade 2 Cormack-Lehane view. After verification of endotracheal tube placement, a healthy fetus was delivered 45 seconds later. At completion of the surgery, the patient remained intubated and was transferred to the intensive care unit for further evaluation. A lorazepam infusion was initiated and the patient demonstrated no further seizure activity. Imaging of the brain via computed tomography revealed no structural abnormalities and further evaluation by neurology was unremarkable. The patient was discharged with nifedipine and levetiracetam for blood pressure and seizure control. The patient experienced no further seizure activity and did not demonstrate neurological deficits post discharge.

Discussion

Preeclampsia is a complex, multiorgan disease of unknown etiology that occurs in approximately 6-8% of pregnancies in the United States, and is the third leading cause of maternal death.¹ After 20 weeks of gestation, symptoms of systolic blood pressure (SBP) sustained above 140 mmHg or diastolic blood pressure (DBP) above 90 mmHg, coupled with proteinuria greater than 300 mg per 24 hours, confirm a diagnosis of preeclampsia. A diagnosis of severe preeclampsia is made when SBP remains elevated above 160 mmHg or DBP above 110 mmHg, proteinuria greater than 5 grams in 24 hours, or when evidence of end-organ damage exists. Eclampsia, which occurs in approximately 1% of the population, is a new onset of seizures or unexplained coma, occurring during or after pregnancy, with no other identifiable cause.^{1,2}

Careful management of the pre-eclamptic parturient is essential to aid in the prevention of eclampsia. Turner¹ suggests that managing hypertension and minimizing seizure risk are the most important goals of therapy with delivery of the fetus and placenta being the most definitive. Hypertension is commonly managed with antihypertensive medications such as labetalol, hydralazine, or calcium channel blockers. Nitroglycerin and nitroprusside are usually reserved for hypertensive emergencies.¹ Seizure prophylaxis is attempted with infused magnesium sulfate (MgSO₄). A bolus of 4-6 grams is recommended followed by a continuous infusion of 1-2 grams/hour. This generally results in a goal serum concentration of 5-8 mg/dl, which should raise the seizure threshold sufficiently to prevent seizure activity.¹

Once the pre-eclamptic parturient progresses to eclampsia, prompt intervention is required to minimize maternal and fetal morbidity and mortality. Subai² outlines a multi-step approach to management of the eclamptic parturient with the goals of maternal safety, respiratory support, and maintenance of cardiac function being paramount. Maternal safety measures employed include utilizing padded side rails, bite blocks, and restraints as necessary.² Seizure activity may result in hypoventilation and respiratory acidosis. Oxygenation is improved by administering oxygen via facemask. Continuous pulse oximetry should be monitored and arterial blood gas measurements obtained if oxygen saturation decreases to < 92%.² Termination and prevention of recurrent seizure activity is the next step in Subai's² recommendations. A bolus infusion of 6 grams MgSO₄ is recommended with a follow on continuous infusion of 2 grams/hour. Subai states that approximately 10% of the population will have additional seizures following this course of action and an additional 2 grams bolus infusion of MgSO₄ may be required. Additionally, seizures refractory to MgSO₄ infusion may require administration of barbiturate. Control of maternal hypertension is then addressed to minimize the risk of maternal cerebrovascular events and to ensure adequate uteroplacental perfusion. Subai² further states that the presence of eclampsia is not an absolute indication for cesarean section, however, assessment of maternal and fetal well being may dictate that an emergent delivery is indicated.

Regional anesthesia is the preferred method for cesarean deliveries in preeclamptic patients due to better hemodynamic control and avoidance of airway instrumentation³. Direct laryngoscopy may cause an acute increase in systemic and pulmonary hypertension, placing the parturient at risk

for cerebral hemorrhage, myocardial infarction, cardiac arrhythmias, and pulmonary edema^{3,4}. The decrease in uterine perfusion may also compromise oxygen delivery to the fetus. In addition to the aforementioned hemodynamic changes, Barash et al. raise the prospect of impaired airway anatomy visualization due to increased tongue size and swollen epiglottis or pharynx as an additional consideration. Thrombocytopenia and impaired coagulation may also cause significant airway bleeding during attempted laryngoscopy. Current literature supports both spinal and epidural anesthetic techniques. Dyer et al. explored the hemodynamic changes associated with spinal anesthesia in severe preeclampsia. Researchers concluded that spinal anesthesia produced clinically insignificant changes in cardiac output⁵. Either regional technique may also improve uteroplacental perfusion in the volume repleted patient placed in the left lateral position⁴.

The Practice Guidelines for Obstetric Anesthesia published by the American Association of Anesthesiologists⁶ provide little direct guidance for the management of the eclamptic parturient. They advocate early epidural placement in complicated obstetric patients as a measure to improve maternal and fetal outcomes and reduce maternal complications. The literature clearly states that general anesthetics carry a higher risk in the parturient population and avoidance when possible is prudent. This patient presented with severe preeclampsia that rapidly progressed to eclampsia. Medical management undertaken by the obstetric team was unsuccessful in controlling the patient's seizure activity. As

this case report demonstrates, earlier administration of epidural anesthesia may have resulted in avoidance of an emergent general anesthetic. Anesthesia professionals must continue to conduct careful individualized patient assessments and an evaluation of laboratory findings to formulate the most appropriate anesthetic plan for their patients.

References

1. Turner, JA: Severe preeclampsia: anesthetic implications of the disease and its management. *American Journal of Therapeutics*. 2009;16:284-288.
2. Subai, MB: Diagnosis, prevention, and management of eclampsia. *Obstetrics and Gynecology*. 2005;105(2):402-410.
3. Wali A, Suresh, M. Maternal morbidity, mortality, and risk assessment. *Anesthesiology Clinics*. 2008;26:197-230.
4. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia* 5th ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2006:1167.
5. Dyer RA, Piercy JL, Reed AR, Lombard CJ, Schoeman LK, James MF. Hemodynamic changes associated with spinal anesthesia for cesarean delivery in severe preeclampsia. *Anesthesiology*. 2008;108:802-811.
6. Practice guidelines for obstetric anesthesia. An updated report by the american society of anesthesiologists task force on obstetric anesthesia. *Anesthesiology*. 2007;106:843-6.

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Blood Loss in Pediatric Partial Nephrectomy for Wilms Tumor

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Keywords: Wilms tumor, bilateral partial nephrectomy, general anesthesia, blood loss, intra-operative blood transfusion

Wilms tumor is the most common childhood abdominal malignancy with an incidence of 5 to 7.8 per 1 million children under 15 years of age and accounts for 6% of all malignancies. It occurs bilaterally in 5% of patients.¹ Therapy includes surgery, chemotherapy, and radiation. Important anesthetic considerations include abdominal distention with delayed gastric emptying and potentially large intra-operative blood losses.¹ However, during nephrectomy, the requirement for blood transfusion has not been widely researched; therefore, there are no clear guidelines available for managing operative blood loss, especially in the pediatric population.²

Case Report

A 2 year old, 14.2 kg, 98cm, ASA Physical Status III male presented for a bilateral partial nephrectomy. The patient had developed abdominal distention and vomiting, and was diagnosed with Stage V Wilms Tumor confirmed by renal biopsy. Twelve weeks of chemotherapy followed.

The patient was born at full term without complications. He had a past history of pneumonia at age 2 not requiring hospitalization. He was allergic to amoxicillin. Medications included: enalapril, minoxidil, labetalol, trimethoprim-sulfamethoxazole and isradipine. On physical exam, a firm non-tender mass was noted in the left upper quadrant and laboratory values revealed a hemoglobin of

9.6g/dL and hematocrit of 27.7%. The remainder of the physical examination, laboratory tests, and vital signs were unremarkable. On the morning of surgery, no medication was administered to the patient and pre-operative vital signs included: blood pressure 93/79, heart rate 124bpm, respiratory rate 23, O₂ sat 100% on room air, and oral temperature 37° C.

Induction of anesthesia followed the application of standard monitors and pre-oxygenation. Fentanyl, propofol, and rocuronium were administered via the subcutaneous port, which remained continuously accessed throughout the case; direct laryngoscopy and oral intubation with a size 4.5 tube was successful. A nasogastric tube was inserted, as well as a peripheral IV and right radial arterial line. Temperature was monitored via an esophageal probe. A thoracic epidural catheter was placed, and a continuous infusion of 0.125% bupivacaine (4mL/hr) was initiated. The patient was turned into the prone position for surgery and an under and lower body forced air warming device was used. Antibiotics were administered prior to incision and at scheduled intervals.

A combination of oxygen, air, and Sevoflurane® (end tidal ranging between 0.6 and 1 MAC), was used for maintenance of anesthesia, along with intermittent doses of fentanyl and rocuronium. The surgery lasted 7 hours. Anesthesia was complicated by several episodes of hypotension, a systolic blood pressure less than 70mmHg, lasting 5-10 minutes per episode. CVP measurements ranged from 8-16mmHg. Total urine output equaled 155mL, and total

blood loss, which was slow but steady, was estimated at 700mL. Infused IV fluids included 4,200mL of lactated ringers, 654mL of packed red blood cells, 200mL of fresh frozen plasma, and 750mL of 5% albumin. All intravenous fluids were warmed, and the patient remained normothermic.

The epidural infusion rate was decreased, until off, during hypotensive episodes in an attempt minimize the sympathetic block and hypotension produced by the neuraxial block. IV fentanyl was administered on an as needed basis, totaling 160mcg at the end of the case.

At the end of the procedure, the patient was transferred, intubated, to the PICU. On postoperative day 1 the patient exhibited facial edema, bilateral basilar crackles, and metabolic acidosis (pH 7.27, pO₂ 101, pCO₂ 42, HCO₃ 19, BE -7.6). Vital signs remained stable, and the hemoglobin and hematocrit were 13.6g/dL and 38.4%. The patient was extubated on postoperative day 2 and discharged from the hospital on postoperative day 9 with no further complications.

Discussion

Massive blood loss is defined as the loss of one blood volume in a 24 hour period and, in an operative setting, is estimated to be 2-3mL/kg/minute or 50% of blood volume in a 3 hour period.³ Failure to maintain circulating blood volume results in inadequate oxygen delivery, hemodynamic instability, and manifestations of shock, while massive blood transfusions have been linked to cardiovascular instability, pulmonary dysfunction, metabolic derangement, increased risk of infection, and peri-operative coagulopathy.⁴ Therefore, accurate and continuous monitoring of blood

loss and timely replacement are essential, and the inability to prevent complications of over or under treatment may lead to adverse pediatric patient outcomes.^{3,5}

There are no guidelines particular to managing operative blood loss in the pediatric patient undergoing a nephrectomy.² The extent of the disease, vascularity or restrictive anatomy can lead to large blood losses.⁶ With the advantage of preserving renal function, the partial nephrectomy for removing small localized lesions has become widely accepted, although it is technically more complicated, and the risk of blood loss appears to be greater.²

In high risk cases, such as when pathology involves the renal vein or vena cava, the use of invasive monitoring and large bore peripheral IV catheters or central line catheters should be considered.^{1,7} Strategies to prevent blood loss include antifibrinolytics and control of arterial blood pressure while autologous transfusions and intra-operative blood salvage are options for blood replacement.⁷

Algorithms and protocols for transfusion therapy may be guided by laboratory tests; but, in instances of rapid blood loss, there is a lag between the real time situation and the availability of these lab results.⁵ In such cases, treatment is empirical and usually protocol driven.⁵

Careful assessment of blood loss by weighing blood soaked sponges, using calibrated miniature suction bottles, and making visual estimates of blood loss on surgical drapes allows for determination of total blood loss.³ However, it is difficult to measure blood loss accurately in the case of rapid hemorrhage.⁵ In these circumstances, the amount of fluid required to maintain

circulating blood volume should be used, as indicated by CVP and blood pressure⁵, as was the instance in the pediatric bilateral partial nephrectomy. For this case, 1,313mL was the total calculated fluid deficit, including NPO deficit, hourly maintenance, and 3rd space total. This deficit was replaced, plus additional crystalloid was administered to maintain a mean arterial blood pressure of 70 mmHg and a CVP of 9-10cmH2O.

Maximal allowable blood loss should be assessed before the induction of anesthesia. This measurement should take into account the estimated blood volume; age of the patient; pre- and intra-operative hematocrit, and cardiopulmonary and general medical conditions.³ The maximal allowable blood loss (MABL) equation should be used to determine the appropriate time to initiate red blood cell transfusion.³

$$\text{MABL} = \frac{(\text{Starting Hgb} - \text{Allowable low Hgb}) \times \text{EBV}}{\text{Starting Hgb}}$$

The nephrectomy patient's initial hematocrit was 27.7%. The transfusion trigger was a hematocrit of 24% with MABL to be approximately 124mL.

Once the transfusion threshold is reached, all further losses up to one blood volume should be replaced half by packed red blood cell (PRBC) concentrates and half by non-blood products.⁵ If a colloid solution, such as albumin, is used prior to reaching the threshold, it should be administered milliliter for milliliter of blood loss, while crystalloid solutions replacement should be at two to three times the amount of blood lost.³

The National Institute of Health Consensus Conference on Peri-operative Red Cell

Transfusion suggests that healthy patients with hemoglobin values of 10g/dL or greater rarely require transfusion, whereas those with hemoglobin values less than 7g/dL frequently require transfusion.³ Hemoglobin may be decreased temporarily as low as 7g/dL with monitoring of blood pressure and arterial and mixed venous PO₂.³ It is also suggested that the hemoglobin level be increased to 8g/dL with a hematocrit greater than 25% postoperatively when the patient requires more oxygen uptake for increased metabolic needs.³

According to Paterson⁵, with each blood volume lost, there is an approximate dilution of platelets by 50%. After the loss of two or three blood volumes, platelet transfusion threshold is reached, and platelet transfusion may be given with each blood volume transfused to reach a goal of 50,000/mm³.⁵ Administration of cryoprecipitate after the loss of three blood volumes is controversial.⁵ If hemostasis is controlled, a fibrinogen level should be sent to the laboratory before transfusion.⁵

Initiating the administration of PRBCs, FFP, and platelets should be triggered after the loss of two blood volumes.^{2,5,6} At this point, with expected continuing bleeding, all further losses should be replaced with whole blood, and calcium administration should be considered for every blood volume lost to treat hypocalcaemia resulting from the rapid infusion of anticoagulated blood products.⁵

It is important to understand the physiology of the pediatric patient, the pathophysiology and disease process for which they present prior to surgery, and the surgical procedure.⁸ "Regardless of the anticipated blood loss for any procedure, the revision of transfusion triggers, a mutually agreed on transfusion algorithm, and reduced blood sampling can

result in blood savings for all pediatric patients”⁸.

References

1. Hammer G, Hall S, Davis P. Anesthesia for general abdominal, thoracic, urologic, and bariatric surgery. In Motoyama EK, Davis PJ, eds. *Smith's Anesthesia for Infants and Children*. 7th ed. Philadelphia: Mosby Elsevier; 2006:685-722.
2. Shvarts O, Tsui K, Smith RB, et al. Blood loss and the need for transfusion in patients who undergo partial or radical nephrectomy for renal cell carcinoma. *J Urol*. 2000;164:1160-1163.
3. Cohen I, Motoyama E. Pediatric intraoperative and postoperative management. In Motoyama EK, Davis PJ, eds. *Smith's Anesthesia for Infants and Children*. 7th ed. Philadelphia: Mosby Elsevier; 2006:359-395.
4. Pietrini D, Piastra M, Lamperti M. New trends in pediatric anesthesia. *Minerva Med*. 2009;75:191-199.
5. Paterson NA. Validation of a theoretically derived model for the management of massive blood loss in pediatric patients: a case report. *Paediatr Anaesth*. 2009;19:535-540.
6. Gilbert WB, Smith JA. Blood use strategies in urologic surgery. *Urology* 2000;55:461-467.
7. Bhananker SM, Ramamoorthy C, Geiduschek JM, et al. Anesthesia related cardiac arrest in children: update from the pediatric perioperative cardiac arrest registry. *Anesth Analg*. 2007;105:344-450.
8. Weldon BC. Blood conservation in pediatric anesthesia. *Anesthesiology Clin*. 2005;23: 347-361.

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Myocardial Infarction During Laparoscopic Nissen

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Keywords: myocardial infarction, laparoscopic complications, intraoperative hypotension, cardiac risk assessment, coronary ischemia

Cardiac complications, including perioperative myocardial infarction, are associated with significant risk for morbidity and mortality in patients undergoing non-cardiac surgery. Since many of these procedures are elective, there is adequate time to identify patients at increased risk using validated risk indices.¹ The following case report describes a patient undergoing laparoscopic nissen fundoplication who

exhibited ST segment elevation intraoperatively, which further progressed to acute myocardial infarction. The potential risk factors, complications, treatment, and prevention of myocardial ischemia will be discussed.

Case Report

A 45-year-old female, weighing 43kg (BMI 15.3) was scheduled for a diagnostic laparotomy with nissen fundoplication. Past medical history included hypertension, gastroesophageal reflux and a hiatal hernia. Her medications included famotidine and

metoprolol, which were both continued on the morning of surgery. Family history was pertinent for a sibling with cardiac death at age 50. Surgical history included a laparotomy and esophagogastroduodenoscopy performed five days prior to this procedure in which it was noted that the systolic blood pressure remained in the 60s for fifteen minutes following induction. The patient denied chest pain, shoulder pain, or shortness of breath, but reported weakness and nausea and vomiting for one week. Estimated functional capacity using Duke Activity Status Index was 4.7 peak metabolic equivalents.

Physical exam in the preoperative holding area demonstrated a blood pressure of 128/77, heart rate 57 bpm with a regular rate, respiratory rate 20, room air oxygen saturation 98% and clear breath sounds bilaterally. Preoperative chest x-ray and laboratory studies were unremarkable, except for a hematocrit of 31.2%. An electrocardiogram revealed sinus bradycardia and mild left ventricular hypertrophy. Baseline ST segments were within normal limits.

Midazolam 0.5 mg was administered intravenously (IV) in the preoperative area. Upon entering the operating room suite, 100% oxygen was administered via facemask. A rapid sequence induction of anesthesia was initiated with cricoid pressure, fentanyl 50mcg, lidocaine 60mg, propofol 80mg, and succinylcholine 60mg. Neuromuscular blockade was achieved with vecuronium and anesthesia maintained with sevoflurane and fentanyl.

The surgeon reported stomach perforation with initial trochar insertion, but the patient appeared hemodynamically stable. Approximately fifteen minutes after initial incision, 1-2mm ST segment elevations

were noted in inferior EKG leads. The patient remained in sinus bradycardia with a systolic blood pressure in the 80s. Oxygenation was maintained at 100% and the patient medicated with nitroglycerin 0.4mg sublingual tablet and fentanyl 25mcg. One minute after nitroglycerin administration, ST segment elevation returned to baseline, while the patient's heart rate remained sinus bradycardia in the 50s and systolic blood pressure decreased to 70s. Ephedrine 10 mg and calcium chloride 500 mg were then administered IV with a normal saline bolus of 250 ml. The systolic blood pressure subsequently increased to the low 100s. Thirty minutes later, Nitroglycerin 0.4mg sublingual tablet and magnesium sulfate 1 gm IV were administered for a second episode of ST segment elevations in inferior leads and ventricular ectopy. An arterial blood gas at that time did not demonstrate any abnormalities.

The remainder of the case was uneventful with an estimated blood loss of 200ml. Patient was admitted to the intensive care unit with a cardiology consult. Post operative chest x-ray, ABG, and chemistries were unremarkable. Significant post operative laboratory results included: hematocrit 28.9%, peak CPK 636 ng/mL, ckmb 30%, MB 4.8 ng/mL, and troponin 4.2 ng/mL. Echocardiogram on the day of surgery demonstrated normal cardiac function and the patient refused any additional cardiac testing. The remainder of the hospital course with uneventful and the patient was discharged on post operative day three.

Discussion

Cardiac complications are one of the most significant risks to patients undergoing noncardiac surgery. It is critical that a thorough preoperative evaluation is

conducted on all patients undergoing major surgery. The risk of perioperative cardiac complications can be assessed using standard indices such as the Goldman cardiac risk index, evaluation of patient's functional status, patient's current cardiovascular status and co morbidities, and the underlying surgical risks (type and length of surgical procedure).²

Laparoscopic procedures are gaining popularity over open procedures as they are minimally invasive, allow for faster wound healing, shorter hospitalization times, decreased pain and decreased bleeding. Risks of laparoscopy include carbon dioxide embolism, pneumoperitoneum, pneumothorax, subcutaneous emphysema, and damage to visceral organs. Carbon dioxide insufflations can cause hypercarbia, which may increase sympathetic tone and lead to hypertension and tachycardia.³ Increased intraabdominal pressures may cause hypotension due to decreased venous return and may impair ventilation leading to respiratory acidosis.³ Furthermore, placing the patient in a head up position reduces cardiac preload and cardiac index. The combined effects of anesthesia, head up position and initial peritoneal insufflations can reduced cardiac index as much as 50 %.⁴

Multiple mechanisms may be responsible for myocardial infarction which all appear to result from an imbalance in myocardial oxygen supply and demand. The potential triggers for myocardial ischemia include surgical stress, increased release of catecholamines, hypotension, tachycardia, and an inflammatory response.⁵ There is increased risk of plaque disruption and higher intravascular shear stress when tachycardia is combined with increased catecholamine levels.⁶ To prevent the effects of tachycardia, the 2007 American College of Cardiology and American Heart

Association guidelines recommend initiation of beta blocker therapy preoperatively and titration to a goal heart rate less than 65 beats/min.⁷ Benefits of beta blocker therapy include reduction of myocardial oxygen demand, increased myocardial oxygen delivery by prolongation of coronary diastolic filling time, and prevention of arrhythmias.⁸

Once signs of ischemia were detected, immediate interventions were performed to optimize the balance between oxygen supply and demand and prevent further injury. Anesthesia was maintained at an appropriate depth with a balance of narcotics and inhalational agent. The fraction of inspired oxygen was maintained at 1.0. Additional beta blocker medication would have been contraindicated due to a heart rate in the 40s. Nitroglycerin was administered to reduce infarct size and oxygen demand when ST segment elevation in inferior leads was noted on cardiac monitor. Surgeons were notified of hemodynamic changes and ST segment elevations throughout the operative procedure. Magnesium sulfate was administered for recurrent polymorphic ventricular ectopy. Review of the literature suggests additional intravenous fluid boluses of normal saline solution to restore intravascular volume instead of calcium chloride administration to transiently increase cardiac output and blood pressure.⁹

It is apparent in the above case study that intraoperative myocardial infarctions, although rare, do occur. This patient had a personal history of hypertension and a strong family history of cardiovascular disease and premature cardiac death. Estimated functional capacity was 4.7 METS. The Goldman Cardiac Risk Index was calculated as 3, which estimates a 0.7% risk of life-threatening complications.¹⁰ Five days prior to surgery the patient had

documented hypotension with systolic blood pressures in the sixties for fifteen minutes post induction of general anesthesia. With hypotension, coronary perfusion decreases as diastolic blood pressure falls below the levels of autoregulation. Kotter et al found that 25% of ischemic events were associated with a 20% decrease in systolic blood pressure.¹¹

Additional catecholamine surges from surgical stress, hemodynamic effects of insufflation and stomach perforation, created an imbalance of oxygen supply and demand, which lead to ST segment elevations and reperfusion arrhythmias. Additionally, acidosis may have contributed to the arrhythmias. In a 2000 study by Kuntz et al, subcutaneous tissue pH can decrease from 7.35 to 6.81 and blood pH can decrease to 7.17 during laparoscopic surgery.³ Intraabdominal insufflations with carbon dioxide may lead to decreased tissue and subcutaneous blood flow in the abdominal wall. In this case, stomach perforation with initial trochar insertion may also have contributed to greater absorption of carbon dioxide in the blood and ventilation-perfusion mismatch

Review of the literature suggests that the best strategy to employ in managing these cases is to conduct a thorough preoperative assessment, evaluate patient and surgical cardiac risk factors, and perioperatively optimize cardiac status utilizing the most recent American Heart Association guidelines.

References

1. Kertai MD, Klein J, Bax JJ, Poldermans D. Predicting Perioperative cardiac risk, *Prog Cardiovas Dis*. 2005 Jan-Feb;47(4):240-57.
2. Shammash JB, Mohler ER, Joffe I, Morgan J, Kimmel S. Perioperative myocardial infarction after noncardiac surgery. In Basow DS, ed. *UpToDate*. Waltham, MA; 2009.
3. Kuntz C, Wunsch A, Bodeker C, et al. Effect of pressure and gas type on intraabdominal, subcutaneous, and blood pH in Laparoscopy. *Surg Endosc*. 2000;14(4):367-371.
4. Joris JL, Noirot DP, Legrand MJ, Jacquet NJ, Lamy ML. Hemodynamic Changes during Laparoscopic Cholecystectomy. *Anesth and Analg*. 2003; 76:1067-1071.
5. Devereaux PJ, Goldman L, Cook DJ, Gilbert K, Leslie K, Guyatt GH. Perioperative cardiac events in patients undergoing noncardiac surgery: a review of the magnitude of the problem, the pathophysiology of the events and methods to estimate and communicate risk, *CMAJ*. 2005;173:627-34.
6. London MJ. Multilead precordial ST-segment monitoring: "the next generation?" *Anesthesiology*. 2002;96(2):259-262.
7. Fleisher LA, Beckman JA, Brown KA, et al. ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery) developed in collaboration with the American Society of Echocardiography, American Society of Nuclear Cardiology, Heart Rhythm Society, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society for Vascular Surgery. *JACC*. 2007;50:e159.

8. Poldermans D, Boersma E. Beta – blocker therapy in noncardiac surgery. *NEJM*. 2005; 353:412.
9. Antman EM, Anbe DT, Armstrong PW, et al. ACC.AHA guidelines for the management of patients with ST-Elevation myocardial infarction-executive summary: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing committee to revise the 1999 guidelines for the management of patients with acute myocardial infarction). *JACC*.2004;44(3):671-719.
10. Goldman L, Caldera DL, Nussbaum SR, et al. Multifactorial index of cardiac risk in noncardiac surgical procedures. *NEJM*. 1977;297:845-50.
11. Kotter A, Kotrly K, Kalbfleisch J, Vucins EJ, Kampine JP. Myocardial ischemia during cardiovascular surgery as detected by an ST segment trend monitoring. *J Cardiothorac Anesth*. 1987;190-199.

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Hereditary Spastic Paraplegia and Neuromuscular Blockade

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Keywords: hereditary spastic paraplegia, Strumpell’s disease, neurological disease, neuromuscular blocking agent, muscle relaxant

Patients with neurological conditions often present a challenge for anesthetic planning. Anesthesia professionals must stay current regarding knowledge of neurological diseases in order to diligently plan an appropriate anesthetic for patients of this population. These conditions often alter the types and amounts of medications administered. Hereditary spastic paraplegia (HSP) is an inherited neurodegenerative disorder which affects corticospinal tracts within the spinal cord. It often results in progressive spasticity and stiffness of lower extremity muscles. Various forms of HSP exist. HSP affects around three out of 100,000 individuals in the United States and Europe.¹

Case Report

A 59 year-old female, 84.8 kg and 64 inches tall, presented to the operating room (OR) for an exploratory laparotomy with possible total abdominal hysterectomy, bilateral salpingo- oophorectomy and lymph node biopsy. She had no drug allergies. Previous surgeries included exploratory laparotomy, wrist surgery, cesarean section and suprapubic catheter placement. Medical history included hypertension, recent unintended weight loss, hereditary spastic paraplegia, acute renal failure, gastroesophageal reflux disease and chronic constipation. Current medications included donepezil, losartan, multivitamin, nitrofurantoin, omeprazole, polyethylene glycol, senna, potassium chloride, spironolactone, triamterene and hydrochlorothiazide, venlafaxine, tizanidine, and dantrolene. The patient had been admitted one week prior with shortness of breath, upper gastro-intestinal bleed, nausea and vomiting, and abdominal distention. An

esophagogastroduodenoscopy was inconclusive. A paracentesis was nondiagnostic. An abdominal/pelvic CT scan was completed and confirmed a mass near the left ovary.

The patient was transferred to the OR suite. Standard monitors were applied. Vital signs included a blood pressure of 161/75, heart rate of 105 beats/minute and an SpO₂ of 94%. Midazolam 2 mg intravenous (IV) and fentanyl 100 mcg IV were given. She was denitrogenated. A modified rapid sequence induction was performed with rocuronium 5 mg IV, lidocaine 40 mg IV and propofol 170 mg IV. After confirmation of easy mask ventilation, rocuronium 35 mg IV was given. Direct laryngoscopy was successful. A 7.0 mm endotracheal tube (ETT) was inserted into the trachea, and placement was confirmed. Volume control was utilized for mechanical ventilation. The inhalational agent used was sevoflurane. Total narcotics given were fentanyl 550 mcg IV and hydromorphone 1 mg IV. Total rocuronium was 60 mg IV. Surgical time was two hours and 50 minutes.

An awake extubation was planned. Train of four count (TOF) was 4/4 at case completion. Neuromuscular blockade was antagonized with neostigmine 4 mg IV after glycopyrrolate 0.4 mg IV. Spontaneous respirations returned. Tidal volume was 450 mL. SpO₂ was 97%. She was slow to wake. Hand grasp and head lift were weak. Eye opening was minimal. After five minutes, nalbuphine 10 mg IV was given. Five minutes later, she had sustained eye contact and head lift, and a firmer hand grasp. The oral cavity was suctioned and the ETT was removed. Oxygen was administered via nasal cannula. RR was 14 breaths/minute and regular. SpO₂ was 97%. She was taken to the recovery room. Within 10 minutes, her RR was 24 breaths/minute, irregular and

shallow with accessory muscle use. SpO₂ was 91-94%. Propofol 100 mg IV was given. The trachea was reintubated. Previous ventilator settings were resumed. Extubation occurred two hours and 15 minutes later. Respiratory status remained stable.

Discussion

The use of neuromuscular blocking agents can be complex in patients with paraplegia. The use of succinylcholine is contraindicated in these patients as it may induce hyperkalemia due to upregulation of nicotinic acetylcholine receptors.^{1,2} Nondepolarizing muscle relaxants should also be used cautiously because of a possible exaggerated muscle relaxant response.³ Past literature has alleged that regional anesthesia (RA) may exacerbate neurological symptoms, but recent information supports the use of RA in this patient population.⁴ Likewise, recent reports of administration of general anesthesia (GA) for patients with HSP have documented equivalent outcomes when compared to RA.⁵ Another study suggests that although it appears RA remains the preferred anesthetic technique for the management of patients with HSP, GA with the use of nondepolarizing muscle blockade can be a safe alternative if RA is contraindicated.⁶ For this particular case, the decision for GA was supported since the length of the procedure and the extent of the patient's condition were unknown. This decision was justified as the surgical incision was extensive, the ovarian tumor was 25 cm x 18 cm, and a total abdominal hysterectomy with bilateral salpingo oophorectomy and lymph node biopsies subsequently followed.

When caring for a patient with HSP, one must be very diligent in determining extubation readiness, especially if a neuromuscular blocking agent (NMBA) was

administered to the patient. Methods to reduce the risk of residual neuromuscular blockade should always be utilized in this patient population. Long acting NMBAs should be avoided if possible. Routine neuromuscular monitoring during surgery should be performed with all patients receiving a NMBA. This can be achieved with a standard peripheral nerve stimulator.⁷ In a randomized study, patients were placed either in a group to receive TOF monitoring in the OR or to receive no intraoperative neuromuscular monitoring. Upon arrival to the recovery room, electromyography was used to quantify TOF ratios. Results showed 15% of patients in the TOF monitoring group had TOF ratios <0.7 in the recovery room, compared to 47% of subjects who had no intraoperative TOF monitoring.⁸ The use of acceleromyography monitoring during surgical procedures and prior to tracheal extubation may also reduce the incidence of residual paralysis. This tool is more sensitive for detecting residual neuromuscular block when compared to standard peripheral nerve stimulators. If significant muscle weakness is suspected, acceleromyography should be used prior to tracheal extubation. Total twitch suppression should be avoided and is usually not surgically necessary. NMBAs should routinely be antagonized with an anticholinesterase agent. Even though anticholinesterase agents do have adverse effects, benefits of reversal outweigh these adverse effects. Anticholinesterase agents should also be given as early as possible since full reversal for higher levels of neuromuscular blockade can require 20-30 minutes.⁷ Even when neostigmine is administered with a 4/4 TOF count upon case completion, up to 1/3 of patients may still have TOF ratios <0.9 20 minutes later.⁹

For this patient, multiple methods were used to reduce the risk of residual neuromuscular

blockade. No long acting neuromuscular blocking agents were used. Instead, zemuron, an intermediate-duration agent was administered. Frequent neuromuscular monitoring was carried out with a peripheral nerve stimulator to determine TOF count. The patient did not experience total twitch suppression. Since this patient was redosed with rocuronium one time, an anticholinesterase was given at least 20 minutes prior to bandage placement even though TOF count was 4/4 upon case completion. After a maximal neostigmine dose was administered and nalbuphine was administered, extubation criteria was achieved in the operating room. TOF count remained 4/4, tidal volumes were at least 450 mL, respirations were regular and nonlabored and SpO₂ remained at least 97% prior to extubation. The patient had adequate head lift for 5 seconds, strong bilateral hand grasps, eye contact for 5 seconds, nodded appropriately to questions and was calm. Acceleromyography was not utilized in determining extubation readiness for this patient.

Another factor which was taken into consideration during this procedure was that this patient also received dantrolene and tizanidine as scheduled medications. Dantrolene, used for acute treatment of malignant hyperthermia and for treatment of disorders causing spasticity, enhances the effects of nondepolarizing NMBAs.¹ Tizanidine is a centrally acting alpha 2-adrenergic agonist which is used for management of spasticity.¹⁰

In summary, the GA case was carried out in a similar fashion to what is suggested in the current literature for patients with HSP requiring a GA with the use of a neuromuscular blocking agent. Although this patient had all necessary clinical signs indicating that extubation would be successful, more time was needed for the

nondepolarizing muscle relaxant to be metabolized. By using all appropriate methods to reduce the risk of residual neuromuscular blockade, the risk of residual paralysis may be reduced, but not completely eliminated. Since current information states that patients with neurodegenerative diseases may be sensitive to nondepolarizing muscle relaxants, it may have been beneficial to transport this patient to the recovery room with the ETT in place and extubation delayed.

References

1. Miller RD, Fleisher LA, Johns RA, Savarese, JJ, Wiener-Kronish JP, Young WL. Pharmacology of muscle relaxants and their antagonists. Naguib M, Lien CA, eds. *Miller's anesthesia*. 6th ed. Philadelphia: Elsevier Churchill Livingstone; 2005:481-572.
2. Babu D. Hereditary spastic Paraplegia. Healthline. <http://www.healthline.com/galecontent/hereditary-spastic-paraplegia>. Accessed January 8, 2010.
3. Deruddre S, Marie M, Benhamou D. Subarachnoid anesthesia for cesarean delivery in a parturient with Strumpell-Lorrain Disease. *Anesth Analg*. 2006;102:1910-1911.
4. Aldrete JA, Reza-Medina M, Daud O, et al. Exacerbation of preexisting neurological deficits by neuraxial anesthesia: report of 7 cases. *J Clin Anesth*. 2005;17:304-13.
5. Kunisawa T, Takahata O, Takayama K, et al. Anesthetic management of a patient with hereditary spastic paraplegia. *Masui* 2002;51:64-6.
6. McIver T, Jolley D, Pescod D. General anesthesia and caesarean section for a patient with hereditary spastic paraparesis (Strumpell's disease). *Int J Obstet Anesth*. 2007;60:190-191.
7. Murphy GS. Residual neuromuscular blockade: incidence, assessment, and relevance in the postoperative period. *Minerva Anesthesiol*. 2006;72:97-109.
8. Shorten GD, Merk H, Sieber T. Perioperative train-of-four monitoring and residual curarization. *Can J Anaesth*. 1995;42:711-5.
9. Murphy GS, Szokol JW, Marymont JH, Franklin M, Avram MJ, Vender JS. Residual paralysis at the time of tracheal extubation. *Anesth Analg*. 2005;100:1840-5.
10. Kamen L, Henney HR, Runyan JD. A practical overview of tizanidine use for spasticity secondary to multiple sclerosis, stroke, and spinal cord injury. *Curr Med Res Opin*. 2008;24:425-39.

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Laryngeal Mask Airway Vs. Endotracheal Tube in the Asthmatic

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Keywords: laryngeal mask airway, asthma, gastroesophageal reflux disease, aspiration, endotracheal tube

Laryngeal Mask Airways are utilized as an alternative ventilation device to the facemask and endotracheal tube. They provide anesthesia practitioners flexibility in monitoring and controlling the patient's

anesthetic.¹ This report discusses which type of airway device is best suited for a patient with asthma and gastroesophageal reflux disease (GERD). LMA's are beneficial in an asthmatic patient because they reduce airway stimulation, thus decreasing the risk for bronchospasm. However, utilizing a LMA in a patient with GERD may increase the risk for aspiration. Selecting the proper airway device to ensure patient safety is top priority for the anesthesia professional.

Case Report

A 52 year old female weighing 82 kg presented for right total knee arthroplasty due to a three year history of knee pain. The patient's co-existing conditions and diseases included; poorly controlled asthma, mitral valve prolapse, hypertension, stroke, irritable bowel syndrome, well-controlled GERD, rheumatoid arthritis, fibromyalgia, and Meniere's disease. The patient's medications included: omeprazole, furosemide, albuterol, ipratropium, zolpidem, acetaminophen, and salicylic acid. Auscultation of the patient's lungs revealed bilateral wheezes to both upper and lower lobes. An albuterol and ipratropium nebulizer treatment was administered preoperatively after these findings. Patient allergies included erythromycin, penicillin, and eggs.

Regional anesthesia was offered and refused. Because of the patient's poorly controlled asthma and well controlled GERD the decision was made to administer general anesthesia through a #4 LMA classic. The patient was transported to the operating room and general anesthesia was induced with midazolam 2 mg IV, fentanyl 100 mcg IV, and sodium thiopental 250 mg IV. Respirations were then controlled by a mechanical ventilator. The mechanical ventilator was set on synchronized

intermittent mechanical ventilation (SIMV) at a rate of 10 breaths per minute and a tidal volume of 600ml with a peak inspiratory pressure of 20 cm H₂O. The patient's lungs were clear to auscultation. Proper placement of the LMA was confirmed with positive end tidal CO₂ and equal bilateral breath sounds. General anesthesia was maintained with sevoflurane 2.5% in oxygen at 2 L/min and air at 1 L/min.

Oxygenation became difficult and the SpO₂ progressively deteriorated. Capnography revealed end tidal CO₂ values between the ranges of 65 and 70 mm Hg. To rule out the possibility of an inappropriately seated LMA, the airway device was removed. It was then reinserted and reconfirmed. The diagnosis of bronchospasm was made upon assessment of wheezing breath sounds, tidal volumes of 400 mL and difficult ventilation. Mechanical ventilation was discontinued and the patient was manually ventilated with 100% FiO₂ oxygen. Despite efforts to improve oxygenation SpO₂ values remained low while end tidal CO₂ values remained high. A decision was made to place an endotracheal tube. Succinylcholine 100 mg IV and fentanyl 150 mcg were administered prior to the trachea being intubated and the respirations again controlled by the mechanical ventilator. The ventilator was set for a rate of 12 breaths per minute and a tidal volume of 700 mL. Peak pressures were 18 cm H₂O with clear bilateral breath sounds. Oxygenation immediately improved and SpO₂ and end tidal CO₂ values returned to baseline. Sevoflurane values were decreased and the patient began breathing spontaneously.

Prior to emergence fentanyl 50 mcg IV was administered and sevoflurane was discontinued, while the oxygen liter flow rate was increased to 10. The anesthesia professional opted to wake the patient prior

to extubating the trachea. Oxygen was administered via face mask at a liter flow rate of 8 on arrival to the recovery room. The post-operative recovery period was uneventful.

Discussion

Asthma is defined as a chronic disease characterized by an over reactive airway, chronic airway inflammation, and reversible expiratory airflow obstruction which is the result of small airway narrowing.³ Literature states the LMA may cause less laryngeal stimulation and thus reduce the risk of bronchospasm.² Other benefits of the LMA include: greater hemodynamic stability during induction and emergence, ease of placement, lower incidence of coughing and sore throat, and decreased anesthetic need for airway tolerance.² However, the LMA does not protect against aspiration and some practitioners may consider GERD a contraindication to placement of an LMA.

Contraindications for the laryngeal mask airway include gastroesophageal reflux, poor lung function, obesity, and cases requiring high positive pressure ventilation. Given the patients poorly controlled asthma and active wheezing preoperatively, and history of well controlled GERD, we felt a LMA would therefore be a better option. Although aspiration risk is the major adverse effect, literature states it has been estimated to occur at a comparable rate with the facemask or endotracheal intubation.⁴ Due to the low pressure seal of the LMA high inspiratory pressures may result in gastric insufflation. To minimize cuff leaks and gastric insufflation peak inspiratory pressure should be kept less than 20 cm H₂O.

Interestingly, studies have found that gastroesophageal reflux and asthma often are co-existing medical conditions. GERD

symptoms include pain, cough, and wheezing which all can worsen the patient's asthma condition. According to the literature asthmatic individuals with reflux have a dramatic decrease in lower esophageal sphincter tone, more frequent episodes of reflux, and a longer exposure time to oesophageal acid.⁵

This case is similar to this information in that the patient experienced both GERD and asthma symptoms. Oxygenation difficulties led the anesthesia professionals to diagnose the patient with a bronchospasm. The patient's anesthetic depth may have been inadequate upon surgical start which may have lead to the bronchospasm. High peak inspiratory pressures were required to provide adequate tidal volumes. Hand ventilation was initiated to adequately ventilate the patient and aimed at reducing end tidal CO₂ values. Sevoflurane, an excellent bronchodilator, was increased to 4% to possibly dilate the small airways of the patient making ventilation easier. Bag ventilation may have over pressurized the low pressure seal of the LMA causing gastric insufflation and placing the patient at increased risk of aspiration. The decision was then made by the anesthesia professionals to remove the LMA and intubate the patient's trachea when the efforts made to correct the inadequate oxygenation failed. Endotracheal tube insertion secured the patient's airway and resulted in Spo₂ and end tidal CO₂ values to return to baseline. Baseline values included a Spo₂ value of 98% and a beginning end tidal CO₂ value of 35 mm Hg. The mechanical ventilator was set with a slow inspiratory flow rate which allows optimal distribution of ventilation-to-perfusion. The inspiratory/expiratory ratio was set at 1:4. Expiratory airflow obstruction is a characteristic of asthma and adequate time is needed for passive exhalation to prevent air

trapping which often occurs in asthmatics.³ The patient's oxygenation vastly improved. An orogastric tube was inserted into the esophagus and advanced into the bowel, then placed on low intermittent suction. This step aimed to alleviate any gastric insufflation that may have occurred.

According to the literature although endotracheal tubes greatly reduce the risk of pulmonary aspiration they do not completely eliminate it. A study, conducted by Blunt, Young, Patil and Haddock, found that in anesthetized patients leakage around a lubricated ETT cuff occurred in 11% of patients and as high as 83% with unlubricated cuffs.⁶

Albuterol, a beta adrenergic receptor agonist, stimulates beta receptors on muscles around the airway causing them to relax and open.³ Albuterol was administered by meter dose inhaler through the end tidal CO₂ port and three valsalva breaths were given to ensure the drug reached the patient's small airways. The albuterol was administered as a pretreatment prior to preparing the patient for extubation to minimize the risk of bronchospasm. In addition to albuterol, lidocaine 1-2 mg/kg IV and opioids prior to emergence can be administered to reduce the risk of bronchospasm.

In order to decrease hyperreactive airway reflexes during emergence the literature states that it is important to remove the tracheal tube when the patient is in a deep plane of anesthesia.³ The anesthesia professionals opted to awaken the patient and allow protective reflexes to return due to the possibility of gastric insufflation and pulmonary aspiration. Both emergence and recovery room transfer were uneventful.

Although refused by the patient, the anesthesia professional felt regional

anesthesia would have been a more appropriate option. Regional anesthesia allows the patient to control their own airway and eliminates the need for airway instrumentation. General anesthesia was preferred by the patient, leading the anesthesia professional to place the LMA. LMAs are favorable in the asthmatic patient because they cause less airway irritation than an ETT but do not protect the patient against aspiration. The risk of bronchospasm is not eliminated with a LMA, but it is less compared to an ETT. Anesthesia professionals should ensure that pulmonary function is optimized preoperatively regardless of what airway device is used. Patient characteristics must be taken into consideration when selecting an airway device.

References

1. Middleton P. Insertion techniques of the laryngeal mask airway: A literature review. *J Periop Prac* [serial online]. 2009; 19: 31-35. Available at <http://web.ebscohost.com.ezproxy.undm.edlibrary.org/ehost/pdf?vid=4&hid=108&sid=b13ddf3e-5318-449a-8666-8ac6c2a7a9c0%40sessionmgr110>. Accessed December 16, 2009. PMID: 19260359.
2. Afzal M. Airway Management in Pediatric Anesthesia: Laryngeal Mask Airway vs Endotracheal Tube. *The Internet Journal of Anesthesiology*. 2007; 13. Available at: http://www.ispub.com/journal/the_internet_journal_of_anesthesiology/volume_13_number_1_1/article/airway_management_in_pediatric_anesthesia_laryngeal_mask_airway_vs_endotracheal_tube.htm l. Accessed December 16, 2009. ISSN: 1092-406X.

3. Stoelting R, Dierdorf S. *Handbook for Anesthesia and Co-Existing Disease* 2nd ed. Churchill Livingstone; 2002.
4. Dunn P. ed. *Clinical Anesthesia Procedures of the Massachusetts General Hospital* 7th ed. Lippincott Williams & Wilkins; 2007.
5. Gibson PG, Henry R, Coughlan J. Gastro-esophageal reflux treatment for asthma in adults and children. *Cochrane Database of Systematic Reviews*. 2003. Available at: <http://www.mrw.interscience.wiley.com.ezproxy.undmedlibrary.org/cochrane/clsysrev/articles/CD001496/frame.html>.
6. Khazin V, Ezri T, Yishai R, et al. Gastroesophageal regurgitation during anesthesia and controlled ventilation with six airway devices. *J Clin Anesth* [serial online]. 2008; 7:508-513. Available at: <http://www.ncbi.nlm.nih.gov.ezproxy.undmedlibrary.org/sites/entrez>. Accessed December 22, 2009. PMID: 19019665.

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Subcutaneous Emphysema During Laparoscopic Hernia Repair

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Keywords: carbon dioxide, inguinal hernia repair, insufflation, laparoscopic surgery, subcutaneous emphysema

Laparoscopic surgery is frequently used for intra-abdominal procedures such as inguinal hernia repair. It is a popular, minimally invasive surgical approach. The following case study reviews a laparoscopic left inguinal hernia repair in which the adult patient experienced subcutaneous emphysema. Patient risk factors associated with the development of subcutaneous emphysema during a laparoscopic procedure are also reviewed.

Case Report

A 33-year-old male was scheduled for laparoscopic left inguinal hernia repair. The patient weighed 70 kg and was 175 cm tall. Past medical history was positive for an inguinal hernia. The patient denied medication use at home. Laboratory values were within the normal range. The patient

had an uneventful intravenous (IV) induction of general anesthesia with endotracheal tube placement, and was maintained on desflurane.

Thirty minutes after induction, and 5 minutes after insufflation of the extraperitoneal space, the patient's end-tidal CO₂ (E_tCO₂) began to rise from the initial measurement of 32 mmHg. Insufflation pressure at this time was 17 mm Hg. In response to the rising E_tCO₂, the minute ventilation was increased over the next 20 minutes. Despite changes in the ventilator settings, the EtCO₂ continued to climb, and reached a maximum value of 59 mmHg. On exam, it was noted that the patient's face was edematous. Crepitus was noted to the patient's thorax, neck and face, with closure of the palpebral fissure. The surgeon was notified of the presence of subcutaneous emphysema. The surgeon instructed the anesthesia practitioners to massage the patient's face in a caudad direction, which

was done for 5 minutes. At that time, the procedure was complete and the insufflation was released. Neuromuscular blockade was antagonized. The patient was mechanically ventilated until the EtCO₂ stabilized between 40-45 mm Hg. The patient then resumed spontaneous ventilation.

The trachea was extubated when the patient met standard extubation criteria and the patient was transported on room air to the post-anesthesia care unit while spontaneously ventilating without difficulty. The total operating room time was less than sixty minutes. The patient's neck remained swollen, with some crepitus noted to the thorax. The development of subcutaneous emphysema was explained to the patient and the patient's spouse and they both verbalized understanding.

Discussion

Laparoscopy is an endoscopic surgical approach that employs a pneumoperitoneum to visualize the surgical field. A pneumoperitoneum is the presence of air or gas, in this instance CO₂, within the peritoneum. This is an approach that was first employed in the 1970s for diagnosing gynecologic conditions. It has since evolved to be used for gastrointestinal, gynecologic, urologic and vascular surgery. The development of CO₂ subcutaneous emphysema is considered to be a frequent side effect of laparoscopic inguinal hernia repair. In the instance of inguinal hernia repair, the extraperitoneal space is intentionally insufflated.¹ The trocars used in laparoscopy pass through two layers in the abdominal wall, the skin and the muscle layer. The trocar is meant to seal the site and not allow CO₂ to escape. If the inner tissue seal is loose and the skin seal is tight, subcutaneous emphysema may result.² Laparoscopy is advantageous over open

techniques because it reduces postoperative pain, pulmonary impairment and incidence of ileus. It also contributes to shortened hospital stays, less scarring and earlier ambulation.³

In a prospective study of 100 patients undergoing laparoscopic hernia repair, Saggari et al. found that 99% of patients developed varying degrees of subcutaneous emphysema.⁴ Saggari et al. explains that the incidence of subcutaneous emphysema development is underreported, and there are specific factors contributing to the development of this common side effect.⁴ These factors include higher insufflation pressure, prolonged operating time, the use of six or more surgical ports, a Body Mass Index (BMI) of less than 25, and a higher E_tCO₂ (starting, peak and difference). Factors applicable to the case report patient include a BMI of 22.9 and a starting E_tCO₂ of 32 mm Hg with a peak of 59 mm Hg and of difference of 17 mm Hg. According to Saggari et al., the incidence of developing subcutaneous emphysema with a BMI of less than 25 is 66.7% (*p*-value = 0.029). The occurrence of developing subcutaneous emphysema with a starting E_tCO₂ of greater than 30 mm Hg is 75% (*p*-value = 0.011), with a peak of greater than 40 mm Hg is 88.9% (*p*-value = 0.000), and difference between start and peak of greater than 10 mm Hg is 85.7% (*p*-value = 0.000).⁴

The development of subcutaneous emphysema is an underreported problem, which may be due to it being a seemingly harmless complication unless hypercarbia becomes a threat to the patient.² Pneumothorax and pneumopericardium have been rare but associated complications of subcutaneous emphysema.² More common complications are related to patient discomfort, short-term disfigurement,

anxiety, respiratory failure, and longer hospital stays.⁵

While subcutaneous emphysema will spontaneously resolve between 1 and 4 days postoperatively,⁴ massage has been successfully implemented.⁵ Massage has also been paired with the placement of one or more modified fenestrated catheters.⁵ This can allow for air to escape when massage of the subcutaneous tissue is directed toward the preplaced catheter. Successful treatment with placement of a 14-gauge angiocath catheter has been documented. Srinivas et al. explains the placement of modified angiocaths using sterile technique. The angiocaths were modified with fenestrations using a scalpel while the angiocath remained on the steel stylette.

Despite the fact that subcutaneous emphysema is a common if not unavoidable complication of laparoscopic inguinal hernia repair, the patient from the case report was not prepared for this complication. While massage was the applied treatment, the placement of a catheter may have allowed air to escape faster. Massage did ease the disfigurement in this patient, but the subcutaneous emphysema had to resolve spontaneously. The patient may have had more rapid resolution with the placement of one or more subcutaneous catheters.

Patients who are undergoing laparoscopic procedures are often fully draped with dim operating room lighting and in positions (Trendelenburg or reverse Trendelenburg) that may make it difficult to visualize changes in the patient's appearance. It is the vigilant anesthesia professional's responsibility to be aware of the possibility

of the development of subcutaneous emphysema. In addition, considering the patient's risk factors of higher insufflation pressure, prolonged operating time, the use of six or more surgical ports, a Body Mass Index (BMI) of less than 25, and a higher E_tCO₂ (starting, peak and difference) is vital to maintaining total patient care.

References

1. Joris JL. Anesthesia for Laparoscopic Surgery. In Miller RD, Fleisher LA, Johns RA, Savarese JJ, Wiener-Kronish JP, Young WL, eds. *Miller's Anesthesia*. 6th ed. Philadelphia, PA: Elsevier Churchill Livingstone; 2005:2288.
2. Singh K, Singhal A, Saggarr VR, Sharma B, Sarangi R. Subcutaneous carbon dioxide emphysema following endoscopic extraperitoneal hernia repair: possible mechanisms. *J Laparoendosc Adv Surg Tech*. 2004;14:317-320.
3. Morgan GE, Mikhail MS, Murray. *Clinical Anesthesiology*. 4th ed. New York, NY: McGraw-Hill; 2006:582.
4. Saggarr VR, Singhal A, Singh K, Sharma, B, Sarangi R. Factors influencing the development of subcutaneous carbon dioxide emphysema in laparoscopic totally extraperitoneal inguinal hernia repair. *J Laparoendosc Adv Surg Tech*. 2008;18:213-216.
5. Srinivas R, Singh N, Agarwal R, Aggarwal AN. Management of extensive subcutaneous emphysema and pneumomediastinum by micro-drainage: time for a re-think? *Singapore Med J*. 2007;48:323-326.

Mentor: Kevin C. Buettner, CRNA, MS

Loss of Motor Evoked Potentials during Distraction

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Keywords: motor evoked potentials, pediatric distraction, pediatric anesthesia for spinal surgery, scoliosis surgery, pediatric spinal instrumentation

The anesthesia management for a short procedure, such as distraction, can be challenging when patient immobility is necessary and the loss of motor evoked potentials (MEPs) are undesirable.

Distraction, the lengthening of growing rods, is typically done every 6 months after the initial spinal instrumentation surgery, is a technique that allows for the pediatric patient to grow in height but more importantly prevents pulmonary compromise.¹ Since this surgical procedure is typically performed twice a year from ages 9-14, evaluating the record of the patient's past distractions, and having alternative plans for anesthesia is vital for patient safety and surgeon satisfaction.¹

Case Report

A 9 year old female weighing 54.3 kg with a height of 142 cm presented for distraction of growing rods. Past medical history stated congenital scoliosis involving the thorax and lumbar regions. This patient had no neurological deficits, normal pulmonary function, or no other comorbidities. The patient had no known allergies and was not taking any medications. Vital signs and lab values were within normal ranges.

Upon patient's arrival in the operating room, standard monitors were applied. Once vital signs were established, she was administered oxygen 4 liters/minute, nitrous oxide 4 liters/minute, and sevoflurane 8% per mask.

Fentanyl 100mcg IV followed by propofol 100mg IV were administered prior to intubation. The patient's trachea was successfully intubated without difficulty. After tracheal intubation, the nitrous oxide was discontinued and anesthesia was maintained with oxygen 1 liter/minute, air 1 liter/minute with a sevoflurane end tidal of 1.4%, in addition to a propofol continuous infusion of 75mcg/kg/min.

The prone patient did not move with incision, but once the surgeon attempted distraction, the patient moved her upper extremities. Fentanyl 25mcg IV was given along with a propofol bolus of 30mg IV. Following additional undesired movements, the patient was given fentanyl 75mcg IV and the propofol infusion was increased to 100mcg/kg/min. Within minutes of surgical stimulation the patient moved once more. Midazolam 1mg IV was administered at this time. Finally sevoflurane was steadily increased to an end tidal of 2.3%. Under the deeper anesthesia the patient did not move, but MEPs were lost while sensory evoked potentials remained intact. Throughout the case, the patient's systolic blood pressure was 100-120 mmHg with a heart rate ranging from 70-80 beats per minute.

The surgeon finished the distraction within 5 minutes. Immediately post-distraction, the sevoflurane was decreased to an end tidal of 1.3% and the propofol infusion was decreased to 75mcg/kg/min. The patient's MEPs returned to normal limits within 5 minutes. The total time for loss of MEPs, was approximately 10 minutes.

The patient was unable to be extubated in the operating room due to apnea. In the recovery room, the patient's respirations became regular at 18 breaths per minute, and the trachea was extubated. A normal neurological assessment was documented in recovery area. There were no neurological sequelae from the loss of the MEPs that transiently occurred intraoperatively.

Discussion

The concern in this case was the loss of motor evoked potentials which is suboptimal during spinal surgery. From a technical standpoint, MEP monitoring provides intraoperative detection of altered neural motor function due to altered blood flow caused by hypotension or a vascular insult.² Studies have shown not only decreased neural perfusion as the reason for the loss or change in MEPs, but also anesthesia can increase the latency, decrease the amplitude, or cause the complete loss of MEPs.³ In order to obtain an optimal interpretation of MEPs, astute anesthesia management and knowledge of MEPs are important.

In this particular case, initially fentanyl and propofol were given to inhibit patient movement. Narcotics do not significantly impact MEPs, but dose dependent amounts of propofol can potentially increase the latency and decrease the amplitude of MEPs.⁴

Since the patient continued to move with surgical stimulation after the additional intravenous fentanyl and propofol, intravenous midazolam was administered. According to Miller, midazolam increases latency and decreases amplitude of motor evoked potentials.⁴ This patient demonstrated no significant depression of motor evoked potentials 5 minutes after the administration of midazolam 1mg intravenously.

Lastly, an increase in the percentage of inspired sevoflurane was attempted to inhibit patient movement while the surgeon performed distraction of growing rod. All inhaled anesthetic gases have the potential to increase latency and decrease amplitude of MEPs.⁴ The potential for depression of MEPs increases as the inspired gas concentration increases.⁴ With the increase in end tidal sevoflurane, the patient's MEPs were lost.

Other anesthetic agents may have been a better option in preventing patient movement than midazolam and increasing the inspired amount of sevoflurane. Research suggests intravenous agents, such as opiates, ketamine, etomidate, and low dose propofol, cause less motor evoked potential depression than inhalational agents.⁴

Utilizing a continuous dose of intravenous remifentanyl may have stopped patient movement without interfering with the MEPs. Remifentanyl is an opioid which has a very short duration of action compared to fentanyl, but can cause hypotension, chest wall rigidity, and bradycardia in pediatrics.⁴ A study by Tobias et al⁵ utilized a propofol infusion starting at 100 mcg/kg/min along with a remifentanil infusion at 0.2 mcg/kg/min which provided an optimal surgical environment for spinal surgeries. If a continuous infusion of remifentanyl had been administered instead of fentanyl boluses, the remifentanyl may have provided a constant analgesic level without prolonging the patient's emergence.

Ketamine, an N-methyl-D-aspartate receptor antagonist, would have been another possible alternative anesthetic agent that may have inhibited patient movement during deep surgical stimulation. One study

suggested a low dose bolus of ketamine at 0.5mg/kg can be utilized to enhance anesthesia without inhibiting MEPs.⁶ Since distraction can be accomplished within minutes, ketamine 27mg given intravenously may have been effective in preventing patient movement while enhancing MEP monitoring.

Low dose intravenous etomidate is another possible drug option to inhibit patient movement. Etomidate administered intravenously at 0.1 mg/kg has been found to enhance MEP monitoring where as etomidate at higher doses decrease the amplitude of MEPs in monkeys.⁷ Since this study was performed on monkeys, other drug options may be preferable until conclusions are drawn using human samples.

The use of nitrous oxide has the potential to enhance anesthesia for short procedures such as distraction of growing rods. Nitrous oxide up to 50%, in combination with opioid, ketamine, or low dose propofol infusion, provides analgesia, increases the depth of anesthesia, and can be quickly eliminated because of its low solubility coefficient.³ Nitrous oxide could have been a possible solution after the first bolus of intravenous fentanyl had proven ineffective in preventing patient movement.

In conclusion, having a prepared, alternative anesthetic plan is essential when dealing with spinal surgeries during which MEPs are being monitored. Since distraction is a short surgical procedure, a TIVA using continuous propofol 100mcg/kg/min and remifentanyl 0.2 mcg/kg/min infusions would have been an appropriate plan of anesthesia. With this particular patient, considering how much anesthesia it took to inhibit movement, a ketamine bolus of 0.5 mg/kg or <50% N2O supplement might have

been necessary to allow the surgeon to perform the distraction in a timely manner.

References

1. Maruyama T, Takeshita K. Surgical treatment of scoliosis: A review of techniques currently applied. *Scoliosis*. 2008;3:1-6.
2. Schwartz D, Auerbach J, Dormans J, et al. Neurophysiological detection of impending spinal cord injury during scoliosis surgery. *J Bone Joint Surg Am*. 2007;89:2440-9.
3. Frei F, Ryhult S, Duitmann E, Hasler C, Luetsch J, Erb T. Intraoperative monitoring of motor-evoked potentials in children undergoing spinal surgery. *Spine*. 2007;32:911-917.
4. Miller R. *Miller's Anesthesia*. 6th ed. Philadelphia: Elsevier Churchill & Livingstone; 2005: Chapter Authors: Mahla M, Black S, Cucchiara R. 1533,1539-1540,2377.
5. Tobias J, Goble T, Bates G, Anderson J, Hoernschemeyer D. Effects of dexmedetomidine on intraoperative motor and somatosensory evoked potential monitoring during spinal surgery in adolescents. *Pediatr Anesth*. 2008;18:1082-1088.
6. Zaarour C, Engelhardt T, Strantzas S, Pehora C, Lewis S, Crawford M. Effect of low-dose ketamine on voltage requirement for transcranial electrical motor evoked potentials in children. *Spine*. 2007;32(22):627-630.
7. Sloan T, Rogers J. Dose and timing effect of etomidate on motor evoked potentials elicited by transcranial electric or magnetic stimulation in the monkey and baboon. *JCMC*. 2009;23(4):253-261.

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Cervical and Facial Subcutaneous Emphysema

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Keywords: laparoscopic surgery, subcutaneous emphysema, hiatal hernia repair, nissen fundoplication, airway compromise

Laparoscopic surgery has been utilized since 1937.¹ Its popularity and frequency of use has increased tremendously since its origination due to multiple benefits over traditional open techniques. These benefits include smaller incisions, decreased postoperative pain, less postoperative pulmonary impairment, shorter hospital stays, a reduction in postoperative ileus, earlier ambulation, smaller surgical scars, and lower cost.^{1,2} In order to visualize the surgical field during a laparoscopic surgery, a pneumoperitoneum is created by insufflating carbon dioxide (CO₂). Even though laparoscopic procedures offer numerous advantages, there are still risks and complications associated with laparoscopy such as bowel perforation, hemorrhage, gas embolism, pneumothorax, pneumomediastinum, and subcutaneous emphysema.²⁻⁴ It is imperative that anesthesia professionals are not only aware of these possible complications, but are able to take measures to prevent unfavorable outcomes associated with these complications.

Case Report

A 65 year old, 67 in, 70 kg female presented for a laparoscopic hiatal hernia repair with nissen fundoplication related to a history of gastroesophageal reflux disease (GERD) and hiatal hernia. Past medical history included: herpes zoster with occasional herpetic neuralgia, transient hypothyroidism, osteoarthritis, diverticulosis, tubular

adenoma, and adenomatous polyps. Past surgical history included a hysterectomy, oral surgery, and colonoscopy. The patient denied any history of anesthesia related complications or family history of anesthesia complications. The patient's current medications included: sertraline HCl, trazadone HCl, and vitamin B complex. The patient denied any known drug allergies. The electrocardiogram showed sinus bradycardia with sinus arrhythmia. The chest radiograph, preoperative labs, baseline vital signs, and physical and airway exams were unremarkable.

The patient received the following medications in the preoperative holding area: midazolam 2 mg and metoclopramide 10 mg intravenously and transdermal scopolamine 1.5 mg. The patient was transferred to the operating room and preoxygenated via face mask using 100% FiO₂ while standard monitors were applied. A rapid sequence induction with cricoid pressure was performed using fentanyl 125 mcg, rocuronium 5 mg, lidocaine 80 mg, propofol, 150 mg, and succinylcholine 100 mg IV. A 7 mm endotracheal tube (ETT) was placed atraumatically to a depth of 20 cm at the lip. Prior to the incision the patient received cefazolin 1 gm, dexamethasone 8 mg, ondansetron 4 mg, and cisatracurium 6 mg intravenously.

Anesthesia was maintained with sevoflurane 2% in air at 1 L/min and O₂ at 2 L/min, fentanyl for analgesia, and cisatracurium for neuromuscular blockade. Initial ventilation settings were volume control, rate of 10, with tidal volume of 550 ml. Adjustments in tidal volume and rate were made during the

case to maintain end-tidal CO₂ between 30-45 mmHg. During the case, the patient developed significant subcutaneous emphysema in the face and neck. The surgeon was immediately notified, endotracheal tube placement was confirmed and vesicular lung sounds were auscultated in all fields bilaterally. Due to the minimal amount of procedural time remaining and the extent of the facial subcutaneous emphysema, it was determined that the patient would remain intubated until the emphysema resolved and removal of the ETT was deemed safe. Near completion of the case, the patient received midazolam 2 mg, cisatracurium 4 mg, and ketorolac 30 mg intravenously.

Upon completion of the surgery, the patient was prepared for transport to the intensive care unit (ICU) for postoperative care. Immediately prior to transport to the ICU, the patient received midazolam 2 mg IV. Upon arrival to the ICU, mechanical ventilation was resumed. A midazolam infusion was started at 3 mg/hr and later a fentanyl infusion was added at 50 mcg/hr with a 25 mcg bolus. Sedation and analgesia were continued until resolution of the facial subcutaneous emphysema. After resolution of the facial subcutaneous emphysema and meeting extubation criteria, approximately 7 hours after the completion of surgery, the patient was extubated. The patient stated that she did not remember anything about the procedure. The patient was discharged the following day without further problems.

Discussion

The pneumoperitoneum created during laparoscopic procedures can create multiple complications such as hypercapnia, atelectasis, gas embolism, dysrhythmias, pharyngeal emphysema, pneumothorax, pneumomediastinum, and subcutaneous

emphysema.¹⁻⁴ The incidence of subcutaneous emphysema experienced during laparoscopic surgery ranges from 0.3% to 3%.³ The rate of incidence for pneumodissection into the neck post laparoscopic reflux surgery has been reported by researchers to be as high as 80%. The pneumodissection in these cases is usually due to a division of the phrenoesophageal ligament and mobilization of the distal esophagus thus allowing the dissection of CO₂.⁵

Several risk factors are associated with increased potential for the development of subcutaneous emphysema such as extraperitoneal laparoscopic procedures, increased age, multiple surgical ports, prolonged surgical time, and high insufflation pressures. The most influential of these are high insufflation pressures and prolonged surgical time (over 200 minutes).³

According to Arturi, four mechanisms can be used to explain the development of subcutaneous emphysema during CO₂ insufflation to obtain a pneumoperitoneum. The first mechanism involves improper placement of the Verres needle, in which there is incomplete penetration of the peritoneal cavity prior to insufflation, resulting in an accumulation of CO₂ in the subcutaneous tissue or in between the fascia and peritoneum. The second mechanism involves the dissection of CO₂ into the subcutaneous tissue through a trochar site after establishing a pneumoperitoneum. This mechanism results in subcutaneous emphysema present in the neck, face, and chest wall. This can be accompanied by either a pneumomediastinum and/or a pneumothorax, which result from the third mechanism, dissection along the aorta and inferior vena cava openings in the diaphragm. The fourth mechanism involves the use of excessive intra-abdominal

pressures, with the potential for the dissection of CO₂ outside the peritoneal cavity, which may result in a pneumoretroperitoneum. Intra-abdominal pressures should be kept within the range of 10 to 20 mmHg.⁴

Typically, the first recognized sign of subcutaneous emphysema and CO₂ absorption is high end-tidal CO₂. Subcutaneous emphysema is visually identified by a smooth bulging of the skin, which upon palpation produces a crackling sensation.³ Subcutaneous emphysema can be categorized into 4 categories ranging from 0 to 3. Category 0 is no subcutaneous emphysema present. Mild emphysema around the trocar insertion sites of groin is category 1. Subcutaneous emphysema extending into the abdomen and thigh would be indicative of category 2. Massive emphysema is categorized as 3 with subcutaneous emphysema extending into the chest, neck, and face region.¹

Once the development of subcutaneous emphysema has been identified a prompt evaluation of the patient's respiratory status should be conducted. This should include auscultation for bilateral breath sounds to rule out a pneumothorax and/ or pneumomediastinum, verification of endotracheal tube placement, and arterial blood gas analysis if necessary. The surgeon should be notified and the surgeon should inspect the diaphragm for defects in addition to instituting measures to reduce complications. The extent of the subcutaneous emphysema should be monitored closely.⁴ Mild subcutaneous emphysema typically resolves quickly and is not associated with negative patient outcomes.³ An increase in minute ventilation (MV) by 20% to 30% by adjusting tidal volume and/ or respiratory rate may be required to maintain eucapnia.¹ Massive

emphysema can be associated with several postoperative complications such as pharyngeal swelling, prolonged hypercapnia, airway compromise, facial swelling, temporary visual impairment, postoperative pain, neck swelling, and hypoventilation.^{1,3} Uncorrected acidosis associated with hypercapnia can result in arrhythmias, increased intracranial pressure, and depressed central nervous system.³ In the event that subcutaneous emphysema is still present upon completion of surgery a thorough airway and respiratory evaluation should be conducted in order to determine if the airway might be compromised following extubation.^{3,4} Direct visualization via laryngoscopy may be necessary to evaluate supraglottic edema.⁴ Short-term postoperative ventilation may be necessary to allow the CO₂ to diffuse out of the subcutaneous tissue and resolve any potential airway compromise.³ Post operative tests should include a chest radiograph and arterial blood gas. A postoperative chest radiograph is not only helpful to determine the extent of the subcutaneous emphysema, but to rule out other pathology such as pneumomediastinum or pneumothorax. Postoperative arterial blood gases can be utilized to evaluate the level of hypercapnia.^{1,3,4}

Laparoscopic surgical procedures have become increasingly popular due to the numerous benefits over open procedures. However, all surgical procedures have associated risks or complications. The incidence of subcutaneous emphysema associated with the creation of a pneumoperitoneum required for laparoscopic procedures ranges from 0.3% to 3%. The most influential risk factors for the development of subcutaneous emphysema are high insufflation pressures (intra-abdominal pressures should be kept

within the range of 10 to 20 mmHg) and prolonged surgical time (over 200 minutes). The first recognized sign of subcutaneous emphysema and CO₂ absorption is high end-tidal CO₂. It can be visually identified by a smooth bulging of the skin, which upon palpation produces a crackling sensation. Upon identification of subcutaneous emphysema, an immediate evaluation of the patient's respiratory status and airway must be conducted followed by notification of the surgeon and adjustment to minute ventilation to maintain eucapnia. At the completion of surgery, another thorough airway and respiratory evaluation should be conducted in order to determine if the airway might be compromised following extubation. Anesthesia professionals must be aware of the potential complications associated with laparoscopic procedures, risk factors, preventative measures, symptoms, and treatment in order to avoid potential negative patient outcomes.

References

1. Worrell JB, Cleary DT. Massive subcutaneous emphysema and hypercarbia: complications of carbon dioxide absorption during extraperitoneal and intraperitoneal laparoscopic surgery. *AANA J.* 2002;70:456-461.
2. Morgan GE, Mikhail MS, Murray MJ. *Clinical Anesthesiology.* 4th ed. McGraw-Hill;2006:582-584.
3. Lindsey S. Subcutaneous carbon dioxide emphysema following laparoscopic salpingo-oophorectomy: A case report. *AANA J.* 2008;76:282-285.
4. Arturi MA, Eckert T. Subcutaneous emphysema and potential airway compromise in laparoscopic-assisted procedures: A case report. *AANA J.* 1995;63:498-500.
5. Jen A, Sanelli PC, Honrado C, Huang C. Cervical subcutaneous emphysema after lower abdominal laparoscopic surgery. *Otolaryngol Head Neck Surg.* 2003;128:157-159.

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Negative Pressure Pulmonary Edema

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Keywords: Negative pressure pulmonary edema, upper airway obstruction, intra-thoracic pressure, pediatric, complication.

Negative pressure pulmonary edema (NPPE) occurs in approximately 0.05 to 0.1% of all patients receiving general anesthesia.^{1,4} In the patients with significant upper airway obstruction requiring intervention, 11% may develop NPPE.^{2,6,7,12} Risk factors for NPPE are multi-factorial, but young,

muscular male patients are especially prone due to their ability to generate large negative intrapleural pressures.^{4,5,7,11,14} Symptoms are usually seen immediately post-extubation; however, onset may occur several hours later.^{1,4,7} Because of the significant morbidity associated with NPPE, rapid diagnosis and treatment is crucial.^{1,2,4-6,10,12,15}

Case Report

A 12 year-old male presented for an elective right orchiectomy. He was 168 cm, 62 kg, and his surgical history was negative. His only medical problem was seasonal allergies for which he took oral Cetirizine 5 mg daily. The patient had an upper respiratory infection 1 week prior to surgery that included fever up to 100 degrees C, cough and sore throat. Preoperatively, he was afebrile and denied a sore throat, cough or sputum. He had a heart rate of 76 bpm with a regular rate and rhythm and lungs were clear to auscultation. He was assessed to have a Mallampati class II airway with a long slender neck and a thyromental distance of 6 cm.

The patient was brought into the operating room and an inhalation induction of oxygen 3 liters/minute, nitrous oxide 7 liters/minute, and sevoflurane 8% was delivered via face mask. The patient was easily mask ventilated with an oral airway. Fentanyl 30 mcg and rocuronium 15 mg were administered prior to intubation. His vocal cords were noted to be anterior and deep, but intubation was accomplished with a Miller 2 blade and a 6.5 mm ID cuffed oral endotracheal tube. There was no air leak at 20 cm H₂O pressure with the cuff of the endotracheal tube filled with 2 ml of air. Anesthesia was maintained with sevoflurane 3%, oxygen 1 liter/minute and nitrous oxide 2 liter/minute.

At the completion of surgery the patient was spontaneously breathing with 300-350 ml tidal volumes at a rate of 20 breaths per minute. Neostigmine 5 mg and glycopyrrolate 0.8 mg were administered. The patient was noted to have 4 twitches without fade using train-of-four monitoring per peripheral nerve stimulator, moving extremities to command, and eyes were in

the midline position. The patient's oropharynx was carefully suctioned before extubation, his oxygen saturation on room air was 99% and the patient was brought to the post anesthesia care unit (PACU). On arrival in PACU, his oxygen saturation was 99% on 40% oxygen via face tent. The patient coughed and became apneic. Jaw thrust and 100% oxygen were applied, as his oxygen saturation declined to 78%. The patient was assisted with a bag-mask device and his oxygen saturation rose to 94% within 2 minutes. Incentive spirometry and oxygen via humidified face mask were administered. After a short time he was noted to have pink frothy sputum and a chest x-ray confirmed pulmonary edema. The patient was admitted for overnight observation, continued oxygen therapy, and discharged in the morning on room air.

Discussion

NPPE is most frequently seen with the onset or relief of an upper airway obstruction, with an 11 to 40% occurrence of severe morbidity and mortality.^{4,6} NPPE can be caused by a plethora of factors associated with an upper airway obstruction. The patient's history of recent upper airway illness, an anatomically difficult airway for intubation and strong male chest musculature increased his risk of NPPE.¹¹⁻¹⁴

The causes of upper airway obstruction can also include: soft tissue masses, croup, epiglottitis, obesity, short neck, sleep apnea, near strangulation, allergic factors, occlusion of an artificial airway, gastroesophageal reflux with acid aspiration, upper digestive tract surgery, use of neuromuscular blocking for tracheal intubation, vigorous suctioning, strong hiccups during inhalational induction of general anesthesia, and most commonly laryngospasm.^{1, 3-8, 11-14}

Laryngospasm, possibly due to oral secretions, slow emergence from inhalational anesthetic or post-operative pain, is a frequent cause of NPPE in children.^{3, 9, 12-14}

Excessive fluid administration and preexisting heart and lung disease may also lead to NPPE.^{2, 5, 10} The Starling equation explains the net movement of fluid from intravascular to extravascular space.^{1, 13, 14} The balance of hydrostatic and oncotic pressures results in a small filtration of fluid into the interstitial space; therefore, pulmonary edema occurs when the volume of interstitial fluid exceeds the re-absorptive capabilities of the pulmonary lymphatic system.^{1, 2, 5, 6, 10, 12, 14} With acute airway obstruction there is an increase in inspiratory flow resistance. Consequently, there can be a strong negative intra-thoracic pressure of -50 to -100 cm H₂O against a closed glottis (known as a modified Mueller maneuver).^{2, 4, 6-9, 11, 13, 14} Intense negative intra-thoracic pressure increases the hydrostatic pressure gradient across the capillary wall and fluid begins to accumulate in the alveolar and interstitial spaces.^{1, 3, 6, 7, 11, 13, 14} Negative intra-thoracic pressure can increase preload to the right ventricle and the pulmonary capillary bed.^{1-3, 6, 8, 11}

With NPPE there is systemic vasoconstriction, a release of catecholamines, histamine, serotonin and kinins in response to hypoxia, and increased left ventricular afterload.^{2, 3, 6, 11, 12-14} The increased afterload increases the pressure in the left atrium and pulmonary capillary venous pressure, again favoring the formation of pulmonary edema.^{1, 11, 14} There can potentially be alveolar capillary membrane disruption (known as stress failure) resulting in further transudation into the interstitial space.^{1, 5-9, 11-13} NPPE causes impaired oxygen diffusion and ventilation-

perfusion mismatch.⁷ NPPE manifests as rapidly decreasing oxygen saturation, stridor, abnormal pulmonary chest x-ray with normal cardiac contour, and the hallmark finding of pink frothy sputum.^{1, 2, 5, 6, 11} This patient demonstrated crackles, wheeze and tachypnea in PACU. Other findings may include cyanosis, anxiety, or diaphoresis with the sympathetic stress stimulation.^{1, 2, 5, 6, 14} In extreme cases of NPPE frank pulmonary hemorrhage may occur.^{1, 3} With the onset of abnormal clinical findings we reviewed the differential diagnoses of pulmonary embolism, acute respiratory distress syndrome, intravascular fluid overload, cardiac dysfunction and NPPE.^{2, 5, 7}

The treatment of NPPE should include rapidly relieving the airway obstruction via a jaw thrust maneuver or inserting an oral or nasal airway and positive pressure ventilation via mask.^{7, 11} In this case jaw thrust and positive pressure ventilation maneuvers were sufficient to relieve the obstruction. In severe cases an artificial airway or endotracheal tube and the use of positive end-expiratory pressure of 5-10 cm H₂O may be necessary to assist in maintaining adequate oxygenation.^{1, 6-8, 11} Succinylcholine 0.1-0.2 mg/kg IV may be helpful in relieving the postoperative laryngospasm, or a dose of 1-2 mg/kg may be given to reintubate.^{5, 7, 14} Cool humidified oxygen via face tent and racemic epinephrine administered by nebulizer (0.5 ml of 2.25% solution diluted to a volume of 2.5 ml) was beneficial for this patient after the obstruction was relieved.¹⁴ Most literature regarding NPPE agrees that diuretics and steroids are not proven to be advantageous and should be employed with discretion depending on the patient's volume status.^{1, 2, 5, 6, 7, 14}

There are no specific techniques to prevent NPPE except care to prevent laryngospasm and laryngeal edema.⁶ The patient's oropharynx was thoroughly suctioned before extubation and positive pressure via face mask was administered after extubation. A topical laryngotracheal anesthesia of 2 ml lidocaine 1% and 2 ml Tetracaine 2% could have been used before intubation, or intravenous administration of lidocaine 2 mg/kg 1 minute prior to extubation has been recommended for adult patients.^{4, 6, 14} As recommended by literature, the patient was also noted to have a leak around the endotracheal tube at less than 15 to 20 cm H₂O.¹⁴ All patients at risk for upper airway obstruction should be extubated with an oral airway in place if necessary and with fully recovered airway reflexes.^{4, 11} The patient was fully awake for extubation so an oral airway was not placed. Surgery should be delayed if abnormal respiratory symptoms are present and the patient should be

observed in PACU for a judicious time if respiratory difficulties are noted.^{4, 11}

Although no abnormal respiratory symptoms were present preoperatively this patient was at risk for NPPE due to his gender, physique, and airway status. As with this patient, prompt and appropriate treatment should resolve NPPE within 24 to 48 hours.^{1, 6, 8} Retrospectively although the surgical procedure was uneventful, the patient had a more challenging airway than anticipated, compounded with a fairly recent upper respiratory infection. The goal for anesthesia should be to provide safe, quality care with attention to potential risks and complications. Due to his risk factors, the potential risk of NPPE should have been discussed with the patient's parents when obtaining anesthesia consent. This case study reminds anesthesia professionals of the small but very real risk of NPPE in certain populations of patients.

References

1. Thiagarajan RR, Laussen PR. Negative pressure pulmonary edema in children-pathogenesis and clinical management. *Paediatr Anaesth*. 2007;17:307-310.
2. Sharma S, Ashish J. Negative-pressure pulmonary oedema after septoplasty. *Internet J Anesthesiol*. 2008; 17(1). http://www.ispub.com/journal/the_internet_journal_of_anesthesiology/volume_17_number_1_1/article/negative_pressure_pulmonary_oedema_after_septoplasty.html. Accessed December 15, 2009.
3. Omar I, Moustafa Musa Syam, Takroui MS. Negative pressure hemoptysis due to airway obstruction on emergence from general anesthesia for arthroscopy. *Internet J Anesthesiol*. 2008; 17(2). http://www.ispub.com/journal/the_internet_journal_of_anesthesiology/volume_17_number_2_1/article/negative_pressure_hemoptysis_due_to_airway_obstruction_on_emergence_from_general_anesthesia_for_arthroscopy.html. Accessed December 15, 2009.
4. Kesimci E, Aslan B, Gumus T, Kanbak O. Management of unpredicted postoperative negative pressure pulmonary edema: a report of two cases. *Internet J Anesthesiol*. 2007; 12(1). http://www.ispub.com/journal/the_internet_journal_of_anesthesiology/volume_12_number_1_1/article/management_of_unpred_cted_postoperat_ve_negat_ve_pressure_pulmonary_edema_a_report_of_two_cases.html. Accessed December 15, 2009.
5. Davidson S, Guinn C, Gacharna D. Diagnosis and treatment of negative pressure pulmonary edema in a pediatric patient: a case report. *AANA J*. 2004; 72(5):337-338.

6. Lowery JE, Myers LL. Florid negative pressure pulmonary edema. *Internet J Otorhinolaryngol*. 2008; 7(2). http://www.ispub.com/journal/the_internet_journal_of_otorhinolaryngology/volume_7_number_2_6/article/florid_negative_pressure_pulmonary_edema.html. Accessed December 15, 2009.
7. Langenderfer B. A case of negative pressure pulmonary edema after breast implant surgery. *Internet J Pulm Med*. 2005; 5(2). http://www.ispub.com/journal/the_internet_journal_of_pulmonary_medicine/volume_5_number_2_26/article/a_case_of_negative_pressure_pulmonary_edema_after_breast_implant_surgery.html. Accessed December 15, 2009.
8. Ikeda H, Asato R, Chin K, et al. Negative-pressure pulmonary edema after resection of mediastinum thyroid goiter. *Acta Otolaryngol*. 2006; 126:886-888.
9. Vasudevan A, Mahesh N. Acute respiratory distress syndrome in a child with cerebral palsy. *Internet J Anesthesiol*. 2009; 20(2). http://www.ispub.com/journal/the_internet_journal_of_anesthesiology/volume_20_number_2/article/acute-respiratory-distress-syndrome-in-a-child-with-cerebral-palsy.html. Accessed December 15, 2009.
10. Hannania S, Barak M, Katz Y. Unilateral negative-pressure pulmonary edema in an infant during bronchoscopy. *Pediatrics*. 2004; 113(5):501-503.
11. Lathan SR, Silverman ME, Thomas BL, Waters WC. Postoperative pulmonary edema. *South Med J*. 1999; 92(3):313-315.
12. Devys JM, Cadi P, Nivoche Y. Protein concentration in pulmonary oedema fluid for negative pressure pulmonary oedema in children. *Paediatr Anaesth*. 1999; 10:557-558.
13. Taha S, Bartelmaos T, Kassas C, Khatib M, Baraka A. Complicated negative pressure pulmonary edema in a child with cerebral palsy. *Paediatr Anaesth*. 2002; 12:181-186.
14. Thomas CL, Palmer TJ, Shipley P. Negative pressure pulmonary edema after a tonsillectomy and adenoidectomy in a pediatric patient: case report and review. *AANA J*. 1999; 67(5):425-430.

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Anesthesia Considerations for Marfan's Syndrome

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Keywords: Marfan's syndrome, mitral regurgitation, aortic regurgitation, aortic aneurysm

Marfan's syndrome (MFS) is an inheritable disorder of the connective tissue.¹ Approximately 75% of individuals with MFS inherit the disorder and 25% are due to sporadic mutation in the fibrillin (FBN1)

gene, which encodes for fibrillin-1.¹ Fibrillin-1 is important in the structural support and elasticity of connective tissues.² Diagnosis of MFS is primarily based on physical examination focusing on the skeletal, ocular and cardiovascular system.³ It is difficult to diagnose MFS because signs of the disorder may vary. Anesthesia practitioners must recognize classic features

to prevent anesthetizing an undiagnosed MFS, which can lead to fatal cardiovascular and pulmonary complications.

Case Report

The patient was a 37 year-old male who presented with a chief complaint of shortness of breath for months and recent worsening of symptoms. The diagnoses upon admission were severe mitral regurgitation, moderate aortic regurgitation, congestive heart failure (CHF) and an incidental abdominal aortic aneurysm all related to a previously undiagnosed MFS.

Physical examination revealed a tall (188 cm) and thin (72 kg) patient with long slender extremities, fingers and toes. Cardiac exam revealed a systolic murmur III/VI heard at the apex with radiation to the axilla and marked jugular vein distention. The following drugs were administered upon admission: sodium nitroprusside (SNP) infusion, furosemide, benazapril, and metoprolol.

Laboratory data were unremarkable. Findings on chest X-ray revealed left atrial enlargement and pulmonary congestion. A transthoracic echocardiogram revealed mitral valve prolapse, severe mitral regurgitation and moderate aortic regurgitation. Cardiac catheterization also confirmed pulmonary hypertension and valvular dysfunction. A computed tomography of his chest, abdomen and pelvis revealed an aortic aneurysm with a 5.8 cm dilation and cardiomegaly with four-chamber enlargement. The patient was to undergo a mitral valve replacement in addition to a Bentall procedure, which includes replacement of the ascending aorta and aortic valve.³

The patient was preoxygenated and standard monitors placed. Midazolam 2 mg was given prior to arterial line insertion.

Induction was initiated with titration of ketamine 50 mg, propofol 100 mg, fentanyl 200 mcg, morphine 4 mg and cisatracurium 14 mg. The patient was ventilated with isoflurane 2% in O₂ 100% for three minutes followed by intubation of the trachea with an 8.0 mm endotracheal tube. A right internal jugular venous single lumen 9 french catheter was placed in addition to a pulmonary artery catheter. The patient had an uneventful bypass period and surgical replacement of the mitral and aortic valve and the ascending aorta was completed. Upon coming off bypass, dopamine and epinephrine drips were initiated for support of cardiac output.

Post bypass transfusions of packed red blood cells (3 units), fresh frozen plasma (4 units), and platelets (2 units) were done. The patient remained intubated and the following intravenous drips were continued upon transport to the cardiothoracic intensive care unit: aminocaproic acid, propofol, amiodarone, epinephrine, dopamine, nitroglycerin, and regular insulin. The patient was extubated on postoperative day two. After an uneventful hospitalization, the patient was discharged on postoperative day seven.

Discussion

The criteria to diagnose MFS in an individual with no family history are abnormalities in at least three major body systems classic of MFS, as in this patient.⁴ There is no specific laboratory test to diagnose MFS.⁵ Genetic analysis can be done for those with a family history to identify specific genetic mutation, but modern technology still cannot identify mutations in all genes that cause connective tissue disorders.⁴

This patient had cardinal features of MFS. He was disproportionately tall and had long, slender extremities, fingers and toes. Scoliosis and pectus excavatum or pectus carinatum of the sternum are common but was not present in this patient.⁴ He had a small jaw and a high palate, which did not affect airway management. Airway management in MFS patients is rarely difficult.⁵

Diagnostic tests, such as a chest X-ray should be done to evaluate for chest wall or spine abnormalities and for signs of CHF. Detection of tracheal deviation secondary to an aortic aneurysm can be evaluated. Further evaluation should include an echocardiogram to assess heart valves and aorta.¹ A CT scan of the chest, abdomen and pelvis should be done, as in this patient, because a chest X-ray may not show evidence of an aortic aneurysm. An aortic aneurysm grows insidiously in MFS and, if not detected preoperatively, puts the patient at risk for aortic dissection under general anesthesia.

The most serious complication associated with MFS involves the cardiovascular system. Eighty-seven percent of MFS patients die from cardiovascular abnormalities.³ A heart murmur or abnormal reading on electrocardiogram may warrant further investigation into cardiac function and status as did in our patient.⁶ Aortic dilation, dissection, or rupture and valve prolapse are caused from the lack of integrity of the connective tissue in the aorta and heart valves.³ In MFS, a dilated aortic root eventually leads to a distortion of the aortic valve resulting in aortic regurgitation (AR).^{1,3}

Mitral valve prolapse is the most prevalent valvular abnormality in MFS patients,

affecting 35-100% of MFS patients.¹ The pathophysiology of mitral and aortic regurgitation is volume overload of the LV.⁷ The body's circulatory compensation for these two disease processes are eccentric hypertrophy, peripheral vasodilatation and tachycardia to promote forward flow.⁷ These compensatory mechanisms explain why our patient remained asymptomatic for years.⁶ Afterload reduction with losartan, furosemide and SNP was initiated to treat heart failure and pulmonary congestion.⁸ Although factors, such as slow heart rate can increase regurgitation in mitral regurgitation (MR), beta-blocker therapy was cautiously started in this patient. Beta-blockers have negative inotropic effects to slow the growth of the aortic root and leads to decreased aortic regurgitation and dissection, congestive heart failure, and improved survival rates in patients with MFS.¹

Intraoperative goals were to maintain baseline blood pressure and heart rate and prevent sudden increases in myocardial contractility. In a patient with AR and MR, cardiac output is best when the heart is full and beating reasonably fast with the blood pressure that is low-normal.⁵ Placement of an arterial line before stimulating events can be helpful in the management of blood pressure. Blunting sympathetic responses to preoperative anxiety, direct laryngoscopy and surgical incision with anxiolytics and opioids should be considered to prevent increases in systemic vasoconstriction, which can worsen regurgitation and/or dilation of the aorta.^{1,7} Myocardial depressants, such as propofol, was carefully titrated. Morphine was used to mitigate further pulmonary congestion due to its vasodilatory effects. The use of ketamine was considered during induction for its hypnotic and analgesic effects, and its

ability to preserve sympathetic outflow. The pharmacodynamic effects of ketamine were used to override the potential myocardial depressant or bradycardic effects of other anesthetic medications when given concurrently. Furthermore, ketamine is efficacious in decreasing pulmonary artery pressures, which may have been beneficial to our patient.⁸

Mechanical ventilation must be carefully initiated with obstructive and restrictive lung volumes in mind. A MFS patient may not have chest wall abnormalities as in our patient, but attention was paid in avoiding excessive tidal volumes and airway pressures due to increase risk for pneumothorax from abnormal pulmonary collagen fibers.^{10, 11}

In conclusion, the management of this patient was complex in which considerations for MR, AR and aortic aneurysm were of utmost importance. The management plan implemented by the anesthesia team was directed at maintaining baseline blood pressure and heart rate and optimizing pulmonary artery pressures. Slow titration of induction agents was carefully done. The use of a balanced anesthetic technique is beneficial in that the pharmacologic effects of one drug can offset the side effects of another drug. Cardiovascular and pulmonary abnormalities are prevalent in patients with MFS and a thorough preoperative assessment is needed to prevent complications.

References

1. Milewicz, DM, Dietz, HC and Miller DC. Treatment of Aortic Disease in Patients with Marfan Syndrome. *Circulation*. 2005;111:150-157.
2. Dietz HC, Loeys B, Carta L, Ramirez F. Recent progress towards a molecular understanding of Marfan syndrome. *Am J Med Genet*. 2005;139(1): 4-9.
3. Pyeritz R. Marfan syndrome: 30 years of research equals 30 years of additional life expectancy. *Heart*. 2009;95:173-175.
4. De Paepe A, Devereux RB, Dietz HC, et al. Revised diagnostic criteria for the Marfan Syndrome. *Am J Med Genet*. 1996; 62:417-426.
5. Kamat S, Travasso B, Borkar D, et al. Anaesthetic considerations in a patient with Marfan's syndrome for maxillary corrective osteotomy. *Indian J Anaesth*. 2006;50(1):51-54.
6. Bonow RO, Carabello B, Chatterjee K, et al. ACC/AHA 2006 guidelines for the management of patients with valvular heart disease. *J Am Coll Cardiol*. 2006; 48:111-122; 142-149.
7. Hines, RL, Marschall, KE. *Stoelting's Anesthesia and Co-Existing Disease*. 5th ed. Philadelphia PA: Elsevier; 2008: 33-40.
8. Subramaniam K, Yared JP. Management of pulmonary hypertension in the operating room. *Semin Cardiothorac Vasc Anesth*. 2007; 11(2):119-36.
9. Varadarajan P, Joshi N, Appel D, et al. Effect of Beta-blocker therapy on survival in patients with severe mitral regurgitation and normal left ventricular ejection fraction. *Am J Cardiol*. 2008; 102(5):611-615.
10. Rigante D, Giuseppe S, Bush A. Persistent spontaneous pneumothorax in an adolescent with Marfan's Syndrome and pulmonary bullous dysplasia. *Respiration*. 2001;68:621-624.
11. Turner JA, Stanley NN. Fragile lung in the Marfan syndrome. *Thorax*. 1976;31:771-775.

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Subdural Placement of a Continuous Lumbar Epidural Catheter

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Keywords: subdural block, subarachnoid block, lumbar epidural, epidural complications, high spinal, total spinal

Lumbar epidural placement in a parturient is associated with numerous risks and although rare, these complications can be devastating to both the mother and the fetus. Subdural block is particularly uncommon and the incidence has been determined to approximate 0.1-0.82 percent.¹ Despite the low occurrence, the clinical presentation is variable and can be perplexing for the anesthetist. The diagnosis is frequently determined after the signs and symptoms occur.² Delay in diagnosis and appropriate interventions place the parturient and fetus at risk for permanent neurologic injury. The following discussion describes a case report in which a subdural block may have occurred despite lack of definitive confirmation.

Case Report

A 29 year old gravida 2 para 1 female presented in active labor at 39 weeks gestation. Her past medical history was significant for gastroesophageal reflux disease and obesity. The patient was 5 feet 3 inches and weighed 107.5 kg. She requested a labor epidural for pain relief. Informed consent was obtained following a physical assessment.

Prior to beginning the procedure, the parturient received 1000 ml bolus of 0.9% normal saline. Blood pressure (BP), heart rate (HR), oxygen saturation (SpO₂), and fetal heart rate (FHR) were continuously monitored.

The L3-4 interspace was identified and marked with the patient in the sitting position. The epidural space was located using a 17 gauge Touhy needle and loss of resistance (LOR) was determined by using saline. The author was unable to locate the space upon first attempt. The CRNA then attempted and was successful at acquiring LOR at 7 centimeters (cm). The epidural catheter was easily threaded and no parasthesias were noted. The catheter was pulled back to 13 cm at the skin and no CSF was aspirated. Three ml of Lidocaine 1.5% with epinephrine 1:200,000 was administered as a test dose. No signs of intravascular or subarachnoid injection were evident. The remaining 2 milliliters (ml) of the test solution was then administered. After securing the catheter in place, 6 ml of 0.25% bupivacaine was given. Upon assisting the patient back into bed, she reported a persistent sharp pain in her head. The catheter was withdrawn 1 centimeter. The parturient indicated the pain had subsided. A continuous infusion of 0.1% bupivacaine with 2 mcg/ml of fentanyl was then initiated at 12 ml/hour. Vital signs and fetal heart rate (FHR) remained stable throughout the procedure.

Approximately 50 minutes following epidural placement, the anesthesia personnel were notified that the patient had developed respiratory distress. The patient was breathing spontaneously though her respirations were rapid and shallow. Weakness in upper extremity motor strength was apparent and she was unable to move her lower extremities. The continuous infusion had been stopped prior to arrival of

anesthesia personnel. Oxygen was administered at 6 liters per minute via face mask. A T2 sensory block was evident upon assessment. BP decreased slightly from baseline to between 95-102 systolic over 54-55 diastolic. Two doses of ephedrine 5 mg were administered to maintain a systolic pressure above 100 mmHg. The FHR remained stable throughout the crises. The catheter was left in place. Close monitoring and supportive measures were continued. Six hours after the event, the block had completely regressed and the patient delivered a healthy baby girl.

Discussion

The subdural space has been referred to as a potential space existing between the dura and arachnoid layers extending from S2 to the cranium.^{3,4} However, recent evidence has revealed that it more accurately represents a dissection between the meningeal layers caused by trauma.² This explanation accounts for the variability in subdural block presentation. Common signs noted in the literature include negative CSF aspiration, negative signs and symptoms after a test dose, delayed onset of symptoms (>20 minutes), a high and unusual sensory block, motor sparing, and mild to moderate effects on blood pressure and heart rate.¹⁻⁶

Subdural placement of an epidural catheter can be radiographically confirmed. Contrast is injected through the catheter followed by computed tomographic (CT) scan to evaluate the distribution of the dye.⁵ A characteristic pattern of subdural placement involves cephalad spread with some root delineation.⁴ Despite this definitive diagnostic tool, it is not often utilized.

The current case presented with the two major diagnostic criteria associated with subdural block: a negative aspiration test

and unexpected widespread sensory blockade.² In a review of the literature, Hoftman and Ferrante found 89% (62 cases) of radiographically confirmed cases reported no CSF upon aspiration of the catheter.² Among those cases, 95% (59 cases) reported a negative test dose.²

The nature of the sensory and motor block itself is often inconsistent. Some case reports indicate an extensive sensory block extending to the upper extremities and face, while sparing sensory innervation to the sacral region.^{2,5,6} Sensory involvement, however, can vary between inadequate or patchy to extensive as described above. Hoftman and Ferrante found that the sensory spread to be excessive in 74% (45), patchy or asymmetrical in 33% (20), and restricted in 11% (7) of cases reviewed.² Motor blockade also differs among case reports. In some, motor blockade was not observed.^{2,5} The characteristic spread of local anesthetic in the subdural space explains this phenomenon. The posterior and lateral compartments of the space have a greater capacity to accommodate fluid, therefore local anesthetic is more likely to accumulate in these regions when injected.⁵ This pattern partially excludes the anterior roots of the spinal cord resulting in the observed motor and sympathetic sparing.⁵ In this case report, motor block to the lower extremities was profound, however sympathetic blockade seemed minimal as BP and HR remained relatively stable.

Another common characteristic of subdural injection includes delayed onset of symptoms.⁴ In this case report, symptoms of extensive sensory and motor block did not appear until almost 50 minutes following initial placement. This presentation clearly differentiates a subdural from a subarachnoid block. When local anesthetic is placed into the intrathecal space, sensory

and motor blockade is rapid and profound. In an article reporting 5 cases of intrathecal injection with a test dose, symptoms appeared within 1 to 3 minutes following injection.⁷ Furthermore, a significant decrease in BP (>20%) was noted with subarachnoid placement.⁷ In this case report, BP and HR remained stable even after the patient developed symptoms of high sensory blockade.

Subdural blocks often present as a high spinal. If not treated, permanent neurologic injury or even death may occur.⁶ Interventions are supportive in nature and advanced cardiac life support measures may be required. In this case report, the initial action was to stop the continuous local anesthetic infusion and to call for help. When anesthesia personnel arrived, the first assessment involved the patient's level of consciousness and her ability to maintain a patent airway. It was evident that the parturient was struggling to clear secretions because the high sensory blockade had weakened her diaphragm and intercostal muscles. The head of the bed was elevated to help support respirations and to allow gravity to assist in decreasing the level of blockade. Oxygen was applied despite normal oxygen saturation so as to maximize oxygen delivery to the fetus and mother.

This case report was not proven to be a subdural block with radiography. Other possible explanations included catheter migration into the subarachnoid space or slow leakage of local anesthetic through a dural tear made on the initial attempt to find the epidural space. Another possibility is conversion from a subdural to a subarachnoid block as a result of fluid accumulation leading to rupture through the dural membrane.

In summary, subdural block is a rare complication to epidural anesthesia, but it can have catastrophic consequences. In order to appropriately intervene, it is important to know the common signs and symptoms as well as the risk factors associated with this type of block. Difficult placement and prior spinal surgery have been shown to increase the risk of placing the catheter in the subdural space.⁵ In this case report, the patient experienced an unusual symptom of a persistent sharp pain in her head, a side effect not reported in the literature. If this were to occur in the future, it would be prudent to remove the catheter and attempt placement in a different interspace.

References

1. Lubenow T, Keh-Wong E, Kristof K, et al. Inadvertent subdural injection: A complication of an epidural block. *Anesth Analg* 1988;67:175-179.
2. Hoftman N, Ferrante F. Diagnosis of unintentional subdural anesthesia/analgesia: Analyzing radiographically proven cases to define the clinical entity and to develop a diagnostic algorithm. *Reg Anesth Pain Med*. 2009;34:12-16.
3. Polley LS, Glosten B. Epidural and spinal analgesia/anesthesia. In Chestnut DH, ed. *Obstetric Anesthesia*. 3rd ed. Philadelphia:Elsevier;2004, 324-348.
4. Jenkins JG. Some immediate serious complications of obstetric analgesia and anesthesia: A prospective study of 145 550 epidurals. *Int J Obstet Anesth*. 2005;14:37-42.
5. Chen SH, Chiueh HY, Hung CT, Tsai SC, Wong SY. Extensive sensory block caused by accidental subdural catheterization during epidural labor analgesia. *Chang Gung Med J*. 2006;29:607-611.

6. Forrester DJ, Mukherji SK, Mayer DC, Spielman FJ. Dilute infusion for labor, obscure subdural catheter, and life-threatening block at cesarean delivery. *Anesth Analg.* 1999;89:1267-1268.

7. Richardson MG, Lee AC, Wissler RN. High spinal anesthesia after epidural test dose administration in five obstetric patients. *Reg Anesth.* 1996;21:119-123.

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Myasthenia Gravis Remission and Neuromuscular Considerations

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Keywords: myasthenia gravis, remission, neuromuscular blockade, vecuronium, sevoflurane

This case highlights the implications of myasthenia gravis when developing an anesthetic plan. Additionally, this report discusses the successful use of neuromuscular antagonism in a seropositive patient with high levels of acetylcholine receptor antibodies that demonstrated no signs or symptoms of current disease and was considered to be in remission at the time of general anesthesia.

Case Report

A 46 year old Caucasian female (122 kg, 167.6 cm) presented for septoplasty with a chief complaint of difficulty breathing through her nose. The patient had a past surgical history of thymectomy. Her past medical history included rheumatoid arthritis, depression, asthma, and myasthenia gravis. The patient reported delayed emergence with her prior surgery. The patient was taking pyridostigmine 60 mg four times daily by mouth and had taken her morning dose. Her acetylcholine receptor antibody level was 0.9 nmol/L (expected 0 nmol/L) but the patient stated she was in remission. On physical exam the patient had stable vital signs and was afebrile. The

patient did not display notable muscle weakness and review of systems was normal.

A 20 gauge intravenous (IV) catheter was placed in the patient's right hand and lactated ringers solution was initiated. The patient was given midazolam 2 mg IV in the preoperative holding area prior to transport to the operating room (OR). Once in the OR the patient was connected to standard non-invasive monitors. The patient was able to move herself onto the operating table. Baseline vitals were obtained.

Oxygen was administered at 8 l/min via a face mask for 3 minutes. General anesthesia was induced with fentanyl 50 mcg, lidocaine 50 mg and propofol 180 mg IV. After loss of corneal reflexes, the patient was ventilated with oxygen and then given vecuronium 2 mg IV. After 2 minutes, laryngoscopy was performed with a MAC 3 under direct visualization. A size 7.0 endotracheal tube (ETT) was placed through the vocal cords. Once bilateral breath sounds and end tidal CO₂ (ETCO₂) were confirmed, sevoflurane was initiated at 2% in oxygen at 1 L/min. The patient was ventilated with synchronized intermittent mandatory ventilation (SMIV) at tidal volumes (TV) of 600ml, respiratory rate (RR) of 12, and positive end expiratory pressure of 2cm H₂O.

During the remainder of the case, the patient received fentanyl 50mcg IV, dexamethasone 4 mg IV, metoclopramide 10 mg IV, famotidine 20 mg IV, and ondansetron 4 mg IV.

Prior to discontinuing sevoflurane, neuromuscular function was assessed with train of four (TOF) peripheral nerve stimulation. Four out of four twitches were present without significant fade two hours after administration of the neuromuscular blocker. Neuromuscular antagonist agents were administered: neostigmine 3 mg IV and glycopyrrolate 0.4 mg IV. The ETT was removed when the patient demonstrated TV of 600 ml, a sustained head lift, and maintained adequate oxygenation levels without respiratory assistance.

The patient was transferred to the post anesthesia care unit with supplemental oxygen via a face mask and SpO₂ monitoring. After an uneventful two hour recovery, the patient was discharged to home by the attending anesthesia practitioner.

Discussion

Characterized by weakness and easy skeletal muscle fatigue, myasthenia gravis requires anesthesia practitioners to be knowledgeable and provide care with special consideration of the disease process.¹ The pathology of myasthenia gravis is caused by an autoimmune destruction of post synaptic acetylcholine receptors in the neuromuscular junction.¹ The current five treatment modalities for myasthenia gravis include: cholinesterase inhibitors (usually pyridostigmine), corticosteroids, immunosuppressive drugs, plasmapheresis and surgical thymectomy.²

With treatment, partial to complete

remission of the disease may be achieved.¹ In fact, 46% of patients who have undergone thymectomy develop complete remission and 50% become asymptomatic with improved treatment management.³ Even in well managed patients, it is recommended that those undergoing elective surgery wait until their condition is stable. Preferably the patient will not be on steroids at the time of surgery.²

When preparing for surgery and induction of anesthesia, pre-medication with benzodiazepines is often avoided because of potential respiratory depression but each patient should be evaluated independently. For practitioners seeking to avoid benzodiazepines, a suggested alternative is nitrous oxide-oxygen sedation, which has been reported to be safe and effective in reducing emotional preoperative stress.² Nitrous oxide administration, however, is not common or practical practice in pre-operative settings. The patient in this report complained of anxiety preoperative holding area. Showing no signs of respiratory compromise as evidenced by adequate oxygen saturations and ease of respirations, it was decided to administer midazolam 2mg IV. The patient was then transported to the operating room with ongoing practitioner assessment and observation.

It is also important to note that antibiotics may be a source of impaired neuromuscular transmission. Penicillin and its derivatives are suitable choices and do not produce neuromuscular relaxing properties.² Ampicillin is the exception which on a rare occasion has been reported to cause muscular relaxation.² Aminoglycosides are to be avoided because of known impairment of neuromuscular transmission and clinically significant weakness.² Antibiotics should be given in consultation with the neurologist. It was decided not to give the patient

antibiotics for this procedure.

Induction of anesthesia for patients with myasthenia gravis requires special consideration. Patients display extreme sensitivity to non-depolarizing neuromuscular antagonists (NdNMA) and may demonstrate varied responses to succinylcholine. These responses range from a normal effect to succinylcholine resistance, and if properly treated with pyridostigmine a prolonged effect.^{1,3,5,6} The degree of sensitivity to neuromuscular antagonism is variable even among patients in complete remission.³ Some myasthenia gravis patients with no signs or symptoms of disease will demonstrate sensitivity to neuromuscular antagonists which leads some resources to suggest avoiding neuromuscular blockade entirely.⁵ However, it has been reported that intermediate acting non-depolarizing neuromuscular antagonists such as atracurium and vecuronium are eliminated rapidly and can be used safely in reduced doses.⁴

This patient had an elevated acetylcholine receptor antibody level. Although antibodies are present in 85-90% of patients, a study by Itoh, Shibata, and Shunichi in 2002 found that presence and quantity of antibodies does not increase a patient's sensitivity to the vecuronium.^{4,7} In fact, no statistical difference was found between myasthenia gravis patients with antibodies and those without antibodies in their response to vecuronium.⁷ It is interesting to note that this patient did have antibodies. The available options for intermediate acting NdNMA were vecuronium or rocuronium. Vecuronium was chosen out of practitioner preference, a decision not specifically guided by research. The normal intubating dose of vecuronium for a patient weighing 122 kg would have been 9.76 mg to 12.2 mg. After induction, we chose to give 2 mg

IV vecuronium, a significantly reduced amount with the aim to facilitate intubation but avoid potential complications.

Maintenance of anesthesia also requires consideration of disease pathology. Inhaled agents produce neuromuscular depression.⁹ Investigation after the case, found that the neuromuscular depressant effects of sevoflurane, as used in this case, are seen in both control patients and patients with myasthenia gravis causing persistent neuromuscular fade at the end of anesthesia as measured by TOF.^{8,9} TOF fade was not observed at the end of this case, but recent research indicates that desflurane may have been a better choice.¹⁰ A study by Gritti et al published in 2009 evaluated the neuromuscular effects of desflurane in myasthenia gravis patients as compared to total intravenous anesthesia with propofol and remifentanyl.¹⁰ Both the TIVA group and the desflurane plus remifentanyl group obtained reversible muscle relaxation without clinical neuromuscular depressant implications, thus allowing for a fast recovery.¹⁰

Management of patients with myasthenia gravis can be challenging for anesthesia practitioners. This case demonstrates the safe administration of benzodiazepine to a well managed myasthenia gravis patient, and the safe use of vecuronium, which was dosed at approximately 20% of the standard intubating dose of 0.08 to 0.1 mg/kg. This case also indicates the usefulness of sevoflurane for anesthetic maintenance but research suggests that desflurane and remifentanyl or TIVA with propofol and remifentanyl are perhaps more appropriate choices for anesthetic maintenance. Ultimately, there are many factors to consider when caring for a patient with myasthenia gravis; many have been reviewed in this case report.

References

1. Morgan GE, Mikhail M, Murray M. *Clinical Anesthesiology*. 4th ed. San Francisco, CA: McGraw-Hill; 2006:818-819.
2. Jamal BT, Herb K. Perioperative management of patients with myasthenia gravis: prevention, recognition, and treatment. *Surg Oral Med Oral Pathol Oral Radiol Endod*. 2009;107: 612-615.
3. Barash PG, Cullen SF, Stoeltin RK. *Clinical Anesthesia*. 6th ed. Philadelphia, PA: Lippincott William & Wilkins; 2009: 626-627.
4. Baraka A. Anaesthesia and myasthenia gravis. *Can J Anesth*. 1992; 39(5): 476-486.
5. Hurford WE. *Clinical Anesthesia Procedure of the Massachusetts General Hospital*. 6th ed. Philadelphia, PA: Lippincott William & Wilkins; 2002: 186-187.
6. Tripathi M, Kaushik S, Dubey P. The effect of use of pyridostigmine and requirement of vecuronium in patients with myasthenia gravis. *J Postgrad Med*. 2003;49: 311-315.
7. Itoh H, Shibata K, Nitta S. Sensitivity to vecuronium in seropositive and seronegative patients with myasthenia gravis. *Anesth Analg*. 2002; 95(1): 109-113.
8. Kwak YL, Choi YS, Shim JK, Choi EM. Persistent train-of-four in myasthenia gravis patients after sevoflurane anesthesia. *Br J Anaesth*. 2008; 100(5):724-730.
9. Nitahara K, Sugi Y, Higa K, Shono S, Hamada T. Neuromuscular effects of sevoflurane in myasthenia gravis patients. *Br J Anaesth*. 2007;98(3):337-341.
10. Gritti P, Carrara B, Khotcholava M, Bortolotti G, Giardini D, Lanterna LA, Benigni A, Sonzogni V. The use of desflurane or propofol in combination with remifentanyl in myasthenic patients undergoing a video-assisted thoracoscopic-extended thymectomy. *Acta Anaesthesiologica Scandinavica*. 2009; 53(3): 380-389.

Mentor: Tracy Lanes, CRNA, MSN

Undiagnosed Subglottic Laryngotracheal Stenosis

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Keywords: Tracheal stenosis, oxygenation, ventilation, complications, airway obstruction

Tracheal obstruction may be due to trauma, infection, tumor, aspirated foreign bodies, or intubation. Even with high volume, low pressure endotracheal tubes (ETT) used today, endotracheal intubation is the most common cause of acquired tracheal stenosis.^{1,2} Nearly 10% of intubated critically-ill patients will develop tracheal

stenosis.³ Despite the possibility of symptoms presenting over time, these symptoms can quickly progress to a life-threatening situation. Symptoms of tracheal stenosis can range from mild respiratory wheezing upon exertion to severe stridor and respiratory distress. The variety of causes and severity of symptoms make this disease difficult to manage.

Case Report

A 32-year-old Hispanic woman, 157.5 cm and 73.9 kg, presented for an elective ileostomy takedown. Her past medical history included a diagnosis of stage 3 ovarian cancer fifteen months prior. At that time she underwent an exploratory laparotomy with bilateral salpingo-oophorectomy and an ileocectomy with adjuvant chemotherapy. Her postoperative course was complicated by a bowel perforation requiring multiple exploratory laparotomies and abdominal wash outs and a consequent loop ileostomy. She remained intubated for 14 days in the intensive care unit (ICU) and after being transferred to the Medical-Surgical ward for 6 days, was discharged home.

Physical examination for the current surgical procedure revealed a Mallampati class II airway and full range in neck extension. Lung sounds were clear bilaterally. Her preoperative chest radiograph was normal with a midline trachea and the patient appeared comfortable. The previous anesthesia records were not available for airway management review.

In the operating room, standard monitors were placed and the patient was preoxygenated for five minutes. An intravenous induction was initiated and mask ventilation was easily performed. A GlideScope (Verathon Inc, Bothell, Washington) was used during intubation for clinical practice. Upon the initial intubation attempt, the ETT met mild resistance; however, a forty-five degree clockwise turn of the ETT allowed for smooth insertion past the vocal cords. Bilateral breath sounds were assessed as equal and clear. The ETT was secured at 21 cm at the lip.

During surgical preparation, 52 minutes after induction, an acute increase in peak inspiratory pressures from 22 cm H₂O to 40 cm H₂O and a subsequent increase in end-tidal CO₂ from 34 mm Hg to > 50 mmHg was noted. Manual ventilations became difficult and a flexible bronchoscope was inserted through the ETT. Tracheal stenosis was suspected based on visualization of a narrowed segment of the trachea. The 7.0-mm ETT was removed and a 6.0-mm ETT was placed under direct visualization by the attending anesthesiologist. Passage of the 6.0-mm ETT was met with moderate resistance but confirmation of end-tidal CO₂ and bilateral breath sounds were assessed. Manual ventilations with an oxygen inspired concentration of 100% maintained an SpO₂>95%. An intraoperative consultation was requested with the ear, nose, and throat (ENT) service, who confirmed an 85% airway occlusion via a flexible bronchoscope below the vocal cords. The decision was made, at that point, to perform a tracheostomy and cancel the original surgical procedure. Serial tracheal dilations from a 4.0-mm ETT to a Shiley (Covidien-Nellcor, Inc. Boulder, Colorado) 6.0-mm tracheostomy tube was accomplished.

The patient stayed in the ICU overnight and was discharged home 7 days after her tracheostomy was placed. During the post-operative evaluation the patient's mother explained that the patient had been experiencing shortness of breath, while doing simple chores, over the past 8 months. Her mother stated that the patient did not disclose her symptoms fearing this would cause a delay in her surgical procedure. At her follow-up ENT visit the decision was made for an endoscopic laser treatment and serial dilations with a concurrent ileostomy takedown.

Discussion

This case emphasizes the need for a high level of suspicion for tracheal stenosis in patients who present with a history of prolonged intubation. Complications, as illustrated in this case, may become apparent intraoperatively when they are more difficult to manage. The patient's only known predictor of tracheal stenosis was her prior history of intubation. In addition, no evidence of tracheal stenosis was identified during physical assessment and the chest radiograph did not illustrate any narrowing of the trachea. The patient's denial of symptoms significantly impacted the outcome of her elective surgery.

The time it takes for symptoms of tracheal stenosis to appear can vary. In a retrospective study, Sarper et al. stated for patients with tracheal stenosis after intubation, the average duration of intubation was 10.9 days and the average onset of symptoms post-extubation was 32.3 days.⁴ Sajal and Sarmishtha published a case report where a patient developed a dry cough and audible wheeze three months post-extubation after being intubated for four days.⁵ Furthermore, in a retrospective study, Andrews and Pearson found the length of time between extubation and recognition of stenosis varied between one day and two years.⁶ These symptoms ranged from mild symptoms of shortness of breath during rest, with no relief after bronchodilator treatment, to acute respiratory distress presenting in the emergency room. Once symptoms of tracheal stenosis appear, they can often be misdiagnosed as asthma and tracheal stenosis is not diagnosed at initial presentation in as many as 44% of patients.⁶

Endotracheal cuff pressure exceeding the mucosal capillary perfusion pressure is

thought to be the trigger for tracheal stenosis.⁷ The increased pressure leads to mucosal ischemia causing ulcerations of the tracheal cartilages. The lesions heal by fibrosis, leading to a progressive tracheal narrowing. A history of progressive dyspnea and wheeze unresponsive to bronchodilators, coupled with a high index of suspicion in patients who have a history of prolonged tracheal intubation, are the most important indicators of tracheal stenosis.⁷ Unfortunately, the patient never reported her shortness of breath experienced during mild exertion such as when doing chores around the house. Furthermore, her symptoms disappeared with rest.

The technique most commonly used to definitively diagnose tracheal stenosis is flexible bronchoscopy, which allows for a determination of the location and severity of the stenotic lesion. Chest radiographs are usually normal and do not help as a diagnostic tool.⁸ In this case, the patient, indeed, presented with a normal chest radiograph. When her symptoms of tracheal stenosis occurred intraoperatively, the definitive diagnosis and the severity of tracheal stenosis were determined using the flexible bronchoscope. The stenosis began 2.4 cm below the level of the true vocal folds at the C7 level and was measured 2.1 cm in length.

The treatment modalities for tracheal stenosis include serial tracheal dilations, laser dissection, and tracheal resection. The choice of treatment modality is based on severity and recurrence of tracheal stenosis. Current practice suggests a trial of tracheal dilation before proceeding to major reconstruction.⁹ In this case, the patient was scheduled for endoscopic laser treatment and serial dilations.

Undiagnosed tracheal stenosis can place a patient in a potentially life-threatening situation. The literature suggests the diagnosis of tracheal stenosis can be elusive, as the symptoms can take as long as 2 years to develop. A patient who denies symptoms of tracheal stenosis can mislead the anesthesia professional, who may subsequently overlook this life-threatening situation. In this case report, the patient denied any shortness of breath or respiratory distress. With clear bilateral breath sounds and the absence of stridor, there was no suspicion of tracheal stenosis. Symptoms of tracheal stenosis should be thoroughly evaluated during the preoperative physical examination. The potential for life-threatening events during surgery should be emphasized to the patient, especially when the patient presents with a history of prolonged intubation.

References

1. Pena J, Cicero R, Marin J, et al. Laryngotracheal reconstruction in subglottic stenosis: an ancient problem still present. *Otolaryngol Head Neck Surg* 2001;125:397-400.
2. Zietek E, Matyja G, Kawczynski M. Stenosis of the larynx and trachea: diagnostics and treatment. *Otolaryngol Pol* 2001;55:515-520.
3. Stauffer JL, Olson DE, Petty TL. Complications and consequences of endotracheal intubation and tracheotomy. A prospective study of 150 critically ill adult patients. *Am J Med*. 1981;70:65-76.
4. Sarper A, Ayten A, Eser I, Ozbudak O, Demircan A. Tracheal stenosis after tracheostomy or intubation. *Tex Heart Inst J*. 2005;32:154-158.
5. Sajal D, Sarmishtha D. Post intubation tracheal stenosis. *Indian J Crit Care Med*. 2008;12:194-197.
6. Andrews M, Pearson F. An analysis of 59 cases of tracheal stenosis following tracheostomy with cuffed tube and assisted ventilation, with special reference to diagnosis and treatment. *Br J Surg*. 1973;60(3):208-212.
7. Spittle N, McCluskey A. Tracheal stenosis after intubation. *BMJ* 2000;321(21):1000-1002.
8. Tzouveleakis A, Kouliatsis G, Oikonomou A, et al. Post-intubation pulmonary embolism and tracheal stenosis: a case report and review of the literature. *Resp Med*. 2008;102:1208-1212.
9. Herrington HC, Weber SM, Anderson PE. Modern management of laryngotracheal stenosis. *Laryngoscope*. 2006;116:1553-1557.

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Dexmedetomidine for Awake Carotid Endarterectomy

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Keywords: carotid endarterectomy, dexmedetomidine, regional anesthesia

Carotid endarterectomy (CEA) is an established surgical procedure performed to

decrease the incidence of stroke in patients with atherosclerotic carotid artery disease.¹ Carotid endarterectomy performed under a regional technique offers the advantage of continuous neurological monitoring, the

most sensitive method for detecting insufficient cerebral perfusion.²

Dexmedetomidine (DEX) may be a useful adjunct to provide sedation for a regional anesthetic. Dexmedetomidine is a highly selective and potent alpha 2-adrenergic agonist that decreases opioid and inhalational agent requirements, decreases plasma catecholamine concentrations, and only mildly depresses ventilation while allowing for continuous neurologic assessment.³

Case Report

A 52 year old, 145kg, 72 inch male with carotid atherosclerotic disease presented for a left CEA. The patient had experienced a transient ischemic attack, manifested by right arm weakness and numbness which resolved in less than 24 hours. A computer tomography angiogram revealed greater than 60% stenosis of the left carotid artery. His medical history was also significant for CPAP-dependent obstructive sleep apnea, morbid obesity (BMI 45), moderately controlled hypertension (blood pressure range: 125/74-157/82), hyperlipidemia, and gastroesophageal reflux disease. His current medication profile included: lisinopril, simvastatin, clopidogrel, aspirin, and omeprazole. Past surgical history included an appendectomy, tonsillectomy, and bilateral knee arthroscopies without any anesthetic complications. His functional status was evaluated as greater than 4 metabolic equivalents. His electrocardiogram showed sinus bradycardia without ST segment or T-wave abnormalities. An adenosine stress test revealed normal myocardial perfusion, and an echocardiogram demonstrated an ejection fraction greater than 60% without valvular or other structural abnormalities. His coagulation studies, electrolytes and liver

function studies were all within normal limits.

A regional anesthetic with a DEX infusion for sedation was planned for the procedure. In the holding area, the patient received midazolam 2 mg intravenously (IV). Standard monitors were applied upon arrival to the operating room, and a 20 gauge arterial line was inserted in his right radial artery. Oxygen was delivered via face mask with end tidal CO₂ monitoring. A deep and superficial cervical plexus block was performed on the lateral portion of the left neck, with 0.5% ropivacaine and epinephrine 1:400,000, 35 ml total. Midazolam 1 mg and fentanyl 50 mcg were titrated IV during administration of the block. A loading dose of DEX 0.7mcg/kg over 10 minutes was initiated approximately 5 minutes after block insertion, followed by a continuous infusion of 0.2 mcg/kg/hr. A negative Allis test verified adequate anesthesia.

Additional medications administered intraoperatively included metoprolol 4 mg, nitroglycerine 100 mcg, labetalol 10 mg, heparin 10,000 units prior to clamping of the carotid artery, and protamine 12.5 mg IV. Fentanyl was titrated in 25 mcg aliquots for perioperative analgesia and midazolam was titrated for anxiety in 1 mg aliquots. The patient remained easily arousable and hemodynamically stable throughout the procedure. The patient's level of consciousness was assessed for speech deficit, contralateral arm strength, confusion, and agitation during clamping of the internal carotid artery. The surgeon also inserted a carotid shunt. Operative time was 3 hours and 50 minutes and total doses of sedative medications included fentanyl 250 mcg, midazolam 6 mg and dexmedetomidine 7 mg. Upon conclusion of surgery, the patient was alert, orientated, and hemodynamically stable.

Discussion

Carotid endarterectomy is a common vascular surgery utilized to decrease the risk of stroke in susceptible patients. The most significant perioperative complications of CEA are myocardial infarction and stroke, with symptomatic patients having a higher incidence of stroke compared to asymptomatic patients.^{4,5} When providing anesthesia for this procedure, anesthesia practitioners must ensure hemodynamic stability to protect the heart and brain from ischemia, and titrate medications in order to render an awake patient at the end of surgery to evaluate neurological function.²

Anesthesia for CEA may be provided via a general, regional, or local anesthetic technique. Most studies comparing these anesthetic techniques have indicated no differences in morbidity or mortality.^{2,6} A general anesthetic offers the benefits of a quiet surgical field and medication administration to decrease cerebral metabolic demand, but fails to provide a consistent, reliable monitor for cerebral ischemia.⁷ The greatest advantage of a regional or local anesthetic technique is an awake patient for direct neurological monitoring, but disadvantages may include patient discomfort, loss of cooperation, poor access to the airway, local anesthetic toxicity, and potential conversion to a general anesthetic.² A regional technique was implemented for this patient due to surgeon preference, lack of language barriers, and the benefits of continuous neurologic assessment in the symptomatic patient. The greatest anesthetic concern in utilizing a regional technique for this patient was providing sufficient sedation without compromising airway patency or neurological monitoring, as morbidly obese patients with history of obstructive sleep apnea may demonstrate increased sensitivity

to sedative medications and be more difficult to intubate.^{8,9}

Dexmedetomidine (DEX) is a highly selective, sedative, α_2 -adrenergic agonist, with an α_2 : α_1 adrenoceptor ratio of 1600:1.^{10,11} α_2 agonist effects are mediated via a G-coupled protein mechanism and produce sedation by inhibitory action in the locus ceruleus of the brain stem.¹¹ Additional sites of action for α_2 agonists are the spinal cord (analgesia), heart (bradycardia), and peripheral vasculature (vasodilation and vasoconstriction).¹¹ The sympatholytic action of DEX results from activating a negative feedback loop, with inhibition of endogenous norepinephrine release.¹² Caution should be utilized when administering DEX to patients with ventricular dysfunction or advanced heart blocks, as the drug has been associated with severe bradycardia and cardiac arrest when infused as an adjunct with general anesthesia.³ An exaggerated hypotensive response may also be observed in patients with hypovolemia, diabetes mellitus and chronic hypertension.¹⁰

Dexmedetomidine may be an attractive alternative to the primary use of opioids for titratable sedation during CEA when used in conjunction with a regional anesthetic technique. DEX only mildly depresses respiratory ventilation, an appealing attribute when delivering sedation to a morbidly obese patient.³ We decreased the DEX loading dose to 0.7mcg/kg from the recommended 1 mcg/kg over 10 minutes and also maintained the DEX continuous infusion on the lower end of the recommended rate at 0.2mcg/kg/hr (recommended maintenance dose ranges from 0.2 to 1 mcg/kg/hr).¹⁰ We decreased the dosage because of the potential for an exaggerated hypotensive response due the

patients NPO status and history of chronic hypertension; as well as the possibility of a compromised airway due to the patient's body habitus and probable increased sensitivity to sedative medications due to OSA.

A cornerstone in the anesthetic management of patients undergoing a CEA is to optimize hemodynamics to prevent brain ischemia. Utilizing DEX for sedation raises the concern that patients with compromised cerebral circulation may suffer ischemia, as DEX can decrease cerebral blood flow without decreasing cerebral metabolic rate. This may potentially result in an increased need for an intracarotid shunt during cross clamping. Bekker et al. found that DEX administration is associated with less perioperative hypertension and tachycardia and does not increase the need for intra-arterial shunting compared with opioid based anesthetics for CEA.¹³ This result may be due to reduced sympathetic tone and lower catecholamine levels while maintaining adequate sedation.¹³ In a small randomized trial, McCutcheon et al. compared a conventional sedation technique with midazolam/fentanyl (STD) versus DEX in terms of hemodynamic control, adverse cardiac and neurological events and patient satisfaction. Researchers concluded there were no statistical differences between groups requiring treatment for hypotension and bradycardia, number of interventions required per patient, or the need for intraarterial shunting.¹⁴ The number of patients requiring postoperative analgesia was significantly lower in the DEX group in the PACU, as opposed to an increase in analgesic requirements for the STD group. A postoperative questionnaire revealed that patients may remember intraoperative discomfort because of the lack of amnesic effects with small concentrations of DEX.¹⁴

Our experience with DEX as an adjunct to regional anesthesia was promising. Throughout the procedure, the patient remained hemodynamically stable and neurologically intact. The outcome of this case supports the integration of DEX as an adjunct in combination with a regional anesthetic for use in awake CEA.

References

1. Luchetti M, Canell M, Zoppi M, Massei R. Comparison of regional anesthesia versus combined regional and general anesthesia for elective carotid endarterectomy: a small exploratory study. *Reg Anesth Pain Med.* 2008;33:340-345.
2. Miller R, Eriksson L, Fleisher L, Wiener-Kronish J. *Miller's Anesthesia.* 7th ed. Philadelphia: Churchill Livingstone; 2010:2026-2028.
3. Stoelting RK, Hillier SG. *Pharmacology & Physiology in Anesthetic Practice.* 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2006:344.
4. Bevilacqua S, Romagnoli S, Ciappi F, et al. Anesthesia for carotid endarterectomy: the third option. Patient cooperation during general anesthesia. *Anesth Analg.* 2009;108:1929-1936.
5. Reuter NP, Charette SD, Sticca RP. Cerebral protection during carotid endarterectomy. *Am J Surg.* 2004;188:772-777.
6. Yastrebov K. Intraoperative management: carotid endarterectomies. *Anesthesiol Clin North America.* 2004;22:265-287.
7. Guay J. Regional anesthesia for carotid surgery. *Curr Opin Anaesthesiol.* 2008;21:638-644.
8. Passannante AN, Rock P. Anesthetic management of patients with obesity and sleep

- apnea. *Anesthesiol Clin North America*. 2005;23:479-491.
9. Benumof JL. Obstructive sleep apnea in the adult obese patient: implications for airway management. *J Clin Anesth*. 2001;13:144-156.
 10. Precedex (dexmedetomidine) [package insert]. Lake Forest, IL: Hospira; October 2008.
 11. Kamibayashi T, Maze M, Weiskopf RB. Clinical uses of alpha₂-adrenergic agonists. *Anesthesiology*. 2000;93(5):1345-1349.
 12. Brady T. Anesthetic management of a pituitary tumor resection with dexmedetomidine. *AANA J*. 2010;78(2):125-127.
 13. Bekker A, Gold M, Ahmed R, et al. Dexmedetomidine does not increase the incidence of intracarotid shunting in patients undergoing awake carotid endarterectomy. *Survey of Anesthesiology*. 2007;51:179-180.
 14. McCutcheon, C, Orme, R, Scott, D, Davies, M, McGlade, D. A comparison of Dexmedetomidine versus conventional therapy for sedation and hemodynamic control during carotid endarterectomy performed under regional anesthesia. *Anesth Analg*. 2006;102:668-675.

Mentor: MAJ Denise McFarland, CRNA, MSN

Editorial

I'm pleased to put forth an issue full of interesting case reports for your review this summer. We have over a dozen reports from 9 Nurse Anesthesia Programs. In addition, we have several new section editors and reviewers, some new and some who had been involved in the past that I am pleased to welcome back into the fold:

Section Editors

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Thank you so much for answering my call for help with the ISJNA! This does not mean we don't have opportunities for those of you who still want to get involved. Please contact me if you are interested in serving as a reviewer, or would like to take the next step and become a section editor!



Vicki C. Coopmans, CRNA, PhD
Editor