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Topics in This Issue

Organ Procurement
Congenital Anomalies
Lumbar Plexus Blockade
Craniotomy
Antithrombin III Deficiency
Dexmedetomidine in Peds
Delirium Tremens
Kippel-Weber Syndrome
Marfan Syndrome
Insulinoma
Obstructive Sleep Apnea
Multiple Sclerosis
Glucose Control
Uncuffed ETTs



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Front Cover: Danielle Rawson, BSN, SRNA, University of Pennsylvania, places an epidural for a radical cystectomy.

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Anesthesia Management for Organ Procurement from a Brain-Dead Donor
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Keywords: Organ procurement, Brain-Dead, Organ harvest, Organ donor, organ transplant

Currently, there are over 95,000 candidates on the waiting list for organ transplantation. Unfortunately, there is a shortage between the demand for organ transplants and supply of available organs.¹ In the United States, in order to meet the demands of organ transplantation, solid organs such as the heart, liver, pancreas, kidney, and bowel are recovered mostly from brain-dead organ donors with the assistance of state and federal law established voluntary programs.²

Despite the unnecessary need for sedation and analgesia for brain-dead patients, the role of anesthesia professionals is still essential during organ retrieval from these donors.³ The focus of anesthesia care for these patients changes from patient preservation to organ preservation.³

Case Report

A 37 year old male was brought into the emergency room complaining of left sided weakness and loss of motor sensation. The patient weighed 95 kg and was 70 inches tall. His medical history only included bipolar disorder for which he took fluoxetine. His prior surgical history included a cholecystectomy without any anesthesia complications. A CT scan revealed a large temporal and parietal lobe subarachnoid hemorrhage. In the emergency room, the patient was subsequently intubated due to mental status changes and irregular breathing. The patient underwent emergency right sided craniotomy and evacuation of hematoma. The patient was

found to have an intracerebral hematoma. He remained intubated with a poor postoperative prognosis. The patient developed subfalcial herniation with early uncal herniation. The patient's pupils were fixed and dilated. Electroencephalogram revealed an isoelectric waveform. An apnea test was also conducted. The patient was found to have absent brain stem reflex. Diagnosis of brain death was confirmed and the patient was pronounced dead. An organ procurement organization was called to evaluate the patient for the possibility of organ donation.

A left subclavian central venous (CVP) catheter was in place along with a left radial arterial line. The patient was started on a levothyroxine infusion at 20 mEq/hr titrated for a systolic blood pressure (SBP) of > 100 mmHg with a maximum dose of 40 mEq/hr. An infusion of 0.45% saline solution at 200 ml/hr was also initiated. The patient also had a dopamine and phenylephrine infusion to maintain SBP greater 100 mmHg. The patient was started on cefazolin and cefepime intravenous antibiotics. A vasopressin 1 unit IV bolus was given. The patient was continued on mechanical ventilation with the following settings: VT 800, AC 12, FiO₂ 100%, and PEEP of 10 cmH₂O.

The patient's family consented to organ donation and the transplant, surgical, and anesthesia teams were notified regarding the organ retrieval. Prior to the harvest laboratory tests revealed the following values: hemoglobin 12.9 g/dL, hematocrit 39%, platelet count 165,000/cc, white blood cell count 17,200 cells/cc, Na⁺ 141 mEq/L, K⁺ 4.0 mEq/L, Cl⁻ 110 mEq/L, CO₂ 19

mEq/L, BUN 9 g/dL, and Cr 0.8 mg/dL, PT 15.1 secs, INR 1.2, and PTT 32 secs. Arterial blood gas showed a pH 7.31, PaCO₂ 33 mmHg, PaO₂ 83, HCO₃ 17 mEq/L, and BE -9 mEq/L. Preoperative vital signs were: BP 116/69, HR 112, 95% O₂Sat on 100% FiO₂.

Multiple 0.45% saline solutions and 5% albumin were obtained in preparation for the organ procurement, along with mannitol 60 gms, furosemide 100 mg, and heparin 50,000 units.

Upon arrival to the operating room, mechanical ventilation was continued with the same settings. Routine monitoring was continued along with CVP monitoring. The anesthesia team was instructed by the transplant team to keep the SBP > 100 mmHg and the CVP between 5 to 10 mmHg. The patient was continued on 100% O₂. Pancuronium 10 mg IV was given. The dopamine infusion was continued at 10 mcg/kg/min and the levothyroxine infusion continued at 40 mEq/hr. The phenylephrine infusion was also continued and titrated to keep the SBP > 100 mmHg. CVP on arrival to the operating was between 13 to 15 mmHg. The patient's SaO₂ ranged from 95 to 100%. The transplant team was constantly informed regarding the patient's blood pressure and CVP values.

Declines in systolic blood pressure below 100 mmHg were treated with 100 – 200 mcg IV boluses of phenylephrine and upward titration of the infusion. Intraoperative heart rate was between 110 to 120 beats/min. Aggressive infusion of warm 0.45% saline solution was accomplished using pressure bags. Body temperature was kept above 37° C. Almost an hour into the surgery, the patient's CVP was 17-18 mmHg, at which time furosemide 40 mg IV was given intravenously and the CVP dropped to 13 –

15 mmHg. Blood loss during the first hour was approximately 700 ml. and urine output was between 800 to 1000 ml every 30 minutes.

Heparin 30,000 units IV was administered prior to the cross clamping of the aorta. Anesthesia care ended with the occlusion of the proximal aorta. All monitoring services and mechanical ventilation were discontinued. The heart valves, eyes, and vascular organs were retrieved from the patient.

Discussion

Anesthetic requirement of the brain dead organ donor entails the management of the physiological events resulting from brain death. This involves maintenance of optimal organ perfusion, cellular oxygenation, and fluid, electrolyte and acid-base balance.³ Overall the perioperative management goals follow the “rule of 100s:” a systolic blood pressure of greater than 100 mmHg, urine output of greater than 100 ml per hour, PaO₂ of greater than 100 mmHg, and a hemoglobin level of greater than 100 g/L.³

Hemodynamic management of brain dead donor involves the treatment of hypotension and possible arrhythmias. Loss of descending vasomotor control gives rise to vasodilation, blunted vasomotor reflexes and impaired cardiac contractility. The resulting hypotension is further exacerbated by traumatic blood loss, diuresis from diabetes insipidus, prior fluid restriction for cerebral edema, and diuretic therapy.⁴ Attaining cardiac stability is essential to optimize organ preservation and eventually better operative graft function.⁴

Therapeutic management of hypotension involves the maintenance of intravascular volume and colloid oncotic pressure, and a

hemoglobin concentration of above 10 g/dL.⁴ Either colloid or crystalloids can be used to restore intravascular volume. Hypotonic solutions such as 0.45% saline should be used to reduce the incidence of hypernatremia in donors.⁴ A CVP of 6 to 10 mmHg should be maintained by the administration of blood components, crystalloids and albumin. To prevent the incidence of renal tubular injury and impaired postoperative renal graft, the use of hydroxyethyl starch for volume expansion is avoided.⁵ After adequate volume resuscitation is achieved, vasoactive drugs are infused to improve cardiac contractility and vascular resistance.⁴ Dopamine at a less than 10 mcg/kg/min has been the primary vasopressor of choice in managing hemodynamic instability. Donors requiring doses greater than 10 mcg/kg/min suggests the need for additional vasoactive agents. Unlike dopamine, dobutamine, and epinephrine, the use of norepinephrine has been associated with an increased incidence of graft failure.⁴ The use of phenylephrine can diminish splanchnic perfusion and jeopardize abdominal organ viability. If necessary vasopressin 0.5 to 1.5 U/hr can be administered.²

Transfusion management should be aimed at providing adequate oxygen delivery by maintaining a hematocrit of greater than 30% percent through PRBC infusion.⁶ Pressure bag infusers should be readily available for the transfusion of fluids and blood products.³ Traumatized and necrotic tissue can lead to coagulation disorders with the release of thromboplastin, cerebral gangliosides, and plasminogen rich substrate.⁵ A profound state of coagulopathy can develop from these factors when combined with ongoing hemorrhage, transfusion, hypothermia, acidosis, and dilution of coagulation factors. Coagulopathies (indicated by an

International Normalized Ratio (INR) greater than 2.0 or a platelet count less than 80,000/cc) must be corrected and may necessitate the transfusion of clotting factors such as fresh frozen plasma and or platelets.⁵

The management of respiratory function is essential to improve the quality of the organs to be donated. Respiratory complications occurring during brain death include injury to the lung, neurogenic pulmonary edema, and aspiration pneumonitis.⁵ Recommendations for the management of respiratory function of brain dead donors include the following ventilatory strategies: (1) maintaining a PaO₂ of greater than 100 mmHg with low FiO₂ and PEEP of 5 cmH₂O (2) use of VT of 10-12 ml/kg and minute ventilation that maintains a PaCO₂ of 30-35 mmHg, a pH of 7.35 to 7.45 and peak airway pressure of less than 30 mmHg.⁷

Endocrine changes also accompany brain death. Vasopressin, thyroid hormones, corticosteroids, and insulin are administered to manage these endocrine changes, to improve cardiac function and to improve graft survival.⁴ Diabetes insipidus commonly occurs when pituitary function is affected. This condition is accompanied by massive polyuria, hypovolemia, hyperosmolarity, hypernatremia, hypokalemia, and other electrolyte abnormalities.⁴ Diabetes insipidus is confirmed by the presence of hypoosmolar urine (300 mOsm/l), serum hyperosmolarity (310 mOsm/l), and hypernatremia (serum sodium > 150 mEq/l).² Part of the management of organ donors is to restore serum electrolyte values and osmolarities, and replace free water losses. Vasopressin (titrated from 2 µg/kg/min) or desmopressin (titrated from 0.3 µg/kg/min) can be used to maintain a urine output of 1.5 to 3 ml/kg/h.⁶

In addition, hormone replacement therapy can also be initiated for hemodynamic stabilization prior to organ procurement. It has been suggested that after brain stem ischemia, there is a reduction in plasma free triiodothyronine (T3), which is believed to be responsible for impairment of myocardial cell metabolism and myocardial contractility. However, the use of hormone replacement therapy still remains controversial.⁸

As mentioned earlier, urine output of 1.5 to 3 ml/kg/h or greater than 100 ml/hr should be maintained. However, oliguria can be present reflecting hypovolemia and hypotension. Persistent oliguria despite a normovolemic state may reflect renal dysfunction resulting from earlier episodes of renal ischemia. Depending on the recommendation of the transplant team, furosemide or low dose dopamine can be used to treat oliguria.⁴

Loss of hypothalamic regulation and an inability to shiver or vasoconstrict predisposes the donor to hypothermia. Hypothermia can lead to further cardiac dysfunction, arrhythmias, coagulopathy, a leftward shift of the oxyhemoglobin dissociation curve, and cold-induced diuresis.⁵ Aggressive methods should be initiated to maintain a core temperature greater than 35° C. Replacement fluids should be warmed, inhaled gases humidified, and warming blankets used.⁶

Reflex neuromuscular activity, ranging from muscle twitching to complex limb and trunk movement can be expected since spinal cord reflexes may be present.³ A long-acting muscle relaxant, such as pancuronium, should be given at the beginning of the procedure to facilitate surgical extraction and eliminate reflex neuromuscular activity.⁶ Anesthesia care ends with

proximal aortic cross clamping. At this point, all monitoring and supportive therapy are discontinued.⁶

Brain-dead organ donors entail a wide array of physiological derangements. The management of these donors for organ procurement presents a significant challenge to anesthesia practitioners, with the clinical focus shifting from patient preservation to organ preservation.⁴ It is important for anesthesia practitioners to have a good understanding of the pathophysiological changes that occur with brain death. However, understanding these changes is not enough; communication between the surgical and transplant team and the anesthesia practitioner is important to optimize donor care and ultimately prevent graft dysfunction.

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Pediatric Anesthesia for Congenital Anomalies

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Keywords: anesthesia, management, pediatric, congenital, anomalies

Health issues associated with birth defects account for 30% of all pediatric hospital admissions nationwide.¹ Recent advances in healthcare and medical technology have dramatically increased survival rates of patients with congenital anomalies and previously nonviable degrees of prematurity.² As a result, more children and adults with a history of prematurity and multiple congenital anomalies are presenting for surgery and requiring anesthesia. Providing anesthesia for these patients requires an understanding of how these defects affect the patient's physiology and response to anesthetics. A thorough preoperative examination is essential to developing an appropriate anesthetic plan.

Case Report

A 28-inch tall, 8 Kg, 18-month-old female with recurrent otitis, esotropia, and amblyopia presented to the operating room for bilateral tympanotomy with tubes and 2 muscle strabismus repair. Extensive past medical history included prematurity (23 and 5/7 weeks) with resuscitation at birth

(chest compressions, positive pressure ventilation, and epinephrine), oral aversion, necrotizing enterocolitis, gastroesophageal reflux disease (GERD), right middle cerebral artery infarct, multiple intracerebral hemorrhages with resultant encephalomalacia, cerebral palsy (CP), osteopenia, and respiratory distress syndrome. Past surgical history included repair of a perforated bowel and patent ductus arteriosus. The patient's medication regimen included albuterol, budesonide suspension, and lansoprazole suspension. A recent echocardiogram revealed a patent foramen ovale (PFO), small left to right atrial shunt, mild tricuspid insufficiency, and increased pulmonary artery pressures. A recent swallowing study also noted evidence of silent aspiration.

An extensive interview with the parents did not reveal any new problems. Preoperative vital signs were blood pressure 82/54, heart rate 105, and room air oxygen saturation 97%. Physical exam findings were consistent with the patient's comorbidities. The patient's lung sounds were clear and skin color was normal. A limited airway exam did not predict difficulties. The patient was calm in the holding room and no

premedication was given. She was carried to the operating room in warm blankets and placed on a warmed operating room table. Monitors including 3 lead ECG, blood pressure cuff, pulse oximetry, precordial stethoscope, and peripheral nerve stimulator were placed on the patient. A smooth inhalation induction was performed with 8 liters per minute of oxygen and 8% sevoflurane. The surgeon inserted the tympanostomy tubes after an adequate depth of anesthesia was achieved. An IV was started and extreme care was taken to avoid intravenous injection of air. After successfully obtaining IV access, rocuronium was given to facilitate intubation. The patient was intubated with a 3-millimeter internal diameter, cuffed endotracheal tube under direct vision using a Miller 1 blade. Bilateral breath sounds and end tidal carbon dioxide were confirmed and mechanical ventilation was initiated. Fentanyl, rocuronium, and sevoflurane were used for anesthetic maintenance. The case proceeded uneventfully. After completion of the procedure neuromuscular blockade was antagonized with neostigmine and atropine. When the patient was able to maintain spontaneous ventilation with adequate tidal volumes, demonstrated an oxygen saturation of at least 95 %, and began swallowing and reaching for the endotracheal tube, the endotracheal tube was removed. She was taken to the recovery room in the lateral decubitus position with blow-by oxygen. Her PACU course was uneventful. The patient remained in the hospital overnight, and was discharged home the next day with no apparent anesthetic or procedure-related complications.

Discussion

Pediatric patients presenting for eye surgery are prone to other congenital problems that may impact anesthetic management.² In this

case, the extensive past medical history and anesthetic records were reviewed. Prioritizing each of the patient's problems and considering them in context created the final anesthetic plan.

Patients with strabismus have an increased risk of malignant hyperthermia (MH).² However, this patient had undergone a previous anesthetic in which MH triggers were used without sequela. After careful consideration, the decision was made not to avoid MH triggers in this case.

The patient also presented with a PFO. This condition is often subclinical, and is actually present in approximately 25 percent of individuals on autopsy.³ However, a PFO becomes increasingly relevant during the perioperative period because these patients have an increased risk of hypoxemia and embolism.³ The level of increased risk is dependent on the size of the defect and the nature of the procedure.³ Increases in pulmonary vascular resistance and right to left shunt can allow deoxygenated blood, thrombi or air to enter the systemic circulation.³

Strenuous events which are likely to occur with pediatric patients, including coughing and crying, can both contribute to the degree of right to left shunt. As a result, avoiding anxiety, coughing and crying are important components of the anesthetic plan. Oral premedication was available to attenuate anxiety and crying during the preoperative separation period. However, it is likely that this would have been difficult to administer due to oral aversion and was not necessary since the patient remained calm preoperatively. During intubation and maintenance, neuromuscular blockade was employed to avoid coughing and straining due to the endotracheal tube. Furthermore, the decision was made to extubate the

patient as soon as extubation parameters were met, before significant coughing occurred.

This patient also had CP, a collection of motor disorders with various manifestations resulting from neurological injury occurring before 2 years of age.⁴ This is pertinent to the anesthetic because these children may have GERD, increased secretions, decreased ability to cough, seizures, decreased ability to regulate temperature, and altered responses to muscle relaxants.^{4,5} Several associated findings were present in this patient. A rapid sequence induction could have been considered because of the history of GERD and aspiration. However, this would have required the insertion of an IV catheter before induction and would have made avoiding anxiety and crying more difficult.

Neuromuscular blockade should be carefully considered with CP patients. Children with CP are at an increased risk of hyperkalemia with succinylcholine administration due to extrajunctional acetylcholine receptors.^{4,5} They are also more sensitive to succinylcholine, requiring a decreased dose.^{4,5} On the other hand, CP patients tend to be less sensitive to non-depolarizing neuromuscular blockers.^{4,5} As a result there is a decreased duration of action and an increased dose may be required. During this case, the standard recommended dose of non-depolarizing muscle relaxants was administered based on clinical effect.

Various aspects of this patient's comorbidities were taken into consideration when developing the anesthetic plan. As a

result of continued advances in neonatal care, it is likely that patients will continue to present for surgery and anesthesia with an ever-increasing number and severity of comorbid conditions. Safe, comprehensive anesthesia care for these complicated patients must take into consideration the effects of their various co-morbidities and the risk and benefits of available options.

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Placement of a Continuous Lumbar Plexus Block

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Keywords: Regional anesthesia, Lumbar plexus, Continuous, total hip arthroplasty

Continuous lumbar plexus blockade (CLPB) is an effective method for intraoperative anesthetic requirement reduction and post operative pain management.¹⁻⁶ The most common use of CLPB is for surgery of the hip and knee. The lumbar plexus originates from the anterior rami of the first to the fourth lumbar vertebrae. The plexus then descends caudally and anteriorly between the quadratus lumborum and the psoas major muscles bilaterally. The lumbar plexus contains lateral femoral cutaneous, femoral and obturator nerves. These nerves innervate the anterior extensors and medial adductors of the thigh and hip. The posterior psoas approach to the lumbar plexus provides reliable anatomical landmarks, easy patient positioning and reliable nerve stimulation that is easy to recognize.⁴ This case report details the successful application and management of a continuous lumbar plexus block with an On-Q® elastomeric infusion pump for post operative pain management after total hip arthroplasty.

Case Report

A 77kg, 47 year old female patient presented for total hip arthroplasty to the pre-anesthesia testing clinic. The patient was found to be an American Society of Anesthesiologist (ASA) class two. The patient had a significant chronic pain and degenerative joint disease history. All other physical assessments and laboratory data were within normal limits. A general anesthetic with endotracheal intubation

combined with a CLPB was planned. An On-Q® infusion pump for postoperative pain management was recommended by the anesthesia practitioner, chosen by the patient and planned in collaboration with the surgeon.

On the morning of surgery the patient was interviewed, assessed and taken to the preoperative holding area where standard American Association of Nurse Anesthetists (AANA) monitors were placed. Oxygen via nasal cannula was applied, the patient was placed in the right lateral decubitus position and 2.0 mg IV midazolam was administered. The iliac crest, posterior superior iliac spine, midline of the fourth and fifth lumbar vertebrae, and fourth lumbar transverse process were identified and clearly indicated with a skin marking pen.

The patient was verbally informed and reassured during each step in CLPB placement. The skin was prepped and draped in standard sterile fashion with betadine and a 1.0% lidocaine skin wheal with deeper Stimuplex needle path localization was performed. A 4 inch Stimuplex 17gauge Touhy needle was used to engage the lumbar plexus, with the stimulation level set at 1.5 mA.

At 4 cm depth the fourth transverse process was encountered. The needle path was directed slightly cephalad to track superiorly beyond the transverse process. At approximately 6 cm needle depth, a distinct quadriceps twitch with patellar tendon snap was elicited. The needle stimulation level was reduced to 0.36mA at which point the

twitch was lost. After negative blood aspiration and no patient complaints of paresthesia, 30ml of 0.25% bupivacaine with 1:200,000 epinephrine was administered in 5 ml aliquots with negative aspiration at each interval.

After expansion of the nerve sheath with local anesthetic, a continuous catheter was inserted through the Touhy needle into the lumbar plexus to a depth of 5 cm past the tip of the needle. The catheter produced a negative blood aspiration. The catheter was secured and clearly identified at the insertion site of the dressing and the injection port as a peripheral nerve catheter using premanufactured stickers supplied in the CLPB kit. All patient vital signs were stable throughout CLPB placement and the patient reported being relaxed and comfortable. Significant vein engorgement of the left lower extremity was recognized five minutes after block placement. Sympathectomy indicated the onset of the CLPB and gave a qualitative indicator of proper placement of the local anesthetic.

The patient was then taken to the operating room and AANA standard monitors were placed. The patient was induced by standard sequence with a combination of lidocaine 80 mg, fentanyl 125 mcg, propofol 150 mg and rocuronium 25 mg. A 7.5 mm cuffed endotracheal tube was placed by direct laryngeal visualization without difficulty. The patient was maintained with desflurane at 3.5-4.5% and positioned laterally for the procedure. Skin incision was made 27 minutes after the CLPB local anesthetic was administered. The patient had no significant change in vital signs with surgical stimulation. The patient required only 100 mcg of supplemental fentanyl during the procedure.

At the conclusion of the surgical procedure the patient was extubated and taken to the PACU. The patient reported no pain upon arrival to the PACU. A thorough neurological, sensory and motor exam was performed 15 minutes after arrival in the PACU. The patient was able to slightly bend the left knee, wiggle toes, plantar and dorsi flex the feet bilaterally. The patient was turned laterally by the PACU team and the dressing and insertion site of the CLPB catheter was reassessed. The On-Q® pump infusion was inspected for proper filling by the pharmacy and an infusion of 0.2% ropivacaine at 8 ml per hour was initiated after negative aspiration from the catheter.

The patient was interviewed and assessed by the anesthesia team at 6, 11, 19, 26, 35 and 46 hours after the continuous infusion was started. The patient's highest pain score was three out of ten, at 25 hours postoperatively. The patient reported being "very satisfied" with the CLPB and the On-Q® pump infusion device. Upon removal of the catheter at 46 hours post operatively, the insertion site was found to be absent of redness, swelling or any indication of reaction at the skin surface. The entire catheter was removed intact and inspected for damage and shearing. The surgical team assumed responsibility for pain management of the patient at this point. The patient was discharged 24 hours later. A follow up phone interview at five days post operatively found the patient still "very satisfied" with the CLPB, the On-Q® pump administration device and the gradual transition from the CLPB to oral pain medication 46 hours after surgery.

Discussion

The CLPB combined with an On-Q® pump for postoperative pain management is an excellent anesthetic tool for total hip

arthroplasty procedures. This case study illustrates the optimal anesthetic goals being obtained by the patient, surgical and anesthetic team. Specifically for this case, the intraoperative anesthetic requirement for the procedure was significantly reduced, the postoperative course for the patient was unremarkable and the patient reported being very satisfied with her anesthetic experience.

However, the CLPB technique is not without potential complications. For example, inadvertent total spinal anesthetic has been reported from a posterior psoas approach to the lumbar plexus with a continuous catheter.¹ In addition, Sciatic nerve palsy after CLPB and total hip arthroplasty has also been reported.² In each case rapid recognition and collaboration of the anesthetic and surgical team averted potential deleterious consequences.^{1,2} These cases illustrate significant potential complications to the use of CLPB for surgical and postoperative pain management.^{1,2} However, in a large analysis of continuous peripheral nerve blocks the most common significant event associated with CLPB was a 6 % rate of vascular puncture during placement.³ This indicates that aspiration of the needle during block placement may be the best way to avoid the most common adverse complication associated with CLPB placement.³

A significant insight for CLPB placement was found by Capedevilla and colleagues. While the distance between the skin and the transverse process may significantly differ among patients, the distance from the transverse process to the lumbar plexus is consistently 18 millimeters.⁴ When attempting to engage the lumbar plexus in a patient with difficult landmarks, this key piece of information provides a consistent needle depth marker for troubleshooting difficult block placement.⁴

A recent large meta-analysis found that CLPB was significantly superior to oral or intravenous opioids for postoperative pain management.⁵ The rate of nausea, vomiting, pruritis and sedation was significantly increased with oral or intravenous opiates.⁵ Moreover, the use of continuous peripheral nerve block provided better postoperative analgesia.⁵ Another meta-analysis also found that the potential clinical benefits of regional anesthesia include decreased nausea, vomiting, postoperative pain and decreased postanesthesia care unit use.⁶

The onset and duration profile of local anesthetics is not the only important factors to consider for a particular peripheral nerve block. Recent research into the effects of local anesthetics on muscle tissues may drive a paradigm shift related to local anesthetic choice. A study of the myotoxic effects of bupivacaine and ropivacaine in pigs discovered that both drugs induce significant skeletal muscle damage, but the negative histological changes of bupivacaine were consistently and significantly more pronounced.⁷ Another recent study on the myotoxic effects of bupivacaine, levobupivacaine and ropivacaine in rat psoas muscle confirmed that bupivacaine is consistently and significantly more myotoxic than ropivacaine.⁸ Currently, the trend of using bupivacaine in place of ropivacaine seems to be related to practitioner familiarity and a significant cost savings with bupivacaine. This emerging data may dictate a paradigm shift for anesthesia practitioners and the choice of local anesthetic for peripheral nerve blocks.

This case report illustrates the optimal use of CLPB techniques for total hip arthroplasty procedures. Current research provides ample evidence for the superiority of peripheral nerve blockade for surgical and postoperative pain management.¹⁻⁶ When

considering an anesthetic plan for a particular patient and surgical procedure it is important for anesthesia practitioners to contrast the potential risk and benefit profile of each option with the needs of the patient and the requests of the surgical team. This knowledge allows providers to intelligently individualize the anesthetic plan for each patient.

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Utilization of Hypothermia During Emergency Craniotomy

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Keywords: Hypothermia, Craniotomy, Traumatic brain injury

Intraoperative maintenance of temperature is a standard of care. Techniques to sustain normothermia such as increasing the ambient room temperature, the use of forced air warming blankets, and warmed intravenous fluids are some of the measures taken by anesthesia practitioners to ensure

normothermia. Under certain circumstances such as craniotomy or coronary artery bypass grafting, hypothermia can be utilized to promote a favorable clinical outcome.¹

Case Report

A 35 year old, 55 kilogram female was found on the side of a road with lacerations to the face, hands and knees. Emergency

medical technicians noted hemorrhaging in the nasal area, airway, and auricles. It was uncertain how long the woman was exposed to the ambient temperature of -7 degrees Celsius (°C), or what had happened to her. Neurological status revealed equal, sluggish pupils to light; the patient became agitated and combative when aroused by painful stimuli. Initial Glasgow Coma Scale (GCS) of 8-9 was recorded by the emergency medical support team. Her cervical spine was immobilized and the trachea was intubated with a #7.5 millimeter endotracheal tube (ETT). Vital signs upon arrival to the emergency department: rectal temperature of 30.06°C, electrocardiogram revealed a heart rate (HR) of 39 beats per minute (bpm) in a sinus rhythm with notable premature ventricular contractions, shallow respirations 6 breaths per minute and blood pressure (BP) 91/45 mm/Hg. Notable laboratory data revealed: serum potassium 2.7 mEq/L, serum carbon dioxide 20 mEq/L, serum calcium 7.8 mg/dl, serum glucose 217 mg/dl, serum ethanol 0.166%, and urine toxicology screen positive for cannabis.

Following initial treatment in the field and stabilization at a small hospital, the patient was airlifted to a level one trauma center. Computed axial tomography (CAT) showed a large left epidural hematoma with a middle meningeal artery tear. There was a 3.3 mm shift from the midline, the ventricles were not dilated. There was also a complex, left-sided basal skull fracture involving the sphenoid, temporal, and occipital bones. There was no evidence of acute abdominal, pelvic, or cervical trauma on CT. No past medical or surgical history was available.

Upon arrival to the operating room (OR) the patient was arousable to stimuli with stable vital signs and a core temperature of 34.4°C. Endotracheal tube placement was confirmed and then connected to the anesthesia

machine; the patient was induced with thiopental 300 mg. General anesthesia was maintained with isoflurane end tidal concentration of 1.1% and 2 liters/minute of oxygen. Two 18 gauge peripheral lines were placed along with a radial arterial line. An esophageal temperature probe was inserted and the rectal temperature probe previously placed was maintained for comparison.

It was discussed and agreed upon by the surgeon, anesthesiologist, nurse anesthetist and student nurse anesthetist to keep the patient at the core temperature at which she presented with to the OR, 34.4°C. The patient had earlier demonstrated some of the side effects of hypothermia (bradycardia, arrhythmias, hypotension, and increased glucose) and gradual re-warming measures began to stabilize these side effects, the team decided to stop re-warming and utilize the hypothermia during the procedure for cerebral protection. Warming blankets and fluid warmers were in place if needed, however the case progressed without the use of these devices.

Isoflurane, midazolam, fentanyl, rocuronium, mannitol, and oxygen were utilized for the case. Core temperature was sustained between 33.5-34.5°C and all vital signs remained stable as the procedure progressed unremarkably. The patient received 1200cc normal saline, blood loss was calculated to be 150cc and urine output was 850cc during a surgical time of 98 minutes.

The patient was transported to the intensive care unit (ICU) uneventfully. Five hours post operatively the patient remained ventilated, intracranial pressure (ICP) tracings were 3-6 mmHg, HR 78 bpm, BP 135/57 mmHg, cerebral perfusion pressure 77-80 mmHg, and temperature 36.4°C. GCS score of 9/15 due to the ETT in place. The

patient was able to follow commands appropriately with all extremities. On postoperative day one the arterial line and ICP monitoring was discontinued, the Glasgow Coma Scale was 10+ out of 15 with the ETT in place. Vital signs and labs were stable, temperature was 36.7°C, and the patient was being weaned off the ventilator.

Discussion

The hypothalamus utilizes a negative feedback mechanism to regulate a core body temperature of 37°C. Hypothermia is defined as an abnormally low body temperature of 36°C where systemic heat loss is greater than heat production². This can be achieved via four mechanisms: radiation is the loss of heat from warm surface such as the body to a cooler environment which accounts for the majority of heat loss; convection, which is the outcome of a temperature gradient between body and ambient air; conductive heat loss, which occurs when a warm body comes in contact with a cold operating room table; and evaporative loss, which is related to respiratory passage, opened wound exposure, and perspiration.³

Physiological side effects related to hypothermia are well known and potentially fatal. As a patient's temperature decreases, the sympathetic nervous system is stimulated and this causes heart rate, stroke volume, peripheral vascular resistance, and myocardial oxygen demand to increase initially. As hypothermia progresses, the sympathetic response diminishes. Around 34°C ventricular arrhythmias increase and around 32°C bradycardia ensues. This can lead to spontaneous ventricular fibrillation. Central nervous system membrane conduction and chemical processes slow resulting in electroencephalographic wave forms to slow at 35°C; below 32°C a coma

results. Coagulation and the clotting cascade depend on a normothermic environment for proper enzyme function; at abnormally low temperatures thrombin becomes inhibited, platelets become sequestered in the liver and functioning circulating platelets decrease. Initially respirations become stimulated; however, around 34°C respiratory rate and response to PaCO₂ decreases. Respiratory drive will cease around 24°C. Drug metabolism and elimination both become prolonged due to decreased renal blood flow resulting in diminished enzyme activity of the liver and a lower glomerular filtration rate.³

Although hypothermia does present the anesthesia practitioner with major challenges, there are known benefits to hypothermia. There is a reduced basal and metabolic rate, improved myocardial protection and tissue and organ preservation. There is also an overall reduction in the cerebral metabolic rate of oxygen consumption (CMRO₂), an approximate 7% decrease for each 1 degree decrease in temperature. Finally, the minimum alveolar concentration decreases.⁴

Thirty-three degrees Celsius has been considered the lower limit for the head injured patient.⁵ Multiple sources acknowledge the benefits of hypothermia; however, there are different thoughts about achieving hypothermia. For instance, two small clinical trials of 82 and 87 patients respectively, showed induced hypothermia, moderate 32-33°C to mild 33-35°C, in patients with a GCS of 8 or less, had improved neurological outcomes.^{6,7} However, in a recent multi-center trial of 392, patients with traumatic brain injury (TBI) who presented hypothermic and were not re-warmed had overall better outcomes than patients who presented with TBI and hypothermia was induced.¹ This patient

presented to the OR hypothermic and since the side effects of the hypothermia were recognized prior to the arrival to the OR and treatment mitigated the effects, the team decided to utilize the hypothermia during the case and follow cautiously.

This case was presented to review the side effects of hypothermia, discuss the possible benefits of hypothermia, and remind the anesthesia practitioner to be aware of options during an intense setting, such as emergency surgery for the TBI patient.

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Antithrombin III Deficiency during Cardiac Surgery

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Keywords: Antithrombin III, coagulopathy, cardiopulmonary bypass, thrombophilia, thrombosis

Cardiac and pulmonary surgical procedures that utilize cardiopulmonary bypass (CPB) present a need for the anesthesia practitioner to administer systemic anticoagulants prior to initiation of CPB. Anticoagulation is often accomplished with the administration of heparin, using weight-based dosing guidelines. However, patients may present with coagulation disorders that can make the

goal of systemic anticoagulation problematic.

Case Report

A 38-year-old African-American male with a medical history of mitral valve regurgitation secondary to streptococcal endocarditis presented for a mechanical mitral valve replacement. The patient weighed 87 kilograms and was approximately 183 centimeters in height. He reported a penicillin allergy and denied any

current medication usage, history of prior surgeries, or history of hemorrhagic diathesis or abnormal bruising. Preoperative vital signs, electrolytes, and coagulation profile were unremarkable. The patient had a preoperative hemoglobin of 10.4 g/dl and platelet count of 286,000/mm³.

Anesthesia was induced intravenously with etomidate and fentanyl, and neuromuscular blockade was achieved with pancuronium. An endotracheal tube was placed uneventfully. Anesthesia was maintained with desflurane in an oxygen-air mixture with intravenous aliquots of pancuronium to maintain neuromuscular blockade at train-of-four 0/4. The patient underwent placement of a 8.0 french introducer and pulmonary artery (PA) catheter with technical difficulty. Attempts at placing the catheters by the SRNA, CRNA, two anesthesiologists, and the cardiac surgeon via the patient's right internal jugular were unsuccessful. Ultimately, the PA catheter was successfully placed via the patient's left subclavian vein. A right radial arterial line was placed without complication. A transesophageal echocardiography (TEE) probe was placed atraumatically and baseline mitral valve regurgitation was documented in the presence of normal left ventricular functioning.

Prior to surgical incision, an initial dose of two million kallikrein inhibitor units (KIU) of aprotinin was administered after a 10,000 KIU test dose. Later, a continuous infusion of aprotinin at 500,000 KIU/hr was initiated to minimize intraoperative blood loss. A baseline activated clotting time (ACT) was found to be 128 seconds (Normal value: 100-130 seconds).¹ After surgical incision and in preparation for CPB, an initial heparin dose of 30,000 units was administered. A subsequent ACT was found to be 413 seconds and an additional 10,000

units of heparin were administered prior to initiation of CPB. Cardiopulmonary bypass was initiated and the next ACT was found to have decreased to 387 seconds. Four units of fresh frozen plasma (FFP) were administered, and subsequent ACTs ranged between 450-600 seconds. No additional heparin was administered. CPB was terminated uneventfully after nearly two hours. Utilizing fixed dosing, the patient received a total of 350 mg of protamine sulfate without complication. The aprotinin infusion was stopped ten minutes prior to completion of skin closure for a total infusion of two million KIU. Normal valve and ventricular function was observed on the TEE at the conclusion of the procedure. The patient received a total of two units of packed red blood cells and 750 mL of blood was returned to the patient via autologous blood salvage. Minimal bleeding was observed from the incision at the conclusion of the procedure.

The patient was transferred to the cardiac intensive care unit and remained intubated, sedated, and with a train-of-four of 0/4 after a 5 mg bolus of pancuronium prior to transport. A mediastinal chest tube was in place to water seal with minimal serosanguinous drainage noted. Vital signs remained within normal limits upon report to the intensive-care unit nurse.

Discussion

Antithrombin III (AT) deficiency is a rare genetic abnormality,² representing approximately one to two percent of congenital coagulopathies.³ This deficiency occurs more commonly as a sequela of hepatic dysfunction or prolonged heparin administration,² and has been associated with an increased incidence of venous thrombosis.⁴ The main concern with AT deficiency in regards to cardiac surgery

requiring CPB is an increased heparin requirement and difficulty achieving therapeutic levels of intraoperative anticoagulation (heparin resistance) as seen in this case study. It is fairly common for patients who have been on prolonged preoperative heparin infusions to have significantly elevated heparin requirements secondary to a consumptive coagulopathy.⁵ These elevated intraoperative heparin requirements lead to increased protamine requirements for heparin reversal as well as an increased incidence of postoperative bleeding.⁶ The patient in this case study did not receive heparin preoperatively, which eliminated heparin as a precipitating factor for his anticoagulation resistance. An alternative explanation for the alteration in AT activity in this patient may be related to hepatic dysfunction. Since AT is primarily synthesized in the liver, patients with hepatic dysfunction are at increased risk for alteration in AT activity. Baseline hepatic function of this patient was unknown and therefore could not be eliminated as a cause of this patient's increased intraoperative heparin requirements and likely AT deficiency.

Activated clotting time (ACT) is a test that is widely utilized to monitor coagulation during CPB. It has been noted that visible clot formation is uncommon with values of 300 seconds. Current clinical standards recommend ACT values of greater than 480 seconds during CPB.⁷ While the ACT is an effective and accurate test for monitoring whole blood coagulation, it is not without limitations. Activated clotting time values obtained from different machines may produce statistically significant variations, and even sequential testing done on the same machine may produce some variability.^{7,8} Another concern with ACT monitoring is the common misconception that ACT values reflect systemic heparin

concentration. While ACT values do often correlate with heparin dosing, they may be significantly affected by a number of other variables. An ACT tests for whole blood coagulation, and factors such as hemodilution, hypothermia, and other agents such as aprotinin may alter ACT values.⁷ Altered ACT values seen with deep hypothermia during CPB actually reflect significant decreases in AT activity.⁹ This patient's hematocrit values remained close to baseline levels of 26 percent, and the patient maintained a core temperature of 36.8 prior to CPB, eliminating these as potential causes. In the face of normal hematocrit values such as in this patient, administration of FFP is a common therapeutic choice. However, some literature suggests FFP be given only after administration of 600 units/kg of Heparin.¹⁰ This patient received approximately 460 units/kg of Heparin prior to transfusion of FFP. Commercial AT preparations are also a viable choice and because they are heat treated they provide a lower incidence of viral transmission.⁵ AT preparations are typically given as a bolus of 1000 units (approximately equivalent to 2-4 units of FFP),¹¹ which can increase a patient's AT activity anywhere from 10-67 percent.¹² However, the expense of these preparations can be prohibitive and limits their use clinically.

When resistance to heparin is encountered intraoperatively during or preceding CPB, options must be considered beyond simply administering larger doses of heparin. In the case of consumptive coagulopathies like AT deficiency, the treatment is often replenishment of missing factors through administration of whole blood, FFP, or other specific preparations. However, in some cases such as heparin-induced thrombocytopenia, other anticoagulation options besides heparin must be considered

for surgeries requiring CPB. Other antithrombin agents like argatroban,¹³ lepirudin, and bivalirudin¹⁴ have been used successfully for anticoagulation during CPB. Non-CPB techniques for cardiac surgeries also remain an option as anticoagulation may not be required. Early cardiac surgery was done without the benefit of CPB as a matter of necessity prior to the invention of the heart-lung machine in the 1950s. Procedures such as coronary revascularization done without CPB have seen resurgence in recent years. While utilizing newer cardiac stabilizing devices, off pump cardiac procedures have been shown in some studies to have lower short-term morbidity. The long-term benefits of these procedures remain a subject of debate.¹⁵ Valve replacement surgery requires CPB and anticoagulation, and discussed treatment options need to be considered for patients presenting for these procedures with suspected AT deficiency.

Antithrombin III deficiency, whether congenital or acquired, is a concern for the patient undergoing CPB. Since it is possible that this deficiency will be unknown prior to the procedure, anesthesia practitioners should be aware of patient populations that are at greater risk for this condition. Patients with known hepatic dysfunction as well as those who have been on prolonged heparin infusions should be considered at risk for AT deficiency, especially prior to procedures requiring CPB. In situations where AT deficiency is suspected, it may be beneficial to have FFP available prior to the procedure so that administration may be expedited if needed. Often the only indication the clinician may encounter intraoperatively is a resistance to heparin manifested by minimal changes in or even subtherapeutic ACT values. It should be noted that while there are numerous congenital and acquired causes for this

phenomenon, the treatment is essentially the same. Additionally, if a patient experiences continued signs of thrombophilia postoperatively and acquired causes of AT deficiency have been ruled out, a definitive diagnosis of AT deficiency could be made by obtaining a functional amidolytic antithrombin assay to establish quantitative values of serum antithrombin activity.²

Administration of FFP, whole blood, or preparations of AT will replenish coagulation factors that are either congenitally deficient, are decreased secondary to hepatic dysfunction, or have been consumed through binding with heparin or other substances. It is not clear from available clinical data which scenario was experienced by this patient. Intraoperatively, treatment successfully corrected his coagulopathy and the surgical procedure continued uneventfully.

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Pediatric Dexmedetomidine Use in Total Intravenous Anesthesia

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Keywords: dexmedetomidine; total intravenous anesthesia; α_2 -adrenergic agonists; motor evoked potentials; somatosensory evoked potentials

The utilization of α_2 -adrenergic agonists such as clonidine and dexmedetomidine in anesthesia has gained popularity in recent years due to their abilities to provide

sedation, analgesia, anxiolysis, and decreased anesthetic requirements.¹ Dexmedetomidine is increasingly being utilized due to its shorter half-life when compared to clonidine as well as its highly selective α_2 -agonism.² Studies have shown dexmedetomidine to produce a neuroprotective effect, and therefore it is important to understand the effects of

dexmedetomidine on neurophysiologic monitoring as it may be an appealing adjunct for neuroanesthesia.¹ A few case reports, similar to this one, have shown alterations in motor evoked potentials (MEPs) with the anesthetic administration of a dexmedetomidine infusion.

Case Report

An 11 year-old female, weighing 47.6 kilograms, presented for a posterior spinal fusion with instrumentation and multiple spinal osteotomies. Neurophysiologic monitoring was planned to include somatosensory evoked potentials (SSEPs) and MEPs. Her past medical history was significant only for idiopathic scoliosis with spine curvature progressing significantly over the past year. Preoperative pulmonary function tests were not performed as the patient did not have an excessively large curve or reactive airway disease. She was not taking any medications and had no past surgical history. Complete blood count and coagulation studies were all within normal limits. Urinalysis was negative for human chorionic gonadotropin. The patient was typed and crossed for 2 units of packed red blood cells.

Thirty minutes prior to departure to the operating room, the patient was premedicated with oral midazolam 12.5 milligrams (mg). Upon arrival in the operating room, standard non-invasive monitors were applied, and she was preoxygenated with 100% fraction of inspired oxygen (FIO₂) at 10 liters per minute (L/min) via face mask. An inhalation induction was performed on the stretcher with administration of nitrous oxide at 4 L/min and oxygen at 2 L/min before insertion of an 18-gauge peripheral intravenous catheter (PIV). Once the PIV was in place, nitrous oxide was discontinued

and sevoflurane was titrated up to 8%. Glycopyrrolate 0.2 mg and propofol 100 mg were administered IV prior to tracheal intubation. No neuromuscular blocking agents were administered due to planned MEP monitoring. The patient's trachea was intubated without incident, and sevoflurane was decreased to 2%.

After induction, a second 18-gauge PIV and a radial arterial line were inserted. Following line placement, sevoflurane was discontinued, an IV propofol infusion was started at 150 micrograms per kilogram per minute (mcg/kg/min), IV dexmedetomidine infusion was started at 1 mcg/kg per hour (mcg/kg/hour), and IV remifentanyl infusion was started at 0.2 mcg/kg/min. The patient was positioned prone on the operating room table.

Baseline SSEPs and MEPs were recorded by the neurophysiologist, and they were within normal limits. After burst suppression was noted by the neurophysiologist, the propofol infusion was decreased from 150 to 100 mcg/kg/min without changing the other IV infusions. Approximately one hour later, MEP amplitudes for bilateral upper and lower extremities decreased and eventually disappeared. However, SSEPs remained within normal limits for bilateral upper and lower extremities. At the time of MEP disappearance, all vital signs were within normal limits and the spine had not yet been instrumented. The electrophysiologist ruled out equipment failure and notified the surgeon of the change. Due to the electrophysiologist's lack of experience with dexmedetomidine, he recommended that we discontinue the infusion temporarily. After discontinuation of the dexmedetomidine infusion, MEPs returned to baseline within 20 – 30 minutes. There were not any changes in SSEPs throughout the case.

The patient's emergence was uneventful. The propofol and remifentanyl infusions were titrated off before emergence, and morphine 10 mg was administered IV for postoperative analgesia. The endotracheal tube was removed without incident and the patient was transported to the post anesthesia recovery unit (PACU) uneventfully.

Discussion

Clonidine is an α_2 -adrenergic agonist that is increasingly being used in anesthesia practice due to its sedative and analgesic properties. Alpha-2 receptors can be found on blood vessels where they mediate vasoconstriction, and on sympathetic terminals where they inhibit norepinephrine release.³ In addition to decreasing anesthetic and analgesic requirements, clonidine also preserves respiratory function, but it has a relatively long half-life of 6-12 hours.³ Dexmedetomidine is another α_2 -adrenergic agonist that has a shorter 2-hour half-life and a much higher ratio of α_2 to α_1 activity than clonidine—1600 to 1 as opposed to 200 to 1, respectively.² Due to its full α_2 -agonism, dexmedetomidine may have increased sedative and analgesic effects when compared to clonidine without untoward α_1 -mediated cardiovascular effects.²

The Food and Drug Administration (FDA) approved and released dexmedetomidine in December 1999.⁴ It was clinically approved as an adjunct in the sedation of intubated and mechanically-ventilated adult intensive care unit patients.⁴ Two randomized, double-blind, parallel-group, placebo-controlled multicenter clinical trials were cited by the FDA comparing dexmedetomidine to placebo for the sedation of mechanically ventilated patients.

Both studies showed that the placebo groups required significantly more rescue medication (midazolam in one study and propofol in another study) to maintain sedation within specified parameters.⁴ Since its FDA approval, much research has been performed exploring dexmedetomidine use in adults.

By activating receptors in the medullary vasomotor center, centrally-acting α_2 -adrenergic agonists like clonidine and dexmedetomidine reduce norepinephrine causing a central sympatholysis which leads to decreased heart rate (HR) and blood pressure (BP).⁵ The presynaptic α_2 -adrenergic receptor is a negative feedback receptor, therefore agonists at this receptor cause a decrease in catecholamine release from the nerve terminal.⁵ Action of α_2 -adrenergic agonists on the locus ceruleus, which is located in the brainstem, plays a prominent role in the sedation and anxiolysis produced.⁵ Parasympathetic outflow is stimulated and firing of inhibitory neurons is increased from the locus ceruleus.⁵ Activation of α_2 -adrenergic receptors in the dorsal horn of the spinal cord leads to the inhibition of substance P release and subsequent analgesic effects.⁵ Overall, dexmedetomidine produces sedation, anxiolysis, analgesia, blunting of the sympathetic nervous system, and a decrease in minimum alveolar concentration of inhalational anesthetics.

In contrast to the extensive research of dexmedetomidine use in adults, there are mainly only anecdotal reports of its use in children.⁶ The FDA warns that the use of dexmedetomidine in young, healthy people with high vagal tone has been associated with episodes of bradycardia and sinus arrest. Therefore, dexmedetomidine is not recommended for use in patients under 18 years of age as more clinical studies are

required to establish its safety and efficacy in this population.⁴

Dexmedetomidine is utilized by clinicians during TIVA in order to decrease propofol requirements and facilitate a more rapid patient emergence from anesthesia. This was the main reason for utilizing Dexmedetomidine for this particular case.

Similar to this case report, there have been a few case reports highlighting the loss of MEPs during pediatric surgery with dexmedetomidine administration. In the aforementioned case, it was unlikely that direct spinal cord injury had occurred as the loss of MEPs for bilateral upper and lower extremities transpired simultaneously. Also, hypoperfusion to the spinal cord was unlikely as mean arterial pressures were within optimal ranges. In two other case reports, loss and decreased amplitude of MEPs occurred during TIVA with propofol, remifentanyl, and dexmedetomidine.⁷ Similarly in these cases, neuromonitoring equipment failure, surgical manipulation of the spine, and spinal cord hypoperfusion were all ruled out as causes for weakened MEP amplitudes.⁷ It was thought that excessive doses of dexmedetomidine were responsible for the loss or decrease of the MEP signal in both cases.⁷ Also, it was noted that SSEPs signals remained unchanged, which is consistent with the findings of this case.⁷

In addition, research has been performed in animals looking at the effects of dexmedetomidine on MEPs and SSEPs. One group of researchers examined the effects of dexmedetomidine on MEPs in rabbits, and they found that dexmedetomidine decreased MEPs amplitudes in a dose-dependent manner.¹ Another group of researchers observed the effects of a range of dexmedetomidine doses on SSEPs in rats.⁸ In this study, it was found that

dexmedetomidine did not suppress SSEPs amplitudes, and it actually tended to increase the amplitudes.⁸ The results of both of these studies are consistent with the events of this case report, and they confirm the need for more research regarding dexmedetomidine's effects on neurophysiologic monitoring.

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General Anesthesia in a Patient with Delirium Tremens

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Keywords: alcohol abuse, alcohol withdrawal, benzodiazepines, delirium tremens, Wernicke syndrome

Delirium tremens (DT) is a potentially life threatening emergency caused by acute alcohol withdrawal. Symptoms may begin within a few hours after the cessation of ingesting alcohol, but may not peak until 48-72 hours later.¹ Approximately 5% of patients experiencing alcohol withdrawal will exhibit symptoms of delirium tremens.² Symptoms include hallucinations, combative behavior, hypertension, tachycardia, severe agitation, tremors, and seizures. The mortality rate for patients experiencing DTs is 5-15%.³ Current treatments are supportive and include treating the symptoms and reducing the risk of mortality. This can be done with benzodiazepines, antihypertensive medications and the correction of fluid and electrolyte imbalances.

Case Report

A 54 year-old male with a self-reported history of alcohol use of 7-8 beers per night presented to the emergency room with abdominal pain. He reported a 15-20 pound weight loss and severe abdominal pain in the last few weeks. Significant medical history

included diverticulitis and nephrolithiasis. He was admitted to a medical/surgical unit with pneumonitis intestinalis and toxic megacolon. At that time he was placed on an alcohol withdrawal protocol. Multiple colonoscopies were attempted for bowel decompression without relief. During this time the patient became more agitated and combative prompting lorazepam boluses PRN every 4 hours for sedation. Four days after failed conservative treatment, the patient was taken to the operating room for a colectomy. This procedure was completed without incident and the patient was returned to the ICU with an endotracheal tube in place and mechanically ventilated. Propofol and lorazepam infusions were initiated for sedation.

The following day, the patient returned to the operating room for an exploratory laparotomy, ileostomy, and bilateral ureteral stents. The trachea remained intubated, cisatracurium 16 mg and propofol 50 mg were given prior to transport. Transport was made with EKG, arterial blood pressure, pulse oximetry, and end tidal carbon dioxide monitoring with manual ventilation by ambu® bag. In the operating room the patient was placed in the lithotomy position with his arms tucked at his sides.

Sevoflurane was initiated at 0.5 MAC and maintained throughout the procedure at 0.5-1.0 MAC. Infusions of total parenteral nutrition at 85 ml/hr, regular insulin 1.5 units/hr, lactated ringers 50 ml/hr, and propofol 25 mcg/kg/min were maintained throughout the procedure. Scheduled medications included metronidazole, ciprofloxacin, and lorazepam 1-2 mg every 4 hours. A free flow IV of 0.9% saline was dedicated for the use of anesthesia medication administration and circulated through a fluid warming unit.

During the case hourly blood samples were obtained to include: arterial blood gases, basic metabolic profile, and hemoglobin levels. All labs were within normal limits with the exception of the serum potassium which was 2.8 mEq/L. Potassium chloride 80 mEq was infused over four hours via central intravenous catheter. Insulin was titrated according to a sliding scale. Hypotension was treated with bolus doses of phenylephrine and titration of sevoflurane. Tachycardia was treated with bolus doses of esmolol. All other medications were continued as previously scheduled.

The procedure was completed without any adverse incidents. The patient was transported to the intensive care unit and remained mechanically ventilated for the next seven days. He was then moved to a medical/surgical unit where he remained for continued medical treatment.

Discussion

Acute alcohol withdrawal is caused by the abrupt cessation of alcohol. This causes the patient to have increased activity of the autonomic nervous system as reflected by the symptoms the patient experiences.⁴ Alcohol affects the brain in a variety of ways. The brain maintains its neurochemical

balance through the use of excitatory and inhibitory neurotransmitters. The primary excitatory neurotransmitter is glutamate, which acts through the N-methyl-D-aspartate (NMDA) receptor and the primary inhibitory neurotransmitter is gamma-aminobutyric acid (GABA) which acts through the GABA-alpha (GABA-A) receptor.⁵

Alcohol has inhibitory effects on the NMDA receptor, and chronic exposure causes an up-regulation. When there is abrupt cessation of alcohol, the result is brain hyperexcitability which is manifested by anxiety, irritability, agitation, and tremors. Alcohol also enhances the effects of GABA on GABA-a receptors resulting in decreased brain excitability. Chronic exposure results in a down-regulation of GABA-A receptors which is evidenced by tolerance to alcohol.⁵

Chronic alcohol use can have many effects on a patient's condition during anesthesia. Therefore it may be challenging when choosing an anesthetic technique, especially if the patient is experiencing delirium tremens. The patient can have enhanced or reduced sensitivity to anesthetic agents. On average, alcohol accounts for half of an alcoholic's caloric intake so they are often malnourished.⁶ This was apparent in this patient as not only did he report decreased food intake while continuing alcohol use, he also was having abdominal pain which together caused him to have a recent 15-20 pound weight loss.

As the treatment of alcohol withdrawal is supportive, a patient is at increased anesthetic risk during this time. If the procedure is elective, it should be delayed until the patient's symptoms are controlled. If the procedure is not elective such as in this case, careful management is required to protect the patient and decrease morbidity

and mortality. Benzodiazepines are helpful and are one of the first line medications used in a patient experiencing DTs. These drugs have a high therapeutic index and act on the benzodiazepine-GABA_A-chloride receptor complex. This causes enhanced chloride flux, hyperpolarization of the membrane, and neural inhibitory effects.¹ In this patient, this was one of the initial treatments used.

Another goal in the management of this patient was to correct fluid and electrolyte imbalances. This can be done with intravenous infusions, multivitamins and thiamine. Thiamine is essential in the body's energy metabolism. Thiamine deficiency is a factor in the development of Wernicke syndrome, which can lead to irreversible dementia.⁴ There are no diagnostic tests to confirm Wernicke syndrome, therefore mental status changes are often used for diagnosis. Fifteen to twenty percent of patients hospitalized with Wernicke syndrome will die from the disorder; for this reason, patients experiencing alcohol withdrawal should receive thiamine as soon as treatment begins.⁴ This patient received an intravenous infusion during initial treatment, which included the essential amounts of multivitamins, thiamine, dextrose and folic acid to replace the body's deficiency. The patient was then continued on thiamine 100 mg and folic acid each day when oral intake was possible.

Prior to transport and during the procedure, the patient remained tachycardic with his heart rate remaining between 100-120 beats per minute. This may have been due to a variety of reasons. This may have been a symptom of DT, related to the pain of surgery itself, or dehydration. Fentanyl was titrated without a change in symptoms, IV fluids were continued, and esmolol was given to treat the heart rate. Esmolol is a beta-1 adrenergic receptor blocking agent

with a very short duration of action. Bolus doses of IV esmolol were the most effective in controlling the heart rate. This worked well for this patient because it reduced the heart rate by 10-20% without causing changes in the blood pressure. Esmolol dosing was later discontinued in the intensive care unit.

An anesthetic for a patient experiencing DT is a challenge with an increased risk for morbidity and mortality. If the case is elective, it is best for the patient to have surgery postponed until they have been without alcohol for at least ten days.⁵ If it is not elective, special care must be taken with the patient to prevent further decreases in health status. Careful titration of medications and fluids must be made to keep the patient's vital signs stable. It may also be necessary to leave the patient mechanically ventilated with sedation. In the end, the decision is made by the anesthesia professionals and the surgeon whether the surgery is required. If surgery can not be delayed, the main goal is to keep the patient safe through the management of the symptoms they are experiencing.

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Klippel-Trenaunay-Weber Syndrome

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Key words: Klippel-Trenaunay-Weber syndrome; Kasabach-Merritt syndrome; coagulopathy; anesthesia; arteriovenous malformation

Klippel-Trenaunay-Weber Syndrome (KTWS) is a rare, non-hereditary disorder resulting in the triad of varicose veins, overgrowth of soft tissue and bone leading to extremity hypertrophy, and cutaneous and deep hemangiomas.¹ In patients with KTWS, thromboembolic events such as deep vein thrombosis (DVT) and pulmonary embolus (PE) are relatively common.² It has been estimated that the occurrence of PE in these patients is 14% to 22%.³ Also, it is possible for these patients to present with spinal hemangiomas and to bear an increased risk of coagulopathy and hemorrhage intraoperatively.⁴ The anesthetic management of patients with this syndrome will be discussed.

Case Report

A 38 year-old male with a height of 5 feet 11 inches and a weight of 121 kilograms presented for left knee arthroscopy with complaint of left knee pain and a preoperative diagnosis of left knee degenerative joint disease (DJD). His past medical history was significant for Klippel-Trenaunay-Weber Syndrome diagnosed at age 9, gastroesophageal reflux disease (GERD), and obesity. He was currently

taking ranitidine for GERD and had no known drug allergies. His surgical history was significant for bilateral saphenous vein strippings in 1999 and 2001 and bilateral knee arthroscopies in 1991. All the procedures were performed under general anesthesia.

Laboratory studies included a platelet count of 197,000 per microliter, a prothrombin time (PT) of 11.7 seconds, an activated partial thromboplastin (aPTT) time of 27 seconds, and an international normalized ratio (INR) of 1. A duplex scan of both lower extremities revealed scattered varicose veins, but no deep vein thromboses. The results of a chest x-ray were within normal limits.

The physical examination revealed a port-wine stain covering the patient's upper back and both upper extremities, becoming patchy on the distal forearms. Hypertrophy was evident in the extremities, with more extensive hypertrophy in the lower extremities. His airway was Mallampati class II with long, protruding upper incisors and a recessed chin.

The patient was premedicated with intravenous (IV) midazolam 2 milligrams (mg) and glycopyrrolate 0.2 mg via an 18-gauge peripheral IV line en route to the operating room. After standard monitors

were applied, the patient was preoxygenated via face mask with 100% oxygen delivered at a rate of 10 liters per minute (L/min). The patient had controlled GERD with no associated symptomatology, therefore a decision was made to perform general anesthesia with a size 5 laryngeal mask airway (LMA). An IV induction was performed with lidocaine 80 mg followed by propofol 300 mg. Continuous end-tidal carbon dioxide (CO₂) was confirmed and metoclopramide 10 mg was administered post induction. General anesthesia was maintained with end-tidal sevoflurane 2 % with 100% oxygen at 2 L/min. Fentanyl 150 micrograms was titrated to the patient's respiratory rate.

Approximately 30 minutes before emergence from anesthesia, ondansetron 4 mg was administered IV. Emergence from anesthesia was uneventful, and the LMA was discontinued without complication. Oxygen was administered via nasal cannula at 4 L/min during transfer to the post-anesthesia care unit (PACU). He remained in the PACU for about 3 hours and was then discharged to home.

Discussion

In 1900, noted French physicians Klippel and Trenaunay first described a syndrome in 2 patients presenting with a port-wine stain and varicosities of an extremity associated with hypertrophy of the bony and soft tissue of the affected limb.¹ In 1918, Parkes Weber, unaware of Klippel and Trenaunay's report, described a patient with the three aforementioned symptoms as well as an arteriovenous malformation of the affected extremity.⁵ Therefore, the syndrome characterized by the above triad plus arteriovenous malformation is called either KTWS or Parkes Weber syndrome.

KTWS is a rare congenital anomaly and as of 1999 only about 1,000 cases have been reported.² As a result, few case reports discussing the anesthetic management of patients with KTWS appear in the literature. Anesthesia practitioners must understand the possible complications that can occur when patients with KTWS have surgery and properly assess them preoperatively. A complete blood count and coagulation studies are a necessity preoperatively as large varicosities and hemangiomas can lead to anemia and localized coagulopathy.⁵ Furthermore, preoperative coagulation studies are important due to the potential for disseminated intravascular coagulation in KTWS patients, possibly due to the trapping of platelets in hemangiomas leading to activation of the clotting cascade.¹ A rare but severe complication of KTWS is Kasabach-Merritt syndrome (KMS), a consumptive coagulopathy secondary to hemorrhage from arteriovenous malformations (AVMs). KMS is characterized by severe thrombocytopenia, hypofibrinogenemia, and increased fibrinopeptide A and fibrinogen degradation products.^{1,6}

Due to the risk of major hemorrhage in KTWS patients, large-bore IV access is recommended as well as having cross-matched and/or cell saver blood available for some surgical procedures.¹ The risk of hemorrhage is of great importance during childbirth or cesarean section. Parturients with KTWS should probably be crossmatched for coagulation factors such as platelets or cryoprecipitate as they may have AVMs involving the vulva or uterus that can cause platelet destruction and coagulopathy.^{5,7} Pregnancy puts patients at increased risk for complications of KTWS, but despite discouragement of pregnancy in these patients, at least 17 cases have been reported.⁵ Pregnant KTWS patients should be assessed for uterine vascular

malformations via prenatal ultrasound. In addition, they should be examined for smaller vascular malformations of the spine and brain via magnetic resonance imaging (MRI) as an increased risk of cerebrovascular accidents exists with these malformations.⁷ Anesthesia practitioners should be consulted during the intrapartum period to determine the type of anesthesia that should be used so that it can be administered promptly for an emergent or scheduled cesarean section or labor.⁵

Regional anesthesia is usually not recommended for KTWS patients as needle insertion could cause trauma to hemangiomas leading to epidural or spinal hematomas.^{4,7} However, Stein and his colleagues successfully administered regional anesthesia and analgesia to KTWS parturients, who desired to be awake during the birth of their babies.⁵ Preoperative computerized axial tomography (CAT scan) and MRI of the spine can detect hemangiomas, or benign skin lesions made up of dense masses of dilated blood vessels. Regional anesthesia can be safely administered if no vascular anomalies are present.⁴ Nonetheless, the anesthetist should be aware that hematoma onset is evidenced by severe back pain that progresses to paraplegia. Prompt diagnosis is necessary so that a decompression laminectomy may be performed as soon as possible.⁴

All the aforementioned factors were considered in planning the anesthetic for this patient. Preoperative complete blood count and coagulation studies were within normal limits. Because minimal blood loss was anticipated, an 18-gauge IV was deemed appropriate. General anesthesia was chosen for our patient as he had not had any preoperative spinal imaging studies to assess for vascular malformations. Although the intraoperative blood loss was greater than

anticipated, the amount was far less than his allowable blood loss and adequately replaced with crystalloids.

KTWS is a rare disorder that anesthesia practitioners should be aware of due to the potential for serious complications. Administering anesthesia to a patient with this syndrome reinforced the importance of a thorough preoperative assessment, including additional imaging studies, as the safety of the patient is top priority. As with every surgical patient, being prepared in advance for any possible complication is of utmost importance.

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Anesthetic Considerations in a Patient with Marfan Syndrome

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Key words: Marfan syndrome, aortic dilatation, anesthesia, Multiple Endocrine Neoplasia syndrome, pheochromocytoma

Marfan syndrome (MFS) is a familial disorder of connective tissue inherited as an autosomal dominant trait with prominent manifestations in the skeletal, ocular, cardiovascular, and pulmonary systems.¹⁻⁵ Although relatively rare, prevalence has been estimated at 2-3 per 10,000.² In patients with MFS, severe mitral valve prolapse (MVP) and aortic dilation and dissection are the leading causes of mortality.¹⁻⁴ To minimize morbidity and mortality, patients with MFS present for varying surgical interventions.¹⁻⁴ This case report discusses the anesthetic management of patients with this syndrome.

Case Report

A 19 year-old male with a height of 180.3 centimeters and a weight of 67.8 kilograms presented for left laparoscopic adrenalectomy with a preoperative diagnosis of left adrenal pheochromocytoma. Past medical history was significant for Marfan syndrome diagnosed at age 5, Multiple Endocrine Neoplasia syndrome Type IIB diagnosed at age 10, and hypothyroidism. He was currently taking levothyroxine for hypothyroidism, fexofenadine for seasonal allergies, and phenoxybenzamine for the past 2 weeks preoperatively. No known drug

allergies were reported. Surgical history was significant for total thyroidectomy in 1998 and right laparoscopic adrenalectomy for pheochromocytoma in 2004, both under general anesthesia.

Laboratory results were unremarkable. Electrocardiogram exhibited normal sinus rhythm. Echocardiogram showed moderate MVP with moderate mitral regurgitation (MR) and cardiomegaly with left atrial enlargement but no aortic dilation. Chest radiograph (CXR) demonstrated neither active disease nor compression of the left mainstem bronchus.

Physical examination revealed a tall, thin, asymmetrical faced male with thoracolumbar scoliosis, pectus cavernosm, disproportionate arm span and arachnodactyly. His airway was Mallampati class II with a high arched palate and a prognathic jaw. Auscultation was significant for an apical systolic click.

The patient was premedicated with oral midazolam 10 milligrams (mg) 30 minutes prior to entering the operating room, and after standard monitors were applied, the patient was preoxygenated via face mask with 100% fraction of inspired oxygen delivered at a rate of 10 liters per minute (L/min). A brief desaturation due to a difficult mask fit required assistance with a double-handed jaw thrust. An intravenous

(IV) induction was performed with lidocaine 70 mg followed by propofol 150 mg, fentanyl 100 micrograms (mcg), and rocuronium 40 mg via a 20-gauge peripheral IV line. The trachea was intubated with a size 8.0 endotracheal tube and continuous end-tidal carbon dioxide was confirmed. Ventilation was then mechanically controlled.

General anesthesia was maintained with end-tidal desflurane 7.0% with oxygen flow of 2 L/min, IV fentanyl 200 mcg total titrated before incision, and IV rocuronium 96 mg total titrated to maintain $\frac{1}{4}$ TOF. After induction, dexamethasone 10 mg was administered IV and a left radial arterial line and a right internal jugular cordis were placed. 30 minutes prior to incision, IV ampicillin 2 grams and IV gentamycin 80 mg were administered. Phentolamine, esmolol, nitroprusside, epinephrine, and norepinephrine were available. 6 L of lactated Ringers solution and 750 milliliters (mL) of Albumin were administered over the 6-hour surgery. Urine output totaled at 445 mL and blood loss totaled at 350 mL Dopamine was initiated at 3 mcg/kg/min for renal support. The operation otherwise was uneventful.

30 minutes before emergence, IV ondansetron 4 mg was administered. The trachea was extubated when criteria were met following completion of the surgical procedure. 6 L/min oxygen via face mask was administered throughout transfer to the post-operative care unit (PACU). After 2 hours of observation in the PACU, he was transferred to the intensive care unit for overnight monitoring where no events were recorded.

Discussion

In 1896 Antoine Marfan, a French pediatrician, was the first to present a child with unusual anomalies in her skeletal system.³ In 1931, MFS was found to be an autosomal dominant disorder and later known to be caused from mutations in the FBN1 gene on chromosome 15.^{2,3,5} To date more than 500 mutations specific to each family's pedigree have been identified.²

Prior to 1995, diagnostic criterion for MFS were based primarily upon certain abnormalities in the skeletal, ocular, and cardiovascular systems.⁴ The Ghent nosology has since taken over specifying major criteria present in at least two different organ systems as well as a third organ system involved as either a major or a minor criteria.^{4,5} In addition to the aforementioned organs, the pulmonary system has recently been included to the list of minor criteria.⁵ The majority of patients with MFS exhibit a marfanoid musculoskeletal body habitus, bilateral ectopia lentis, aortic involvement, and a family history of MFS varying in onset and severity.^{4,6} Even without meeting criteria for MFS, a marfanoid appearance still puts patients at high risk of aortic root aneurysms.⁶

The Marfan phenotype is based on the traditional characteristics of unusually tall, thin children with excessive longitudinal bone growth and long "spidery" fingers (arachnodactyly).⁷ Height often exceeds 6 feet 1 inches and more severe forms of MFS are seen at heights greater than 6 feet 3 inches.⁶ Disproportionate arm span exceeding height is commonly seen, and long, narrow, asymmetrical faces are often seen as well. This in combination with a prognathic jaw and atlantoaxial instability complicates mask ventilation and

intubation.⁷ Additionally, thoracolumbar scoliosis and pectus excavatum can further inhibit ventilation and oxygenation.⁷

Restrictive pulmonary disease may result from the above skeletal abnormalities consistently decreasing total lung capacity especially seen with lower values of forced vital capacity and forced expiratory volume in 1 second.⁸ Fortunately, the patient previously mentioned demonstrated pectus cavernosum and respiratory compromise was not an issue. Interestingly, the literature does not support any evidence of a connective tissue defect in the lung parenchyma thus a Marfan patient who lacks this chest wall deformity does not have any inhibition of pulmonary function.⁸ However, spontaneous pneumothorax and apical blebs have been connected to MFS and stated as minor criteria.⁵

Cardiovascular manifestations of MFS pose the greatest threat in terms of morbidity and mortality.² Degeneration of elastic fibers in vascular media, referred to as cystic medial necrosis, initiates dilatation of the mitral annulus, aortic root and arch leading to valvular dysfunction and aneurysm formation. 60-80% of adults with MFS exhibit dilatation of the sinus of Valsalva.² Unrecognized dilatation is complicated by aortic dissection and rupture leading to premature death.^{1-4,7} Aortic root dilatation can be identified early in childhood by echocardiogram and variably progresses to aortic regurgitation in adulthood.^{3,4,7} The risk of dissection or rupture exponentially increases around adolescence, however, rupture can occur at any point in the lifespan.^{4,7} Root replacement is based upon aortic dimension, rate of increase, and family history.³ Aortic diameters exceeding 6 centimeters generally requires surgery.⁷ Beta-Blocking therapy has become the mainstay in non-operated Marfan patients

reducing the wall tension on the great vessels and the development of aortic dissection.⁹

Other common cardiovascular manifestations of MFS include MVP with severe MR as seen in the patient discussed.^{2,3,5} Significant valvular regurgitation can lead to compression of the left mainstem bronchus from an enlarged left atrium thus requiring replacement.^{1,3} On preoperative assessment, a systolic click will be heard on systole at the apex of the heart demonstrating MR.¹⁰ Main pulmonary artery dilatation, a relatively unknown cardiovascular manifestation, has also been linked to MFS predisposing patients to left ventricular dilatation and dysfunction.^{2,5} Congestive heart failure and angina pectoris may also develop before the fourth decade of life.¹

In considering the question of any type of surgery in MFS patients, anesthesia practitioners are faced with several issues. Preoperatively, identification of high-risk groups of serious cardiovascular complications is imperative. Echocardiograms may find an early diagnosis of valvular dysfunction, congestive heart failure, or aortic dilatation and aneurysm.⁴ Medications used preoperatively for cardiovascular failure should be continued.^{2,4,9} CXR may be useful in detecting apical blebs; pulmonary function tests may be needed to identify restrictive lung disease.^{5,8} A difficult mask fit should be anticipated and atlantoaxial instability must be identified prior to laryngoscopy.⁷ No specific anesthetic recommendations are reported, but the induction and maintenance technique should avoid hypertension and tachycardia to prevent aneurysm rupture and to preserve myocardial oxygenation. With unknown aortic wall thickness, a transesophageal

echocardiography should be considered and rapid aortic expansion should always be anticipated. In summary, a thorough preoperative assessment must be initiated in the Marfan patient and hemodynamic stress on the aorta must be minimized throughout surgery.

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Anesthesia During Surgical Removal of Suspected Insulinoma Bradley J. Young, BSN

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Key Words: anesthesia, insulinoma, pancreatic tumor, hypoglycemia

An insulinoma is a tumor of the pancreas derived from the beta cells of the islets of Langerhans. It has the ability to synthesize and secrete insulin, yet it is autonomous of the normal feedback mechanisms.¹ Fewer than 5% of insulinomas are larger than 3 cm. Ninety percent or more of all insulinomas are benign.² The diagnosis of an insulinoma is usually made biochemically with low

blood sugar, elevated insulin, pro-insulin and C-peptide levels, and confirmed by medical imaging or angiography. The definitive treatment is surgery.^{1,2} The purpose of this case report is to present the anesthesia management of a patient with probable insulinoma.

Case Report

A 55-year-old, 5'3", 134 kg, ASA 3 female presented for exploratory laparoscopy and

resection of insulin-secreting tumors. Her medical history was significant for two previous hypoglycemic events with seizures in the prior three months, palpitations, tobacco use, hypercholesterolemia, obesity, esophageal varices, portal hypertension, non-alcoholic steatohepatitis with cirrhosis, gastroesophageal reflux disease, osteoarthritis and depression. Her surgical history included salpingoophorectomy, partial nephrectomy due to a benign tumor and removal of multiple epithelial cysts from her uterus. Her daily medications included inderal, atorvastatin, venlafaxine, fexofenadine, esomeprazole, and lactulose.

The initial diagnostic workup revealed high concentrations of insulin after prolonged fasting, increased concentrations of C peptide and several small abdominal masses found by ultrasound. The patient was admitted to the hospital the night before surgery and ordered to have nothing by mouth. Blood glucose was monitored every hour and an intravenous (IV) infusion of 5% dextrose in ringer's lactate was started to maintain blood glucose 80-120 mg/dL.

The morning of surgery significant labs included a blood glucose concentration of 92 mg/dL and potassium concentration of 4.3 mEq/L. Her heart was in normal sinus rhythm at 65 beats/min, blood pressure was 123/72 mmHg and oxygen saturation was 95% on room air.

In the preoperative holding area, the patient was given famotidine 20 mg, sodium bicitrate 30 mL, metoclopramide 10 mg and midazolam 2 mg. Upon arrival to the operating room, standard monitors were applied and preoxygenation via face mask was performed for five minutes. A modified rapid sequence induction with propofol 80 mg, fentanyl 100 mcg and cisatracurium 11 mg was performed while maintaining

cricoid pressure. The trachea was intubated with a 7.5 mmID endotracheal tube. Following induction, an arterial line and a central venous pressure (CVP) catheter were placed. Anesthesia was maintained with a mixture of oxygen, air and isoflurane.

Following induction, the patient's blood pressure and heart rate remained within normal limits. The infusion of 5% dextrose in ringer's lactate was continued intraoperatively. An IV infusion of the somatostatin analog octreotide was started at 50 mcg/hr. Blood glucose was measured every 10 minutes during the initial surgical period. Once the surgeon began to remove the suspected insulinomas, blood glucose was checked every five minutes and the 5% dextrose in ringer's lactate was stopped. After removal of all suspected insulin-secreting tumors, the patient's blood glucose concentration continued to be maintained at 100-120 mg/dL. All tissue samples were identified by pathology as benign and non-insulin secreting. Intra-operative ultrasound was consequently performed and no additional masses could be found. The surgeon then performed a distal pancreatectomy. This portion of the surgery was complicated by the rapid loss of 1,200cc of blood. Two units of packed red blood cells (PRBC) were administered.

Following completion of the surgery the patient was not extubated per the surgeon's request. She was transferred to the surgical intensive care unit (SICU) for close monitoring where she remained intubated for two days. She also required additional blood component administration, and continued to have blood glucose instability. Further diagnostic testing was scheduled to evaluate the cause of her continued hypoglycemic events.

Discussion

A case of suspected insulinoma requires careful preparation and vigilant monitoring by the anesthesia professional. In this case, the patient had many of the typical signs of insulinoma. Patients with insulinoma have symptoms of hypoglycemia resulting from neuroglycopenia and increased catecholamine release.² Neuroglycopenic symptoms are most common and include recurrent headache, lethargy, diplopia and blurred vision, particularly with exercise or fasting. Severe hypoglycemia could result in seizures, coma and permanent neurological damage. Symptoms resulting from the catecholaminergic response, such as tremulousness, palpitations, tachycardia, diaphoresis, hunger and anxiety may also be present but are less common.^{1,2} The diagnosis of true hypoglycemia is based on Whipple's triad: 1) symptoms and signs of hypoglycemia, 2) concomitant plasma glucose level of 45 mg/dL (2.5 mmol/L) or less and 3) reversibility of symptoms with administration of glucose.² This patient had many of the classic symptoms of hypoglycemia. She also had a non-invasive ultrasound that identified multiple small abdominal masses. Insulinoma can be localized 65-70% of the time by non-invasive means including ultrasound, CT scan or MRI techniques.^{2,3}

There has been much controversy over glucose management during the resection of an insulinoma. Intraoperative hypoglycemia is a real danger because the classic signs and symptoms of hypoglycemia are masked by anesthesia.⁴ In addition to preventing hypoglycemia, it is important to avoid hyperglycemia because insulin secretion from beta islet cells is stimulated by high blood glucose levels. For this reason, preoperative corticosteroid therapy is best avoided.⁵ In a classic study by Muir et al., it

was concluded that intermittent sampling (every 15 min) of plasma glucose is adequate to protect patients from dangerous hypoglycemia provided plasma glucose is kept above 60 mg/dL.^{4,6}

There are contrasting approaches to the perioperative management of glucose in patients undergoing resection of insulinoma. Although a continuous IV infusion of a dextrose containing solution is not absolutely indicated, it is commonly used to achieve the goal of maintaining a blood glucose concentration greater than 60 mg/dL.^{4,6} If administering a dextrose containing solution, close communication with the surgeon is essential. As the surgeon removes the suspected insulinoma, hyperglycemic rebound potentially will occur. This is characterized by a rise in plasma glucose values within 30 minutes after excision of the tumor. This plasma glucose rise is an indicator of total tumor removal. The approach of administering glucose-containing infusions has been criticized because of subsequent inability to detect hyperglycemic rebound after excision of the tumor.^{4,6} By contrast, others who deliberately maintain moderate hypoglycemia by using non-glucose containing IV solutions and intermittent glucose determinations may place the patient at risk for developing dangerous hypoglycemia between measurements. Most anesthesia professionals who employ this technique consider it important to detect hyperglycemic rebound. However, the manipulation of an insulinoma by the surgeon can result in massive insulin release from the tumor with subsequent hypoglycemia. In this situation, anesthesia professionals often times must provide IV boluses of dextrose. This action may mask evidence of hyperglycemic rebound.^{4,6}

During this case, the surgeon requested perioperative use of octreotide. Octreotide suppresses insulin release from insulinomas and appears to reduce perioperative morbidity in settings where a mechanical pancreas is not available.⁴ Five percent dextrose in ringer's lactate was administered to maintain blood glucose concentrations near 100 mg/dL. In this case, using close communication with the surgeon, we stopped the dextrose infusion when the surgeon removed the first suspected insulinoma. According to Chari et al., dextrose infusions should be stopped soon after tumor removal to prevent excessive hyperglycemic response in the postoperative period.⁵ Although the blood glucose was maintained between 100-120 mg/dL before tumor resection, blood glucose monitoring was increased to every 5 minutes after tumor resection so that hyperglycemic rebound would be monitored and documented. Glucose measurements remained within the same range over the next 30 minutes. The lack of hyperglycemic rebound, as well as the failure to visually identify any additional insulin-secreting tumors, led the surgeon to perform an intraoperative ultrasound and further surgical intervention.

This case presented some unique challenges due to the patient's complex medical history and diagnosis of suspected insulinoma. It is important for the anesthesia practitioner to be aware of the various approaches to managing a patient with insulinoma. Frequent blood glucose monitoring and open

communication with the surgeon are essential elements during this operation.

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Upper Airway Surgery and Obstructive Sleep Apnea

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Keywords: Obstructive sleep apnea, uvulopalatopharyngoplasty, upper airway

surgery, continuous positive airway pressure, obesity.

Obstructive sleep apnea (OSA) affects 9% of men and 4% of women.¹ Up to 90% of the patients with OSA are undiagnosed.² A major risk factor is obesity. OSA has several systemic consequences including hypertension, stroke, myocardial infarction, pulmonary hypertension, cardiac dysrhythmias, cardiac angina, fatigue, anxiety, depression, and heart failure. Treatments range from conservative measures, including continuous positive airway pressure (CPAP), to surgical intervention for severe cases and those in which conservative measures fail.³ This case report discusses a surgical treatment option to improve airway patency in patients with OSA and anesthetic considerations.

Case Report

A 39 year old male, ASA II, Mallampati class 2, BMI 28 kg/m², 95 kg, with a diagnosis of OSA and deviated septum presented for elective surgery. The patient, unable to tolerate CPAP, elected to undergo a septoplasty for correction of a deviated septum and uvulopalatopharyngoplasty (UPPP) for correction of OSA-related obstruction. The patient reported daytime hypersomnolence and his significant other reported heavy snoring with apneic periods during the night. Allergies included amoxicillin. Current medications included venlafaxine for depression. The patient's medical history was negative for coexisting diseases with the exception of smoking cessation four years prior. The patient's laboratory values were all within normal range. The patient was transferred to the operating room. ECG, blood pressure, and pulse oximetry monitors were applied. The patient was pre-oxygenated with 100% oxygen for 5 minutes. One hundred micrograms (mcg) of fentanyl were administered intravenously (IV) followed by lidocaine 100 milligrams (mg) and propofol

200 mg. When a state of unconsciousness was achieved the patient was mask ventilated to ensure adequate ventilation prior to administration of rocuronium 50 mg IV. Once the train of four (TOF) was 0/4 twitches using a nerve stimulator on the facial nerve, direct laryngoscopy was performed. An 8.0 endotracheal tube (ETT) was placed and confirmed by auscultation and CO₂ production. Anesthesia maintenance was achieved using desflurane, titrated based on the patient's vital signs. Upon completion of the surgery, the patient had 3/4 twitches and neostigmine 3 mg and glycopyrrolate 0.6 mg were administered IV. Neuromuscular blockade was fully reversed with 4/4 on the TOF and sustained tetanus present. Extubation occurred when the patient was fully awake, able to follow commands, and protect his airway. Upon extubation, the patient was transported to the post anesthesia care unit (PACU) and placed on oxygen via nasal cannula. The patient was monitored closely for signs and symptoms of apnea, hypopnea, hypercarbia, and hypoxia. He was able to maintain and protect his airway without intervention from staff. The patient was awake, alert and oriented and denied any pain or discomfort. After 30 minutes in the PACU the patient was transferred to a post-surgical unit. The patient was monitored overnight for apnea/hypopnea related to his OSA. After an uneventful night, the patient was discharged.

Discussion

The true incidence of mortality and morbidity related to OSA and anesthesia is unknown. A recent Pennsylvania Patient Safety Advisory noted more than 250 reports of adverse events related to OSA since June of 2004. Twenty percent of the adverse events were classified as serious with three deaths occurring in ambulatory and hospital settings.⁴ A survey of

anesthesiologists in Canada found that 27% had experienced complications in patients with OSA.⁵ Numerous case reports of complications have been reported and concern has been raised by many individuals and organizations.⁶ The nurse anesthetist should approach the patient with diagnosed or suspected OSA conservatively and be prepared for the following complications: difficult intubation, postoperative apnea, hypopnea, hypoxia, hypercarbia, hypertension, and cardiac dysrhythmias.^{3,6} Obstructive sleep apnea is an obstruction of airflow to the lungs caused by upper airway collapse. Obstruction of airflow, despite continued efforts to breathe, results in hypoxemia, hypercarbia, and a decrease in pH. Activation of the central nervous system (CNS) results in the wakening of the patient and an increase in airway patency. As CNS arousal decreases, the process begins anew.^{2,3,6}

Anatomic factors associated with OSA include enlarged tonsils, retrognathia, micrognathia, macroglossia, and increased fatty tissue in the lateral walls of the pharynx. These factors decrease the anterior/posterior (AP) diameter of the pharynx, predisposing the patient to airway obstruction during sleep. Obesity is the major risk factor associated with OSA due to an increase in soft tissue and fat located around the pharynx. Other factors include smoking, the use of alcohol, and nasal congestion.^{2,4,5} Patients with sleep apnea are at an increased risk of hypertension, stroke, myocardial infarction, pulmonary hypertension, cardiac dysrhythmias, cardiac angina, fatigue, anxiety, depression, and heart failure.³

Signs and symptoms of OSA are nocturnal obstructive breathing, insomnia, and daytime hypersomnolence. Nocturnal obstructive breathing includes snoring,

snorting, gasping, choking, and/or witnessed apneic periods. Patients with OSA complain of not being able to fall asleep, stay asleep, or they wake early with the inability to fall asleep again. Patients complain of hypersomnolence during sedentary tasks such as watching television or working on a computer.^{3,6}

Diagnosis of OSA begins with a thorough interview of the patient. The patient's significant other may shed light on the patient's symptoms of OSA. The anesthetist should have a high degree of suspicion during the preoperative interview since the majority of patients with OSA are not diagnosed. If signs and symptoms of sleep apnea are present, the patient may then be referred to a sleep lab for a polysomnography. Polysomnography, the gold standard for diagnosis, is an overnight test which monitors the patient in categories such as EEG, ECG, air flow, pulse oximetry, electro-oculograms, chin-electromyograms, and respirations.⁷ If the results of the polysomnography are positive for OSA, the severity of the disease is then determined based on the number of apneas/hypopnea episodes the patient experiences during each hour of sleep.

Surgical treatments for OSA include uvulopalatopharyngoplasty (UPPP) and treatment of correctable airway-related abnormalities. UPPP is the removal of soft tissue in the back of the throat such as the uvula, the soft palate and other tissues around it. Enlarged tonsils and adenoids are often removed as well. The purpose of UPPP is to increase the AP diameter of the upper airway thereby improving airway patency during sleep. Success rates with UPPP have been shown to be approximately 40%.⁸

Patients with OSA undergoing anesthesia are at a higher risk for difficult airway management and increased episodes of apnea, especially during the recovery phase. Medications administered during the perioperative period may predispose the patient to upper airway collapse related to anesthetic medications and postoperative analgesics. In addition, disruptions in REM sleep may predispose the patient to an increased risk of perioperative apneas. During emergence the patient should be positioned with the head of the bed elevated, neuromuscular blockade should be fully reversed, and the patient should be awake, able to follow commands and able to protect their own airway prior to extubation. If the patient needs to be re-intubated, the trauma from the surgery may cause an impaired view of the trachea thus leading to a difficult intubation. Supplemental oxygen and pulse oximetry should be administered to the patient upon extubation and throughout the initial recovery period. Surgical intervention in the upper airway results in airway edema, and patients should be closely monitored during the postoperative phase. Patients on CPAP prior to surgery should utilize CPAP during periods of rest and sleep in the postoperative period, unless contraindicated by the surgical procedure. The patient should be monitored in an appropriate environment in the postoperative period.^{3,6}

Patients with OSA undergoing surgical procedures are at significant risk for mortality and morbidity. The anesthetist should be prepared for the potential of a difficult intubation, be judicious in the administration of opioids, and ensure neuromuscular blockade is fully reversed and the patient fully awake for extubation. During the recovery phase the patient with OSA should be monitored closely for respiratory depression and arrest. Patients with OSA undergoing upper airway surgical

procedures for the treatment of OSA related symptoms are at increased risk for apnea/hypopnea during the postoperative period related to airway edema and the administration of anesthetics and analgesics.

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Anesthetic Management of Multiple Sclerosis
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Keywords: multiple sclerosis, neuromuscular disease, demyelinating autoimmune disease, anesthesia, neurology

Multiple Sclerosis (MS) is a demyelinating autoimmune disease that attacks the white matter of the central nervous system. It is predominately diagnosed in adults aged 20 to 40 years, and is two times more likely to affect females than males.¹ Depending upon the severity of the disease, it can be debilitating. In the United States MS affects approximately 350,000 people and almost two million people worldwide.¹ It is likely that an anesthesia professional will encounter a patient affected by MS, due to the high prevalence of this disease.

Case Report

A 42 year old, 130 kg 177 cm, female with a history of MS presented for Achilles tendon repair. The patient's past medical history included obesity and left optic neuritis secondary to MS. Surgical history included a total abdominal hysterectomy, anterior and posterior bladder suspension and a cholecystectomy with no anesthesia complications. Medications included glatiramer. Preoperative assessment revealed ASA physical status II and her airway was classified as a Mallampati III. The neurological evaluation revealed no muscular weakness and she denied any disability specific to her MS.

In the holding area, midazolam 2 mg intravenous (IV), glycopyrrolate 0.2 mg IV and cefazolin 2 g IV were administered. Upon arrival to the operating room, standard monitors were applied and fentanyl 100 mcg IV was administered. Induction of anesthesia was performed with lidocaine 30 mg IV, propofol 250 mg IV, rocuronium 30 mg IV, and an additional fentanyl 150 mcg IV. Using direct laryngoscopy with a Macintosh 3 blade a 7.0 cuffed oral endotracheal tube was placed to 21 cm at the lips. An esophageal temperature probe was inserted and general anesthesia was maintained with 4 to 5 % desflurane, 0.5 L/min of air and 0.5 L/min of oxygen.

The prone position was utilized for surgery and an upper body warming device was applied. Once patient positioning was accomplished, morphine 10 mg IV, ondansetron 4 mg IV, famotidine 20 mg IV, and dexamethasone 10 mg IV were administered. The maintenance phase was uneventful and the train of four (TOF) was monitored via the ulnar nerve. No additional neuromuscular blockade was administered.

At the conclusion of the surgery, the TOF revealed 3 out of 4 twitches and neuromuscular blockade was antagonized with neostigmine 3 mg IV and glycopyrrolate 0.6 mg IV. The total surgery time was 64 minutes. Extubation criteria

were met, including a tidal volume of 375 ml and a sustained head lift greater than 5 seconds. The trachea was extubated without complication. Upon arrival to the PACU, pain and nausea were denied and vital signs were stable.

Discussion

Multiple sclerosis is a disease of the central nervous system which is characterized by multiple sites of demyelination and inflammation in the brain and spinal cord.¹ Research data suggests there is a genetic component linked to MS, however, the exact etiology related to the predisposition and progression of the disease is unknown.¹ It is also speculated that there is a complex series of immunological events in susceptible individuals.² A virus, such as herpes simplex or Epstein-Barr, triggers an inflammatory process that causes T-cell mediated reactions to myelin in the brain. Myelin is replaced by scar tissue leaving the axon exposed, slowing the speed of neuronal transmission. In early stages of the disease, affected nerve tissue is able to repair the myelin and symptoms can resolve.³ With disease progression, myelin is permanently destroyed, and nerve impulse transmission is slowed. During this time, muscle weakness and fatigue worsen. MS progression is characterized by relapses and remissions in about 80% of patients or primary progression in 20% of patients where each new relapse brings additional functional deficits.⁴

Clinical manifestations are varied since lesions can occur anywhere in the white matter of the brain stem and/or spinal cord. Common symptoms include visual abnormalities, difficulty walking, bowel retention, urinary incontinence and temperature intolerance. Brain stem involvement can produce diplopia, cardiac

dysrhythmias, autonomic dysfunction, and, ultimately, respiratory failure.²

The diagnosis of MS is based on a combination of clinical symptoms and diagnostic testing such as magnetic resonance imaging (MRI), lumbar puncture (LP) or somatosensory-evoked potentials (SSEPs). The most common diagnostic test is a cranial MRI which documents the presence of the demyelinated plaques in the brain and also measures the progression of the disease.⁵

To date, there is no cure for MS. Treatment involves changing immune and inflammatory responses while decreasing the patient's symptoms. Corticosteroids are the primary treatment for the inflammatory responses. In addition, immunotherapy is used to decrease the occurrence of acute attacks and the progression of MS by inhibiting the autoimmune response. Interferon beta 1-a (Avonex), interferon beta 1-b (Betaseron) and Glatiramer (Copaxone) may reduce the frequency of attacks by 30%.⁴

There are many potential drug interactions unique to the MS patient. There is an increased incidence of cardiotoxicity in this patient population who are taking immunosuppressant drugs. Also, autonomic dysfunction may exaggerate hypotensive effects of volatile anesthetics.¹ When patients are treated with corticosteroids, a stress dose of steroids is indicated immediately prior to surgery and intraoperatively.¹

The most recent medication marketed for MS in Canada is Sativex. Sativex is a cannabis extract that is administered via oromucosal spray. United States (US) clinical trials started in 2007, but the drug will not be available commercially for

approximately 3 to 4 years. Sativex contains approximately 50% ethanol and should be used cautiously in patients who are alcoholics or have liver disease. In addition, sedatives and hypnotics may enhance sedative effects of Sativex.⁶ In 2007, a study by Collin et al, revealed that Sativex significantly reduces intractable spasticity in people with MS.⁷ Anecdotal evidence suggests that Sativex is effective in reducing central neuropathic pain and is well tolerated.

Patients with MS have specific anesthetic needs. Careful preoperative and postoperative assessments should be performed to detect any new symptoms, such as visual changes or an increase in musculoskeletal weakness. The preoperative consent should state that the stresses of surgery and anesthesia may worsen the symptoms.¹ These patients may have respiratory dysfunction due to their neurological disability or a complicated airway due to the presence of cervical disk disease.⁸ Succinylcholine should not be utilized due to the abnormally high release of potassium from the extrajunctional receptors in patients with neuromuscular diseases.¹ In addition, their response to nondepolarizing muscle relaxants may be prolonged related to extrajunctional cholinergic receptors in upper motor neuron lesions, and therefore a smaller dose may be indicated.⁹

It is imperative that extubation criteria are assessed and met prior to extubation to insure full return of neuromuscular function. Temperature control is also extremely important. Demyelinated nerve fibers are sensitive to increases in temperature. A study performed by Flensher and Lindererona (2002), utilized a cooling suit to cool patients with MS. The patients reported less fatigue and an improvement of

symptoms related to decreasing their core temperature.¹⁰

There are no studies that indicate regional anesthesia is better than general anesthesia for MS patients. Data on perioperative outcomes regarding which type of anesthesia are inconclusive. According to Fleisher (2004), it is reasonable to limit the use of regional anesthesia with MS patients until more data is available to establish its benefits.¹¹

According to the National Multiple Sclerosis Society (NMSS), surgery for patients with MS will not exacerbate their symptoms nor worsen their disease. The risks of general anesthesia for patients with MS are the same as for those who do not have this disease.¹² However, surgical risks are increased in a small percentage of MS patients who are in the end stages of MS.¹² These patients are severely weakened by MS and usually have respiratory problems that place them at greater risk for anesthetic complications. Current recommendations state that elective surgery should be avoided during periods of relapse.¹² MS patients are complex and unpredictable. Anesthesia professionals must formulate a safe and efficient plan that allows for the unique considerations of MS patients.

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Perioperative Glucose Control in the Diabetic Patient

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Key Words: Diabetes Mellitus, Anesthesia, Insulin, Blood Glucose, Infection

Diabetes mellitus (DM) in the United States is attributed to an aging population, increased obesity, sedentary lifestyles and changes in diet. Estimates from the American Diabetes Association (ADA) state that 12-25% of hospitalized patients have DM and are more likely to undergo surgery than their non-diabetic counterparts.¹ Complications associated with DM include retinopathy, nephropathy, neuropathy and atherosclerosis, directly increase the need of surgical intervention and often contribute to occurrence of surgical infections and vasculopathies.² Strict glycolic control has profound effects on reducing these

complications in the diabetic surgical patient and is considered standard practice in anesthesia.^{2,3}

Case Report

A 52 year old male presented for incision and drainage of a shoulder abscess after rotator cuff repair performed three weeks earlier. His past medical history was significant for Type I DM and gastroesophageal reflux (GERD). Medications included acetaminophen/oxycodone, esomeprazole, 70 NPH/ 30 regular insulin, and gargine insulin. He reported non-compliance with home glucose monitoring and insulin administration.

Pre-operative vital signs were as follows: blood pressure 131/88 mmHg, heart rate 65 beats per minute, respiratory rate 20 breaths per minute, temperature 98.1 degrees Fahrenheit, and oxygen saturation 97% on room air. Significant lab studies included a glucose level of 373 mg/dL.

Physical exam revealed a regular rate and rhythm and lung sounds which were clear to auscultation. His ASA physical status was classified as II-E. Preoperatively, metoclopramide 10 mg and famotidine 20 mg were administered intravenously (IV). Midazolam 2 mg IV was given immediately prior to transport to the operating room.

Upon arrival to the operating room, standard monitors were applied, and oxygen per face mask at 8 L/minute was instituted. A rapid sequence induction with cricoid pressure was performed with fentanyl 100 mcg, lidocaine 100 mg, propofol 180 mg, vecuronium 1 mg and succinylcholine 80 mg. Direct visual oral intubation of the trachea was achieved with a 7.5 mm reinforced endotracheal tube. The patient was placed in a sitting position. Anesthesia was maintained with 1.6-2.5% sevoflurane. An additional 100 mcg of fentanyl and vecuronium, 6mg, was administered throughout the case. Ten units of regular insulin was given IV. A repeat blood glucose level of 324 mg/dL was obtained one hour later. Regular insulin 5 units IV was administered at that time. Cefazolin 2 g IV was infused after wound cultures were obtained.

Neuromuscular blockade was antagonized at the conclusion of the procedure and the trachea was extubated when extubation criteria were met. Estimated blood loss was 50 mL and crystalloid replacement totaled 1.6 L. The patient was transported to the recovery room on oxygen, 10 L/min per face

mask. The blood glucose level was noted to be 312 mg/dL. The patient was admitted to the general surgical floor for glucose management and IV antibiotics. One day following the procedure, the patient denied any complaints of pain, nausea or anesthetic complications.

Discussion

DM is a disease characterized by elevated blood glucose from a defect in carbohydrate metabolism secondary to an absolute or relative lack of insulin. The two main types of DM include Type 1 or insulin dependent diabetes and Type 2 or non-insulin dependent diabetes. Type 1 DM is the result of an inflammatory or autoimmune response which destroys β cells in the pancreas. Since endogenous insulin is absent, administration of exogenous insulin is essential for treatment. Type 2 DM is a result of genetic and environmental factors which lead to insulin resistance and impaired insulin function. Multi-modal treatment includes diet, exercise, drugs that stimulate endogenous insulin secretion, drugs that increase insulin sensitivity and exogenous insulin.³

Hyperglycemia and hypoglycemia are common complications associated with the diabetic surgical patient. Perioperative stress causes an increase in the secretion of epinephrine, norepinephrine, cortisol and growth hormone. These hormones cause glycogenolysis, gluconeogenesis, and protein catabolism which promotes hyperglycemia, acidosis and ketosis in patients with DM.^{2,4} Hypoglycemia can result from prolonged fasting, long acting hypoglycemic drugs and renal insufficiency. Maintaining "tight" control of a patient's blood glucose level has been shown to reduce mortality and morbidity in a variety of surgical procedures.⁵ The target range

should be less than 150 mg/dL with ideal values falling between 70-110 mg/dL.⁶

Poorly controlled DM contributes to post surgical wound infection and impaired wound healing. Infection contributes up to 66% of postoperative complications in the patient with DM and is responsible for 20% of postoperative deaths in this population.⁶ Infection and impaired wound healing is a result of impaired phagolytic activity, suppressed collagen synthesis and decreased granulocyte chemotaxis. These pathologies occur when blood glucose levels are greater than 200 mg/dL.⁷ As compensatory mechanisms are impaired in the diabetic patient, insulin supplementation is often required to minimize glucose regulation defects. The key to intraoperative glucose management is the use of a fast acting IV insulin preparation, whether in infusion or bolus form. Regular insulin can lower blood sugar levels by 10-30mg/dL for every one unit of IV insulin administered.^{3,6} It is important to note that subcutaneous insulin has limited intraoperative use as its absorption may be unreliable.^{2,6,7} Two major complications that must be considered when using IV insulin include hypoglycemia and the potential for hypokalemia if glucose levels are lowered too rapidly. It is important to implement heightened vigilance, frequent glucose measurements, and rapid dosing adjustments in glucose and insulin infusions in order to minimize or avoid hypoglycemia.³

Anesthesia professionals should be cognizant that DM produces dysfunction in most organs. Perioperative management can affect both short and long term complications. Preoperative evaluation should focus on eliciting evidence of end organ damage. This may include myocardial ischemia, renal dysfunction, polyneuropathy and joint immobility. Common problems for

diabetic patients include GERD, diarrhea, delayed gastric emptying, proteinuria, coronary artery disease (CAD), hypertension (HTN), joint stiffness or immobility, and elevated blood potassium, creatinine and urea levels.^{3,8} Preoperative testing should include an EKG, electrolyte studies, fasting blood glucose, and renal function testing in addition to other appropriate testing based on the invasiveness of the planned surgery.

While there is no apparent indication that one type of anesthetic is better for patients with diabetes, the stress response may be reduced with combinations of local or regional anesthesia and the administration of opioids.⁹ The specific agents and monitoring techniques used with anesthesia should be guided by the severity of systemic diseases, such as CAD, nephropathy and HTN. Beta adrenergic blockers and antihypertensive drugs should be readily accessible during the perioperative phase. Neuromuscular blockers that do not rely on renal function will be more rapidly eliminated in the diabetic patients with altered renal function. Limited joint mobility, resulting from abnormal deposition of collagen, requires careful positioning of the patient to prevent neuromuscular or neurovascular injuries. For this same reason, fixation of cervical vertebrae may make direct laryngoscopy and tracheal intubation difficult, and alternative airway management devices should be available.³

DM has a significant impact on metabolic changes in the perioperative period. It is becoming increasingly apparent that aggressive management of hyperglycemia, HTN and nephropathy markedly improve patient outcomes. While the type and duration of surgery influence the degree of preoperative testing and intraoperative management plan, anesthesia professionals should remain alert and vigilant to

alterations in systemic and metabolic functions caused by DM and make modifications in anesthesia care based on the best available research. Interventions as outlined above will increase the likelihood of achieving comparable rates of infection between diabetic and non-diabetic surgical patients.

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Pediatric Aspiration with Uncuffed Endotracheal Tube

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Customary practice favors the use of uncuffed endotracheal tubes (ETT) for pediatric patients undergoing general anesthesia requiring endotracheal intubation. This is in spite of the fact that the traditional use of uncuffed ETTs has been proposed as a contributor to the incidence of aspiration.¹

² Given that patients undergoing a tonsillectomy and adenoidectomy (T&A) are prone to emesis, the conventional use of uncuffed ETTs places the pediatric patient undergoing a T&A at high risk for aspiration. It is not surprising that the occurrence of anesthesia-related aspiration in the pediatric population has been reported to be two to four times greater than in adults.³ This case study will demonstrate

that using an uncuffed ETT may not protect the airway in the event of emesis.

Case Report

A two and a half year old male weighing 25 pounds (11.4 kg) presented to an outpatient surgical center for a T&A with bilateral myringotomy and insertion of tympanostomy tubes. The patient was assigned an ASA II classification for a past medical history significant for asthma and recurrent otitis media. The patient's last asthma attack was approximately seven months prior to the scheduled surgery. The patient's past surgical history included tympanostomy tube placement without surgical or anesthetic complication. Upon physical exam, the patient's lungs were auscultated and found to be bilaterally equal and clear. The parents reported that the child had no allergies and that he had had nothing to eat or drink for eight hours.

The child was taken into the operating room and standard monitors were placed. An uneventful mask induction was performed with sevoflurane and intravenous access was immediately obtained. Propofol 20 mg with lidocaine 10 mg, fentanyl 10 mcg, dexamethasone 2.25 mg and ondansetron 1.5 mg were administered intravenously. The trachea was intubated with a size 4.5 uncuffed ETT and adequate tidal volumes were maintained with positive pressure ventilation. Subsequently, respiration was controlled by a mechanical ventilator.

Anesthesia was maintained during the surgery with sevoflurane and nitrous oxide. Vital signs remained stable throughout the procedure with no decrease in oxygen saturation from baseline. Upon completion of the surgery, sevoflurane and nitrous oxide were discontinued and 100% oxygen was administered. During emergence, the patient

began to forcefully cough and gag while still in a light plane of anesthesia. While intubated and coughing the child had several bouts of opaque emesis with particulate matter. Despite suctioning of the oropharynx, emesis was noted in the lumen of the ETT. Emesis was removed from the airway by passing a suction catheter down the ETT. When the patient was fully awake, the ETT was removed.

The patient was taken to the recovery room once his condition was determined to be stable. He was crying and sitting up in bed. At this time, he had another episode of emesis. The parents were again queried regarding the child's fasting prior to surgery and they again denied allowing the child to eat or drink anything after midnight. Post-operatively the patient's vital signs remained stable (including blood oxygen saturation) and no abnormal lung sounds were audible. Because of the presumption of pulmonary aspiration, he was admitted to a nearby hospital for overnight observation and further medical workup. While hospitalized the patient was treated with antibiotics and closely monitored for pulmonary compromise over a 24-hour period. During his hospitalization a chest X-ray was performed with findings of poor inspiration and compression of both lungs. Upon discharge, his blood oxygen saturation reading was 100% on room air and a course of amoxicillin was prescribed.

Discussion

The second most common pediatric surgical procedure is a T&A.⁴ This surgical procedure is associated with risks of bleeding, airway obstruction and increased incidence of emesis leading to unplanned hospitalization.^{5,6} This type of procedure is commonly performed in outpatient surgery centers where the ability to provide intensive

post operative care and monitoring is not available.

In this case study, despite the effort to minimize the risk of emesis by using anti-emetics of proven efficacy (dexamethasone and ondansetron), the patient vomited and subsequently aspirated.^{4, 6-8} The consequences of this event included increased cost due to transfer and hospitalization, possible serious pulmonary injury and undue stress and anxiety imposed upon the patient and his parents.

Gastric decompression might have reduced the likelihood of vomiting in this case, however, this practice has not been shown to be efficacious in patients undergoing a T&A.⁹ Some authors advocate extubating the pediatric patient while he/she is in a deep plane of anesthesia.^{5, 10} Advocates of extubating while in a surgical plane of anesthesia suggest that this may eliminate bucking and gagging on the ETT during emergence and possibly reduce the risk of emesis. The alternative opinion holds that extubation after the patient is completely awake is safer because the airway reflexes are intact. In this case, the ETT was secured in place until the patient was alert; however, this did not prevent pulmonary aspiration.

There is discussion in the literature challenging the use of uncuffed ETTs in the pediatric population.^{1, 2, 11} Established medical practice dictates the use of uncuffed ETTs for children under the age of eight to ten years old. Uncuffed ETTs are believed to decrease the risk of post-intubation croup, minimize the risk of accidental barotrauma and allow for a larger internal diameter which facilitates breathing.^{1, 5} It has been argued that the ETT size and duration of intubation are better predictors of mucosal damage leading to postintubation croup than the presence of a cuff as demonstrated by

two landmark studies published over the last several decades.^{1, 12, 13} It is easy to understand that a cuffed or uncuffed ETT that is too large exerts undue pressure on the mucosa of the subglottic region and places the patient at risk for trauma potentially leading to major airway complications.¹

Disadvantages to using an uncuffed ETT include the increased risk of pulmonary aspiration due to the incomplete seal of the airway, possible need for multiple laryngoscopies secondary to improper size, leakage of anesthetic agent in the operating room and inability to provide adequate positive pressure ventilation.^{1, 2, 11, 14} In this case, it is clear that despite the presence of an appropriately-sized uncuffed ETT in the airway, the patient was not protected from aspiration.

It can be speculated that had a cuffed ETT been used, the airway would have been better protected and complications avoided. It has been proposed that the use of an appropriately sized cuffed ETT with careful monitoring of depth and cuff pressure, can reduce the risk of aspiration without increasing the risk of post extubation croup.^{1, 2, 11, 14} The use of a cuffed ETT in this situation would have provided improved airway protection and would have reduced the risk of aspiration. In retrospect, a cuffed ETT with minimal occlusive pressure may have been a better approach for administering anesthesia to a pediatric patient undergoing a procedure with a high risk of emesis.

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