

Section 1
General

1

Obesity/difficult airway

Ruchir Gupta

STEM 1

A 38 year old, 190 kg 5'6" male is scheduled for a laparoscopic gastric bypass.

PMH: Patient has DM, HTN, GERD, OSA requiring CPAP. His past medical history is also significant for hypertension, severe GERD, hiatal hernia, depression, and obstructive sleep apnea requiring CPAP.

PE: P 74 bpm, BP 130/68 mmHg, RR 14, T 36°C, 97% on room air. On physical exam, his Mallampati score is III and his cervical range of motion is normal. His dentition is intact. Auscultation reveals normal breath sounds bilaterally with regular rate and rhythm.

EKG: NSR.

Labs: Serum glucose is 153 mg/dL, otherwise his complete blood count (CBC) and basic chemistry panel are within normal limits.

Preoperative

Obesity

1. How is body mass index (BMI) calculated?

BMI is calculated by dividing the weight in kg by the height in meters squared (kg/m²).

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Obstructive sleep apnea

2. How is obstructive sleep apnea (OSA) different from obstructive sleep hypopnea (OSH)?

OSA is cessation of airflow for >10 seconds, five or more times per hour of sleep. There is a >4% decrease in O₂ while the patient is asleep. By contrast, in obstructive sleep hypopnea (OSH) there is a decrease in airflow of >50% for more than 10 seconds, 15 or more times per hour of sleep. The decrease in O₂ saturation during these episodes is also >4%.

3. How is the definitive diagnosis of OSA made?

Although OSA may be suspected by history, physical examination, and/ or comorbidities, a definitive diagnosis requires a formal sleep study.

4. What are the systemic manifestations of OSA?

OSA affects multiple organ systems:

Cardiac – patients usually have hypertension and LVH. Pulmonary HTN may also be present.

Pulmonary – increased V/Q mismatch from decreased FRC and atelectasis.

GI – patients often have the stomach displaced upward due to extra-abdominal pressure placing them at risk for GERD.

Renal – the patient may have hypertensive nephropathy.

Neuro – hypersomnolence is frequently observed with increased sensitivity to anesthetic agents.

5. How is OSA distinguished from the Pickwickian syndrome?

OSA is defined as cessation of airflow for >10 seconds, five or more times per hour of sleep. Pickwickian syndrome (PS) is characterized by chronic hypoventilation which is worse during sleep, resulting in an elevation in PaCO₂ levels. Indeed, the diagnosis of PS is BMI >30, PaCO₂ >44 mmHg and no alternate explanation for hypoventilation. Patients with PS often have coexisting OSA, but not every patient with one disease will necessarily have the other. Patients with PS frequently develop sequelae from the CO₂ retention: polycythemia, cor pulmonale, and somnolence.

6. How does this patient's history of OSA affect your anesthetic management?

Preoperatively, a thorough H&P must be done to assess for any comorbid conditions. I would use sedative drugs cautiously because of increased sensitivity in OSA patients to CNS depressants such as midazolam. A thorough airway exam must also be done because most of these patients are difficult airways. Intraoperatively, care must be taken to preoxygenate these patients thoroughly as they are prone to rapid desaturation secondary to low FRC. I would also have the difficult airway cart in the room and consider an awake intubation if the airway was nonreassuring. During the course of the surgery, I would use a multimodal pain management regimen including IV NSAIDs, IV Tylenol, rectus sheath block, and local wound infiltration to decrease my narcotic use. Postoperatively, I will extubate in the head-up position to improve pulmonary

mechanics and extubate only after the patient is fully awake. I will also have a CPAP machine ready in the PACU and continue to monitor the patient closely in the PACU for any episodes of apnea and desaturation.

Intraoperative

Monitors

1. What monitors will you use for this patient?

I would use the standard ASA monitors (pulse oximeter, noninvasive blood pressure measurements, a five-lead EKG, a temperature probe, and a capnograph), a neuromuscular blockade monitor, and if PIV access is difficult, I will place a central line. I will also ask for a Foley catheter to be placed.

2. The nurse informs you that she has a large size BP cuff. What will you do?

I could attempt to place a standard size BP cuff on the forearm or the lower leg. I could also place a large size cuff or alternatively, an arterial line.

Premedication

3. What premedication if any will you give this patient?

To avoid possible respiratory depression in morbidly obese patients with OSA, premedication is used sparingly, if at all. Thus, if the patient was anxious, I would start by reassuring the patient. If the patient was still anxious, then after monitors have been placed in the operating room, I would administer a low dose of midazolam and/or ketamine. Due to risk of aspiration, I would administer sodium citrate to decrease stomach acidity, and promotility agents such as metoclopramide to enhance gastric emptying.

Intubation

4. How would you intubate this patient?

Based on information provided, management of the patient's airway may be difficult and so after preoxygenating the patient with 100% oxygen, I would perform an awake oral fiberoptic intubation. I would topicalize the airway with 1–2% lidocaine spray or nebulizer and perform a superior laryngeal nerve block by injecting 2 mL of 2% lidocaine, just anterior to the cornu of the hyoid bones, and a transtracheal recurrent laryngeal nerve block.

5. Your resident accidentally administers a bolus of propofol to the patient to prevent him from moving during the AFOI. A few seconds later, the patient becomes apneic and begins to desaturate. What will you do?

I will immediately call for help and perform mask ventilation in an attempt to improve this patient's oxygenation while attempting to awaken the patient.

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6. **You mask ventilate the patient and obtain some EtCO₂, but the patient continues to desaturate, with an oxygen saturation of 90% and decreasing. Mask ventilation is getting progressively more difficult. What will you do?**

I will again call for help if it has not yet arrived and perform direct laryngoscopy. If that did not work or I am unable to mask ventilate the patient, I would place an LMA and ventilate through that device.

7. **You successfully place an LMA and the desaturation improves. Should you cancel the case once the patient awakens?**

It depends on the status of the patient. If there is a great deal of oral swelling and bleeding secondary to my ventilation and direct laryngoscopy attempts, I would delay this case for a few days until the swelling has subsided. If, however, the oropharynx seemed uninjured and there were no signs of bleeding or swelling, I would reattempt my awake fiberoptic and proceed as scheduled.

Maintenance of anesthesia

8. **The airway is now secured with an ETT. What agents will you use to maintain anesthesia?**

I would choose short-acting agents to reduce the risk of postoperative sedation. Desflurane is desirable because it is relatively insoluble in fat, has a fast wake-up, faster return of airway reflexes, and decreased amount of hepatic metabolism. For muscle relaxation, I will choose cisatracurium or rocuronium.

Postoperative

Extubation

1. **Would you extubate this patient? If so, how?**

Assuming there were no adverse intraoperative events and significant volume shifts, then I would want to extubate this patient immediately at the end of the procedure. I would have emergency airway equipment on standby, place the patient in the sitting position to optimize pulmonary mechanics, ensure the patient has been adequately reversed, and have the patient spontaneously breathing with adequate tidal volumes and a normal respiratory rate. Once the patient is responding to commands and able to protect his airway, I will extubate.

2. **What respiratory parameters can be used for extubation?**

Respiratory rates between 10 and 30 breaths per minute, SaO₂ greater than 95% on FiO₂ of 0.4, vital capacity greater than 10 mL/kg of IBW, and tidal volume greater than 5 mL/kg of IBW.

Hyperglycemia

3. **The nurse in the PACU pages you to tell you the patient's blood glucose is 253 mg/dL. Why is controlling blood glucose in the perioperative period important?**

The consequences of acute hyperglycemia include impaired immune response with increased risk of infection, impaired wound healing, dehydration, and electrolyte disturbances secondary to the osmotic diuretic effect of high serum glucose levels. This diabetic patient, who takes oral hypoglycemic medication, is at risk for developing potentially fatal nonketotic hyperosmolar coma.

Pain control

4. **What is your plan for postoperative pain management in this patient?**

I would provide this patient with non-opioid analgesics such as Tylenol and NSAIDs. I would also have asked the surgeon to place local anesthesia around the incisions intraoperatively. In addition, for pain refractory to these medications, a patient-controlled analgesia (PCA) is helpful in providing relief without risking overdosing since the patient must be awake and alert to administer the medicine. Due to the presence of OSA I would place this patient in a monitored setting overnight.

5. **A colleague suggests the use of patient-controlled epidural analgesia (PCEA), instead of PCA. What are some benefits of PCEA, in comparison to PCA?**

The benefit of PCEA over PCA is that less opioid medication is used because local anesthesia can also be delivered epidurally. The lower opioid requirement results in fewer side effects such as respiratory complications while also ensuring effective analgesia.

Mental status change

6. **A few hours later, the PACU nurse informs you that the patient is "disoriented." What is your differential diagnosis?**

My differential includes:

- (1) Unstable vital signs: hypoxia, hypotension, malignant arrhythmia.
- (2) Anesthesia-induced causes: residual anesthesia, narcotic overdose or reaction.
- (3) Possible delirium tremens from withdrawal from a previously unknown substance abuse agent.
- (4) Potential metabolic and endocrinologic causes: hyponatremia, hypokalemia, hypocalcemia, hypoglycemia, hyperglycemia, hypothermia, hypothyroidism, and Addison's disease, among others.
- (5) Neurologic causes include post-ictal state, cerebral edema, and stroke.

7. **How will you respond?**

First, I will ensure that the patient is adequately oxygenating and ventilating and the vital signs are stable. Next, I will perform a focused history and physical.

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From the history, I will review the anesthetic record as well as the nurse's notes to see what medications the patient has received, when the symptoms began, have they been getting worse or better, and the severity of the disorientation. From the physical, I will listen for breath sounds and look for neurologic signs, including pupillary size and any focal neurologic deficits. Finally, I will order STAT labs including ABG, electrolytes with glucose, and a CBC to evaluate for anemia. If a stroke is suspected, a STAT head CT and neurology consult would be obtained.

FURTHER READING

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Myasthenia gravis

Shimon Frankel

STEM 2

A 54 year old female presents for laparoscopic resection of the colon. Patient has a history of colon cancer and myasthenia gravis for which she is on pyridostigmine and prednisone. VS: BP 121/55 mmHg, RR 12, HR 64 bpm, room air saturation 99%. Hb 11.4 g/dL.

Preoperative

Assessment of severity

- 1. How would you assess if this patient is optimized for surgery?**
I would obtain a focused history and physical. From the history, I want to know if her disease is limited to ocular muscles, or if there is involvement of her extremities or respiratory/laryngeal muscles as evidenced by trouble chewing, swallowing, or talking. I would also want to know if the patient ever had surgery before, any prolonged intubations or problems with anesthesia, any episodes of myasthenic crisis, and whether her symptoms have been stable over the past few weeks. From the physical, I would assess bilateral motor strength in all of her extremities, and see if she displays any signs of pharyngeal or respiratory muscle involvement.
- 2. What is myasthenia gravis (MG)? What other medical conditions is MG closely associated with?**
MG is an autoimmune disorder involving antibodies to the alpha-subunit of the postsynaptic nicotinic acetylcholine receptor at the neuromuscular junction leading to a decreased number of functional receptors. With repeated

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stimulation and decreased acetylcholine release, fatigue occurs. The extent of involvement can be limited to ocular muscles, but muscles of respiration and swallowing can also be involved, causing respiratory distress or aspiration.

MG is occasionally associated with thymus hyperplasia, thymomas, or autoimmune diseases such as thyroid disease, pernicious anemia, rheumatoid arthritis, and women with certain HLA types.

3. Would you obtain pulmonary function tests (PFTs)? Why? Why not?

I would not obtain PFTs in this patient unless there was a severe respiratory component as evidenced by my H&P. If the patient had severe respiratory symptoms, negative inspiratory force (NIF) and forced vital capacity (FVC) could be used as a reference to determine optimal conditions for extubation or the need for postoperative mechanical ventilation. They may also help in determining appropriateness for ambulatory surgery. If the patient has a thymoma, flow volume loops (FVLs) can demonstrate the extent of impairment and whether it is fixed or dynamic.

Medication for MG

4. Should the patient continue the morning dose of pyridostigmine on the day of surgery? Why?

Although there is no clear-cut answer, I would recommend continuing the pyridostigmine in order to avoid any possible respiratory difficulties prior to surgery. The disadvantage of continuing cholinesterase inhibition is the possibility of prolonged motor block with succinylcholine, and the possibility of developing a cholinergic crisis.

5. Why is this patient on corticosteroids?

MG is an autoimmune disease. Corticosteroids suppress the immune system and attenuate the production of these abnormal autoantibodies to the acetylcholine receptors at the motor endplate. Treatment of MG usually begins with cholinesterase inhibitors. For more advanced disease, corticosteroids and thymectomy are used. In more severe cases, immunosuppressants (such as azathioprine or cyclosporine) and plasmapheresis are used. IVIG is used for myasthenic crisis.

6. Would you administer preoperative steroids? Why? Why not?

Yes, I would administer a stress dose of steroids, hydrocortisone 100 mg every 8 hours on the day of surgery and then taper it postoperatively. Patients taking chronic glucocorticoids may have suppressed adrenal function. Thus I would continue her chronic dose perioperatively and be alert for signs of adrenal insufficiency.

7. Would you sedate this patient preoperatively?

If there is a question as to her respiratory reserve I would avoid sedative premedication. If the symptoms are primarily ocular, a small dose of a

benzodiazepine would be acceptable, especially since emotional stress could exacerbate myasthenia.

8. Is there anything you would tell this patient about her postoperative course?

I would inform the patient and family that symptoms may worsen perioperatively, but will usually return to baseline. I would also mention the possibility of postoperative mechanical ventilation.

Lambert-Eaton syndrome

9. What is Lambert-Eaton myasthenic syndrome (LEMS)?

This is a disease of motor weakness that is often characterized by proximal limb weakness, especially of the lower extremities. It is caused by autoantibodies to the voltage-gated calcium channels of the presynaptic membrane. Calcium is not able to enter the nerve ending and allow acetylcholine release. Weakness typically improves with repetitive use as this enables buildup of calcium allowing for acetylcholine release. Patients are sensitive to both depolarizing and nondepolarizing muscle relaxants and patients may need postoperative respiratory support.

LEMS is often seen as a paraneoplastic syndrome related to small cell cancer of the lung. It may also be seen with sarcoidosis, other malignancies, thyroiditis, and collagen vascular diseases.

Intraoperative

Anesthesia

1. Would you select a general or a regional technique for this patient?

Assuming there are no contraindications, I would prefer a regional anesthetic technique for this case because it avoids muscle relaxation, instrumenting of the airway, and the need for postoperative ventilator support. Patients with MG are usually more resistant to succinylcholine and more sensitive to nondepolarizing muscle relaxants.

2. If you decide on a regional technique, would you select a spinal or an epidural approach?

I would select a continuous epidural approach because it would allow me to run a constant infusion if the operation takes longer than was originally planned. Additionally, it allows me to administer local anesthetics and narcotics postoperatively for pain.

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Induction

3. How will you induce general anesthesia in this patient if she refuses a regional technique?

Assuming the patient has a normal airway, I will preoxygenate with 100% oxygen and perform a slow controlled induction with short-acting agents such as remifentanyl, lidocaine, and propofol.

4. The surgeon says he will need muscle relaxation for the case. How do you respond?

I would try to avoid muscle relaxants if possible because even patients with minimal disease may have increased sensitivity to NDMR. Additionally, inhalational agents often provide adequate muscle relaxation in myasthenic patients to perform surgery. If needed, I would use intermediate-acting muscle relaxants in increments of 0.1–0.2 times the ED₉₅.

5. How would your induction technique change if the patient were at high risk of aspiration?

I would perform a rapid sequence intubation using succinylcholine at a dose of 1.5–2 mg/kg. Patients with MG are resistant to succinylcholine, but at higher doses it should provide rapid intubating conditions.

Muscle relaxation

6. Would you use a nerve stimulator during the case? Why? Why not?

Yes I would but I would bear in mind that it may not be reliable in myasthenic patients because the distribution of muscle weakness is often uneven and many patients may exhibit fade even in the preoperative period.

7. What is the impact of preoperative cholinesterase inhibitor administration on neuromuscular blockade and reversal?

Preoperative pyridostigmine inhibits plasma cholinesterase and succinylcholine may cause a prolonged block. Reversal of residual nondepolarizing neuromuscular blockade at the end of surgery may be unsuccessful because acetylcholinesterase is already maximally inhibited.

Postoperative

Postoperative weakness

1. Are you going to extubate this patient at the end of the case?

It depends on how the case went and whether the patient is meeting extubation criteria. I would not extubate if more than minimal doses of nondepolarizing muscle blocking agents or narcotics were used since the patient may not recover respiratory function immediately.