

ANESTHESIOLOGY™ 2014

OCTOBER 11-15 | NEW ORLEANS, LA

Session: L146

Session: L169

Cardiopulmonary Collapse During Laparoscopic Nissen Fundoplication

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Disclosures: This presenter has no financial relationships with commercial interests

Stem Case and Key Questions Content

A 46-year-old, 158 cm- 200 lb female is scheduled for laparoscopic guided paraesophageal sliding hiatal hernia repair under general endotracheal anesthesia. She has 20-pack-year history of smoking, gastroesophageal reflux disease, HTN and diabetes. Preoperative vital signs, labs and EKG are within normal limits. She has a short thick neck with MP class III airway.

Key Questions:

1. What are some of the advantages of laparoscopic surgery over open procedures?
2. What is your anesthetic plan with regard to induction and maintenance of anesthesia? Should nitrous oxide be used in this case?
3. How would you manage postoperative pain?

Case continuation:

After induction, anesthesia is maintained with sevoflurane, air and oxygen (FiO₂ = 50). The patient is placed on the volume control ventilation (RR 10; TV 600; peak airway pressure of 20 mmHg). Patient's arms were tucked at the side prior to prepping and draping. Thereafter the abdomen was insufflated with CO₂. Up until the time of insufflation, the patient exhibited stable vital signs (BP 120/70 mm Hg, and HR 72 bpm). After abdominal insufflation, the patient's heart rate abruptly dropped to 20-30 bpm. This is followed almost instantaneously by systole.

1. What do you think is going on?
2. What should we do?

Case continues:

These changes in vital signs are transient. After discussion with the surgeon, the decision is made to proceed with surgery. The patient is placed in extreme reverse Trendelenberg position. The case proceeds uneventfully for 45 minutes. In response to an elevated CO₂, respiratory rate is increased to 12 breaths per minute. This is associated with a decrease in EtCO₂ from 46 mmHg to 35 mmHg. In spite of this change in ventilatory rate, EtCO₂ continues to increase, thus necessitating further increases in rate.

1. What should you do?
2. What do you think is going on?
3. What is the cause of rapid increase in the EtCO₂ in this case?

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Case continues:

On physical examination, the patient's face and neck appear swollen. Thereafter, rapidly expanding subcutaneous emphysema is noted in the face and neck.

1. What is the mechanism of development of subcutaneous emphysema in this clinical setting?
2. What else you should look for or worry about with this presentation?

Case continues:

In response to an end tidal CO₂ of 50 mmHg, the respiratory rate is increased to 14 breaths/minute. After an additional hour, with the surgeon dissecting very close to the diaphragm, the patients BP abruptly goes from 120/60 mmHg to 60/40, EtCO₂ goes from 55 mmHg to 30 mmHg, and Peak airway pressure increases from 24 cmH₂O to 60 cmH₂O. Within 2 minutes after this sequence of events, Oxygen saturation begins to drop. Hand ventilation is begun.

1. What is the first thing you will do?
2. What is your differential diagnosis?

Case continues:

Surgeon follows your advice while the hemodynamics respond to your interventions & are quickly restored back to normal, EtCO₂ and oxygenation improve. Now, the surgeon wants to know what is going on. Is this because patient is not tolerating the pneumoperitoneum? Is there anything he can do to help? He states this patient will really benefit from laparoscopic approach as compared to open Nissen Fundoplication.

1. Will the tube thoracotomy/ chest tube help?
2. Can we make some ventilatory changes to prevent this from happening again?
3. Would you consider PEEP?
4. How about Changing Insufflation pressure?
5. What about I: E ratio? Would changing the minute ventilation help?
6. Is the application of PEEP will be of any help?

Model Discussion Content

Laparoscopic cholecystectomy was first described in human beings in 1985, since then it has revolutionized general surgery. The overall adverse event rate reported with open procedure has been 22.4% with a mortality rate of 1.95%, a rate which could be much higher among elderly. It is well established that open procedures produce significant derangement of pulmonary mechanics, independent of the effects of general anesthesia. Both Vital capacity and functional residual capacity are significantly reduced in the immediate post-operative period with gradual restoration during the second to third post-operative days. These effects are primarily attributed to incisional pain and reflex diaphragmatic dysfunction. Laparoscopic approach has emerged as a popular alternative to traditional abdominal surgery because it combines the benefits of shorter post-operative course, rapid return to normal activities, significantly less post-operative pain and ileus. Various studies have reported significant improvement in forced vital capacity (FVC), forced expiratory volume in one second (FEV₁) and forced expiratory flow (FEV) among patients undergoing laparoscopic surgical procedures.

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Physiological Changes during Laparoscopic Surgery

Trocar insertion: can cause bleeding from injury to abdominal wall vessels, perforation of the hollow viscus and injury to other intra-abdominal organs.

Positioning: Even though head up position improves diaphragmatic and respiratory function, the pooling of blood away from the heart may cause significant hypotension due to the decrease in the preload. Pneumoperitoneum related changes: Modern laparoscopic surgery employs variable flow generators that automatically terminate flow when the preset pressure (usually 12-15 mm Hg) is reached. Several authors have noted no significant change in the cardiac output (CO) despite an increase in mean arterial pressure (MAP) and end-tidal carbon dioxide (EtCO₂) during CO₂ pneumoperitoneum. However, if the intra-abdominal pressure is increased further (upto a maximum of 25 mmHg) then the airway pressure, the blood pressure, intra- thoracic pressure, central venous pressure, heart rate all tend to increase along with an increase in the EtCO₂. Actually, a moderate increase in IAP may increase CO and cardiac filling. On the other hand further increases in IAP are usually associated with significant decreases in CO and cardiac filling, an effect which can be significantly pronounced in reverse trendelenburg position. Overall, it appears that increases in IAP may have two opposite effects. Blood is forced out of the abdomen and inferior vena cava into the central circulation while there is an increased tendency for blood to pool peripherally (thereby decreasing preload). Further, initiation of insufflation has been associated with bradycardia and asystole due probably to intense stimulation and parasympathetic outflow.

Respiratory effects: The potential effects of pneumoperitoneum induced hypercarbia, acidosis and hypoxia may be encountered under general anesthesia, which can be especially pronounced among patients with preexisting respiratory diseases and is caused primarily by a decrease in functional residual capacity (FRC) secondary to increase in IAP. The decrease in FRC may not return to normal for up to 2-3 days. This effect may further get accentuated by translocation of abdominal CO₂ in to the pleural cavity either through a congenital defect in the diaphragm or due to inadvertent creation of pleuro-peritoneal communication. Yet another effect that must be kept in mind is that increases in IAP during laparoscopy may predispose to regurgitation of gastric contents.

Carbon dioxide: Since peritoneal insufflation is required to facilitate exposure and visualization of the abdominal contents and surgery, CO₂ turns out to be the ideal gas because of its ready availability, low cost, efficacy and high permeability (easy removal). The CO₂ is readily absorbed in to systemic circulation due to its high permeability, from where it can be readily removed by hyperventilation. Its high solubility is in part due to rapid buffering capacity of the blood. Further the risk of CO₂ embolization causing significant hemodynamic instability is greatly reduced due to its high solubility and easy removability from the lungs by hyperventilation. An acute increase in the PaCO₂ in the presence of inhalation agents can predispose the patients to cardiac arrhythmias. Studies have shown that the PaCO₂ level is highest after the completion of the procedure. Hypercarbia can increase plasma catecholamine levels that can lead to significant increases in patients' systemic blood pressure, heart rate and cardiac output. It is also evident

that CO₂ dissecting in to the tissue spaces gets absorbed at much higher rate leading to precipitous elevations in EtCO₂ as compared to when it is localized in the abdominal cavity under positive pressure.

Nitrous Oxide: Use of N₂O remains controversial because of the concerns about its ability to produce bowel distension although this issue has not been studied. In spite of similar diffusion capacities of the two gases (N₂O and CO₂), N₂O is more likely to cause significant distension of air-filled closed spaces since the solubility of CO₂ far exceeds that of N₂O. With regard to the concern regarding nausea and vomiting, studies have not been able to show significant difference between the two groups.

Cardiopulmonary complications of laparoscopic procedures: Pneumothorax (capnothorax) is an infrequent complication of laparoscopic surgery of the upper abdomen especially during Nissen Fundoplication due to the dissection involving structures close to pleuro-peritoneal junction near the diaphragm. Most pneumothoracis are of little consequence since the CO₂ is highly dissolvable gas and is readily removed with hyperventilation of the patient though the lungs. However, under certain circumstances when the rate of accumulation of CO₂ pneumothorax exceeds the rate of removal, cardiopulmonary collapse due to tension pneumothorax can develop. The diagnosis is essentially clinical since in most cases the condition has been reported to develop precipitously. The combination of increase in airway pressures (caused by decrease in pulmonary compliance), decreased or absent breath sounds on the affected side, sudden persistent hypotension, ensuing hypoxemia and a drop in EtCO₂ with widening of the A-a gradient for CO₂ are suggestive of the developing capnothorax. Examination of the chest may reveal presence of significant subcutaneous emphysema. The drop in EtCO₂ that accompanies tension pneumothorax usually follows a period of significant increases in EtCO₂ caused by brisk absorption of CO₂ from the subcutaneous and pleural spaces. The shape of capnogram usually remains unchanged. The drop in EtCO₂ can be due to a number of other causes e.g. development of auto- PEEP due to need for hyperventilation to maintain normocarbica in susceptible patients, metabolic derangements, pulmonary air/CO₂ embolism, pulmonary thrombo-embolism or myocardial ischemia/infarction and most importantly precipitous drop in CO. Interestingly, none of the above mentioned causes of sudden drop in EtCO₂ are usually accompanied with high ventilatory pressures. Emergent bronchoscopy can quickly rule out problems with endotracheal tube mal-positioning whereas urgent intraoperative chest radiography will usually confirm the diagnosis. Once the diagnosis is established, multiple treatment options have been utilized to treat the condition. Application of positive end expiratory pressure has been recommended to decrease the incidence and extent of clinically significant pneumothorax. Once the tension pneumothorax is suspected, immediate discontinuation of surgery with desufflation of the pneumoperitoneum should be performed. This in itself may help with the airway pressure and may make ventilation of the patient easier. Drop in EtCO₂ is usually indicative of significant drop in cardiac output, and may necessitate institution of fluid boluses and vasopressor therapy to maintain perfusion pressure and CO. In the mean time, hyperventilation of the patient may help with removal of CO₂ thereby reducing the size of capnothorax. In extreme situations a tube thoracostomy or transdiaphragmatic decompression

of the pleural cavity (on the left side) may become necessary with immediate resolution of the problem. Once the condition of the patient has stabilized, how to accomplish rest of the procedure needs to be addressed with the surgical team. The capnothorax is caused by the movement of CO₂ from the abdominal cavity into the chest along the peritoneo- pleural pressure gradient. Since the gradient exists only during late and end-expiratory phase of the positive- pressure-ventilation cycle, it has been suggested that maneuvers aimed at minimizing this gradient could prevent accumulation of the capnothorax. This is accomplished by application of PEEP while simultaneously decreasing the CO₂ insufflation pressure to counter the peritoneo-pleural pressure gradient of CO₂. In addition, increasing respiratory rate and decreasing the expiratory time by altering the inspiratory to expiratory time ratio has the dual effect. On one hand it decreases the time duration for which the peritoneo-pleural gradient is positive, while on the other hand it aids in the removal of absorbed CO₂ from the lungs through hyperventilation. Since the benefits of laparoscopic surgery are clearly established, every effort should be made to accomplish the procedure laparoscopically.

CO₂ Embolism and its Treatment: CO₂ embolism occurs during the insufflation of the abdomen when inadvertently, a large amount of CO₂ is directly injected in to a large vessel. This usually happens during blind insufflation through the Veress needle and is usually suspected if the abdominal cavity does not distend uniformly. If significant volume of CO₂ embolizes then hypotension, hypoxia and cardiac arrest can develop. Sudden decrease in EtCO₂, hypotension and cyanosis are early indicators and the diagnosis is usually confirmed with pericardial/transesophageal or transesophageal echocardiography. In addition, aspiration of foamy blood from the central catheter confirms gas embolism. Since CO₂ is an extremely soluble gas in blood, its embolism is far less life threatening as compared with air embolism. Lethal volume of CO₂ embolism is about 20-30 times that of air. Once suspected, therapy includes support of hemodynamics with fluids, pressors and if time permits- pulmonary artery catheter. Abdominal insufflation should be immediately discontinued, FiO₂ should be increased to 1.0, patient should be hyperventilated, PEEP is applied to minimize air entrapment and the patient is placed in steep left lateral decubitus trendelenburg position. Also a central venous catheter is helpful in aspirating air from the right heart.

Bradycardia/ Cardiac Asystole: Sudden precipitous bradycardia and asystole has been sporadically reported with abdominal insufflation during laparoscopic procedures. Cause is not entirely clear, however it is suggested that it is caused by intense stimulation of vagal activity with sudden and brisk abdominal distension and pressurization. Close vigilance and prompt intervention with administration of atropine and epinephrine usually suffices.

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