Anesthetic considerations for esophageal perforation

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The author reviews the anatomy of the esophagus and discusses the pathophysiology of non-instrumental esophageal perforation. To illustrate the anesthetic considerations necessary for esophageal surgery, he includes a case study.

To the anesthesia practitioner, the esophagus is the source of one of the most feared complications of anesthesia—gastric aspiration. However, this muscular conduit is also one of the anesthetist's most useful monitors of heartbeat, respirations and temperature via specialized probes.

The esophagus is often taken for granted until a pathological process compromises its function. Esophageal perforation is perhaps the most serious pathophysiologic condition that can occur, and is one that requires immediate surgical intervention. This condition presents a serious challenge to the anesthetist.

Anatomy

By the fourth week of gestation, the esophagus of the fetus is discernible as a definite conduit between the pharynx and stomach. By the eighth week, the esophagus and trachea are sufficiently developed to be recognized as two separate structures.

The esophagus is a hollow muscular conduit that extends from the pharynx at the level of cervical six through the posterior mediastinum and diaphragm to the stomach. In the neck, it lies in the midline but as it enters the thorax, it deviates slightly to the left and descends behind the left mainstem bronchus. As it extends from the neck to the bifurcation of the trachea, it lies between the vertebræ and the trachea. At this level, the esophagus is indented by the arch of the aorta on the left side; and on the right, it is covered by parietal pleura.

Below the tracheal bifurcation, the esophagus pitches slightly to the right of the midline and maintains contact on the left with the descending thoracic aorta and anteriorly with the left atrium. Just prior to entering the diaphragm, it deviates again behind the pericardial sac. It extends across the esophageal hiatus of the diaphragm, and lies anterior to the aorta in contact with the left parietal pleura.

The esophagus narrows in three distinct areas: at its origin, at the level of the arch of the aorta and left bronchus, and at the distal end as it approaches the diaphragm. The cricopharyngeal narrowing is usually the narrowest point in the gastrointestinal tract, a fact of considerable teleologic importance.

The entire length of the esophagus is lined with an aqueous epithelium containing superficial and deep mucous glands. There are two layers to the muscle coat, an inner circular layer and an outer longitudinal layer. In the cervical portion, the musculature is primarily striated and
voluntary, while the thoracic portion is a mixture of voluntary and smooth muscle. The lower third of the esophagus is composed entirely of smooth muscle.

The blood supply to the cervical portion of the esophagus comes from the inferior thyroid artery, and from branches of the aorta in the thoracic portion. Innervation is via both sympathetic and parasympathetic nervous systems. Parasympathetic connections are via the vagi, and sympathetic connections are via the branches from the celiac plexus and mediastinal branches of the thoracic sympathetic chain.

Pathophysiology

In 1724, Boerhaave was credited with the first description of postemetic rupture of the esophagus.5 His classic report, a consequence of gastrogenic overindulgence, preceded reports of the successful management of such cases by more than two centuries.6

The causes of non-instrumental perforation of the esophagus are numerous, and include coughing, blunt abdominal trauma, defecation, exposure to compressed air, labor of childbirth, convulsions, vomiting and even normal swallowing.7 Causes also include postsurgical procedures such as pneumonectomy, vagotomy and diaphragmatic hernia repair. Advanced age and the presence of specific diseases are the major causes for weakening of the structure of the esophagus.8

Once perforation has occurred, the negative intrathoracic pressure from normal breathing acts as a suction device to deliver gastric contents (including bile, gastric juice, anaerobic organisms, and partly digested food) into the thorax. These substances initiate a virulent necrotizing infection, thus compromising the function of the heart and lungs.

Clinical symptoms include fever, respiratory distress (indicating pleural involvement), severe dyspnea or systemic arterial desaturation, pneumothorax and low cardiac output. A chest x-ray may indicate right or left sided white-out depending on the site of perforation.

Post-emetic perforations usually involve the distal portion of the esophagus. These major linear tears require immediate surgical intervention. Mortality rates increase significantly, up to 80%, when time elapsed after perforation exceeds 24 hours.

Anesthetic considerations

The patient with a perforated esophagus can present with septic or hypovolemic shock, alveolar hypoventilation and consequently, respiratory acidosis. Serial determination of blood pressure, pulse, temperature, as well as measurement of central venous pressure (CVP), urinary output and arterial blood gas tensions are essential. Baseline electrolytes and liver enzyme measurements are necessary to determine the patient's metabolic acid-base balance, as well as to determine whether liver disease could have contributed to the esophageal weakening and subsequent perforation under increased pressure. In addition, the administration of broad-spectrum antibiotics and the correction of fluid and electrolyte imbalances prior to induction of anesthesia are very important.

The induction/intubation of the patient is perhaps the most critical segment of the anesthetic management. It must be performed swiftly and with minimal stimulation so as to prevent further retching, vomiting and possible enlargement of the perforation. Consequently, an awake intubation may be hazardous for these very reasons.

The utilization of drugs which minimize cardiac depression is necessary to prepare the patient for intubation because of the possibility of hypovolemic or septic shock. Large doses of Pentothal® (thiopental), 3-4 mg/kg, may be too risky. The safest course of action is to administer ketamine or incremental doses of fentanyl until the patient is nearly unresponsive. Then, with close supervision of the respirations, a paralyzing dose of Anectine® (succinylcholine) is administered, and the patient is intubated. It is also extremely important to give the patient a defasciculating dose of curare so as to minimize the increase in gastric pressure associated with the administration of Anectine®.

Because of low arterial oxygen partial pressures and increased body temperature, the choice of a maintenance anesthetic is limited to one in which 100% oxygen can be administered. Halothane or Ethrane® (enflurane) with 100% oxygen can be utilized; if blood chemistries indicate the possibility of liver disease, enflurane may be the anesthetic agent of choice.

If the cardiovascular system is labile, a low dosage of an inhalational agent, .1-.5%, supplemented with fentanyl may be warranted. Incremental doses of ketamine may also be indicated if severe cardiac compromise has occurred. Fluid requirements can be assessed through CVP measurement, urine output, and vital signs. A deterioration in arterial oxygen saturation may indicate fluid sequestering in the alveoli from fluid overload.

544
The insertion of an arterial line is of paramount importance to determine the adequacy of ventilation and oxygenation during anesthesia, and also to serve as a source for serial arterial blood gas (ABG) measurements. Postoperative ventilation is necessary because of the thoracic approach used to accomplish the repair and because of the unilateral lung white-out present. Therefore, a nasotracheal tube is inserted in place of the orotracheal tube because of the likelihood of long-term postoperative ventilation. (Nasotracheal intubation is tolerated better during long term mechanical ventilation. It also allows for more efficient oral care during the postoperative period.)

Two of the most serious postoperative complications include a leaking of the anastomosis and mediastinal infection. A nasogastric tube is inserted during surgery to aid in healing the anastomosis and to prevent the build-up of gastric pressure.

Once the initial surgical intervention and anesthesia has been completed, the anesthetist's next challenge is weaning the patient off mechanical ventilation, a process that may require days or weeks.

The following is a case study that illustrates these anesthetic considerations.

**Case study**

The patient, a 53-year-old woman, 5'3"., weighing 60 kg, was admitted to the emergency room complaining of knife-like chest pain and dyspnea. Upon questioning, she stated that the onset of pain occurred simultaneously with an episode of vomiting. Increasing dyspnea and chest pain had brought her to the emergency room 10 hours after the vomiting episode.

The ECG indicated no abnormalities. Blood work was as follows: Hct. 32, Hgb. 10.1, WBC 13.5, electrolytes within normal limits, moderately elevated SGOT and LDH. Vital signs were: blood pressure 100/84, pulse 120, and respirations 32. Arterial blood gas results were: PaO₂ 62, PaCO₂ 31, HCO₃ 24, pH 7.47. Adjusted PaO₂ value with normocarbia was 53 torr, with moderate to severe hypoxemia. A mixture of 40% FIO₂ was administered via mask. The chest x-rays indicated left-sided pleural effusion which progressed to total white-out within two hours. The diagnosis of esophageal perforation was affirmed by the results of the upper GI series.

When the patient arrived in the operating room, she still complained of chest pain. Dyspnea still persisted, even with the administration of 40% O₂. At this point, one peripheral IV was infusing. Vital signs were: 96/78-124-34. A positive tilt test for blood pressure changes indicated hypovolemia, which had been caused by a combination of blood loss and lack of fluid intake. The patient was mildly febrile at 100°. Blood was typed and cross-matched, six units were made available. Earlier, in the emergency room, the patient had been given antibiotics.

The patient elicited a negative cardiovascular, pulmonary, neurologic, and renal history. A history of alcoholism did exist but with no incidence of jaundice. (This could have explained the elevated liver enzyme levels.)

Anesthetic management consisted of the following. A 14-gauge peripheral IV was started along with a central line via the left subclavian. There was an initial CVP reading of 5. An arterial line was inserted into the left radial artery. The patient was preoxygenated, and was monitored via an ECG and continuous blood pressure readings.

A defasciculating dose of 3 mg curare was given intravenously. Ketamine was chosen as the induction agent because of the labile state of the cardiovascular system. The 75 mg dosage of ketamine was followed immediately with 100 mg of Anectine®. The trachea was intubated with cricoid pressure. Pavulon® (pancuronium) 4 mg was administered once the effects of the Anectine® had dissipated as shown on the nerve stimulator.

The patient was then placed in the right lateral position. Oxygen (100%) was administered until the surgical incision was made. Vital signs were monitored for any changes. Heart rate and blood pressure increased upon incision, therefore, Ethrane® in doses starting at .5% was administered. The patient responded favorably, and a level of 1% Ethrane® with 100% O₂ was administered once the effects of the Anectine® had dissipated as shown on the nerve stimulator.

Upon entering the thorax, the surgeon evacuated two units of blood. After the administration of two units of packed cells and upon raising the CVP from a value of 2 to 8, the patient was once again stabilized. For purposes of postoperative ventilation, the orotracheal tube was replaced with a nasotracheal tube, and a nasogastric tube was also inserted. Urine output, by a Foley catheter, ranged from .5-1ml/kg/hr.

The first arterial blood gas reading on 100% O₂ was: PaO₂ 68, PaCO₂ 32, HCO₃ 22, pH 7.49. The minute ventilation was 8,000cc with 10 cm PEEP. At this point, the surgery was completed. The anesthetist’s next challenge is weaning the patient off mechanical ventilation, a process that may require days or weeks.
tilation, the PaO₂ rose to 78. Increasing PEEP had a deleterious effect on blood pressure, so 10 cm became the maximum value utilized.

Total fluid requirements of the patient amounted to 3,000 cc Ringer’s Lactate solution, 2 units of packed cells, and 2 units of plasma protein fraction.

At the termination of the case, relaxants were not reversed because it was necessary to ventilate the patient.

For the first two postoperative days the patient was maintained with controlled ventilation at 8,000cc M.V., with 10 cm. PEEP which maintained PaO₂ values between 70-80 torr. On the third day, IMV was instituted at a rate of 8 and a tidal volume of 800cc. PaO₂ was 72 torr with a PaCO₂ of 38 torr and a pH of 7.39, all acceptable figures. The white-out of the left lung was showing signs of resolution.

IMV was decreased by a rate of 2 breaths/minute/day, down to a rate of 2, at which point the patient was placed on a T-piece with 40% O₂. This treatment progression was based on serial blood gas determinations and the response of the patient.

On a T-piece, arterial blood gases were: PaO₂ 76, PaCO₂ 36, HCO₃ 22, pH 7.36. Acceptable VD/VT ratio with a satisfactory inspiratory effort and the ability to double minute ventilation enabled the patient to be extubated on the sixth postoperative day. Convalescence was uneventful.

REFERENCES


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It should be noted that the opinions stated in this article are those of the author and are not reflective of the official opinions of the Department of Defense or the U.S. Army Nurse Corps.