Gastroesophageal Reflux and Aspiration of Gastric Contents in Anesthetic Practice

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General anesthesia may cause aspiration of gastroesophageal contents because of depression of protective reflexes during loss of consciousness. Some patients may also be at risk for pulmonary aspiration because of retention of gastric contents caused by pain, inadequate starvation, or gastrointestinal pathology. Although knowledge of the problems associated with aspiration has increased, in the past few decades, the incidence and associated mortality rates in the perioperative period have not changed much. With the increase in the extent of surgery performed in the ill, more elderly population, the incidence of regurgitation and aspiration would be expected to increase. The failure to demonstrate any change in the incidence of aspiration perhaps results from improvements in clinical management. This review article discusses the physiologic factors associated with an increased risk of gastroesophageal reflux (GER) and aspiration and some of the methods used to prevent aspiration.

Mechanisms that prevent regurgitation and aspiration include the lower esophageal sphincter (LES), the upper esophageal sphincter (UES), and the laryngeal reflexes. Impairment of these mechanisms may increase the risk of aspiration pneumonitis. Patients are at increased risk if there is a minimum gastric volume of 0.4 mL/kg and the pH of the gastric contents is <2.5. However, volume of fluid aspirated into the lungs does not necessarily relate to volume within the stomach, although some evidence supports a dose–response relation for both the gastric volume instilled directly into the lung and gastric acidity. A reduction in LES pressure is the major physiologic derangement in patients with GER during anesthesia. The tendency to regurgitation is not dependent on the LES pressure itself, but on the difference between the LES pressure and gastric pressure (barrier pressure). Anesthetics and techniques may reduce LES pressure and therefore promote GER because of a reduction of barrier pressure. Antiemetics, cholinergic drugs, succinylcholine, and antacids increase LES pressure, whereas anticholinergics, thiopental, opioids, and inhaled drugs reduce LES pressure. Atracurium, vecuronium, ranitidine, and cimetidine have no effect on LES pressure. The UES helps to prevent aspiration by sealing off the upper esophagus from the hypopharynx. Its function and tone are altered during anesthesia induction. Most anesthesia techniques are likely to reduce UES tone and increase the likelihood of regurgitation of material from the esophagus into the hypopharynx. The protective airway reflexes may be impaired at any time during the perioperative period, particularly in the elderly who have less active airway reflexes and should be considered at increased risk of aspirating pharyngeal material.

Methods to minimize regurgitation and aspiration include control of gastric contents, reduction in GER, prevention of pulmonary aspiration, and attenuation of the effects of aspiration. The first two involve preoperative starvation, a decrease in gastric acidity, facilitation of gastric drainage, and maintenance of a competent LES. Preoperative starvation is designed to minimize the risk and degree of regurgitation and possible pulmonary aspiration during anesthesia. The methods used to assess gastric emptying include paracetamol absorption, electrical impedance tomography, radiolabeled diet, ultrasonography, aspiration of gastric contents under direct vision with a gastroscope, polyethylene glycol dilution, and blind aspiration of gastric contents. In recent studies on preoperative starvation, the volume of aspirated gastric contents after a shorter fasting period after clear fluids, milk, or solids has been compared with the traditional fasting period of at least 6 hr.

The two main issues involving gastric acidity in the preoperative period are the type and timing of drugs to increase gastric pH and the situations in which these drugs should be used. The two most commonly used groups of drugs are H2 antagonists and proton pump inhibitors. Although these drugs increase gastric pH and reduce gastric volumes, evidence does not support their routine use because of the infrequent incidence of aspiration and the multiple factors associated with this complication.

Nasogastric tubes are commonly inserted in patients at risk for aspiration. Current debates regarding the use of nasogastric tubes involve whether they should be inserted and then removed before induction of
anesthesia in the emergency situation, whether they should be inserted during elective abdominal surgery, the effect of size on GER and aspiration in mechanically ventilated patients, and the inclusion of an occlusive balloon to prevent GER. In emergencies, cricoid pressure is applied during rapid sequence induction; evidence suggests that the efficacy of cricoid pressure is not decreased by the presence of a nasogastric tube placed before anesthesia. Other evidence indicates that a nasogastric tube may facilitate regurgitation. One study also showed that there is no difference in the incidence of GER and aspiration based on the gauge of the tube used.

Cricoid pressure is standard practice during anesthesia induction in patients with a potentially full stomach, although there is no convincing evidence that cricoid pressure has reduced the incidence of aspiration or mortality. One issue is the magnitude of force that should be applied to the cricoid cartilage; it should be sufficient to prevent aspiration but not so great as to cause airway obstruction or allow the possibility of esophageal rupture in the event of vomiting. The force applied should be low and then increased only when the patient becomes unconscious. The pressure should be in an upward and backward direction to improve the view at laryngoscopy.

The effect of the method of airway control on the risk of aspiration is another consideration. Tracheal intubation is the gold standard in protecting the airway from aspiration in anesthetized patients. Evidence indicates that evaluation of the seal provided by the cuff is required. Even with improvements in cuff design to prevent leakage, lung infection is a problem in patients undergoing mechanical ventilation. One mechanism of this type of infection involves microaspiration of secretions passing below the glottis through small channels between the cuff and tracheal mucosa. New tubes that allow continuous aspiration of subglottic secretions reduce significantly the incidence of ventilator-associated pneumonia.

New airway devices, developed in the past 20 yr, include the laryngeal mask airway (LMA), the cuffed oropharyngeal airway (COPA), the esophageal–tracheal combination tube (ETC), and the laryngeal tube. Use of the LMA is associated with a reduction in barrier pressure at the LES. Some authors have suggested that the use of the LMA during either spontaneous ventilation or positive-pressure ventilation may promote GER, by high negative intrathoracic pressure generated during inspiration and occurrence of gastric insufflation, respectively. The timing of the removal of the LMA has been studied. pH was significantly increased in patients in whom the LMA remained in situ until mouth opening occurred to command.

The COPA is an airway device that was not designed to protect the airway from aspiration; however, it assists in the maintenance of an unobstructed airway and may reduce one of the factors predisposing to regurgitation and aspiration of gastric contents. It has an inflatable cuff that forms a seal in the proximal laryngopharynx. However, its use often requires airway adjustment maneuvers, such as jaw lift, head rotation, and neck extension. In comparison studies, the LMA performed better than the COPA by providing a better seal. In the presence of ventilation with positive pressure, the possibility of gastric insufflation and gastric aspiration may be increased.

The ETC is a double-lumen tube with a high-volume, low-pressure trachoeosophageal distal cuff and a proximal pharyngeal balloon. Use of the device requires sufficient training and is associated with some complications, such as sore throat, dysphagia, and hematoma. One serious complication is esophageal laceration, which may be the pathway for the development of subcutaneous emphysema. The ETC is not recommended for routine use unless it is inserted under direct vision.

The laryngeal tube has a proximal pharyngeal cuff and a distal esophageal cuff. Its single lumen allows ventilation through the port between the two cuffs. However, free gastric drainage is not possible with an inflated esophageal cuff. Its ease of blind placement is one advantage over the ETC.

Available evidence presented in this review should assist the anesthesiologist in clinical decision-making and lead to improvements in anesthetic management.

Comment: Every anesthetist is alert to the fact that patients may regurgitate and aspirate stomach contents into the lungs. However, I believe that because the condition is rare, many of us have actually become blasé with regard to this particular danger; some anesthetists try to reduce the acceptable starvation time, use positive-pressure ventilation through a laryngeal mask while insufflating the abdomen with gases during laparoscopy, then place the patient in a head-down position, and still nothing comes out. This article is therefore a timely, thorough review of the causes and prevention of GER and should be read by all trainees.

The article consists of 19 pages, is extensively referenced, and discusses the effects of lung aspiration, factors predisposing to aspiration, and methods to prevent the condition, including lowering the volume of stomach contents, cricoid force, reducing stomach acidity, and manipulating the various sphincter tones. If I have to criticize the manuscript, I would say that the authors do not fully explain why certain drugs raise or lower the lower esophageal sphincter (LES) tone, and there is no mention of the classic description of aspiration pneumonia by Mendelson,1 a starting point for any discussion about GER.

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REFERENCES
Moderate Hypothermia Depresses Arterial Baroreflex Control of Heart Rate During, and Delays Its Recovery After, General Anesthesia in Humans

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The effects of hypothermia on arterial baroreflex function were examined during general anesthesia and after its administration in 20 healthy volunteers. The participants were randomly assigned to a normothermic group (10 each, active forced-air warming) or to a hypothermic group (no active warming) during anesthesia. Measurement of R-R intervals and systolic blood pressure were taken at conscious baseline and at 20, 60, and 120 min after induction and at 20, 60, 120, and 180 min after emergence from general anesthesia with sevoflurane for 2 hr. Ventilation was controlled mechanically, and the end-tidal sevoflurane concentration was kept at 2% during anesthesia. Baroreflex responses were elicited by bolus intravenous injections of phenylephrine and nitroprusside. Linear portions of the baroreflex curves related to R-R intervals and systolic blood pressure were ascertained to obtain baroslopes. During anesthesia, the mean lowest tympanic temperature of the hypothermia group (33.9 ± 0.5°C [mean ± standard deviation]) was markedly lower than that of the normothermia group (36.1 ± 0.7°C). There were decreases of 19 to 39% and 27 to 53%, respectively, in the baroslopes of the pressor and depressor tests after general anesthesia in the hypothermia group compared with the normothermia group. The baroslopes of the normothermia group returned to baseline 60 min after anesthesia, whereas the pressor test sensitivity of the hypothermia group was markedly less than that of the normothermia group for the entire recovery period.

The results show that moderate hypothermia enhances anesthesia-induced depression of baroreflex function to anesthetized humans and delays its recovery after general anesthesia.

Comment: During surgery, approximately half of patients reduce their core body temperature to less than 36°C, and approximately one third reduce their core body temperature to less than 35°C. Shivering is an important complication of intraoperative hypothermia. Major consequences of mild perioperative hypothermia include myocardial ischemia, increased intraoperative blood loss, surgical wound infection, and longer hospital stays. Perioperative hypothermia, in addition, produces undesirable neuroendocrine stress hormone responses. Hemodynamic consequences may be caused by a direct effect of hypothermia on the cardiovascular and central nervous system or short-term baroreceptor dysfunction.

Dr. Tanaka and colleagues from Akita University School of Medicine in Japan studied the effects of normothermia involving active forced-air warming and hypothermia (no active warming) during anesthesia in 20 healthy volunteers. Baroreceptor reflex function was assessed using R-R interval and systolic blood pressure responses triggered by bolus intravenous injections of phenylephrine and nitroprusside.

The study findings were dramatic. Moderate hypothermia to a mean of 33.9 ± 0.5°C significantly increased the depression of baroreflex function associated with anesthesia and delayed its recovery after 2% sevoflurane administration of 2 hours’ duration. The volunteer patient population and the lack of surgical stimulation may preclude extrapolation of the study findings to the usual clinical surgery context. The 20 volunteers were young individuals with ASA physical status class I, with no history of drug intake, and without concomitant administration of anesthetic drugs, such as opioid and neuromuscular blocking agents. The neurohumoral response to surgical stimulation may attenuate the reported anesthesia-induced baroreceptor sensitivity changes.

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