Chapter 41

Gastrointestinal Disease

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Conditions Associated with the Oral Cavity and Pharynx

Animals with trauma or space-occupying masses of the head and neck frequently require general anesthesia. These patients are often difficult to intubate to secure and maintain a patent airway. Fractures of the mandible, maxilla, or temporomandibular joint may not permit examination to determine the range of jaw motion. Masticatory myositis may prevent opening of the mouth even while the patient is anesthetized. General anesthesia without a secure airway may result in aspiration, which can be fatal. Anesthetic management of these conditions should be initiated by preparing for placement of a tracheostomy tube. In dogs, a combination of intramuscular (IM) acepromazine (0.05 mg/kg) and intravenous (IV) hydromorphone (0.2 mg/kg) will induce a neuroleptanalgesic state with sufficient muscle relaxation to enable examination of the mouth and jaw. If the animal's laryngeal function or its ability to open its mouth is questionable, IV fentanyl (0.005 mg/kg) should be substituted for hydromorphone because there is a small, but real, risk of vomiting associated with hydromorphone administration. Owing to opioid-induced bradycardia, premedication with an anticholinergic agent is recommended. In dogs weighing over 18 kg, a maximum dose of 4 mg of hydromorphone is suggested to prevent excessive respiratory depression. In cats, a low dose of ketamine (4 mg/kg IM) combined with acepromazine (0.05 mg/kg) usually enables examination of the oral cavity.

IM or subcutaneous (SC) administration of xylazine in dogs or cats may cause emesis (Table 41.1). Dogs and cats with oral or pharyngeal masses are at high risk for aspiration pneumonia, so the use of xylazine, medetomidine, morphine, and other drugs that commonly induce emesis should be avoided. Xylazine administration in large animals is not associated with emesis and may be used to sedate horses (0.5 to 1.0 mg/kg IV) or cattle (0.1 mg/kg IM) for oral examination.

Removal of Esophageal Foreign Bodies

General anesthesia may be required for removal of esophageal foreign bodies in a variety of species. In a horse that has a foreign object lodged in the esophagus (i.e., choke), passage of a nasogastric tube or endoscope is facilitated by sedation with xylazine (0.5 mg/kg) or detomidine (10 to 20 μg/kg). If the foreign body cannot be retrieved or dislodged, the horse is anesthetized. Relaxation of the striated muscular coat of the esophagus may aid in removal of the obstruction. Skeletal muscle relaxation is enhanced at deeper planes of anesthesia. Muscle relaxation may also be improved by induction of anesthesia with guaifenesin (5% solution in 5% dextrose combined with 0.3% thiopental given to effect) or by administration of a neuromuscular blocker. In horses and cats, the proximal two-thirds of the esophagus has a striated muscle layer, whereas in dogs, the entire esophagus contains striated muscle.[1] General anesthesia is required for endoscopic foreign-body removal in dogs and cats. In anesthetized dogs and horses, skeletal muscle relaxation of the esophagus may be improved with a short-acting muscle relaxant (e.g., atracurium, 0.2 mg/kg IV). Administration of atracurium must be accompanied by tracheal intubation and support of ventilation. In some species, esophageal stricture can be a sequelae to esophageal foreign bodies. These patients require general anesthesia in order to perform balloon dilation or bougienage.

Anesthesia for Dogs with Gastric Dilation-Volvulus

It has been estimated that there are between 40,000 and 60,000 cases of canine gastric dilation-volvulus per year in the United States.[2] The condition is associated with multiple system problems (Table 41.2) resulting in a high mortality rate (40% to 60%).[2] Stomach distention severely restricts ventilation and decreases cardiac function. Metabolic alkalosis may develop from gastric sequestration of hydrogen ions. Later in the course of the disease, metabolic acidosis may occur from decreased cardiac output and poor ventilation, resulting in tissue hypoxia and lactate production. Consequently, cardiac arrhythmias such as sinus tachycardia, atrial fibrillation, ventricular tachycardia, or ventricular premature contractions are frequently observed. Because the metabolic status is difficult to predict, it is suggested that serum electrolytes, blood pH, and plasma bicarbonate concentration be measured prior to anesthesia. In one study, hypokalemia was

Table 41.1. Gastrointestinal effects of drugs used during anesthesia.

Drug	Effect
Acepromazine	Antiemetic
Atropine	Decreased motility
Cholinesterase inhibitors	Increased motility
Detomidine	Decreased motility
Diazepam	Appetite stimulant
Halothane	Decreased mucosal blood flow
Morphine	Emetic and gastrointestinal stimulant
Xylazine	Decreased motility; emetic

Table 41.2. Problems associated with gastric dilation-volvulus.

Problem	Significance
Acidosis/alkalosis	Measure pH
Cardiac arrhythmias	Attempt correction prior to anesthesia
Gastric necrosis	May cause arrhythmias
Hypokalemia/hyperkalemia	Measure K+
Impaired venous return	Correct by decompressing stomach Treat shock
Respiratory impairment	Correct by decompressing stomach Ventilate
Peritonitis	Begin antibiotics; poor prognosis
Shock	Correct underlying problems

present in 33% of the dogs with gastric dilation-volvulus.[3] Electrolyte abnormalities, acid-base imbalance, and gastric distension should be corrected as soon as possible. The derangements in acid-base balance frequently precipitate shock if left untreated. Restoration of circulating plasma volume should be initiated using high doses of isotonic saline solution (90 mL/kg IV) or hypertonic saline solution (7%, 4 mL/kg IV). In dogs with experimentally induced gastric dilation-volvulus and shock, a combination of 7% hypertonic saline in 6% dextran 70 (5 mL/kg IV) was superior to isotonic saline (60 ml/kg IV) for resuscitation.[4] It is suggested that fluids be administered through one or two large-bore IV catheters.

Cardiac arrhythmias should be identified and treated prior to induction of anesthesia. In many cases, cardiac arrhythmias may not require antiarrhythmic agents and may respond to fluid therapy and correction of acid-base and/or electrolyte abnormalities. If antiarrhythmic therapy is necessary, lidocaine (2 to 4 mg/kg slow IV bolus followed by 25 to 100 $\mu g \cdot kg^{-1} \cdot min^{-1}$) and procainamide (0.5 to 2.0 mg/kg IV, and then 20 to 40 $\mu g \cdot kg^{-1} \cdot min^{-1}$) separately or in combination have been used for treating premature ventricular contractions or ventricular tachycardia. Quinidine (6 to 8 mg/kg IM, every 6 h) has also been recommended for treatment of arrhythmias in dogs with gastric dilation-volvulus. Postoperative treatment of cardiac arrhythmias may also be necessary. A continuous lidocaine infusion may be prepared by adding 25 mL of 2% lidocaine to each 500 mL of IV fluid administered at a rate of 50 to 100 $\mu g \cdot kg^{-1} \cdot min^{-1}$.

Administration of oxygen via face mask should begin before induction of anesthesia. The use of large doses of arrhythmogenic anesthetic agents such as thiobarbiturates or halothane should be avoided. The use of α_2-agonists should be avoided in these cases because they may cause aerophagia and depressed cardiac output.[5] In addition, xylazine causes decreased gastroesophageal sphincter pressure and may allow increased gastric reflux.[6] α_2-Agonists decrease intestinal motility in dogs and may prolong recovery of normal gastrointestinal function following correction of gastric distension.[7] Neuroleptanalgesic combinations such as diazepam (0.2 mg/kg IV) and hydromorphone (0.1 mg/kg IV) are good choices for induction of anesthesia in dogs with unstable cardiovascular function. Opioids may decrease intestinal motility, but this effect is usually of minor clinical significance.[8] Propofol or diazepam-ketamine has also been suggested as a good choice for induction of anesthesia. For maintenance of anesthesia, isoflurane or sevoflurane would be excellent inhalants. Nitrous oxide is contraindicated prior to gastric decompression because it will equilibrate with gas in the stomach, increasing the intragastric volume and pressure.

Succinylcholine initially causes contraction of skeletal muscle. The distended stomach is predisposed to rupture if succinylcholine is administered prior to relief of excessive gastric pressure. If a neuromuscular blocking agent is used in the anesthetic management of gastric dilation-volvulus, it should be a nondepolarizing drug such as pancuronium or atracurium.

Of the dogs that die of gastric dilation-volvulus, 50% die on the day of surgery.[9] Death is usually associated with septic shock or peritonitis secondary to gastric necrosis or perforation.[10] Reperfusion injury has also been implicated as a factor associated with the high mortality from this condition.[11] Iron-chelating drugs such as deferoxamine have been evaluated for their ability to decrease injury in anoxic tissues that are subsequently reperfused.

Anesthetic Management of Horses with Acute Abdominal Distress

Acute abdominal distress in horses is often characterized by severe pain. Pain elicits a variety of responses that include release of catecholamines and corticosteroids.[12] The stress response and sympathetic stimulation resulting from pain are detrimental to an animal's well-being. It is often difficult to perform diagnostic procedures or examine a horse in severe pain. Judicious use of analgesics in horses with acute abdominal distress is essential.

Because of their strong analgesic and sedative effects, the α_2-agonists have been used extensively in horses with acute abdominal pain. Depending on the amount of xylazine, detomidine, or romifidine administered in the preoperative period, the induction dose of anesthetic may need to be decreased. Cardiovascular depression associated with α_2-agonists will often persist longer than either sedation or analgesia. Even though the sedative effects of these drugs may have waned, the anesthetic requirement will be decreased. At a comparable dose, detomidine is more po-

tent than xylazine and has a longer analgesic and sedative action.[13–15] High doses of detomidine (i.e., 0.02 mg/kg IV) decrease cardiac output by 50%.[16] The duration of cardiovascular side effects may limit the use of detomidine and romifidine as premedicants for horses undergoing surgery to correct intestinal problems.

Xylazine is a suitable alternative to detomidine in compromised horses. However, the usual 1.1 mg/kg IV dose of xylazine is often decreased to prevent excessive cardiovascular depression. Xylazine prolongs gastrointestinal transit time in a variety of species.[17] In horses, xylazine (0.55 mg/kg IV) has been shown to decrease the motility index of circular and longitudinal muscle layers for 30 min.[18] In ponies, xylazine (1.1 mg/kg IV) increased intestinal vascular resistance, motility, and oxygen consumption.[19] In a study of cecal and right ventral colon myoelectric activity in ponies, xylazine (0.5 mg/kg IV) and/or butorphanol (0.04 mg/kg IV) resulted in decreased coordinated spike bursts for 20 min or longer.[20] It should be appreciated that the results of these studies were derived from horses or ponies with healthy gastrointestinal function. Clinically, it appears that these effects are rarely a disadvantage to using xylazine for preanesthetic medication in horses with colic. Furthermore, a study comparing myoelectric activity in equine intestine following three anesthetic regimens found no prolonged effect associated with (a) xylazine and ketamine, (b) thiopental and halothane, or (c) thiopental in guaifenesin and halothane.[21] The conclusion was that the particular regimen of general anesthesia is relatively unimportant in the development of motility disturbances in horses after anesthesia.

Excessive cardiovascular depression is avoided in compromised patients by decreasing the preanesthetic dose of xylazine (0.1 to 0.5 mg/kg IV) and the subsequent administration of diazepam (0.02 to 0.04 mg/kg IV) or guaifenesin (55 to 80 mg/kg IV) when inducing anesthesia with ketamine (1 to 2 mg/kg IV). Induction may be accomplished with ketamine as a bolus or in combination with guaifenesin (1 to 2 g ketamine/1 L 5% guaifenesin). An alternative induction regimen is to combine thiopental (2 to 4 mg/kg) with guaifenesin, given to effect. Induction of anesthesia when using thiopental and guaifenesin does not depend on preanesthetic heavy sedation and muscle relaxation to prevent rigidity or excitement as does induction with ketamine.

Anesthesia may be maintained with either halothane or isoflurane (1.5% to 2.5%) in oxygen. Halothane has been associated with a 62% decrease in intestinal blood flow in ponies anesthetized at 1 minimal alveolar concentration (MAC).[22] However, clinically significant deleterious effects of halothane on recovery from colic surgery have not been demonstrated. Horses anesthetized with isoflurane have higher cardiac outputs than horses anesthetized with a similar MAC of halothane.[23] Isoflurane anesthesia in horses undergoing surgery for colic is associated with a higher heart rate and lower arterial CO_2 concentration as compared with halothane.[24] Horses anesthetized with isoflurane recover more rapidly than those anesthetized with halothane.[24,25] Thus, there are several advantages to using isoflurane over halothane for horses undergoing surgery for colic.

Damaged intestinal tissue may release toxins into the systemic circulation, which can lead to cardiovascular dysfunction and decreased tissue perfusion. Release of eicosanoids (leukotrienes, prostacyclins, prostaglandins, and thromboxanes) has been associated with colonic volvulus in ponies.[26] Administration of flunixin meglumine may counteract the deleterious effects of toxins released during abdominal surgery. In addition, generation of free radicals in the equine intestine following anoxia and subsequent reoxygenation has been demonstrated in vitro.[27] Such studies may be the basis for the empirical intraoperative treatment of certain cases of equine colic with free-radical quenchers such as dimethylsulfoxide (DMSO).

Opioid agonists such as oxymorphone, morphine, and meperidine can be safely used in horses experiencing pain.[28] These drugs frequently induce undesirable excitement in horses that are pain free, unless preceded with a suitable sedative or tranquilizer (e.g., xylazine or acepromazine). Use of an opioid agonist-antagonist such as butorphanol may be advantageous because this drug provides analgesia but has a "ceiling" on respiratory depression.[29] However, numerous other pain medications also are suitable for use in horses, including nonsteroidal anti-inflammatory drugs, local anesthetic agents, and α_2-adrenoceptor agonists.[30]

In addition to pain control, support of the respiratory and cardiovascular systems is paramount in managing anesthesia for colic surgery. The bulk and weight of the gastrointestinal tract filled with ingesta and/or gas impair venous return to the heart and consequently decrease cardiac output. Diaphragmatic excursion and pulmonary function are impaired by a full stomach during anesthesia. These effects decrease tissue perfusion and oxygenation, creating metabolic acidosis and complicating the anesthetic management of horses. Evaluation of acid-base status will aid in determining adequacy of ventilation. Respiratory acidosis ($pH < 7.2$) can be avoided by use of controlled ventilation. Aggressive fluid therapy with lactated Ringer's solution will aid in correcting mild to moderate metabolic acidosis if normovolemia is reestablished.[31] It is not uncommon for a horse to require 30 L or more of isotonic IV fluids during colic surgery.

Monitoring arterial blood pressure is recommended in all species to provide some indirect information concerning cardiac output and tissue perfusion. Low tissue perfusion has been implicated in the occurrence of postanesthetic myositis.[32] Maintenance of a mean arterial blood pressure above 70 mm Hg may be achieved by fluid administration, by adjusting anesthetic depth, and by careful infusion of dobutamine (3 to 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ IV) or another sympathomimetic drug. Improved cardiac output will result when IV fluids are administered to hemoconcentrated patients. A decreased packed cell volume will decrease blood viscosity and improve cardiac output. Hypertonic saline (7% at 4 mL/kg IV) has been recommended for use in hypovolemic horses to expand plasma volume rapidly.[33] One advantage of hypertonic saline administration over isotonic crystalloid solutions is that a small volume is required. Correction of an extracellular volume deficit via administration of a small volume of hypertonic saline is accomplished more rapidly than with the administration of a large volume of isotonic fluid. However, the beneficial effects of hypertonic saline administration are short-lived and should be preserved with subsequent isotonic crystalloid therapy during anesthesia.

Cardiac arrhythmias may occur during anesthesia for surgical correction of an intestinal disorder. Bradycardia caused by increased vagal tone elicited from intestinal manipulation has been observed. Anticholinergic agents are occasionally used in horses for treatment of vagally induced bradycardia. However, high doses of these drugs may decrease gastrointestinal motility for up to 12 h in horses.[34] Horses treated with anticholinergics are more likely to develop postanesthetic colic if they have been fed within 4 to 6 h of anesthesia.[35] Perhaps a more important concern during anesthesia is the effect of atropine administration on cardiac arrhythmogenicity. Administration of atropine to halothane-anesthetized horses was associated with an increased incidence of tachycardia after administration of epinephrine or dobutamine.[36,37] However, horses given low doses of atropine in combination with detomidine, guaifenesin, diazepam, ketamine, and halothane anesthesia were not predisposed to adverse cardiovascular effects when dobutamine was administered for control of arterial blood pressure.[38]

Displaced Abomasum

This is a frequent problem in adult dairy cattle and occasionally in other ruminants such as llamas. In adult cattle, a standing laparotomy is the standard surgical approach. Regional anesthesia for standing laparotomy may be accomplished by a number of techniques, including the distal paravertebral block, the proximal paravertebral block, and the line block. For small ruminants such as goats, sheep, or llamas, general anesthesia will provide a more immobilized patient on which to perform surgery.

Particular anesthetic concerns for animals with displaced abomasum include disturbances in acid-base balance and electrolyte abnormalities. Similar to the pathogenesis of gastric dilation-volvulus in dogs, displaced abomasum in ruminants may initially present with metabolic alkalosis caused by abomasal sequestration of hydrogen chloride. Hypokalemia is a common concurrent finding because potassium is excreted by the kidneys in an attempt to retain hydrogen ions in response to metabolic alkalosis. As the disease progresses, metabolic acidosis occurs because of poor tissue perfusion and lactate accumulation. Shock, followed by death, is expected in untreated animals. Dehydration, poor circulatory volume, and electrolyte abnormalities must be corrected. Serum chloride less than 79 mEq/L and heart rate greater than 100 beats/min are associated with a poor prognosis.[39]

Disorders of the Pancreas

Acute pancreatitis in dogs and cats is frequently associated with vomiting, anorexia, and abdominal pain. However, the diagnosis of acute pancreatitis is often difficult to make antemortem. Classic laboratory findings associated with pancreatitis (increased amylase and lipase activity) may not be observed. Conversely, increased pancreatic enzyme activity is not specific for pancreatitis. Intestinal foreign bodies are frequently associated with increased lipase activity. Exploratory laparotomy in healthy dogs (without signs of pancreatitis), in which abdominal tissues were examined but not surgically altered, was associated with a threefold increase in serum lipase activity.[40] Morphine administration may cause elevation of amylase and lipase activity by increasing smooth muscle contraction in the pancreatic duct (sphincter of Oddi).[41] In addition, many dogs with pancreatitis have concurrent disease, such as diabetes mellitus, hyperadrenocorticism, renal failure, neoplasia, congestive heart failure, or autoimmune disorders.[42] Acute pancreatitis has been induced by drugs, including corticosteroids, nonsteroidal anti-inflammatory agents, organophosphates, thiazide diuretics, sulfonamides, tetracycline, azothioprine, furosemide, and estrogen.[43] Thus, it is likely that animals with acute pancreatitis are often anesthetized for reasons unrelated to diagnosis or treatment of pancreatitis. However, in some situations, such as acute necrotizing pancreatitis or pancreatic phlegmon, surgical therapy may be indicated. Endoscopic, surgical, or laparoscopic techniques can be used to place enterostomy tubes for enteral nutrition. Iatrogenic pancreatitis may also occur after abdominal surgery. Humans occasionally develop acute pancreatitis after renal transplantation, gastrectomy, and biliary tract surgery.[44] The "incidental" finding of acute pancreatitis is not uncommon at the necropsy of many patients with an unknown cause of death.

Medical management of acute pancreatitis basically consists of maintaining adequate fluid therapy and nothing per os. The use of plasma and analgesics has also been advocated for management of pancreatitis. Establishing normal pancreatic circulation is paramount for tissue healing. Preanesthetic preparation of patients with pancreatitis is accomplished by withholding oral intake of food and water and administering IV fluids to correct hydration and/or electrolyte imbalances.

The choice of anesthetics for use in a patient with pancreatitis is often based on other complicating factors identified for the patient. Intravenously administered α_2-adrenergic agents have a hyperglycemic effect owing to inhibition of insulin release by the beta cells in the islets of Langerhans of the pancreas. This effect has been observed with epinephrine and with the potent sedative-analgesics such as detomidine and xylazine.[45–49] While α_2-adrenergic agonist–induced hyperglycemia is avoided by pretreatment with α_2-adrenergic antagonists, sedative and analgesic effects are similarly prevented.[45–51] It is unknown whether the α_2-adrenergic effects on the pancreas are of clinical significance in patients with pancreatitis. However, a conservative approach to anesthetic management of these patients generally avoids use of these drugs. Opioid analgesics (hydromorphone, buprenorphine, or butorphanol) are a suitable alternative to provide sedation and analgesia prior to induction of anesthesia. Morphine causes spasm of the sphincter of Oddi at the termination of the common biliary and pancreatic duct in 1% to 3% of the human population and may be associated with complications in animals with pancreatitis.[41] Other opioids cause less spasm of this sphincter and are useful when indicated for treatment of pain associated with pancreatitis. In humans, because of better pain relief with fewer side effects, epidural administration of morphine is preferred for treatment of pain associated with pancreatitis.[52]

There is no clear best choice for induction of anesthesia for patients with pancreatitis. Halothane would not be the anesthetic of

choice in patients with concurrent liver disease or those with cardiac dysrhythmias. Maintenance of anesthesia with isoflurane or sevoflurane is preferred in such cases. During surgery, the anesthetist should attempt to provide vigilant monitoring of anesthetic depth, prevention of hypotension, and maintenance of adequate vascular volume.

Obesity

Obese patients often have underlying physiological problems in addition to the condition for which anesthesia is required. Evaluation of obese animals for presence of pancreatitis, diabetes mellitus, hepatic insufficiency, or cardiac disease should be included in the diagnostic workup. An obese animal's veins may be more difficult to locate and catheterize. Drug dose should be adjusted to a patient's lean weight to avoid overdosing with anesthetic drugs. Obese animals anesthetized with halothane will also have a longer recovery time than other patients because of significant sequestration of the anesthetic in fat. Isoflurane and sevoflurane are more desirable inhalation anesthetics for obese animals because of their minimal biotransformation and low tissue solubility.

Preoperative hypoxemia (Pickwickian syndrome) due to hypoventilation is a common feature of obesity in humans and is markedly worsened by anesthesia. Obesity decreases the ventilatory capacity of patients during anesthesia, owing to decreased chest wall compliance. Hypoventilation may occur because of limited diaphragmatic excursion from the increased weight of the abdominal contents. The increased mass of the pharyngeal tissues and tongue may lead to upper-airway obstruction after premedication with tranquilizers or during induction of anesthesia. Obese patients given sedatives or tranquilizers prior to anesthesia should be continuously observed for airway obstruction. Rapid control of the airway at induction and positive-pressure ventilation during anesthesia are recommended. During recovery from anesthesia, obese patients should be kept intubated until they will no longer tolerate the endotracheal tube. Obese animals must regain normal muscle function to maintain an adequate tidal volume and a patent airway after extubation.

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