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CHAPTER 119
GASTROINTESTINAL HEMORRHAGE

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KEY POINTS

- Gastrointestinal hemorrhage is an important cause of blood loss anemia.
- In dogs and cats gastrointestinal ulceration is the most commonly reported cause of gastrointestinal hemorrhage.
- Nonsteroidal antiinflammatory drugs and hepatic disease are frequent causes of gastrointestinal ulceration in dogs.
- Neoplasia is a common cause of gastrointestinal ulceration in cats.
- Severe thrombocytopenia should not be overlooked as a cause of gastrointestinal hemorrhage in dogs.
- Hematemesis and melena suggest gastrointestinal hemorrhage but are not always noted.
- With acute severe gastrointestinal hemorrhage, the primary objective is to assess rapidly the patient's cardiovascular status and institute resuscitative efforts if shock is present.
- It is reasonable to administer gastrointestinal protectants before confirming the cause of gastrointestinal hemorrhage.
- Most cases of gastrointestinal hemorrhage respond well to medical treatment, although surgery may be indicated in others.

Gastrointestinal (GI) hemorrhage is an important cause of blood loss anemia and a potentially life-threatening condition in dogs.[1] It is reported less frequently in cats. It may be acute or chronic, occult (no visible blood) or overt (grossly visible blood), and can vary from mild, self-limiting states to severe life-threatening conditions. Significant GI hemorrhage often can be detected during history and physical examination. However, on occasion even acute severe GI hemorrhage may be overlooked if signs localizing blood loss to the GI tract are not present or if concurrent disease obscures the diagnosis.[2,3] In addition, because even mild cases may progress to life-threatening events, it is important to identify rapidly patients with GI hemorrhage and institute therapies to prevent their deterioration.

ETIOLOGY

GI hemorrhage in dogs and cats can be the result of a primary insult to the GI tract or may be secondary to a systemic disease process. It may originate in the esophagus, stomach, small intestine, or large intestine. As such, a number of pathologic processes have been associated with GI hemorrhage. In general, these can be divided into three broad categories: diseases causing ulcers, diseases causing coagulopathies, and diseases associated with vascular anomalies. Some diseases are difficult to classify into one of the above categories, and animals may have single or multiple predisposing causes.[1,4] Diseases associated with GI ulceration and/or GI hemorrhage in dogs and cats are listed in Box 119-1.

The most common cause of GI hemorrhage in dogs and cats is GI ulceration.[3-6] The severity of GI hemorrhage associated with ulcers varies with the degree and extent of mucosal erosion. With erosion into an underlying artery, the magnitude of bleeding is related to the size of the arterial defect and the diameter of the artery.[7] Nonsteroidal antiinflammatory drugs (NSAIDs) and hepatic disease are the most commonly reported risk factors for ulcers in dogs (Figure 119-1).[4] Neoplasia is a common risk factor for ulcers in cats; systemic mastocytosis, gastrinoma, intestinal lymphosarcoma, and adenocarcinoma are the most commonly reported tumors.[3] Inflammatory bowel disease also may be an important nonneoplastic cause of GI ulceration in cats and dogs.[3,8] Stress ulcers are a frequent cause of GI hemorrhage in critically ill human patients and have been reported in dogs and cats after hypovolemia and surgery.[3,9] The true incidence and significance of stress ulcers in critically ill cats and dogs has not been determined but should be considered in patients that develop GI hemorrhage while in the hospital.

Coagulation disorders associated with GI hemorrhage include rodenticide toxicity, disseminated intravascular coagulation, coagulation factor deficiencies (factor XII and prekallikrein deficiency), and thrombocytopenia.[1,5] Thrombocytopenia is the most common coagulation disorder resulting in GI hemorrhage in dogs and should

FIGURE 119-1 Severe hematemesis in a dog subsequent to ingestion of naproxen, a nonselective nonsteroidal anti-inflammatory drug used in humans. Although this case involved accidental ingestion, gastrointestinal hemorrhage has been reported in animals after administration of nonsteroidal antiinflammatory drugs at recommended therapeutic dosages.

BOX 119-1 ***Diseases Associated with Gastrointestinal Ulceration and Hemorrhage in Dogs and Cats***

Drug Administration

NSAIDs
Glucocorticoids

Systemic and Metabolic Diseases

Hepatic disease
Uremia
Pancreatitis
Hypoadrenocorticism

Ischemic Events

GDV
Mesenteric volvulus
Mesenteric thrombosis
Intussusception

Neurologic Disease

Head trauma
IVDD
Mucosal trauma
Foreign bodies

Fungal Infections

Pythium
Histoplasma

Bacterial Infections

Salmonella
Clostridium spp.
Campylobacter
Helicobacter (controversial)

Parasitic Infections

Hookworms
Whipworms
Coccidia
Roundworms

Viral Infections

Parvovirus
Coronavirus

Algal Infections

Protothecosis

Systemic neoplasia

Mastocytosis
Gastrinoma

Gastrointestinal Neoplasia

Lymphoma
Adenocarcinoma
Leiomyoma
Leiomyosarcoma
Hemangioma

Stress of Critical Illness

Major surgery
Hypovolemia
Sepsis

Miscellaneous

IBD
Polyps
Idiopathic eosinophilic masses
HGE

GDV, Gastric dilatation-volvulus; *GI,* gastrointestinal; *HGE,* hemorrhagic gastroenteritis; *IBD,* inflammatory bowel disease; *IVDD,* intervertebral disk disease; *NSAIDs,* nonsteroidal antiinflammatory drugs.

not be overlooked.[1] Coagulation disorders resulting in GI hemorrhage appear to be less common in cats.

Vascular anomalies, because of the high incidence of varices, are a common cause of GI hemorrhage in humans. In contrast, only a few cases of vascular anomaly have been reported in the veterinary literature, and it appears to be an infrequent cause of GI hemorrhage in dogs and cats.[10] It should be considered when more common causes of GI hemorrhage have been ruled out.

HISTORY AND PHYSICAL EXAMINATION

With extensive hemorrhage, vomiting, diarrhea, or ulcer perforation, patients with GI hemorrhage may be presented in a state of shock resulting from blood loss, hypovolemia, endotoxemia, or sepsis. Examination findings consistent with shock include tachycardia, diminished or thready arterial pulses (particularly peripheral), cool extremities, prolonged capillary refill time, and pale mucous membranes. Immediate resuscitative therapies to reverse the state of shock take precedence (see Chapters 5 and 60), and localization of the site of hemorrhage and tailored therapies may have to be delayed until the cardiovascular system is stable.

Once resuscitative efforts have commenced, a complete history and physical examination should be performed. Hematemesis (vomitus with the appearance of coffee grounds or frank blood), hematochezia (passage of bright red or frank blood with or without stool), or melena (black, tarry stool) suggests the GI tract as a source of hemorrhage. However, these signs are not always evident clinically and may not appear until significant GI hemorrhage has occurred.[3,4,11] With duodenal hemorrhage, if reflux of duodenal contents into the stomach is insufficient, blood may not be visible in the vomitus.[12] However, when it is present, hematemesis suggests ongoing blood loss.[13] Diseases of the nasal cavity and oropharynx occasionally can cause hematemesis and melena from swallowing blood of epistaxis or hemoptysis (coughing of blood). In addition, activated charcoal, metronidazole, bismuth (Pepto-Bismol), and diets high in iron (liver, unsweetened baking chocolate) can result in dark stools and should not be confused with melena.[14]

A history of aspirin or other NSAID administration is not uncommon.[4,11,15] Case reports exist of GI ulceration, hemorrhage, and GI perforation occurring in veterinary patients that have received selective cyclooxygenase inhibitors at recommended therapeutic dosages.[11] Decrease or loss of appetite with or without other signs of GI disease should prompt consideration of GI side effects in any patients receiving NSAIDs. The medication should be discontinued and the patient should be examined. In cases of thrombocytopenia or coagulation disorders, there may be a history of bleeding from other sites of the body, including the nasal cavities or urinary tract. Thorough examination of the mucosal surfaces may reveal petechiae in severely thrombocytopenic patients. A search for subcutaneous nodules or masses may detect underlying mast cell tumors.

Because GI hemorrhage may be insidious in onset, especially when chronic, the abdomen should be examined carefully. Abdominal palpation may localize areas of pain (tenderness, voluntary or involuntary guarding) or induce nausea, identify masses or foreign objects, or detect abdominal distention or a fluid wave. Splenomegaly or hepatomegaly may be identified in patients with mastocytosis, other neoplasia, or hepatic diseases. A careful rectal examination should be performed to detect frank blood or melena and to look for masses or foreign bodies.

Localizing the site of GI hemorrhage is important because the cause, diagnostic tests, and therapies for upper and lower GI hemorrhage may vary.[5,14] Although hemorrhage from any site in the GI tract can be serious, upper GI hemorrhage tends to be more severe.[13,14] Hematemesis or melena suggests upper GI hemorrhage.[14] However, it is the amount of time the blood remains in the GI tract and not necessarily the site of bleeding that determines its color.[14,15] Delayed GI transit time and retention of blood in the colon could result in melena associated with a lower GI tract lesion.[14,16] Hematochezia is usually reflective of large intestinal, rectal, or anal hemorrhage;

however, severe acute intestinal hemorrhage can act as a cathartic, significantly decreasing GI transit time.[13-15] This may result in the passage of frank blood in the stool after significant blood loss into the upper GI tract.[13,15]

DIAGNOSTIC TESTS

GI hemorrhage is confirmed when a source of bleeding is localized to the GI tract. Patients with signs of shock should have emergency minimum blood tests performed (hematocrit, total protein, blood urea nitrogen [BUN], glucose and, if available, pH, lactate, and electrolytes) while resuscitative efforts and a search for the underlying cause are undertaken. In cases suspected to have hemoabdomen or septic peritonitis, abdominocentesis, emergency abdominal sonography, and possibly diagnostic peritoneal lavage are warranted and may be performed during initial resuscitation of the patient. Once resuscitative efforts have commenced or the patient's condition has stabilized, other diagnostic modalities should be considered.

Tests to Help Detect Presence of Gastrointestinal Hemorrhage

Certain hematologic and biochemical abnormalities are suggestive of GI hemorrhage. Anemia of undetermined origin should prompt consideration of GI hemorrhage. The finding of microcytic, hypochromic anemia (iron deficiency anemia) is reported with chronic GI hemorrhage.[4] However, because iron deficiency anemia takes time to develop, normocytic normochromic anemia is more common in cases of recent GI hemorrhage.[1,4] A high BUN-to-creatinine ratio (greater than 20) has been reported with GI hemorrhage.[16] This phenomenon has been explained by volume depletion and intestinal absorption of proteins, including digested blood, into the circulatory system.[16] However, diseases resulting in increased protein metabolism (fever, burns, infections, starvation, and administration of glucocorticoids) also may result in an increased BUN-to-creatinine ratio.[1,16] Large bowel hemorrhage reportedly has little effect on BUN levels, and many dogs with GI hemorrhage do not have an elevation in the BUN concentration.[1,16,17]

In equivocal cases of GI hemorrhage a fecal occult blood test (most of which rely on the peroxidase activity of hemoglobin) may be performed. Although helpful for detecting occult GI hemorrhage, diets containing red meat or having high peroxidase activity, such as fish, fruits, or vegetables, can cause false-positive results.[18] Animals should be fed a meat-free diet for at least 72 hours before a fecal occult blood test.[19] The presence of peroxidase-producing bacteria within the GI tract also may cause false-positive results.[18] Despite false-positive results a negative fecal occult blood test result does rule out significant GI hemorrhage.[2] When significant gastric hemorrhage is suspected, passage of a nasogastric tube and aspiration of the stomach contents may confirm and help localize the site of GI hemorrhage. However, this procedure may cause discomfort, and false-negative results have been reported.[12,17]

Tests to Help Identify Underlying Causes

A coagulation profile, complete blood count, routine biochemistry profile, electrolytes, adrenocorticotropic hormone stimulation testing, imaging, and endoscopy often are indicated to try to identify the underlying cause of GI hemorrhage.

The coagulation profile may identify coagulopathies such as rodenticide intoxication or clotting factor deficiencies. It also may detect prolonged bleeding times that are not the direct cause of GI hemorrhage. The platelet count is important, because immune-mediated thrombocytopenia is a common cause of moderate to severe GI hemorrhage in dogs.[1] An elevated hematocrit in a patient with acute hemorrhagic diarrhea and a relatively normal plasma protein concentration is suggestive of hemorrhagic gastroenteritis.[19]

Biochemical markers reflective of hepatic and renal disease may be evident (alkaline phosphatase, alanine aminotransferase, aspartate aminotransferase, and bilirubin in cases of hepatic disease; and urea, creatinine, and phosphorus in cases of renal disease). Because hypoadrenocorticism has been reported as a cause of severe GI hemorrhage in the dog, electrolyte levels should be evaluated and an adrenocorticotropic hormone (ACTH) stimulation test performed if another cause for GI hemorrhage cannot be found.[20] Fecal smears, cultures, and parvovirus testing may be indicated if infectious disease is suspected. Measurement of gastrin levels is recommended in cases of recurrent GI ulceration and in cases that fail to respond to medical therapy.[4]

Radiographs may detect foreign bodies, masses, or free air in the peritoneal cavity. Pneumoperitoneum is suggestive of GI perforation in a patient that has not undergone recent abdominal surgery. Although contrast radiographs may identify gastrointestinal mucosal defects, they generally have been replaced by ultrasonography and endoscopy.[4,17] Ultrasonography may identify foreign bodies and masses and may help to identify concurrent GI perforation when present.[21,22] The use of ultrasonography to identify ulcers in dogs has been described. It allows evaluation of the intestinal wall structure and thickness and can detect the presence of a defect or crater.[22] When used serially, it may help determine changes in response to therapy and has suggested the need for surgery in some instances.[22] Ultrasonography also has been reported in the assessment of cats with GI ulceration.[3]

Endoscopy is considered the most sensitive test to evaluate upper GI tract hemorrhage and ulcers, although patients must be resuscitated optimally before the procedure.[7,17] It often provides a diagnosis, helps assess prognosis, and may have therapeutic benefits (i.e., foreign body retrieval). In addition to allowing direct visualization of the mucosa, it permits biopsies for histology and culture, which may be required to identify lesions and infectious diseases (i.e., neoplasia, inflammatory bowel disease, protothecosis). The disadvantages of endoscopy include the need for anesthesia, its limitation to the proximal GI tract and colon, the potential to exacerbate GI hemorrhage, and the possibility of causing iatrogenic ulcer perforation.[15]

If the above diagnostic procedures fail to identify the cause of significant ongoing GI hemorrhage, abdominal exploratory surgery, scintigraphy using technetium-labeled red blood cells, and arteriography should be considered.[2,17,19] Scintigraphy has been demonstrated to aid in localization of GI hemorrhage in dogs, and arteriography may help identity GI vascular anomalies.[2,10,19]

TREATMENT

The treatment priority in patients with GI hemorrhage is to stabilize the cardiovascular system, control ongoing hemorrhage, treat existing ulcers, prevent bacterial translocation, and to identify and address the underlying cause. Because of the large number of disease conditions that can result in GI hemorrhage, therapy directed toward correcting the underlying cause is variable (i.e., surgery for foreign bodies or tumors, steroids for hypoadrenocorticism, immunosuppressives for immune-mediated thrombocytopenia, discontinuation of NSAIDs). In considering the underlying cause, it is important to consider related or unrelated coagulation abnormalities (i.e., liver disease causing ulceration and a clotting factor deficiency) and to address concurrent diseases that may exacerbate GI hemorrhage (i.e., uremia in a patient on NSAIDs).

Medical Management

The initial priority is to identify rapidly and reverse any signs of shock (see Chapters 5 and 60). Depending on the duration and extent of blood loss, administration of packed red blood cells, whole blood, or hemoglobin-based oxygen-carrying solution may be indicated. In the patient with severe acute GI hemorrhage, this often is implemented as part of the initial resuscitation protocol. Guidelines regarding when to transfuse patients with GI hemorrhage that are anemic but cardiovascularly stable are not well established in veterinary medicine and are controversial in human medicine.[23] The hematocrit at which a patient requires a transfusion varies depending on the degree and rate of blood loss, hemodynamic status, initial and subsequent hematocrits, presence of concurrent illness, and severity of clinical signs.[24] If the patient displays clinical signs attributable to a decrease in oxygen delivery (i.e., tachycardia, hyperlactatemia, tachypnea) or if serial measurements reveal a decreasing hematocrit after initiating therapy, a blood transfusion is indicated.[24] The need for general anesthesia and surgery also may influence the decision of when to transfuse.

If GI hemorrhage is the result of a primary coagulopathy or is exacerbated by a secondary coagulopathy (i.e., disseminated intravascular coagulation, hepatic failure, shock, or dilution with aggressive fluid therapy), fresh frozen plasma should be considered. In patients with persistent GI hemorrhage as a result of thrombocytopenia, vincristine may increase the release of platelets from the bone marrow, although the function of these platelets has been questioned.[25]

The use of iced saline gastric lavage to decrease GI hemorrhage is no longer recommended[5,6]; it has not been proven to slow hemorrhage, is known to cause discomfort and rapidly can lower core body temperature, which prolongs bleeding in experimental canine studies.[6,15]

Animals with hematemesis and melena should be treated for GI ulcers until proven otherwise. Medications known to cause ulcers should be discontinued (i.e., NSAIDs). Given the association between GI hemorrhage and steroids in dogs, unless they are considered essential to therapy (i.e., hypoadrenocorticism, immune-mediated diseases), they also should be discontinued.

It is reasonable to administer GI protectants before confirming the cause of GI hemorrhage, given that ulcers are the most common cause of GI hemorrhage in dogs and cats, and GI protectants have a wide safety margin. In addition, intraluminal gastric acid neutralization may slow GI hemorrhage by promoting mucosal homeostasis.[7,26] Commonly used GI protectants include acid suppressants such as histamine-2 receptor antagonists (cimetidine, ranitidine, famotidine) and proton pump inhibitors (omeprazole, pantoprazole), mucosal binding agents such as sucralfate, and synthetic prostaglandins such as misoprostol. There are no veterinary studies to conclude which gastroprotectants or combination of gastroprotectants is most efficacious in the management of GI ulcers. However, a study demonstrated that famotidine (0.5 mg/kg IV q12h), omeprazole (1 mg/kg PO q24h), and pantoprazole (1 mg/kg IV q24h) significantly suppressed gastric acid secretion in dogs, but ranitidine (2 mg/kg IV q24h) failed to show significant gastric acid suppression at the dosage evaluated.[26] In cases of NSAID toxicity, misoprostol may provide additional benefit (see Chapters 76 and 161).

In deciding which medications to use, clinicians should give consideration to the route of drug administration because absorption of medications administered orally in critically ill patients has been questioned, particularly if GI hypoperfusion is present. Many dogs with GI hemorrhage are also vomiting, which may further limit the utility of oral medications. In patients that have persistent vomiting, antiemetics can be used. Metoclopramide, given as a constant intravenous infusion (1 to 2 mg/kg q24h), often is tried initially. Cases refractory to metoclopramide may benefit from additional antiemetics such as ondansetron. Because many causes of GI hemorrhage are associated with discomfort and pain, analgesics such as an opioid should be considered.

Although controversial, in cases with significant GI hemorrhage and suspected GI mucosal barrier compromise, broad-spectrum antibiotics (i.e., a penicillin and an aminoglycoside or fluoroquinolone, or a combination of a cephalosporin, metronidazole, and an aminoglycoside or fluoroquinolone) are warranted because of the risk of bacterial translocation. Broad-spectrum antibiotics also are recommended in patients that are septic. Ideally, samples for culture and susceptibility (i.e., urine and blood) should be collected before starting antibiotic therapy. In cases in which GI mucosal barrier compromise is not believed to be a factor (i.e., idiopathic immune mediated thrombocytopenia) and there is no evidence of sepsis, supportive therapy and addressing the underlying cause supersedes the administration of broad-spectrum antibiotics. A recent study evaluating the efficacy of amoxicillin/clavulanic acid in dogs with aseptic idiopathic acute hemorrhagic gastroenteritis found no difference in morbidity or mortality in patients treated with antibiotics compared with those given a placebo.[27]

Endoscopy, Interventional Radiology, and Surgery

Most cases of GI hemorrhage can be managed medically. In cases of severe GI ulceration and hemorrhage refractory to medical treatment, endoscopic hemostasis may be beneficial. Upper GI endoscopy is recommended for the diagnosis and treatment of upper GI bleeding in people: the source of bleeding can be identified in up to 95% of cases and endoscopic therapy is reported to be effective in 80% to 90% of patients.[28] Ulcer hemostasis has been described by injecting epinephrine or 98% alcohol through an endoscope sclerotomy needle into the base of an ulcer.[7,29] The combination of epinephrine injection and use of either endoclips, endoscopic cautery (thermal, electric, or laser), or fibrin/thrombin injections currently is recommended in people to control GI hemorrhage unresponsive to medical management.[7,12,30] In people, endoscopic therapy also is indicated for active arterial bleeding as well as visualization of a nonbleeding vessel or an adherent blood clot because both findings are associated with high risk of rebleeding (50% and 25% to 30%, respectively).[30] Surgery can be avoided in most cases but is indicated for preexisting surgical disease (foreign body, tumor, septic abdomen) in patients at risk of exsanguination or perforation (based on endoscopy or serial sonographic evaluation), or if the patient fails to respond to medical therapy. An equally efficacious alternative to surgery with lower morbidity in human studies is percutaneous angiography and embolization, which may be applicable to veterinary patients.[30,31]

PROGNOSIS

Many cases of GI hemorrhage are self-limiting and the prognosis varies with the underlying cause. In cases of moderate to severe GI hemorrhage requiring a blood transfusion, the prognosis is reportedly fair to poor, with a mortality rate of 29% to 45%.[1]

REFERENCES

1. Waldrop JE, Rozanski EA, Freeman LM, et al: Packed red blood cell transfusions in dogs with gastrointestinal hemorrhage: 55 cases (1999-2001), J Am Anim Hosp Assoc 39:523, 2003.
2. Washabau RJ: Acute gastrointestinal hemorrhage. Part I. Approach to patients, Comp Cont Educ Pract Vet 1:1317, 1996.

3. Liptak JM, Hunt GB, Barrs VRD, et al: Gastroduodenal ulceration in cats: eight cases and a review of the literature, J Feline Med Surg 4:27, 2002.
4. Stanton ME, Ronald BM: Gastroduodenal ulceration in dogs: retrospective study of 43 cases and literature review, J Vet Intern Med 3:238, 1989.
5. Washabau RJ: Acute gastrointestinal hemorrhage. Part II. Causes and therapy, Comp Cont Educ Pract Vet 1:1327, 1996.
6. Kirk RW, Bonagura JD, editors: Kirk's current veterinary therapy XI, St Louis, 1992, Saunders.
7. Palmer K: Management of haematemesis and melaena, Postgrad Med J 80:399, 2004.
8. Lyles SE, Panciera GK, Saunders GK, et al: Idiopathic eosinophilic masses of the gastrointestinal tract in dogs, J Vet Intern Med 23:818-823, 2009.
9. Hinton LE, McLoughlin MA, Johnson SE, et al: Spontaneous gastroduodenal perforation 16 dogs and 7 cats (1982-1999), J Am Anim Hosp Assoc 38:176, 2002.
10. Gelens HCJ, Moreau RE, Stalis IH, et al: Arteriovenous fistula of the jejunum associated with gastrointestinal hemorrhage in a dog, J Am Vet Med Assoc 202:1867, 1993.
11. Enberg TB, Braun LD, Kuzma AB: Gastrointestinal perforation in five dogs associated with the administration of meloxicam, J Vet Emerg Crit Care 16:34, 2006.
12. Kupfer Y, Cappell MS, Tessler S: Acute gastrointestinal bleeding in the intensive care unit, Gastroenterol Clin North Am 29:275, 2000.
13. Zuckerman GR: Acute gastrointestinal bleeding: clinical essentials for the initial evaluation and risk assessment by the primary care clinician, J Am Osteopath Assoc 100:S4, 2000.
14. Case VL: Melena and hematochezia. In Ettinger SJ, Feldman EC, editors: Textbook of veterinary internal medicine, ed 7, St Louis, 2010, Saunders.
15. Shaw N, Burrows CF, King RR: Massive gastric hemorrhage induced by buffered aspirin in a Greyhound, J Am Anim Hosp Assoc 33:215, 1997.
16. Prause LC, Grauer GF: Association of gastrointestinal hemorrhage with increased blood urea nitrogen and BUN/creatinine ratio in dogs: a literature review and retrospective study, Vet Clin Pathol 27:107, 1998.
17. Steiner J, editor: Small animal gastroenterology, Hannover, 2008, Schluetersche (distributed by Manson publishing).
18. Tuffli SP, Gaschen F, Neiger R: Effect of dietary factors on the detection of fecal occult blood in cats, J Vet Diagn Invest 13:177, 2001.
19. Hall EJ, German AJ: Diseases of the small intestine. In Ettinger SJ, Feldman EC, editors: Textbook of veterinary internal medicine, ed 7, St Louis, 2010, Saunders.
20. Medinger TL, Williams DA, Bruyette DS: Severe gastrointestinal tract hemorrhage in three dogs with hypoadrenocorticism, J Am Vet Med Assoc 202:1869, 1993.
21. Boysen SR, Tidwell AS, Penninck DG: Ultrasonographic findings in dogs and cats with gastrointestinal perforation, Vet Radiol Ultrasound 44:556, 2003.
22. Penninck DG, Matz M, Tidwell AS: Ultrasonographic detection of gastric ulceration, Vet Radiol Ultrasound 38:308, 1997.
23. Villanueva C, Colomo A, Bosch A, et al: Transfusion strategies for acute upper gastrointestinal bleeding, N Eng J Med 368(1):11-21, 2013.
24. Maltz GS, Siegel JE, Carson JL: Hematologic management of gastrointestinal bleedings, Gastroenterol Clin North Am 29:169, 2000.
25. Rozanski EA, Callan MB, Hughes DH, et al: Comparison of platelet count recovery with use of vincristine and prednisone or prednisone alone for treatment of severe immune-mediated thrombocytopenia in dogs, J Am Vet Med Assoc 220:477, 2002.
26. Bersenas AM, Mathews KA, Allen DG, et al: Effects of ranitidine, famotidine, pantoprazole, and omeprazole on intragastric pH in dogs, Am J Vet Res 66:425, 2005.
27. Unterer S, Strohmeyer BD, Kruse C, et al: Treatment of aseptic dogs with hemorrhagic gastroenteritis with amoxicillin/clavulanic acid: a prospective blinded study, J Vet Intern Med 25:973-979, 2011.
28. Millward S: ACR appropriateness criteria on treatment of acute nonvariceal gastrointestinal tract bleeding, J Am Coll Radiol 5:550-554, 2008.
29. Matz ME: Endoscopy. In Wingfield WE, Raffe MR, editors: The veterinary ICU book, Jackson Hole, Wyo, 2002, Teton NewMedia.
30. Dineson L, Benson M: Managing acute upper gastrointestinal bleeding in the acute assessment unit, Clin Med 12(6):589-593, 2012.
31. Ripoll C, Banares R, Baceiro I, et al: Comparison of transcatheter arterial embolization and surgery for treatment of bleeding peptic ulcer after endoscopic treatment failure, J Vasc Radiol 15:447-550, 2004.