Extrahepatic biliary obstruction (EHBO) is an uncommon diagnosis in veterinary patients; however, when seen, EHBO is often associated with significant systemic compromise that can present a considerable diagnostic and therapeutic challenge. A variety of underlying disease processes can be the cause of obstruction, so a thorough diagnostic evaluation of the patient is required. The underlying cause may significantly affect the prognosis as well as the approach to therapy.

The most common causes of EHBO in dogs are pancreatitis, neoplasia, biliary mucoceles, cholangitis, and cholelithiasis. In cats, a complex of inflammatory diseases that includes pancreatitis, cholangiohepatitis, cholecystitis with or without cholelithiasis, and neoplasia is most commonly responsible. Other less common causes in cats include parasitic infection, diaphragmatic hernia, and foreign body obstruction.

Patients with EHBO often have multisystemic organ dysfunction, much of which has been attributed to the occurrence of systemic endotoxemia in humans and experimental animal models. It is hypothesized that the absence of bile salts in the intestinal tract leads to bacterial overgrowth and endotoxin absorption. Impaired clearance of endotoxins caused by impaired reticuloendothelial function is also thought to contribute to the development of systemic endotoxemia. Impaired myocardial contractility, hypotension, coagulopathies, gastrointestinal hemorrhage, renal dysfunction, and delayed wound healing have all been shown to result. Aggressive supportive therapy as well as early surgical intervention is advised in most patients because they rapidly become systemically compromised.

**DIAGNOSTIC CRITERIA**

**Historical Information**

**Gender Predisposition**
- There are no known gender predispositions for any of the individual underlying diseases known to cause EHBO.

**Age Predisposition**
- A very wide age range of animals can be affected, although certain underlying diseases can be associated with certain age groups.
  - Neoplasia is usually seen in older patients, although some animals as young as 2 years of age can be affected.
  - Biliary mucoceles are seen in dogs 3 to 15 years of age with a median age of 10 to 11 years. Biliary mucoceles are not reported in cats.
- In cats, a very wide age range has been reported for all causes of EHBO.

**Breed Predisposition**
- No breed predispositions have been noted in either dogs or cats.

**Owner Observations**
- Icterus.
- Lethargy.
- Anorexia.
Vomiting.
Weight loss.

Other Historical Considerations/Predispositions
Clinical signs often wax and wane, and patients may be presented several weeks or months after the clinical signs begin.

Physical Examination Findings
- Icterus.
- Weight loss.
- Dehydration.
- Ascites.
- Palpable cranial abdominal mass.
- Diarrhea.

Laboratory Findings
- Hyperbilirubinemia (reference range: dogs, 0.3–0.9 mg/dl; cats, 0.1–0.8 mg/dl).
- Increased serum alkaline phosphatase (reference range: dogs, 20–155 U/L; cats, 22–87 U/L).
- Increased γ-glutamyl transferase (reference range: dogs, 7–24 U/L; cats, 5–19 U/L).
- Hypoalbuminemia (reference range: dogs, 2.5–3.7 g/dl; cats, 2.4–3.8 g/dl).
- Urinalysis: Bilirubinuria or bilirubin crystals in the urine are common.
- Coagulation profile: One-step prothrombin time (OSPT) and the PIVKA (proteins induced by absence of vitamin K) test are the most sensitive early coagulation tests but are often not abnormal until 10 to 14 days after obstruction. Often in chronic cases, evidence of a mixed hemostatic disorder with increases in OSPT and activated partial thromboplastin time along with thrombocytopenia can indicate disseminated intravascular coagulation.
- Fecal examination: Acholic feces are occasionally found in patients with complete biliary obstructions because of a complete lack of bile pigments in the intestines. Evidence of trematode eggs in the feces may indicate fluke infestation, which is a reported cause of EHBO, especially in cats.

Other Diagnostic Findings
- Radiography: Plain radiographs cannot be used to diagnose EHBO but may show a space-occupying lesion in the cranial abdomen that may be evidence of an enlarged gallbladder or a mass. Approximately 50% of canine choledoliths and more than 80% of feline choledoliths are radiopaque and may be visible on abdominal radiography. Choledoliths may be seen in the gall-

KEY TO COSTS
$ indicates relative costs of any diagnostic and treatment regimens listed.
  $ costs less than $250
  $$ costs between $250 and $500
  $$$ costs between $500 and $1,000
  $$$$ costs more than $1,000
bladder or may be visualized in the bile duct, although their causative role in EHBO cannot be assessed. Biliary mucoceles may, if large, be visualized as a large soft tissue opacity in the cranial abdomen. If EHBO has culminated in rupture of the biliary tract and bile peritonitis, there may be a loss of detail on abdominal radiographic views.

- Abdominal ultrasonography: Abdominal ultrasonography is an excellent imaging technique used to evaluate the biliary tract in dogs and cats. The normal diameter of the bile duct is approximately 2 to 4 mm in dogs and cats. One of the earliest signs of EHBO is distension of the bile duct, which occurs within 48 hours, soon followed by distension of the hepatic ducts with intrahepatic duct distension present within 1 week in most cases.

  — It should be noted that ultrasonographic distension of the biliary tree does not indicate active obstruction because long-term persistence of distension can result from a previous episode of EHBO. Monitoring the degree of obstruction over several days may be helpful in discerning active obstruction from a previous episode.

  — Patients with gallbladder mucoceles frequently present with enlarged gallbladders that have a typical immobile stellate or finely striated ultrasonographic appearance (the so-called kiwi gallbladder). Choleliths can usually be identified by their focal echogenic appearance and acoustic shadowing. The area around the major duodenal papilla is a common location for obstructive lesions and should be evaluated thoroughly for evidence of neoplasia, pancreatitis, or choleliths causing EHBO.

- Hepatobiliary scintigraphy: A variety of radiopharmaceutical agents have been used for hepatobiliary scintigraphy in both dogs and cats, including technetium–diisopropyl iminodiacetic acid and technetium–methoxyisobutylisonitrile. Scintigraphic evaluation of hepatic uptake, biliary accumulation, and excretion into the intestine is performed. Nonvisualization of the intestine by 3 hours after radiopharmaceutical injection is used as the scintigraphic criterion for diagnosis of EHBO. The main disadvantage of scintigraphy is that it is incapable of giving accurate information as to the exact site or cause of obstruction. Specialized equipment and expertise are required. Local radiation safety protocols for handling animals administered radiopharmaceuticals must also be observed.

- Computed tomography and magnetic resonance imaging: Advanced imaging techniques will likely be used more frequently to evaluate the biliary tract in the future, although little information on their use is available in the veterinary literature.

### ON THE NEWS FRONT

- Endoscopic retrograde cholangiopancreatography: Used extensively for both diagnostic and therapeutic interventions in humans, this technique has now been described in dogs and is likely to become routine in the future. After catheterization of the major duodenal papilla, injection of iodinated contrast agent allows visualization of both the pancreatic duct system and the biliary tract. Therapeutic interventions, such as cholelith removal or stent placement, may also be possible in the future after a diagnosis has been established.

- Abdominocentesis: If abdominal effusion is present, obtaining a sample by abdominocentesis is vital to rule out bile peritonitis. The most common causes of biliary leakage and secondary bile peritonitis are an increase in intracolic pressure secondary to EHBO and direct perforation by a cholelith or neoplasm that has eroded through the wall of the bile duct. If the bilirubin concentration of the abdominal fluid is two or more times greater than that in the serum, bile peritonitis is likely to be present.

- Exploratory laparotomy: Exploratory laparotomy should be considered an important part of the diagnostic plan, especially if diagnostic imaging findings are equivocal but clinical signs or laboratory findings are still suggestive or if diagnostic imaging is unavailable. Palpation of the gallbladder can often allow assessment of obstruction, which is usually accompanied by bile duct distension. If gallbladder palpation does not allow assessment of patency or if there is a discontinuity to the biliary tract resulting in bile leakage upon palpation of the gallbladder, catheterization of the biliary tract is necessary to assess patency. Catheterization can either be performed normograde or retrograde using a 5- to 12-Fr red rubber catheter, depending on the size of the patient. Either a catheter can be passed from the gallbladder down the bile duct to enter the duodenum (normograde) or a 3- to 4-cm antimesenteric duodenotomy over the area of the major duodenal papilla, 3 to 5 cm aboral to the pylorus, can be performed. The catheter can then be passed retrograde up through the bile duct into the gallbladder.

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STANDARDS of CARE: EMERGENCY AND CRITICAL CARE MEDICINE
Summary of Diagnostic Criteria

- History of lethargy, anorexia, vomiting, and weight loss.
- Physical examination may show icterus, dehydration, pain on abdominal palpation, and possibly ascites with or without evidence of a cranial abdominal mass effect.
- Hyperbilirubinemia.
- Elevated serum levels of cholestatic enzymes (alkaline phosphatase, γ-glutamyl transferase).
- Diagnostic imaging evidence of progressive biliary tract distension, most commonly through ultrasonographic or scintigraphic studies.

Diagnostic Differentials

- Prehepatic icterus is the result of hemolytic disease and can be caused by a variety of underlying factors. Differentiation between pre- and posthepatic icterus is accomplished through evaluation of hematocrit, erythrocyte morphology, liver enzymes, and the results of diagnostic imaging of the biliary tract. Fractionation of total bilirubin into conjugated and unconjugated bilirubin is not helpful in small animals.
- Primary hepatic disease caused by inflammatory, infectious, degenerative, or neoplastic processes can mimic the clinical signs of extrahepatic biliary diseases. These can usually be differentiated using serum biochemical evaluation and diagnostic imaging. Inflammatory disease of the liver often accompanies extrahepatic biliary diseases, especially in cats, in which an inflammatory disease complex involving the intestine, pancreas, biliary tract, and liver exists. Involvement of the liver in extrahepatic biliary disease or primary hepatic disease should be assessed by either ultrasonography-guided aspiration or needle biopsy of the liver or by collection of biopsy samples at laparotomy.

TREATMENT RECOMMENDATIONS

Initial Treatment

- Initial patient stabilization: Many patients with EHBO present late in the course of their disease process. They are often systemically compromised and require hemodynamic resuscitation before surgical intervention.
- Fluid therapy: Fluid therapy should be instituted promptly. An estimate of the fluid deficit should be made based on degree of dehydration (assessed by degree of skin tenting and dryness of mucous membranes) and hemodynamic parameters such as heart rate, pulse quality, capillary refill time, temperature of the extremities, and (if possible) measurement of arterial blood pressure and central venous pressure.

The fluid deficit is replaced with an isotonic crystalloid solution (Normosol-R, Plasmalyte). Solutions with bicarbonate precursors, such as lactate (lactated Ringer’s) and acetate (Normosol-R, Plasmalyte), are alkalinizing and are especially useful in patients with metabolic acidosis, which is commonly found in patients with EHBO.

The rate of fluid administration varies widely based on the degree of hemodynamic compromise. Dogs with mild to moderate hypovolemic shock can be administered fluids rates of 30 to 60 ml/kg for the first hour. Fluid dose rates in cats are generally reduced by one-third to one-half. In cases of severe hypovolemic shock, up to 90 ml/kg/hr in dogs and 60 ml/kg/hr in cats can be given initially. Intercurrent respiratory or cardiac compromise should be ruled out before administering large volumes of fluids. After replacement, maintenance fluid (isotonic crystalloid solution) can be administered at 10 ml/kg/hr during surgery. In patients with low colloid oncotic pressure, a colloid such as dex-
tran (dogs, 10–20 ml/kg; cats, 5–10 ml/kg) or het-astarch (dogs, 10–20 ml/kg; cats, 5–10 ml/kg; boluses of 5 ml/kg can be given to dogs and by slow infusion over 30 minutes to cats) may be necessary to maintain vascular volume. The need for colloid administration may be assessed by monitoring serum albumin, total protein, or, ideally, direct measurement of colloid oncotic pressure. $\$

- **Antimicrobial administration:** Positive cultures of bile in patients with EHBO have been recorded in 38% of dogs and 50% of cats in previous studies. Many different bacteria have been cultured, including *Escherichia coli*, *Clostridium* spp, *Enterococcus* spp, and *Bacteroides* spp, among others. IV antimicrobial coverage should be initiated soon after diagnosis because bactibilia is common and associated with an increased mortality rate in cases with concurrent bile peritonitis and potentially associated with higher wound infection rates postoperatively. Empirically selected antimicrobial agents should be broad spectrum with good action against enteric gram-negative and anaerobic bacteria and preferably be excreted in bile. A second-generation cephalosporin (e.g., cefoxitin, 15 mg/kg IV tid for dogs and cats) is a suitable choice, although it lacks efficacy against enterococci. Ampicillin (22 mg/kg IV tid for dogs and cats) can be added to widen the spectrum to include *Enterococcus* spp. $\$

- **Blood products:** Results of coagulation tests dictate the need for blood products to supplement fluid therapy in the resuscitation regimen. Fresh-frozen plasma should be administered at a rate of 10 ml/kg q4–6h as needed in animals with laboratory-demonstrated coagulopathy or excessive surgical bleeding. In animals that are anemic, packed erythrocytes (at a dose of 10 ml/kg) can be used. Whole blood is especially useful in patients with concurrent coagulopathy because it contains active coagulation factors as well as erythrocytes. A dose of 2 ml/kg of whole blood increases the packed cell volume (PCV) by about 1%. The formula shown in the box on page 6 can be used to estimate the dose of whole blood. $\$–$$\$

- **Vitamin K supplementation:** Because of the absence of bile salts in the intestines of patients with EHBO, fat emulsification is impaired, leading to malabsorption of vitamin K. This results in decreased activation of vitamin K–dependent coagulation factors II, VII, IX, and X. If the results of coagulation tests are normal, there may be no need for supplementation. However, if there is clinical bleeding or laboratory evidence of vitamin K–dependent coagulopathy (increased prothrombin time or PIVKA), then supplementation is advised. Vitamin K is administered at 0.1 to 0.2 mg/kg SC sid. $\$

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**Alternative/Optional Treatments/Therapy**

**Surgical Management $$$$$$$**

In patients with demonstrated EHBO, surgical management should not be delayed beyond the period of resuscitation. In rare cases, it is possible for pancreatitis-induced EHBO to be responsive to medical management and be reversible, but in my experience this is unusual, and many cases require surgical decompression. In general, if an inability to demonstrate patency of the bile duct by normograde or retrograde catheterization of the duct exists, a cholecystoenterostomy should be considered. The procedure of choice is a cholecystoduodenostomy, which maintains more normal gastrointestinal physiology than cholecystojjunostomy. In some cases, however, excessive tension is encountered when an attempt is made to anastomose the gallbladder to the duodenum, mandating the use of the jejunum in the anastomoses. Because the normal bile duct has a small lumen (only 2–4 mm in diameter in cats), choledochoduodenostomy is ill advised except in complex cases in which both passage of bile through the major duodenal papilla or a cholecystoenterostomy are impossible (e.g., cases of EHBO accompanied by gallbladder necrosis) and significant ductal dilation exists.

In cases in which patency of the bile duct can be shown by catheterization but biochemical and diagnostic imaging tests have confirmed functional EHBO, biliary stenting can be considered. Biliary stenting is an option in patients with potentially reversible disease processes (e.g., pancreatitis), in those with trauma to the bile duct, and as a palliative measure in patients with neoplasia.

Another option for temporary rerouting of the biliary system is the placement of a cholecystostomy tube that drains bile through the body wall to a closed collection system. This can be helpful in the management of pancreatitis-related EHBO if resolution of the pancreatitis is anticipated in time. Cholecystectomy is advised in cases of cholelithiasis, biliary mucocele, gallbladder neoplasia, or trauma to the gallbladder if patency of the bile duct can be confirmed.

**Cholecystoduodenostomy**

The patient is positioned in dorsal recumbency and prepared for aseptic surgery. A full exploratory laparotomy is performed. After a decision has been made to perform a cholecystoduodenostomy, the gallbladder must be dissected from its hepatic fossa to allow complete mobilization. This is usually performed by blunt dissection from the fossa with Metzenbaum scissors, cotton-tipped applicators, or the inner cannula of a Poole suction tip. Care needs to be taken to avoid twisting of the cystic duct or damage to the cystic artery. Any bleeding from the hepatic parenchyma
Whole Blood Doses for Patients with EHBO

<table>
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<tr>
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<th>Dose in Dogs (ml)</th>
<th>Dose in Cats (ml)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Body weight × 90 ×</td>
<td>Body weight × 45 ×</td>
</tr>
<tr>
<td>Desired PCV – Actual PCV</td>
<td>Donor PCV</td>
<td>Desired PCV – Actual PCV</td>
</tr>
</tbody>
</table>

should be well controlled by application of a sheet of oxidized cellulose hemostatic agent (Surgicel, Ethicon, Inc.) to the area.

Once mobilized, the gallbladder is positioned adjacent to the antimesenteric border of the duodenum. An incision is created through the long axis of the gallbladder, and bile is suctioned. A duodenotomy of similar dimension is created. Suturing is begun at one end of the far walls of gallbladder and duodenum. A simple continuous suture line is used to oppose one side of the gallbladder wall incision to the corresponding side of the duodenotomy using 3-0 or 4-0 monofilament absorbable suture material. A second continuous line of suture is then used on the near walls to complete the anastomosis. Some authors recommend a two-layer closure, but this tends to further narrow the lumen, which is not desirable. The stoma should be created as large as possible. A small stoma (<2.5 cm) may predispose the patient to obstruction from stricture formation, resulting in the retention of intestinal chyme within the gallbladder, leading to ascending cholangiohepatitis.

Biliary Stenting

An antimesenteric duodenotomy incision is made over the area of the major duodenal papilla 3 to 5 cm aboral to the pylorus. Patency of the biliary tract is checked by attempting to express the gallbladder and looking for bile exiting at the major duodenal papilla. If possible, a 5- to 10-cm section of red rubber catheter (5 to 12 Fr) is passed retrograde up through the major duodenal papilla to lie across the site of the lesion. The gallbladder is emptied of bile and samples are collected for culture and susceptibility testing. The catheter is anchored to the submucosa of the duodenum with one or two interrupted monofilament absorbable sutures (e.g., polydioxanone or poliglecaprone 25). The duodenotomy is closed with a simple, interrupted pattern of 3-0 or 4-0 monofilament absorbable suture material. In some cases, biliary stents will pass in the feces, usually within 6 months postoperatively. In my opinion, radiographs should be taken 3 months after surgery, and if the stent remains in place (if red rubber catheters are used, they are radiolucent enough to be visualized on plain radiographs) and the disease process has resolved, consideration should be given to endoscopic stent removal, which can be performed without difficulty. Little experience exists with biliary stenting in cats, but I caution that reobstruction has been seen clinically. This may be because of an inability of the feline bile duct to accommodate larger stents (3.5- to 5-Fr catheters are most commonly used).

Choledochotomy

Choledochotomy should be avoided if there is an alternative because dehiscence of the repair is common. Choledochotomy is most often performed when a cholelith is lodged in the bile duct. Attempts should be made to milk choleliths back into the gallbladder with subsequent removal via cholecystotomy or cholecystectomy. However, it is possible to flush the cholelith or parts of it into the hepatic ducts, from where they can be difficult to recover. Many choleliths are very brittle and can fragment easily. If choledochotomy is deemed necessary, an incision is made over the cholelith, which is removed followed by suturing the duct with 4-0 to 6-0 monofilament absorbable suture in a simple, continuous pattern.

Sphincter-Altering Procedures

Occasionally, if a cholelith is lodged in the terminal bile duct adjacent to the major duodenal papilla, it is possible to perform a sphincterotomy by incising over a small section of the intramural bile duct oral to the papilla to allow the cholelith to be removed from within the lumen of the duodenum. It is helpful to place a red rubber catheter into the papilla to ensure that the incision only enters the bile duct and does not damage the pancreatic tissue or pancreatic duct. This procedure is infrequently necessary but represents another option for management of choleliths that are difficult to dislodge from the terminal bile duct. In many cases, it is impossible to pass a catheter into the bile duct if a large stone is lodged close to the major duodenal papilla.

Cholecystectomy

The gallbladder is completely dissected free from the hepatic fossa, as already described. At its base, the cystic duct, along with the cystic artery that runs adjacent to it, is doubly ligated with 2-0 or 3-0 monofilament absorbable suture material (some authors recommend the use of nonabsorbable suture material). The gallbladder can then be removed, and the stump should be checked carefully for any leakage of bile.

Before cholecystectomy is done, patency of the bile duct must be confirmed. If this procedure is being performed as part of management of a biliary mucocele, I recommend flushing the bile duct in an effort to ensure that gelatinous bile that has entered the bile duct from
the gallbladder does not cause ongoing obstruction after cholecystectomy.

Supportive Treatment

• **IV fluid therapy:** Postoperatively, isotonic crystalloids should be continued at a rate of 2 to 5 ml/kg/hr in addition to replacing any ongoing losses caused by vomiting, diarrhea, or effusion into the body cavities. Serum electrolyte levels, especially potassium and phosphate, should be monitored and added to maintenance fluids as needed.

• **Antimicrobial therapy:** Therapeutic antimicrobial selection and use should be based on bacterial culture and susceptibility results from bile and liver samples obtained at surgery. Until culture and susceptibility results are available, a broad-spectrum antimicrobial with good activity against gram-negative and anaerobic bacteria should be instituted. If a positive culture is obtained, the appropriate antimicrobial should be administered for 4 to 6 weeks.

• **Nutritional support:** Because of the systemic compromise these patients endure, nutrition is an important part of supportive care. In many cases, patients will be partially or completely anorectic for several days or longer. In patients that are still voluntarily eating, there is no need to place a feeding tube. However, in anorectic patients that are systemically ill, placement of a feeding tube may be advised. Esophagostomy tubes are easily placed in cats and are generally well tolerated. Gastrostomy tubes can be placed during surgery or by a percutaneous endoscopic technique. Surgically placed jejunostomy tubes may be preferable in patients that have underlying pancreatic pathology to prevent excessive pancreatic stimulation during feeding.

• **Analgesia:** Opioid analgesia is routinely administered for 48 hours after surgery or until such time as the patient appears to be comfortable on alternative analgesic agents. Although opioids, especially morphine, increase tone in the sphincter of Oddi and can therefore be associated with biliary stasis as well as a decrease in pancreatic secretions in people, little is known about this phenomenon in small animals. It is thought that butorphanol (0.2–0.4 mg/kg IV or IM) and buprenorphine (0.01 mg/kg IV or IM) may be better choices than morphine for this reason. Fentanyl patches (Table 1) can also be used to provide 48 to 72 hours of continuous analgesia in the postoperative period.

NSAIDs should be used with caution in vomiting animals and in those that have undergone cholecystoenterostomy because these patients can be predisposed to gastrointestinal ulceration as a result of the physiologic alterations in gastric acid secretions that can occur with biliary rerouting.

Multiple NSAIDs are available for postoperative pain in dogs. I prefer deracoxib (1–4 mg/kg PO sid) and carprofen (2.2 mg/kg PO bid). Only one NSAID is currently licensed for postoperative use in cats in the United States: meloxicam given as a single SC dose of 0.3 mg/kg. When NSAIDs are prescribed, concurrent administration of sucralfate (250 mg/15 kg PO qid) with or without an H₂-receptor antagonist such as famotidine (0.5 mg/kg PO sid) or ranitidine (2 mg/kg PO bid–tid) is recommended.

Patient Monitoring

• **Biochemical panels** should be repeated, initially on a daily basis, until resolution of hyperbilirubinemia and abnormalities in cholestatic liver enzymes resolve.

• **Coagulation profiles** should be performed postoperatively on a daily basis until parameters have returned to normal. Until that time, further treatment with blood products may be necessary.

• **Patients should be closely monitored for any signs of dehiscence of cholecystoenterostomy or duodenotomy incisions.** This is most likely to occur 3 to 5 days after surgery. Clinical signs may include increasing abdominal pain and development of abdominal effusion. Monitoring for evidence of sepsis, such as injected mucous membranes, tachycardia, and hypotension, is important, as is laboratory evidence of worsening hyperbilirubinemia or hypoproteinemia.

• **Rodent models** have shown impaired wound healing in EHBO, so patients should be closely monitored to assess for the progression of normal healing.

Home Management

• **Analgesia:** Dogs should be given 5 to 7 days of ongoing analgesic coverage after surgery. Either NSAID or fentanyl patches may be suitable (see above).

• **Dietary management:** Dogs do not require long-term dietary modification if they are eating at home.

<table>
<thead>
<tr>
<th>Animal</th>
<th>Dose</th>
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<tbody>
<tr>
<td>Small dogs (&lt;5 kg)</td>
<td>25-µg patch with half of patch covered by tape (do not cut patch)</td>
</tr>
<tr>
<td>Dogs 5–10 kg</td>
<td>25-µg patch</td>
</tr>
<tr>
<td>Dogs 10–20 kg</td>
<td>50-µg patch</td>
</tr>
<tr>
<td>Dogs 20–30 kg</td>
<td>75-µg patch</td>
</tr>
<tr>
<td>Dogs &gt;30 kg</td>
<td>100-µg patch</td>
</tr>
</tbody>
</table>

**TABLE 1: Fentanyl Patch Doses**
and EHBO has resolved. Ongoing management of feeding tubes may be necessary at home in patients that are slow to resume eating normally.

- **Rest:** Dogs should be allowed only short leash walks for the first 2 weeks to allow for healing of the laparotomy incision.

- **Owners should be vigilant for recurrence of icterus,** which may indicate recurrence of obstruction or an episode of ascending cholangiohepatitis. Episodes of cholangiohepatitis usually respond to supportive care such as fluid therapy and antimicrobials.

- **Suture removal** is done 10 to 14 days after surgery.

**Milestones/Recovery Time Frames**

- Hyperbilirubinemia and elevations in cholestatic liver enzymes should start to decrease the day after surgery and usually continue to decrease into the normal range in about 1 week. If this does not occur or if the serum total bilirubin starts to increase again after surgery, be suspicious of a complication such as recurrence of obstruction or cholangiohepatitis.
- **Icterus** should start to resolve within a few days after surgery but may take several weeks to resolve completely.
- Continuous blood pressure, central venous pressure, and electrocardiographic monitoring should be performed for 2 to 3 days after surgery or until parameters normalize.
- Most patients start to eat on their own after 2 to 3 days, although parenteral feeding may be required in some patients that continue to be anorectic for longer periods.

**Treatment Contraindications**

- Patient stabilization and resuscitation should be attempted before proceeding with surgical intervention.
- Surgical intervention is recommended early in the course of disease because in my experience, progressive hemodynamic compromise occurs rapidly in patients with EHBO.

**PROGNOSIS**

The prognosis for animals with EHBO is guarded in cats and dogs because of the potential for severe hemodynamic compromise. Perioperative mortality rates of 40% to 50% are reported in dogs, and a 57% mortality rate is reported in cats. The prognosis in patients that do survive the perioperative period depends on the underlying etiology.

**Favorable Criteria**

- **Underlying etiology:** Neoplasia carries a particularly poor prognosis because most are pancreatic or biliary adenocarcinomas, which have often metastasized at the time of surgery. Cholangitis or cholangiohepatitis with or without cholelithiasis and gallbladder mucoceles is often associated with a better prognosis, although mortality can still be high.

**Unfavorable Criteria**

- **Azotemia** on presentation is a poor prognostic indicator. Renal perfusion is often compromised because of renal artery vasoconstriction and systemic hypotension. Acute renal failure is a common cause of postoperative mortality in humans with EHBO and is also recorded in dogs and cats.
- **Postoperative hypotension** is a poor prognostic sign. Many patients become noticeably hypotensive during surgery because they have compromised myocardial function and an inability to respond normally to vasopressor agents.
- **Coagulation abnormalities** worsen the prognosis. Attention to correcting these deficits is a very important part of preoperative management.
- The presence of **septic bile peritonitis** is a poor prognostic indicator. These patients need particularly aggressive resuscitation and close monitoring.

**RECOMMENDED READING**