Canine Extrahepatic Biliary Tract Disease and Surgery

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ABSTRACT: Diseases that require surgery of the extrahepatic biliary system in dogs are primarily acquired and include extrahepatic biliary tract obstruction, gallbladder mucocele, traumatic injury, and cholecystitis. The main goals of surgery are to confirm the underlying disease process, establish a patent biliary system, and minimize perioperative complications. Surgical biliary tract diseases are associated with a high mortality rate, and early surgical intervention with appropriate supportive care may improve the prognosis.

The incidence of disorders restricted to the gallbladder and biliary tree is low compared with the many parenchymal hepatic conditions that occur in animals. Diseases of the extrahepatic biliary system are often confused with other intraabdominal disorders because their course and clinical signs are similar. Diseases that require surgery of the extrahepatic biliary system in dogs are primarily acquired and include extrahepatic biliary tract obstruction (EHBO), traumatic injury, and cholecystitis. The main goals of surgery are to confirm the underlying disease process, establish a patent biliary system, and minimize perioperative complications. This article reviews the common disorders of the extrahepatic biliary system in dogs, including common clinical signs, typical clinical pathologic findings, surgical procedures, and common surgical complications.

ANATOMY AND PHYSIOLOGY OF THE BILIARY SYSTEM

The canine extrahepatic biliary system comprises the gallbladder, cystic duct, hepatic ducts, bile duct, and major duodenal papilla (Figure 1). In dogs, bile flows from the bile canaliculi into the interlobular ducts and then the lobar ducts before leaving the liver. Lobar ducts drain into hepatic ducts, through which bile passes into the common bile duct. The gallbladder lies within a fossa between the right medial and quadrate lobes of the liver. The gallbladder is drained by the cystic duct and is the portion of duct before the hepatic ducts enter the common bile duct (Figure 2).

In a medium-sized dog (33 to 44 lb [15 to 20 kg]), the gallbladder holds approximately 15 ml of bile. In dogs, the common bile duct terminates near the minor pancreatic duct at the major duodenal papilla (Figure 2). In a medium-sized dog, the common bile duct is approximately 5 cm long and 2.5 mm in diameter and empties...
into the duodenum 2.5 to 6 cm distal to the pylorus at the major duodenal papilla after coursing intramurally for approximately 2 cm. The blood supply to the gallbladder and common bile duct is derived from the left branch of the proper hepatic artery.

In dogs, the pH of bile is usually greater than 6. Bile is acidified by secretion of hydrogen ions (H+) and not by reabsorption of bicarbonate ions (HCO₃⁻). More than 90% of biliary solids are composed of bile acids. Bile salts are kept in solution by incorporation into micelles. Taurocholate and taurocholic acid are the primary salt and acid, respectively, in bile. Bile acids are synthesized from cholesterol, conjugated in hepatocytes, and secreted continuously into bile canaliculi. Bile salts are formed within hepatocytes and secreted as sodium ion (Na⁺) and potassium ion (K⁺) salts of bile acids. They are secreted into the duodenum, reabsorbed in the ileum, and then carried back to the liver for reexcretion. Ninety percent of bile is recirculated bile salts.

Bilirubin is the major bile pigment and a product of hemoprotein catabolism. The gallbladder reabsorbs more than 50% of the calcium in bile. This maintains free calcium ions (Ca²⁺) at a low concentration in bile, which is thought to protect against cholelith formation.

Filling of the gallbladder with bile occurs continuously through hepatic secretion and passive gallbladder distention. This is a low-flow, low-pressure system. The sphincter of Oddi is the functional sphincter located at the terminal portion of the common bile duct. Rhythmic contractions of the sphincter regulate duodenal bile flow into spurts rather than continuous flow. The sphincter functions as a one-way valve that can regulate pressure within the biliary tract and also provides resistance to the retrograde passage of duodenal contents or pancreatic secretions into the biliary tree.

Cholecystokinin is a hormone produced and secreted by the duodenal mucosa. It is the principal hormone responsible for stimulation of gallbladder contraction. Cholecystectomy, ileal resection, and a rise in cholestyrmine increase the release of cholecystokinin. Motilin and the cholinergic pathway also stimulate gallbladder contraction and bile flow.

Unconjugated bile acids are cytotoxic and induce tissue inflammation. They alter the permeability of vascular structures within the peritoneum, resulting in transudation of fluid and then transmural migration of enteric organisms into the peritoneal cavity. Although virtually all bile acids derived from the biliary tree are conjugated, a bacterial infection or low pH within the biliary tree can result in bile acid deconjugation.

Bile salts enhance absorption of fat-soluble vitamins (i.e., vitamins A, D, E, and K). Decreased production,
inactivation of bile salts, or biliary obstruction can contribute to a clinically important decrease in vitamin K–dependent coagulation factors.\(^2,6\)

### CLINICAL SIGNS OF EXTRAHEPATIC BILIARY DISEASE

Clinical signs in dogs with surgical diseases of the biliary tree are nonspecific and mimic other abdominal disorders. Signs may wax and wane for several weeks before presentation.\(^2\) In humans, biliary system pain is severe and can be difficult to distinguish from esophageal pain and angina. Such discomfort in animal patients seems to be rare, although, occasionally, animals with cholelithiasis are examined for episodic abdominal discomfort, vomiting, and diarrhea.\(^1–3\) Most animals with bile duct obstruction are not likely to be examined until clinical signs of icterus develop. Patients with EHBO are overtly icteric, have lost weight, and present with vague signs of illness (Figure 3). Clinical icterus becomes evident when serum bilirubin levels become greater than 1.5 to 2 mg/dl.\(^6,8,9\) Right cranial abdominal masses can often be palpated in patients with biliary tract disease secondary to pancreatitis or neoplasia. Detection of acholic feces in an icteric animal is consistent with a diagnosis of bile duct obstruction.

### DIAGNOSTIC EVALUATION

The primary diagnostic problem in evaluating dogs with icterus is differentiating among an extrahepatic obstructive lesion, a primary hepatic parenchymal disease process, and some systemic diseases. The first requires surgical intervention, and the latter are managed medically. A difficulty in diagnosing biliary tract obstruction is that animals may not demonstrate clinical signs or hematologic abnormalities for weeks to months after the obstruction. This has been documented in experimental bile duct ligation studies.\(^4\)

Bile duct obstruction causes an increase in total serum bilirubin, with more than 90% being direct or conjugated bilirubin, along with corresponding bilirubinuria. Dogs have a low renal threshold for excretion of conjugated bilirubin, and, with obstruction of the bile duct, renal excretion of bilirubin becomes important for elimination of the pigment.\(^1,6\) Bilirubinuria may be the first sign of bile duct obstruction in dogs and may precede the development of jaundice. If the obstruction is complete, urobilinogen will be absent from the urine. Because detection of urobilinogen in urine depends on many variables (e.g., exposure to light, drugs, sensitivity of detection methods), the absence of urobilinogen should be interpreted with caution.\(^2\)

Acute experimental biliary obstruction in dogs results in rapid elevation of serum alkaline phosphatase and bilirubin after a latent period of up to 6 hours.\(^10\) One study\(^4\) identified an elevation of conjugated and total serum bilirubin levels 24 hours after experimental common bile duct ligation and reported that total bilirubin levels reached their maximum at 5 to 6 days after experimental obstruction. However, the magnitude of some serum liver enzyme elevations has no correlation with the degree of hepatobiliary injury or obstruction.\(^1\)

If a dog is septic or necrotizing cholecystitis is present, fever and leukocytosis with a left shift are often present.\(^1,2\) Mild to moderate nonregenerative anemia may also be observed.

Abnormalities in coagulation tests are uncommon in patients with EHBO because dogs present too early in the course of the disease for abnormalities to be detected.\(^1,11\) When coagulopathies are identified, prothrombin time is usually elevated before the partial thromboplastin time because of the short half-life of factor VII.

### Imaging Modalities

Radiography should be used in patients with clinical signs and laboratory abnormalities consistent with biliary disease. In dogs and cats, 14% to 50% of choleliths are
mineralized and, therefore, radiopaque.\textsuperscript{1,2,7,12,15} Gas within the biliary structures is likely due to emphysematous cholecystitis, cholangitis, choleodochitis, or an abscess and warrants prompt surgical and antimicrobial therapy.\textsuperscript{12,24}

Abdominal ultrasonography is currently the most useful and practical technique for demonstrating gallbladder and common bile duct dilation associated with obstruction in small animals. Ultrasonography is a sensitive and specific indicator of the cause of EHBO.\textsuperscript{15} Ultrasonographic findings of bile duct distention secondary to obstruction may be identified in 62\% to 100\% of cases involving humans and dogs.\textsuperscript{13,16,37} Gallstones are infrequently radiopaque but are readily identified with abdominal ultrasonography. The degree of biliary tract dilation in obstructed dogs and humans is variable. Therefore, duct size allows only a crude estimation of the duration of obstruction.\textsuperscript{4,9} It is important to note that biliary obstruction can be diagnosed before the onset of clinical icterus with the use of abdominal ultrasonography. Minimal intrahepatic ductule distention is a subtle abnormality but can be identified via ultrasonography as early as 4 hours after experimental biliary occlusion; however, the absence of gallbladder dilation does not exclude EHBO because the gallbladder may be contracted because of inflammation.\textsuperscript{4,20}

Oral, intravenous, and cholangiographic contrast studies can be conducted. High serum bilirubin concentrations, hypoalbuminemia, icterus, hepatocellular disease, pancreatitis, peritonitis, biliary obstruction, cholecystitis, or concurrent sulfonamide and salicylate administration may cause decreased hepatic concentration of the contrast, resulting in poor opacification of the extrahepatic biliary system.\textsuperscript{3,9,12,13} Percutaneous transhepatic cholangiography, endoscopic retrograde cholangiopancreatography, and cholecystocentesis are reliable techniques for diagnosing EHBO in humans but are not routinely used in dogs.\textsuperscript{1,3,4,9,12}

Hepatobiliary scintigraphy can be a valuable diagnostic tool in differentiating EHBO from hepatoportal disease.\textsuperscript{9} \textsuperscript{99m}Tc-DISHDA is used as the radio nuclide, and high serum bilirubin concentrations at the time of hepatobiliary scintigraphy do not interfere with the diagnostic usefulness of the findings.

In humans, severe complications from vagal stimulation, which have progressed to cardiac arrest, have been described in patients with acute cholecystitis undergoing vigorous diagnostic or therapeutic manipulation of the gallbladder. Similar problems have been observed in dogs undergoing simple gallbladder aspiration.\textsuperscript{1,17,18}

### Microbiology

Partial or complete EHBO can lead to bile stasis and promote aerobic and anaerobic bacterial growth. Bile is thought to be sterile in normal dogs and cats.\textsuperscript{19,20} In humans, infections of the biliary tree usually begin in the gallbladder and spread into the biliary system rather than ascend from the gut.\textsuperscript{1} In one study\textsuperscript{21} of dogs with bile peritonitis, 10 of 17 had positive cultures for bacteria in their abdominal effusion, and multiple organisms were isolated in eight of 10. The most common isolate was Escherichia coli, followed by Enterococcus, Enterobacter, Klebsiella, Streptococcus, Pseudomonas, Bacteroides, and Acinetobacter spp. In another study\textsuperscript{26} of nine dogs with EHBO, six of nine cultures were positive for aerobic bacteria, which included \textit{E. coli} as well as Enterobacter, Enterococcus, Staphylococcus, Micrococcus, and Streptococcus spp. In four of six dogs, a mixed population of bacteria was isolated.

In one report\textsuperscript{7} of 29 dogs with cholelithiasis, 20 dogs had cultures of a cholelith or bile and 14 of 20 had positive growth. The most common organisms grown were \textit{E. coli} as well as \textit{Streptococcus, Enterococcus, and Klebsiella spp.}

### EXTRAHEPATIC CAUSES OF BILIARY TRACT OBSTRUCTION

The incidence of biliary tract obstruction is much lower in dogs than in humans because intraluminal extrahepatic obstruction due to cholelithiasis is uncommon in dogs.\textsuperscript{3,4,19} A number of other disorders cause EHBO in dogs; mechanical obstructions of the duct due to neoplastic diseases of the liver, gallbladder, bile ducts, pancreas, gastrointestinal (GI) tract, or lymphatic tissue are common.\textsuperscript{1,3–5,22} Abscesses, granulomas, and fibrosis secondary to trauma or, more commonly, inflammation from pancreatitis can also cause extraluminal obstruction.\textsuperscript{4,5,22,23}

Pancreatic disease is the most common cause of EHBO in dogs.\textsuperscript{5} Scar tissue can form in or around the common bile duct, or the duct can be compressed by fibrotic or inflamed pancreatic tissue, pancreatic cysts, or pancreatic abscesses. In a study\textsuperscript{23} evaluating dogs undergoing surgery of the extrahepatic biliary tract, 17 of 60 cases were caused by EHBO. Five of these were due to neoplasia, and 12 were secondary to pancreatitis.

Intraluminal obstruction is less common but may be caused by cholelithiasis, choledocholithiasis, or impalpable bile.\textsuperscript{3,5} It has been reported that biliary obstruction lasting over 6 weeks can lead to biliary cirrhosis.\textsuperscript{1} In the presence of complete bile duct obstruction, bile may become colorless (white bile) because of reduced secretion of bilirubin and increased production of mucin.\textsuperscript{1,3,25}
Cholelithiasis

Until recently, cholelithiasis has been considered an uncommon disease in dogs and cats (Figure 4). It has been reported\(^2,6,26\) that dogs with cholelithiasis and choledocholithiasis account for less than 1% of patients with liver disease. Cholelithiasis is a condition of senior, female small-breed dogs and is often an incidental finding at necropsy or via radiology.\(^7\) As a clinical problem, cholelithiasis has a high incidence in miniature schnauzers and miniature poodles.\(^4\) Although most choledochooliths are believed to form in the gallbladder, some form in the ducts\(^27,28\) (Figure 5). Calcium salts are the major component of pigmented gallstones, and the availability of ionized calcium may be important in gallstone formation in dogs.\(^5\)

It is believed that choleliths are typically clinically silent because they are common, incidental postmortem findings.\(^1,5–7,26\) As many as 75% of reported cases of cholelithiasis have been diagnosed at necropsy with no reported association with clinical signs.\(^26\) Clinical signs associated with cholelithiasis are thought to be more commonly related to cholecystitis. The prevalence of cholelithiasis causing cholecystitis is estimated to be less than 1% of dogs with biliary disease.\(^13\) Choleliths have been associated with vomiting, anorexia, icterus, fever, and abdominal pain.\(^1,2,5–7,13,25\)

Gallstones are readily diagnosed using abdominal ultrasonography.\(^1,7\) There is great variation in the size, shape, number, and composition of choleliths. The composition is cholesterol; a combination of bilirubin, calcium carbonate, calcium phosphate, and glycoproteins; or both.\(^2,7,13,25\) Cholesterol stones are large, white, and lightweight and occur in animals on high-cholesterol diets.\(^26\) Canine cholelithiasis may be rare because of decreased concentrations of cholesterol in canine bile compared with those in human bile. Pigment stones are yellow, dark brown, or black stones that vary in weight and fragility and are more common in dogs. The cause of pigment stone formation is most likely related to infectious cholecystitis. The mechanism of formation is thought to be similar to that of urinary calculi. Solid particles of dead cells or inspissated material act as a nidus for crystallization.\(^1,7,25,26\) In addition, some constituents of bile are resorbed during biliary stasis more easily than others, leaving highly desiccated residues within the gallbladder.

Pancreatic Diseases

Mortality rates in dogs with pancreatitis that underwent biliary tract surgery were 50% to 100%.\(^2,24\) Early detection and aggressive management of pancreatic necrosis or abscessation was shown to improve survival (Figure 6). There have been a few reports\(^29–32\) of pancreatic pseudocysts causing EHBO in dogs. These pseudocysts form as a result of pancreatic duct disruption from acute or chronic pancreatitis or trauma (Figure 7). CT has been used in differentiating between pancreatic pseudocysts and pancreatic abscesses, although ultrasonography has also been useful in diagnosing and clinically monitoring
pancreatic pseudocysts.²⁹,³³ Treatment involves draining the pseudocyst into the GI tract and omentalization of the pseudocyst. Other conditions associated with the pancreas that cause EHBO are pancreatic neoplasia, abscesses, cysts, and granulomas. These causes are less common than obstruction caused by chronic pancreatitis.

Neoplastic Diseases
Primary neoplasms of the gallbladder and common bile duct are rare in domestic animals, comprising 2% of all canine hepatic tumors.²,²²,²⁵ Up to 20% of animals with EHBO have neoplasia of the pancreas or the bile duct.²² Adenomas of the gallbladder are benign localized tumors originating from the glandular epithelium. The tumor projects into the lumen and does not infiltrate the deeper parts of the gallbladder wall.²⁵ Adenocarcinomas of the gallbladder invade the wall, form incomplete tubules and undifferentiated cells, undergo necrosis, and may extend into the liver and adjacent peritoneum. Metastasis to the lungs and perineural lymphatics has been reported.

Mucocele
A biliary mucocele is distention of a bile-containing structure or cavity due to inappropriate accumulation of mucus.²,¹⁶,¹⁴–³⁷ (Figure 8). Biliary mucoceles occur in dogs older than 6 years of age and have no breed or sex predilection.² EHBO does not appear to play a primary role in gallbladder mucocele formation, and no inflammatory or infectious cause has been associated with gallbladder mucoceles.³⁶ Biliary stasis, decreased gallbladder motility, and altered absorption of water from the gallbladder lumen predispose animals to biliary sludge formation. Biliary sludge may be a precipitating factor for the development of canine biliary mucoceles.

There are reports of a 50% to 60% incidence of gallbladder rupture in patients with biliary mucoceles and a 13% to 80% incidence of a histopathologic diagnosis of gallbladder wall necrosis. Ruptured mucoceles contain semisolidified bile and leak slowly, causing acute but local chemical, sterile peritonitis. In some dogs with ruptured mucoceles, the solidified mucocele can be found intact, floating freely in the abdomen.

In most dogs with gallbladder mucoceles, the disease is contained within the gallbladder and does not involve the hepatic ducts or the common bile duct. In some cases, gelatinous biliary material is present within the common bile duct and is easily dislodged or flushed through the duodenal papilla (Figure 9). Dogs with biliary mucoceles that undergo cholecystectomy and survive the immediate perioperative period have an excellent prognosis.³⁶,³⁷

Necrotizing Cholecystitis
Necrotizing cholecystitis has reportedly been a common cause of gallbladder rupture in dogs.²¹ Impairment of the cystic arterial circulation by occlusion, bacterial infection, or cystic duct obstruction from cholediths, neoplasia, or an adjacent inflammatory process can lead to cholecystitis.³ In most cases of necrotizing cholecysti-
tis, a combination of surface irritants and impaired cystic duct flow is thought to be present.

Necrotizing cholecystitis has been classified in the human and veterinary literature in a class system:

• **Class I** includes patients with necrotizing cholecystitis without gallbladder rupture.
• **Class II** includes patients with acute necrotizing cholecystitis with gallbladder perforation and peritonitis.
• **Class III** identifies patients with chronic cholecystitis with cholecystic and omental hepatic adhesions with fistulas to other abdominal structures.

In a study of dogs with necrotizing cholecystitis, patients had a mean age of 9.5 years and a mean duration of clinical signs before presentation of 3 days. Histopathologic findings in the gallbladders of these dogs were similar to findings in dogs with other posthepatic biliary diseases and causes of cholestasis. In 78% of dogs in this study, gallbladder rupture was identified at surgery. The degree of necrosis seen via histopathology suggested that most of the dogs had acute exacerbation of chronic cholecystitis. Necrotizing cholecystitis leading to gallbladder rupture occurred secondary to common bile duct obstruction in 66% of dogs, and 81% of dogs had positive bacterial cultures from the gallbladder wall. The authors suggested that delayed diagnosis and treatment of necrotizing cholecystitis is directly related to a poor prognosis and recommend aggressive pursuit of a diagnosis. Underlying Cushing’s disease may be a contributing factor in the development of necrotizing cholecystitis in dogs because they may have a compromised immune system.

A histopathologic review of gallbladders from dogs with necrotizing cholecystitis was recently conducted at the University of Pennsylvania. Twelve dogs had evidence of gallbladder wall infarction as the cause of necrotizing cholecystitis. Thrombus formation in the cystic artery, atheromatous vascular changes, and underlying endocrine disease may be involved in the development of necrotizing cholecystitis. All 12 dogs underwent cholecystectomy, and there was a 33% mortality rate.

**Emphysematous Cholecystitis**

Emphysematous cholecystitis has been reported infrequently in dogs. This inflammatory condition induces gas formation within the wall and/or lumen of the gallbladder and is usually secondary to a combination of gallbladder wall ischemia and proliferation of gas-forming bacteria such as *E. coli* and *Clostridium perfringens*. It has been associated with diabetes mellitus but has also been observed in nondiabetic dogs. Cholecystectomy and treatment with appropriate antibiotics based on a culture and sensitivity are indicated. In a study of dogs with necrotizing cholecystitis, five of 23 dogs were also diagnosed with emphysematous cholecystitis, and three of five had positive cultures for *E. coli*.

**Trauma**

Trauma to the biliary tract is a common cause of bile peritonitis in veterinary patients. The most common
traumatic cause is being hit by a car, but gunshot wounds, bite wounds, and other penetrating abdominal wounds have also been reported as causes.22,39 Coexisting injuries involving other major abdominal organs usually do not occur. Blunt trauma causes rupture of the common bile duct more frequently than rupture of the gallbladder. Rupture has also been reported in the distal common bile duct, the cystic duct, and hepatic ducts. The most common site of common bile duct rupture is just distal to the entrance of the last hepatic duct.2,5,40,41 The pathogenesis of bile duct rupture from trauma includes the short cystic duct, rapid gallbladder emptying, and a simultaneous shearing force applied to the duct.

Recognition of biliary tract trauma is most often delayed, and clinical signs result from ensuing bile peritonitis.22 Bile peritonitis is usually the cause of death in patients with traumatic extrahepatic biliary tract rupture.

**Bile Peritonitis**

Bile peritonitis, or bilious ascites, is the inflammatory response of the peritoneum to the presence of bile. Bile peritonitis is caused by rupture of the extrahepatic biliary system. The poorly vascularized fundus is the area most susceptible to rupture in the canine gallbladder.7,41–43 Inspissated bile may cause focal or localized peritonitis rather than generalized bile peritonitis. If the bilirubin concentration of the abdominal effusion is greater than twice that of the serum concentration, it is diagnostic of bile peritonitis.21 There is a 27% to 45% survival rate for dogs with septic bile peritonitis and an 87% to 100% survival rate for dogs with sterile bile peritonitis.21,24 Humans with sterile biliary effusion may have vague symptoms that last for an average of 30 days before surgical treatment and have a mortality rate of less than 10%. Humans with septic bile peritonitis have an acute presentation with abdominal pain and shock and a mortality rate of greater than 20%.44

Clinical signs associated with bile peritonitis include vomiting, anorexia, diarrhea, weight loss, icterus, abdominal distention, fever, and abdominal pain. The onset of clinical signs in dogs with a ruptured biliary tract and the degree of peritonitis present depend on the volume of liquid bile and the concentration of bile salts. Many studies1,2,13,21,45 have identified a prolonged period (i.e., 3 to 30 days) from rupture to presentation for bile peritonitis. Previous studies13,45 have reported that a prolonged clinical course and delayed treatment of bile peritonitis lead to a higher mortality rate. Other studies22,21 have contradicted this finding.

Bile acids are toxic to tissues and cause permeability changes and tissue necrosis, which encourage bacterial growth. Sources of bacteria are thought to be endogenous anaerobic bacteria from the liver and intestine as well as hematogenous bacteria. The toxicity of bile acids depends not only on the amount of bile in the peritoneal cavity but also on the concentration of bile acids in the bile.22 Bile acids cause erythrocyte lysis, electrolyte imbalance, hypoproteinemia, anemia, and marked dehydration due to the alkaline pH and hyperosmolality of bile acids. Bile peritonitis is reportedly more lethal in dogs than in humans because of the higher content of taurocholic acid in canine bile. Taurocholic acid is considered much more toxic than the glycocholic acid component that predominates in human bile.

Survivors of biliary surgery for bile peritonitis have a significantly lower leukocyte count (mean: 20,608/µl) compared with nonsurvivors (mean: 35,712/µl). The same trend was observed in dogs with circulating bands (survivors had a mean count of 686/µl, and nonsurvivors had a mean of 4,852/µl).21

**Figure 9.** Gelatinous biliary material within the common bile duct of a dog at necropsy. The dog also had a gallbladder mucocele.
Three surgical approaches to the extrahepatic biliary system of dogs have been described. Ventral midline celiotomy is most commonly performed. This approach can be extended through the sternebrae or paracostally on the right side. A third approach involves a thoracotomy through the right seventh or eighth intercostal space.

In many cases of biliary obstruction secondary to hepatic neoplasm, it is necessary not only to use a bile rerouting procedure but also to remove some of the liver. It has been reported that up to 40% of the liver can be resected without major complications in dogs with obstructive jaundice.

Many animals requiring surgery of the extrahepatic biliary tract are nutritionally compromised. The potential for long hospital stays, concurrent or developing pancreatitis, and underlying systemic disease frequently necessitates the placement of distal duodenal or jejunal feeding tubes during laparotomy.

Gallbladder

Cholecystectomy is preferred over cholecystotomy for stone removal because cholecystectomy removes the reservoir for subsequent stone accumulation and may have a lower morbidity and mortality rate than does cholecystotomy. The gallbladder wall does not seal well immediately after cholecystocentesis or cholecystotomy. A double-layer closure and/or placing an omental patch over the incision in the gallbladder is typically indicated. Regardless of underlying disease requiring cholecystectomy, it is important to evaluate and flush the bile ducts to remove stones, debris, or sludge that may lead to reobstruction of the extrahepatic biliary tract.

Ducts

Primary repair of ruptured hepatic ducts, the cystic duct, or the common bile duct can be performed. Longitudinal tension on the suture line of a repaired bile duct can cause severe stenosis. A ruptured hepatic duct can be sacrificed because collateral drainage of bile develops. If a large tear or defect exists in the cystic duct, cholecystectomy can be performed. Because of the size of the canine common bile duct (i.e., 3 mm) compared with the human bile duct (i.e., 10 mm), choledochoenteric anastomosis is very difficult. Historically, rerouting procedures such as cholecystojejunostomy or cholecystoduodenostomy are preferred over primary repair techniques of the common bile duct; however, stenting across the common bile duct defect and suturing the tear provide a good outcome.

Biliary–Enteric Anastomosis and Enteric Stoma

The mucosa appositional technique of cholecystoduodenostomy is currently recommended for biliary redirection in dogs. The primary concern with cholecystoduodenostomy and cholecystojejunostomy is enterobiliary reflux. Cholecystojejunostomy may decrease the chances of enterobiliary reflux but increases the risk of peptic ulceration of the duodenum due to the altered physiology of the GI tract (Figure 10). When bile is diverted from the duodenum to the jejunum via a rerouting procedure, fat digestion is decreased, gastric acid secretion is increased, and neutralization of gastric acid in the duodenum is decreased. Duodenal ulcers may develop as a sequela. It has been proposed that a mechanism of decreased duodenal inhibition of gastric acid secretion plays a role in the development of duodenal ulceration. The use of H₂-blockers and proton pump inhibitors may be useful in limiting these ulcers.

Jejunal interposition (i.e., cholecystojejunoduodenostomy) may provide the most physiologic biliary diversion; however, long-term results of this procedure have not been investigated in dogs and cats. Other complications of biliary-enteric anastomosis commonly include leakage, dehiscence, stricture, and, if the enteric-biliary stoma is too small, cholangiohepatitis.

The most critical factor in biliary-enteric anastomosis is creating a large enough opening to permit drainage of refluxed intestinal contents from the biliary tract back...
into the intestine. The cholecystoenterotomy opening should be 2.5 to 4 cm long to minimize postoperative problems such as cholangitis associated with inadequate draining of refluxed intestinal contents. Clinical manifestations of cholangitis are rare when an adequately sized stoma is created.\textsuperscript{1,3,23} Cholangiohepatitis occurs when duodenal or jejunal contents are allowed to sit in the gallbladder for long periods and ascending infection occurs. Creating an incision in the gallbladder from the fundus to the beginning of the cystic duct and a corresponding incision of equal length in the intestine generally creates a stoma of adequate length. Bacterial growth within the biliary tract is expected during the initial postoperative period; however, clinical cholangitis usually does not develop unless the biliary tree becomes obstructed or the stoma is too small.

A liver biopsy should be performed during surgery for biliary disease. Histopathologic evaluation of the liver parenchyma may provide useful information regarding the underlying disease process, its effect on the liver, long-term management of the patient, and prognosis.\textsuperscript{6}

**Biliary Stents and Tubes**

Biliary decompression by means of a cholecystostomy tube may be indicated in critically ill patients until they are stable enough for a more complicated rerouting procedure.\textsuperscript{8} It is important to remember that treatment of biliary tract sepsis without biliary tract decompression is ineffective because no antibiotics achieve adequate levels in the biliary tree in the presence of EHBO.\textsuperscript{1}

In a recent report,\textsuperscript{49} eight dogs had a choledochal tube stent placed to relieve obstructions of the common bile duct and duodenal papilla due to extraluminal compression. A duodenotomy was created and the stent (i.e., red rubber catheter) placed up the duodenal papilla and into the common bile duct. The stent was then sutured in place to the mucosa of the duodenum with one or two stay sutures of an absorbable material. A hemostatic clip was placed in the lumen of the duodenal side of the stent to allow radiographic assessment of the position of the stent (Figure 11). Theoretically, after the suture breaks, the stent is passed in the feces. Discharge of the stent in the eight dogs was unpredictable; one dog required endoscopic removal of the stent during endoscopy for small intestinal biopsies. Complications of choledochal stenting include obstruction of the stent with bile concretions and promotion of stricture formation. Stricture formation can occur because the red rubber catheters may incite an inflammatory response. Intraluminal tubes may interfere with normal bile drainage, promoting cholangitis.

**Perioperative Complications**

Excessive bleeding can occur following blunt dissection and retraction of the gallbladder from the hepatic fossa, particularly in dogs with a coagulopathy.\textsuperscript{3} Assessments of coagulation factors and platelet deficiency or dysfunction should be conducted preoperatively. In dogs with hemorrhage from the hepatic fossa, a hemostatic agent can be placed in the fossa or an omental pedicle can be sutured over the area. In dogs with potential bleeding diathesis, freeing the gallbladder from the fossa can be partially or completely avoided if a duodenal or jejunal loop can be anatomically positioned adjacent to the gallbladder and biliary-enteric anastomosis can be successfully performed with minimal tension on the sutures.

Dehiscence of a biliary-enteric anastomosis can occur with improper suture placement, ischemic injury to the gallbladder, or excessive tension across the anastomosis site. An early postoperative diagnosis of bile leakage can be difficult to make because of the insidious onset of clinical signs associated with bile peritonitis. Placing a closed-suction drain intraoperatively in the cranioven-
The blood supply to the gallbladder must be preserved when performing decompressive or rerouting surgery of the biliary tract. Injury to the cystic artery can result in necrotizing cholecystitis and bile peritonitis within 48 to 72 hours. If necrosis of the gallbladder or damage (i.e., iatrogenic; torsion or infarction of the cystic artery) to the cystic arteries occurs postoperatively, cholecystectomy should be performed and the duodenal segment of the biliary-enteric anastomosis closed.

Attempts should be made to salvage the common bile duct and duodenal papilla.

Pancreatitis can result from excessive intraoperative traction and manipulation of the pancreas, which causes iatrogenic injury to the pancreatic parenchyma, ductal system, or blood supply. The pancreas is a target organ for ischemic damage resulting from systemic disturbances such as shock and sepsis. Thus the development of pancreatitis after surgery may be unrelated to manipulation of the pancreas.

**Concurrent Therapy**

If jaundice is present because of a common bile duct occlusion, vitamin K (Merck) should be administered intramuscularly or subcutaneously 12 to 24 hours before surgery. Once the flow of bile has been reestablished, enteral vitamin K (Merck) therapy can be instituted and continued for 2 weeks.

**Mortality and Risk Factors**

Surgery of the biliary tract in dogs is accompanied by significant morbidity and mortality despite recent advances in surgical techniques and supportive care. Mortality in dogs undergoing extrahepatic biliary tract surgery is 28% to 63%. Currently, it is not clear which animals undergoing extrahepatic biliary tract surgery are at high risk for mortality and may benefit from aggressive preoperative and postoperative supportive care. In humans, several studies have identified preoperative factors that define groups of patients at high risk for postoperative mortality. Mortality rates of 8% to 30% have been reported in humans undergoing biliary tract surgery for obstructive jaundice. In humans, risk factors affecting mortality after surgery for obstructive jaundice include anemia, hyperbilirubinemia, and the presence of a malignant lesion; risk factors affecting mortality after biliary tract surgery include malignancy, age (>60 years), fever, anemia, leukocytosis, azotemia, hypoalbuminemia, hyperbilirubinemia, and an increased serum alkaline phosphatase level. It has also been shown that as the number of risk factors in a patient increases, so does the correlation with mortality.

In a retrospective study of dogs undergoing extrahepatic biliary tract surgery, azotemia, postoperative hypotension, and a prolonged partial thromboplastin time were associated with increased risk for mortality.

**SUMMARY**

Clinicians should be aware of the common clinical signs, pathophysiologic changes, diagnostic tools, and surgical techniques associated with extrahepatic biliary tract disease in dogs. Particular attention is required in stabilizing patients with biliary tract disease in preparation for surgery. A high mortality rate is associated with surgical biliary tract diseases, and early surgical intervention with appropriate supportive care may improve the prognosis.

**REFERENCES**

5. Fossum TW: Surgery of the extrahepatic biliary system, in Fossum TW,
Canine Extrahepatic Biliary Tract Disease and Surgery


ARTICLE #4 CE TEST

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I. The extrahepatic biliary system does not include the

a. gallbladder.

b. interlobular ducts.

c. hepatic ducts.

d. common bile duct.

e. cystic duct.
2. Bile salts enhance absorption of fat-soluble vitamins, which do not include vitamin
   a. A.  d. E.
   b. B.  e. K.
   c. D.

3. Clinical signs of icterus develop at serum total bilirubin levels as low as _____ mg/dl.
   a. 1 to 1.5  d. 8 to 9
   b. 1.5 to 2  e. 10 to 10.5
   c. 4 to 4.5

4. Bilirubinuria may be the first sign of bile duct obstruction in dogs and may precede the development of jaundice because dogs
   a. have a low renal threshold for excretion of unconjugated bilirubin.
   b. are unable to deposit conjugated bilirubin in their mucous membranes.
   c. are predisposed to renal failure from hyperbilirubinemia.
   d. have a low renal threshold for excretion of conjugated bilirubin.
   e. cannot unconjugate bilirubin.

5. Which statement is incorrect?
   a. Hepatobiliary scintigraphy can be a valuable diagnostic tool in differentiating EHBO from hepatocellular disease.
   b. Biliary obstruction can be diagnosed before the onset of clinical icterus with the use of abdominal ultrasonography.
   c. Bile is thought to be sterile in normal dogs and cats.
   d. Cholelithiasis always occurs in a sterile environment.
   e. Infection of the extrahepatic biliary tract commonly involves multiple organisms.

6. Reported causes of extraluminal EHBO do not include
   a. pancreatitis.
   b. pancreatic pseudocyst.
   c. pancreatic tumors.
   d. portal vein infarction.
   e. hepatic neoplasia.

7. Which statement regarding cholelithiasis is incorrect?
   a. Cholelithiasis is a condition of senior, female small-breed dogs and is often an incidental finding at necropsy or via radiology.
   b. Choleliths have been associated with vomiting, anorexia, icterus, fever, or abdominal pain.
   c. Choleliths commonly form in the lobar ducts and travel into the gallbladder or common bile duct.
   d. Pigment stones are yellow, dark brown, or black; vary in weight and fragility; and are common in dogs with cholelithiasis.
   e. Canine cholelithiasis may be rare because of decreased concentrations of cholesterol in canine bile compared with those in human bile.

8. Which statement regarding gallbladder mucoceles is incorrect?
   a. Biliary mucoceles occur in dogs older than 6 years of age and have no breed or sex predilection.
   b. In some cases, gelatinous biliary material is present within the common bile duct and needs to be flushed out.
   c. Gallbladder mucoceles occur secondary to neoplastic changes in the hepatic ductule system.
   d. There are reports of a 50% to 60% incidence of gallbladder rupture in patients with biliary mucoceles.
   e. Dogs with biliary mucoceles that undergo cholecystectomy and survive the immediate perioperative period have an excellent prognosis.

9. Which statement regarding bile peritonitis is incorrect?
   a. The pathogenesis of bile duct rupture from trauma includes the short cystic duct, rapid gallbladder emptying, and a simultaneous shearing force applied to the duct.
   b. The poorly vascularized fundus is the area most susceptible to rupture in the canine gallbladder.
   c. The presence of bile in the peritoneum causes immediate, rapid, and severe peritonitis.
   d. Dogs with septic bile peritonitis have a higher mortality than do dogs with sterile bile peritonitis.
   e. Bile peritonitis is usually the cause of death in patients with traumatic extrahepatic biliary tract rupture.

10. Complications of biliary-enteric anastomosis do not include
    a. stricture.
    b. dehiscence.
    c. enterobiliary reflux.
    d. cholecystojejunal intussusception.
    e. hemorrhage from the hepatic fossa.