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Feline Liver Disease

Susan A. Morrison, D.V.M.

Feline liver disease can be a diagnostic and therapeutic challenge. There are features of hepatic metabolism and anatomy unique to the cat that are important for the understanding of feline liver disorders. This article provides a general overview of the pathophysiology, diagnosis, and management of various feline primary liver disorders (an in-depth discussion of specific therapeutic considerations is included in reference 1). However, it is important to realize that in the cat certain biochemical parameters associated with the liver may be altered secondary to other systemic diseases such as hyperthyroidism, diabetes mellitus, endotoxemia, septicemia, anorexia, hyperthermia, thromboembolism, and cardiovascular failure. Consequently, a complete metabolic evaluation should be done to help determine the primary cause of changes in the liver.

Anatomy

The hepatic blood supply is made up of two-thirds deoxygenated blood from the portal vein and one-third oxygenated blood from the hepatic artery. Those vessels and a bile ductule make up the portal triads at the periphery of the hepatic lobules. Blood flows from the periphery of the hepatic lobules within the sinusoids toward the hepatic vein. Therefore, the hepatocytes, particularly those near the central vein farthest from the portal triad blood supply, are most sensitive to hypoxic injury.

The major anatomic feature distinguishing the feline hepatic anatomy from the canine is that in the cat one of the pancreatic ducts joins the common bile duct before its entry into the duodenum. Because of that close anatomical association, retrograde flow of enzyme- and bile-rich fluid from the enteric lumen could cause inflammation and resulting fibrosis in either the pancreatic or the hepatic system. (Refs. 1, 11)

Physiology

A unique feature in feline hepatic physiology is the deficiency in glucuronyl transferase. That enzyme is necessary for glucuronic acid conjugation. Most substances are excreted by cats as water-soluble ionic compounds because, once secreted, they cannot diffuse back across the tubular cell membranes and are trapped outside the body. Glucuronic-acid conjugation is the major pathway by which fat-soluble compounds are changed to water-soluble ones. The natural deficiency of that pathway in the cat can result in toxicity from insufficient excretion of many substances, especially drugs such as aspirin and morphine.

Another important physiologic feature of cats is the inability to synthesize arginine. That amino acid is involved in the conversion of ammonia to urea. Lack of appropriate dietary sources induces muscle catabolism to make arginine available, increasing the susceptibility of poorly nourished cats to hyperammonemia and resultant encephalopathy.

The rest of the feline liver functions are glycogen formation and storage; gluconeogenesis; protein, albumin, and fat synthesis; carbohydrate metabolism; transport-globulin synthesis; vitamin storage; clotting factor synthesis; and hormone and bile metabolism. They show no features unique to the cat. (Ref. 8)

Clinical Signs

Cats often are not presented until late in the course of liver disease because the species is so efficient in masking clinical signs. The associated clinical signs are common to many other diseases; they include anorexia, vomiting, diarrhea, fever, weight loss, dehydration, seizures, and occasional polyuria and polydipsia. Because those findings are not specific for liver disease, a thorough metabolic evaluation is necessary.

Hepatomegaly and jaundice may be noticed on physical examination. In general, cats do not present with small livers. Chorioretinitis or anterior uveitis can be seen with systemic diseases that may affect the liver, such as feline infectious peritonitis and toxoplasmosis. Therefore, a thorough fundoscopic examination is always warranted. (Refs. 1, 8, 9, 11)

Hematologic Abnormalities

Hemogram A nonregenerative anemia is very often seen in cats with hepatic disorders. Possible causes for the condition are decreased protein metabolism, decreased synthesis of globulin necessary for erythropoiesis, decreased survival time for red blood cells, and anemia resulting from chronic disease. A regenerative anemia secondary to blood loss from coagulation abnormalities may also occur. Poikilocytes such as schistocytes may be seen.

The white-blood-cell count may be normal, or a leukocytosis resulting from stress, necrosis, or infection may exist. (Refs. 8, 11)

Coagulation Profile The prothrombin time (PT) and partial thromboplastin time (PTT) may be prolonged by hepatobiliary disease for two reasons. First, in severe chronic hepatocellular disease, the liver may be insufficient in its production of the clotting-factor proteins I, II, V, VII, IX, and X. Second, the clotting factors whose production depends on the presence of vitamin K (II, VII, IX, and X) may be depleted during complete biliary obstruction because vitamin K is fat soluble, and bile is necessary for it to be absorbed from the intestines.

Severe diffuse hepatocellular injury can cause the release of tissue thromboplastins and a decreased ability to clear clot promoting factors, resulting in disseminated intravascular coagulation.

Thrombocytopenia and thrombocytopenia also may occur in cats with liver disease. Fibrinogen production may be decreased by hepatic insufficiency or be increased by inflammatory hepatic disease or cholestasis. (Refs. 3, 11)

Urinalysis

Isosthenuria may result from the medullary washout that is caused by a diseased liver's decreased production of urea, which is necessary for the maintenance of the medullary concentration gradient. Bilirubin metabolism and the significance of bilirubinemia and urobilinogen are addressed in the section entitled "Plasma Proteins."

Biochemical Testing

Liver Enzymes: Alanine Amino Transferase and Aspartate Aminotransferase Alanine

amino transferase (ALT) is a liver-specific enzyme located within the hepatocytes in the cat. The serum ALT value increases with changes in hepatocellular membrane permeability that may occur with hepatic injury such as necrosis, inflammation, fatty infiltration, hypoxia, and cholestasis. The serum half-life is less than twenty-four hours; therefore, continued elevation of ALT activity suggests ongoing hepatocellular injury.

Aspartate aminotransferase (AST) is an enzyme present in the cat primarily in cardiac muscle, skeletal muscle, hepatocytes, and erythrocytes. Like ALT activity, serum AST activity increases with hepatocellular injury. However, its presence in other tissues makes serum elevations of AST less liver specific.

Serum ALT and AST activities can also be increased by drug-induced microsomal enzyme induction in the dog. That does not seem to occur in the cat. (Refs. 3, 11)

Serum Alkaline Phosphatase Serum alkaline phosphatase (SAP) is produced in many tissues. However, because of the enzyme's small concentrations and brief half-life, only hepatic and bone origin are considered to be of clinical significance in the cat.

The SAP of hepatic origin is a microsomal enzyme produced by tissues of the biliary tree during intrahepatic or extrahepatic induced cholestasis. In the dog, SAP can also be induced by drugs such as glucocorticoids. That phenomenon has not been recognized in the cat.

Another species difference is the short half-life of SAP in the cat, about six hours compared to seventy-two hours in the dog. Therefore, even mild increases (2- to 3-fold) are significant in the cat.

The SAP of bone origin may contribute to two- to threefold increases in the enzyme in young, growing, kittens and in mature cats in association with primary bone tumors, secondary renal hyperparathyroidism, and hyperthyroidism. (Refs. 3, 11)

Gamma Glutamyl Transferase Gamma glutamyl transferase (GGT) is located within the cytosol and is membrane bound in most cells. The GGT of hepatic origin is the most clinically significant in the cat and increases primarily as a result of intrahepatic or extrahepatic cholestasis, similarly to SAP. However, GGT seems to be more sensitive to hepatic disorders than SAP is and may be increased more markedly in the cat in the presence of cirrhosis, major bile-duct obstruction, or intrahepatic cholestasis. Idiopathic hepatic lipidosis is the one condition in which GGT values in the cat do not parallel SAP values. In cats with that disorder, SAP may be increased five- to twentyfold while GGT may remain normal or only slightly elevated. In the dog, similar enzyme-inducing agents that affect SAP also will affect GGT. That has not been recognized in the cat. (Refs. 3, 4)

Plasma Proteins

Albumin Albumin is synthesized in the liver. That function is maintained until there is severe hepatic failure (80% reduction in functional mass). Hypoalbuminemia is uncommon in cats with liver disease, but when present, indicates end-stage liver disease.

Albumin is the plasma protein that is primarily responsible for the plasma colloid osmotic pressure. When albumin values decline below 2.0 grams per deciliter, extravascular fluid accumulation is likely to develop in the form of ascites, anasarca, pleural effusion, or localized peripheral edema. Therefore, a hypoalbuminemic patient receiving intravenous fluid therapy must be closely monitored for overhydration. (Refs. 3, 11)

Globulin In a cat with normal hepatic function, antigens from the portal circulation are cleared by the hepatic reticuloendothelial system. In a cat with chronic liver disease, hepatic reticuloendothelial function is insufficient, allowing antigens to reach the systemic circulation and resulting in increased immunoglobulin production.

There is also a compensatory regulatory mechanism that attempts to maintain plasma-colloid osmotic pressure by increasing serum globulins when concurrent severe hypoalbuminemia exists. (Refs. 3, 11)

Glucose Serum glucose values may be increased or decreased in cats with liver disease. Cats are prone to stress hyperglycemia. Cats with liver disease therefore may present with hyperglycemia due to stress or an associated diabetes mellitus.

Hypoglycemia, although uncommon in cats with liver disease, may be present if hepatic glycogen storage and gluconeogenesis is insufficient to maintain normal blood-glucose levels. (Ref. 11)

Blood Ammonia Ammonia is an end product of protein metabolism that is produced by intestinal microbial urease activity on endogenous urea and dietary amines. It is then transported to the liver via the portal circulation, where it is detoxified to urea. Normal control of blood ammonia concentration requires functional hepatic mass, a normal hepatic portal circulation, and sufficient dietary arginine and other urea-cycle enzymes. Elevations of blood-ammonia levels after fasting or during ammonia-tolerance testing may indicate an abnormality in one, or in a combination, of those components. (Ref. 3)

Bilirubin Bilirubin is synthesized primarily from heme pigments of senescent red blood cells. Unconjugated water insoluble bilirubin is protein bound in circulation and transported to the liver, where it is taken up by the hepatocytes and conjugated to form water-soluble bilirubin glucuronide. That conjugated bilirubin may then be stored within the hepatocytes or

excreted in the bile. The biliary excretion is the rate-limiting step. In the small intestine, bilirubin is degraded to urobilinogen. The majority of urobilinogen is oxidized and excreted in the feces. Ten to 15 percent is reabsorbed and secreted in the urine or recirculated through the liver.

Quantitative differences in unconjugated and conjugated bilirubin in the cat are of little diagnostic importance clinically, because unconjugated bilirubinemia exists only temporarily without associated conjugated bilirubinemia. The appearance of urobilinogen in feline urine indicates an intact enterohepatic circulation. However, the presence of even trace amounts of bilirubin in the urine of the cat (unlike in the dog) is considered abnormal and an indication of hemolytic or hepatobiliary disease.

Jaundice develops when total serum bilirubin increases to more than 1.5 to 2.0 milligrams per deciliter as a result of a circulatory overload of heme pigments. That can be caused by hemolytic disease, insufficient hepatic uptake, storage and conjugation of bilirubin during intrahepatic disease, and decreased biliary excretion from either intrahepatic or extrahepatic cholestasis.

It has been proposed that there is a distinction between biochemical jaundice (<3 mg% of serum bilirubin) and clinical jaundice (>3 mg%) in the cat because the cat is prone to hyperbilirubinemia caused by fasting (in which the serum bilirubin does not usually exceed 3 mg%). Anorexia can deplete the intrahepatocellular protein with which bilirubin is bound and stored. A deficiency or saturation of that protein affects the ability of the hepatocyte to retain the bilirubin. Therefore, biochemical jaundice (<3 mg%) does not necessarily indicate specific hemolytic or hepatobiliary disease in the cat. (Refs. 3, 8, 11)

Liver-Function Tests

Liver-function tests such as indocyanine green or sulfobromophthalein clearance and ammonia tolerance have been well described in the literature. Because of its sensitivity, specificity, safety, and convenience, the preferred liver-function test at our clinic is the measurement of bile-acid levels during fasting and two hours postprandial.

As previously described, circulating bile acids are conjugated in the liver and transported via the biliary system to the gallbladder for storage. Following a meal, gallbladder contraction is induced that results in secretion of bile into the intestines. In the intestine the bile acids function in the digestion and absorption of fat. Most of the bile acids are reabsorbed in the ileum and extracted from circulation by the liver. That enterohepatic circulation is very efficient in the normal cat. Ileal malabsorption of bile acids may cause decreased serum values of bile acid during fasting and post-

prandial. Conversely, increased serum values may be caused by impaired hepatic circulation, uptake, storage, or excretion of bile acids. In our laboratory, normal bile-acid values in the cat are ≤ 2.0 micromoles per liter during fasting and 10.0 micromoles per liter two hours postprandial.

Although biochemical abnormalities may be strongly suggestive of hepatobiliary disease in the cat, they are not adequate assessments of liver function. Liver-function tests are necessary to confirm actual decreased functional hepatic mass, thus justifying more-invasive diagnostic techniques such as liver biopsy or exploratory laparotomy. In our clinic a bile-acid value consistently exceeding 20 micromoles per liter in a cat warrants a liver biopsy.

In most cases a liver biopsy is necessary to establish a specific diagnosis of hepatobiliary disease in the cat, a prognosis, and an appropriate therapeutic regimen. One of three techniques may be used: (1) percutaneous needle biopsy (blind or ultrasound guided), (2) laparoscopy, (3) surgical laparotomy. Those techniques have been well reviewed in the literature. The risks of each technique must be weighed against the benefits of the information gained. When an extrahepatic biliary obstruction is suspected, surgical laparotomy is the technique of choice.

A coagulation profile should be evaluated before any biopsy technique is performed. Despite obtaining normal coagulation-test values, fresh whole blood should also be available for transfusion during a biopsy because there is a change in coagulation test values only if a coagulation factor is <30 percent of normal activity.

Serologic testing for feline leukemia virus, feline infectious peritonitis, and toxoplasmosis should also be included for all feline patients suspected of liver disease, because of the association of those diseases with some feline liver disorders. (Refs. 3, 5, 7–11)

Intrahepatic Diseases

Hepatic Lipidosis Hepatic lipidosis is a common liver disease in cats, in which hepatocellular accumulation of triglycerides develops from a disruption in hepatic lipid metabolism and results in severe liver dysfunction. In a review of 150 cases, the most-common clinical findings reported were anorexia of two to three weeks duration, jaundice, hepatomegaly, weight loss, and dehydration. Hepatic encephalopathy and bleeding disorders also have been reported.

Hepatic lipidosis can be initiated by a number of endocrine, nutritional, metabolic, toxic, and anoxic causes (see the table). Often in the cat an underlying disease cannot be identified, and the condition is termed idiopathic hepatic lipidosis (IHL).

Sources of hepatic lipids are dietary fatty acids and triglycerides released from adipose tissues. Within the hepatocyte, fatty acids may be oxidized or reesterified and secreted as lipoproteins. Several causes of IHL have been proposed, including overnutrition, protein-carbohydrate imbalance, and obesity. In cats, overnutrition may lead to intrahepatocellular fat accumulation by saturating the oxidation and transport pathways.

A protein-carbohydrate imbalance can occur when a relative protein deficiency leads to an insufficiency of transport proteins, which are necessary for the hepatocellular secretion of triglycerides. Concurrent ingestion of carbohydrates causes further mobilization of fatty acids to the liver. If the caloric intake of those carbohydrates is not sufficient, incomplete oxidation of the fatty acids will further enhance the lipid accumulation.

Obesity has been a repeated historical finding in many cats afflicted with idiopathic hepatic lipidosis. In obese people, peripheral insulin resistance resulting in hepatic fat accumulation has been documented. Although that relationship has not yet been documented in the cat, obesity and subsequent fatty-acid mobilization may overwhelm the ability of insulin to suppress the fatty acids. That can result in an insulin-resistant state that would allow continued release of fatty acids from adipose tissue, thus promoting hepatic lipid accumulation. There have been a few reports of successful use of low-dose insulin treatment of IHL in cats. However, its effectiveness remains speculative and is not advised by most authors.

Clinicopathologic abnormalities associated with IHL include marked elevations in serum-aminotransferase and alkaline-phosphatase activities. As previously mentioned, in cats with IHL, the magnitude of the increase of GGT does not seem to correlate with the remarkable magnitude of the increase of SAP.

The definitive diagnosis of IHL is based on histopathologic findings of hepatocellular vacuolation and the absence of another concurrent disease or hepatobiliary disorder that could promote hepatic lipid accumulation.

Although initial treatment includes restoring hydration, electrolyte, and vitamin B deficits, the most-important aspect of therapy is the long-term maintenance of a positive caloric and protein balance. Placement of a nasogastric, gastrotomy, or pharyngostomy tube is usually necessary to achieve that balance.

Appetite stimulants such as the benzodiazepines diazepam (Valium®—Roche) and oxazepam (Serax®—Wyeth) can be used as an alternative to force-feeding. That treatment has been met with limited success at our clinic.

The total caloric intake should be 80 to 100 kilocalories per kilogram per day. If the

patient is being tube-fed, that requirement can be fulfilled with a well-balanced commercial cat food made into a gruel. Initially, the total daily requirement should be divided into six to eight feedings (5–10 ml/kg each). The amount of each feeding can be gradually increased while the daily number of feedings is decreased.

The total daily fluid requirement of 50 to 60 milliliters per kilogram can be supplied orally, subcutaneously, or intravenously. Because a protein-carbohydrate imbalance has been suggested as a possible cause of hepatic lipidosis, the indiscriminate use of glucose supplementation could be detrimental to the lipidotic cat. It has been shown that the intravenous infusion of glucose stimulates hepatic fatty-acid production and if protein intake is inadequate, promotes the accumulation of lipid within the liver. Therefore, parenteral glucose supplementation should only be provided to a lipidotic cat that is hypoglycemic. (Refs. 1–3, 6, 8, 9, 11)

Cholangiohepatitis Complex The term *cholangiohepatitis* is used to describe a complex of related inflammatory hepatobiliary disorders. Those have been categorized according to the predominant inflammatory cellular infiltrate seen on histopathologic examination: (1) suppurative (neutrophils) cholangitis-cholangiohepatitis, (2) chronic nonsuppurative (lymphocytes, plasma cells) cholangiohepatitis, (3) biliary cirrhosis (diffuse fibrosis of the extrahepatic and sometimes intrahepatic biliary system). Although little is known about that complex of diseases, it is believed by some that each represents a different stage in the progression of one syndrome.

Clinical signs are nonspecific and may include intermittent anorexia, fever, lethargy, vomiting, soft stools, and jaundice. Biochemical testing usually shows marked increases in serum bilirubin and ALT (2- to 40-fold) and moderate increases in SAP and GGT (2- to 5-fold). Definitive diagnosis and treatment are based on histopathologic examination, culture (aerobic and anaerobic), and sensitivity of bile and hepatic tissue.

Three to five weeks of specific antibiotic therapy is indicated for the suppurative form of the disease. The nonsuppurative form requires immunosuppressive treatment with glucocorticoids (prednisolone: 2.2 mg/kg SID initially, then tapered to 1.1 mg/kg/day every other day to control the signs). The aim of therapy is to control the disease process. However, a complete cure is rare.

Biliary cirrhosis is believed to be the final stage of the cholangiohepatitis complex. It is uncommon in cats because those with cholangiohepatitis rarely survive long enough to develop that sequela. There is extensive fibrosis and evidence of chronic inflammation in the hepatobiliary system and most often in the pancreas.

At that stage, serum GGT may be the

Liver Diseases Commonly Encountered in Cats

Extrahepatic Bile-Duct Obstruction

Common bile-duct strictures:
 pancreatic inflammation/fibrosis
 duodenal inflammation
 neoplasia (bile-duct adenocarcinoma)
 cholangitis
 Common bile-duct obstructions:
 cholelithiasis
 sludged bile
 parasites (liver flukes)

Cholangitis-Cholangiohepatitis Syndrome

Inflammation ascending up biliary tree:
 pancreatitis
 duodenal inflammation
 spontaneous reflux of pancreatic enzymes, ingesta, microorganisms
 Cholelithiasis
 Sludged bile (cause or effect)
 Parasites (liver flukes)
 Copper accumulation (?)
 Immunological mediation

Biliary Cirrhosis

Chronic extrahepatic bile-duct obstruction
 Chronic cholangitis-cholangiohepatitis syndrome

Hepatic Necrosis

Toxins:
 bacterial
 fungal
 drugs (acetaminophen)
 organophosphates
 heavy metals
 plants
 Hypoxia:
 anemia
 shock
 congestive heart failure
 thromboembolism
 Infectious diseases:
 feline infectious peritonitis
 calcivirus
 toxoplasmosis
 septicemia
 endotoxemia

Hepatic Lipidosis

Obesity
 Prolonged overnutrition
 Chronic anorexia
 Protein malnutrition
 Endocrine disorders (diabetes mellitus)

Hypoxia

Toxins:
 bacterial
 drugs (tetracycline)
 chemicals (carbon tetrachloride, alcohol)
 plants
 Idiopathic

Neoplasia

Primary (rare):
 hepatocellular carcinoma
 bile-duct carcinoma
 hemangiosarcoma
 Secondary:
 lymphosarcoma
 myeloproliferative disease
 systemic mastocytosis

Portosystemic Vascular Anastomosis

Congenital:
 left gastric vein
 portal azygous
 portal vena cava
 multiple portal vena cava
 patent ductus venosus
 Secondary (rare)

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only liver enzyme that is increased, because the rest of the liver enzymes would have been depleted by the previous chronic inflammation. In contrast to that of the dog, the cirrhotic liver in the cat is not small and may, in fact, be enlarged. Hypoalbuminemia and ascites, although a rare sequela of feline liver disease, may be present at that advanced stage. The prognosis is very poor. Medical management of the stage is primarily supportive and seeks to maintain adequate hydration and nutrition. Ideally, the nutrient requirements should be provided by a primarily carbohydrate diet in which the minimum protein requirement is supplied by a protein of high biologic value. However, severe protein restriction is not recommended, because cats require a high level of dietary protein (especially of arginine) to decrease the potential for catabolism of body proteins for energy. A multiple-vitamin supplement should also be included.

Corticosteroid administration at that stage is not recommended for three reasons: (1) corticosteroids may induce sodium retention, thus promoting ascites; (2) corticosteroids can induce protein catabolism, thus favoring the development of hepatic encephalopathy; and (3) cortico-

steroids may hasten the general loss of body condition. (Refs. 1, 3, 9, 11)

Hepatic Necrosis Acute hepatic necrosis in the cat may be caused by infectious diseases such as endotoxemia or septicemia; drug, chemical, or plant toxicities; or hypoxia (see the table).

Serum ALT and AST activities may be markedly increased, twenty- to fortyfold, while GGT and SAT values may be normal or increased about threefold. During a period of two to three weeks following the injury, those enzymes will gradually decline to normal levels. Cats with acute hepatic necrosis may be hyperbilirubinemic or have coagulation abnormalities that do not correct with vitamin K if the lesion is diffuse.

Initial treatment should involve removal or binding of the toxic substance (if such is suspected) by using saline enemas or activated charcoal given by stomach tube. Supportive care should include parenteral antibiotics and fluid therapy with B complex vitamins, potassium, and glucose supplementation. Dietary considerations are the same as those included in the section on hepatic lipidosis. (Refs. 1, 3, 11)

Extrahepatic Bile-Duct Obstruction Extrahepatic bile-duct obstruction (EHBDO)

may be caused by neoplasia or inflammation and stricture of the bile duct or its associated organs (pancreas, duodenum). Less commonly, choleliths or parasitic migration may result in EHBDO in the cat. The presenting clinical signs may include anorexia, fever, intermittent vomiting, and weight loss. Jaundice also may be present.

Biochemical testing usually reveals marked increases in serum bilirubin, cholesterol, GGT, SAP, and transaminase activities. Both fasting and postprandial serum bile-acid values show dramatic increases of up to 100-fold.

If complete biliary obstruction exists, urine urobilinogen decreases to undetectable levels, stools become acholic, and bleeding tendencies develop that are responsive to vitamin-K administration.

Radiographic evaluation of the abdomen or the thorax may occasionally be helpful in identifying an abdominal mass or a metastatic disease, respectively. Ultrasonography may be used to determine if extrahepatic biliary obstruction is present and can sometimes better elucidate the etiology, such as cholelithiasis or obstructive masses.

Surgical intervention and correction are ultimately necessary in the majority of

cases of EHBDO. That may involve a cholecystotomy, a biliary culture, and a liver biopsy if cholelithiasis is present or require more-extensive biliary-diversion techniques if a stricture of the common bile duct, a nonresectable mass, or severe pancreatic fibrosis is present.

A coagulation profile and thoracic radiography to check for metastatic disease should be included in the presurgical evaluation of patients with EHBDO.

Prophylactic use of vitamin K for forty-eight hours before surgery is strongly recommended even if normal coagulation tests have been obtained, because there may be a factor deficiency of up to 70 percent.

If cholelithiasis is present, postoperative care should include long-term (4–6 weeks) antibiotic therapy based on biliary culture and sensitivity results, and management of any underlying hepatic disorder determined by the liver biopsy. Postoperative hydrocholeretic treatment using dehydrocholic acid (10–15 mg/kg PO TID) has also been advised. Supportive care with balanced electrolyte fluid therapy, B vitamins, and glucose and potassium supplements will be necessary in all postoperative cases. (Refs. 1, 3, 11)

Portosystemic Vascular Anomalies Cats with portosystemic vascular anomalies (PSVA) may demonstrate encephalopathic signs, poor growth rate, anorexia, ptyalism, depression, vomiting, diarrhea, and polydipsia. The neurologic signs may or may not be associated with meals.

Physical-examination results may be normal. Biochemical parameters may be normal or show mild decreases in blood-urea nitrogen (BUN) and blood glucose. Very mild increases in ALT and AST may occasionally be seen. The most-remarkable abnormality in cats with PSVA is the decreased hepatic function that may be demonstrated by increased bile-acid values.

Ultrasonography or mesenteric angiography is necessary to confirm a diagnosis of PSVA. Surgical correction by partial to complete ligation of the anomalous vessel is the recommended therapy. Medical therapy before surgery primarily involves the prevention of hepatic encephalopathy. Balanced electrolyte fluid therapy supplemented with potassium and 2.5 to 5.0 percent dextrose is usually necessary to correct dehydration and prevent the hypoglycemia that patients with PSVA are especially prone to develop. Hypoglycemia, hypokalemia, and metabolic alka-

losis should be avoided, because they exacerbate hepatic encephalopathy. (Refs. 1, 5, 11)

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About the Cornell Feline Health Center

The ultimate purpose of the Cornell Feline Health Center is to improve the health of cats by developing methods to prevent or cure feline diseases and by providing continuing education to veterinarians and cat owners. The Cornell Feline Health Center is a nonprofit organization supported largely by private tax-deductible contributions. Correspondence may be directed to:

Cornell Feline Health Center
Cornell University
College of Veterinary Medicine
Ithaca, New York 14853-6401

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