GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Hepatectomy refers to removal either of the entire liver (total hepatectomy) or of a portion of the liver (partial hepatectomy).

PREOPERATIVE MANAGEMENT

The liver is the largest gland in the body. It is the primary site of the metabolism (detoxification) of many substances and plays a central role in the metabolism of protein, fat, and carbohydrates. Unfortunately, clinical signs of hepatic disease may not be apparent until the disease is advanced and dysfunction is irreversible. Hepatic failure may affect many other organ systems, including the central nervous system (CNS), kidneys, intestines, and heart.

The liver produces most of the plasma proteins, including albumin, α-globulins and β-globulins, fibrinogen, and prothrombin. Hypoalbuminemia is common in patients with advanced hepatic disease. Fluid therapy may further dilute the serum albumin; plasma or colloid infusions should be considered in these patients, in addition to electrolyte solutions. Albumin levels below 2 g/dl may be associated with delayed wound healing. Electrolyte abnormalities are common in patients with hepatic disease but except for potassium alterations are seldom severe. Coagulopathies may occur because of diminished synthesis of clotting factors or consumption. Preoperative evaluation of clotting function, especially the mucosal bleeding time, is warranted; transfusions with fresh whole blood may reduce intraoperative hemorrhage in selected patients. Some patients with hepatic disease are anemic because of nutritional deficiencies, coagulation abnormalities, or gastrointestinal hemorrhage. Animals with a hematocrit below 20% or anemic animals that are clinically hypoxic or weak should be given preoperative blood transfusions (see Table 5-4 on p. 551). Many patients with liver disease are anorexic and may require nutritional supplementation before surgery (see Chapter 11). Hypoglycemia sometimes occurs with severe hepatic insufficiency; monitoring of blood glucose levels and supplementation of fluids with glucose may be needed. Patients with massive ascites may have ventilatory disturbances as a result of diaphragmatic displacement and restriction of lung expansion. In such patients, removing some abdominal fluid before induction of anesthesia may help prevent hypoventilation.

NOTE: The cranial location of the liver may make hepatic biopsy somewhat difficult, particularly in large, deep-chested breeds or when the liver is abnormally small. In these animals, extend the incision as far cranially as necessary to facilitate hepatic exposure.

ANESTHESIA

In animals with hepatic dysfunction, the ability to metabolize and inactivate some drugs may be impaired because of decreased hepatic metabolism, hepatic blood flow, volume of distribution (i.e., of drugs that are highly protein bound), and extraction efficiency. Consequently, drugs commonly used to anesthetize veterinary patients may have a prolonged duration of action or altered function. Acetylpromazine is believed to lower the seizure threshold and should not be used in patients with severe hepatic insufficiency and/or encephalopathy. It also lowers systemic vascular resistance and blood pressure and may alter the metabolism of some drugs (i.e., procaine and succinylcholine).

Diazepam (Box 20-1) is useful as a premedicant or induction agent in patients with hepatic dysfunction because it causes mild, dose-related CNS depression, does not depress the cardiopulmonary system, raises the seizure threshold, and can be antagonized with flumazenil. Diazepam is best used in conjunction with an opioid because it may disinhibit some behaviors when used alone. It should be used with caution in hypoalbuminemic patients. Most opioids have little or no adverse effect on the liver; however, intravenous morphine should be avoided in dogs with hepatic dysfunc-
**BOX 20-1**

Selected Anesthetic Agents for Animals With Hepatic Disease Causing Hepatic Insufficiency*

| Premedication | Diazepam (0.2 mg/kg IV) plus etomidate (0.5–1.5 mg/kg IV) or propofol (4 mg/kg IV to effect); as an alternative, if the patient is not vomiting, use mask induction or give thiopental at reduced doses |
| Maintenance | Isoflurane or sevoﬂurane |

*See p. ••• for recommendations for patients with portosystemic shunts.
†Use 0.01 mg/kg in cats.
SC, Subcutaneous; IM, intramuscular; IV, intravenous.

**BOX 20-2**

Antibiotics in Animals With Hepatocellular Compromise

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ampicillin</td>
<td>22 mg/kg IV, IM, or SC, tid to qid</td>
</tr>
<tr>
<td>Metronidazole (Flagyl)</td>
<td>10–15 mg/kg PO, tid</td>
</tr>
<tr>
<td>Cefazolin (Ancef, Keftzol)</td>
<td>20 mg/kg IV or IM, tid to qid</td>
</tr>
<tr>
<td>Clindamycin (Antirobe)</td>
<td>11 mg/kg IV or PO, bid to tid</td>
</tr>
</tbody>
</table>

IV, Intravenous; IM, intramuscular; SC, subcutaneous; tid, three times a day; qid, four times a day; PO, oral; bid, twice a day.

NOTE: Metronidazole, when administered at doses above 50 mg/kg of body weight per day, can cause severe neurologic signs (e.g., central vestibular signs including ataxia, nystagmus, head tilt, and seizures) in some dogs.

**SURGICAL ANATOMY**

The diaphragmatic surface (parietal surface) of the liver is convex and lies mainly in touch with the diaphragm. The visceral surface faces caudoventrally and to the left, and contacts the stomach, duodenum, pancreas, and right kidney. There are six hepatic lobes (Fig. 20-1). The borders of the liver are normally sharp, but appear more rounded in young animals and in those with infiltrated, congested, or scarred livers. The liver has two afferent blood supplies, a low-pressure portal system and a high-pressure arterial system. The portal vein drains the stomach, intestines, pancreas, and spleen and supplies four fifths of the blood that enters the liver. The remainder of the afferent blood supply is from the proper hepatic arteries. These arteries are branches of the common hepatic artery and may number between two and five. The efferent drainage of the liver is through the hepatic veins. In the fetal pup, the ductus venosus shunts blood from...
the umbilical vein to the hepatic venous system. The ductus venosus becomes fibrotic after birth and is known as the ligamentum venosum. Bile, formed in the liver, is discharged into bile canaliculi lying between the hepatocytes. These canaliculi unite to form interlobular ducts that ultimately merge to form lobar or bile ducts (see p. ***). The portal vein, bile ducts, hepatic artery, lymphatics, and nerves are contained in the lacelike and nonsupporting portion of the lesser omentum known as the hepatoduodenal ligament.

NOTE: Use caution when dissecting around the pylorus to prevent damaging the common bile duct.

**SURGICAL TECHNIQUE**

Surgery of the liver is complicated by the fact that hepatic tissue is friable. Because of the sparsity of fibrous protein in the liver, sharp dissection is difficult and results in retraction of blood vessels and bile ducts in the friable stroma. Ligation of structures (i.e., blood vessels and bile ducts) after they have been cut is extremely difficult. Packing the liver firmly enough to obtain hemostasis may cause compressed cells to become ischemic and necrotic. Maintaining hepatic blood supply is important because the liver normally harbors pathogenic anaerobes. For these reasons, surgery of the liver requires techniques different from those used in surgery on most other abdominal organs.

Hepatic biopsies are commonly indicated in patients known to have or suspected of having hepatic disease. The biopsies may be obtained percutaneously, with laparoscopy (see p. **), or at surgery. Partial hepatectomies are less commonly performed, but may be indicated for focal neoplasms or trauma. The standard approach for hepatic surgery is a cranial ventral midline abdominal incision. The caudal aspect of the sternum can be split if additional exposure is needed.

NOTE: Be sure to obtain a liver biopsy in all patients with clinical signs or laboratory abnormalities consistent with hepatic disease, or whenever the liver appears grossly abnormal.

**Percutaneous Liver Biopsy**

Percutaneous core biopsies or fine-needle aspirations are relatively inexpensive, easy techniques that can be sensitive and specific for focal lesions (i.e., tumors such as carcinomas and lymphosarcoma) when used with ultrasound guidance. However, with the exception of feline hepatic lipodiosis, these percutaneous techniques are very insensitive and unreliable in patients with diffuse hepatic disease (i.e., inflammation, fibrosis, cirrhosis, and necrosis) and those that may have congenital vascular shunts, and are not recommended in these cases. Animals with clinical bleeding, severe thrombocytopenia (i.e., fewer than 20,000 platelets/μl), cavitory lesions, a prolonged mucosal bleeding time, or highly vascular lesions (determined with ultrasound) generally should not have percutaneous core biopsies because of the risk of uncontrollable hemorrhage or abdominal infection. Caution is also recommended with fine-needle aspiration in these patients.

Tissue core biopsies may be obtained with a TruCut biopsy (Fig. 20-2), a large-bore needle, or an automated biopsy device (e.g., Bard Biopty Instrument). For histopathologic

**FIG. 20-1** Anatomy of the liver.
specimens, the TruCut needle should be removed from the syringe or gun and placed in formalin. Once the sample has been fixed, it should be removed from the needle for processing. A core biopsy should use the largest gauge needle that may safely be used in the patient, typically a 14-gauge needle. If core biopsies are performed, at least two or three (2 cm long) samples should be obtained. Core biopsies are generally only taken from one liver lobe (i.e., the left liver lobe so as to minimize the chance of lacerating the bile ducts or gallbladder, which are on the right side). However, this is a significant limitation because hepatic lesions may not be present in all liver lobes. Finally, extreme care must be taken to ensure that the core biopsy needle will not pass through the liver and lacerate structures (e.g., veins, stomach, intestines, diaphragm, lungs, and heart) under the hepatic lobe from which a biopsy is being taken.

Fine-needle aspirates may be obtained using two different techniques. First, a hand-held syringe or an aspiration gun with a syringe may be attached to a 20- to 25-gauge, 1- to 3-inch needle. A syringe with a small amount of air is then attached to the needle, and the cells are blown out onto a slide. In the second technique, the needle is repeatedly passed through the liver without any suction being applied. After several passes, the needle is attached to a syringe and the contents are blown out onto a slide. Fine-needle aspiration is most likely to be diagnostic in patients with diffuse hepatic neoplasia (e.g., lymphosarcoma) or feline idiopathic hepatic lipidosis. However, inability to diagnose these conditions on a fine-needle aspirate does not preclude disease, and even if one of these conditions is found, there could be other, undiagnosed diseases present. This latter possibility is particularly important in cats because almost all sick, anorexic cats will have some fat vacuoles in the hepatocytes (Willard et al, 1999). However, before clinical illness due to hepatic lipidosis can be diagnosed, one must determine that there is sufficient fat in the hepatocytes to be causing hepatic dysfunction. Furthermore, diagnosing hepatic lipidosis does not guarantee that there is not other hepatic disease present that was not found by the fine needle technique. Cytologic evaluation of ultrasound-guided, fine-needle aspirates is most likely to agree with histopathologic findings when the animal has vacuolar hepatopathy; however, this condition was commonly misdiagnosed by cytology. In a recent study, overall agreement between cytology and histopathology in dogs and cats was 30.3% and 51.2%, respectively (Wang et al, 2004). In another study, in which morphologic diagnoses were made by use of an 18-gauge needle, concurrence with wedge biopsy specimens was approximately 50% in both dogs and cats (Cole et al, 2002). The substantial limitations of diagnosing hepatic disease in dogs and cats by percutaneous techniques should be recognized by clinicians (Cohen et al, 2003).

**NOTE:** Percutaneous biopsies may be obtained under tranquilization or heavy sedation using a transthoracic or transabdominal approach, although the former should only be used if the latter is not possible to prevent laceration of the liver during respiration. The latter is described here.

**Percutaneous blind biopsy.** With the animal in dorsal recumbency, clip the hair from the area surrounding the xiphoid process and prepare it for aseptic surgery. Make a small incision in the skin on the left side between the costal arch and the xiphoid process. Insert the biopsy needle through the skin incision in a craniodorsal direction, angling it slightly toward the left of midline. Advance the needle until ultrasound guidance shows the needle to be positioned at the surface of the liver. Advance the biopsy needle into the hepatic tissue and obtain the biopsy sample (see Fig. 20-2).
Percutaneous ultrasound-guided biopsy. There are three ultrasound-guided methods that can be used to biopsy or aspirate hepatic structures or lesions. Any of these techniques may be used when obtaining samples of the liver. If diffuse disease is suspected, the left liver should be sampled to avoid the main biliary structures. The first technique is called the indirect guidance method and is not generally recommended. It entails using ultrasound to find the structure of interest and then the ultrasound probe is removed. The needle is passed blindly in the area of interest. This technique is only applicable when the target is extremely large and direct visualization of the needle is not required as it is passed into the structure for biopsy or aspiration.

The other two techniques allow direct visualization of the needle as it is passed into the liver. It is critical that the needle pass at an oblique angle to the ultrasound beam for it to be seen on the image. A needle that passes parallel to the beam will not create useful echoes. The second technique is the freehand technique. This method requires good hand-eye coordination and takes practice to master it. Many sonographers prefer this technique because it allows the operator more choices in approaching the structure.

Handle the ultrasound probe with one hand (usually the nondominant hand) and the needle with the opposite hand. Use the probe to visualize the structure of interest and then pass the needle into it and obtain the biopsy.

The last technique is the needle guide technique. Many ultrasound machines have a manufactured needle guide, which fits onto the transducer. This equipment will guide the needle in a preset angle and maintain the orientation to the transducer. Software on the machine will project the needle path onto the screen, which allows accurate placement of the needle. One problem with this approach is that the needle guide is often bulky and thus the choice of approaches may be limited, particularly in small animals. Because the needle is passed through the needle guide, sterile technique is essential.

Sterilize the guide before use. Apply a sterile covering to the transducer (a sterile surgical glove works well). Place coupling gel into the glove, and then fit it over the transducer. Then attach the guide. Pass the needle through the needle guide, and obtain the biopsy.

Laparoscopic Liver Biopsy

Laparoscopy (see Chapter 14) offers the clinician several advantages over other hepatic biopsy techniques. First, laparoscopy not uncommonly finds lesions missed by ultrasound, allowing the clinician to take a biopsy from clearly abnormal areas that would have been missed if using a percutaneous technique (Cardi et al, 1997). Second, laparoscopy allows one to obtain better tissue samples than is possible with percutaneous techniques (sufficient hepatic tissue can readily be obtained for histopathology, mineral analysis, and culture), and it allows biopsies from multiple liver lobes (as opposed to just one lobe, as commonly occurs with core needle techniques). At the same time, the endoscopist can quickly look around the abdomen and examine the peritoneum, omentum, stomach, pancreas, intestines, and/or kidneys to see if there is any other unsuspected disease. Finally, laparoscopy can be done quickly (i.e., <20 minutes), and the patient routinely is able to be discharged within hours of completion of the procedure. Coagulopathies are not an absolute contraindication unless they are severe; electrocautery and coagulation-enhancing materials can be used, but are seldom needed.

Hepatic biopsy is obtained by using “double spoon” type forceps.

If no focal abnormalities are found, open the forceps and place them around the edge of the liver lobe. Then push the forceps into the lobe until the entire cup of the biopsy forceps is filled with hepatic tissue. Tightly close the jaws, and pull the sample off the lobe. If a biopsy of a different area of the liver lobe is desired, open the forceps and thrust the lower jaw into the liver lobe at the desired spot. Once the lower jaw is thrust as far as desired into the liver lobe, close the upper jaw over the lower jaw, and retrieve the tissue sample.

NOTE: Hemorrhage routinely occurs when the sample is torn off the liver; but remember that the laparoscope will magnify any hemorrhage that occurs. Typically, there is less than 1 to 5 ml of blood lost with a biopsy.

Surgical Liver Biopsy

Biopsies of the liver should be routinely done during exploratory laparotomy in animals known to have or suspected of having liver disease. Surgical biopsy allows the entire liver to be thoroughly inspected and palpated, and biopsies taken of focal lesions for histopathologic examination, culture, or copper analysis. Furthermore, hemorrhage from the biopsy site can be readily identified and controlled with proper technique. If generalized hepatic disease is present, the sample can be taken from the most accessible site (marginal biopsy samples). With focal disease, the entire liver should be carefully palpated for intraparenchymal nodules or cavities and representative samples obtained. The information gained from histologic examination of the patient’s liver may prove beneficial in determining the prognosis, diagnosis, and long-term management of hepatic dysfunction.

A biopsy of the hepatic margin may be obtained by the “guillotine” method.

Place a loop of suture around the protruding margin of a liver lobe. Pull the ligature tight, and allow it to crush through the hepatic parenchyma before tying it (Fig. 20-3, A). As the suture tears through the soft hepatic tissue, vessels and biliary ducts are ligated. Hold the liver gently between the fingers and, using a sharp blade, cut the hepatic tissue approximately 5 mm distal to the ligature.
(allowing the stump of crushed tissue to remain with the ligature). To prevent crushing the biopsy sample and causing artifacts, do not handle it with tissue forceps. Place a portion of the sample in formalin for histologic examination; reserve the remainder for culture and cytologic study. Check the biopsy site for hemorrhage. If hemorrhage continues, place a pledget of absorbable gelatin foam over the site. As an alternative, if a biopsy of a focal (nonmarginal) area of the liver is to be taken, use a punch biopsy or TruCut biopsy (see Fig. 20-2), or place several overlapping guillotine sutures around the margin of the lesion and excise it (Fig. 20-3, B). Use caution with a punch biopsy to avoid penetrating more than half the thickness of the liver with each biopsy. Apply pressure to the site until bleeding stops. If hemorrhage continues, place a pledget of absorbable gelatin sponge over the site.

Biopsies may also be obtained using a biopsy punch. The punch is used to excise a small portion of the liver, and a pledget of absorbable gelatin sponge is placed into the defect until bleeding stops.

**Partial Lobectomy**

Partial lobectomy may be indicated in some cases when the disease involves only a portion of a liver lobe (e.g., peripheral hepatic arteriovenous [A-V] fistulae, focal neoplasia, hepatic abscesses, or trauma). Partial lobectomy may be challenging because of the difficulty in obtaining hemostasis and should be done with extreme caution in animals with bleeding disorders. Stapling instruments have been used for both partial and complete lobectomies, but discretion should be exercised in their use because hemorrhage may occur if the staples do not adequately compress hepatic tissue.
Determine the line of separation between normal hepatic parenchyma and that to be removed, and sharply incise the liver capsule along the selected site (Fig. 20-4, A). Bluntly fracture the liver with the fingers (Fig. 20-4, B) or the blunt end of a Bard-Parker scalpel handle, and expose the parenchymal vessels. Ligate large vessels (hemoclips may be used), and electrocoagulate small bleeders encountered during the dissection (Fig. 20-4, C). As an alternative, place a stapling device (Autosuture TA 90, 55, or 30) across the base of the lobe and deploy the staples. Excise the hepatic parenchyma distal to the ligatures or staples. Before closing the abdomen, make sure the raw surface of the liver is dry and free of hemorrhage. In small dogs and cats, several overlapping guillotine sutures (as described previously) may be placed along the entire line of demarcation (Fig. 20-5). Be sure the entire width of the hepatic parenchyma is included in the sutures. After tightening the sutures securely, use a sharp blade to cut the hepatic tissue distal to the ligature, allowing a stump of crushed tissue to remain with the ligature.

**Complete Lobectomy**

Complete lobectomy may be indicated for some focal lesions involving one or two hepatic lobes (e.g., traumatic lacerations of the liver or hepatic A-V fistulae). The left lobes of the liver (i.e., left lateral and left medial lobes) maintain their separation near the hilus more than do the other lobes; therefore the left lobes often can be removed in small dogs and cats by placing a single encircling ligature around the base of the lobe.

For the right lateral and caudate lobes, careful dissection around the hepatic caudal vena cava usually is necessary. Before performing the dissection, pass umbilical tape around the portal vein, celiac artery, cranial mesenteric arteries, and caudal vena cava in front of and behind the liver. The tape is passed through rubber tubing, which can be used to occlude the hepatic blood supply if uncontrollable hemorrhage occurs.

For the left lobes in small dogs and cats, crush the parenchyma near the hilus with the fingers or a forceps. Place an encircling ligature around the crushed area and tie. For the left lobes in larger dogs and for the right and caudate lobes, carefully dissect, if necessary, the lobe from the caudal vena cava. Isolate the blood vessels and biliary ducts near the hilus and ligate them. Double ligate or oversew the ends of large vessels. Resect the parenchymal tissue, leaving a stump of tissue distal to the ligatures to prevent retraction of the hepatic tissue from the ligatures and subsequent hemorrhage.
HEALING OF THE LIVER

The liver is unique among the visceral organs in its healing properties. It has relatively little connective tissue stroma, is highly susceptible to small changes in blood flow, and has an enormous regenerative capacity. With regeneration, adequate liver function is possible in patients even after 80% of the organ has been removed or destroyed. Lacerations of the liver should be closed only when bleeding is profuse. If lacerations are sutured, they should be closed in a manner that does not create an internal pocket of bile or blood or cause ischemia of the surrounding cells. The proper hepatic artery can be ligated as an emergency measure to control hemorrhage from extensive liver lacerations. Complex fractures or severe contusions should be treated by hepatic lobectomy if ligation of the hepatic artery does not result in hemostasis.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Guillotine biopsies often are performed with large (0 or 2-0) chromic gut suture or polyglactin 910. Suture with good knot security (e.g., silk suture) may facilitate partial hepatectomy. Polydioxanone or polyglyconate suture may also be used for vessel ligation in complete and partial lobectomies.

POSTOPERATIVE CARE AND ASSESSMENT

Recovery from anesthesia should be closely monitored in animals with severe hepatic dysfunction. Because of the increased half-life of some drugs in patients with hepatic dysfunction, recovery may be prolonged. Intravenous fluids should be provided until the patient is able to maintain hydration, but care must be taken not to overhydrate hypoalbuminemic patients. Blood glucose levels should be monitored; transient hypoglycemia is common after removal of large portions of the liver. Albumin levels should be monitored. If the patient becomes severely hypoalbuminemic (i.e., less than 2 g/dl) or has substantial worsening of third space fluid accumulation, one should consider administering plasma, whole blood, or synthetic colloids (e.g., hetastarch). Clotting times may be assessed if hemorrhage or petechiation occurs. Antibiotics given during surgery should be continued for 2 to 3 days if partial hepatectomy has been performed. Nutritional supplementation may be necessary in some patients during the early postoperative period, particularly if the animal is anorexic or has severe vomiting or diarrhea (see Chapter 11).

Analgesics (e.g., hydromorphone, butorphanol, and buprenorphine) should be provided to patients after surgery (see Table 13-4 on p. •••). For severe pain, a fentanyl-lidocaine-ketamine combination given as a continuous rate infusion (CRI) may be indicated (see Table 13-7 on p. •••).

COMPLICATIONS

Biopsy samples may be useless for diagnosis if the tissue sample is crushed, fragmented, or too small or if the specimen contains predominantly blood or necrotic portions of mass lesions. Bile peritonitis may occur if the gallbladder or bile ducts are inadvertently penetrated. One study found the complication rate in 246 animals undergoing ultrasound-guided biopsy of abdominal structures to be 1.2%. The most important complication may be the propensity for an incorrect diagnosis when this technique is used (see previous discussion).

The most common and serious complication of hepatic surgery is hemorrhage. This may result from ligatures slipping off friable hepatic tissue. Care should be taken to ensure that a stump of tissue remains distal to the ligature when encircling sutures are used for biopsy or partial hepatectomy. With hepatic trauma anaerobic bacteria may proliferate in hypoxic portions of the liver and cause sepsis; therefore broad-spectrum antibiotics should be used in patients with severe hepatic trauma and in those undergoing hepatic surgery. Complications after major hepatic resection may include portal hypertension, ascites, fever, hemorrhage, or persistent bile drainage.

SPECIAL AGE CONSIDERATIONS

Portosystemic shunt ligation (see p. •••) often is performed in young animals, which are particularly prone to hypoglycemia. Serum glucose concentrations should be carefully monitored. Hypothermia, also a particular problem in young patients, reduces the minimum alveolar concentration (MAC) of inhalants used for anesthetic maintenance.

References


**Suggested Reading**

This study showed that the normal liver of healthy dogs harbors different bacterial species. The types of bacteria seen are reviewed along with histologic findings.

### SPECIFIC DISEASES

**PORTOSYSTEMIC VASCULAR ANOMALIES**

**DEFINITIONS**

Portosystemic vascular anomalies (PSVA) or portosystemic shunts (PSS) are anomalous vessels that allow normal portal blood to drain from the stomach, intestines, pancreas, and spleen and pass directly into the systemic circulation without first passing through the liver. The term portacaval shunt is frequently used; however, this term technically refers to a specific type of vascular anomaly (i.e., portal vein to caudal vena cava). Extrahepatic shunts are vascular anomalies located outside the hepatic parenchyma; intrahepatic portosystemic shunts (IHPPS) are located in the liver. The term “hepatic microvascular dysplasia” seems to be in the process of being replaced with “portal vein hypoplasia.” Portal vein hypoplasia appears to be a condition characterized by small or absent intrahepatic portal vessels and portal arteriolar hyperplasia that is associated with microscopic shunting of blood through the liver without a macroscopic portosystemic shunt.

**GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY**

When portal blood bypasses the liver, many substances that are normally metabolized or excreted in the liver enter the systemic circulation. Also, important hepatotrophic substances from the pancreas (e.g., insulin) and intestines do not reach the liver, resulting in hepatic atrophy or failure of the liver to attain normal size. Hepatic insufficiency or hepatic encephalopathy frequently occurs. Hepatic encephalopathy is a clinical syndrome of altered CNS function resulting from hepatic insufficiency. A variety of substances (i.e., ammonia, methionine/mercaptans, short-chain fatty acids, alterations in the ratio between circulating levels of branched-chain and aromatic amino acids, and γ-aminobutyric acid) have been incriminated in the resulting elaboration of false neurotransmitters.

Portosystemic shunts may be broadly categorized as intrahepatic or extrahepatic. Extrahepatic shunts may be congenital or acquired. Congenital extrahepatic shunts usually are single anomalous vessels that allow abnormal blood flow from the portal vein to the systemic circulation. Extrahepatic PSS account for nearly 63% of single shunts in dogs; they also occur in cats. Many different types of PSS have been described in dogs, including (1) portal vein to caudal vena cava; (2) portal vein to azygos vein; (3) left gastric vein to caudal vena cava; (4) splenic vein to caudal vena cava; (5) left gastric, cranial mesenteric, caudal mesenteric, or gastroduodenal vein to caudal vena cava; and (6) combinations of the above (Fig. 20-6). Cats most commonly have a large single
vessel that empties directly into the prehepatic vena cava; however, they may have atypical PSS connections and the shunt may flow into any systemic vessel including the renal, phrenicoabdominal, azygos, or internal thoracic veins. Intrahepatic shunts usually are congenital, singular shunts, which occur because the ductus venosus fails to close after birth, or they may arise when other portal to hepatic vein or caudal vena cava anastomoses exist. IHPSS constitute about 35% of single shunts in dogs and approximately 10% in cats.

Acquired extrahepatic shunts typically are multiple and represent about 20% of all canine PSS. They are thought to arise partly because of increased resistance to portal blood flow and subsequent portal hypertension. This hypertension causes normal, nonfunctional microvascular connections, which are present at birth, between portal and systemic veins to become functional. Multiple shunts are most commonly associated with chronic, severe hepatic disease (i.e., cirrhosis), but have been reported secondary to hepatoportal fibrosis in young dogs. Venoocclusive hepatic disease has been reported as a cause of multiple PSS in young cocker spaniels. Multiple shunts most commonly occur in the left renal area and the root of the mesentery (Fig. 20-7), and connections to the caudal vena cava or azygos veins usually are seen.

Our knowledge of hepatic microvascular dysplasia (i.e., portal vein hypoplasia [PVH]) is currently being redefined, and it may be different by the time this chapter is published. Current thought is that PVH is a congenital condition characterized by small or absent intrahepatic portal vessels and portal arteriolar hyperplasia, which allows an abnormal communication between the portal and systemic circulations. This condition is sometimes difficult to differentiate from congenital PSS because both may have similarly increased serum bile acid concentrations. Furthermore, the histologic lesions of PSS and PVH are identical, and PVH is common in some breeds that are at increased risk for PSS. In general, most dogs with PVH are asymptomatic and do not have obvious microhepatica, but exceptions exist. In particular, there is concern that PVH can progress and result in noncirrhotic portal hypertension and/or hepatoportal fibrosis in some patients. Currently, PVH is diagnosed by hepatic histology plus elimination of PSS. Mesenteric portograms and nuclear scintigraphy in dogs with PVH should be normal.

A-V fistulae account for about 2% of single shunts and may be congenital or acquired. Acquired A-V fistulae occur secondary to trauma, tumors, surgical procedures, or degenerative processes that cause arteries to rupture into adjacent veins. The fistulae typically are macroscopic communications that form between branches of the hepatic artery and portal vein; however, microscopic hepatic A-V fistulae have also been suspected. As congenital lesions, they are believed to develop as a result of failure of the common embryologic capillary plexus to differentiate into an artery or a vein. Affected animals usually develop portal hypertension and multiple collateral shunting vessels, resulting in an acute
onset of low protein transudative ascites between the ages of 2 and 18 months (Figs. 20-8 and 20-9). In contrast, dogs with congenital PSS rarely have ascites.

**DIAGNOSIS**

**Clinical Presentation**

**Signalment.** Purebred dogs are at increased risk for PSS and PVH. Domestic shorthair cats are most commonly affected, although these aberrations also occur in purebred cats (i.e., Himalayan). Single PSS usually are congenital and are most commonly diagnosed in animals under 1 year of age, although dogs as old as 13 years have been diagnosed. Extrahepatic shunts have been most frequently diagnosed in miniature and toy-breed dogs (e.g., Yorkshire terriers, Maltese, Silky terriers, miniature schnauzers, poodles, Lhasa apso, Bichon Frise, Jack Russell Terriers, Shih Tzu, and Pekingese). They are clearly hereditary in Yorkshire terriers (Tobias, 2003; Tobias and Rorbach, 2003) and may be genetic in other breeds as well. Intrahepatic PSS are more commonly diagnosed in large-breed dogs (e.g., German shepherds, golden retrievers, Doberman pinschers, Labrador retrievers, Irish setters, Samoyeds, and Irish wolfhounds). Small-breed dogs most likely to have IHPSS are toy and miniature poodles. There may be a hereditary basis for IHPSS in Irish wolfhounds. Congenital extrahepatic and IHPSS have been reported in cats. There is no convincing gender predisposition for these anomalies in either species.

**NOTE:** In general, small-breed dogs are more likely to have extrahepatic shunts, and large-breed dogs are more likely to have IHPSS.

Multiple shunts are most commonly diagnosed in animals between 1 and 7 years of age; however, multiple acquired PSS that occur secondary to hepatoporal fibrosis have been reported in dogs as young as 4 months of age. Breeds most commonly affected include the German shepherd, Doberman pinscher, and cocker spaniel. Multiple acquired shunts have been described in cats.

Most dogs with hepatic A-V fistulae have been young (i.e., under 1½ years of age) at the time of diagnosis. Congenital hepatic A-V fistulae have rarely been reported in cats.

**History.** The presenting history for animals with PSS varies considerably. Affected animals usually are evaluated because of failure to grow, small body stature, or weight loss. Other common abnormalities include intermittent anorexia, depression, vomiting, polydipsia or polyuria, ptyalism (especially in cats), pica, amaurosis, and behavioral changes. Some animals are presented for evaluation of urinary dysfunction (i.e., hematuria, dysuria, pollakiuria, stranguria, and urethral obstruction) associated with urate urolithiasis (see later discussion). Signs of hepatic encephalopathy can vary tremendously from those that are extremely mild and hard to identify as a significant abnormality (e.g., lethargy, being “tired,” being “slow”) to severe changes (e.g., ataxia, weakness, stupor, head pressing, circling, amaurosis, pacing, seizures, or coma). These signs may be constant or intermittent and sometimes, but not invariably, worsen after eating (especially a high-protein diet composed of animal protein). Hepatic encephalopathy may also worsen after gastrointestinal hemorrhage (e.g., caused by parasites or ulceration). In a recent study, 82% of dogs had CNS signs, 76% had gastrointestinal signs, and 39% had urinary signs (Mehl et al, 2005). In addition to ptyalism, affected cats typically show episodic central blindness.

**NOTE:** Consider PSS in any young animal with a prolonged response to anesthetic agents or tranquilizers that require hepatic metabolism for clearance. These may be the first abnormalities you note in some affected animals.

The most common presenting sign in dogs with hepatic A-V fistulae is sudden onset of depression, ascites, and vomiting. Despite the chronic nature of this condition, the animal often has an acute onset of gastrointestinal or neurologic signs. The ascites typically is a pure transudate despite a serum albumin more than 1.8 g/dl.

**NOTE:** Animals with hepatic A-V fistulae may be presented for evaluation of gastrointestinal foreign bodies. Presumably gastric irritation causes pica in these animals.

**Physical Examination Findings**

Most animals with PSS have microhepatica, and the kidneys may feel prominent or plump. A golden or copper color to the iris has been observed in many cats with PSS. Neurologic abnormalities may be noted (see previous discussion). Ptyalism is a common finding in cats, but rare in dogs. Animals with hepatic A-V fistulae may have a palpably enlarged liver (rare) or ascites. An audible bruit sometimes can be auscultated in the cranial abdomen of affected animals.

**Diagnostic Imaging**

Survey abdominal radiographs are an important part of screening for congenital PSS. Microhepatica is expected in affected patients and may vary from mild to marked. It is extremely rare to find a dog with PSS that does not have some degree of microhepatica, and failure to find this change is an indication to look for diseases other than congenital PSS. Plain abdominal radiographs are more sensitive in finding microhepatica than abdominal ultrasound.

Definitive diagnosis of PSS is made by surgical identification of the shunt, intraoperative positive contrast portography, ultrasound identification of the shunt, or nuclear hepatic scintigraphy. Various positive contrast techniques have been described, including splenoporation, cranial mesenteric arterial portography, celiac arteriography, splenic portal catheterization, and jejunal vein portography. Jejunal vein portography is the simplest and most effective
Ultrasound-guided splenoportography can also be performed in large-breed dogs. The most consistent finding on survey abdominal radiographs is microhepatica. Ultrasound has become the diagnostic tool of choice for imaging PSS. Both intrahepatic and extrahepatic shunts have been identified with this technique; however, an inconclusive ultrasound examination does not rule out PSS. Ultrasound diagnosis of PSS is dependent upon operator experience and the time allotted to examine the patient. Occasionally a dilated intrahepatic vessel or the communication of an intrahepatic shunt with the caudal vena cava is noted (Fig. 20-10). With extrahepatic shunts, overlying bowel may obscure the shunt, but a small liver with few detectable hepatic or portal veins may be noted. Increased hepatic arterial detection using Doppler is also a common finding. The bladder and renal pelves should be assessed for calculi because urate stones usually are radiolucent and difficult to see on survey abdominal radiographs. Ultrasound scanning is also useful to identify the anechoic, tortuous vessels seen with hepatic A-V fistulae. Pulsed wave Doppler ultrasound may assist in making a diagnosis of hepatic A-V fistulae. Visualization of retrograde (hepatofugal), pulsatile flow in the abnormal portal vein branch and the main portal vein may allow one to discern the involved liver lobe and resect it.

Nuclear scintigraphy is a rapid, noninvasive method of documenting abnormal hepatic blood flow. Sodium pertechnetate technetium 99m (99mTc) is typically used in scintigraphic studies to detect PSS. After colonic administration of 99mTc, the time when activity in the region of the liver is first noted is compared with the time when activity appears in the region of the heart (Fig. 20-11). Animals with liver-to-heart intervals longer than 12 seconds are generally considered clinically normal. Sometimes studies in very small animals can be difficult to interpret because of the close proximity of the liver and heart, and occasional studies must be repeated if the 99mTc is not rapidly absorbed from the colon. If a study must be repeated, it must be done the following day to allow the body to eliminate the technetium from the invalid study. False positive results have not been reported; however, false negative results may occur if a small shunt involves only a peripheral portion of the portal system. PVH will have a normal scintigraphic study, which distinguishes it from PSS. Computed tomography (CT) angiography appears to be a useful technique for identifying PSS, including multiple acquired shunts; however, there are too few studies to know the accuracy of this technique. A new scintigraphic technique has recently been reported (Morandi et al, 2005; Cole et al, 2005)—transsplenic portal scintigraphy. This technique is unique in that it uses ultrasound guidance to inject a small amount of 99mTc into the parenchyma of the spleen. Dynamic phase imaging of the splenic vein drainage yields a nuclear angiogram of the portal system and is useful for detecting the presence of either single or multiple extrahepatic shunts. An advantage of this technique is that a very small amount of radioactivity is used, thus the animal can be released from radiation isolation shortly after the procedure, depending on the state’s release criteria. Furthermore, the authors have found that transsplenic portal scintigraphy yields higher quality images than transrectal scintigraphy.

NOTE: Nuclear scintigraphy is a useful, noninvasive screening tool for diagnosing congenital or acquired shunts, and distinguishes them from PVH.

Laboratory Findings

Hematologic, serum biochemical, and urine analysis of animals with PSS may disclose various abnormalities, but dogs can have a congenital PSS without any abnormalities on
complete blood count (CBC) or serum biochemistry panel. Hematologic abnormalities may include microcytosis with normochromic erythrocytes, mild nonregenerative anemia, target cell formation, or poikilocytosis. Low serum iron concentrations appear to be related to the development of microcytosis in dogs with PSS. Biochemical tests often reveal a reduction in the serum albumin, cholesterol, and/or blood urea nitrogen (BUN) concentrations. Low serum albumin is a common finding in dogs; however, some dogs (and most cats) with PSS have normal albumin levels. Low BUN results from reduced conversion of ammonia to urea in the hepatic urea cycle, but the polyuria-polydipsia seen in many patients may contribute. Other abnormalities occasionally include mild to major increases in serum alanine aminotransferase, aspartate aminotransferase, and alkaline phosphatase. The serum bilirubin concentration usually is normal. Fasting hypoglycemia rarely occurs. Functional measurements of coagulation (i.e., prothrombin time, activated partial thromboplastin time, and activated coagulation time) usually are normal. Routine urinalysis may disclose dilute urine or ammonium biurate crystals. Hyperuricemia and hyperammonemia lead to increased urinary excretion of urate and ammonia, promoting urinary precipitation of ammonium biurate crystals. Hematuria, pyuria, and proteinuria may occur if urate calculi form. The hematologic and biochemical profiles of canine hepatic A-V fistulae can be similar to those of dogs with single or multiple PSS.

Hepatic function tests are important in screening for congenital PSS. Serum bile acids have been the standard hepatic function test in dogs and cats for years, but it is now recognized that they have some major limitations. First, it is critical to measure both preprandial and postprandial serum bile acid concentrations; approximately 20% of dogs have a higher preprandial value. Second, some dogs with very high serum bile acid concentrations (>150 μmol/L) do not have clinically significant hepatic disease, whereas some dogs with congenital PSS have serum bile acid concentrations that are only moderately increased (e.g., 50 to 60 μmol/L, normal <30 μmol/L). Third, unlike what is expected for most biochemical determinations, there can be substantial variation in bile acid concentrations from day to day (as much as or greater than 100%). Currently, urinary bile acids (Balkman et al, 2003; Trainor et al, 2003) appear to be about as useful as serum bile acids, but may have the advantage of being easier to collect (i.e., the owner can bring in a urine sample as opposed to bringing in the patient), especially in cats.

Hyperammonemia is very specific for hepatic insufficiency, but simply measuring resting blood ammonia is very insensitive, even in patients experiencing hepatic encephalopathy. The ammonia tolerance test (ATT) is a very sensitive test, but performing the test is difficult (many animals vomit or defecate the ammonia chloride that is administered). Measuring 6- to 8-hour postprandial blood ammonia concentrations appears to be more sensitive than measuring fasting blood ammonia (Walker et al, 2001), but is probably less sensitive than the ATT. The biggest disadvantage of measuring blood ammonia is that it is easy to obtain artifactual values if instructions in collecting, storing, and preparing the blood are not followed exactly. This test must be run in-house; it cannot be sent to an outside lab.

**DIFFERENTIAL DIAGNOSIS**

PSS must be differentiated from other diseases that cause hepatic insufficiency (e.g., cirrhosis) or neurologic abnormalities (e.g., hydrocephalus and epilepsy) in dogs and cats. Performing survey abdominal radiographs (to look for microhepatica) and a hepatic function test (usually preprandial and postprandial serum bile acids) are the typical means of screening for congenital PSS. If either of these tests is normal, then congenital PSS is unlikely, and other diseases must be seriously considered.

**MEDICAL MANAGEMENT**

Surgery is the treatment of choice for most animals with PSS because hepatic function may continue to deteriorate as long as most of the blood is shunted away from the liver. The life expectancy of animals that are managed medically generally is reported to be 2 months to 2 years; however, one study suggested that the older a dog is when presented for treatment, the better its prognosis on conservative treatment (Watson and Heritage, 1998). In particular, medical management must be considered in asymptomatic dogs that have been fortuitously diagnosed and dogs older than 7 years old that have minimal clinical signs. In these patients, one must weigh the reported 7% mortality associated with corrective surgery versus the likelihood of substantial deterioration if surgery is not done. Although not proven, intuitively it appears that the patients described above, if they have only modest changes on serum biochemistry panel and only modest to moderate microhepatica on abdominal radiographs, may be the best candidates for medical management. If there are histologic changes (e.g., bridging hepatic fibrosis or bridging biliary hyperplasia) that would seem to make postsurgical complications (e.g., portal hypertension) more likely, then medical management should probably be chosen over surgery. However, surgery is desirable in that restoration of hepatotropic factors in the liver postoperatively should promote hepatic regeneration. Medical management should be initiated before surgical intervention in animals with substantial signs of hepatic encephalopathy (some argue this should be routine; see later discussion in the Preoperative Management section).

The goals of medical therapy are to identify and correct factors predisposing to hepatic encephalopathy (i.e., reduce absorption of toxins produced by intestinal bacteria, diminish the interaction between enteric bacteria and nitrogenous substances, and avoid drugs that predispose to encephalopathy), and to decrease oxidative damage to hepatocytes. Precipitating factors for hepatic encephalopathy include high-protein meals (especially meat), bacterial infections, gastrointestinal bleeding, blood transfusions, inappropriate drug therapy, and electrolyte and acid-base abnormalities. General supportive care of the patient with hepatic encephalopathy should include fluid therapy (0.9% sodium chloride...
Secondary to hepatic insufficiency. Recommendations on the management of coma that occurs more effective, and some patients must be anesthetized with propofol. A medical text should be consulted for additional information.

Diazepam is often ineffective in encephalopathic dogs having seizures; phenobarbital may be identified and corrected. Neomycin or lactulose (or both) should be given. Acid-base disturbances, and supplementation of potassium as needed. A highly digestible diet in which the primary source of calories is carbohydrates should be fed. For long-term dietary management, feed the lowest-protein diet (preferentially vegetable protein or cottage cheese) the animal will tolerate. Moderately protein-restricted diets (i.e., k/d or in some animals u/d; Hill’s Pet Products) that contain high levels of branched-chain amino acids and arginine are often used, but there is no good evidence that preferentially feeding branched-chain as opposed to aromatic amino acids benefits dogs with PSS. Antibiotics (Box 20-3) are used to reduce the enteric flora that produce many of the toxins (i.e., ammonia) thought to cause hepatic encephalopathy. Oral neomycin frequently is used for this purpose, but it should be avoided in animals that are azotemic. Metronidazole or ampicillin (either oral or parenteral) also reduces intestinal ammonia concentrations. Lactulose is a synthetic disaccharide that acidifies colonic contents and traps ammonium ions in the lumen (see Box 20-3). It is also an osmotic cathartic that shortens the intestinal transit time and reduces the production and absorption of ammonia. Lactulose may be given orally or as a retention enema. Side effects of lactulose administration may include diarrhea, vomiting, anorexia, and increased gastrointestinal loss of potassium and water. Treatment of animals that are presented for operative seizures. The high incidence of postligation seizures. The high incidence of postligation seizures. There is currently debate as to the value of pretreating surgical candidates with potassium bromide (dogs) or phenobarbital (cats) to attempt to lessen the incidence of postoperative seizures. The high incidence of postligation sei-
Fluid and electrolyte imbalances should be corrected preoperatively. Perioperative antibiotics (e.g., cephalosporins) are recommended for patients with PSS. See the previous section on Medical Management for other recommendations.

**Anesthesia**

Extreme care must be exercised when anesthetizing an animal having PSS. Because of the reduced liver function and abnormal hepatic blood flow, drug absorption, metabolism, and clearance are notably reduced. In addition, drugs that are highly protein bound are affected by the low albumin concentrations that may accompany PSS (i.e., resulting in increased levels of circulating, unbound drugs). Therefore drugs that are metabolized by the liver (e.g., phenothiazine tranquilizers) and those that are highly protein bound (e.g., diazepam) should be avoided. Benzodiazepines may also negatively affect neurologic function in hepatoencephalopathic patients. A reversible opioid may be administered with an anticholinergic, followed by mask or chamber induction with isoflurane or sevoflurane and oxygen and endotracheal intubation (Box 20-4). Propofol, a noncumulative, nonbarbiturate hypnotic, has been used successfully at reduced doses for induction of patients with PSS (Box 20-4). Blood glucose levels should be monitored because patients with PSS may have reduced hepatic glycogen stores. Care should be taken to prevent hypothermia. Inotropic support (i.e., dobutamine [2 to 10 µg/kg/minute IV] or dopamine [2 to 10 µg/kg/minute IV]) may be necessary in some patients. These patients should be monitored for arrhythmias or tachycardia.

**Surgical Anatomy**

The canine portal vein varies from 3 to 8 cm long, depending on the animal’s size. On a radiographic contrast study of the normal portal system, the portal system usually originates at the level of the first lumbar vertebra (Fig. 20-13). Knowledge of the anatomy of the portal and hepatic venous systems is imperative to locate shunts, particularly intrahepatic shunts (Fig. 20-14). The portal vein is formed by the confluence of the cranial and caudal mesenteric veins and the splenic vein. The splenic vein enters the portal vein at the level of the thoracolumbar junction. The phrenicoabdominal veins ter-

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**FIG. 20-12**
An ameroid constrictor.

**FIG. 20-13**
Mesenteric portogram in a dog with a normal portal system. The portal system originates at the level of the first lumbar vertebra. Note the hepatic vasculature.

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**BOX 20-4**

**Selected Anesthetic Protocols for Animals With Portosystemic Shunts**

**Dogs**

**Premedication**

Give atropine (0.02–0.04 mg/kg SC or IM) or glycopyrrolate (0.005–0.011 mg/kg SC or IM) plus hydromorphone (0.1–0.2 mg/kg SC or IM)

**Induction**

Mask induce with isoflurane or sevoflurane or administer propofol (4 mg/kg IV to effect)

**Maintenance**

Isoflurane or sevoflurane

**Cats**

**Premedication**

Give atropine (0.02–0.04 mg/kg SC or IM) or glycopyrrolate (0.005–0.011 mg/kg SC or IM) plus butorphanol (0.2–0.4 mg/kg SC or IM) or buprenorphine (5–15 µg/kg IM) or hydromorphone (0.05 mg/kg SC or IM)

**Induction**

Chamber induce with isoflurane or sevoflurane or administer propofol (4 mg/kg IV to effect)

**Maintenance**

Isoflurane or sevoflurane

SC, Subcutaneous; IM, intramuscular; IV, intravenous.
terminate in the caudal vena cava about 1 cm cranial to the renal veins. Any vein that enters the caudal vena cava cranial to the phrenicoabdominal veins (before the hepatic veins) may be considered an anomalous structure.

NOTE: Examine the caudal vena cava carefully. The only vessels that should enter the caudal vena cava between the renal veins and the hepatic veins are the small phrenicoabdominal veins.

Positioning
A standard ventral midline celiotomy is performed from the xiphoid cartilage caudally, and the portal system is examined. For IHPSS and A-V fistulae, the incision may need to extend cranially through the xiphoid process and caudal sternebrae.

SURGICAL TECHNIQUE
Single extrahepatic shunts generally are treated by placing an ameroid constrictor or cellophane band on the vessel. Because the vessel slowly becomes occluded, portal hypertension is rare and most surgeons no longer evaluate portal pressures in conjunction with this surgery. When compared with surgical ligation, surgical time is shortened and intraoperative and postoperative complications are decreased with ameroid constrictors (Murphy et al, 2001). In rare cases, animals with IHPSS may have an ameroid constrictor or cellophane band placed on the vessel; however, IHPSS generally requires ligation. Two additional techniques, intravascular coil occlusion and placement of a portacaval veno-graft using the jugular vein (with an ameroid constrictor placed on it) with complete ligation of the intrahepatic shunt, have also been used for intrahepatic shunt occlusion. With the latter technique, an unacceptably high incidence of multiple, extrahepatic acquired shunts was noted at long-term follow-up (Kyles et al, 2001).

Animals with congenital PSS should generally have a liver biopsy performed when the shunt is attenuated. However, if the gross appearance of the liver is abnormal (especially if it is rough or irregular), a biopsy of the liver should be taken and the histology report received before attenuating the shunt. PSS patients with chronic fibrotic or biliary hyperplastic changes may be at increased risk for portal hypertension following shunt attenuation.

Animals with multiple hepatic shunts secondary to acquired hepatic disease often benefit from medical management directed at the cause of the hepatic disease (e.g., anti-inflammatory, antifibrotic drugs, antioxidants), and subsequent ascites and encephalopathy (e.g., dietary restriction of protein and salt, diuretics). Good medical care may result in long-term survival and a good quality of life; non-cirrhotic portal hypertension in particular may respond...
well. Caudal vena cava banding has been suggested to raise the systemic venous pressure in the abdomen to or slightly above that of the portal venous system, but this technique is seldom performed. Multiple extrahepatic PSS usually are evident upon exploratory laparotomy in these patients. Findings that may be noted include enlarged mesenteric veins, a larger than normal portal vein, and anomalous connections between the portal venous system and the systemic venous circulation. The most common location for multiple PSS is the area of the left kidney; however, anomalous venous connections between the mesenteric circulation and the caudal vena cava or its tributaries may be noted throughout the abdomen (Fig. 20-15). Portoazygos connections have also been observed in clinical cases. Care should be taken when incising the abdominal wall in patients suspected of having multiple PSS because large, dilated vessels may be present in the falciform ligament or the greater omentum or both. Trauma to these vessels upon abdominal entry may cause significant hemorrhage. Dissection of the falciform ligament usually requires ligation or cauterization of multiple vessels. Because many patients with multiple extrahepatic PSS have ascites, suction should be available upon entry into the abdomen to evacuate ascitic fluid.

**NOTE:** If you find multiple shunts in an animal, be sure a biopsy is taken from the liver.

A-V fistulae are treated by removing the affected liver lobe or in rare cases by ligating the fistulae directly. Portal pressure should be measured in conjunction with occlusion of IHPSS or vena cava banding. The normal portal pressure in dogs is 8 to 13 cm H₂O, which is 7 to 8 cm H₂O higher than the systemic venous pressure (Box 20-5). However, in animals with single PSS, the resting portal pressure often is closer to the systemic venous pressure. Excessive portal venous pressure can result in splanchnic congestion, portal hypertension, and death.

### Ameroid Constrictor Placement on Extrahepatic Shunts

Perform a midline abdominal incision. Identify the portal vein by retracting the duodenum to the left and ventrally. Locate the caudal vena cava, renal veins, phrenicoabdominal veins, and portal vein (ventral to the caudal vena cava at the most dorsal aspect of the mesoduodenum). Note any veins entering the caudal vena cava proximal to the phrenicoabdominal veins. If the shunt has not been identified, open the omental bursa and retract the stomach cranially, the duodenum to the right and ventrally, and the left lobe of the pancreas caudally. Identify shunts that communicate with the caudal vena cava through the epiploic foramen by observing abnormal tributaries of the portal vein, left gastric vein, or splenic vein. Once the shunt has been identified, select the appropriately sized ameroid constrictor. Generally, use a 3.5- or 5-mm ameroid in most small dogs with extrahepatic shunts. Dissect around the shunt vessel to allow placement of the device, but avoid dissecting a large area next to the vessel. Excessive dissection may allow movement of the ameroid on the vessel and predispose to premature kinking of the vessel.

**NOTE:** The ameroid should fit on the vessel without compromising the lumen; however, avoid using an ameroid that is too large because the weight of the device may cause the vessel to kink, obstructing flow prematurely.
Part II  Soft Tissue Surgery

Cellophane Banding of Extrahepatic Shunts
Fold a 10 cm long × 1.2 cm wide strip of cellophane (purchased from a store and sterilized, or use MS 350 grade cellophane [Cello Paper Pty]) longitudinally to form a three-layer strip that is approximately 4 mm wide.

The cellophane may be attached in either of two ways; (1) causing partial obstruction of the shunt at the time of placement (Hunt et al, 2004), or (2) where the cellophane causes no shunt occlusion initially. The second technique is easier to perform, eliminates the need for monitoring portal pressures, and may result in a more favorable outcome than the first technique (H. Seim, personal communication).

For the first technique, pass the cellophane around the shunt and a pin of predetermined size, and place a titanium clip on the strip. To determine pin size in dogs less than 10 kg (which will determine the diameter of the cellophane band), evaluate changes in heart rate, arterial pressure, intestinal color and motility, and pancreatic color when the shunt is totally occluded, or measure portal pressures (see later discussion). If elevations in heart rate are minimal (less than 10 beats/min) and if arterial systolic pressure does not decrease more than 10 mm Hg, use a 2-mm pin to determine the final diameter of the band. If changes are moderate, use a 2.5-mm pin; and if they appear to be severe, use a 3-mm pin. Secure the cellophane band with two titanium ligacips (Autosuture, Ethicon).

For the second technique, prepare the cellophane as described above, and place it around the vessel without causing any occlusion of the shunt. Secure the cellophane with a hemoclip(s).

Ligation of Single Extrahepatic Shunts
If placement of an ameroid constrictor is not possible, the vessel may be ligated or attenuated; however, extreme care must be taken to prevent causing portal hypertension.

Identify the anomalous vessel, isolate it, and pass 2-0 silk suture around the vessel (Fig. 20-16). If jejunal portography was not performed (see later discussion), exteriorize a segment of jejunum and insert a 20- to 22-gauge over-the-needle catheter (Angiocath, Abbocath) into a jejunal vein. Do not damage the corresponding jejunal artery. Obtain baseline portal pressures. Temporarily occlude the shunt, and observe portal pressures during this manipulation.

Once you have positively identified the shunt, slowly tighten the ligature while monitoring the portal pressure. If possible, completely occlude the shunting vessel, but do not allow postligation portal pressures to exceed 10 cm H₂O (8 mm Hg) above baseline pressures or 20 to 23 cm H₂O (15 to 18 mm Hg). If intraoperative Doppler ultrasound is available, once hepatopetal flow is established in the cranial portal vein and shunt, do not occlude the shunt further (Szatmari et al, 2004). You may be able only to attenuate the vessel. Observe the viscera for evidence of splanchnic congestion for 5 to 10 minutes. If excessive splanchnic congestion is noted, loosen the suture. Remove the jejunal vein catheter and ligate the vein. Examine the kidneys and bladder for calculi. If cystic calculi are present and if the patient is in stable condition, remove the calculi during the shunt ligation surgery. If operative time has been lengthy or if renal calculi are present, it may be best to schedule a second surgery. Obtain a liver biopsy (see p. •••) before closing the abdomen.

Jejunal Portography
Positive contrast radiographs can determine if the shunt is extrahepatic or intrahepatic. If the caudal extent of the PSS is cranial to T13, the shunt is probably intrahepatic. If the caudal extent of the shunt is caudal to T13, it probably is extrahepatic. Sensitivity of this procedure may be less in dorsal and right lateral recumbency than in left lateral recumbency (Scrivani et al, 2001).

Exteriorize a loop of jejunum. Identify a jejunal vein near the mesenteric border of the intestine and place one or two sutures around the vessel. Insert a 20- to 22-gauge over-the-needle catheter into the vessel (Fig. 20-17), and use the preplaced sutures to secure it to the vessel. Attach a heparinized extension set and three-way stopcock to the catheter.
Inject a water-soluble contrast agent (e.g., Renovist) (2 ml/kg of body weight) as a bolus into the catheter, and make an exposure while the last milliliter is injected. If necessary, make both lateral and ventrodorsal projections to help fully define the location of the shunt (Fig. 20-18). The catheter can also be used for pressure measurement.

With multiple hepatic shunts, radiographic confirmation of the shunts is rarely necessary. The technique of intraoperative mesenteric portography in these patients is the same as for single PSS, except that exposures should be delayed approximately 3 or 4 seconds after the start of injection of the contrast material to allow filling of the shunting vessels.

Transvenous Retrograde Portography

Transvenous retrograde portography provides a method of identifying and characterizing shunts without the need for abdominal surgery (Miller et al, 2002). This technique may be particularly useful in dogs in which the presence of a shunt is questioned because of an atypical history (e.g., a much older patient), discordant findings between nuclear scintigraphy and ultrasonography, or when surgical identification of a shunt was unsuccessful in an animal in which one was strongly suspected preoperatively.

Place the patient in left lateral recumbency, and aseptically prepare the right jugular furrow. In patients that weigh less than 10 kg, catheterize the jugular vein using a percutaneous technique. If the patient weighs more than 10 kg, perform a jugular venous cutdown to facilitate insertion of a large catheter into the vein.

The large catheter is required in big dogs to totally occlude the caudal vena cava.

Following insertion into the introcuder or venotomy site, direct the occluding catheter down the cranial vena cava and then in a dorsal direction into the azygos vein. Advance the catheter into the azygos vein as far as is possible without resistance. Inflate the balloon only enough to occlude the vein.

Occasionally, when larger balloon catheters are used, inflation of the balloon is unnecessary because the catheter itself may occlude the lumen sufficiently to allow for retrograde filling of the azygos vein. This seems to be especially true when the azygos vein is normal.

Make a vigorous hand injection of contrast (1 to 2 ml/kg) during fluoroscopic evaluation.

This injection normally results in retrograde filling of the intercostal and vertebral vessels. It is common to see the contrast flow in retrograde fashion into the abdominal cava.

Record the entire injection on videotape. If necessary, make an additional injection if hard copy of the study is required. Make initial images in the lateral projection; perform ventrodorsal or oblique views when indicated.

After the initial injection into the azygos vein, withdraw the catheter into the right atrium and then advance it caudally through the right atrium and into the caudal vena cava.

NOTE: In patients weighing less than 10 kg, use a 7.5 French dual-lumen flow balloon (Swan-Ganz) catheter; in patients that weigh more than 10 kg, use variably sized balloon dilation catheters to facilitate occlusion of the caudal vena cava.
Part II  Soft Tissue Surgery

Advance the catheter to a position immediately cranial to the diaphragm. Once the catheter is in position, inflate the balloon enough to completely occlude the caudal vena cava. Once the caudal vena cava has been occluded, make a vigorous hand injection of contrast (1 to 2 ml/kg) during fluoroscopic evaluation (Figs. 20-19 and 20-20). Occlusion of the caudal vena cava results in retrograde filling of the abdominal cava and the shunt.

In some cases the retrograde filling of the shunt is suboptimal. In those cases, positive pressure ventilation (20 cm H₂O for 5 to 8 seconds) during the injection usually results in improved retrograde filling of the shunt.

**NOTE:** It is imperative that the occlusion of the caudal vena cava and subsequent contrast injection be made immediately cranial to the diaphragm. If the occluding balloon is placed at the level of or caudal to the diaphragm, the ostia of shunts that arise in a cranial position may be occluded by the balloon, resulting in a false negative study.

Once the shunt has been identified, attempt selective catheterization of the shunt.

Selective catheterization with a flow-directed balloon allows for more specific opacification of the shunt, providing more detailed anatomic information. In addition, the configuration of the flow-directed balloon catheter allows for measurement of portal pressure both when the shunt is open and when it is occluded by the inflated balloon. Furthermore, selective catheterization of the shunt provides the opportunity to leave the catheter in the shunt lumen, facilitating intraoperative identification of the anomalous vessel.

Remove the catheter after the vessel has been identified and isolated. Remove the introducer and apply local pressure or sacrifice (ligate) the jugular vein and close the skin routinely.

The timing of removal of the introducer and catheters depends on whether the surgeon desires the catheter to be left in the lumen of the shunt during surgery.

**Ligation or Attenuation of Intrahepatic Shunts**

Both intravascular and extravascular methods have been described for ligation of IHPSS. Ligation of IHPSS can be extremely challenging because the vessel often is difficult to locate. Occasionally the shunt can be identified as a palpable depression or soft spot in a liver lobe, or it may be seen entering the caudal vena cava if it is not completely encircled by hepatic parenchymal tissue. Intraoperative ultrasound scans have been used to help identify the shunt in hepatic tissue, but this technique is not always successful. Intrahepatic shunts are classified as left, central, or right sided. Left and central divisional shunts account for a majority of shunts (see Fig. 20-14). Left sided IHPSS (patent ductus venosus) are typically located in the left lateral or medial hepatic lobes. Ligation or attenuation of the left hepatic vein may be performed in these animals. Central shunts are generally found in the right medial lobe, whereas right shunts are typically located in the right lateral or caudate lobes. An intravascular technique involving temporary hepatic vascular occlusion in conjunction with caudal caval venotomy was described by Breznock for intrahepatic shunt occlusion; however, because this procedure is technically difficult and surgery time is prolonged, many surgeons prefer extravascular techniques. Isolation and obstruction of the specific branch of the portal vein supplying the IHPSS have been described. Indirect passage of suture for ligation of right...
sided intrahepatic PSS was recently reported (Tobias et al, 2004). The ligature should encircle the right portal branch approximately 4 mm lateral to its bifurcation from the parent vein (see later discussion).

**NOTE:** Warn owners that ligation of IHPSS is difficult because the shunts are often hard to identify at surgery.

### Isolation and Ligation of IHPSS Involving the Left Medial or Lateral Liver Lobes

Many shunts can be found cranial to the liver. Extend the abdominal incision proximally into the caudal sternebrae. Incise the diaphragm if necessary. Incise the left triangular ligament, and free the left lateral liver lobe so that it can be retracted to the right. Use a combination of sharp and blunt dissection to isolate the anomalous vessel at its junction with the hepatic vein. Place a single silk ligature around the vessel and attenuate flow while measuring portal pressure. Alternately, ligate or attenuate the left hepatic artery as it enters the liver while measuring portal pressure.

### Isolation and Ligation of Right Sided IHPSS

If necessary, ligate the right hepatic duct. Pass a Carmalt forceps from the dog’s right to left over the dorsal surface of the main portal vein, just caudal to its bifurcation, but cranial to the termination of the gastroduodenal vein (Fig. 20-21). Grasp one end of a 2-0 silk suture, and pull it back over the portal vein. Then pass the forceps from the dog’s right to left, dorsal to the left portal vein and within 5 mm of its bifurcation from the main portal vein. Grasp the opposite end of the suture, and pull it back over the vein.

### Partial Hepatectomy for Removal of Hepatic Arteriovenous Fistula

Treatment of a hepatic A-V fistula involves removal of the affected lobes and abnormal vascular structures. This has been done with or without temporary hepatic vascular occlusion. If temporary vascular occlusion is used, the vascular clamps and occlusive ligatures should be released within 15 minutes.

> Extend the abdominal incision cranially through the caudal sternebrae, and incise the diaphragm down to and partly around the hiatus of the caudal vena cava. Place moistened umbilical tapes around the thoracic portion of the caudal vena cava, the abdominal portion of the caudal vena cava (between the liver and renal veins), and the portal vein (just proximal to the first hepatic branch). Pass the umbilical tapes through a piece of rubber tubing (Rumel tourniquet). Identify, isolate, and ligate the phrenicoabdominal veins, and isolate the celiac and cranial mesenteric arteries. Place a purse-string suture in the portal vein or a splenic tributary, and pass a 3.5 or 5 French catheter into the vessel to monitor portal pressure. Monitor blood pressure carefully during surgery; manipulation and ligation of the fistula may cause sudden, severe fluctuations. Isolate the affected lobes by dissection of the triangular, coronary, and hepatorenal ligaments and the ligaments of the lesser omentum. Identify the hepatic arterial branch supplying the affected lobe and temporarily occlude it to see if pressure in the fistula diminishes.
Double ligate the arterial supply of the fistula with non-absorbable suture (e.g., 2-0 silk). Isolate the portal branch and biliary ducts to the affected lobe and double ligate them. Temporarily occlude the vasculature by tightening the pre-placed umbilical tape ligatures and by placing vascular clamps on the celiac and cranial mesenteric arteries. Sharply dissect the liver parenchyma to resect the affected lobe. Ligate any vascular structures not already occluded and control hemorrhage by packing the area for several minutes. Sometimes the affected portion of the liver can be removed by partial hepatectomy without performing vascular occlusion as described here.

**SUTURE MATERIALS AND SPECIAL INSTRUMENTS**

Generally 3.5- and 5-mm ameroid constrictors are used for occlusion of single extrahepatic shunts. Blunt-tipped, right-angled, or Mixter forceps are useful for dissecting around venous structures. Shunt ligation usually is performed with silk suture because of the relative knot security this suture affords. Delayed wound healing may be a problem if the patient is hypoproteinemic. To prevent dehiscence, a long-lasting absorbable suture material, such as polydioxanone or a nonabsorbable suture material, should be used to close the linea alba.

**NOTE:** Ameroid constrictors are available through Research Instruments Northwest, 1369 N. 47th Ave., Sweet Home, OR 97386 (541-753-2018).

Right-angled forceps (e.g., Mixter, gallbladder or gall duct, or thoracic forceps) are widely available from many instrument manufacturers or suppliers, including Weck, Miltex, V. Mueller, Scanlan, and Codman.

**POSTOPERATIVE CARE AND ASSESSMENT**

Generally, animals can be sent home the day after placement of an ameroid constrictor. Continuing medical management and feeding a low-protein diet may be necessary until the shunt vessel occludes and the hepatic parenchyma regenerates. The patient should be reevaluated 2 to 3 months after surgery and tested for evidence of improved hepatic function (i.e., normal serum albumin). The animal may be weaned off medications and returned to a normal diet when hepatic function is determined to be reasonable.

When shunt ligation or attenuation is performed, intensive care management and close observation of the patient are extremely important because portal hypertension may develop several hours after the procedure. Hypertension and splanchnic congestion may be evidenced as a painful abdomen, hemorrhagic diarrhea, endotoxic shock, and death. Many shunt patients have a painful abdomen during the early postoperative period, which makes it difficult to recognize life-threatening portal hypertension. However, should signs of endotoxic shock or hemorrhagic diarrhea or other signs of a deteriorating condition occur, emergency surgery to remove or loosen the ligature around the shunting vessel is advisable. Portal vein thrombosis may occur in single PSS cases in which the shunt has been partly ligated; it is a potentially life-threatening complication. If a shunt is only partly ligated, some authors recommend a single anticoagulant dose of regular heparin at the time of shunt attenuation. Ascites may occur after single shunt ligation; it is usually self-limiting, resolving in 1 to 3 weeks. Diuretics may be used if drainage occurs from the incision site or if the animal experiences dyspnea or discomfort from abdominal distention.

Status epilepticus after PSS ligation has been reported. These seizures generally are first noted 2 to 3 days after shunt ligation; their cause is unknown. This complication seems more common in cats, so much so that some recommend routine pretreatment of cats undergoing surgery for PSS with phenobarbital (not potassium bromide, which can cause respiratory problems in cats). Diazepam is not recommended for these patients; constant rate infusion of propofol seems to be the most effective therapy (Heldman et al, 1999). The patient is anesthetized for 24 hours and then awakened. If seizures recur, then the patient is reanesthetized and awakened in another 24 hours. This cycle is repeated until control is achieved. Long-term anticonvulsant therapy may be required. Owners should be counseled that permanent neurologic abnormalities, such as blindness, may occur (especially in cats). Medical management of hepatic encephalopathy should be continued postoperatively until the hepatic parenchyma regenerates; this may take several months. If the clinical signs have not improved within 2 to 3 months, nuclear scintigraphy or jejunal portography should be repeated.

**PROGNOSIS**

Overall mortality in a recent study was 8.7% for extrahepatic shunts and 20% for IHPS (Winkler et al, 2003). Most dogs have an excellent or good outcome after placement of an ameroid constrictor. The complication rate for ameroid constrictors in the aforementioned study was 15.4%. Complications occurred in approximately 10% of dogs, and the mortality rate was 7.1% in another study (Mehl et al, 2005). Potential complications include hemorrhage, ascites, seizures, and/or coagulopathies. Portal hypertension may be secondary to kinking of the shunt; limiting dissection around the vessel may reduce this complication. The cause of seizures in these patients is not well understood (see previous discussion), but animals that have grand mal seizures after surgery are more likely to die than those that have partial seizures characterized by disorientation, hyperesthesia, vocalization, salivation, and/or jaw clenching. In one study, 12% of dogs developed neurologic signs within 6 days of surgical attenuation of congenital extrahepatic shunts (Tisdall et al, 2000). Prophylactic treatment with phenobarbital did not significantly reduce the incidence of neurologic sequelae, but it may have made the seizures less severe. In addition, development of multiple acquired
shunts may occur after placement of an ameroid constrictor (see previous discussion). With ligation, the surgical mortality is relatively high, a fact that reflects the many variables and unknown factors that exist in relation to portal physiology and dynamics.

Patients that only tolerate partial shunt occlusion and have persistence of clinical signs postoperatively require dietary and medical management. In such animals, reoperation and total shunt occlusion are recommended. Dogs that tolerate complete ligation of their shunt tend to have fewer clinical signs than those that tolerate only partial occlusion at surgery. Factors that appear to be significant predictors of continued portosystemic shunting include low preoperative albumin concentrations, high port pressure during incomplete temporary occlusion of the PSS, and high portal pressure difference (Mehl et al, 2005). Predictors of an unsuccessful long-term outcome include lower preoperative albumin concentration, high WBC count, high portal pressure measured during complete temporary occlusion of the PSS, postoperative seizures, and continued shunting detected via portal scintigraphy. Outcome after placement of cellophane bands seems to be similar to that after placement of ameroid constrictors.

Hemorrhage, hypotension, and acute hepatic congestion are possible complications during surgical correction of IHPSS in dogs. Packed cell volume and total protein may be positive prognostic indicators for long-term survival in dogs with IHPSS, whereas low body weight (less than 15 kg) and low total protein, albumin, and BUN may be negative prognostic factors (Papazoglou et al, 2002). The long-term prognosis is good for dogs with hepatic A-V fistulae that survive surgery.

References


Suggested Reading


Results of this study suggest that long-term outcome of ameroid constrictor occlusion of PSS in cats is poor.


This retrospective study of 233 dogs and 9 cats with PSS affirmed that breed has a significant influence on shunt anatomy in dogs. Unusual or inoperable shunts are more likely to occur in breeds that are not predisposed to congenital PSS.
CAVITARY HEPATIC LESIONS

DEFINITIONS

Cavitary hepatic lesions usually are cysts or abscesses, although occasionally large neoplastic lesions, such as hemangiomas and adenomas, cavitate. Hepatic abscesses are localized collections of pus in the hepatic parenchyma. Hepatic cysts are closed, fluid-filled sacs lined by secretory epithelium.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Hepatic abscesses are rare in dogs and cats and usually are associated with extrahepatic infection (i.e., ascending biliary tract infections, hematogenous infection via the portal vein or hepatic artery, or direct extension from areas adjacent to the liver), hepatic trauma (i.e., surgical biopsy, penetrating wounds, or blunt trauma), or neoplasia. Despite the normal presence of bacteria in the liver of dogs, hepatic abscesses seldom occur. This may be related to a well-developed local defense system provided by the liver’s rich blood supply and the phagocytic ability of reticuloendothelial cells.

Hepatic abscesses are most often recognized as a complication of omphalophlebitis in puppies and usually are diagnosed at necropsy. Diabetes mellitus has been associated with hepatic abscesses. The organisms most often isolated from hepatic abscesses in dogs include *Escherichia coli* and *Clostridium* spp. Small abscesses may not cause clinical signs and may resorb without therapy.

Hepatic cysts usually are incidental findings, although in rare cases they become large enough to interfere with the function of adjacent organs. A single hepatic cyst may be noted, or several cysts may be present in the same or different lobes. Concurrent polycystic renal disease has been reported in cats. If hepatic cysts are present in an animal with clinical evidence of hepatic dysfunction, liver biopsy often is warranted to determine the cause.

DIAGNOSIS

Clinical Presentation

Signalment. No gender or breed predisposition has been reported for hepatic abscesses or cysts.

History. Clinical signs of hepatic abscessation vary and may include anorexia, lethargy, weight loss, vomiting, and intermittent abdominal pain. Most animals with hepatic cysts are asymptomatic; however, some cysts cause abdominal distention. Secondary infections of hepatic cysts may cause clinical signs similar to those of hepatic abscesses.

Physical Examination Findings

Physical examination findings in patients with hepatic abscessation typically include persistent fever, hepatomegaly, and abdominal enlargement. Palpation of a firm abdominal mass and notable abdominal distention may be noted in some animals with hepatic cysts. In a recent study of hepatic abscesses in 14 cats, clinical signs were vague and included anorexia, lethargy, and weight loss. Fever was present in only 23% of the cats, whereas 31% were hypothermic (Sergeeff et al., 2004). Clinical signs of sepsis are common in cats with small multifocal abscesses and in those with microabscesses.

Diagnostic Imaging

Small hepatic cysts often are incidental radiographic and sonographic findings. Large hepatic cysts usually are well-defined, radiopaque structures in the cranial abdomen (Fig. 20-22). Abdominal radiographs may demonstrate hepatomegaly in animals with hepatic abscesses, but a well-defined hepatic mass is seldom evident. Occasionally, gas is noted in the hepatic parenchyma, which strongly suggests abscessation caused by gas-forming bacteria. Ultrasonography is the most useful diagnostic test for defining hepatic abscesses and cysts in dogs and cats. Hepatic abscesses appear as hypoechoic or anechoic structures that may contain mixed echogenecities, depending on the cellularity. The abscesses may appear solitary, multifocal, and small, or they may be microabscesses. Scintigraphy, CT, and magnetic resonance imaging (MRI) also are highly sensitive in diagnosing hepatic lesions, but these techniques are used less often than ultrasonography. Ultrasonography-guided fine-needle aspirations of hepatic abscesses can be performed before surgery; however, there is a risk that the abscess will rupture or drain into the abdomen and cause diffuse peritonitis. Fluid removed from cysts during fine-needle aspiration usually is transudative.

NOTE: Evaluate the kidneys for cystic disease in cats with hepatic cysts. Both conditions may be present.

Laboratory Findings

Laboratory abnormalities are seldom present with hepatic cysts. They are variable in animals with hepatic abscesses, but may include an inflammatory leukogram and nonregen-
operative anemia. Serum biochemical abnormalities may include hypoalbuminemia, hypokalemia, hyperglycemia, and elevated hepatic enzymes; however, elevation of alanine transaminase activity is not a consistent finding. *E. coli* is the organism most commonly isolated from cats with hepatic abscesses (Sergeeff et al, 2004).

**DIFFERENTIAL DIAGNOSIS**

Hepatic cysts, abscesses, neoplasms, and parasitic lesions must be differentiated. Hepatic abscesses often are difficult to diagnose because they produce nonspecific signs that may be masked by associated disease processes. Large neoplastic hepatic lesions may necrose and become secondarily infected. Infection of hepatic cysts is also possible. Therefore, histologic evaluation of surgically resected tissue is important.

**MEDICAL MANAGEMENT**

Medical management of hepatic abscesses entails administration of fluid/electrolyte/acid-base therapy plus appropriate antibiotic therapy. Percutaneous ultrasound-assisted drainage and alcoholization using 95% ethanol has been reported for treatment of focal abscesses in 5 dogs and a cat (Zatelli et al, 2005).

To perform this technique, position a spinal needle attached to extension tubing and a syringe into the abscess using ultrasound guidance and drain the fluid. Use a syringe that is twice the volume of the estimated amount of exudate in the lesion. Before removing the spinal needle, inject a volume of alcohol equal to half the volume of exudate removed. Leave the ethanol in the abscess cavity for 3 minutes, and then gently remove it. Submit the exudate for culture and susceptibility testing. Continue appropriate antibiotic therapy for an additional 30 days.

If surgery is elected, resection of hepatic abscesses is indicated as soon as the animal is an acceptable anesthetic risk. Preoperative antibiotic therapy may be based on culture and sensitivity results if fine-needle aspiration has been performed, or antibiotics with bactericidal activity against anaerobes and gram-negative bacteria (e.g., ticarcillin/clavulanic acid plus enrofloxacin, or cefoxitin; or clindamycin plus enrofloxacin, or ticarcillin/clavulanic acid plus amikacin [Box 20-6]) may be given empirically. Parenteral antibiotics are indicated in the perioperative period. Combination therapy may be necessary, particularly if multiple organisms are isolated. Percutaneous drainage of hepatic cysts and sclerosis of the cyst lining have not been reported in dogs or cats.

**SURGICAL TREATMENT**

Whether hepatic cysts should be removed when diagnosed in asymptomatic animals is not clear. Although these cysts could enlarge or become infected and cause clinical signs, little information is available about the long-term follow-up of large hepatic cysts that are not surgically resected in dogs or cats. Hepatic cysts associated with clinical signs and hepatic abscesses should be promptly resected.

**Preoperative Management**

Symptomatic animals should be in stable condition before surgery. Antibiotics may be initiated before surgery, or in some animals they may be administered after intraoperative cultures have been taken.

**Anesthesia**

See pp. •••••• for the anesthetic management of animals with hepatic disease.

**Surgical Anatomy**

See p. •••• for the surgical anatomy of the liver.

**Positioning**

The animal is positioned in dorsal recumbency for a midline abdominal incision. The prepped area should extend from mid thorax to the pubis.

**SURGICAL TECHNIQUE**

Hepatic abscesses and cysts generally are treated by partial hepatectomy (see pp. ••••••). If hepatectomy cannot safely be performed and the cyst completely removed, it may be omentalized (Friend et al, 2001). Although there is less concern about spillage of cystic contents into the abdomen, it is wise to try to remove the cyst without entering the lumen. Culture of hepatic cysts may be optional if the fluid does not
appear infected cytologically; however, some cysts can develop secondary bacterial infections.

Pack the area surrounding the liver with moistened laparotomy sponges to diminish intraoperative contamination if the lumen of the abscess or cyst is entered. If possible, resect the affected portion of the liver without entering the lesion. Culture the lesion, and submit it for histologic examination. Palpate the remainder of the liver parenchyma for other nodules, and explore the abdominal cavity for associated infection or disease.

For omentalization, identify a segment of the omentum that will extend into the cyst cavity. Remove as much of the wall of the cyst as possible, and spread the omentum over the remaining cyst and adjacent liver. Tack it gently in place to the remaining cyst capsule.

**SUTURE MATERIALS AND SPECIAL INSTRUMENTS**

See p. ••• for recommendations for suture choices during partial hepatectomy.

**POSTOPERATIVE CARE AND ASSESSMENT**

Fluid therapy for animals with hepatic abscesses should be continued until the animal is drinking normally. Antibiotic therapy should be continued for 7 to 10 days. The animal should be monitored for peritonitis (i.e., leukocytosis, fever, abdominal fluid, abdominal pain) if abdominal contamination occurred. Minimal postoperative care is needed for most animals with hepatic cysts.

**PROGNOSIS**

The prognosis for animals with hepatic abscesses depends on the rapidity with which the abscess is diagnosed, whether concurrent peritonitis is present, and the animal’s overall health. The overall mortality rate in a recent report of 14 cats with hepatic abscesses was 70% (Sergeeff et al, 2004). The prognosis for animals with hepatic cysts (with or without surgery) is good unless concurrent hepatic or renal disease exists.

**References**


**HEPATOBILIARY NEOPLASIA**

**DEFINITIONS**

Hepatocellular tumors arise from hepatocytes; cholangiocellular neoplasms arise from intrahepatic or extrahepatic bile duct epithelium. The term hepatoma has been used to refer both to hepatocellular carcinomas and to hepatocellular adenomas. Cholangiocellular carcinomas are also known as bile duct carcinomas.

**GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY**

Primary hepatic neoplasms are uncommon in dogs and cats. They may be of epithelial or mesenchymal origin (Box 20-7). Hepatocellular carcinomas and cholangiocellular carcinomas are the most commonly diagnosed primary hepatic malignancies in dogs. Hepatocellular carcinomas may involve a single liver lobe or may be nodular or diffuse, involving multiple lobes. In cats, cholangiocellular adenomas are the most common primary tumor. Hepatic carcinoids are rare tumors that arise from neuroectodermal cells in the liver. Biliary cystadenomas are uncommon benign liver tumors of older cats that may occur as focal or multifocal cystic lesions. Benign hepatic masses (i.e., adenomas or cysts) often are incidental findings at necropsy. They may be more common than malignant tumors in both species, but often go undiagnosed because they seldom cause clinical signs. Cholangiocellular carcinomas arise primarily from intrahepatic bile duct epithelium; neoplasms of the extrahepatic bile duct and gallbladder are rare.

Malignant primary hepatic tumors have typically been considered to be highly metastatic; however, a median survival of greater than 1460 days and a metastatic rate of only 4.8% have been reported after lobectomy for hepatocellular carcinoma (Liptak et al, 2004). They may metastasize by direct extension to other liver lobes or adjacent organs, or they may spread distantly via lymphatics or blood. Epithelial tumors most often metastasize to the regional lymph nodes and lungs. Mesenchymal tumors most often metastasize to the spleen.

Metastatic neoplasia is more common in the liver than are primary tumors. The liver is a common site for metastasis because it acts as a filter between the abdominal organs and the systemic circulation. Lymphosarcoma is the most common secondary hepatic tumor. Other tumors that commonly metastasize to the liver are pancreatic adenocarcino-

**BOX 20-7**

Primary Hepatic Neoplasia in Dogs and Cats

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<td>- Cholangiocellular carcinoma</td>
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<td>- Extraskeletal osteosarcoma</td>
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**INSTRUMENTS**

See p. ••• for recommendations for suture choices during partial hepatectomy.
mas, hemangiosarcomas, insulinomas, and tumors of the alimentary and urinary tracts.

**DIAGNOSIS**

**Clinical Presentation**

**Signalment.** Primary hepatic neoplasia usually is a disease of aged dogs and cats. There is no known breed predisposition. Hepatocellular carcinomas may be more common in male dogs, whereas cholangiocellular carcinomas may be more common in cats and female dogs. Dogs with metastatic liver cancer may be slightly younger (7.8 years of age) than those with primary hepatic malignancy (10 years of age).

**History.** Many animals with primary hepatic neoplasia are presented for treatment of signs associated with hepatic failure. The animal may be lethargic, weak, anorexic, losing weight, or vomiting or may have polyuria or polydipsia. The clinical signs associated with metastatic hepatic neoplasia vary considerably. Primary hepatic tumors and metastatic hemangiosarcomas may rupture and bleed, causing signs of shock.

**Physical Examination Findings**

The most significant finding on physical examination of most primary hepatic tumors is an enlarged liver; however, hepatic carcinoids may not cause significant hepatomegaly. Additional findings may include jaundice and ascites. Hemangiosarcomas and hepatocellular adenomas may rupture and cause hemoperitoneum, shock, and pale oral mucous membranes. Notable hepatomegaly is less common with metastatic neoplasia; however, lymphosarcoma often causes diffuse hepatic enlargement.

**Diagnostic Imaging**

Survey radiographs help localize the mass to the liver (Fig. 20-23) and may reveal extrhepatic metastasis. Thoracic radiographs are indicated whenever hepatic neoplasia is suspected because pulmonary metastasis is common. Ultrasonography often localizes and defines the extent of disease. Ultrasound-guided biopsies may allow presurgical diagnosis (see p. •••), but these tumors are often highly vascular and can bleed profusely. Although conventional ultrasound may detect lymph node enlargement and therefore help define metastasis in dogs with hepatic neoplasia, Doppler and contrast-enhanced ultrasound may prove to be even more sensitive diagnostic aids (Nyman et al, 2004).

**Laboratory Findings**

Neutrophilia and biochemical abnormalities compatible with hepatic disease (elevated serum alanine transaminase, aspartate transaminase, and serum alkaline phosphatase) are expected but often absent in animals with hepatic neoplasia. They are nonspecific, but recognition may prompt further evaluation of the hepatobiliary system. Mild to moderate anemia is less commonly associated with hepatic neoplasia. Serum bilirubin concentrations may be elevated, particularly if extrahepatic biliary obstruction occurs. Occasionally, hypoglycemia causes clinical signs. Albumin levels usually are normal in patients with primary hepatic neoplasia. Biochemical abnormalities seldom correlate with the extent of hepatic involvement with either primary or metastatic tumors.

**DIFFERENTIAL DIAGNOSIS**

Primary hepatobiliary tumors must be differentiated from regenerative nodules, abscesses, hematomas, and cysts. Histologic and/or cytologic evaluation of fine-needle aspirates or biopsy specimens is necessary to distinguish definitively between these lesions (see p. •••). Percutaneous biopsies should not be performed in animals with clinical bleeding disorders or if the lesions appear cavitary or highly vascular. Cytologic evaluation of abdominal fluid is seldom helpful in differentiating between these lesions.

**MEDICAL MANAGEMENT**

Surgical excision of primary malignant hepatic tumors is the treatment of choice. Unfortunately, these tumors often are not diagnosed until they are large and metastasis has occurred. Because they usually are diagnosed in older animals, concurrent cardiac, renal, or other metabolic problems are...
common. Medical therapy should aim at correcting fluid and electrolyte imbalances and providing nutrition to improve the chances of surviving surgery.

**SURGICAL TREATMENT**

If the tumor is localized to a single lobe or confined to the gallbladder, surgical resection may be curative. Partial hepatectomy and cholecystectomy are described on pp. *** and ***, respectively. Ultrasound is often used to screen for metastasis, but it is not a particularly sensitive way to detect these lesions, particularly if the metastases are on serosal surfaces. Although not as sensitive as an exploratory laparotomy, laparoscopy is more sensitive than ultrasound for finding metastasis. One can first perform laparoscopy to look for gross evidence of metastasis before proceeding to surgery to resect a tumor.

Surgical biopsies should be performed on all animals with hepatomegaly or nodularity because differentiation of lesions requires histopathologic evaluation. Finding multiple hepatic masses does not diagnose metastatic disease; many benign lesions can present as multiple hepatic nodules (e.g., regenerative nodules associated with cirrhosis or lobular collapse) or even neoplasia because primary hepatic tumors may spread to other portions of the liver. Multiple benign masses may be seen in the liver. If neoplasia is suspected, the draining lymph nodes and surrounding organs should be carefully assessed for metastasis. Hepatocellular tumors are most commonly found in the left medial and left lateral liver lobes.

**PREOPERATIVE MANAGEMENT**

The animal’s condition should be stabilized before surgery if possible. Fluid therapy should be initiated and electrolyte imbalances corrected. Blood transfusions (see Table 5-4 on p. ***) should be given to severely anemic animals (i.e., packed cell volume less than 20%), especially if bleeding tendencies are present (i.e., petechiation, ecchymosis, or hemorrhage). If the animal has clinical evidence of a significant coagulopathy on the mucosal bleeding time (i.e., bleeding time >5 to 7 minutes) or is severely thrombocytopenic (i.e., fewer than 20,000 platelets/µl), consider plasma or whole blood transfusions and ensure hemostasis at surgery. Many patients with a prolonged one-stage prothrombin time (OSPT or PT) and partial thromboplastin time (PTT) do not have bleeding problems at surgery, but they should be monitored for such before, during, and after surgery. If the patient has massive ascites, slow removal of some fluid before induction of anesthesia may help prevent hypoventilation associated with positioning the patient while it is prepared for surgery.

**Anesthesia**

Ventilation of patients with ascites requires support (i.e., intermittent positive-pressure ventilation [IPPV]). Compression of the caudal vena cava in patients with large hepatic masses or massive ascites may diminish venous return and reduce cardiac output. See pp. *** for additional comments about the anesthetic management of patients with hepatic disease.

**Surgical Anatomy**

See p. *** for the surgical anatomy of the liver.

**Positioning**

Exploration of the liver generally is performed through a cranial ventral midline abdominal incision (see p. ***). The incision may be extended paracostally to allow enhanced visualization and manipulation of large tumors. The prepped area should extend from midthorax to the pubis.

**SURGICAL TECHNIQUE**

See pp. *** and ** for a description of surgical techniques for partial hepatectomy or cholecystectomy, respectively.

**SUTURE MATERIALS AND SPECIAL INSTRUMENTS**

Absorbable suture material is used for hepatic biopsy (see p. ***). Ligation of the cystic duct for cholecystectomy generally is done with nonabsorbable suture material (see p. ***).

**POSTOPERATIVE CARE AND ASSESSMENT**

Postoperative nutritional support of patients with hepatic neoplasia often is necessary (see Chapter 11). Nonresectable primary hepatic tumors seldom respond to chemotherapy or radiation therapy. Chemotherapy may palliate hepatic lymphosarcoma. For other considerations in animals undergoing partial hepatectomy, see p. ***.

**PROGNOSIS**

The prognosis for dogs and cats with primary hepatobiliary malignancies often is poor; however, some dogs may live for a year or longer with aggressive therapy. In a recent report of cats with malignant, nonlymphomatous hepatobiliary disease that underwent surgery, the median length of the survival was 0.1 month (range, less than 1 day to 4 months). The high rate of metastasis and degree of invasion make surgical resection unlikely to be curative in most patients. Benign tumors may be surgically resected, and long-term survival of patients with benign hepatic tumors has been reported. Survival times in cats with hepatobiliary cystadenomas has ranged from 12 to 44 months after surgery.

**References**


**HEPATIC LOBE TORSION**

**DEFINITIONS**

Hepatic lobe torsion occurs when a liver lobe twists around its axis.
Liver lobe torsions are rare in dogs and cats. Torsion of the left lateral lobe is most common, presumably because it has greater mobility, is larger, and is relatively more separated from the adjacent lobes than are the other liver lobes. In most animals the cause is unknown, but congenital absence or traumatic rupture of hepatic ligaments is generally suspected. A ruptured hepatocellular carcinoma was diagnosed in one cat with torsion of the right medial liver lobe (Swann and Brown, 2001). When a liver lobe twists on its axis, it creates venous obstruction, causing increased hydrostatic pressure, ascites, and thrombosis. The liver lobe will eventually necrose.

**Clinical Presentation**

**Signalment.** No breed or sex predisposition has been identified for hepatic lobe torsions in dogs or cats. Most reported dogs have been middle-aged.

**History.** Clinical signs of hepatic lobe torsion are often nonspecific and may include depression, lethargy, anorexia, collapse, and/or abdominal enlargement of one to several days’ duration. Acute death may occur.

**Physical Examination Findings**

Physical examination findings may include pain on abdominal palpation and the presence of ascites. The animal should be examined carefully for signs of trauma.

**Diagnostic Imaging**

Survey radiographs may show ascites and should be reviewed for signs of associated trauma (e.g., diaphragmatic hernia). Ultrasonography may help define the lesion and localize it to a defined area of the liver.

**Laboratory Findings**

Blood work abnormalities are nonspecific and are not helpful in identifying hepatic lobe torsion. Neutrophilia may be present and biochemical abnormalities compatible with hepatic disease (elevated serum alanine transaminase, aspartate transaminase, and serum alkaline phosphatase) are commonly seen.

**Differential Diagnosis**

Liver lobe torsion must be differentiated from nonsurgical diseases of the liver, such as hepatitis.

**Medical Management**

Surgical excision of torsed hepatic lobes is warranted. The animal should be stabilized before surgery.

**Surgical Treatment**

Surgical resection of the devitalized lobe is warranted. The technique for hepatic lobectomy is described on p. •••.

**Preoperative Management**

The animal’s condition should be stabilized before surgery if possible. Fluid therapy should be initiated and electrolyte imbalances corrected. If the patient has massive ascites, slow removal of some fluid before induction of anesthesia may help prevent hypoventilation associated with positioning the patient while it is prepared for surgery.

**Anesthesia**

Ventilation of patients with ascites may require support (i.e., IPPV). See pp. ••• for additional comments about the anesthetic management of patients with hepatic disease.

**Surgical Anatomy**

The liver lobe is supported by a series of ligaments, including the left and right lateral triangular ligaments, which extend from the left and right lateral hepatic lobes to the muscular portion of the diaphragm; left and right lateral coronary ligaments, which attach the right and left lobes to the central tendinous portion of the diaphragm; and the falciform ligament, which attaches to the liver, abdominal wall, and sternal portion of the diaphragm.

**Positioning**

Exploration of the liver generally is performed through a cranial ventral midline abdominal incision (see p. •••). The prepped area should extend from midthorax to the pubis.

**Surgical Technique**

See pp. ••• for a description of the surgical technique for removal of a liver lobe. Histologic evaluation of the excised liver lobe is warranted, as the devitalized mass may be similar in appearance to a hepatic tumor.

**Suture Materials and Special Instruments**

Absorbable suture material is used for liver lobectomy (see p. •••).

**Postoperative Care and Assessment**

Instructions on postoperative care of the patient after liver lobe resection are provided on p. •••. Dogs with associated diaphragmatic hernias or other trauma should be carefully observed for evidence of respiratory distress after surgery. Fluid support should be continued until the animal is eating and drinking sufficiently on its own. Pain medication should be given postoperatively (see p. •••).

**Prognosis**

The prognosis for dogs and cats with hepatic lobe torsion is good if the underlying disease can be effectively treated.

**Reference**