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Portosystemic Shunts

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Introduction

Portosystemic shunts (PSS) are vascular anomalies that divert blood from the abdominal viscera to the heart, bypassing the hepatic sinusoids and carrying intestinal absorption products directly to the systemic circulation. Portosystemic shunts can occur as congenital anomalies, or may develop secondary to liver disease and portal hypertension. While clinical signs from multiple acquired shunts must be managed medically, congenital PSS have been successfully treated with surgery in many dogs and cats.

Anatomy

Congenital PSS usually occur as single large vessels, while acquired shunts are numerous and often small in size. Common types of single congenital portovascular anomalies include intrahepatic portocaval shunts, such as patent ductus venosus, and extrahepatic portocaval or portal-azygos shunts. Shunts may connect the portal vein with the caudal vena cava directly, or may originate from a portal tributary, such as the left gastric vein, and terminate on a caval tributary, such as the phrenic vein. In a small percentage of dogs, the prehepatic portal vein is also congenitally absent.

Signalment, History, and Clinical Signs

Congenital intrahepatic and extrahepatic PSS are usually diagnosed in immature animals. No sex predilection is evident. Intrahepatic PSS are found primarily in large breed dogs such as Irish Wolfhounds, Old English sheepdogs, Golden and Labrador Retrievers, and Samoyeds, and in medium-sized breeds such as Australian shepherds and Australian Cattle dogs. Extrahepatic PSS occur primarily in small breed dogs such as the Yorkshire terrier, Schnauzer, poodle, Maltese, Shih Tzu, and dachshund. In cats PSS are most often extrahepatic. Congenital shunts are hereditary in Irish Wolfhounds and are thought to be hereditary in Yorkshire Terriers, Maltese, and Cairn Terriers.

Clinical signs associated with portosystemic shunts commonly involve the nervous system, gastrointestinal tract, and urinary tract. General clinical signs include poor growth rate, weight loss, fever, and anesthetic or tranquilizer intolerance. Neurologic dysfunction is seen in most animals with PSS and includes lethargy and depression, ataxia, seizures, behavioral changes, and blindness. Head pressing, circling, and development of a head tilt have also been reported. Gastrointestinal clinical abnormalities include anorexia, vomiting, and diarrhea. Some dogs have no apparent signs or present with signs of cystitis or urinary tract obstruction. Many cats have hypersalivation and some have unusual copper colored irises.

Hepatic Encephalopathy

Hepatic encephalopathy has been recognized in animals with PSS, end-stage liver
disease, and congenital urea cycle enzyme deficiencies. Clinical signs include depression, dementia, stupor, and coma. Muscle tremors, motor abnormalities, and focal and generalized seizures have also been reported. The etiology of hepatic encephalopathy is probably dependent on several factors, including circulating toxins, alterations in amino acid concentrations, and increased cerebral sensitivity to drugs and toxins. Toxins which have been implicated in hepatic encephalopathy include ammonia, mercaptans, short chain fatty acids, indoles, aromatic amino acids, and biogenic amines.

Precipitating factors of hepatic encephalopathy include diuretics, protein overload, hypokalemia, alkalosis, transfusion of stored red cells, hypoxia, hypovolemia, gastrointestinal hemorrhage, infection, and constipation. Increased cerebral sensitivity to sedative, analgesic, and anesthetic agents may induce coma in animals with PSS, even when normal dosages of the drugs are used. Protein overload and gastrointestinal hemorrhage provide substrates for bacterial production of ammonia, and constipation can increase retention and absorption of ammonia and other encephalopathic substances. Blood which has been stored for 24 hours contains 170 ug of ammonia/dL, and ammonia concentrations will continue to increase with prolonged storage.

Results of Diagnostic Tests

Abnormalities found on hemograms of animals with PSS include leukocytosis, anemia, and microcytosis. Some animals with congenital PSS have prolonged PTTs; however, this does not usually result in a clinically significant problem. Biochemical abnormalities associated with PSS in dogs include decreases in blood urea nitrogen, protein, albumin, glucose, and cholesterol; and increases in serum alanine aminotransferase and alkaline phosphatase. Increase in alkaline phosphatase is most likely from bone growth, since cholestasis is not usually a problem in animals with PSS. Cats with PSS usually have normal albumin and cholesterol concentrations. Urine abnormalities include low urine specific gravity and ammonium biurate crystalluria. At magnifications of 400x or more, ammonium biurate crystals often have a spikey, thornapple or starfish shape and golden color. Because of increased urinary excretion of ammonia and uric acid, dogs and cats may also develop uroliths. Urate uroliths are often radiolucent and therefore may not be detectable on survey radiographs unless they are combined with struvite. Abnormal urine sediment suggestive of cystitis (hematuria, pyuria, and proteinuria) has been described in animals with PSS and may be associated with crystalluria or urolithiasis.

Hepatic histologic changes in animals with PSS include generalized congestion of central veins and sinusoids, lobular collapse, bile duct proliferation, hypoplasia of intrahepatic portal tributaries, proliferation of small vessels and lymphatics, diffuse fatty infiltration, hepatocellular atrophy, and cytoplasmic vacuolization. These pathology changes can also be seen in dogs with hepatic microvascular dysplasia that do not have single congenital shunts. Pathologic changes may be present in the central nervous system, especially in encephalopathic animals.

Diagnosis of Portosystemic Shunts: Liver Function Tests

Although history, physical examination, and routine laboratory tests may be suggestive of portosystemic shunting, function tests such as ammonia tolerance test (ATT) and measurement of fasting and postprandial serum bile acid concentrations are more reliable for diagnosing liver dysfunction.

Serum bile acids are synthesized in the liver from cholesterol. After conjugation with taurine, they are secreted into bile and stored in the gallbladder. During food intake, neurohumoral and hormonal factors such as cholecystokinin stimulate gallbladder contraction and excretion of bile acids into the small intestines where they form micelles that enhance lipid emulsification and absorption. At least 95% of intestinal bile acids are actively reabsorbed in the ileum and are transported by portal blood back to the liver via the enterohepatic cycle. Normally postprandial bile acid concentrations are minimally increased because of rapid first-pass hepatic extraction. Serum bile acid concentrations are elevated with cholestasis, jaundice, and portosystemic shunting. They are not significantly affected by dehydration, hypovolemia, or passive hepatic congestion, although results can be mildly affected by lipemia and hemolysis. No special techniques are required for handling and storage of serum for bile acid samples. Prolonged fasting may result in normal bile acid concentrations in animals with PSS; therefore, fasting and 2-hour postprandial samples should be analyzed. If the animal is sensitive to high protein meals, a low protein diet mixed with a few milliliters of corn oil can be used to stimulate gastrointestinal motility and cholecystokinin activity. Occasionally fasting bile acid concentrations are greater than those measured postprandially due to spontaneous interdigestive contraction of the gallbladder.

Normal hepatic function is essential for conversion of ammonia to urea. Increased resting ammonia concentration indicates decreased hepatic mass or shunting of portal blood. Concentrations of blood ammonia are not well correlated with severity of hepatic encephalopathy.
and ammonia levels may be normal in 7% to 21% of dogs with PSS, especially after prolonged fasting. The ammonia tolerance test was developed to provide a more accurate diagnosis of liver dysfunction. A heparinized baseline sample is taken after a 12 hour fast, and ammonium chloride is administered orally by stomach tube or in gelatin capsules (0.1 g/kg, maximum 3 grams), or as an enema (2 ml/kg of a 5% solution inserted 20 to 35 cm into the colon). A second blood sample is obtained 30 minutes after ammonium chloride administration. Blood samples are transported on ice for immediate plasma separation and analysis. Normal values vary with the method of analysis; results in animals with PSS should be compared to a control sample from a healthy animal to ensure accuracy. Improper sample cooling, incomplete plasma separation, or delays in sample analysis will result in falsely elevated values because of erythrocyte and plasma generation of ammonia. Results are invalid after oral ammonium chloride administration if vomiting occurs, and after rectal administration if diarrhea or shallow rectal instillation occurs.

Diagnostic Imaging

Diagnostic imaging of microhepatica from survey abdominal radiographs is usually based on an upright, more cranial stomach position. Renomegaly has been reported in dogs with PSS; its etiology has not been determined. Urate calculi normally are radiolucent but occasionally will be seen in the renal pelvis, ureter, or bladder on survey films.

To accurately diagnose a portosystemic shunt and determine its location, imaging techniques such as angiography, ultrasonography, scintigraphy, and magnetic resonance angiography can be utilized. Intraoperative mesenteric portography provides excellent visualization of the portal system but usually requires a celiotomy. The dog is anesthetized and a small laparotomy is performed. Water-soluble contrast medium (maximum total dose, 2 ml/kg) is injected into a catheterized jejunal or splenic vein, and one or more radiographs are taken during completion of the injection. Alternatively, the spleen can be injected directly and percutaneously in a sedated dog. There is a risk of splenic laceration with this technique, however, and images are often of poorer quality compared to direct intravascular injection.

Because no dilution of contrast material occurs, intraoperative mesenteric portography provides an excellent image of the shunt if the vessel is not too large. The technique is relatively simple and requires no special equipment. Differentiation of intrahepatic and extrahepatic PSS may be made on most portograms. If the most caudal loop of the shunt or the point where the shunt diverges from the portal vein is cranial to the T-13 vertebra, then the shunt location is probably intrahepatic. The shunt location will vary by one half to three fourths of a vertebral length depending on the phase of respiration.

Diagnosis of PSS may be made with hepatic ultrasonography. Ultrasonographic evidence of PSS includes microhepatica, decreased numbers of hepatic and portal veins, and detection of the anomalous vessel. Extrahepatic PSS are more difficult to diagnose with ultrasonography; their location is often obscured by gas-filled intestines. Overlying ribs and lungs may also interfere with a thorough ultrasonographic evaluation. Colorflow doppler is useful for detecting changes in the direction and rate of blood flow in the portal vein.

Nuclear scintigraphy is a noninvasive means of evaluating dogs for portal venous shunting. In dogs, technetium pertechnetate is extracted from the circulation primarily by the liver. In animals with shunts, the pertechnetate rapidly circulates to the heart and lungs. Normal dogs have a shunt fraction of less than 15% on scintigraphy; most dogs with PSS have fractions greater than 60%.

The ultimate tool for diagnosing PSS is laparotomy. Once experience is obtained, most extrahepatic shunts and at least half of intrahepatic shunts can be identified on exploratory.

Differential diagnoses

Single congenital portosystemic shunts must be differentiated from multiple acquired shunts secondary to portal hypertension, and from hepatic microvascular dysplasia. Hepatic microvascular dysplasia (HMD) signifies a disorganization of the liver’s microscopic architecture which is similar to that of dogs with single congenital shunts. HMD has been reported in small breed dogs such as the Yorkshire terrier, Cairn terrier, Maltese, cocker spaniel, and poodle. Dogs with HMD display biochemical, hematologic, and clinical changes consistent with portosystemic shunting but lack a macroscopic portosystemic shunt. Therefore, in dogs with HMD, portograms and scintigrams are normal. Signs of HMD are managed by low protein diet; lactulose is added if necessary.
Medical management of PSS

Medical management of animals with PSS includes correction of fluid, electrolyte, and glucose imbalances and prevention of hepatic encephalopathy by controlling precipitating factors. Dietary protein is restricted to reduce substrates for ammonia formation by colonic bacteria. Gastrointestinal hemorrhage should be controlled with ranitidine and sucralfate if gastric irritation or ulceration is suspected, and with anthelmintics if parasites are present. Non-absorbable intestinal antibiotics that are effective against urease producing bacteria, such as neomycin, should be administered to decrease bacterial populations. Enemas and cathartics may be used to reduce colonic bacteria and substrates and are especially important in animals with hepatic encephalopathy.

Lactulose, a synthetic disaccharide, is hydrolyzed in the colon to organic acids that increase fecal water loss osmotically and acidify colonic contents. Acidification will trap ammonia as ammonium in the colon and will alter colonic bacterial flora. Alteration of intestinal transit time associated with the osmotic diarrhea will decrease time available for ammonia production and absorption. Lactulose may be given orally or by enema; dosages should be regulated so that feces is soft but formed. Cystitis should be treated with appropriate antibiotics based on urine culture and sensitivity; response may be poor if uroliths are present. Urate uroliths may respond to low protein diets; renal calculi have reportedly dissolves after shunt ligation.

With proper medical management, weight and quality of life stabilize or improve with treatment in most animals. One third of dogs do well with medical management as the sole method of treatment, with many living to 7 years of age or older. Duration of survival with medical management alone has been correlated to age at initial signs and with BUN concentration; dogs that are older at presentation or have a higher BUN live longer. Over half of dogs treated with medical management alone are euthanized, usually within 10 months of diagnosis, because of uncontrollable neurologic signs and, in some cases, progressive hepatic fibrosis and subsequent portal hypertension. Surgery is the treatment of choice for animals with single congenital portosystemic shunts, except perhaps in older dogs with minimal chemistry changes and no clinical signs or urinary tract abnormalities on a protein restricted diet.

Anesthetic Management

Anesthetic agents that are metabolized by the liver (i.e. barbiturates and phenothiazine tranquilizers), highly protein-bound (i.e. diazepam and barbiturates) or hepatotoxic (i.e. halothane) should be used with caution because of poor hepatic function and hypoalbuminemia. Sedation followed by mask induction and maintenance of anesthesia with isoflurane is often the anesthetic regimen of choice. Opioids can be safely used as premedicants and pre-emptive analgesics since they effects are chemically reversible. In our practice we commonly sedate the dogs with ace promazine (0.25 mg total dose) and butorphanol (0.2 – 0.4 mg/kg) and then induce by mask or with propofol.

Exploratory Laparotomy

Definitive diagnosis of extrahepatic PSS can usually be made during exploratory laparotomy, if the veterinarian is familiar with the anatomy of the abdomen. Most extrahepatic PSS terminate on the caudal venal cava; therefore, a thorough understanding of the anatomy of the caudal vena cava is important in differentiating normal and abnormal tributaries.

Renal veins are approximately 8 mm in diameter in an average sized dog, with the right vein located farther cranial than the left. The phrenicoabdominal vein is less than or equal to half the diameter of the renal vein and terminates on the caudal vena cava about 1 cm cranial to the renal veins on the same side. In a normal dog, there are no large vessels entering the caudal vena cava between the renal and hepatic veins. In an animal with a portocaval shunt, the caudal vena cava will appear dilated at the level of the shunt terminus and turbulent flow will be visible within the vein and near the terminus of the large, shunting vessel, which usually has a diameter of 1 cm.

Frequently, extrahepatic PSS terminate on the caudal vena cava at the level of the epiploic foramen. The epiploic foramen is found to the right of midline, at the base of the mesoduodenum. Its boundaries are formed dorsally by the caudal vena cava, ventrally by the portal vein and hepatic artery, cranially by the caudate lobe of liver, and caudally by the celiac artery. To locate the epiploic foramen, gently retract the duodenum ventrally and to the left, exposing the right kidney and caudal vena cava. Extrahepatic PSS may not be immediately obvious; it may be necessary to gently retract the celiac artery caudally to see the PSS terminating on the caudal...
Portocaval shunts entering near the epiploic foramen may be difficult to see if the terminus of the PSS is obscured by an overlying artery, liver lobe, or the pancreas. Portoozygous shunts often traverse the diaphragm at the level of the crura or aortic hiatus, and are obscured by overlying vescera. To improve detection of and access to these shunts, it may be necessary to open the omental bursa by tearing a hole in the superficial, ventral leaf of the greater omentum and retracting the stomach cranially and intestines caudally. Any vein of significant size that visibly penetrates the diaphragm at its lumbar attachments is likely to be a portoozygous shunt. Portoozygous shunts that traverse the diaphragm through the esophageal hiatus may be easier to approach outside of the omental bursa by retracting the liver and stomach to the right so that the cardia and esophagus are visible.

Occasionally a shunt that has not been identified by exploration of the cranial abdomen can be located by thorough examination of the caudal vena cava in the caudal abdomen. Thorough exploration is warranted in all dogs with single congenital PSS because of the possibility, though rare, of a second shunt.

Intrahepatic PSS are more difficult to detect. Experienced surgeons will note enlargement of the portal vein branch to, or hepatic vein draining, the lobe containing the shunt. Not readily visible during surgery, intrahepatic PSS may be located by palpation, ultrasound, catheterization via the portal vein, or measurement of portal pressure changes during digital vascular occlusion. To catheterize the shunt, insert a long jugular catheter through the spleen and into the portal vein, and thread it through the shunt and into the caudal vena cava. Alternatively, place a purse string suture in the portal vein; make an incision into the vein within the purse-string suture, and thread a red rubber catheter into the portal vein and through the shunt. In most dogs it will be necessary to incise the diaphragm to feel the catheter in the caudal vena cava. Once the catheter is in place, the various portal vein branches, liver lobes, and hepatic veins are palpated to locate the origin and insertion of the shunt.

When a shunt is not found, the surgeon should obtain a liver biopsy to rule out other hepatic diseases such as hepatic microvascular dysplasia and perform intraoperative mesenteric or splenic portography to definitively rule out a PSS. If the dog has gastrointestinal disease and increased bile acids, intestinal biopsies should also be taken, since inflammatory bowel disease can be associated with hepatic veno-occlusive disease.

Porsostystemic Shunt Occlusion

Once the PSS is identified and presence of a prehepatic portal vein is verified, shunt occlusion can be attempted. It is critical to ligate the shunt as close to its insertion site as possible so that all tributaries of the shunt are upstream from the occlusion. Portocaval shunts should be occluded at their terminus on the caudal vena cava. Portozygous shunts can be occluded at the abdominal side of the diaphragm. Thorough examination is warranted before ligature placement as portoozygous shunts frequently have small branches from gastric veins that enter the PSS just before it traverses the diaphragm. The diaphragm may be opened if more exposure is needed.

Shunts can be occluded with suture or constricting devices. If suture is to be used to ligate the shunt, then a small opening is made through the fascia around the shunt by dissecting adjacent to the PSS at its terminus. Silk suture (2-0) is frequently used in dogs because of ease of handling and knot security; however, nonabsorbable monofilament is recommended in cats. The shunt should be temporarily occluded for 5-10 minutes while the surgeon evaluates the vescera for evidence of portal hypertension, including pallor or cyanosis of the intestines, increased intestinal peristalsis, cyanosis or edema of the pancreas, and increased mesenteric vascular pulsations. Additionally, the surgeon can measure portal and central venous pressures. To measure portal pressure, a catheter is placed directly into a jejunal vein or through the splenic parenchyma and into a splenic vein. The catheter is secured in place with gut suture and is attached to an extension set, 3-way stopcock, and syringe. A water manometer is attached to the 3-way stopcock, which is rested on the inguinal region of the patient to provide consistent readings during portal pressure measurements.

Recommendations for postligation pressures are to limit the maximum portal pressure to 17 to 24 cm H2O, maximal change in portal pressure to 9-10 cm H2O, and maximal decrease in central venous pressure to 1 cm H2O. Partial ligation should be performed if evidence of portal hypertension is noticed during surgery. Objective pressure measurements should not be used as the sole criteria for degree of shunt attenuation, since blood pressures can vary with depth of anesthesia, hydration status, phase of respiration, degree of splanchnic compliance, and other systemic factors. To perform partial ligation, choose a cylinder (a piece of tubing, steel pin, or rod) that is the approximate diameter that you wish to achieve during shunt occlusion. Place the cylinder next to the shunt and wrap the ligature around the shunt and the cylinder. Tie the ligature and remove the cylinder, then recheck portal pressures and evaluate the color of the vescera.
Abrupt occlusion and partial ligation of PSS have been associated with serious postoperative complications, including perioperative death in 14 to 21%, seizures in 7.5-11%, recurrence of clinical signs in 40-41%, and development of multiple PSS in 7%. Therefore, many surgeons now prefer gradual, complete shunt ligation with devices such as ameroid constrictors (Research Instruments N.W., Inc., Lebanon OR, 97355; kpm@proaxis.com). An ameroid constrictor is an inner ring of casein that is surrounded by a stainless steel sheath. Casein is a hygroscopic substance that swells as it slowly absorbs body fluid. The stainless steel sheath forces the casein to swell inwardly, eventually closing the ring and obliterating the shunt. Ameroid constrictors cause gradual shunt occlusion over 2-5 weeks by direct pressure and by stimulation of a fibrous tissue reaction. Ameroid constrictors are gas sterilized and therefore should not be used until 12 to 24 hours after sterilization to allow residual ethylene oxide to be released from the casein.

Ameroid constrictors come in various sizes, with internal diameters ranging from 3.5 to 9 mm; constrictors with a 5 mm internal diameter are most frequently used for PSS ligation. The choice of ameroid constrictor size for PSS occlusion is based on shunt diameter; therefore, the surgeon should have a selection of sizes available at each surgery. To avoid postoperative portal hypertension, choose a constrictor that does not compress the shunt after placement. In cases where larger constrictors are not available, portal pressures can be measured during partial shunt occlusion and viscera can be evaluated subjectively for signs of portal hypertension to determine whether a smaller constrictor could be used.

Before constrictor placement, the “key”, a small column of casein that completes the constrictor ring, is removed from the ameroid constrictor and set aside in a dry cup. The ameroid constrictor is held securely by a pair of Allis tissue forceps, which prevent rotation of the casein inside of the stainless steel ring. Dissection of the supporting fascia around the PSS should be kept to a minimum when placing an ameroid constrictor to prevent postoperative movement of the ring and acute obstruction of the shunt. Once an opening has been made through the fascia around the PSS, the shunt is flattened by elevating it with open right angle forceps or two silk sutures; the ring is slipped over the shunt and, with a hemostat, the key is replaced within the constrictor to complete the circle. If the key is difficult to place, then a small amount of casein can be shaved off of one of its ends. If the key is lost or unusable, the inner casein ring can be rotated so that its opening faces in the opposite direction from that of the stainless steel ring.

Gas sterilized strips of cellophane have been used to provide partial occlusion of shunts in dogs. Because the strips are flexible, they are easier to place around intrahepatic shunts. The strips are wrapped once around the shunt, compressing it to 2-4 mm in diameter, and are held together with surgical clips. Inflammation caused by the cellophane results in complete occlusion of most shunts in dogs in less than 4-6 weeks.

Blood flow through intrahepatic PSS may be reduced by occluding the portal vein branches leading to, or hepatic veins draining the shunt using the above described extravascular techniques. Alternatively, the shunt can be approached intravascularly during inflow occlusion. Most surgeons prefer extravascular techniques when possible to reduce the risk of complications. Recently thrombogenic coils have been placed via catheters through the jugular vein to gradually obstruct the shunts. Because most animals will not tolerate abrupt PSS occlusion, vessel thrombosis must be regulated with the use of anticoagulants or through delayed placement of additional coils.

Postoperative care

After surgery, animals are monitored closely for seizures, hypothermia, hypoglycemia, and signs of portal hypertension, including shock, pain, and abdominal distension. Most animals will need analgesics; opioids are used most frequently. Sedation with a low dose (0.25 mg total dose IM or IV) of acepromazine may be necessary if dogs are vocalizing or abdominal pressing, since these activities will increase portal pressure. Dogs with ameroid constrictor occlusion usually experience minimal discomfort.

A protein restricted diet and lactulose are continued after surgery until liver function improves. Frequently the animals can be gradually weaned off of the lactulose 4 to 6 weeks after the surgery. Bile acids and albumin are evaluated 2, 4, 6, and 12 months after the surgery to evaluate liver function. Protein in the diet can be gradually increased once bile acids are normal. In dogs with mildly elevated bile acids and normal albumin, it may be necessary to monitor clinical response to diet change to determine whether protein content can be gradually increased.

Treatment of postoperative portal hypertension includes intravenous fluid administration for hypovolemic shock, systemic antibiotics, and immediate surgery to remove the constrictor or ligature. Factors which may increase portal pressure postoperatively include excessive intraoperative fluid administration, increased systemic blood pressure from anesthetic recovery,
and increased intra-abdominal pressure from bandages, pain, or vocalization.

Between 5 and 10% of small breed dogs develop seizures after shunt ligation. Etiology is unknown, and affected animals usually do not respond to fluids, dextrose, or enemas. Seizures are treated with a bolus of diazepam and blood glucose is measured and corrected if low. If the animal continues to seize, the diazepam is repeated and the animal is then placed on a continuous intravenous infusion of propofol (.025 to 1.0 mg/kg/minute) or another barbiturate over 12-24 hours and then gradually weaned off. Cats should also be treated with oral lactulose and neomycin when they are conscious. Prognosis is poor for animals with postoperative seizures, and many that survive continue to have neurologic problems.

Prognosis

Prognosis for successful surgical treatment is best for dogs with extrahepatic shunts; for animals that undergo complete shunt ligation or occlusion with ameroid constrictors; and for those that present with urinary tract signs and no hepatic encephalopathy. Complication rates are much lower and the surgery is much faster when ameroid constrictors are used. In our practice, 85% of dogs treated with ameroid constrictors are clinically normal within 3 months after the surgery. Mortality rates after surgery are high in animals with very low albumin (1.0 g/dL) or any other indication of severe liver disease such as ascites.

Half of dogs that undergo partial shunt ligation with suture develop clinical signs of portosystemic shunting within 2 years after ligation. Some of these animals will respond to further ligation, while others have developed multiple acquired shunts or cannot tolerate further occlusion. Clinical signs in these patients are controlled with lactulose and a protein restricted diet.

Cats commonly develop neurologic signs after surgery and may require frequent doses of lactulose and neomycin (every 6 hours) or treatment with phenobarbital or potassium bromide. Long-term, only one third of cats do well after shunt ligation.

Heredity

Development of shunts is caused by hereditary factors in many breeds. Yorkshire terriers are the breed most commonly affected with single congenital shunts. In fact, the risk of a Yorkshire terrier having a congenital shunt is 59 times greater than mixed breed dogs, and 36 times greater than all other breeds combined. The method by which the genes are transmitted, however, is still unknown. Based on current pedigree and breeding studies, we know that the trait is not sex-linked, simple dominant, or simple recessive. We also know that it can skip generations. Breeding dogs with hepatic microvascular dysplasia (HMD or MVD- a disease of the microscopic liver blood vessels) can result in offspring with single congenital shunts. Additionally, breeding lines of dogs commonly affected with shunts to lines without shunts reduces the incidence of shunt formation in offspring. It is possible that the trait is polygenic (caused by multiple genes), or one of incomplete penetrance or variable expressivity (not all dogs with the gene have a shunt, or the dogs may have various degrees of liver disease).