Minimally invasive treatment of mesenteric arterioporal fistulas in two dogs

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CASE DESCRIPTION
Two Pembroke Welsh Corgis with gastrointestinal signs including inappetence, diarrhea, lethargy, and hypersalivation were referred for evaluation.

CLINICAL FINDINGS
Diagnostic testing included abdominal ultrasonography and CT angiography. One patient had a cranial mesenteric artery-to-mesenteric vein fistula with multiple acquired extrahepatic portosystemic shunts. The second patient had both cranial and caudal mesenteric artery-to-mesenteric vein fistulas and multiple acquired extrahepatic portosystemic shunts.

TREATMENT AND OUTCOME
Both patients underwent minimally invasive coil embolization of the mesenteric arterioporal fistulas, with complete occlusion confirmed by means of angiography at procedure completion. Clinical outcome approximately 1 year after treatment was assessed as fair to good because of recurrence of clinical signs that required medical management in 1 dog and some persistent serum biochemical abnormalities.

CLINICAL RELEVANCE
Outcome for the 2 patients described suggested that coil embolization may be a feasible and effective minimally invasive technique for the treatment of mesenteric arterioporal fistulas in dogs. However, further investigation of the potential for chronic hepatic disease in patients with a history of acquired portosystemic shunts is warranted. (J Am Vet Med Assoc 2017;251:1306–1312)

A 2-year-old 13.7-kg (30.2-lb) spayed female Pembroke Welsh Corgi (dog 1) was referred for evaluation and possible minimally invasive treatment of a hepatic vascular anomaly. The dog had been examined by the referring veterinarian because of inappetence, diarrhea, and lethargy of unknown duration. Results of a physical examination by the referring veterinarian were unremarkable, and results of a CBC and serum biochemical analysis were largely within reference limits (Supplementary Table S1, available at avmajournals.avma.org/doi/supppl/10.2460/javma.251.11.1306). Urinalysis revealed ammonium biurate crystals (21 to 50 crystals/hpf), bilirubinuria (1+), pyuria (4 to 10 WBCs/hpf; reference range, 0 to 3 WBCs/hpf), and proteinuria (2+). The dog was initially treated for 10 days with lactulose (48 mg/kg [22 mg/lb], PO, q 24 h), marbofloxacin (3.6 mg/kg [1.6 mg/lb], PO, q 24 h), and metronidazole (9 mg/kg [4 mg/lb], PO, q 12 h) on the basis of a presumptive diagnosis of an extrahepatic shunt. An abdominal ultrasonographic examination revealed a small, mildly hypechoic liver with a hypovascular pattern. Doppler interrogation showed a 1.9-cm-diameter vascular anomaly medial to the right kidney. A small amount of material (eg, mineralized sediment or multiple small calculi) was present in the urinary bladder. Preprandial (178 µmol/L; reference range, 0 to 13 µmol/L) and postprandial (299 µmol/L; reference range, 0 to 30 µmol/L) serum bile acids concentrations were both high, and plasma protein C activity was slightly low (72%; reference range, 75% to 135%), suggestive of portosystemic shunting. An exploratory laparotomy performed 1 month later revealed multiple extrahepatic portosystemic shunts and confirmed the presence of the vascular anomaly identified on ultrasonographic examination; no treatment was performed at that time. Liver specimens obtained during the exploratory laparotomy were submitted for histologic examination. Results indicated an embryonic appearance of the portal vasculature, the presence of endothelial cells with fusiform and hyperchromatic nuclei, and arterialization of central veins consistent with hepatic microvascular dysplasia (ie, portal vein hypoplasia).

The patient was referred to the Animal Medical Center 2 months later for further diagnostic evaluation and possible minimally invasive (ie, interventional) treatment of the hepatic vascular anomaly. The dog remained inappetent and lethargic despite medical treatment. At the time of referral, the patient was receiving lactulose (66 mg/kg [30 mg/lb], PO, q 12 h) and consuming a low-protein diet. Multiple-phase contrast CT angiography was performed, which included precontrast, arterial, venous, and delayed venous phases. Results revealed a cranial mesenteric artery-to-mesenteric vein fistula with multiple acquired extrahepatic portosystemic shunts. Because the patient’s condition was stable, medical management was continued to facilitate consultation with a
physician interventional radiologist for the purposes of optimal treatment planning.

Two months after CT angiography, the patient returned to our hospital and treatment of the vascular anomaly by means of minimally invasive embolization was pursued. Results of a preoperative physical examination were unremarkable except for a mild fluid wave noted on abdominal palpation, and an unexplained discrepancy in values for indirect systolic arterial pressure (measured by means of Doppler oscillometry) between forelimb (220 mm Hg) and hind limb (85 mm Hg). Repeated abdominal ultrasonography indicated pulsatile flow in the portal vein cranial to the arterioporal fistula and hepatopetal flow. Several small tortuous vessels were also evident adjacent to the left renal vein. The right kidney contained a moderate amount of mineralized material and focal cysts in the cranial pole. A small amount of anechoic peritoneal fluid was also evident, with several hyperechoic foci noted along the dependent portion of the bladder. Results of a CBC were within reference limits; results of a serum biochemical analysis and plasma protein C assay were suggestive of progressive liver disease (Supplementary Table S1).

The dog was premedicated with a combination of oxymorphone (0.075 mg/kg [0.034 mg/lb], IM) and dexmedetomidine (10 µg/kg [4.55 µg/lb], IM). An IV catheter was placed, and general anesthesia was induced 15 to 20 minutes later with propofol (3 mg/kg [1.36 mg/lb]), IV. After endotracheal intubation, anesthesia was maintained with delivery of isoflurane in oxygen. The patient also received maropitant (1 mg/kg [0.45 mg/lb], IV) at the time of premedication, cefoxitin (30 mg/kg [13.6 mg/lb], IV) at the time of anesthetic induction, and a balanced electrolyte solution (10 mL/kg/h [4.5 mL/lb/h], IV) throughout the procedure. Heart rate, respiratory rate, indirect blood pressure (measured by means of Doppler oscillometry), arterial oxygen saturation (measured by means of pulse oximetry), end-tidal carbon dioxide concentration (measured by means of sidestream capnography), and body temperature were monitored continuously throughout the anesthetic period. Data were recorded every 5 minutes.

After standard surgical preparation and draping, the left femoral artery was accessed surgically, followed by placement of a 4F introducer sheath by means of a single-wall-puncture technique. Access was chosen on the basis of the direction and angle of the celiac axis in relation to the aorta. A 4F angled diagnostic angiography catheter was advanced into the cranial mesenteric artery with fluoroscopic guidance, and angiography was performed to delineate the vascular anatomy (Figure 1). The cranial mesenteric artery-to-portal vein fistula was identified, and a microcatheter-microwire combination was used to place 6 platinum embolization coils in the fistula. Angiography was then repeated to confirm that the fistula was no longer patent (Figure 2). The patient recovered...
from anesthesia without apparent complications, and a postoperative abdominal ultrasonographic examination including Doppler interrogation of the area of interest confirmed cessation of blood flow through the arterioporal fistula. Hepatopetal blood flow in the portal vein and its tributaries was present. The patient was discharged the following day and the owner was instructed to continue medical management, which consisted of lactulose (66 mg/kg, PO, q 12 h), metronidazole (9 mg/kg, PO, q 12 h), famotidine (0.7 mg/kg [0.32 mg/lb], PO, q 24 h), tramadol (3.6 mg/kg, PO, q 8 to 12 h), cephalixin (18.2 mg/kg [8.3 mg/lb], PO, q 12 h), and a low-protein diet.8

The dog returned to the hospital approximately 1 month later, at which time the owner reported clinical improvement with increased energy and improved appetite. Abdominal radiography revealed no evidence of migration of the embolization coils. Abdominal ultrasonography showed subjective evidence of improvement in portal vein blood flow, compared with preoperative images (eg, decreased size of portal vascular structures and lack of signs of pulsatile flow [flow velocities not reported]); the coils remained in place without signs of arterialization of the portal vasculature. A CBC and serum biochemical analysis suggested improvement of liver function. Preprandial serum bile acids concentration and plasma protein C activity were improved, compared with preoperative values, although the former was still higher than the upper reference limit (Supplementary Table S1). At the completion of this follow-up visit, the owner was instructed to gradually wean the dog off all medications.

When the dog was examined 3 months after the procedure, the owner continued to report that it appeared to have improved energy but was periodically inappetent. Nonetheless, the owner considered the dog to be improving and had gradually weaned the dog off all medications except famotidine and was no longer feeding the prescribed low-protein diet. A repeated CBC and serum biochemical analysis showed increased activity of liver enzymes, hypocholesterolemia, and decreased plasma protein C activity (Supplementary Table S1). Subsequent 4- and 7-month follow-up visits documented further decreased protein C activity, decreased alanine aminotransferase activity, and resolution of hypocholesterolemia; BUN concentration remained within the reference range. Because of this, the dog was restarted on medical management 4 months after interventional treatment, consisting of metronidazole (9 mg/kg, PO, q 12 h), lactulose (46 mg/kg [21 mg/lb], PO, q 24 h), famotidine (0.7 mg/kg, PO, q 24 h), and the same low-protein diet.

At a 1-year postoperative visit, the dog was reported to be experiencing intermittent clinical signs. Appetite was occasionally decreased, but the owner stated that the dog otherwise had good energy levels and quality of life. Overall, the technical procedural outcome was deemed good on the basis of angiographic evidence of complete closure of the arterioporal fistula at the time of the procedure. Clinical outcome was classified as fair because of the recurrence of clinical signs requiring resumption of medical management, but with serum biochemical values mostly within reference ranges.

A 1-year old 11.4-kg (25 lb) castrated male Pembroke Welsh Corgi (dog 2) with a 2-month history of vomiting, diarrhea, nausea, hypersalivation, and hematuria was referred for evaluation. When the patient was examined by the primary care veterinarian 4.5 months prior to referral, results of physical examination were unremarkable. A CBC, serum biochemical analysis, and urinalysis revealed high liver enzyme activities and ammonium biurate (≥ 3 crystals/hpf) and struvite (≥ 3 crystals/hpf) crystalluria (Supplementary Table S2, available at avmajournals.avma.org/doi/suppl/10.2460/javma.251.11.1306). Abdominal ultrasonography indicated the presence of a 3.0 × 1.7-cm cystic structure cranial to the urinary bladder, immediately caudal to the kidneys. The dilated structure was further assessed with Doppler ultrasonography, which showed arterial blood flow that led to a dilated vein. A cluster of small tortuous vessels was also identified dorsocaudal to the liver that was interpreted as multiple acquired portosystemic shunts. The patient also had a bladder urolith and echogenic material floating within the bladder. Subsequent auscultation of the abdomen revealed an audible bruit, presumably a result of aberrant blood flow. Serum ammonia concentration at that time was within the reference range (0 to 98 μmol/L); however, pre- and postprandial bile acids concentrations and results of a plasma protein C assay were indicative of liver disease, portosystemic shunting, or both. The patient was treated for hepatic encephalopathy with lactulose (114 mg/kg [52 mg/lb], PO, q 12 h), famotidine (0.4 mg/kg [0.18 mg/lb], PO, q 24 h), amoxicillin (17.5 mg/kg [8 mg/lb], PO, q 12 h), and a low-protein diet.4

One month following initial evaluation by the primary care veterinarian, the patient was referred to a different tertiary care hospital, because the specific anatomy of the anomalous vasculature could not be fully defined. At that time, CT angiography revealed a large mass of tortuous vessels in the center of the abdomen and dilation of the associated portal and mesenteric vasculature (Figure 3). The presence of 2 arterioporal fistulas was confirmed; the first originated from the cranial mesenteric artery and formed a large ampulla leading to a direct communication with the associated cranial mesenteric vein. The second consisted of the caudal mesenteric artery forming a large dilation ventral to the sacrum and connecting to the tortuous and dilated caudal mesenteric vein adjacent to the colon. Complex esophageal, mediastinal, diaphragmatic, and testicular acquired portosystemic shunts that likely developed secondary to the arterialized portal vein and subsequent hypertension were also documented. The liver was subjectively small, and there was a mild to moderate volume of peri-
Several weeks later, a CBC, coagulation profile, and routine laboratory testing were performed. The results were similar to those reported previously.

Although the dog had initially appeared inappetent, the appetite and energy level reportedly had improved. The patient continued to respond to medical management. A CBC, serum biochemical analysis, and coagulation panel were repeated; low mean corpuscular volume, high liver enzyme activities, and low albumin and cholesterol concentrations were noted.

The patient was prepared for the procedure as described for dog 1. An initial dose of levetiracetam (60 mg/kg [27.3 mg/lb], IV, once) was also administered preoperatively in an effort to protect against seizure activity, and administration was continued (30 mg/kg, IV, q 8 h) for the duration of hospitalization. A surgical approach was made to the right carotid artery. The artery was ligated cranially and cannulated with a 6F vascular access sheath. With a 4F angled diagnostic angiography catheter, the cranial mesenteric artery-to-cranial mesenteric vein fistula was evaluated by means of angiography in multiple planes (Figure 4). A single orifice was identified, and the feeding blood vessel was selected with a 3F braided peripheral vascular microcatheter. The shunting vessel was occluded with multiple embolization coils. The second arterioportal fistula (caudal mesenteric artery-to-caudal mesenteric vein) was similarly evaluated angiographically (Supplementary Figure S1, available at avmajournals.avma.org/doi/suppl/10.2460/javma.251.11.1306). Again, multiple platinum embolization coils were deployed to occlude the feeder blood vessel and achieve stasis. A final angiogram was then obtained, confirming qualitatively normal hepatopetal and arterial intestinal blood flow. The vascular access sheath was removed, and the carotid artery was ligated caudal to the access site. The patient recovered uneventfully from general anesthesia and was monitored overnight without apparent complications. It was discharged the following day with the owner instructed to administer lactulose (200 mg/kg [91 mg/lb], PO, q 8 h), metronidazole (10 mg/kg, PO, q 12 h), and omeprazole (0.9 mg/kg [0.4 mg/lb], PO, q 12 h) until further directed at a follow-up examination in 1 month. A 2-week course of treatment with amoxicillin-clavulanate (16.4 mg/kg [7.5 mg/lb], PO, q 12 h) was also prescribed. Postoperative repeated laboratory testing performed prior to discharge (1 day after surgery) revealed decreased activity of liver enzymes and a marked decrease in plasma protein C activity, compared with the preoperative value (Supplementary Table S2).

Two weeks after the procedure, the owners reported that the dog’s energy level and appetite had increased. Clinical signs associated with hepatic encephalopathy were reportedly infrequent. However, 4 weeks postoperatively, behavioral changes recurred despite the dog being fed a plant-based diet and receiving treatment with lactulose (233 mg/kg [106 mg/lb], PO, q 8 h), famotidine (1.7 mg/kg [0.8 mg/lb], PO, q 24 h), and amoxicillin (26.3 mg/kg [12 mg/lb], PO, q 12 h) prescribed by the primary care veterinarian. Results of repeated laboratory testing
included increased liver enzyme activities and plasma ammonia concentration. Analysis of a free-catch urine specimen showed moderate struvite (40 to 46 crystals/hpf) and ammonium biurate (0 to 2 crystals/hpf) crystalluria. Plasma protein C activity was also decreased (Supplementary Table S2).

At a 3-month follow-up visit, the dog was reported to be doing very well at home, with increased energy. The previously noted encephalopathic behavior changes had resolved, and plasma ammonia concentration was within the reference range. Two months later (5 months after the procedure), the dog continued to do well, and repeated laboratory testing found decreased BUN and serum albumin concentrations and decreased plasma protein C activity. Pre- and postprandial bile acids concentrations were 21 and 295 µmol/L, respectively (Supplementary Table S2). The preprandial concentration was thought to be erroneous, but repeated testing confirmed the result. Two additional months later (7 months after treatment), repeated serum biochemical analysis showed further decreased serum albumin and BUN concentrations. Results of analysis of a free-catch specimen urine were within reference limits. At the 9- and 11-month postoperative follow-up examinations, medical management was unchanged, and the dog continued to lack clinical signs. Results of a free-catch urine specimen at the 11-month visit were within reference limits with the exception of bilirubinuria (3+). Results of a CBC were within reference ranges, and results of a serum biochemical analysis were largely unchanged from prior results. Technical outcome for this patient was classified as good on the basis of complete attenuation of the arterioportal fistulas as assessed with angiography at the completion of the procedure. Clinical outcome was classified as good on the basis of lack of recurrence of clinical signs during an almost 12-month follow-up period, but with the continued need for medical treatment and the presence of some abnormalities on serum biochemical analysis.

**Discussion**

Cardiovascular anomalies in dogs can be either congenital or acquired. Congenital defects are present at birth and can be the result of genetic, environmental, infectious, toxicologic, pharmaceutical, and nutritional factors. Peripheral vascular anomalies including portosystemic shunts, arteriovenous fistulas, and arteriovenous malformations can also be congenital or acquired.\(^1,2\) Portosystemic vascular anomalies are abnormal vessels that allow portal blood draining the abdominal viscera to pass directly into the systemic vasculature, bypassing the liver. These communications can be broadly categorized as intrahepatic or extrahepatic. Extrahepatic shunts can be congenital or acquired. Congenital extrahepatic shunts are single abnormal communications occurring more frequently in smaller breeds of dogs.\(^3\) These have been reported\(^3\) to account for approximately 63% of all portosystemic vascular anomalies in dogs, and their morphology can be variable. Conversely, intrahepatic shunts are usually congenital, single shunts that comprise approximately 35% of all portosystemic vascular anomalies in dogs.\(^3\) Arteriovenous malformations (including mesenteric arterioportal fistulas) reportedly contribute to the remaining 2% of portosystemic vascular anomalies in dogs and can occur congenitally or secondary to trauma, neoplasia, surgical procedures, or degenerative processes.\(^3\) These anomalies are usually macroscopic and, when congenital, have been suggested to occur as a result of failure of the embryologic capillary plexus to differentiate into an artery or vein.\(^5\) In dogs, arteriovenous malformations are typically congenital and rarely involve the extremities, head, or pelvis.\(^6\)

Arteriovenous malformations are uncommon in human patients, reportedly affecting approximately 2% of portosystemic vascular anomalies in dogs.
1 in 100,000 individuals. Although they can occur throughout the body, the 2 most commonly affected sites are the brain and spinal cord, with gastrointestinal sites being rare. The etiology of arteriovenous malformations is largely unknown, but they are considered to develop in utero, and symptoms may manifest at any age. Symptoms are variable and most (88%) human patients may not notice symptoms until they are severe. Mesenteric arterioportal fistulas are aberrant arteriovenous communications between the high-pressure mesenteric arterial tree and low-pressure portal vein tributaries. Although these malformations are rare, it has been reported that the incidence of mesenteric arterioportal fistulas in human patients is increasing. Presumably this increase is at least in part a result of increased availability of advanced diagnostic imaging modalities and improved survival rates for patients with abdominal trauma. The most common causes of arterioportal fistulas in such patients in 1 study were trauma (28%), iatrogenic injuries (eg, during liver biopsy; 16%), congenital origin (15%), malignant neoplasia (15%), and rupture of a splanchic arterial aneurysm. These arterioportal fistulas have been reported to originate from the hepatic (45%), splenic (30%), superior mesenteric, gastroduodenal, and inferior mesenteric arteries in human patients. They can be completely asymptomatic or manifest with various clinical features including abdominal bruit, portal hypertension, gastrointestinal hemorrhage, malabsorption, chronic mesenteric ischemia, and signs of liver disease and heart failure.

Treatment of mesenteric arterioportal fistulas consists largely of minimally invasive transcatheter embolization or open surgery for more complex cases. It has been reported that human patients with acquired fistulas have a more favorable prognosis, versus those with congenital arterioportal fistulas, because of comparatively minimal physiologic derangements and the absence of concurrent congenital abnormalities when treating patients with acquired lesions. Traditionally, the treatment of choice for patients with arterioportal fistulas has been complete surgical resection. However, consideration must be given to number, size, location, anatomic features, and vascular flow rate when aiming for complete resection. Recently, the approach to treatment in human patients with arteriovenous malformations has shifted away from surgical ligation or resection in favor of a less invasive endovascular approach. In a 2010 review by Krishan et al, 21 patients with arterioportal fistulas were identified, all of whom were treated successfully via an endovascular approach, with only 1 possible procedure-related complication (death as a result of myocardial infarction). This contrasted with the outcome for patients treated by means of an open surgical technique for which morbidity (50%) and mortality (50%) rates were notably higher. The authors defined morbidity as the development of postoperative surgical complications including portal vein thrombosis, biliary fistula, biliary stricture, or stroke. Endovascular treatments for patients with arterioportal fistulas largely consist of the use of embolization coils, vascular plugs, glue embolics, sclerosing agents, or stent grafts. The general aim of such procedures is to target feeder arterial vessels of the fistula. Occlusion of the fistulous tract, pseudoaneurysm, or both has been performed in human patients but is sometimes avoided because of the fragile nature of the involved tissues and the risk of hemorrhage. Occlusion of a typical arteriovenous fistula is generally well tolerated and typically curative in human patients. The major complications associated with coil embolization are inappropriate embolization of vital arterial branches (resulting in ischemic necrosis of adjacent viscera, skin, and other structures) and migration of embolics to distal sites such as the pulmonary vasculature. This can be exacerbated by difficulty in identifying the feeder vessel in patients with chronic lesions and resultant vascular hypertrophy. In contrast, complete surgical resection is considered more likely to achieve curative intent in appropriately selected cases, but has been reported to have a considerably higher risk of hemorrhage in human patients.

In the present report, we described the treatment of mesenteric arterioportal fistulas in 2 Pembroke Welsh Corgis. Both arterioportal fistulas consisted of 1 or more mesenteric artery-to-mesenteric vein fistulas with multiple acquired extrahepatic portosystemic shunts. Despite successful technical outcomes with angiographic confirmation of closure of the mesenteric arterioportal fistulas in both patients described, results of serial laboratory testing remained abnormal and clinical signs recurred in dog 1. Notably, owners of both dogs of the present report were satisfied with the outcomes reported. We suggest that hepatic dysfunction from a persistent lack of portal perfusion because of the extrahepatic portosystemic shunts caused recurrence of clinical signs in the patient described. High-pressure flow of arterial blood into the low-pressure portal vein causes subsequent portal vein hypertension and development of multiple extrahepatic shunts, diverting portal blood from the liver. Any vessel that shunts blood away from the portal vasculature, diverting it directly into the systemic circulation, will not only decrease overall hepatic vascular flow, but also act as a conduit allowing blood to bypass the normal filtration and conjugation systems of the liver. As such, failure of the liver to filter substances such as ammonia, mercaptans, and short chain fatty acids likely contributed to the clinical signs associated with hepatic encephalopathy observed in dog 2 of the present report.

In human patients with arterioportal fistulas, it has been reported that chronic arterialization of the portal vein with hyperkinetic hepatic blood flow results in liver disease, and a similar process may have contributed to the persistent signs of liver dysfunction identified in the 2 patients of the
present report. In an experimental study of dogs, Zuidema et al 13 reported that large arteriportal fistulas produced focal hepatic fibrosis (periportal zones), dilation of the sinusoids, periportal necrotizing vasculitiis, increased hemosiderin deposits, and decreased activity of microsomal enzymes. Therefore, we suggest that liver plasticity in response to an increase in portal blood flow may be limited, such that multiple acquired extrahepatic portosystemic shunts may develop or worsen in severity after embolization. Alternatively, after treatment of an arteriportal fistula by means of coil embolization, total portal vascular pressures and overall hepatic perfusion would be decreased. It is difficult to predict the effect of such changes on liver function; however, it is likely that chronic portal hypertension would be increasingly detrimental over time. 11, 12

If persistent hepatic dysfunction is suspected because of multiple extrahepatic shunts, caval banding can be considered. 14 Suture banding of the caudal vena cava is performed during simultaneous measurement of pressures in the portal vein and caudal vena cava. The suture is tightened until caudal vena cava pressure is increased and slightly exceeds portal pressure. The goal is to achieve reversal of blood flow through the multiple aberrant shunting vessels. However, a study performed by Butler-Howe et al 15 involving experimentally induced portosystemic shunts in dogs demonstrated that although the procedure achieved and maintained target caudal vena cava pressures for 7 days, the calculated hepatic plasma flow was not necessarily persistently improved. As such, it is possible that vena caval banding may not result in long-term improvement.

We are not aware of prior reports of minimally invasive treatment and outcome for mesenteric arteriportal fistulas in veterinary patients. This apparently rare condition occurred in 2 unrelated Pembroke Welsh Corgis. Consequently, similar lesions should be considered when evaluating dogs of this breed with clinical and laboratory signs of liver dysfunction. Interventional management with coil embolization in the 2 patients of this report was technically feasible. However, additional studies are warranted to further evaluate hepatic function in dogs with similar arteriovenous anomalies and to determine the appropriate indications and types of ancillary treatments to improve the outcome for patients with multiple extrahepatic portosystemic shunts.

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Footnotes


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