CANINE PORTOSTYSEMIC SHUNTS: PART 2

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MEDICAL OR SURGICAL TREATMENT OF CONGENITAL PORTOSTYSEMIC SHUNTS?
Surgical attenuation of congenital portosystemic shunts (cPSS) is the recommended treatment for most affected dogs in order to establish normal hepatic portal blood flow and restore liver volume and function.1–4 Multiple acquired portosystemic shunts secondary to chronic portal hypertension are treated medically and are outside the scope of this article.5 Surgically treated dogs have been demonstrated to experience improved survival and long-term quality of life compared with those managed medically.6,7 Unlike medical management alone, surgical correction permits re-direction of portal blood flow toward the liver and restoration of liver volume and function.6 Medical management alone is aimed at treating and mitigating clinical signs associated with hepatic encephalopathy; however, portal blood continues to bypass the liver parenchyma leading to progressive hepatic insufficiency/atrophy.1 In one older study,6 just over 50% of dogs managed medically for cPSS were euthanased after a mean of approximately 10 months; however, one third of dogs survived at least three years. The author is unaware of more recent studies describing solely medical management of cPSS in dogs.

MEDICAL MANAGEMENT OF CONGENITAL PORTOSTYSEMIC SHUNTS
Medical management is recommended for dogs with cPSS when surgery is considered not possible or is declined.1 A two-week period of medical management is also recommended prior to surgical correction in order to treat hepatic encephalopathy and produce more suitable anaesthetic candidates.16 Preoperative medical management consists of administration of antibiotic therapy (typically a beta lactam, metronidazole, or neomycin), a synthetic disaccharide (eg, lactulose), and a soy or other high quality (high biologic value) protein-restricted diet.1 Care should be taken with administering severely protein-restricted diets to puppies with cPSS (resulting in hypoalbuminaemia) and supplementation of commercial protein-restricted diets with a high quality protein (eg, cottage cheese) is advised. Administration of an antiseizure medication, such as levetiracetam, may also be considered, depending on clinician preference in an attempt to prevent the development of postanesthesia neurologic signs.6,18 Antibiotic therapy aims to decrease ammonia production by urease-producing bacteria in the intestinal tract.1 Administration of a synthetic disaccharide is aimed at acidification of the colon (following its conversion to organic acids by colonic bacteria), which results in entrapment of ammonia as ammonium (resulting in decreased absorption) and decreased colonic bacterial numbers; and decreased faecal intestinal transit time as a result of lactulose’s cathartic effect, which results in decreased ammonia absorption.1 Administration of proton pump inhibitors (eg, omeprazole) or H2 (histamine-2) receptor antagonists (eg, famotidine, ranitidine) should be administered to dogs with congenital intrahepatic portosystemic shunts (cIHPSS) due to the high prevalence of gastrointestinal ulceration in these dogs.10 Life-long gastroprotectant administration is currently recommended for dogs that have undergone surgical attenuation of cIHPSS.10

SURGICAL MANAGEMENT OF CONGENITAL PORTOSTYSEMIC SHUNTS
The goal of all techniques is to achieve eventual complete occlusion of the shunting vessel and restore normal hepatic portal blood flow, hepatic volume and function.4 One of tenets of cPSS surgery is to attenuate the anomalous vessel at, or as close as possible to, its insertion into the systemic vein (caudal vena cava or azygous vein) to ensure that all tributaries entering the shunt are attenuated.1 Broadly speaking, cPSS can be attenuated intra- or extravascularly.1 Intravascular techniques include minimally invasive percutaneous coil embolisation,10–14 caval venotomy (cavotomy)30 and portal venotomy.26–30 These techniques are performed almost exclusively for management of cIHPSS, although coil embolisation and attenuation of congenital extrahepatic portosystemic shunts (cEHPSS) using the Amplatzer vascular plug have also been described.25,27,30 Transcaval and transportal venotomy require total hepatic vascular occlusion.15,16 Extravascular techniques can be classified as open (coeliotomy) or laparoscopic,22 but are almost exclusively the former. Care should be taken when entering the abdominal cavity during coeliotomy to prevent inadvertent injury to a shunt located within the falciform ligament (remnant of the umbilical vein).23 Extravascular techniques include suture ligation (usually with silk or polythene),24–30 amnioid ring constrictor placement,26–28,31–37 thin film (cellophane) banding,33,35–40 and placement of a hydraulic occluder.42 There is no strong evidence to recommend one surgical technique over another.43

SURGICAL MANAGEMENT OF EXTRAHEPATIC PORTOSTYSEMIC SHUNTS
Extrahepatic portosystemic shunts can be classified as portocaval (those entering into the vena cava) or portoazygous (those entering into the azygous vein).

• Portocaval shunts: The majority of portocaval cEHPSS insert into the pre-hepatic vena cava at the level of the
epiploic foramen and are attenuated at this location. The epiploic foramen is the opening into the omental bursa and is bounded dorsally by the pre-hepatic caudal vena cava, ventrally by the portal vein (and hepatic artery), and caudally by the coeliac artery (Figure 1). No vessel should enter the pre-hepatic vena cava at this level or between the insertion of the phrenicoabdominal vein and the porta hepatis in the dog. Shunts entering into the pre-hepatic vena cava at the epiploic foramen are identified by grasping the duodenum ventrally and to the left, or by tearing the superficial leaf of the greater omentum and opening into the omental bursa. Isolation of the shunt from surrounding perivascular fascia is performed by careful dissection using a Lahey bile duct forceps or Mixter right angled forceps. Portophrenic shunts may be categorised as portocaval as they insert, either directly or indirectly, into the vena cava. These shunts travel along the greater or lesser curvature of the stomach and ventral surface of the oesophagus, dive under the peritoneal surface of the diaphragm, and enter via the left phrenic vein into the left hepatic vein (which drains into the caudal vena cava) or caudal vena cava. Portophrenic shunts are attenuated on the abdominal surface of the diaphragm by attenuation of the shunt itself or the left phrenic vein.

- **Portoazygous shunts**: Portoazygous shunts typically involve the left (most commonly) or right gastric veins and traverse the diaphragm through the oesophageal hiatus to insert in the azygous vein within the thorax. These shunts may be identified intraabdominally by opening into the omental bursa. In a recent study, just over 90% of portoazygous shunts arose from the left gastric vein. These shunts are typically attenuated on the abdominal surface of the diaphragm or within the thoracic cavity by either by lateral thoracotomy or transdiaphragmatic incision. Or et al. described a minimally invasive transdiaphragmatic approach to permit attenuation of portoazygous shunts at their terminus in the azygous vein within the thorax due to concerns over the possibility of continued shunting through smaller unrecognised tributaries distal to an attenuation device placed on the abdominal surface of the diaphragm. Hunt et al. described two dogs with left gastric-azygous shunts in whom persistent shunting occurred as a result of an unrecognised vessel entering into the left gastric vein cranial to the site of placement of an ameroid constrictor.

**SURGICAL MANAGEMENT OF INTRAHEPATIC PORTOSYSTEMIC SHUNTS**

Surgical management of cHPSS is considered significantly more challenging than that of cEHPSS due to the location of the shunt within hepatic parenchyma. The surgical approach to cHPSS is influenced to a large extent by the morphology of the shunt. These shunts can be classified as right-, central-, or left-divisional. Shunts attenuated by extravascular techniques are approached via ventral midline coeliotomy with or without diaphragmatic incision or caudal median sternotomy.

- **Right-divisional cIHPSS**: Right-divisional cIHPSS enter into the hepatic vena cava (within hepatic parenchyma) and are most commonly managed at a pre-hepatic location by attenuation of the supplying right portal branch, by intrahepatic dissection of liver tissue, or by intravascular techniques. Ameroid ring constrictor (ARC) has been described for surgical management of both cEHPSS and cHPSS. The ARC consists of a compressed ring of casein surrounded by a stainless-steel casing. The construct is incomplete to allow protrusion of the vessel. Casein is a hygroscopic material that swells as it absorbs fluid by the process of imbibition. The outer stainless-steel casing prevents dislodgement of the vessel. Casein has a hygroscopic material that swells as it absorbs fluid by the process of imbibition. The outer stainless-steel casing forces the casein to swell toward the centre rather than outward, causing compression of the enclosed vessel. Vessel closure is achieved by extraluminal compression by casein, perivascular fibrosis and inflammation/soft tissue formation within the ARC, thrombus formation, or a combination of these mechanisms. Reported earliest vessel occlusion times.

- **Central-divisional cIHPSS**: Central-divisional cIHPSS enter either directly into the hepatic vena cava or indirectly via a central hepatic vein and are attenuated similar to right-divisional cIHPSS at a pre-hepatic location, by intrahepatic (to isolate the shunt terminus) or interlobar dissection, or by intravascular techniques (post-hepatic cavitomy or transportal venotomy).

- **Left-divisional cIHPSS**: Left-divisional cIHPSS enter into the left hepatic vein or rarely the post-hepatic caudal vena cava and are attenuated directly in a post-hepatic location just before entering the left hepatic vein or indirectly by attenuation of the left hepatic vein by intrahepatic dissection, or by (pre-hepatic) dissection of the left portal vein branch.

**INDIVIDUAL ATTENUATION TECHNIQUES**

- **Ameroid ring constrictor (ARC)**: Use of the ARC has been described for surgical management of both cEHPSS and cHPSS. The ARC consists of a compressed ring of casein surrounded by a stainless-steel casing. The construct is incomplete to allow placement over the desired anomalous vessel. A key is used to complete the ring and prevent dislodgement of the vessel. Casein is a hygroscopic material that swells as it absorbs fluid by the process of imbibition. The outer stainless-steel casing forces the casein to swell toward the centre rather than outward, causing compression of the enclosed vessel. Vessel closure is achieved by extraluminal compression by casein, perivascular fibrosis and inflammation/soft tissue formation within the ARC, thrombus formation, or a combination of these mechanisms. Reported earliest vessel occlusion times.
range from within four to five weeks,37 within 60 days,31 within three weeks61 and within eight days.32 Gradual attenuation of cEHPSS with a self-retaining polyacrylic acid-silicone device was recently described in six dogs.54 Ameroid constrictors are available in a variety of sizes classified by internal diameter and typically a size that is slightly greater than the anomalous vessel is chosen.1

- **Thin film (cellophane) banding:** True cellophane is composed of regenerated extruded cellulose. In a study by Smith et al.,33 thin film from four different sources was analysed by spectroscopy and microscopy and only one was found to be consistent with cellophane. Thin film banding of both cEHPSS12,13,18,40,41,42 and cIHPSS12,13,18,40,42 has been described. Typically an ~12mm strip is folded twice to form an ~4mm wide, three-layered strip of thin film,28,39,47,56 although a recent study recommended creation of a four-layered strip.38 The thin film is passed around the shunting vessel and secured in place using titanium clips.29,37,41,56 Experimentally, thin film has been demonstrated to induce an initial acute inflammatory response, followed by a slow foreign body reaction, and a more gradual attenuation compared with the ARC.51 There are conflicting reports in the literature concerning the degree to which the shunting vessel should be acutely attenuated intraoperatively.28,39,42,55 In a clinical study by Youmans and Hunt,36 all dogs that had shunt attenuation to <2.5mm achieved complete vessel occlusion by eight to 12 weeks, whereas dogs that had the shunt attenuated to >3mm had evidence of delayed closure and persistent shunting at >12 weeks. Attenuation of the shunting vessel to 50% of its original diameter, or 2-3mm internal diameter has been recommended, provided portal hypertension does not result.26,42 In a study by Frankel et al.,42 complete occlusion of the shunting vessel (based on normalisation of serum post-prandial bile acid concentrations) was achieved in five out of six (83%) dogs that underwent partial (< 3 mm) attenuation; however, mean post-prandial bile acid concentrations were increased at > 6 months. Conversely, all dogs that underwent complete attenuation of the shunting vessel had mean post-prandial bile acid concentrations within reference range at > 6 months. At the author’s institution, in cases where complete attenuation (with silk ligation) cannot be achieved (due to development of portal hypertension), partial attenuation, as much as is tolerated, is performed using thin film.

- **Hydraulic occluder:** There is one report of the use of hydraulic occluders for management of cIHPSS in dogs in the literature.40 Ten dogs, with left-, central- and right-divisional cIHPSS, were treated by application of the hydraulic occluder around the portal branch supplying the shunt. Injections of sterile saline into subcutaneous injection ports were performed at two weeks postoperatively and every two weeks thereafter up to eight weeks.42

- **Suture ligation:** Complete and partial ligation of both cEHPSS42,62,72,12,18 and cIHPSS12,20,34,57 has been described. Most commonly used materials include silk and polypropylene. Complete ligation is not always possible due to development of portal hypertension.29 Due to the larger size of cIHPSS compared with cEHPSS, the majority of cIHPSS can only be partially attenuated at the time of surgery. In a study by Tivers et al.,65 which included 55 dogs with cIHPSS, only 18.2% tolerated complete attenuation, while 81.8% tolerated partial attenuation. In that study, the majority of dogs that did not tolerate complete attenuation at index surgery underwent a planned second surgery. Complete attenuation was tolerated at the time of repeat surgery in over 80% of dogs.22 Conversely, complete ligation was possible in 76% of dogs with cEHPSS in a recent study.27 Complete ligation of cPSS is desired as it has been demonstrated to be associated with a better long-term clinical outcome compared with partial ligation. In an older study by Johnson et al.,44 all dogs that received complete ligation became clinically normal, with excellent quality of life, whereas only some of those dogs that underwent partial ligation became clinically normal. In a study by Hunt and Hughes,61 a significantly higher proportion of dogs that underwent partial ligation (versus complete ligation) experienced long-term recurrence of signs related to portosystemic shunting. In a study by Hottinger et al.,66 all dogs that underwent complete ligation for whom follow-up greater than one year was available were clinically normal, in comparison to ~10% of dogs that underwent partial ligation. Approximately 40% of dogs that underwent partial ligation in that study experienced recurrence of clinical signs.28 In a further study by Komtebedde et al.,68 recurrence of clinical signs related to portosystemic shunting was observed in 50% of dogs with partially ligated cEHPSS after an average of three years.

- **Endovascular coil embolisation:** Coil embolisation of cPSS is almost exclusively performed for management of cIHPSS due to the challenges associated with locating and attenuating these shunts;13,14,31,32 although coil embolisation of cEHPSS has also been described.12,20 Coils are deployed via a peripheral vein (most commonly femoral vein/saphenous vein or jugular vein)42,64 and up to seven to eight coils are placed within the shunting vessel, most commonly following placement of a caval stent to prevent migration of the coil(s) into the systemic circulation.

**Surgical Complications**

Surgical management of cIHPSS is associated with higher rates of perioperative mortality and intra- and post-operative complications than cEHPSS.18,31,42,70

- **Intraoperative complications:** Intraoperative complications are reported in 0-6.7% of dogs with cEHPSS (including only studies that contain a minimum of 10 dogs).28,31,32,34,40,41,42 Intraoperative complications are reported in 0-20% of dogs with cIHPSS (including only studies that contain a minimum of 10 dogs).13,14,20,22,42 Reported complications include development of portal hypertension,13,14,42 cardiac arrest,13,14,42
hypothenia;\textsuperscript{39}  haemodynamic disturbances, including hypotension;\textsuperscript{8,13,20,45-47} injury to/tearing of the shunt vessel/haemorrhage during shunt dissection;\textsuperscript{1,12,13,24-26} sudden death;\textsuperscript{13}  severe hypotension and oliguric renal failure;\textsuperscript{13}  stent misplacement and/or coil migration;\textsuperscript{2,12,13}  caval stent fracture;\textsuperscript{13}  and so forth. Development of severe portal hypertension can be avoided by temporarily arresting blood flow through the shunt using a vessel loop and bulldog clamp and observing for signs of portal hypertension or measuring portal pressures. This will determine whether complete attenuation can be safely performed. Signs of portal hypertension include intestinal hypermotility and pancreatic congestion/cyanosis, increases in heart rate, and decreases in central venous and arterial blood pressures.\textsuperscript{13}  Guidelines for performing partial attenuation include a rise in postattenuation portal pressure of >20 cm H\textsubscript{2}O, an increase in resting portal pressure of >10 cm H\textsubscript{2}O, a decrease in central venous pressure of >1 cm H\textsubscript{2}O and a decrease in mean arterial pressure of >1 mm H\textsubscript{2}O.\textsuperscript{41}  Measurement of portal pressures requires placement of an intravenous cannula in a jejunal vein (tributary of the cranial mesenteric vein) and connection to a pressure transducer.

- **Postoperative complications:** Postoperative complications include haemorrhage/haemoperitoneum;\textsuperscript{13-15}  coagulopathy;\textsuperscript{7,13,24}  hypoglycaemia;\textsuperscript{13}  development of multiple acquired shunts;\textsuperscript{28,31,34,36}  ascites;\textsuperscript{1,23,24,26-28}  postoperative seizures (post-ligation neurologic syndrome);\textsuperscript{5,7,13,20,28,34,47}  wound-related complications;\textsuperscript{13,28}  abdominal distension;\textsuperscript{28}  portal hypertension;\textsuperscript{13,23,24,28,31,38,40}  cardiac dysrhythmias;\textsuperscript{10,41}  incomplete shunt closure/persistent shunt flow;\textsuperscript{56}  hypothermia;\textsuperscript{13}  bleeding from jugular catheter site;\textsuperscript{10,124}  fistulous tract associated with the subcutaneous injection port;\textsuperscript{46}  requirement for implant (hydraulic occluder) revision;\textsuperscript{13}  cardiac arrest;\textsuperscript{10,41}  respiratory arrest;\textsuperscript{13}  pneumonia;\textsuperscript{10,13}  sudden death;\textsuperscript{10,13}  abdominal distension;\textsuperscript{13}  hypoglycaemia;\textsuperscript{13}  hypotension;\textsuperscript{10,13}  gastrointestinal bleeding/ulceration;\textsuperscript{13,132}  and pancreatitis.\textsuperscript{23}

**PROGNOSIS**

The reported perioperative mortality rate following surgical management of cEHPSs is up to 16.7% (including studies that contain a minimum of 10 dogs).\textsuperscript{28,31,34,36,40,42-47} Good to excellent outcomes are reported in 78-94% of dogs undergoing cEHPSs attenuation.\textsuperscript{1}  The perioperative mortality rate for dogs undergoing cIHPSs attenuation (including only studies that include a minimum of 10 dogs) is between 0%-27%.\textsuperscript{10,13,23,28,32,40,42,7,23}  Good-to-excellent outcomes are reported in 50-100% of dogs undergoing attenuation of cIHPS.\textsuperscript{1}

**REFERENCES**

15. White RN, Trower ND, McEvoy FJ, Garden OA, Boswood A.