# CANINE PORTOSTYSEMIC SHUNTS: PART 2

**Ronan A Mullins MVB DECVS, European specialist in small animal surgery and assistant professor of small animal surgery at University College Dublin, provides his second instalment of a comprehensive overview of congenital portosystemic shunts**

## MEDICAL OR SURGICAL TREATMENT OF CONGENITAL PORTOSYSTEMIC 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.[1] Surgically treated dogs have been demonstrated to experience improved survival and long-term quality of life compared with those managed medically.[2,3] Unlike medical management alone, surgical correction permits re-direction of portal blood flow toward the liver and restoration of liver volume and function.[4] 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,[5] just over 50% of dogs managed medically for cPSS were euthanised 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 PORTOSYSTEMIC 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.[1,6] 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) moderately 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 postattenuation neurologic signs.[6-9] 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.[1,10] Life-long gastroprotectant administration is currently recommended for dogs that have undergone surgical attenuation of cIHPSS.[10]

## SURGICAL MANAGEMENT OF CONGENITAL PORTOSYSTEMIC 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)[15] and portal venotomy.[16-19] 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.[11,12,20,21] Transcaval and transportal venotomy require total hepatic vascular occlusion.[16-18] 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 polyethylene),[24-30] ameroid ring constrictor placement,[9,24,26-28,31-37] thin film (cellophane) banding,[13,19,37-41] and placement of a hydraulic occluder.[42] There is no strong evidence to recommend one surgical technique over another.[43]

## SURGICAL MANAGEMENT OF EXTRAHEPATIC PORTOSYSTEMIC 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.[1] 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.[1] 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.[1] 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.[1] Portophrenic shunts are attenuated on the abdominal surface of the diaphragm by attenuation of the shunt itself or the left phrenic vein.[1]

- **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.[44] These shunts may be identified intraabdominally by opening into the omental bursa.[1] In a recent study,[44] just over 90% of portoazygous shunts arose from the left gastric vein. These shunts are typically attenuated on the abdominal surface of the diaphragm[1] or within the thoracic cavity,[19,45,46] either by lateral thoracotomy[19,45] or transdiaphragmatic incision.[46] Or *et al*[46] 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*[47] 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 cIHPSS is considered significantly more challenging than that of cEHPSS due to the location of the shunt within hepatic parenchyma.[18,32,35] The surgical approach to cIHPSS is influenced to a large extent by the morphology of the shunt. These shunts can be classified as right-, central-, or left-divisional.[1] Shunts attenuated by extravascular techniques are approached via ventral midline coeliotomy with or without diaphragmatic incision or caudal median sternotomy.[28,32,35,40]

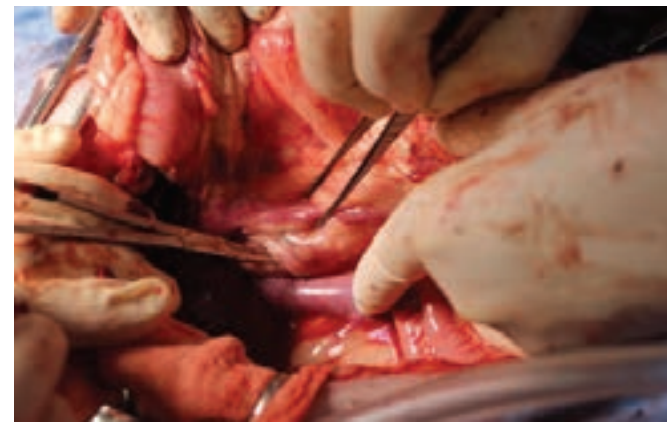

**Figure 1: Intraoperative image demonstrating a haemostat forceps placed in the epiploic foramen in a normal dog without a portosystemic shunt. A DeBakey forceps demonstrates the portal vein. The surgeon's left index finger indicates the pre-hepatic caudal vena cava. The coeliac artery rests on the tips of the haemostat and represents the caudal boundary of the epiploic foramen. Cranial and ventral are to the left and top of the image, respectively.**

- **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,[1,19,35] by intrahepatic dissection of liver tissue,[18] or by intravascular techniques.[1]
- **Central-divisional cIHPSS:** Central-divisional cIHPSS enter either directly into the hepatic vena cava or indirectly via a central hepatic vein,[18] and are attenuated similar to right-divisional cIHPSS at a pre-hepatic location,[1,35] by intrahepatic (to isolate the shunt terminus) or interlobar dissection,[18,48,49] or by intravascular techniques (post-hepatic cavotomy[18] or transportal venotomy).
- **Left-divisional cIHPSS:** Left-divisional cIHPSS enter into the left hepatic vein or rarely the post-hepatic caudal vena cava[18,35] and are attenuated directly in a post-hepatic location just before entering the left hepatic vein[18,35] or indirectly by attenuation of the left hepatic vein,[28] by intrahepatic dissection,[35] or by (pre-hepatic) dissection of the left portal vein branch.[50]

## INDIVIDUAL ATTENUATION TECHNIQUES

- **Ameroid ring constrictor (ARC):** Use of the ARC has been described for surgical management of both cEHPSS[9,24-27,31,34,36,37,47] and cIHPSS.[28,32] The ARC consists of a compressed ring of casein surrounded by a stainless-steel casing.[1] 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.[1] 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 by extraluminal compression by casein, perivascular fibrosis and inflammation/soft tissue formation within the ARC, thrombus formation, or a combination of these mechanisms.[47,51-53] Reported earliest vessel occlusion times

range from within four to five weeks,[31] within 60 days,[31] within three weeks[51] and within eight days.[52] 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*,[55] 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 cEHPSS[22,37,38,40,41,45,56] and cIHPSS[13,19,39,40,57] has been described. Typically an ~12mm strip is folded twice to form an ~4mm wide, three-layered strip of thin film,[38,39,47,56] although a recent study recommended creation of a four-layered strip.[58] The thin film is passed around the shunting vessel and secured in place using titanium clips.[19,37-40,56] 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.[37,38,40,41,56] In a clinical study by Youmans and Hunt,[38] 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.[38,40] In a study by Frankel *et al*,[41] 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 no acute intraoperative attenuation had increased post-prandial bile acid concentrations at just over 2 months but mean 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.[42] 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 until eight weeks.[42]
- **Suture ligation:** Complete and partial ligation of both cEHPSS[24,26,27,29,59-62] and cIHPSS[18,28,60,63] has been described. Most commonly used materials include silk and polypropylene. Complete ligation is not always possible due to development of portal hypertension.[59] 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*,[63] 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.[63] Conversely, complete ligation was possible in 76% of dogs with cEHPSS in a recent study.[59] 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*,[64] 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*,[65] 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.[65] In a further study by Komtebedde *et al*,[66] recurrence of clinical signs related to portosystemic shunting was observed in 50% of dogs with partially ligated cEHPSS after an average of three years.
- **Endovacular coil embolisation:** Coil embolisation of cPPS is almost exclusively performed for management of cIHPSS due to the challenges associated with locating and attenuating these shunts,[11-13,67-69] although coil embolisation of cEHPSS has also been described.[11,12,20] Coils are deployed via a peripheral vein (most commonly femoral vein/saphenous vein or jugular vein),[10,67,69] 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.[18,32,40,70,71]

- **Intraoperative complications:** Intraoperative complications are reported in 0-6.7% of dogs with cEHPSS (including only studies that contain a minimum of 10 dogs).[26,34,37,38,40,61,62] Intraoperative complications are reported in 0-20% of dogs with cIHPSS (including only studies that contain a minimum of 10 dogs).[10,13,32,35,42] Reported complications include development of portal hypertension;[12,14] cardiac arrest;[12]

hypothermia;[26] haemodynamic disturbances, including hypotension;[10,13,26,32,35,42,66] injury to/tearing of the shunt vessel/haemorrhage during shunt dissection;[1,24,34,42] sudden death;[19,34] severe hypotension and oliguric renal failure;[42] stent misplacement and/or coil migration;[10-13,67] caval stent fracture;[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.[1,61] Guidelines for performing partial attenuation include a rise in postattenuation portal pressure of >20cm $H_20$, an increase in resting portal pressure of >10cm $H_20$, a decrease in central venous pressure of >1cm $H_20$ and a decrease in mean arterial pressure of >1mm Hg.[61] 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;[1,34,40] coagulopathy;[28,37] hypoglycaemia;[1] development of multiple acquired shunts;[28,31,34,56] ascites;[10,13,24,28,42] postoperative seizures (post-ligation neurologic syndrome);[6,7,10,13,26,28,34,37-41,56,73] wound-related complications;[13,42] abdominal distension;[10,34] portal hypertension;[1,10,14,26,31,38,40] cardiac dysrhythmias;[10,41] incomplete shunt closure/persistent shunt flow;[56] hypothermia;[19] bleeding from jugular catheter site;[10,14] fistulous tract associated with the subcutaneous injection port;[42] requirement for implant (hydraulic occluder) revision;[42] cardiac arrest;[10,41] respiratory arrest;[13] pneumonia;[10,13] sudden death;[10,19] abdominal distension;[10] hypoglycaemia;[24] hypotension;[13] gastrointestinal bleeding/ulceration;[1,10,13] and pancreatitis.[13]

## 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).[26,31,34,36-38,40,41,61,62] Good to excellent outcomes are reported in 78-94% of dogs undergoing cEHPSS attenuation.[1] The perioperative mortality rate for dogs undergoing cIHPSS attenuation (including only studies that include a minimum of 10 dogs) is between 0%-27%.[10,13,18,28,32,40,42,70] Good-to-excellent outcomes are reported in 50-100% of dogs undergoing attenuation of cIHPSS.[1]

## REFERENCES


1. Berent AC, Tobias KM. Hepatic Vascular Anomalies. In: Johnston SA, Tobias KM , eds. Veterinary Surgery: Small Animal 2nd edition. St. Louis: Elsevier Saunders; 2018: 1852-1886.
2. Greenhalgh SN, Dunning MD, McKinley TJ, *et al*. Comparison of survival after surgical or medical treatment in dogs with a congenital portosystemic shunt. *J Am Vet Med Assoc*. 2010;236(11):1215-20.
3. Greenhalgh SN, Reeve JA, Johnstone T, *et al*. Long-term survival and quality of life in dogs with clinical signs associated with a congenital portosystemic shunt after surgical or medical treatment. *J Am Vet Med Assoc.* 2014;245(5):527-33.
4. Zwingenberger AL, Daniel L, Steffey MA, *et al*. Correlation between liver volume, portal vascular anatomy, and hepatic perfusion in dogs with congenital portosystemic shunt before and after placement of ameroid constrictors. *Vet Surg.* 2014;43(8):926-34.
5. Watson PJ, Herrtage ME. Medical management of congenital portosystemic shunts in 27 dogs – a retrospective study. *J Small Anim Pract*. 1998;39(2):62-8.
6. Strickland R, Tivers MS, Adamantos SE, *et al*. Incidence and risk factors for neurological signs after attenuation of single congenital portosystemic shunts in 253 dogs. *Vet Surg*. 2018;47(6):745-755.
7. Mullins RA, Sanchez Villamil C, de Rooster H, *et al*. Effect of prophylactic treatment with levetiracetam on the incidence of postattenuation seizures in dogs undergoing surgical management of single congenital extrahepatic portosystemic shunts. *Vet Surg*. 2019;48(2):164-172.
8. Brunson BW, Case JB, Ellison GW, *et al*. Evaluation of surgical outcome, complications, and mortality in dogs undergoing preoperative computed tomography angiography for diagnosis of an extrahepatic portosystemic shunt: 124 cases (2005-2014). *Can Vet J*. 2016;57(1):59-64.
9. Fryer KJ, Levine JM, Peycke LE, Thompson JA, Cohen ND. Incidence of postoperative seizures with and without levetiracetam pretreatment in dogs undergoing portosystemic shunt attenuation. *J Vet Intern Med*. 2011;25(6):1379-84.
10. Weisse C, Berent AC, Todd K, *et al*. Endovascular evaluation and treatment of intrahepatic portosystemic shunts in dogs: 100 cases (2001-2011). *J Am Vet Med Assoc*. 2014;244(1):78-94.
11. Bussadori R, Bussadori C, Millán L, Costilla S, Rodríguez-Altónaga JA, Orden MA, Gonzalo-Orden JM. Transvenous coil embolisation for the treatment of single congenital portosystemic shunts in six dogs. *Vet J*. 2008;176(2):221-6.
12. Léveillé R, Johnson SE, Birchard SJ. Transvenous coil embolization of portosystemic shunt in dogs. *Vet Radiol Ultrasound*. 2003;44(1):32-6.
13. Case JB, Marvel SJ, Stiles MC, Maisenbacher HW 3rd, Toskich BB, Smeak DD, Monnet EL. Outcomes of cellophane banding or percutaneous transvenous coil embolization of canine intrahepatic portosystemic shunts. *Vet Surg*. 2018;47(S1):O59-O66.
14. Culp WTN, Zwingenberger AL, Giuffrida MA, Wisner ER, Hunt GB, Steffey MA, Mayhew PD, Marks SL. Prospective evaluation of outcome of dogs with intrahepatic portosystemic shunts treated via percutaneous transvenous coil embolization. *Vet Surg*. 2018;47(1):74-85.
15. White RN, Trower ND, McEvoy FJ, Garden OA, Boswood A.

A method for controlling portal pressure after attenuation of intrahepatic portacaval shunts. *Vet Surg*. 1996;25(5):407-13.
16. Hunt GB, Bellenger CR, Pearson MR. Transportal approach for attenuating intrahepatic portosystemic shunts in dogs. *Vet Surg*. 1996;25(4):300-8.
17. Hunt GB, Youmans KR, Sommerlad S, Swinney G, Nicholson A, Melville L, Hoffman KL, Allan GS. Surgical management of multiple congenital intrahepatic shunts in two dogs: case report. *Vet Surg*. 1998;27(3):262-7.
18. White RN, Burton CA, McEvoy FJ. Surgical treatment of intrahepatic portosystemic shunts in 45 dogs. *Vet Rec*. 1998;142(14):358-65.
19. Hunt GB, Tisdall PL, Webb A, MacPherson GC, Brain P, Malik R. Congenital portosystemic shunts in toy and miniature poodles. *Aust Vet J*. 2000;78(8):530-2.
20. Léveillé R, Pibarot P, Soulez G, Wisner ER. Transvenous coil embolization of an extrahepatic portosystemic shunt in a dog: a naturally occurring model of portosystemic malformations in humans. *Pediatr Radiol*. 2000;30(9):607-9.
21. Hogan DF, Benitez ME, Parnell NK, Green HW 3rd, Sederquist K. Intravascular occlusion for the correction of extrahepatic portosystemic shunts in dogs. *J Vet Intern Med*. 2010;24(5):1048-54.
22. Miller JM, Fowler JD. Laparoscopic portosystemic shunt attenuation in two dogs. *J Am Anim Hosp Assoc*. 2006;42(2):160-4.
23. Brockman DJ, Brown DC, Holt DE. Unusual congenital portosystemic communication resulting from persistence of the extrahepatic umbilical vein. *J Small Anim Pract*. 1998;39(5):244-8.
24. Murphy ST, Ellison GW, Long M, Van Gilder J. A comparison of the Ameroid constrictor versus ligation in the surgical management of single extrahepatic portosystemic shunts. *J Am Anim Hosp Assoc*. 2001;37(4):390-6.
25. Winkler JT, Bohling MW, Tillson DM, Wright JC, Ballagas AJ. Portosystemic shunts: diagnosis, prognosis, and treatment of 64 cases (1993-2001). *J Am Anim Hosp Assoc*. 2003;39(2):169-85.
26. Hurn SD, Edwards GA. Perioperative outcomes after three different single extrahepatic portosystemic shunt attenuation techniques in dogs: partial ligation, complete ligation and ameroid constrictor placement. *Aust Vet J*. 2003;81(11):666-70.
27. Worley DR, Holt DE. Clinical outcome of congenital extrahepatic portosystemic shunt attenuation in dogs aged five years and older: 17 cases (1992-2005). *J Am Vet Med Assoc*. 2008;232(5):722-7.
28. Mehl ML, Kyles AE, Case JB, Kass PH, Zwingenberger A, Gregory CR. Surgical management of left-divisional intrahepatic portosystemic shunts: outcome after partial ligation of, or ameroid ring constrictor placement on, the left hepatic vein in twenty-eight dogs (1995-2005). *Vet Surg*. 2007;36(1):21-30.
29. Bristow P, Tivers M, Packer R, Brockman D, Ortiz V, Newson K, Lipscomb V. Long-term serum bile acid concentrations in 51 dogs after complete extrahepatic congenital portosystemic shunt ligation. *J Small Anim Pract*. 2017;58(8):454-460.
30. Tivers MS, Lipscomb VJ, Bristow P, Brockman DJ. Intrahepatic congenital portosystemic shunts in dogs: short- and long-term outcome of suture attenuation. *J Small Anim Pract*. 2018;59(4):201-210.
31. Vogt JC, Krahwinkel DJ Jr, Bright RM, Daniel GB, Toal RL, Rohrbach B. Gradual occlusion of extrahepatic portosystemic shunts in dogs and cats using the ameroid constrictor. *Vet Surg*. 1996;25(6):495-502.
32. Kyles AE, Gregory CR, Jackson J, Ilkiw JE, Pascoe PJ, Adin C, Samii VF, Herrgesell E. Evaluation of a portocaval venograft and ameroid ring for the occlusion of intrahepatic portocaval shunts in dogs. *Vet Surg*. 2001;30(2):161-9.
33. Gellasch KL, Patricelli AJ, Sicard GK, McAnulty JE. Use of portocaval venografts with ameroid constrictor placement and hepatic lobectomy for treatment of intralobular intrahepatic portocaval shunts in four dogs. *J Am Vet Med Assoc*. 2003;222(4):455-60, 449.
34. Mehl ML, Kyles AE, Hardie EM, *et al*. Evaluation of ameroid ring constrictors for treatment for single extrahepatic portosystemic shunts in dogs: 168 cases (1995-2001). *J Am Vet Med Assoc*. 2005;226(12):2020-30.
35. Bright SR, Williams JM, Niles JD. Outcomes of intrahepatic portosystemic shunts occluded with ameroid constrictors in nine dogs and one cat. *Vet Surg*. 2006;35(3):300-9.
36. Falls EL, Milovancev M, Hunt GB, Daniel L, Mehl ML, Schmiedt CW. Long-term outcome after surgical ameroid ring constrictor placement for treatment of single extrahepatic portosystemic shunts in dogs. *Vet Surg*. 2013;42(8):951-7.
37. Traverson M, Lussier B, Huneault L, Gatineau M. Comparative outcomes between ameroid ring constrictor and cellophane banding for treatment of single congenital extrahepatic portosystemic shunts in 49 dogs (1998-2012). *Vet Surg*. 2018;47(2):179-187.
38. Youmans KR, Hunt GB. Cellophane banding for the gradual attenuation of single extrahepatic portosystemic shunts in eleven dogs. *Aust Vet J*. 1998;76(8):531-7.
39. Connery NA, McAllister H, Skelly C, Pawson P, Bellenger CR. Cellophane banding of congenital intrahepatic portosystemic shunts in two Irish wolfhounds. *J Small Anim Pract*. 2002;43(8):345-9.
40. Hunt GB, Kummeling A, Tisdall PL, Marchevsky AM, Liptak JM, Youmans KR, Goldsmid SE, Beck JA. Outcomes of cellophane banding for congenital portosystemic shunts in 106 dogs and 5 cats. *Vet Surg*. 2004;33:25-31.
41. Frankel D, Seim H, MacPhail C, Monnet E. Evaluation of cellophane banding with and without intraoperative attenuation for treatment of congenital extrahepatic portosystemic shunts in dogs. *J Am Vet Med Assoc*. 2006;228(9):1355-60.
42. Adin CA, Sereda CW, Thompson MS, Wheeler JL, Archer LL. Outcome associated with use of a percutaneously controlled hydraulic occluder for treatment of dogs with intrahepatic portosystemic shunts. *J Am Vet Med Assoc*.

2006;229(11):1749-55.
43. Tivers MS, Upjohn MM, House AK, Brockman DJ, Lipscomb VJ. Treatment of extrahepatic congenital portosystemic shunts in dogs - what is the evidence base? *J Small Anim Pract*. 2012;53(1):3-11.
44. Or M, Ishigaki K, de Rooster H, Kutara K, Asano K. Determination of Porto-Azygos Shunt Anatomy in Dogs by Computed Tomography Angiography. *Vet Surg*. 2016;45(8):1005-1012.
45. Harari J, Lincoln J, Alexander J, *et al*. Lateral thoracotomy and cellophane banding of a congenital portoazygous shunt in a dog. *J Small Ani Pract*. 1990;31:571-573.
46. Or M, Kitshoff A, Devriendt N, De Ridder M, Quist-Rybachuk G, de Rooster H. Transdiaphragmatic Approach to Attenuate Porto-Azygos Shunts Inserting in the Thorax. *Vet Surg*. 2016;45(8):1013-1018.
47. Hunt GB, Culp WT, Mayhew KN, Mayhew P, Steffey MA, Zwingenberger A. Evaluation of in vivo behavior of ameroid ring constrictors in dogs with congenital extrahepatic portosystemic shunts using computed tomography. *Vet Surg*. 2014;43(7):834-42.
48. Kochin EJ, Gregory CR. Surgical attenuation of intrahepatic portosystemic shunts using the cavitron ultrasonic surgical aspirator. *Vet Surg* (Abstract). 1991;20:340.
49. Swalec Tobias KM, Rawlings CA. Surgical techniques for extravascular occlusion of intrahepatic shunts. *Comp Cont Educ Vet Pract*. 1996;18:745-754.
50. Lidbetter DA, Krahwinkel DJ. Gradual occlusion of the left branch of the portal vein with an ameroid constrictor for treatment of an intrahepatic portosystemic shunt. *Aust Vet Practit*. 2001;31:120–124.
51. Youmans KR, Hunt GB. Experimental evaluation of four methods of progressive venous attenuation in dogs. *Vet Surg*. 1999;28(1):38-47.
52. Besancon MF, Kyles AE, Griffey SM, Gregory CR. Evaluation of the characteristics of venous occlusion after placement of an ameroid constrictor in dogs. *Vet Surg*. 2004;33(6):597-605.
53. Adin CA, Gregory CR, Kyles AE, Griffey SM, Kendall L. Effect of petrolatum coating on the rate of occlusion of ameroid constrictors in the peritoneal cavity. *Vet Surg*. 2004;33(1):11-6.
54. Wallace ML, Ellison GW, Giglio RF, Batich CD, Berry CR, Case JB, Kim SE. Gradual attenuation of a congenital extrahepatic portosystemic shunt with a self-retaining polyacrylic acid-silicone device in 6 dogs. *Vet Surg*. 2018;47(5):722-728.
55. Smith RR, Hunt GB, Garcia-Nolen TC, Stump S, Stover SM. Spectroscopic and mechanical evaluation of thin film commonly used for banding congenital portosystemic shunts in dogs. *Vet Surg*. 2013;42(4):478-87.
56. Landon BP, Abraham LA, Charles JA. Use of transcolonic portal scintigraphy to evaluate efficacy of cellophane banding of congenital extrahepatic portosystemic shunts in 16 dogs. *Aust Vet J*. 2008;86(5):169-79.
57. Nelson NC, Nelson LL. Imaging and Clinical Outcomes in 20 Dogs Treated with Thin Film Banding for Extrahepatic Portosystemic Shunts. *Vet Surg*. 2016;45(6):736-45.
58. Joffe MR, Hall E, Tan C, Brunel L. Evaluation of different methods of securing cellophane bands for portosystemic shunt attenuation. *Vet Surg*. 2019;48(1):42-49.
59. Kayanuma H, Koyama R, Kanai E. Feasibility of complete surgical ligation on 72 dogs with singular extrahepatic congenital portosystemic shunt based on portal pressure and comparison of intraoperative mesenteric portovenography. *J Vet Med Sci*. 2019;81(3):361-364.
60. Wolschrijn CF, Mahapokai W, Rothuizen J, Meyer HP, van Sluijs FJ. Gauged attenuation of congenital portosystemic shunts: results in 160 dogs and 15 cats. *Vet Q*. 2000;22(2):94-8.
61. Hunt GB, Hughes J. Outcomes after extrahepatic portosystemic shunt ligation in 49 dogs. *Aust Vet J*. 1999;77(5):303-7.
62. Harvey J, Erb HN. Complete ligation of extrahepatic congenital portosystemic shunts in nonencephalopathic dogs. *Vet Surg*. 1998;27(5):413-6.
63. Tivers MS, Lipscomb VJ, Bristow P, Brockman DJ. Intrahepatic congenital portosystemic shunts in dogs: short- and long-term outcome of suture attenuation. *J Small Anim Pract*. 2018;59(4):201-210.
64. Johnson CA, Armstrong PJ, Hauptman JG. Congenital portosystemic shunts in dogs: 46 cases (1979-1986). *J Am Vet Med Assoc*. 1987;191(11):1478-83.
65. Hottinger HA, Walshaw R, Hauptman JG. Long-term results of complete and partial ligation of congenital portosystemic shunts in dogs. *Vet Surg*. 1995;24(4):331-6.
66. Komtebedde J, Koblik PD, Breznock EM. Long-term clinical outcome after partial ligation of single extrahepatic vascular anomalies in 20 dogs. *Vet Surg*. 1995;24(5):379-83.
67. Gonzalo-Orden JM, Altónaga JR, Costilla S, Gonzalo Cordero JM, Millán L, Recio AO. Transvenous coil embolization of an intrahepatic portosystemic shunt in a dog. *Vet Radiol Ultrasound*. 2000;41(6):516-8.
68. Asano K, Watari T, Kuwabara M, Sasaki Y, Teshima K, Kato Y, Tanaka S. Successful treatment by percutaneous transvenous coil embolization in a small-breed dog with intrahepatic portosystemic shunt. *J Vet Med Sci*. 2003;65(11):1269-72.
69. Knapp T, Navalòn I, Medda M, Pradelli D, Borgonovo S, Crosta C, Bussadori CM. A multimodality imaging approach for guiding a modified endovascular coil embolization of a single intrahepatic portosystemic shunt in dogs. *Res Vet Sci*. 2015;103:156-63.
70. Komtebedde J, Forsyth SF, Breznock EM, Koblik PD. Intrahepatic portosystemic venous anomaly in the dog. Perioperative management and complications. *Vet Surg*. 1991;20(1):37-42.
71. Papazoglou LG, Monnet E, Seim HB 3rd. Survival and prognostic indicators for dogs with intrahepatic portosystemic shunts: 32 cases (1990-2000). *Vet Surg*. 2002;31(6):561-70.
72. Wallace ML, MacPhail CM, Monnet E. Incidence of Postoperative Neurologic Complications in Pugs Following Portosystemic Shunt Attenuation Surgery. *J Am Anim Hosp*