Portosystemic shunt surgery and PO care in cats and dogs

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FOLLOWING diagnosis and appropriate stabilisation of dogs and cats with portosystemic shunts (PSS), as detailed in part one of this article (VT40.45), the authors generally recommend surgery.

The aim of surgical treatment is to occlude the shunting vessel (preferably completely at surgery or gradually over time) so that blood flow is diverted entirely through the liver and the animal can be slowly weaned off medical management.

Certain situations may be influenced by both patient and owner factors, where surgery is not recommended and the patient continues on long-term medical management, as discussed in the previous article.

Finding the shunt (extrahepatic)

Before surgery, the nature of the shunt (intrahepatic or extrahepatic) should be confirmed wherever possible using ultrasound, mesenteric portovenography, computed tomography or magnetic resonance imaging.

The shunting vessel is approached via a ventral midline laparotomy, taking particular care when entering the abdomen because, rarely, shunts can be located in the falciform ligament (Brockman, 1998). In the first instance, exploration of the caudal vena cava (CVC) is advisable and this can be achieved by performing the duodenal manoeuvre, followed by the colonic manoeuvre. In a normal dog, no vessels should enter the CVC between the right renal/phrenicoabdominal and hepatic veins, so any large vessels identified in this region may be a shunt (Figure 1).

Shunts can be very large (15mm or larger) and the blood flow within the shunt and the CVC in this region will be turbulent. Occasionally, CVC dilation can also be seen at the level of shunt.

If a vessel is not visualised using this technique, exploration of the epiploic foramen may be rewarding. This can be performed by opening the two leaves of the omentum and following them dorsally. The boundaries of the epiploic foramen include the CVC (dorsally), the hepatic artery and portal vein (ventrally), and the celiac artery (caudally).

A porto-azygous vessel is usually identified at surgery as a vessel that heads in a craniodorsal
direction arising from a contributory of the hepatic portal vein. Exploration of the diaphragmatic crura and oesophageal hiatus should also be performed, by retracting the liver and stomach to the right to aid visualisation.

Very rarely, more than one shunt may be present. Shunts have been described associated with the phrenic vein, left colic vein or umbilical vein remnant. A full abdominal exploration is, therefore, strongly advised in every case. If no shunt can be found, intravenous mesenteric portovenography is recommended, as is a liver biopsy to rule out microvascular dysplasia (hypoplasia of the portal vein).

**Finding the shunt (intrahepatic)**

Intrahepatic shunts are very challenging to locate because they are usually completely surrounded by hepatic parenchyma.

In a similar fashion to extrahepatic PSS, the hepatic and portal vein branches associated with the shunt may be identifiable as dilated vessels containing turbulent blood flow.

To minimise extensive parenchymal dissection (which may cause profuse haemorrhage, particularly in patients that may have coagulation problems), surgeons have attempted to identify the shunts by palpating the aneurysms associated with them through the liver lobes (Breznock, 1983), performing intraoperative ultrasonography (Wrigley, 1983) or catheterising the shunt via the portal vein (Tobias, 1996). Shunt location can be confirmed by digital occlusion of the vessel and observation of changes in portal pressure or visceral appearance.

**Shunt occlusion (extraheptic)**

Once the shunt has been located, the authors place a Rummel tourniquet (Figure 2) to achieve complete occlusion for five minutes, while carefully observing for signs of portal hypertension. Grossly, changes suggestive of this include intestinal cyanosis, increased intestinal peristalsis, pancreatic cyanosis/oedema (Figure 3) and increased mesenteric vascular pulsations.

Pressure measurements should be carefully assessed while completely occluding the shunt: central venous pressure should not decrease by greater than 7mmHg, arterial blood pressure should be limited to changes of 5mmHg or less and the heart rate should not dramatically change. Changes in excess of this mean that complete ligation is not possible at this stage because the risk of portal hypertension is too great.

Some surgeons also measure portal pressure via a jejunal venous catheter to assess for hypertension. Normal portal pressure is 6mmHg to 10mmHg and animals with PSS have pressure of 0mmHg to 8mmHg. Post-ligation pressure should be limited to between 12mmHg and 17mmHg, with a maximum change of 6mmHg to 7mmHg (Martin, 1987; Swalec, 1991; Tobias, 2003).
If gross clinical changes and pressure measurements are acceptable, the Rummel tourniquet can be removed and a ligature placed (the authors use polypropylene) to achieve complete ligation of the shunt, but only 40 per cent to 68 per cent of dogs and cats can tolerate this (Swalec, 1990; Tobias, 2003).

Partial ligation is possible and this may be performed with suture, followed by complete occlusion at a subsequent laparotomy. In the authors’ experience, owners are often reluctant to proceed with a second procedure and, for this reason, we recommend the use of ameroid constrictors or cellophane banding where complete ligation is not possible. These techniques cause gradual occlusion and fewer postoperative complications (Tobias, 2003), and reduce the need for second surgery.

Ameroid constrictors are casein rings encased in stainless steel (Figure 4). The casein absorbs fluid from the body and swells inwards, compressing the shunt placed within the lumen over four to five weeks, although this can be highly variable (Vogt, 1996; Monnet, 2005). Rings are available in a variety of sizes and should be selected so that the ring fits snugly around the shunt, causing minimal occlusion when first placed. Ideally, dissection around the shunt should be kept to a minimum to reduce movement of the heavy ring, which otherwise may cause kinking and, therefore, premature occlusion of the shunt. For this reason, placement as close as possible to the vena cava is also advised.

Folded strips of cellophane bands are easy to place and cause gradual occlusion of the shunt over eight weeks by inciting a fibrous reaction (Youmans, 1998). In the same manner as the ameroid constrictors, they should not cause any occlusion when first placed, and security of the band is achieved using vascular clips (Figures 5 and 6). Particularly large shunts may not occlude completely after cellophane banding.

Following shunt attenuation, a liver biopsy should be obtained to assess for other hepatic disease, such as copper storage disease. The clinician should be aware that the histopathological changes seen in a liver biopsy from an animal with PSS will be similar to that from an animal with microvascular dysplasia/hypoplasia of the portal vein. Biopsy alone cannot differentiate between the two conditions and a gross shunt must be excluded by visualisation, contrast studies or advanced imaging.

**Shunt occlusion (intrahepatic)**

Complete ligation of intrahepatic shunts is only possible in 27 per cent of cases, but the shunts may also be occluded with cellophane or ameroid constrictors. Because the shunts are often difficult to locate and dissect, treatment often consists of ligation of the portal vein or hepatic vein associated with the shunt. For intrahepatic shunts, the authors prefer to use transvenous coil embolisation, which has also been described for extrahepatic PSS (Leveille, 2003; Schneider, 2005; Bussadori, 2008).
For this technique, a guide wire, under fluoroscopic guidance, is passed from the jugular vein, via the cranial vena cava and right atrium, into the caudal vena cava. A catheter is advanced over the guide wire (Figure 7) and contrast angiography is performed to confirm the location of the shunt, using fluoroscopy and digital subtraction (Figure 8).

The diameter of the caudal vena cava is measured and an appropriately sized, ensheathed, self-expanding nitinol stent (Figure 9) is advanced over the guide wire and deployed under fluoroscopic guidance so that it covers the vessel entry point into the CVC – the left hepatic vein in this case (Figure 10).

Accurate placement is confirmed by repeat angiography, which is especially important because dilation of the CVC in the region of the shunt is not uncommon and may lead to incorrect stent positioning.

A cobra-tipped catheter is passed down the guide wire, through the interstices of the stent in to the shunt, and is used to pass thrombogenic coils in to it (Figure 11).

The stent prevents migration of the coils into the CVC and the catheter tip also allows measurement of portal pressures. Coils are added until the portal pressures increase by 7mmHg to 14mmHg and, in the authors’ experience, three to five coils are required to achieve this.

**Postoperative care**

Following surgery, animals should be constantly monitored in the intensive care unit for at least 24 hours, recording demeanour, heart rate, pulse quality, respiratory rate, blood pressure and urinary output.

Postoperative complications include hypoglycaemia, prolonged anaesthetic recovery, haemorrhage (surgical or gastrointestinal haemorrhage), seizures or portal hypertension, all of which are reported to be common in up to 75 per cent of cats (Kyles, 2002).

Signs of portal hypertension include gastrointestinal haemorrhage, abdominal pain and ascites, but this is uncommonly seen with techniques that cause gradual occlusion.

Other parameters measured at the authors’ institution include blood gas analysis, electrolytes and glucose, and appropriate treatment, such as potassium or glucose-spiked fluids, is provided as required and assessed frequently in the immediate postoperative period.

It is particularly important to assess pain levels closely because increased pain may be an indication of portal hypertension or pancreatitis. Patients recovering from PSS surgery are particularly challenging in this respect because they respond unpredictably to analgesia as a result of their slowed liver metabolism.
For this reason, full opioid analgesia – administered as required – may be more advisable that a static standard protocol of one dose every four hours. The development of hepatic encephalopathy may also be misinterpreted as dysphoria or pain, so careful monitoring by experienced personnel is essential.

Ideally, PSS patients should require no long-term medical management following successful surgical attenuation of the shunt. If animals are otherwise clinically well, they should be reviewed after one month to allow measurement of serum biochemical (particularly bile acids) parameters. At this stage, antibiotics may be stopped if results are normal.

After two months of satisfactory progression, lactulose may be titrated down and stopped. After three to four months, the gradual introduction of a normal diet may be considered. It is important to perform dynamic bile acid assessment at this stage.

Mortality rates for patients treated surgically are illustrated in Table 1. Good-to-excellent outcomes can be expected in up to 94 per cent of dogs undergoing surgery for extrahepatic PSS, regardless of the method used. Where the shunt is intrahepatic, the same results may be expected for 70 to 89 per cent of dogs receiving ameroid constrictors, 76 to 100 per cent undergoing complete occlusion and 50 per cent undergoing cellophane band placement (Berent, 2009).

In cats, an excellent long-term outcome is less guaranteed, being seen in up to 75 per cent of cats undergoing ligation or ameroid constrictor placement, and 80 per cent receiving cellophane bands.

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References and further reading
