CHAPTER 8

Ultrasonographic evaluation of partially attenuated congenital extrahepatic portosystemic shunts in dogs

based on the article by

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Summary

Doppler ultrasonography was used to evaluate the portal vein in fourteen dogs before, immediately after and four weeks after a partial ligation of a congenital extrahepatic portocaval shunt. The preoperatively detected hepatofugal or zero flow in the portal vein segment cranial to the shunt-origin turned to hepatopetal in thirteen dogs by the fourth postoperative week and these dogs became clinically healthy. Only one dog continued to display hepatofugal flow in the portal vein cranial to the shunt-origin and this dog continued to show clinical signs of hepatic encephalopathy. Regarding the shunt-flow, in six dogs the shunt remained functional. In three of the six, in addition to the functional shunt, acquired portosystemic collaterals developed. In the remaining eight dogs the patent shunt was non-functional, since hepatopetal shunt-flow was detected adjacent to the portal vein. The hepatopetal flow in the shunt was the result of the fact that the splenic vein entered the shunt, and the splenic blood was divided: one part flowed via the shunt towards the portal vein preventing the portal blood from shunting, and the other part flowed via the attenuated shunt-segment to the caudal vena cava. Shunting of the splenic venous blood is clinically insignificant. A second surgery for further shunt-attenuation is recommended in cases when the flow-direction remains hepatofugal in the portal vein cranial to the shunt-origin. However, further shunt-attenuation should not be performed, when acquired portosystemic collaterals are present or when the flow is hepatopetal in the shunt adjacent to the portal vein. When the flow is hepatopetal in the entire portal vein postoperatively, regardless of the presence of congenital or acquired shunting, further shunt-attenuation is usually unnecessary, since these dogs are clinically healthy.
Introduction

Portosystemic shunting occurs when anomalous veins allow the portal blood to enter the systemic veins directly without first flowing through the hepatic sinusoids. Portosystemic shunting can be acquired or congenital. **Acquired portosystemic collaterals (APSCs)** develop as the result of sustained hepatic or prehepatic portal hypertension by enlargement of extrahepatic rudimentary vessels, through which no blood normally passes. Collateral-formation is a compensatory mechanism to maintain normal portal pressure by allowing the portal blood to be drained into the lower pressure systemic veins. Portosystemic shunting is considered to be congenital if a single or double anomalous vein is present without a concurrent portal hypertension. **Definitive treatment of congenital portosystemic shunts (CPSSs) involves occlusion of the shunting vessel at a location closest to the systemic venous circulation.**

Portosystemic shunting is considered to be congenital if a single or double anomalous vein is present without a concurrent portal hypertension. Definitive treatment of congenital portosystemic shunts (CPSSs) involves occlusion of the shunting vessel at a location closest to the systemic venous circulation. Attenuating the shunt forces blood to flow through the portal branches, which are often hypoplastic, resulting in subsequent development of portal hypertension. Portal vein hypoplasia may be primary or arise secondary to portal hypoperfusion. The degree of portal hypertension that develops during attenuation of a CPSS depends on the severity of portal vein hypoplasia (i.e. the capacity of the portal branches to absorb the increased blood flow), and on the degree of shunt-attenuation. If severe post-ligation portal hypertension develops, the patient may die during or shortly after the surgery as a result of circulatory collapse or thrombosis of the portal vein. If these acute complications do not occur, APSCs may develop as a result of sustained portal hypertension.

Complete occlusion of a CPSS has been suggested to result in a better outcome compared to partial ligation. However, other groups found no difference in clinical outcome between dogs with partial and complete shunt-ligation. The underlying reasons of poor outcomes in most of the studies remained undetermined. It has been suggested that, if complete shunt-occlusion is not feasible during a surgical attenuation of a CPSS because it would cause the development of a fatal portal hypertension, a second surgery should be performed to attempt a complete shunt-occlusion. The underlying idea is that an initial partial ligation would allow the portal system to adapt to an increased flow and the portal branches would become gradually wider by the time of the second surgery.

A second surgery should only be considered, when portosystemic shunting persists exclusively through the CPSS. When shunting occurs through APSCs, further attenuation of the CPSS is contraindicated. To determine whether post-ligation portosystemic shunting occurs via the CPSS, APSCs or both, mesenteric portography can be used. In addition to involving the use of ionising radiation, this angiographic technique requires general anaesthesia and a laparotomy, both of which are high risk procedures in animals with hepatic insufficiency. Though scintigraphy is suitable to detect and to quantify portosystemic shunting, with this technique it is impossible to distinguish between congenital and acquired shunting. Doppler ultrasonography offers a non-invasive way to examine the abdominal blood vessels in unsedated dogs, and the direction and velocity of flow can easily be determined. However, no reports were found in the literature about the ultrasonographic findings of dogs that underwent partial surgical ligation of a CPSS.

Our aims are to describe the post-ligation hemodynamic changes of the portal system and to suggest ultrasonographic criteria for decision making as to whether a dog should or should not undergo a second laparotomy for complete CPSS-ligation. Furthermore, we wanted to find an explanation at the level of portal hemodynamics as to
why certain dogs with a partially attenuated extrahepatic CPSS have excellent and others poor clinical outcomes.

**Materials and methods**

Of the dogs that underwent a surgical ligation of an extrahepatic CPSS at the Companion Animal Clinic of Utrecht University between April 2001 and April 2003 the ones that were selected fulfilled the following criteria: the CPSS was partially attenuated; the owners returned with their dogs to the clinic for a re-examination four weeks after surgery; and pre- and intraoperative ultrasound examinations were also performed.

Fourteen dogs fulfilled the inclusion criteria, of which three toy breed dogs had congenital extrahepatic right gastric-caval, and ten toy breed dogs and one Rottweiler had congenital extrahepatic spleno-caval shunts. The age of the dogs at surgery ranged between 4 months and 5.5 years. The post-attenuation diameter of the shunt ranged from 1.50 to 5.00 mm.

The diagnosis of CPSS was established preoperatively in unsedated animals using transabdominal ultrasonography (ATL HDI 3000 ultrasound system with 7-4 MHz phased array and 8-5 MHz curved linear array transducers, Philips Medical Systems, Advanced Technical Laboratories Ultrasound) by direct visualization of the CPSS after having measured a high 12-hour-fasting blood ammonia level (normal reference range: 24-45 µmol/l).

Gauged surgical attenuation of the CPSSs was performed by one surgeon (FJvS) according to a reported method2 combined with intraoperative Doppler ultrasonography **(Chapter 7).** The CPSS was attenuated to the narrowest diameter that did not result in cyanosis of the intestines. If signs of portal hypertension such as intestinal cyanosis were seen during the surgery, then the ligature was removed and another one with a larger diameter was placed around the shunting vessel. Portal pressure was not measured. Before and after placing the ligature around the shunt the presence and direction of blood flow was determined in the shunt and in the portal vein cranial as well as caudal to the shunt-origin with intraoperative color Doppler ultrasonography. The results of the postligation intraoperative ultrasonography did not influence the surgeon’s decision making about the degree of shunt-attenuation. All pre-, intra- and postoperative ultrasound examinations were done by one ultrasonographer (VSz).

At the 4-week re-check, the 12-hour-fasting blood ammonia level was measured and an abdominal ultrasound examination was performed. Special attention was paid to the presence of free abdominal fluid and the visibility of the left gonadal vein (criteria to identify the presence of portal hypertension), and to the size and symmetry of the liver (indicator of portal venous perfusion of the liver). Acquired portosystemic collaterals were diagnosed ultrasonographically when the left gonadal vein was dilated **(Fig 1, Chapter 5).** Special efforts were made to image the right and left portal branches.
Dilated left ovarian vein (LOV) as a result of acquired spleno-renal collaterals in a miniature schnauzer four weeks after partial attenuation of a congenital extrahepatic spleno-caval shunt. This color Doppler ultrasound image was made via the left flank with the dog in right lateral recumbency. Arrows indicate the directions of blood flow. LRV Left renal vein, CVC Caudal vena cava. (Full color illustration on page 183.)

Colour Doppler ultrasonography was used to determine the direction of flow in the shunt, the portal branches and the portal vein both cranial and caudal to the origin of the shunt. The direction of flow in the shunt was determined adjacent to the portal vein. Pulsed wave Doppler ultrasonography with uniform insonation method was used to measure the flow velocity in the portal vein caudal to the shunt-origin. The time-averaged mean portal velocity was calculated by a built-in automatic spectrum analyser (HighQ ATL HDI 3000).

The ultrasonographic findings of the preoperative, postligation intraoperative and the 4-week postoperative examinations were compared in each dog.

Based on the ultrasonographic findings four weeks postoperatively, four outcome categories were defined. The outcome was excellent, when the flow-directions in the shunt and in the entire portal vein were hepatopetal (flow towards the liver), and no APSCs were found. The outcome was good, when the flow-direction in the entire portal vein was hepatopetal, but hepatofugal (flow away from the liver) in the shunt, and no APSCs were found. The outcome was fair, when hepatopetal flow was seen in the entire portal vein, the flow-direction was hepatofugal in the shunt, and APSCs were found. The outcome was poor, when the flow-directions were hepatofugal both in the shunt and in the portal vein cranial to the shunt-origin, and no APSCs were found.

Ultrasound-guided core biopsies of the liver were taken for histopathologic examination at the 4-week re-check, when the whole or a part of the liver was small despite the presence of hepatopetal flow in the entire portal vein and there was concurrent hyperammonemia.
Results

**Right gastric caval shunts**

The blood flow directions pre-, immediately post- and 4 weeks postoperatively in these three dogs are demonstrated in Table 1A. At preoperative ultrasonography the left and right portal branches as well as the portal vein cranial to the shunt-origin could not be visualised.

Four weeks postoperatively the owners of all three dogs reported complete resolution of the clinical signs. The blood ammonia levels were normal and ultrasonographic signs of portal hypertension were absent. The liver had normal size and was symmetric. The left and right portal branches were well recognisable. Color Doppler ultrasonography revealed that the direction of blood flow in the entire portal vein and in the portal branches was hepatopetal in each of the three dogs, whereas the flow-direction in the shunt was hepatopetal in two (Fig 2) and hepatofugal in one dog. The time-averaged mean velocity in the portal vein caudal to the shunt-origin ranged from 13.0 to 21.3 cm/s.

![Color Doppler ultrasound image of a congenital extrahepatic right gastric-caval shunt in a Maltese dog, four weeks after partial surgical attenuation made via the left flank with the dog in right lateral recumbency. The image shows the origin of the cranial shunt-loop. The flow-directions in the shunt (SH) and in the entire portal vein are hepatopetal. The portal vein cranial to the shunt-origin (PVcrSH) has the same diameter as the portal vein caudal to the shunt-origin (PVcaudSH). Arrows indicate the direction of blood flow. HA hepatic artery. (Full color illustration on page 183.)](image)

**Figure 2.**

Color Doppler ultrasound image of a congenital extrahepatic right gastric-caval shunt in a Maltese dog, four weeks after partial surgical attenuation made via the left flank with the dog in right lateral recumbency. The image shows the origin of the cranial shunt-loop. The flow-directions in the shunt (SH) and in the entire portal vein are hepatopetal. The portal vein cranial to the shunt-origin (PVcrSH) has the same diameter as the portal vein caudal to the shunt-origin (PVcaudSH). Arrows indicate the direction of blood flow. HA hepatic artery. (Full color illustration on page 183.)
Table 1.
Comparison of the blood flow directions before, immediately after and 4 weeks after partial surgical attenuation of a congenital extrahepatic portosystemic shunt in fourteen dogs.

The outcome categories were defined based on the portal hemodynamics four weeks postoperatively. The arrows indicate the directions of blood flow as it is shown on Figure 4A: the first horizontal arrow represents the portal vein cranial to the shunt-origin, the vertical arrow represents the shunt as it originates from the portal vein and the second horizontal arrow represents the portal vein caudal to the shunt-origin. Hepatopetal flow: ← and ↑, hepatofugal flow: → and ↓, zero flow: -, acquired portosystemic collaterals: ω

A. Right gastric-caval shunts (n=3)

<table>
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<tr>
<th>OUTCOME</th>
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<th>IMMEDIATELY AFTER NARROWING</th>
<th>FOUR WEEKS AFTER NARROWING</th>
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<td>EXCELLENT</td>
<td>←↑←</td>
<td>←↑← (Fig 2) normal liver</td>
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<tr>
<td>GOOD</td>
<td>small liver</td>
<td>←↓←</td>
<td>←↓← normal liver</td>
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B. Spleno-caval shunts (n=11)

<table>
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<th>OUTCOME</th>
<th>BEFORE NARROWING</th>
<th>IMMEDIATELY AFTER NARROWING</th>
<th>FOUR WEEKS AFTER NARROWING</th>
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<td>←↑← (Figs 4B,4C) normal liver</td>
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<td>EXCELLENT</td>
<td>↓← (Fig 5A)</td>
<td>←↓←</td>
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<tr>
<td>GOOD</td>
<td>small liver</td>
<td>←↓← (Fig 5A)</td>
<td>←↓← (Figs 5B,5C) normal liver</td>
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<tr>
<td>FAIR</td>
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<td>←↓← (Figs 1,5B) asymmetric liver</td>
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<tr>
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<tr>
<td>POOR</td>
<td>←↓← (Fig 6A)</td>
<td>→↓← small liver</td>
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Spleno-caval shunts
The blood flow directions pre-, immediately post- and 4 weeks postoperatively in these eleven dogs are demonstrated in Table 1B. The preoperative ultrasonographic findings were identical in all the eleven dogs, namely the liver was small, the left and right portal branches could not be identified, and the flow-directions in the shunt and in the portal vein segment between the shunt-origin and the entering point of the gastroduodenal vein were hepatofugal (Fig 3).
Figure 3.
Transabdominal color Doppler ultrasound image of a congenital extrahepatic spleno-caval shunt before shunt-attenuation. The flow-direction is hepatofugal in the portal vein segment between the origin of the shunt (SH) and the entering point of the gastroduodenal vein (GDV) because the majority of blood from the GDV flows caudally to the shunt. The blood that comes from the intestines and from the spleen is shunting. The direction of the blood flow is indicated by arrows.

Dogs with excellent outcome. Intraoperative ultrasonography immediately after shunt-attenuation revealed the presence of hepatopetal flow in the entire portal vein and in the shunt adjacent to the portal vein in 5 dogs (Fig 4A). In one dog hepatofugal flow was seen in the entire shunt (Table 1B).

Four weeks postoperatively the owners of all six dogs reported complete resolution of the clinical signs. The blood ammonia levels were normal. The liver was symmetric and its size was estimated to be larger than before surgery. Ultrasonographic signs of portal hypertension were absent. The left and right portal branches were well recognisable. Color Doppler ultrasonography revealed that the direction of blood flow in the entire portal vein, in the portal branches and in the shunt adjacent to the portal vein was hepatopetal (Fig 4B). In the shunt-segment between the splenic vein and the portal vein the flow-direction was hepatopetal, and in the shunt segment between the splenic vein and the caudal vena cava it was hepatofugal, since a part of the blood from the splenic vein flowed via the shunting vessel to the portal vein and the other part flowed via the attenuated shunt-segment to the caudal vena cava (Fig 4C).
Figure 4.
See legends on opposite page.
(Full color illustrations on page 184.)
Figure 4. Partially ligated spleno-caval shunt. Scenario of an excellent outcome. The direction of the blood flow is indicated by interrupted arrows.

A. Intraoperative color Doppler ultrasound image made immediately after partial shunt-attenuation reveals hepatopetal flow in the shunt (SH) and in the portal vein both cranial (PVcrSH) and caudal to the shunt origin (PVcaudSH). The portal vein segment cranial to the shunt-origin has the smallest diameter and the highest flow velocity.

B. Longitudinal transabdominal color Doppler ultrasound image made immediately after shunt-attenuation reveals hepatofugal flow in the shunt (SH) and hepatopetal flow in the portal vein both cranial (PVcrSH) and caudal to the shunt origin (PVcaudSH). Compare to Figure 3!

C. Transverse transabdominal color Doppler ultrasound image made via a right intercostal space, four weeks after partial shunt-attenuation. The spleenic venous blood flows to the shunt and from there to the portal vein (PV). AO Aorta

Figure 5. Partially ligated spleno-caval shunt. Scenario of a good outcome. The direction of the blood flow is indicated by interrupted arrows.

A. Intraoperative colour Doppler ultrasound image made immediately after partial shunt-attenuation reveals hepatofugal flow in the shunt (SH) and hepatopetal flow in the portal vein both cranial (PVcrSH) and caudal to the shunt origin (PVcaudSH). Compare to Figure 4A!

B. Longitudinal transabdominal color Doppler ultrasound image made via the right flank, four weeks after partial shunt-attenuation. The flow-direction in the entire portal vein is hepatopetal and in the shunt is hepatofugal. Both the blood of the splenic vein (SPLV) and that of the portal vein caudal to the shunt-origin (PVcaudSH) are shunting. Compare to Figures 3 and 4B! HA Hepatic artery, GDV gastroduodenal vein, PVcrSH Portal vein segment between the shunt-origin and the entering point of the gastroduodenal vein, PVcrGDV Portal vein cranial to the entering point of the gastroduodenal vein

C. Transverse transabdominal color Doppler ultrasound image made via a right intercostal space, four weeks after partial shunt-attenuation. The direction of the blood flow is hepatofugal in the entire SH. This means that the toxin rich blood from the portal vein (PV) flows to the caudal vena cava (CVC). The stenosis of the shunt caused by the ligature is clearly seen next to the CVC (arrow). In the lumen of the CVC the orange colour signal indicates a rapid blood flow (poststenotic jet). AO Aorta

Dog with good outcome. Intraoperative ultrasonography immediately after shunt-attenuation revealed the presence of hepatopetal flow in the entire portal vein and hepatofugal flow in the shunt adjacent to the portal vein (Fig 5A).

Four weeks postoperatively the owner of the dog reported complete resolution of the clinical signs. The blood ammonia level was normal and ultrasonographic signs of portal hypertension were absent. The liver had normal size and was symmetric. The left and right portal branches were well recognisable. Color Doppler ultrasonography revealed hepatopetal flow in the entire portal vein (Fig 5B) and in the portal branches, and hepatofugal flow in the entire shunt (Fig 5C).

Dogs with fair outcome. In two of the three dogs (a 4-month-old cairn terrier and a 8-month-old miniature schnauzer) with fair outcome the postligation intraoperative ultrasonography revealed hepatofugal flow in the shunt and in the portal vein cranial to the shunt-origin (Fig 6A), whereas four weeks after surgery the flow-direction became hepatopetal in the entire portal vein (Fig 5B, Table 1B). Actually, the change of the flow-
direction in the portal vein cranial to the shunt-origin was detected already one day after surgery during a non-scheduled ultrasound examination in one dog (Fig 6B). At the 4-week re-check abdominal ultrasonography revealed a markedly asymmetric liver. In both dogs the left half of the liver was very small, however the right half was enlarged. The diameters of the portal branches corresponded with the liver size, namely the left portal branch was narrower in both cases than the right one, however normally the left branch is wider. Histopathologic examination of the liver biopsy specimens of the small left liver half revealed the presence of portal vein hypoplasia (Figs 7A, B), whereas the hepatic architecture was normal of the specimens that were taken form the enlarged right liver half (Figs 7C, D).

A. Intraoperative color Doppler ultrasound image of a partially ligated congenital extrahepatic spleno-caval shunt immediately after partial shunt-attenuation. The flow-direction is hepatofugal in the shunt (SH) and in the portal vein cranial to the shunt-origin (PVcrSH). There is normal flow in the portal vein caudal to the shunt-origin (PVcaudSH).

B. Longitudinal transabdominal color Doppler ultrasound image via the right flank, one day after partial shunt-attenuation. The flow in the entire portal vein turned to hepatopetal compared to the intraoperative state (see Figure 6A!). Since the portal vein segment cranial to the entering point of the gastroduodenal vein (PVcrGDV) is very thin, the flow velocity is very high in it (aliasing artefact).

The third dog with fair outcome (a 9-month-old Yorkshire terrier) displayed hepatopetal flow both in the shunt and in the portal vein cranial to the shunt-origin and zero flow in the portal vein caudal to the shunt-origin at the intraoperative ultrasonography immediately after shunt-attenuation (Fig 8). However, four weeks after surgery the flow-direction in the shunt was hepatofugal (Fig 5C) and the flow in the entire portal vein hepatopetal (Fig 5B). The liver was symmetric and its size was estimated to be larger than before surgery (Table 1B).
Hypoplasia of the left portal branch. Ultrasound images (A, C) and photomicrographs of biopsy specimens (B, D) of the left (A, B) and right (C, D) liver halves of a cairn terrier four weeks after a partial attenuation of a congenital extrahepatic spleno-caval shunt. Staining (B, D): haematoxylin-eosin, magnification: 20x.

A, B. Portal vein hypoplasia. Histopathologic examination of the biopsy specimen taken from the small left liver lobes reveals unrecognisable portal branches and arterial proliferation at the portal area.

C, D. Histopathologic examination of the biopsy specimen taken from the enlarged right liver lobes reveals normal hepatic architecture with normal portal area. The portal vein is well recognisable (arrow).

Four weeks postoperatively the owners of all the three dogs reported complete resolution of the clinical signs, however the blood ammonia levels were elevated. The left and right portal branches were well recognisable. Color Doppler ultrasonography revealed hepatopetal flow in the entire portal vein (Fig 5B) and in the portal branches, and hepatofugal flow in the entire shunt (Fig 5C). In addition to the functional CPSS, APSCs were seen in each of the three dogs (Fig 1).

Dog with poor outcome. In this dog (a 4-month-old Rottweiler) the results of the pre-, intra- and postoperative ultrasonographic examinations did not differ from one another (Table 1B, Fig 6A). Four weeks postoperatively the owner observed no clinical improvement, and the blood ammonia level was elevated. The whole liver was small and the left and right portal branches could not be identified. Ultrasonographic signs of portal hypertension were absent. Color Doppler ultrasonography revealed hepatofugal flow both in the shunt and in the portal vein cranial to the shunt-origin.
In the eleven dogs with spleno-caval CPSS the time-averaged mean velocity in the portal vein caudal to the shunt-origin ranged from 10.7 to 25.3 cm/s at the 4-week re-check.

**Discussion**

A partially attenuated and patent CPSS is generally assumed to be functional, therefore clinically significant,\textsuperscript{10,12,14,17} otherwise whether a CPSS is partially or completely occluded would not be an issue, and a second surgery that attempts complete occlusion of a partially attenuated shunt would not be considered. However, there is a substantial difference between the anatomy and therefore the postoperative evaluation of an intrahepatic and an extrahepatic CPSS. In cases of intrahepatic CPSSs, the shunt is the direct continuation of the trunk of the portal vein via the left or the right portal branch. Therefore, if colour Doppler ultrasonography detects blood flow via the attenuated segment of the CPSS, it means that the toxin-rich blood of the portal vein keeps shunting into the caudal vena cava, and this can indeed be responsible for hyperammonemia. In cases of extrahepatic CPSSs, however, there is a T-shaped junction at the point of the shunt-origin because the portal vein bifurcates into the shunting vessel as well as continues to the liver, moreover the splenic vein or the gastric veins are in direct connection with the shunting vessel. Therefore, if blood is seen to be shunting via the attenuated part of an extrahepatic
CPSS, it does not mean that the toxin-rich blood of the portal vein is shunting, since the shunting blood may originate from a portal tributary.

When a CPSS is completely occluded during surgery, postoperative hyperammonemia can be the result of: the formation of APSCs; a fully open CPSS due to the degradation of the suture material; or the presence of a second, previously not recognised CPSS.

When the shunt-attenuation is partial, postoperative hyperammonemia can be the result of persistent shunting via the attenuated CPSS with or without the simultaneous presence of APSCs. To differentiate these two conditions is mandatory because the presence of APSCs excludes the possibility of a further shunt-attenuation.

Progressive remission of portosystemic shunting has been documented in partially attenuated intra- and extrahepatic CPSSs, and spontaneous mechanical closure of the CPSS due to thrombosis and scar-formation was suspected to be responsible for this phenomenon. However, the present study has demonstrated that a patent extrahepatic CPSS is not necessarily functional when it diverts only the splenic venous blood, which does not contain more ammonia and other toxins than a systemic vein.

Scintigraphy and portography have been used for follow-up diagnostic imaging of portosystemic shunting. Since scintigraphy is unable to differentiate between portosystemic shunting that occurs via CPSSs and that via APSCs, this technique cannot be recommended for follow-up evaluation of attenuated CPSSs because it does not facilitate decision making about a second surgery. Angiography can differentiate whether shunting occurs via the attenuated CPSS or via APSCs, however the use of ionising radiation and anaesthesia is required. The most commonly used angiographic techniques for evaluating portosystemic shunting are intraoperative mesenteric portography and splenoportography. The advantage of intraoperative mesenteric portography is that a jejunal or the mesenteric vein is catheterised, therefore the route of the toxin-rich blood can be followed, however the procedure involves a laparotomy. In cases when mesenteric portography detects APSCs, a high anaesthetic risk was taken merely for diagnostic purposes.

Though splenoportography can be performed without a laparotomy by ultrasound-guided injection of contrast medium into a parenchymal splenic vein, the results of splenoportograms can be misleading. As color Doppler ultrasonography demonstrated in the present study, the splenic vein plays a central role in the portal hemodynamics in cases of partially attenuated extrahepatic CPSSs. This pressure balancing role of the splenic venous blood is the result of the anatomic location of the splenic vein, namely that it enters the shunt in half way between the portal vein and the caudal vena cava. In cases of a non-functional shunt, one part of the splenic blood keeps flowing through the attenuated shunt to the caudal vena cava, however the other part flows to the portal vein. Though the splenic vein belongs to the portal venous system, it does not contain more toxins than any systemic vein, therefore shunting of the splenic blood has no clinical significance. Moreover, the splenic blood that flows from the shunt to the portal vein prevents the toxin-rich mesenteric blood from shunting. Splenoportography however is unable to differentiate between shunting blood arising from the splenic or mesenteric vein. Therefore, splenoportography indicates portosystemic shunting also in cases when only the blood of the splenic vein flows through the shunt. This may result in false positive findings. This phenomenon explains the observations of Van Vechten and others, namely that despite of the evidence of shunt-patency, shunt-values determined by scintigraphy were within the reference range.

The classification of the outcome in the present study was based on the ultrasonographically detected hemodynamics. If telephone interviews or questionnaires had been used to evaluate the surgical outcome as in other studies, only the Rottweiler would
have had an unfavourable outcome. In fact, in the present study, the Rottweiler was not considered to have a completely unfavourable outcome due to the absence of APSCs development, thus further shunt-attenuation during a second surgery could still theoretically result in a favourable outcome. The outcome was considered to be fair, when APSCs had developed, since their presence indicates a form of portosystemic shunting that cannot be further corrected and may later be the cause of recurrence of the clinical signs. However, all dogs with APSCs were clinically healthy at the 4-week re-check. The present study showed that the clinical signs ceased once the blood from the portal vein reached the liver, namely hepatopetal flow was detected in the portal vein cranial to the shunt-origin, regardless of the presence of APSCs or a functional CPSS. Hepatopetal portal flow cranial to the shunt-origin means that the blood from all portal tributaries flows via the portal branches to the hepatic sinusoids. This is a great improvement of the circulation of the liver compared to the preoperative situation. Preoperatively no blood or only a portion of the blood from the gastroduodenal vein reaches the liver in dogs with an extrahepatic CPSS, and this is insufficient for normal hepatic development and function. The improved postoperative portal circulation of the liver results in enlargement of the liver lobes and the presence of flow in the right and left portal branches.

A major etiological difference in the development of APSCs was found in the three dogs that developed sustained portal hypertension in the present series. In the two dogs with a coinciding primary portal vein hypoplasia, even a temporarily applied complete shunt-occlusion was unable to change the flow-direction in the portal vein segment cranial to the shunt-origin from hepatofugal to hepatopetal during surgery. In contrast to this, in the third dog exaggerated shunt-attenuation was the probable cause of APSCs development. If a slight attenuation of a shunt results in hepatopetal flow in the portal vein segment cranial to the shunt-origin and in the shunt, further shunt-narrowing is unnecessary because it does not reduce shunting of the portal blood, but increases the level of portal hypertension, since a larger portion of the splenic venous blood is forced to flow to the narrow portal vein segment that is cranial to the shunt-origin instead of to the caudal vena cava. The absence of flow in the portal vein caudal to the shunt-origin indicates the presence of portal hypertension. Subsequently, the sustained portal hypertension caused the development of APSCs and resumption of hepatofugal flow in the shunt. If the CPSS had been attenuated to a larger diameter, then the flow-direction in the shunt may have remained hepatopetal and potentially an excellent instead of a fair outcome could have been reached. We recommend that during surgical attenuation of an extrahepatic CPSS ultrasonography should be used and the largest shunt-diameter should be found that ensures hepatopetal flow in the shunt and in the entire portal vein. Further shunt-attenuation is contraindicated because it would increase only the level of portal hypertension, but would not reduce shunting, nor improve the circulation of the liver. When the flow remains hepatofugal in the shunt and in the portal vein cranial to the shunt-origin during a temporarily applied complete shunt-occlusion, the shunt should be attenuated to the narrowest diameter that ensures continuous flow in the portal vein caudal to the shunt-origin. For an excellent outcome whether the CPSS is partially or completely occluded is irrelevant. The degree of attenuation has to be individually determined based on the presence and direction of portal and shunt flow.

In four dogs the ultrasonographic findings were different immediately after shunt-attenuation and four weeks after surgery indicating that clinically relevant hemodynamic changes can still occur postoperatively both to positive and to negative directions. However, the postoperative portal hemodynamic changes may be predicted based on the intraoperative ultrasound findings in the majority of the cases, especially in dogs with fair outcome.
In one dog with excellent outcome the flow-direction in the shunt changed from hepatofugal to hepatopetal. Local inflammation and scar formation around the ligature may have further reduced the shunt-diameter, and the resistance towards the liver may have been reduced by dilation of the small portal branches. As a result, the attenuated shunt-segment had insufficient capacity to allow the whole amount of splenic venous blood to flow through it, thus a part of it was forced to flow to the portal vein.

In two dogs with fair outcome (a cairn terrier and a miniature schnauzer) the flow-direction in the portal vein cranial to the shunt-origin changed from hepatofugal to hepatopetal in the postoperative period. Most likely, shunt-ligation resulted in the development of severe portal hypertension, and the congested portal blood caudal to the shunt-origin was forced to flow to all possible routes to reach the lower pressure systemic venous system: towards the attenuated CPSS; towards the hepatic sinusoids; and towards tiny rudimentary vessels, which eventually became APSCs.

Finally, in one dog with fair outcome (a Yorkshire terrier) the direction of flow changed in the shunt from hepatopetal to hepatofugal subsequently to surgery. Presumably the shunt-attenuation was exaggerated causing the development of severe portal hypertension. The portal vein cranial to the shunt-origin had an insufficient capacity to absorb both the blood from the splenic vein and that from the mesenteric vein. Apparently the vascular bed of the splenic vein seems to have a lower reserve capacity to dilate than that of the jejunal veins, because intraoperative ultrasonography of this dog showed continuous hepatofugal flow in the splenic vein and in the shunt, whereas no flow was detectable in the portal vein caudal to the shunt-origin immediately after shunt-attenuation. The congested portal blood was presumably forced to flow towards all possible routes to reach the lower pressure systemic veins, similarly to the previously described scenario. As a result, normal portal flow caudal to the shunt-origin was restored, but the flow in the shunt became hepatofugal, which means that the shunt became functional again, as well as APSCs developed to solve the severe portal hypertension.

Based on these observations the following conclusion can be drawn: when the flow-direction in a partially attenuated extrahepatic CPSS is hepatopetal adjacent to the portal vein 4 weeks after partial shunt-ligation, no portosystemic shunting occurs, either congenital, nor acquired.

Mean portal flow velocities measured four weeks postoperatively in all the fourteen dogs were in the reference range (18.1±7.6 cm/s) that was established in normal dogs by Nyland and Fisher. Apparently, the first priority of the portal circulation after the onset of portal hypertension due to shunt-ligation is to restore and maintain the physiological blood flow in the portal vein. In the present study portal flow velocity in the portal vein caudal to the shunt-origin did not give any additional information regarding the presence of portal hypertension or the level of portal perfusion. However, hepatopetal portal flow direction cranial to the shunt-origin at the 4-week re-check was correlated with a favourable clinical outcome. Though dogs with APSCs or with functional CPSS were clinically healthy four weeks after surgery, they might have an increased chance for recurrence of the clinical signs in the future.

Primary and secondary portal vein hypoplasia cannot be differentiated by histopathologic examination of the liver. Therefore, a possible unfavourable outcome in dogs with a coexisting primary portal vein hypoplasia cannot be predicted before surgical ligation of a CPSS because secondary portal vein hypoplasia, which is the result of hypoperfusion of the liver, is always present in dogs with CPSS. Once the portal circulation has become normal postoperatively (i.e. hepatopetal flow in the entire portal vein), the histopathologic signs of secondary portal vein hypoplasia disappear. In contrast to this,
primary portal vein hypoplasia cannot be solved by restoring normal portal flow. In two
dogs with fair outcome the evidence of a coexisting primary portal vein hypoplasia was
found both ultrasonographically and histologically at the 4-week re-check. Interestingly, in
both dogs the abnormality affected only the left portal branch. The left portal branch
supplies the larger part of the liver (four liver lobes) and the right portal branch supplies
only two liver lobes, namely the right lateral and the caudate ones. A hypoplastic right
portal branch may not have clinical significance. The reason why only the left portal branch
was hypoplastic remains obscure. Though preoperative diagnosis of coinciding primary
portal vein hypoplasia in dogs with CPSS is currently impossible, its recognition would not
change decision making about whether or not a dog with an extrahepatic CPSS should
undergo surgery. Observations from the present study suggest that every dog with a CPSS
under the age of six years should undergo surgery because even if a coexisting primary
portal vein hypoplasia is present and shunt-attenuation would cause the development of
APSCs, normal portal circulation can be restored by changing the hepatoportal or zero flow
to hepatopetal in the portal vein cranial to the shunt-origin, which subsequently ensures
normal hepatic development and function. The clinical signs of hepatic encephalopathy
usually also disappear once the flow becomes hepatopetal in the entire portal vein because
the amount of mesenteric blood that used to shunt has markedly decreased, and the APSCs
probably divert predominantly the splenic venous blood, since the spleno-renal collaterals
are the most common types of APSCs in dogs.7 The patients of the present study did not
include dogs older than six years since surgical treatment had not been recommended to the
owners if the dog at the time of the diagnosis of a CPSS was over six years of age, because
an earlier study revealed significantly worse clinical outcome in older animals.5 Since
intraoperative and postoperative Doppler ultrasonography was not part of the protocol when
the study of Wolschrijn and others5 was performed, the reasons for the less favourable
outcomes in older dogs remained unknown. A prospective study with the inclusion of older
dogs and of the use of intra- and postoperative ultrasonography should be performed to find
the portal hemodynamic reasons for the less favourable outcomes in older animals.

As a conclusion, a second surgery for further shunt-attenuation should not be
performed when APSCs are detected, or when the shunt-flow is hepatopetal adjacent to the
portal vein. However, further shunt-attenuation during a second surgery is recommended,
when the flow-direction is hepatoportal in the portal vein cranial to the shunt-origin. In
cases, when the flow is hepatopetal in the entire portal vein and hepatoportal in the shunt,
further shunt-attenuation should be considered depending on the presence of clinical signs.

In summary, ultrasonography is a reliable method to evaluate the portal
hemodynamics and to facilitate decision making about a possible surgery following partial
attenuation of a CPSS. Moreover, Doppler ultrasonography gives more information in a
non-invasive way than angiography or diagnostic laparotomy. The minimum amount of
portal hemodynamic information to be collected at a re-check ultrasound examination: the
flow direction in the portal vein cranial to the shunt-origin; the flow-direction in the shunt
adjacent to the portal vein; and the width of the left gonadal vein (indication for the
presence of APSCs).

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References
